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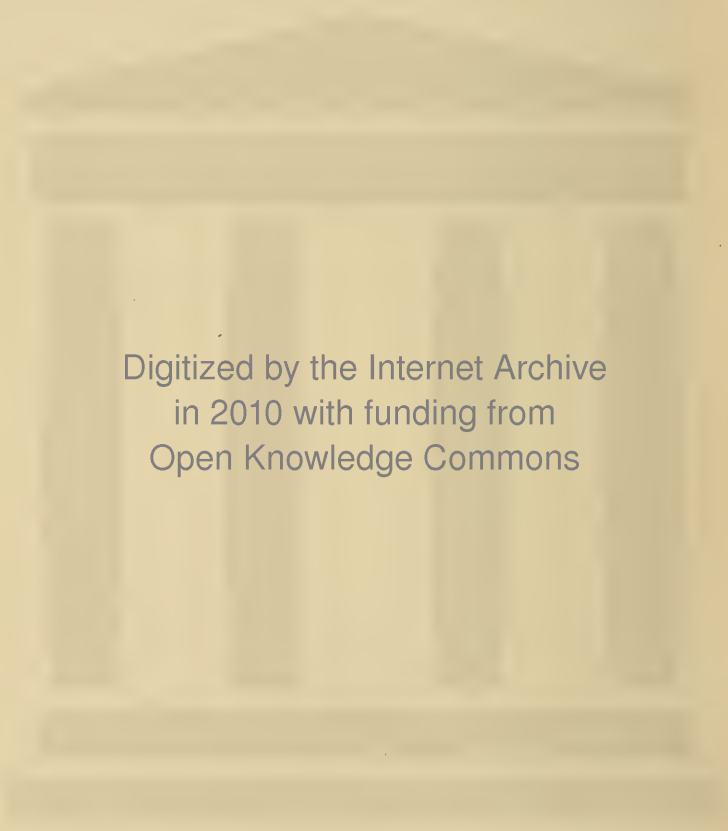
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DISEASES
OF THE
NERVOUS SYSTEM

A TEXT-BOOK OF
NEUROLOGY AND PSYCHIATRY

BY

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PREFACE.

THE diseases of the nervous system are no longer compassed by a description of the gross lesions of the brain, spinal cord, cranial and peripheral nerves. The more limited symptomatology of disorders of these structures, which in this work has been called sensori-motor neurology, has been expanded in two directions—in one by the increase in our knowledge of the historically oldest portion of the nervous system, namely, the sympathetic and autonomic (vegetative) nervous system and in the other by the increase in our knowledge of the mechanisms that operate at the psychic or mental levels.

The vegetative nervous system is in close functional relations with the endocrinous glands, and, although some of the endocrinopathies may ultimately turn out not to be exclusively nervous affections, still these organs of internal secretion are so closely related from all points of view, embryological, anatomical, physiological, pathological, and pharmacodynamic, with the vegetative nervous system that their disordered functions must needs be considered in a work dealing with the diseases of the nervous system. The symptomatology of this region constitutes the borderland of neurology and internal medicine.

At the highest level stand the mental mechanisms in which action receives a symbolic representation. Here the nervous system is also the medium through which that form of physiological or pathological activity called conduct is brought about. These mechanisms, while operating consciously, largely through the sensori-motor channels of adjustment, are also intimately related to the vegetative levels where through the emotions they act unconsciously.

The authors have kept in mind the concept of the individual as a biological unit tending by development and conduct toward certain broadly defined goals and have considered the nervous system as only a part of that larger whole. The part, however, partakes of the unity of the whole and, so far as possible, the attempt has been made to arrange the diseases of the nervous system in accord with this evolutionary concept.

For practical purposes and for the reasons stated the work has therefore been divided into three parts dealing respectively with the vegetative, the sensori-motor, and the psychic levels, the reactions in all of which come to pass through the medium of the nervous system.

Man is not only a metabolic apparatus, accurately adjusted to a marvelous efficiency through the intricacies of the vegetative neurological mechanisms, nor do his sensori-motor functions make him solely a feeling, moving animal, seeking pleasure and avoiding pain, conquering time and space by the enhancement of his sensory possibilities and the magnification of his motor powers; nor yet is he exclusively a psychical machine, which by means of a masterly symbolic handling of the vast horde of realities about him has given him almost unlimited powers. He is all three, and a neurology of today that fails to interpret nervous disturbances in terms of all three of these levels, takes too narrow a view of the function of that master spirit in evolution, the nervous system.

For these reasons the treatise has been called primarily a work on the diseases of the nervous system rather than two books, one on neurology and one on psychiatry, which would perpetuate a distinction which the authors believe to be wholly artificial.

S. E. J.
W. A. W.

NEW YORK AND WASHINGTON, 1915.

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NERVOUS AND MENTAL DISEASES.

CHAPTER I.

METHODS OF NEUROLOGICAL EXAMINATION.

THE student of nervous diseases should have at his command a practical, systematic series of methods for examining his patients. Such a scheme of case examination should above all be orderly, and sufficiently extensive to enable him to make a rapid sizing up of the character of the disturbance under investigation, and not be burdened with details for which an application will be found in only the rarest of disorders.

A comprehensive neurological history should include a careful:

1. *Family history*, especially fashioned to bring out factors of heredity and environment that might throw light upon the disorder under question.

2. *The anamnesis*, or patient's own (*a*) early history, (*b*) the history of the development of the disorder, and (*c*) the general neurological and general clinical examination.

For the sake of convenience, the chief factors to be covered are here given in the form of a printed questionnaire. Such printed schemes have their advantages and their disadvantages. But if carefully and thoughtfully followed the advantages far outweigh the disadvantages. For the beginner in neurology, to whom this book is addressed especially, such a questionnaire, well grounded in the mind, is the first step in the development of a technique of case examination which will prove invaluable. It is not thought that another form may not prove as satisfactory, but some definite scheme is indispensable.

Questionnaire.—Naturally the heading, size, form of binding, of ruling, etc., will be determined by local conditions and by special needs. The definite facts which are noted in the questionnaire are not by any means all of the facts to be collected, and such a printed question blank is given purely as a guide, rather than as a finished product. As a practical blank for out-patient dispensary work it is invaluable, and also for note-taking in private work. In hospital work with nervous disorders, where it is hoped that autopsy material may put opportunities for anatomical research in one's hands, such a

blank is naturally too didactic and inelastic. Its main outlines, however, are still desirable.

The initial facts are the name, in full, maiden name in married women, age, residence, social status, married, single, widow, divorced, and race. In countries where immigration is active, it is desirable to know not only the birthplace of a patient, but also his racial stock.

Family History.—It is desirable, in studying mental disorders particularly, to get a family history as well as an objective history, both from the patient and from other members of the family.

The first factor that needs to be investigated is that of heredity. Viewed in the usual loose light in which it is regarded, the facts gathered concerning heredity are usually worthless; they are worse, they are misleading and pernicious. Better no facts concerning the extremely complicated question of heredity than false ones. In ordinary investigation the research need not be exhaustive. A complete consideration of heredity is best left to special eugenic students, whose business it

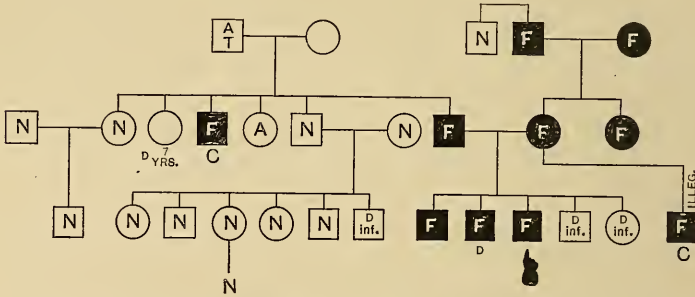


FIG. 1.—Pedigree chart illustrating how two defective parents may have only defective children. A, alcoholic; C, criminalistic; D, infant, died in infancy; F, feeble-minded; N, normal; T, tuberculous. Goddard, 1910. (Taken from Davenport.)

has become to properly collate and weigh the mass of material needed to bring out salient factors in the heredity problem. The main facts noted in the questionnaire are sufficient, save in the consideration of certain disorders in which definite heredity factors are known, such as Huntington's chorea, Thomsen's disease, diabetes insipidus, deaf-mutism, certain optic atrophies, etc.

Consanguinity (Fig. 1) in the parents is first to be inquired into; the degree of relationship should be explicitly expressed.

Consanguinity may or may not have any relation to nervous disorders; it much depends on the stock. Bad factors present in one or another may be absolutely missed in the descendants, for Mendelian laws seem fairly well established. With the presence of dominant neuropathic traits in both ascendants, the chances are less hopeful in the descendants—but not hopeless, since recessive characters are passed on as well. In human fecundity but few of the ova are impregnated, and each ovum is undoubtedly very different. The chance

No.

Diagnosis: *Tabes.*

NAME, *J. Jones.* Age, *42* Occup.: *Clerk. M.S.W.[D.]*
Residence, *444 Spring St.* Race, *U. S. In U. S. 42 yr.*
Date, *Jan. 5, 1910.* Examined by *J.*

Heredity: Parents related: *0*
F., *d. 70; apoplexy.* Mental: *0*
M., *d. 64; cancer.* Nervous: *0*
Children: *Only child.* Epilepsy: *0*
TBC: *0* Diabetes: *0*
Alcohol: *0* Syph.: *0*
Birth: *Normal.* Eruptions: *?*
Walk: *N.* Speak: *N.* Read: *6 years.*
Children's Diseases: *Measles.*
Enuresis: *0* Thumb: *0* Nail-biting, etc.: *0*
Education: *Pub. Sch., High Sch., to 17 years.*
Adult Diseases:
Syphilis: *26.* Treatment: *1 month.*
Shocks: *0* Internal: *Hg.*
Habits: Alc.: *+* Tob.: *++*
Sex: *Moderate.*
Trauma: *0*
Occupation Toxemias: *0*
Convulsions (injury, tongue, urine): *0*
Constitution: *Healthy.* Weight: *150.*
Marriage: *At 32.*
Menses:
Children: *1; d. in infancy (convulsions).*
Mis.: *2; 4 mos.* Dead: *0*

History:

Chief complaint: Slight unsteadiness in gait and severe "rheumatic" neuralgic pains in lower extremity for past four years. Five years ago had a transitory attack of dizziness, with double vision and an eye palsy, cross eye, which lasted two months.

Occasional weakness in bladder, dribbling. Paræsthesiæ occasional, etc.

First page of Questionnaire: Made out from examination of a patient with *Tabes.*

STATUS NERVOSUM

Cranium: Hor., N. Bin., N. Vert., N.
 Sensibility: *O. K.* Deformity: *0*
 Asymmetry: *0*
 I. Smell: {*Ol. ter. O. K.*
 {*Asafet. O. K.*
 II. Sight: {*R. 20/40*
 {*L. 20/100*
 Hemianopsia: *0* Scotomata: *0*
 Fundus: *0* Fields: *Limited; con.*
 III. IV. VI. Eye movements: *O. K.*
 Nystagmus: *0* Palp. fiss.: *R=L.*
 Diplopia: *0; 5 years ago.*
 Pupils: *R > L, 9 and 4 mm.*
 Reflexes: {*Light: Lost L; dim. R.*
 {*Accom.: O. K.*
 {*Symph.: Dim. R > L.*
 {*Consensual: Lost L+R.*
 V. Motor: *O. K.*
 Sensory: *O. K.* Jaw jerk: *O. K.*
 Tender spots: *0* Cornea: *O. K.*
 Conjunctiva: *O. K.*
 VII. At rest: *O. K. R=L.*
 Forehead: *O. K.* Eyes closed: *O. K.*
 Teeth: *O. K.* Whistle: *O. K.*
 Involuntary: *O. K.* Rosenbach: *O. K.*
 VIII. Hearing: *Dim.* Equilibrium: *O. K.*
 Rinné: + Weber: +
 Tinnitus: *0*
 IX. X. XI. XII. Fauces: *O. K.*
 Speech: *O. K.* Reflexes: + *O. K.*
 Tongue: *Straight.* Swallow: *O. K.*
 Scars: *0* Tremor: *0*
 Cardiac: *O. K.* Respiratory: *O. K.*
 Taste: *O. K.*
 Shoulders: *O. K.* Neck: *O. K.*

History—Continued

STATUS NERVOSUM

Upper extremity:

Atrophy: *O*

Hypertrophy: *O*

Spasm: *O*

Hypotonus: ++ Twitching: *O*

Muscular power: *Dim.* *R=L.*

Dynanom.:

Nerve trunks: *Not tender.*

Triceps Rx.: *Dim.* Radix Rx.: *Dim.*

Tremor: *O* Rest: *O* Static: *O*

Ataxia: + F.N.T.: *Atax.* F.F.T.: *Atax.*

Stereognosis: *O. K.*

Light touch: *O. K.* Adiadokok: *O*

Position: *O. K.* Diapason: *Dim.*

Pain: *O. K.* Thermal: *O. K.*

Vasomotor: *O* Trophic: *O*

Trunk:

Power: *O. K.* Deformity: *O*

Spine: *O. K.*

Epigastric Rx.: + Abdom. Rx.: +

Cremaster Rx.: + Anal. Rx.: +

Bladder: *Sluggish.* Rectum: *Sluggish.*

Light touch: *O. K.* Localization: *O. K.*

Deep Sens.: *Dim.* Diapason: *Dim.*

Pain: *O. K.* Thermal: *O. K.*

Equilib: *Unsteady.* Asynergia: +. *Slight.*

Vasomotor: *O* Trophic:

Dermographia: *O*

History—Continued

STATUS NERVOSUM

Lower extremity:

Atrophy: +; *legs flabby.*

Hypertrophy: 0

Spasm: 0 Kernig: 0

Hypotonus: + + Tremor: 0

Muscular power: *Diminished.*

Synergistic tests: *R=L.*

Patellar: 0 Achilles: 0 Clonus: 0

Babinski: 0 Chad: 0 Opp: 0

Nerve: *Tender.* Lasègue: *Tender.*

L. touch: *Dim.* Pain: *O.K.*

Deep: *Dim.* Therm: *O.K.*

Localization: *O.K.* Position: *Impaired.*

Diapason: *Impaired.*

K. H. T.: *Atax.* Romberg: + + +

Gait: *Markedly ataxic.*

Closed eyes: *Made worse.*

Asynergia: *O.K.*

Vasomotor: 0 Trophic: 0

Status corporis: *Medium nutrition.*

Heart: *O.K.* Murmurs: 0

Blood: *O.K.* Wassermann: + +

Lungs: *O.K.* Arteries: *O.K.*

Abdomen: *O.K.* Blood-pressure: 160.

Liver: *O.K.* Skin: *O.K.*

Urine: *O.K.* Joints: *O.K.*

Cerebrosp. fluid:

Wass.: + + Cells: 50. Globulin: + +

Treatment:

Intraspinous injections
of salvarsanized serum
(Swift-Ellis). Course of
mercurial inunctions.

element seems too great to estimate. One feature of consanguinity not to be overlooked is that many relatives (first cousins, second cousins) marry each other because of a neuropathic tendency. It is not impossible then that the evil results of consanguinity sometimes seen is a direct transmission of the neuropathic traits that were primarily responsible for the marriage. At all events the studies of Woods,¹ Cox,² Lundborg,³ Punnett,⁴ Bateson,⁵ Karplus,⁶ and the numerous papers of Karl Pearson,⁷ all give hope of new and more definite outlooks in the study of the relationship of heredity to disease in general, and of disorders of the nervous system in particular.

Abraham⁵ and others have given some suggestive discussions concerning the tendency for related neurotics to marry.

Mental Disorder.—The presence of mental disorder in the direct ancestry is of moment. The evidence drawn from aunts and uncles is of value, while that from cousins is open to the influence of another stock. It is useless to record the fact "insane" without any definite

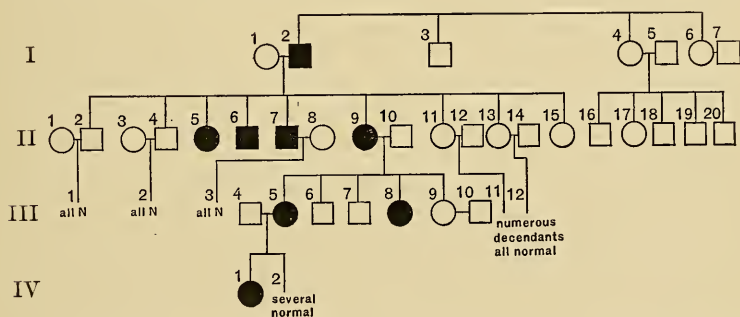


FIG. 2.—Pedigree of a family showing Huntington's chorea. Affected persons (indicated by black symbols) are always derived from affected parents. From original data furnished by Dr. S. E. Jelliffe; Smi family. (Taken from Davenport.)

knowledge of the particular mental disturbance. It is important to reiterate that the old Morel, Zeller view, that all mental disturbances are one disease with different stages is absurd. Insanity as a disease entity has no existence. One should be as specific as possible, for there is a distinct tendency for special types to be passed down as such, a fact that only the more fundamental conceptions of Kraepelin have permitted to be verified. Mental peculiarities should be noted: great avarice, originality, queerness, eccentricities, great capability, marked incapacities, temperamental peculiarities, inability of husband

¹ Heredity in Royalty, 1906.

² Psychiatrischen Neurologische Bladen, 1907.

³ Ueber Degeneration, 1901.

⁴ Mendelism in Relation to Disease.

⁵ Brain, 1906.

⁶ Zur Kenntniss der Variabilität und Vererbung am Zentralnervensystem, 1907.

⁷ Biometrika.

⁸ Die Stellung der Verwandtenehe in der Psychologie der Neurosen, Jahrbuch f. Psychoan. u. Psychopath. Forschungen, 1909.

and wife to get along, suicides (valuable as evidence only if cause be known), all these may be of considerable service in offering a clue to many difficult cases, in psychoneuroses and psychoses particularly. The occurrence of the hysterical reaction in the parents, or in the brothers or sisters should not be overlooked, but deductions therefrom should be founded on precise criteria, not haphazard guessing.

Definite *nervous disorders* that need investigation in the ancestry are, so far as known, but comparatively few. The more important are ties, myoclonias, Huntington's chorea, Thomsen's disease, myopathies, certain congenital brain defects, as cerebellar defects, speech defects, skin defects, optic defects (color blindness). Careful history taking will undoubtedly reveal other defect neuroses, for the very fundamental study of Karplus (loc. cit.) has shown how brain form, brain cytotectonic, cord form, cord myelotectonic, etc., are directly transmitted. Certain occupation neuroses, some migraines, many eye strains, etc., are possibly due to the continuous transmission of structural variations.¹

Epilepsy.—The occurrence of epilepsy as epilepsy means little. Epilepsy, like many another disease so-called, is resolvable into many different factors. The convulsive seizure is only an end result of a vast variety of antecedent events concerning but few of which can one posit any hereditary factors.

Alcoholism.—Alcoholism, if excessive, should never be overlooked. If possible the cause should be recorded, for in the last analysis alcoholism is to be regarded as the individual's attempt to escape certain difficulties. Alcoholism may mean hysterical or compulsive reactions. Periodicity in drinking should be carefully inquired into, particularly in its relation to the cyclothymic constitution.

Migraine.—The hereditary factor in migraine is much exaggerated. The extreme frequency of the disease has served to bring about this confusion.²

Tuberculosis and Diabetes.—Tuberculosis, diabetes, tendency to arthritic disturbances are factors in heredity, the exact significance of which it is hard to estimate; tuberculosis especially, because of its extreme frequency. The presence of diabetes in a parent should not be overlooked. It seems to play a large role in nervous and mental disorders.

Syphilis.—Syphilis as an antecedent factor should never be disregarded. Not only does one meet with congenital tabes, paresis, hydrocephalus, optic atrophies, deafness, etc., but evidence is accumulating that reinforces the belief that a syphilitic heritage is responsible for much nervous and mental disorder, of a less tragic though perhaps more annoying character than those first mentioned. Objective evidences of a syphilitic inheritance should be looked for. Teeth, bony

¹ Compare Adler, Die Mindewertigkeit der Organen.

² See chapter on Migraine in Osler, Modern Medicine, 1915, second edition, vol. 6.

formation, pelvic, chest, and cranial contours, etc. Wassermann tests of the blood of suspected parents may even be necessary to clear up a diagnosis, say of a meningeal disturbance of hidden etiology in an infant, child, or even young adult. The evidence of congenital Wassermann reactions is still too undecided at this date to permit one to be satisfied with the results, especially if negative, obtained from the blood of the patient.

The objective examination passes on to the *birth* of the patient. Was it normal, or instrumental, or of excessive length? Hemorrhage, accidents, or pressure palsies may thus receive their interpretation.

Did the child learn to walk at a normal period, *i. e.*, from nine to fifteen months (Preyer), and if not were there definite facts—excessive weight, intercurrent disease—to explain the tardiness? Was speech

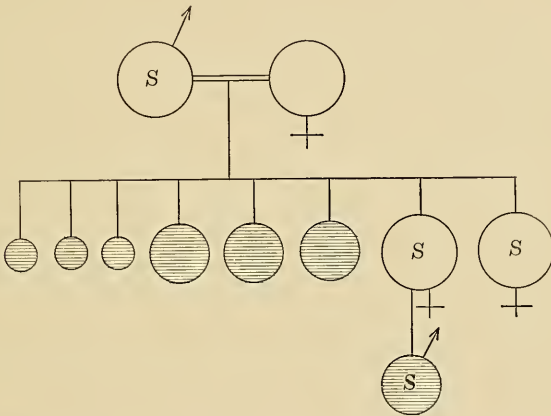


FIG. 3.—Congenital syphilis “unto the third generation.” Syphilitic father of first generation. Second, three premature births, two dead children, and one dying at sixteen months. Congenital syphilitic next, with Hutchinson triad. This patient married, and had a child with snuffles, who died at age of six weeks. Remaining sister had infantile hemiplegia, Hutchinson teeth, and keratitis. (Mott.)

acquired early or late? and did the child learn to read at a normal period (five to eight years, according to opportunities afforded)? These facts are of much importance, especially in estimating mental capacity, and for young children probabilities of development and the need for special training. Children who learn to walk and talk as late as twenty-six to thirty-eight months respectively often remain very backward. The relations of speech to mental development are extremely close. Special tests as the Binet-Simon scale are valuable in placing the intellectual age of the child. (See Chapter on Mental Examination.)

Difficulties in teething, especially when attended with convulsions, should be noted. The rachitic chest form should not be overlooked.

Concerning children’s diseases, much can be said. Severe measles,

or scarlet fever, or other disorders may lay the foundation of a later developing epilepsy; diphtheria may cause various forms of neuritis with, at times, permanently weakened powers. Tuberculosis plays a very important role. The importance of influenza is often overlooked, while the relation of the acute streptococcic infections and their resulting joint involvements to chorea is well established. Excessive fatigue, or exhaustion, either from an infectious disease, apart from its toxemia, or from over-exercise or strain, in rapidly growing children bears a close relation also to choreic reactions.

Very close attention should be paid to the aural affections of children, and the nasopharyngeal cavities scrutinized for adenoids, or other foreign bodies that interfere with free respiration, sound sleep or the proper hygiene of the nasopharyngeal mucous membranes.

Intestinal worms should not be overlooked. They may be the cause of infantile convulsions or of milder neurotic disturbances.

The occurrence of vertigo should be inquired into.

Enuresis should never be overlooked. If continuing past the third or fourth year it affords valuable evidence of a neurotic predisposition. Thumb-sucking, nail-biting, and other little habits may be included in this place. Stammering should be carefully inquired into.

The education of the patient, especially if a mental disorder or a psychoneurosis is under investigation, should be very thoroughly gone into. A knowledge of the earliest impressions gained, the picture books used, the principal childish associations formed, special tastes, animosities or dislikes are essential to the understanding of the neurasthenic, hysterical or allied reactions. The ideals inculcated, the religious and ethical training gained in the early years usually give a definite stamp to the personality and must be known if the adult personality is to be understood. The grosser factors of the classes passed, and the schooling received are absolutely essential in estimating the grade of later mental capacity, and the application of intelligence tests in the study of the psychoses or psychoneuroses.

Adult Diseases.—Syphilis stands in the first rank. Care should be exercised in obtaining a syphilitic history. Did you ever have a chancre? is the usual method of asking the question. The query, How old were you when you had a sore on the penis? although perhaps more abrupt, will give a higher percentage of positive answers, especially in those cases where its previous existence is largely inferred, as in general paresis or tabes. If the direct question is to be avoided, as in the case of many women, married or otherwise, the questions concerning syphilitic symptoms are desirable. The presence of symptoms of continuous sore throat, hair-falling, etc. A physical examination for mucous plaques, leukoplakia of the mouth, and scars on the penis should never be omitted. In women the mucous membranes of the cheeks and sides of the tongue should always be examined.

Further, the Wassermann blood reaction should be obtained in all cases where any doubt exists. Although the percentage of unknown

syphilitic infections is low, nevertheless such exist. A single Wassermann test, positive or negative, is not conclusive, and great care should be taken in the choosing of a proper serologist. The subject of technic is a complicated and immensely important one.¹

The subsequent history of the syphilitic infection is desirable and the character and length of time of treatment should be recorded.

Gonorrhea is not unimportant. It is of special relevancy in all arthritic disorders, in choreas (vaginal discharge) in young children, and in meningeal excitements. Gonorrheal neuritis is known.

Arthritis in its various forms calls for careful observation. Here one would best record observations, and not attempt a diagnosis of the joint conditions.

The role of the infections in mental pathology is very marked. Typhoid fever and influenza both constitute severe infections with marked influence on nervous tissues.

Under Shocks is included sudden mental and moral influences, tending to disturb the emotional life. Loss of money, of parents, husband, children, or loved ones, interference with one's hopes of a career, unfortunate entanglements, all call for investigation. The great importance of emotional disturbance in all nervous reactions should be borne constantly in mind.

As to habits, particular attention should be directed to alcoholism. It plays a most important role in diseases of the nervous system. There is much divergence of opinion as to what may constitute alcoholism. Accurate recording of the exact amounts consumed will afford the student the best criteria by which he later can judge for himself from his own carefully kept records. Other narcotics, as opium and its allies, cocaine, the alcohol hypnotics, bromides, etc., call for record.

Occupation toxemias should not be overlooked. They are daily assuming increased importance in America by reason of the increasing number of dangerous occupations. Workers in lead, arsenic, mercury, copper, pewter, pottery, dyes, sulphur compounds, and others suffer often from obscure symptoms, due to chronic poisonings. Occupation fatigues explain many neurasthenias.

The sexual habits should be inquired into. Liberal indulgence in masturbation, etc., while usually self-corrective, at times works havoc with the nervous system. Sexual repression in the married as well as the unmarried is at times an important element in the neuroses (anxiety neurosis). The rarer anomalous sexual impulses call for investigation in some cases.

Special attention should be paid to convulsive seizures, either occurring in the young or in adult life. Certain facts about such convulsions should never be omitted. These are the presence of dizziness, or of objects revolving (direction to be noted), of the state

¹ Plaut. Wassermann Reaction in Neurology and Psychiatry, Nervous and Mental Disease Monograph Series, No. 5, New York, 1910; Kaplan, Serology in Nervous Diseases, Philadelphia, 1914.

of consciousness; whether there is injury to the body during such attacks; if the tongue be injured; if urine or feces be voided during the attack, and if there be amnesia, complete or partial, following the attack.

The influence of violence or injury to the body is often of extreme importance. If there is accompanying mental shock the fact should not be omitted.

The general constitution of the patient—his or her general capacity for work and fatigue—is to be noted. The question of general temperament, of outlook on life, may be tentatively entered in this place.

In the case of women, special attention should be directed to the menstrual history. The number and character of the births, the health of the children, the number of miscarriages with causes should be recorded.

Subjective History.—The patient's own account of his illness can either precede or follow the outlines of the family history and his general previous condition.

It is advisable for the patient to fix as nearly as possible the date of the onset of his illness. Certain facts which may or may not have had any connection with the malady under study may aid in fixing such a period of transition between health and sickness. The nature of its onset, whether acute and progressive, or acute and regressive, insidious and irregular, or very severe and immediate. What did the patient notice at that time? Then gradually trace, step by step, hour by hour, day by day, week by week, or year by year, the development of the disorder. What new symptoms have been added to the first—what have disappeared; has the picture remained the same, or has it gradually or suddenly altered?

A methodical going over the locomotor, sensory, emotional, intellectual, skin, digestive, respiratory, and secretory systems should follow the patient's own account of the difficulty. Pains felt should be roughly charted and carefully located. If certain symptoms have disappeared, attention should be paid to the mode of their onset and of their departure. It is important to ascertain just what effect the illness has upon the social, familial, physical or psychical life in order to estimate the severity of certain symptoms. Also, has the patient consulted other physicians, or has he visited institutions or other than trained medical men of various kinds? What was the treatment? Its effect?

A thorough subjective anamnesis is one of the most difficult, and at the same time most important features of a neurological or mental examination, particularly the latter. If the gradual evolution of the disorder is carefully elucidated, there is little danger of going wrong. If one hops from one thing to another, however, mistakes will be frequent.

The Objective Examination.—During the subjective examination many facts concerning the general attitude of the patient have been

gained. His expression and carriage, in bed or able to be about. The character of his intelligence and the responsibility of his answers. Is his mind clear and is he oriented?

One soon determines whether one has a purely mental case to deal with, a definite neurological picture, or a borderland or combined condition. The systematic neurological examination is here outlined first; a systematic mental examination is given in a following section.

As has already been noted, a scheme is of great advantage, to beginners particularly. With increasing experience, one may depart from a hard-and-fast method of case-taking, but in the beginning the student should habituate himself to a rigid and exhaustive method if he would avoid careless work.

The cranium should first be inspected. Is it regularly shaped or is there assymetry? Measurements of the vertical, binauricular, and horizontal diameters should be taken.

The position, general form, and character of the ears should be noted, and the arch of the palate observed. The occurrence of isolated, or even several, so-called signs of degeneration may be recorded. There is no necessary connection between such anomalies and nervous or mental disease. They are found in superior as well as inferior deviates. The departure from the average is worthy of record, but the hasty generalizations of the Lombroso school should be avoided. These deviations from the average structures will be discussed later.

Careful and thorough percussion of the skull may reveal local points of tenderness (brain tumor), etc. The presence of cicatrices (epilepsies), depressions (fractures), or abnormal elevations is to be noted. In special cases, x-ray examination of the skull is of great value, and should always be made for suspected fractures, for many brain tumors (acromegaly), etc.

The development of the two sides of the face is to be compared, the width of the nostrils noted, and particular attention given to the character, texture, and color of the hair, and skin of the face and mucous membranes.

Cranial Nerves.—These should be systematically tested. I. *Smell.*—There are no satisfactory quantitative tests for smell apart from special physiological psychological tests that are of service in research work only. The smell in each nostril can be tested separately, preferably by some well-known substance (oil of turpentine) and by a substance resembling well-known foods (asafetida, onions). The nostril of one side is stopped, and with the eyes closed the patient is asked to smell from a bottle; the other side is then tested with the same or a different substance and comparisons are made. Variations in smell are very common, and too much weight is not to be given to smell tests. Influenza interferes with smell tests. Irritating substances, like ammonia, etc., should *not* be used. A search for subjective smell disturbances may be made at the same time. Local conditions, empyema of the antrum, etc., must be excluded. Anosmia,

unilateral or bilateral, is often present in fractures of the skull and in brain tumors.

II. *Eyes.*—Sight should be tested by the Snellen or other type cards. Myopias and astigmatisms are important to bear in mind in testing

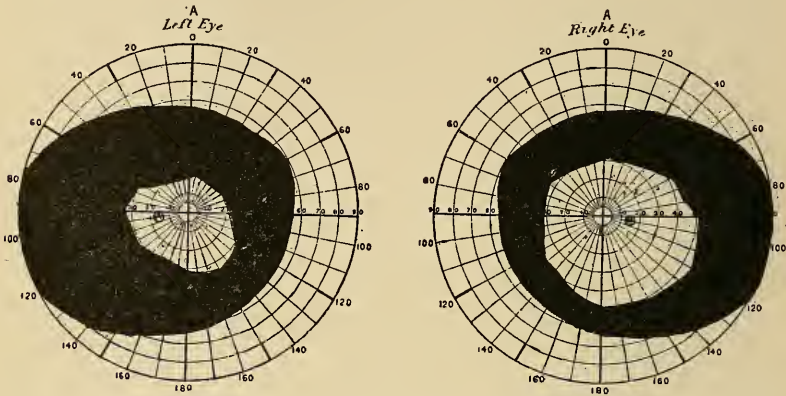


FIG. 4.—Crossed amblyopia in a case of hysteria. (Stewart.)

the sight functions. Hemianopsia should always be searched for. It may be done rapidly by use of the usual finger sight test and if there are any anomalies careful chartings by a perimeter should be made. Scotomata are sought for in the same manner.

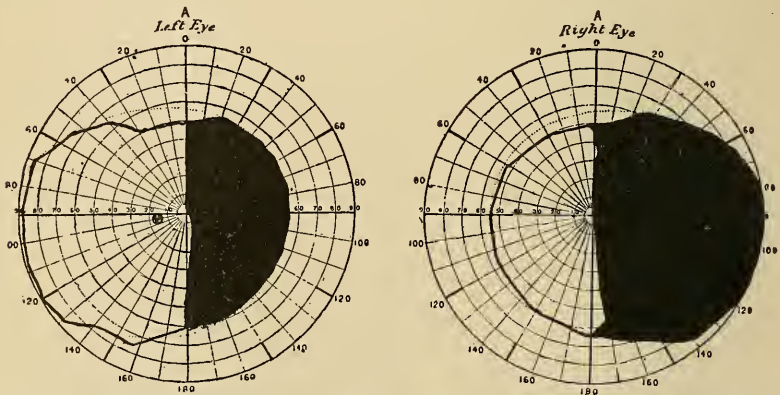


FIG. 5.—Right homonymous hemianopsia in a case of softening of the left occipital lobe.

Color vision is important. Colored wools in the full daylight are matched as to shades. In color-blindness, if of the red-green variety, gray- or straw-colored wools are selected. In total color-blindness the individual confuses all colors: comparative degrees of

brightness alone are distinguished. A number of ridiculous blunders may be made in testing for color-blindness with the wool-tests.

The field of vision should be tested with avoidance of suggestive factors. This may be done with a perimeter, which frequently introduces the error of suggestion, or the rapid finger test employed. The patient sits about three feet from the examiner. Each eye is tested separately. The patient puts his hand over his left eye, and looks fixedly at the examiner's left eye, the right eye being closed. With the left hand held midway between patient and examiner some distance to one side, it is then gradually brought toward the middle line, the fingers being waved slightly. The patient is directed to say "now" as soon as he catches sight of the slightly waving fingers. Four axes should be tested. Variations from the examiner's own fields can be noted. A square of white paper (1 cm.), on a gray card, may be used instead of the fingers. For a general test of the color fields squares of different colors may be used. The most striking features to be sought for are hemianopsia, temporal or nasal; concentric limitations, irregular limitations, quadrant hemianopsias, psychic blindness.

In all cases the fundus should be examined. A knowledge of the eye-ground changes is essential for good neurological investigation. Works on ophthalmology must be consulted for the many anomalies, but the most important to be observed are signs of pressure, of atrophy, of retinal hemorrhage or congestion, and irregular pallors (temporal pallor in multiple sclerosis), edema, choroiditis, retinitis, venous congestion, arterial occlusions, etc. Optic atrophy may exist without blindness.

Third, Fourth, and Sixth Cranial Nerves.—The functions of the third, fourth, and sixth cranial nerves are conveniently tested, first together and later separately. Ability to move both eyes outwardly (external rectus) indicates integrity of the sixth cranial nerve. If the eyes can be moved freely, amply and equally upward, downward, and inward, the third and fourth cranial nerves are usually intact.

Having seen if the eyes move freely in all direction, nystagmus is tested for under the same conditions. It consists of a slow movement of the bulb in one direction, with a rapid jerk back in the opposite direction. Notes on the direction of the slow and rapid movements should be made. They are of great value in determining labyrinthine and cerebellar nystagmus. Nystagmus may be present on central fixation, or only become apparent as the eyes are directed to one side. Slight tremors of the globes on extreme lateral position may be of small diagnostic importance. Nystagmus should be tested for in the vertical and horizontal and also in oblique axes. Rotatory nystagmus may be looked for.

In the presence of a nystagmus, certain supplementary tests are advisable. The most important are the turning stool, and hot and cold water tests. In the former the patient is seated on a revolving

office chair, and turned to the right or left ten revolutions, with moderate rapidity. On arresting the revolutions careful note should be made of the direction of the nystagmus in each eye, and also the length of time it persists. The head may be held erect, and bent forward or backward to test each semicircular canal.

Hot and cold water is syringed gently into the external auditory meatus of each ear. A large bulb syringe is used. Cold causes a definite rotatory nystagmus toward the ear not irrigated. The patient also has vertigo and marked disturbance of equilibrium. If hot water be used the quick nystagmic movement will be toward the ear irrigated, and the ataxia is different. The presence of labyrinthine trouble or cerebellar disorder involving the vestibular apparatus cause modifications in the character of this nystagmus (see Vestibular).

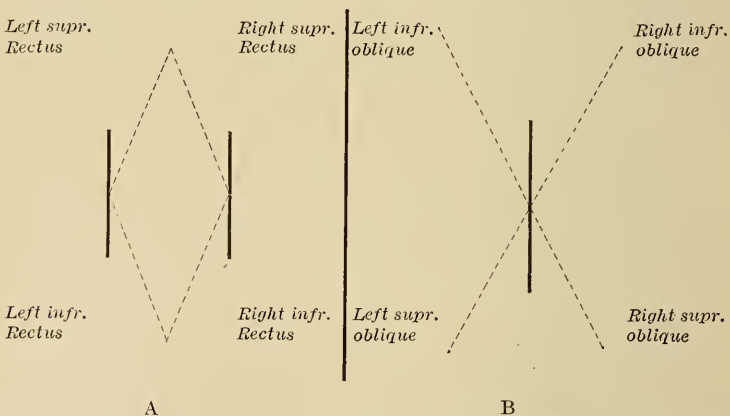


FIG. 6.—Werner's "artificial memory" for the double images in ocular paralyses (Ophthalmic Review, 1886). *A*, shows the position of the images in paralysis of the recti muscles; *B*, in paralysis of the oblique muscles. The dotted lines indicate "false" images, the thick black lines "true" images. (Stewart.)

Diplopia is next tested for. The patient should be asked if he has ever seen double and a single light, or one finger held to the right or left, up or down, used to test if double vision exists. Should it be present the position of the images in relation to one another should be noted; whether they separate or approach as the candle is farther or nearer, and a red glass should be placed before one or the other eye to determine the location of the images, and their relative position.

The accompanying schemes are of value in memorizing the muscles involved (Fig. 6):

Monocular diplopia, seeing double with one eye, is occasionally met with. It is due to gross corneal or eye defects, occasionally in central scotomata, but usually it is a product of suggestion in hysteria.

Pupils.—The size should be compared and noted. $r = 1 : r > 1, 1 > r$, and a rough measure given, 2, 3, 4 mm., as the case may be; mydriasis,

myosis. The form and the presence of irregularities, oval, polygonal, and implantation noted. Particular attention should be directed to the elimination of changes due to drugs, to accidents or injuries, and to inflammatory products.

The reactions to light should then be tested, at first with clear daylight, the patient looking at some distant object. In bed cases, electric hand lamps are very serviceable; with these the light is best directed somewhat obliquely. The reaction may be prompt and wide,

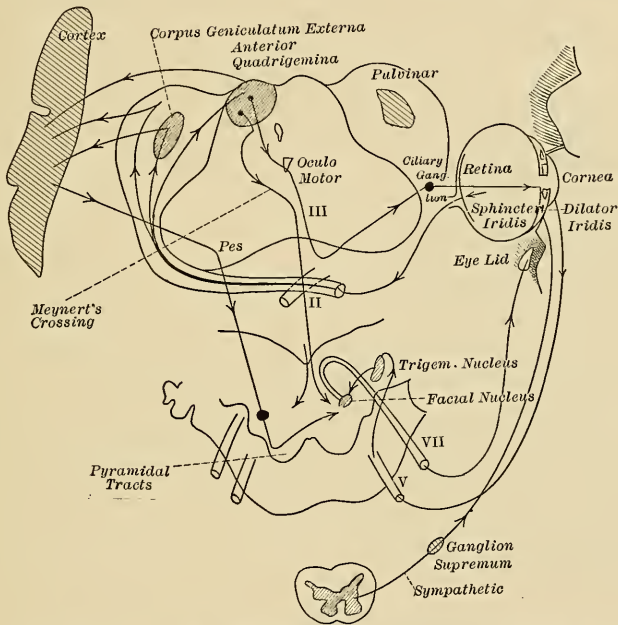


FIG. 7.—The paths of the chief eye reflexes: (1) Pupillary reflex: Retina; optic; X; corpora quadrigemina; X; oculomotor nucleus, oculomotor nerve; ciliary ganglion; sphincter iridis. (2) Closing eyes to light: Retina; optic; X; corpora quadrigemina, Meynert's fibers; X; facial nucleus; facial nerve; lid muscles. (3) Winking on approach of object: Retina; optic; X; corpora quadrigemina or pulvinar, external geniculate; cortex; pyramidal tract; X; facial nucleus; facial nerve; lid. (Same paths also in 2.) (4) Corneal reflex: Cornea; trigeminus; trigem. nucleus; facial nucleus; facial nerve; lid. (Lewandowsky: Funktionen d. Centralnervensystem, p. 122, Fig. 29.)

prompt and restricted, slow, sluggish, diminished or absent. Each eye is to be tested separately, and then the consensual test applied by illuminating one eye and noting the reaction in the other.

Wernicke's hemiopic phenomenon should be sought for in hemianopsias. In this the pupil does not react if the light falls upon the blind segment of the retina.

Tests for the accommodation reflexes are then made. The patient looks at the finger as it is moved near or away from the eye, and there is corresponding contraction and dilatation of the pupils. Here the

reaction may be prompt or slow, with slight or marked amplitude. In blind people the thought of looking at their own nose and across the room may bring out the reaction. The existence of a lost or partially lost light reflex, with unimpaired accommodation reflex, is known as the Argyll-Robertson pupil. It may be present in one or both eyes, and may be partial or complete. It is due to a dissociation of the sensorimotor reactions, and may be present in a variety of disorders, although it is most frequently found in syphilitic disorders (Fig. 7).

The mechanism is variously, although not entirely, satisfactorily explained, because of the complexity of the afferent and efferent fiber tracts and their connections (Lewandowsky scheme). Marina's hypothesis of its peripheral origin (disease of ciliary ganglion) explains most of the tabetic and paretic cases, but does not explain some of the traumatic or mesencephalic cases. Cajal's scheme is as follows:

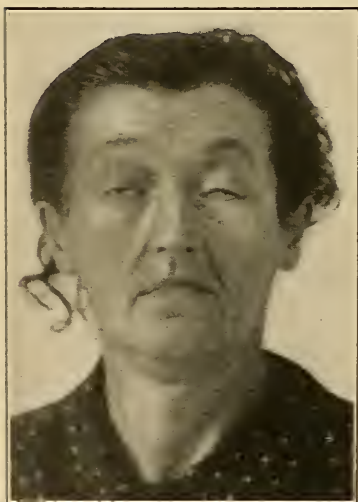


FIG. 8.—Tabes with bilateral ptosis.
Operated upon to hold eyelid open.

1. Retinal neuron with its optic fibers ramifying in the anterior corpora quadrigemina.

2. The mesencephalic neuron with its axis-cylinder formation of the posterior commissure.

3. The neuron of the interstitial nucleus of the tegmentum (calotte) with its collaterals destined to the motor nuclei.

4. The neuron of the bulbar nuclei of the oculomotor communis et externus and of the patheticus with cylinder axes going to the muscles of the eye.

The sympathetic reflex is tested by pinching the skin of the cheek or some other part of the body. The pupils dilate under the influence of painful stimuli.

Ophthalmoplegia externa is the name given to a paralysis of the external eye muscles; ophthalmoplegia interna to those of the pupil, which is widely dilated and immobile to light and convergence. Complete ophthalmoplegia is found when all of the pupillary phenomena are absent and there is loss of all eye movements with ptosis.

Ptosis consists in a drooping of the upper lid, paralysis of levator palpebræ, which is supplied by filaments from the third nerve. Henneberg's reflex, consisting in a spastic action contraction of the orbicularis oris when the hard palate is stroked, may be mentioned with the reflexes of the cranial nerves.

The Fifth (Trigeminus) Nerve.—The motor functions of the fifth nerve are tested by having the patient move his jaw to the right and

left. The examiner's hand, exerting contrary pressure, can determine differences in innervation (external pterygoid, temporals). The finger wrapped in a towel can be used to bite upon to determine variations in bite (masseter, temporal). The state of hardness of the temporals and masseter muscles can be directly palpated. The jaw should be protruded (internal pterygoid).

The use of a tuning fork of low pitch is useful in determining loss of the function of the tensor tympani muscle.

In one sided paralysis of the motor fibers of the fifth nerve, the opened jaw deviates to the paralyzed side by the action of the sound external pterygoid. The patient chews on the sound side. It may or may not be accompanied by sensory changes. Elevation of the eyelid on strong biting is a frequent associated movement.

The jaw reflex may be tested at the same time. With the mouth partly open a pencil or flat object is placed upon the teeth and lightly tapped with the hammer. There is a quick contraction of the masseters and temporals, and usually an associated movement of closing the eyelids.

The *sensory* functions of the fifth nerve demand very careful testing by reason of its wide distribution.

The supra-orbital, infra-orbital and mental points should first be pressed upon to determine the degree of sensitiveness. Then the palpebral, conjunctival, and corneal reflexes should be tested. This is best done with a long pin with a globular glass head. With the patient looking away from the examiner, the palpebral margin is touched with the head of the pin, then the conjunctiva, and finally the choroid, and running along the globe over the cornea the effect is noted. Both eyes should be compared and the tear secretion noted.

The amount of tear secretion may be measured by hanging two small strips of litmus paper on each lower lid, by bending in the paper at the top so as to make a small ledge to hang. The rate of moistening of the two sides will show quantitative variations in the amounts secreted.

The ordinary sensibility should be tested first with a camel's-hair brush—the two sides compared. Then with the point and head of a very sharp pin; then the skin should be pinched on each side and differences noted. Deep pressure over the malar, frontal, and jaw bones made to determine deep pressure sense, and the use of hot and cold test-tubes to learn if variations in thermal sense exist. Finally a slowly vibrating diapason should be applied to the jaw bone to determine its bony sensibility.

The interior of the mouth and the surface of the tongue should not be neglected in these tests, and special care should be taken in outlining the ear areas, and changes within the auditory canal.

Careful inspection of the teeth should not be omitted, and anomalies of dentition carefully noted. The two sides of the bony structures

of the face should be compared for bony atrophies (hemiatrophy) or hypertrophies (acromegaly).

The Seventh (Facial) Nerve.—The seventh (facial) nerve is a mixed nerve. The state of the musculature at rest should first be recorded. The folds and the angles of the mouth and muscle-twitching carefully noted. The patient is then asked to wrinkle his forehead (frown),

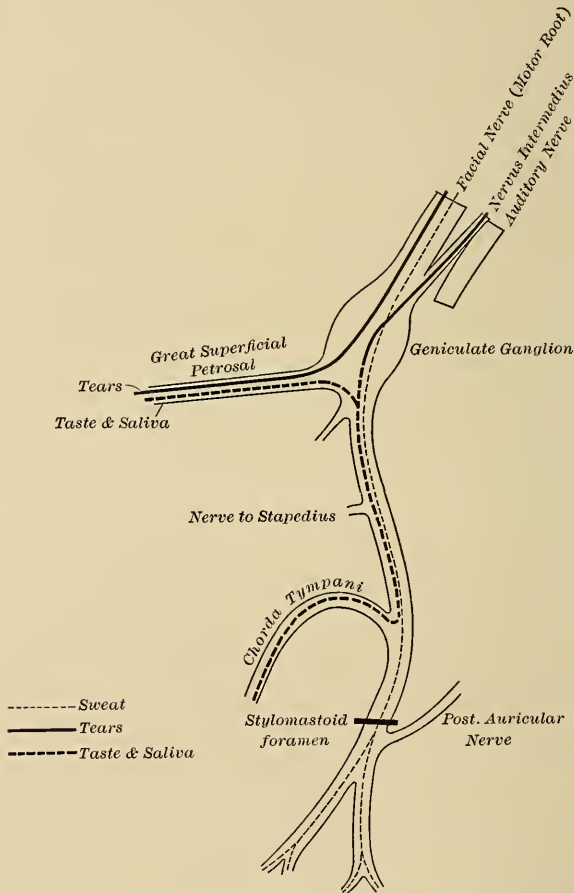


FIG. 9.—Diagram of facial nerve, showing course of secretory and of taste fibers. (Stewart.)

raise the eyebrows, close his eyes tight, show the teeth, whistle, and puff out the cheeks. In this way the musculature of the two sides can be compared. A mild joke will bring out the emotional contractions which are equally important to note. Some individuals can move their ears.

Not only should the presence of paralysis or paresis be carefully

noted, but spasms observed under voluntary and emotional reaction. Their degree, character, and intensity should be carefully recorded.

Sensory, secretory, and motor functions are to be tested, and many differences are to be noted in the distribution of the palsies according to the location of the lesion of this nerve: (1) after its exit from the stylomastoid foramen, (2) within the Fallopian aqueduct, (3) between its emergence from the pons and the geniculate, or (4) within the pons (See Fig. 9).

The Eighth (Cochlear and Vestibular) Nerves.—Here two entirely different nerves with absolutely different functions need to be tested. They are the cochlear nerves (hearing) and the vestibular nerves (equilibrium).

Hearing is roughly tested most satisfactorily by speech. Having first ascertained that the auditory canals are free from wax, the examiner, some six to ten feet away, pronounces three numbers, asking the patient, who has one ear closed, to repeat them after him: the voice is then raised or lowered, and the distance varied to determine the hearing capacity. The opposite ear is tested in the same manner and comparisons made. A watch-tick or tuning-fork may also be used. Careful tests with tuning-forks and whistles are needed in complicated cases.

Bony conduction should also be tested. This is done by putting the vibrating tuning-fork over the mastoid, and the patient indicates when he no longer hears it, the fork is then brought to the auditory meatus to test the air conduction. Rinné's test is positive, *i. e.*, air conduction present after loss of bone conduction is the normal formula: the negative Rinné indicates middle-ear disorder. The tuning-fork on the center of the forehead is heard in both ears equally under normal conditions (Weber). In middle-ear affections it may be heard unequally on the two sides. Positive Weber (*i. e.*, louder on affected side) with negative Rinné is largely indicative of middle-ear disorder. Deafness due to central disorder is usually associated with other localizing signs, yet it may be an isolated phenomenon of beginning tumor, encephalitic process, tabes, multiple sclerosis, etc.

Forks of very slow vibration are of value in determining the functional capacity of the stapedius muscles. Tests with continuous tone series following Bezold's methods are indicated in all complicated cases, since defects in lower or in higher tone perception usually indicate a difference in the site of the lesion.

Certain patients show hyperacusis, tinnitus. Ringing in the ears is largely a subjective sign. It is an evidence usually of middle-ear or of cochlear irritation. The sounds vary greatly. They may be beating, buzzing, or whistling, and may at times be the point of departure of illusions, or even hallucinations. The pulsating types of tinnitus are usually associated with the heart beat. They are found in certain tumors, in aneurysms, or in anxiety states with cardiac irregularities. Continuous tinnitus, low pitched or high pitched, is

the more common. The effects upon the tinnitus of lying down and also the effects of certain drugs, amyl nitrite, etc., are of service in differentiating the causes and probable site of a tinnitus.

Vertigo.—This belongs largely to the symptomatology of the vestibular apparatus. Equilibration is an extremely complex adjustment involving the comparative integrity of a large number of neurons from the periphery to the coordinating centers, now fairly satisfactorily proved to reside in the cerebellum. The vestibular apparatus is the chief ganglion of the cranial end of this whole apparatus or system termed by Sherrington the proprioceptive system. The cerebellum is its chief center.

Tests for Equilibration.—The more standard equilibration tests are the Romberg, and the Babinski asynergic tests. The Romberg test consists in having the patient stand erect with closed eyes, with heels and toes together. Under normal conditions there should be only a very slight swaying, but the person with well-marked Romberg sways sideways, or backward, or forward, or may even fall if the feet are not put apart. Slight degrees of Romberg, or unilateral localization of Romberg, may be brought out by having the patient stand on one or the other foot. Other observations of the Romberg can be made by having the patient bend forward or backward or sideways, and in this way quantitative suggestions may be received as to the localization of the fiber tracts involved.

In certain cerebellar disturbances there is a special loss of equilibrium which Babinski has termed asynergia. This may be tested in a number of different ways. The more valuable are by means of walking. The patient usually has a great deal of ataxia and walks with difficulty. If supported on either side, it is noticed that in walking he shows a marked tendency to walk from underneath himself, so that his legs finally are put out far in advance of his center of gravity. The same type of loss of cerebellar equilibrium may be demonstrated by having the patient stand erect and then slowly bend backward, making an *arc de cercle*. In the normal condition, the muscular adaptation bends the knees forward and the individual assumes a well-balanced position; but in the asynergic, the legs are held straight, the trunk is bent backward, and the patient has a tendency to fall backward.

For the upper extremities a similar type of muscular incoördination shows itself in the typical *diadokokinesis* tests. These tests are made by having the patient perform certain minute complicated movements very rapidly, such as quickly pronating and supinating the hand in a semiflexed position, or making rapid piano-playing movements, or sudden flexions and extensions of the arm on the forearm. In involvements of certain of the cerebellar tracts usually associated with equilibrium there is a marked inability to perform certain of these movements; they are done clumsily, irregularly and slowly, depending on the grade of sensory tract involvement.

Similar tests consist in having the patient reproduce on a piece of

paper single lines with a lead-pencil, when it will be found that they almost invariably overrun.

For testing the trunk the patient should be on the back, with the arms folded, and with the legs drawn up in the obstetrical position. Here considerable variation will be found. The normal individual, if on a fairly hard foundation, can equilibrate fairly well; there is but very little swaying, while the ataxic patient sways considerably. Cerebellar patients also, and certain types of frontal cases, show what is known as cataleptic rigidity in this position. After swaying for a certain length of time the legs and body steadily become fixed with sufficient rigidity to enable them to be photographed. This sign of cataleptic rigidity is considered by Babinski as characteristic of cerebellar defect.

In further testing equilibrium the gait occupies an important place. The patient should be directed to walk along a crack, or a straight line, first with the eyes open; he should be asked to suddenly reverse, inquiry being made if there are symptoms of vertigo in the reversion either to the right or the left, for both sides should be tested, and then the patient should be directed to return with his eyes closed when, if an abnormal equilibrium exists, it will become intensified, and staggering to the right or to the left, forward or backward, will be noted (latero-pulsion, propulsion, or retropulsion).

The subjective sense of giddiness is often extremely complicated. Certain patients complain of objects turning about them, in which case the direction of the moving object, in terms of the hands of a clock, should always be noted. Association of giddiness or vertigo with eye symptoms is very widespread.

The Barany tests by heat and cold, by compressed air, by the revolving chair, have already been mentioned in the consideration of nystagmus, and need not be repeated here. They are primarily tests for the labyrinthine function, the nystagmus being only an accessory phenomenon.

Examination of the Pharynx.—Careful note should be made of the position of the fauces while at rest and during phonation, and of the movements of the soft palate during phonation. The pharyngeal reflexes are tested by touching successively the right and left pillar, and the vomiting reflex is best tested by irritating the back of the pharynx with a wooden spatula.

While under normal conditions the uvula should hang in the middle line, a certain amount of variation in position is very frequently observed.

While the pharynx is being examined, note should be made of the pharyngeal vault, and also of the contour of the posterior pharyngeal walls. Further, the integrity of the superior pharyngeal muscles should be tested by having the patient swallow, and noting whether the act is coördinated, or whether there is regurgitation through the nose.

Examination of Taste.—Taste is a complex function and utilizes at least three different nerves. It is best tested by solutions which are sweet (sugar), bitter (quinine), acid (vinegar), salty (salt solutions). The solutions should be kept in wide-mouthed bottles, and are applied in small quantities by means of a glass rod applied to different parts of the tongue to determine the functional capacity of these parts. Small amounts should be used, and it is best to reserve the bitter test until the last, commencing with the sweet, and following with the sour, the salt, and the bitter. The mouth should be washed out between the tests with the different substances if careful records are to be made.

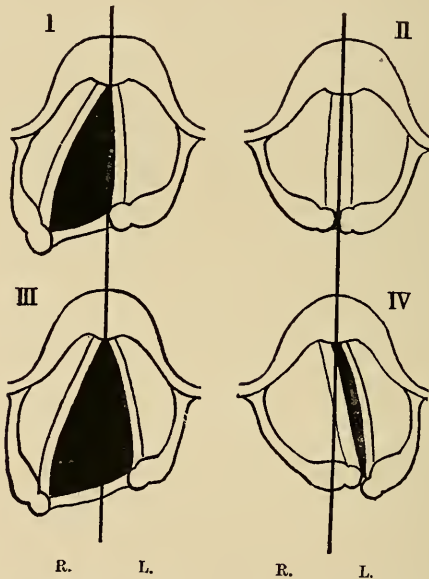


FIG. 10.—Organic laryngeal paralyses. (Barwell.) *I*, left abductor paralysis during inspiration; *II*, left abductor paralysis during phonation; *III*, left recurrent laryngeal paralysis, during inspiration; *IV*, left recurrent laryngeal paralysis, during phonation.

Taste can also be tested by means of a minimal galvanic current when the positive pole will produce an acid reaction, the negative pole an alkaline or salty one.

While testing for taste the condition of the secretion of the buccal mucous membrane can be tested. This is best done by rubbing the floor of the mouth with a glass rod and noting the rapidity of excretion and the amount.

Examination of the Larynx.—This naturally has to be carried out by means of a laryngological mirror, when mal-position or changed position of the vocal cords during inspiration, expiration and phonation are observed. Fig. 10 shows the position of the vocal cords in four characteristic palsies.

Examination of the Speech.—A complete analysis of the function of language will not be entered into in this place, since its modifications are best considered under the general head of aphasia. The chief points of neurological interest to be observed are whether the tongue is protruded in the middle line, whether it is freely movable, up, down, right, and left, and can be made to push out both cheeks. Careful search should be made for scars on the tongue, and the presence of a leukoplakia on the sides of the tongue or of the mucous membrane of the cheeks should not be overlooked.

Tremors of the tongue may be very fine, involving the whole organ, or may be fibrillary or coarse and irregular. Considerable attention should be devoted to the search for tongue tremors.

In testing ordinary speech, certain test phrases are advisable. The patient should be directed to repeat the alphabet, and the numbers up to twenty-five, and should repeat something well known, such as the Lord's Prayer, or some bit of poetry, and during the repetition careful attention should be directed to the enunciation of the individual letters, to the presence of stumbling on words, of running words together, to the omissions of words, or the omission of syllables, and particularly to the repetition and the displacement of syllables. In order to bring out some of these defects, certain test words are utilized. Among the most valuable are the following: Truly Rural; Third Riding Artillery Brigade; Methodist Episcopal; National Intelligencer. Naturally the type of case will suggest certain defects, which can then be seized upon by the examiner, and the speech defects, when present, brought out more clearly.

In stating the speech defects due to laryngeal loss, special attention should be directed to the presence of cough, of stridor, and to the pitch of the breathing.

Stuttering is a spasmodic form of speech disturbance which calls for special mention. Certain patients show a very marked slowing of speech (bradyllalia), while others show an intermittent enunciation, and still others a peculiar, monotonous, semi-sing-song type of enunciation known as "scanning speech." In complicated speech disturbances due to involvement of the hypoglossal nerve one obtains the so-called bulbar thick speech: the patient speaks as though he had a hot potato in his mouth. Further, in extensive speech disturbances due to coexisting lesions in different parts of the speech mechanism one has other disturbances known as anarthria, or more particularly as dysarthria, or "jumbled speech." The subject of aphasic speech will be discussed later.

The Tenth Nerve.—The study of the heart action is referred to the chapter on the Vegetative Nervous System.

The Eleventh Nerve or Spinal Accessory.—This nerve supplies the sternomastoid and the trapezius. Its functional capacity is tested by the ability to raise the shoulders and to turn the neck, pressure being made on the chin in resistance. There are a number of normal

displacements resulting from paralysis of this nerve which will be discussed later under the head of Paralysis.

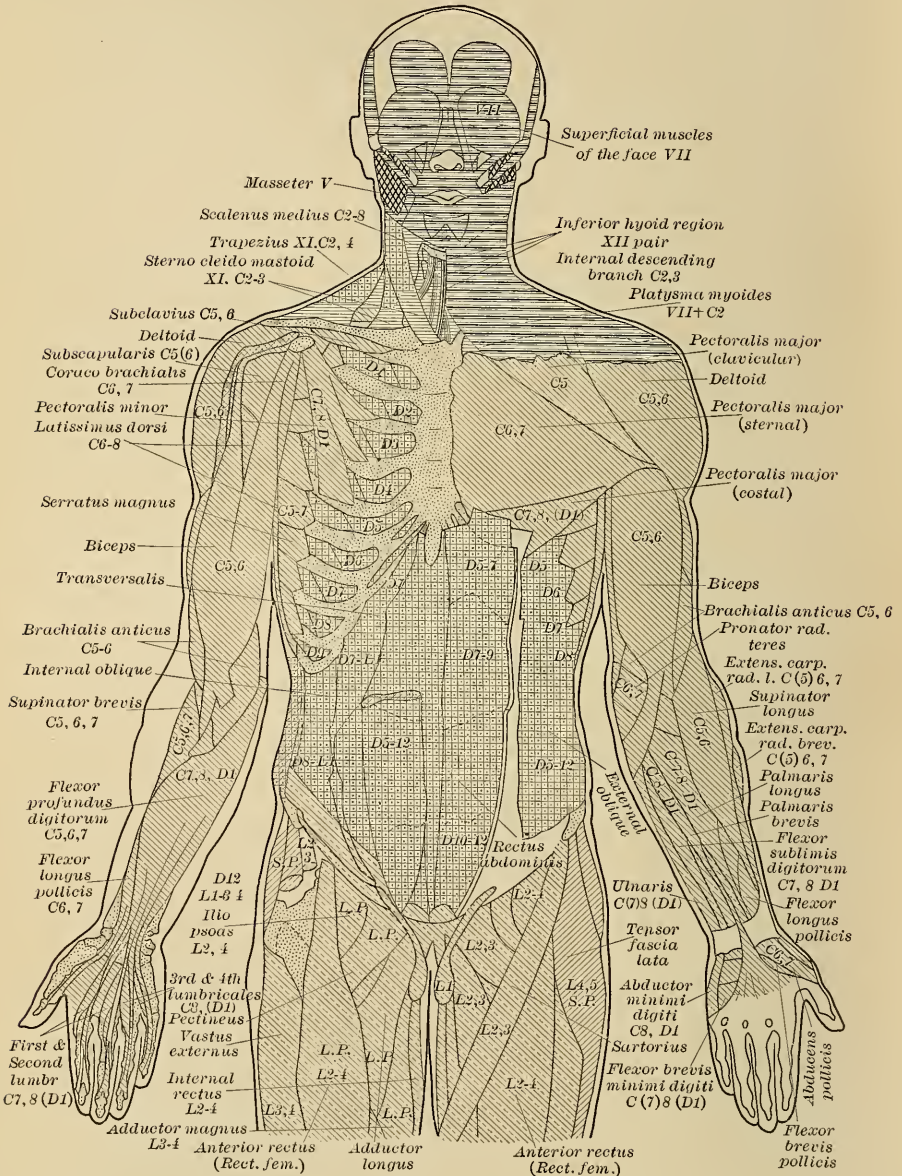


FIG. 11.—Radicular innervation of the muscles of the anterior regions of the head, neck, trunk, and upper extremities. On the left, the superficial muscular layer; on the right, the deep muscular layer. (After Dejerine.) C, cervical; D, dorsal; L, lumbar.

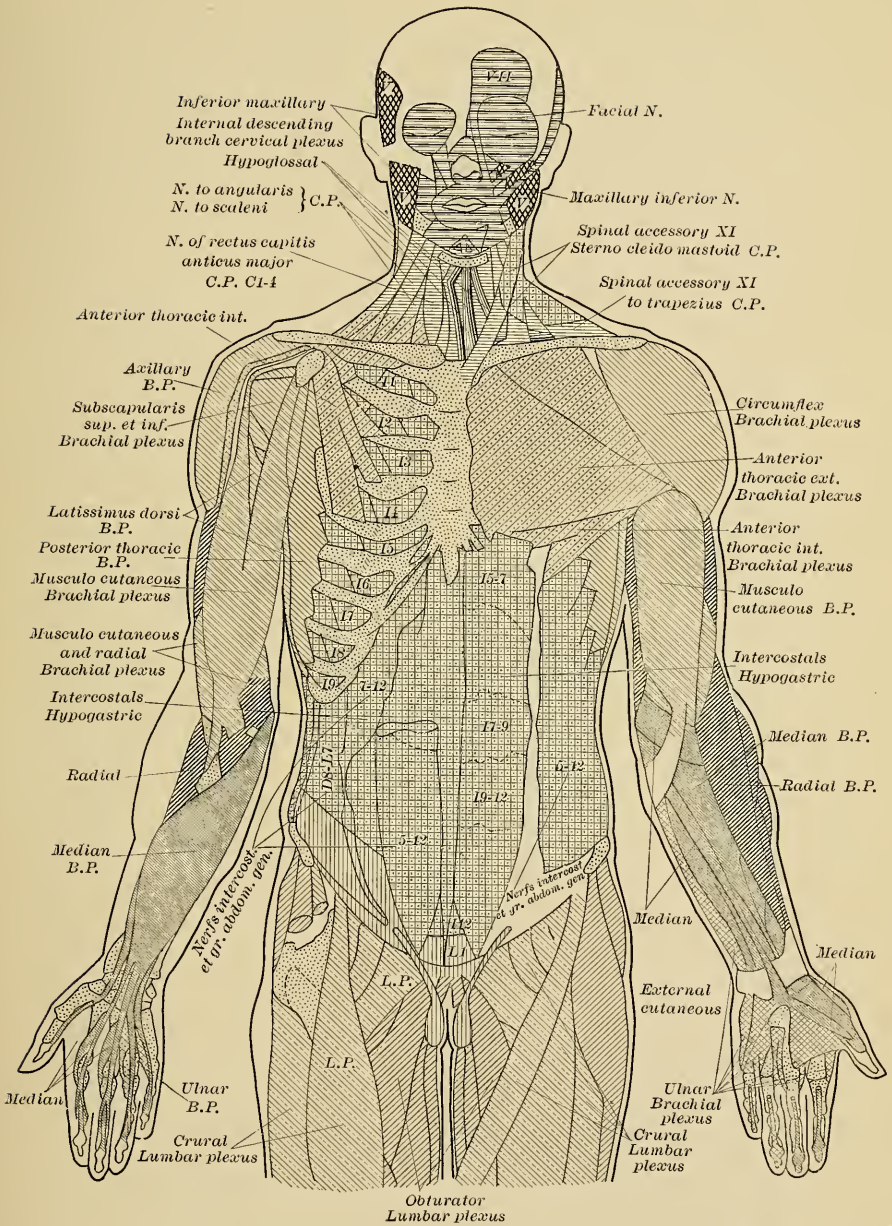


FIG. 12.—Peripheral innervation of the anterior muscles of the head, neck, trunk, and upper extremities. To the left the superficial muscular layer; the deep muscular layer to the right. The same markings on this and the preceding figure are followed for the facial, trigeminal, spinal accessory, hypoglossal, cervical plexus, and intercostal nerves respectively. Different shadings differentiate different innervations in the brachial plexus. (After Dejerine.) The chief plexuses are indicated. C.P., cervical plexus; B.P., brachial plexus; L.P., lumbar plexus; S.P., sacral plexus; D. in Fig. 11 indicates dorsal roots; I.X. in Fig. 12, intercostal nerves.

Ninth, Tenth, and Twelfth Nerves.—The glossopharyngeal, vagus, and hypoglossal nerves may be considered more or less together in their testing. The glossopharyngeal is involved in the function of taste of the posterior third of the tongue and of the soft palate. It is also a nerve of common sensation for the back of the tongue, part of the soft palate, and upper part of the pharynx. It supplies the middle constrictor of the pharynx and the stylopharyngeus with motor fibers.

For further details of the testing of the other nerves see the chapter on Cranial Nerves.

Upper Extremities.—A systematic examination of the upper extremities is next in order, the muscular apparatus first claiming attention. The two sides of the body should be examined systematically. (See Figs. 11 to 14.)

Anomalies of structure should first be noted, such as habit or occupation positions, alterations of posture, etc. Gross differences in the size of the bones, the wrists, hands, etc., should be measured.

Atrophy.—This may be determined by simple palpation and by measurement. After natural differences in the muscular volume are taken into consideration, striking variations should be carefully measured. The circumferences over the biceps and just below the elbows on the two sides should be compared. Special individual muscular atrophies may be picked out later by means of electrical reaction tests. Atrophies of the nails, skin, or hairy structures can be recorded here, or under the heading Neurotrophic Disorders, which appears later in the questionnaire.

Hypertrophy.—This is best brought out by palpation and by measurement. Due weight should be given to natural variations—right and left sides—and to the influence of certain occupations—blacksmiths, iron-workers, etc.

Hypotonus.—This is indicated by unusual flaccidity of the musculature and movements. Sudden pronation or supination of the arm, extension or flexion at the shoulder-, wrist- or elbow-joints, may show sudden sharp resistances, followed by marked flaccidity. Ability to overextension, etc., is a sign of hypotonus.

Spasm.—This indicates hypertonus. When permanently present, contractures result. The particular muscles which may show hypertonus, or spasticity or contractures, should be recorded. In certain spastic conditions the hypertonus may be relieved by passive movements. Such should be sought for.

Muscular Power.—This is first tested by having the patient execute all the chief movements of the shoulders and arms. The chief tests are as follows:

Shoulder, Arm, Hand, and Fingers.—*Deltoid.*—Request the patient to raise the arms laterally to a horizontal position. Inability so to do indicates deltoid paralysis.

Pectoral Muscles.—Stretch out the arms straight in front and then approximate the hands against resistance by the examiner, meanwhile watching both heads of the pectoral muscle.

Latissimus Dorsi.—Raise the arms laterally to a level, then, while keeping them fully extended, bring the arms downward and backward, as if to make the hands meet behind the sacrum. The examiner standing behind the patient resists the movement.

Serratus Magnus.—Desire the patient to push with his hands against those of the examiner or against a solid object. If the serratus has lost its power the scapula will project and the digitations of the muscle, which ordinarily should be visible, will not be seen.

Trapezius.—Ask the patient to raise the shoulders as close to his ears as possible against the pressure of the examiner's hands. This will demonstrate the strength of the upper part of the trapezius. The middle and lower portions are tested by desiring him to bring the scapulæ as close together as possible.

It is hardly possible to detect paralysis of the levator anguli scapulæ and rhomboids unless the trapezius is also involved.

Biceps.—Let the patient flex his extended arm, his elbow resting in the observer's left hand, while the latter's right hand, grasping the wrist of the patient, offers the necessary resistance. Also supinate the hand against resistance.

Triceps.—The triceps may be tested as is the biceps, excepting that the previously flexed arm is to be extended against resistance.

Supinator Longus.—Test as for the biceps, except that the hand should be midway between supination and pronation. If the muscle is paralyzed it will fail to become conspicuous on the radial side of the upper part of the forearm.

Flexors of the Wrist.—Grasping the patient's hand, the palm being upward, desire him to bend the hand up toward his forearm against resistance.

Extensors of the Wrist.—The patient's hand being held palm downward, he is required to bend it backward against resistance. Moderate weakness of the extensors of the wrist may be manifested by asking him to squeeze the examiner's hand, in which case the wrist will become involuntarily flexed, the weakened extensors being unable to counteract the flexors. Marked or complete paralysis of the extensors is wrist-drop.

Flexors of the Fingers.—Because of the usual difference in the strength of the two hands the examiner should cross his forearms and place his right hand in the right hand of the patient, and *vice versa*. Then let the patient squeeze the hands. If the observer keeps his own fingers extended and bunched loosely together he will be able to withstand a very hearty grasp without discomfort.

Adductor Pollicis.—Ask the patient to pinch with his thumb and finger one of the examiner's fingers.

Opponens Pollicis.—Desire the patient to approximate the ends of the little finger and the thumb—while thus approximated the examiner pulls his finger through.

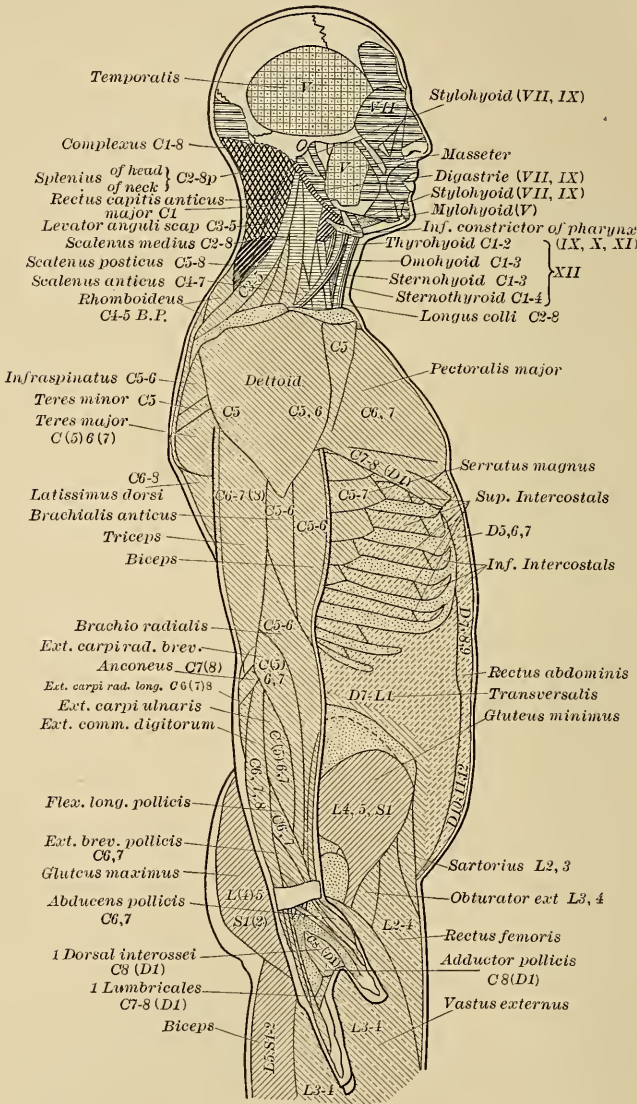


FIG. 13.—Radicular innervation of the lateral aspect of head, neck, trunk, and upper extremity. The platysma myoides, sternocleidomastoid, trapezius, and oblique muscles of the abdomen, gluteus medius, and tensor of the fascia lata have been removed. The shading and cross-hatching follow the muscles and innervation as in Figs. 11 and 12. (After Dejerine.)

The *interossei* and *lumbricales* muscles of the hand flex the proximal phalanges, and extend the middle and terminal phalanges. The dorsal

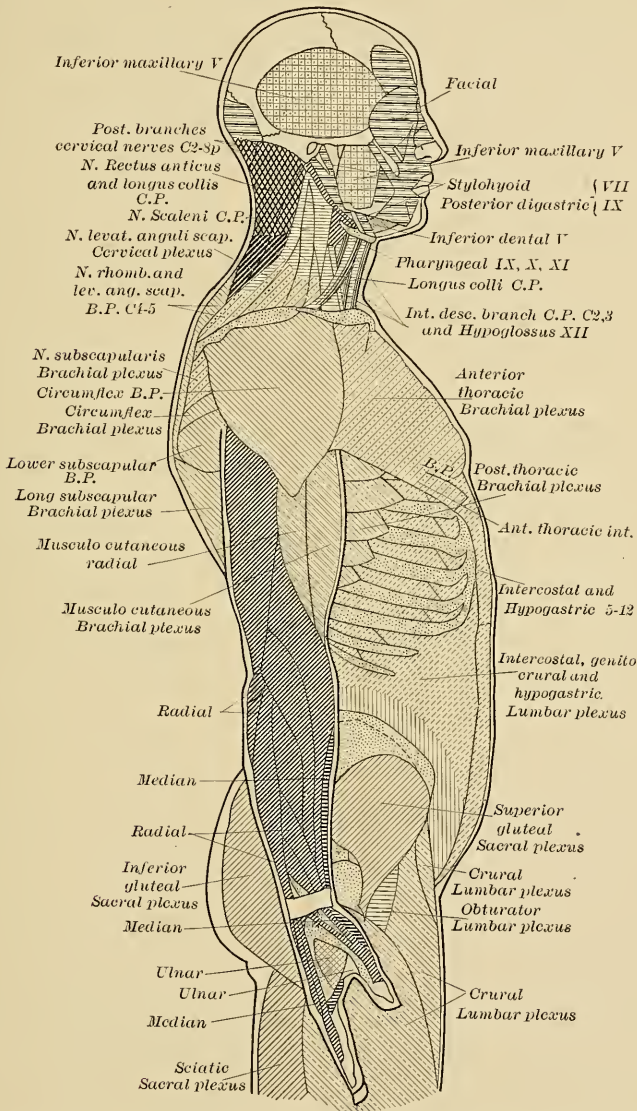


FIG. 14.—Peripheral innervation of the lateral aspect of the head, neck, trunk, and upper extremity. Markings and cross-hatchings, as in previous figure, to indicate the various innervations. Figures C.D.L.P., C.P., B.P., L.P., and S.P. as in previous diagrams. (After Dejerine.)

interossei adduct, the palmar adduct, the fingers from and toward a longitudinal line drawn through the center of the middle finger.

Test by making the patient separate and approximate the fingers, and flex the proximal phalanges, keeping the middle and terminal phalanges extended. Paralysis of these muscles causes "claw-hand."

The strength of the hand grasp is well tested by the dynamometer. Readings of three tests for each hand should be recorded. Dynamometer records are available for definite comparisons. Further, the dynamometer is useful by many repeated readings (20) for obtaining an idea of the fatiguability of muscles (neurasthenia, myasthenia, alterations in attention, etc.). Weiler has constructed a useful dynamometer with a graphic register.

The Muscles of the Trunk.—The erector muscles of the spine are examined by causing the patient to lie face downward and asking him to raise the head and shoulders without assistance from the hands. Unless paralyzed the erectors become clearly visible during the attempt. The abdominal muscles are tested in a similar manner, except that the patient lies in the dorsal position while making an effort to raise the head.

The Lower Extremities.—*The muscles of the lower extremities* are best tested with the patient in the lying position (see Figs. 11 to 16):

Flexors of the Thigh.—The patient lying upon his back, ask him to raise the leg from the bed, against resistance, the knee being kept straight. This determines the strength mainly of the ileopsoas, partly of the quadriceps.

Extensors of Thigh.—The leg being kept straight and the patient lying upon his back, raise the foot and ask him to bring it down upon the bed against resistance. This determines the strength of the gluteus maximus and partly of the hamstring muscles.

Abductors of Thigh.—With the leg across the middle line ask the patient to carry it toward the outer side against resistance, thus testing mainly the gluteus medius.

Inrotators of Thigh.—With the patient on his back, flex the knee to a right angle, grasp the foot, and oppose resistance while he inrotates the thigh, testing mainly the gluteus minimus.

Outrotators of the Thigh.—Similarly test the power of outrotation, thus determining the condition of the obturators, pyriformis, gemelli, and quadratus femoris.

Flexors of the Knee.—The patient lying upon his back, desire him to bend the knee while the examiner resists the movement by grasping the ankle, thus ascertaining the power of the biceps, semimembranosus, and semitendinosus.

Extensors of the Knee.—With the patient in the dorsal position, flex the knee, and by pressure on the sole of the foot resist his endeavor to extend the knee. The quadriceps femoris is the principal muscle concerned.

Plantar Flexors (Extensors) of the Foot.—With the leg straight, resist, by pressure upon the sole of the foot, the patient's endeavor to bring the tarsus in a line with the leg, thus testing the gastroc-

nemius, soleus, peroneus longus and brevis. Have patient stand on toes.

Dorsiflectors of the Foot.—With the leg straight, resist the patient's attempt to bend up the foot, thus testing the tibialis anticus and the peroneus tertius. Marked paralysis of these muscles causes "foot drop." Have patient stand on the heels.

Muscles of the Foot.—The flexors, extensors, interossei, and lumbricales of the toes are examined in a similar manner to those of the fingers. There is a sort of claw-foot analogous to the claw-hand.

Reflexes.—*Superficial and Deep.*—These are then taken up. Those of the cranial nerves have been considered. The important reflexes of the upper extremities are:

Elbow or Triceps Jerk.—This is best tested by supporting the patient's arm at the elbow, allowing the forearm to hang flaccidly or putting it over the edge of a chair. The stroke is made just above the olecranon, and the reaction consists in an extension of the forearm due to contraction of the triceps muscle.

Radius Periosteal Reflex.—The radius periosteal reflex consists in a slight flexion of the arm on the forearm when the radius is tapped just three or four inches above the external condyle.

Supinator Jerk.—The supinator jerk is obtained by striking the muscle about midway between the elbow and wrist, the arm being supported at the wrist. It consists in a slight extension of the pendant wrist.

Reflex.	Method of eliciting.	Response.	Segment.
Biceps.	Tap biceps tendon.	Biceps contracts.	C 5 and C 6.
Triceps.	Tap triceps tendon.	Triceps contracts.	C 5, 6 and 7.
Supinator longus.	Tap radial styloid.	Supinator longus contracts.	C 5 and C 6.
Wrist.	Tap flexor tendons at wrist.	Fingers are flexed.	C 6 to C 8.
Carpometacarpal.	Tap back of wrist.	Fingers are extended.	C 6 to D 1.

Jacobsohn's radius reflex consists in a slight flexion of the fingers, particularly of the terminal phalanges when the radius of the extended outstretched hand supported by the observer's hand is suddenly tapped with a hammer.

The superficial reflexes of the trunk are next in order. Both sides should always be tested and recorded \circ if absent, $+$ if present; $R=L$ or $R>L$, $R<L$, or R, o, L_+ , or *vice versa*, as the case may be.

Epigastric.	Stroke downward from nipple.	Epigastrium dimples.	D 7 to D 9.
Abdominal.	Stroke down from costal margin.	Abdominal muscles contract.	D 11 to L 2.
Cremasteric.	Stroke inner side of thigh.	Testicle is pulled up.	L 1 to L 2.
Gluteal.	Stroke skin over buttocks.	Gluteal muscles contract.	L 4 to L 5.
Bulbocavernosus.	Pinch dorsum of glans penis.	Compressor urethræ contract.	S 3 to S 4.
Superficial anal.	Prick skin of perineum.	External sphincter contracts.	S 5 and conus.

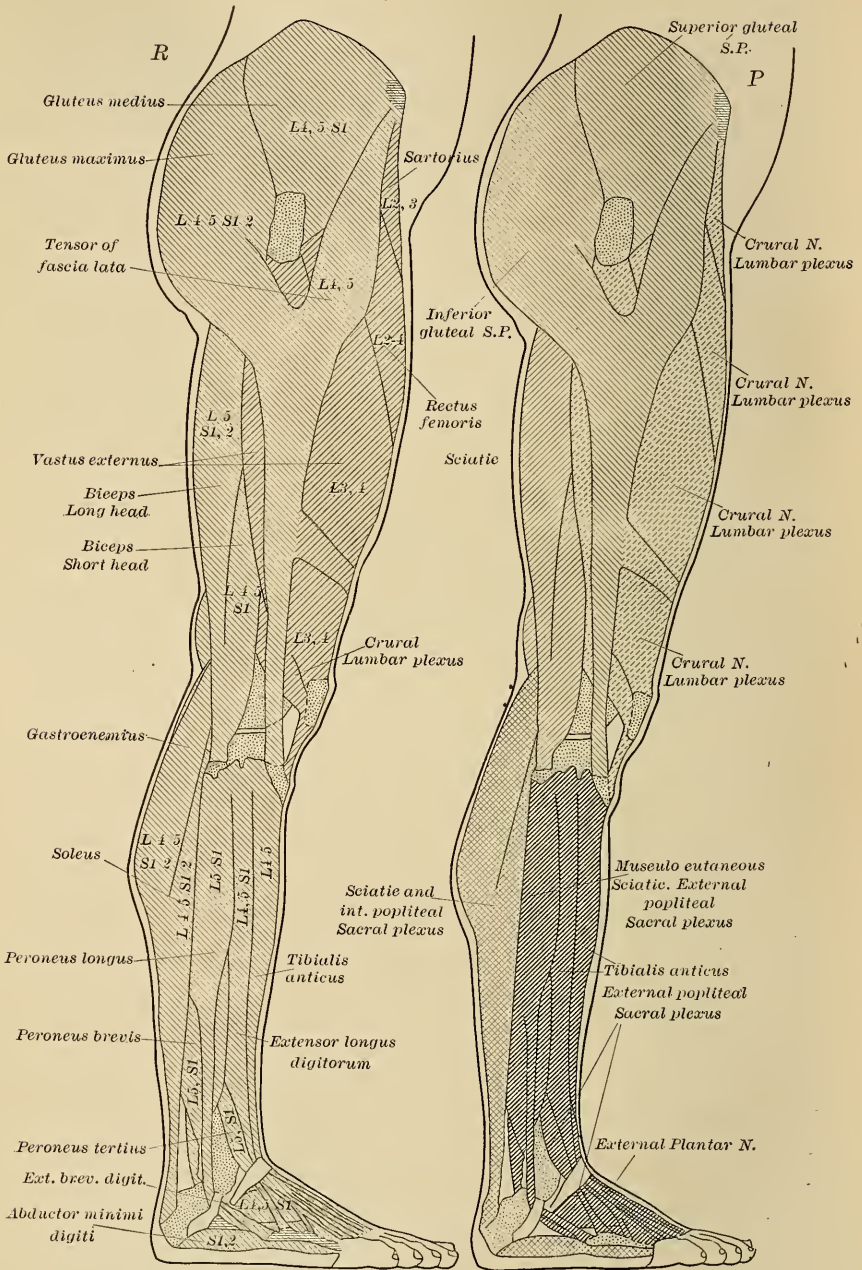


FIG. 15.—Radicular (*R*) (to left) and peripheral (*P*) (to right) innervations of the muscles of the external side of the lower extremity. Letters and abbreviations as in preceding figures. (After Dejerine.)

Reflexes of the Lower Extremities.—The *knee-jerk* (Erb-Westphal's sign) is one of the most familiar. The knee-jerk may be tested in a

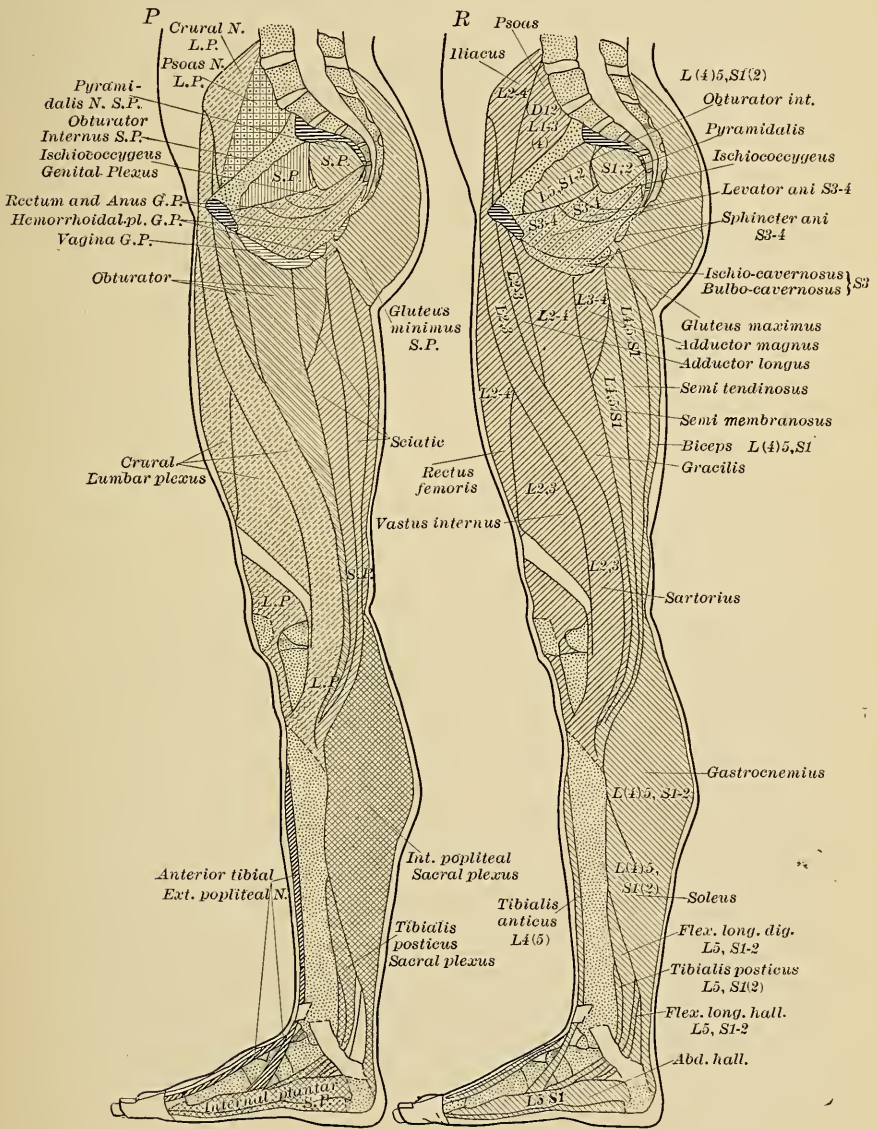


FIG. 16.—Radicular (R) (to right) and peripheral (P) (to left) innervations of the muscles of the internal side of the lower extremities. (After Dejerine.) G.P., genital plexus.

variety of ways. One of the best is to have the patient sit upon a table, which permits the limbs to hang freely, then telling him to look

at the ceiling, or diverting his attention, the tendon just below the patellar is tapped or the patient is directed to cross his leg on the knee. Exaggerated, active, normal, sluggish, or absent responses should be recorded. Another method is to have the patient sitting, and the feet upon the floor, but the legs comfortably extended. The tendon is then tapped. Here a simultaneous tapping of both tendons may be tried.

In certain patients the attention must be diverted, otherwise he holds the leg rigid. This destroys the reflex. One can have the patient repeat the Lord's Prayer, or have him do small sums in arithmetic, or converse with an assistant to divert his attention from the testing.

Jendrassik thought of the expedient of utilising a forced muscular act in the upper extremity to reinforce the knee-jerk. This may be carried out by having the patient make hard fists at a given signal, when the tendon is tapped, or by having him grasp his hands and pull at the given signal. By reinforcement a very weak knee-jerk may be made very evident.

Achilles Jerk.—This is best tested by having the patient kneel upon a chair, the foot being just free of the edge. The Achilles tendon is then tapped, and there results a dorsal flexion of the entire foot.

For patients in bed, the leg should be everted, slightly flexed, and the foot extended to put the tendon on slight tension. One person in a hundred has no knee-jerk.

Ankle-clonus.—To elicit ankle-clonus requires some care. It is best obtained by supporting the patient's leg along the under side, the patient coöperating by thorough relaxation, then the free hand grasps the foot, and makes a sudden upward, dorsal flexion, holding the foot fairly firmly flexed at the end of the movement when a series of clonic extensions and flexions take place. The leg should be slightly everted, and the knee somewhat flexed. A false clonus consists in half a dozen flexions and extensions; true clonus continues for some time.

Plantar Reflex.—By stroking the sole of the foot, either at its external or internal border, a quick plantar flexion of all the toes including the great toe takes place. This is normal plantar flexion. As many individuals are ticklish, there is frequently a sudden jerking of the whole foot, or such a protective movement is manifested only in the tendons of the great toe. This should be distinguished from true dorsal extension or the *Babinski reflex*.¹ This consists in the comparatively slow dorsal extension of the great toe when the plantar reflex is tested and at the same time there is a slight spreading apart of the other toes.

If the patient is in bed—as he should be for testing—a fully developed Babinski sign consists in the slow dorsal raising of the great toe, the spreading of the other toes, a slight rotation of the thigh on the hip,

¹ The term *dorsal extension* is here meant extension of the toes. By Babinski the term *plantar extension* was used. We are using the words in the ordinary sense, disregarding the fact that by some palmar extension is made synonymous with dorsal flexion, and plantar flexion with dorsal extension.

and a contraction of the fascia lata of the thigh. In order to develop the whole test the feet should be warm, the thigh slightly rotated externally, the knee slightly bent, and the stroke made either on the outer or inner border of the sole by either a fairly sharp instrument,



FIG. 17.—Extension of the great toe on irritating the sole. The Babinski plantar extension phenomenon.

the finger nail, or a blunt-pointed instrument. As there is great variation in the thickness of the skin of the soles of the feet, the various ways of bringing out a Babinski phenomenon should be tried in each case. Particular attention should be directed to the dangers of confusing



FIG. 18.—The Chaddock modification of the Babinski, causing great toe extension on stroking beneath the external malleolus. (Chaddock.)

the protective, pulling away motion on tickling, which causes a marked, quick dorsal raising of the great toe, from a true Babinski phenomenon.

Careful record should be made of the irregularities in plantar response. In some patients there will be plantar flexion of the small

toes but no reaction of the big toe. At times this may be as significant as a true dorsal extension. It is to be borne in mind that dorsal raising of the great toe is normal in infants and children up to the age of successful walking.

A number of closely related reflexes have been described, having much the same significance as the Babinski sign, but being less constant, and at times contradictory. These are:

Strümpell Reflex.—This follows forceful pressure over the anterior tibial region with a resultant dorsal extension of the great toe. It is found in a number of conditions other than those of functional disturbance of the pyramidal tracts.

Oppenheim Reflex.—Here the inner surface of the leg is sharply and deeply stroked by the thumb from the middle to the ankle behind the malleolus. It brings out a great toe dorsal extension.

The Paradoxical Reflex.—Called by a variety of names, as described by Gordon, it consists in a dorsal extension of the great toe following the grasping of the deep muscles of the calf and making a forceful indentation along their external border.

Mendel-Bechterew.—This reflex consists of the dorsal extension of the toes, especially the second and fifth, when the dorsum of the foot is tapped about at the base of the middle toes. In reflex irritability this reflex is augmented. If plantar flexion takes place the authors regard it as a sign of organic affection.

Chaddock.—This produces a toe extension by stroking the side of the ankle (Fig. 18).

Reflex.	Method of obtaining.	Result.	Location.
Knee.	Tap patellar tendon.	Leg extended.	L3 and L4.
Achilles.	Tap tendo Achillis.	Foot plantar flexed.	S, S4.
Ankle clonus.	Sudden dorsi flexion foot.	Quick up and down movements.	Pyramidal tracts (L3, S2).
Plantar.	Stroke soles of feet.	Plantar flexion all toes.	L3, S2.
Babinski.	Stroke soles of feet.	Great toe dorsal extension.	Pyramidal tracts (L3, S2).
Oppenheim.	Stroke inner side of calf.	Dorsal extension great toe.	Pyramidal tracts (L3, S2).
Paradoxical.	Deep pressure in calf.	Dorsal extension great toe.	Pyramidal tracts (L3, S2).

Tremors.—The tremors of the upper extremities alone claim attention. Those of the head may be fine and oscillatory, nodding, rhythmic, or jerky. The rapidity of the tremors should be noted.

Tremors of the hand and wrist should first be tested with the pendant hand supported at the wrist. Then with hands extended and fingers wide apart *static* tremors come out. Or with the hands in motion *locomotor* tremors become evident.

Static tremors are either fine and rapid (eight to twelve per second), or coarse and slow (four to six per second). They may be irregular. When involving the whole arm one speaks of movements rather than tremors.

Locomotor tremors are in reality ataxias. They are brought out best by having the patient bring his index fingers from any position slowly to the end of the nose, first, with eyes open, then with eyes closed, test both sides (Finger-nose Test—F. N. T.), or the index fingers should be brought to touch each other (Finger-finger Test—F. F. T.). Here coarse irregular movements (ataxias) may be brought out. The patient's finger may overshoot the nose (asynergia). The patient's ataxia increases markedly as the nose is reached (intention tremor), or increases only just as the object sought is arrived at.

Ataxia in the lower extremities is tested by the Knee-heel Test (K. H. T.), the patient, on his back, is directed to touch the left knee with the right heel, and *vice versa*.

Athetoid movements are coarse, slow, sinuous, progressive, rhythmical movements in the fingers, arm, or trunk.

Choreic movements are irregular, coarse, or fine movements, non-rhythmical and non-coördinated—they are jerky movements.

Associated movements are involuntary movements of the opposite side, induced by a voluntary act. Not infrequently they are quite non-homologous movements.

Localized convulsive movements and Jacksonian epileptic movements consist of sudden convulsive involuntary extensions and flexions without loss of consciousness.

Tics.—These are involuntary, coördinated movements of psychic origin.

Diadokokinesis.—This signifies the ability to perform alternate rapid coördinated movements, of antagonistic muscles. Certain patients show a loss of this ability (adiadokokinesis). The tests most frequently applied are quick pronation and supination of the semi-flexed hand; piano-playing movements or quick flexion and extension of the forearm on the arm. The term is applicable only in the absence of motor paresis or gross anesthesia.

Apraxia.—This, speaking generally, consists in the loss of ability to perform purposeful movements. The tests are to have patient throw a kiss, to make a salute, to make a beckoning gesture, a threatening gesture, or to go through an imaginary act, such as taking a match out of a box and lighting it or blowing it out. It is also elicited testing the necessary movements to employ objects correctly.

EXAMINATION OF SENSORY NERVOUS SYSTEM.

The most important of the tests of the sensory nervous system are for: (1) light touch, (2) pain, (3) thermal sensations, and (4) deep sensibility. Head has suggested the terms epicritic, protopathic, and deep sensibility for the three types of sensibility which he maintains exist.

The Pyramidal Tract in its Corticospinal and Corticonuclear Portions.

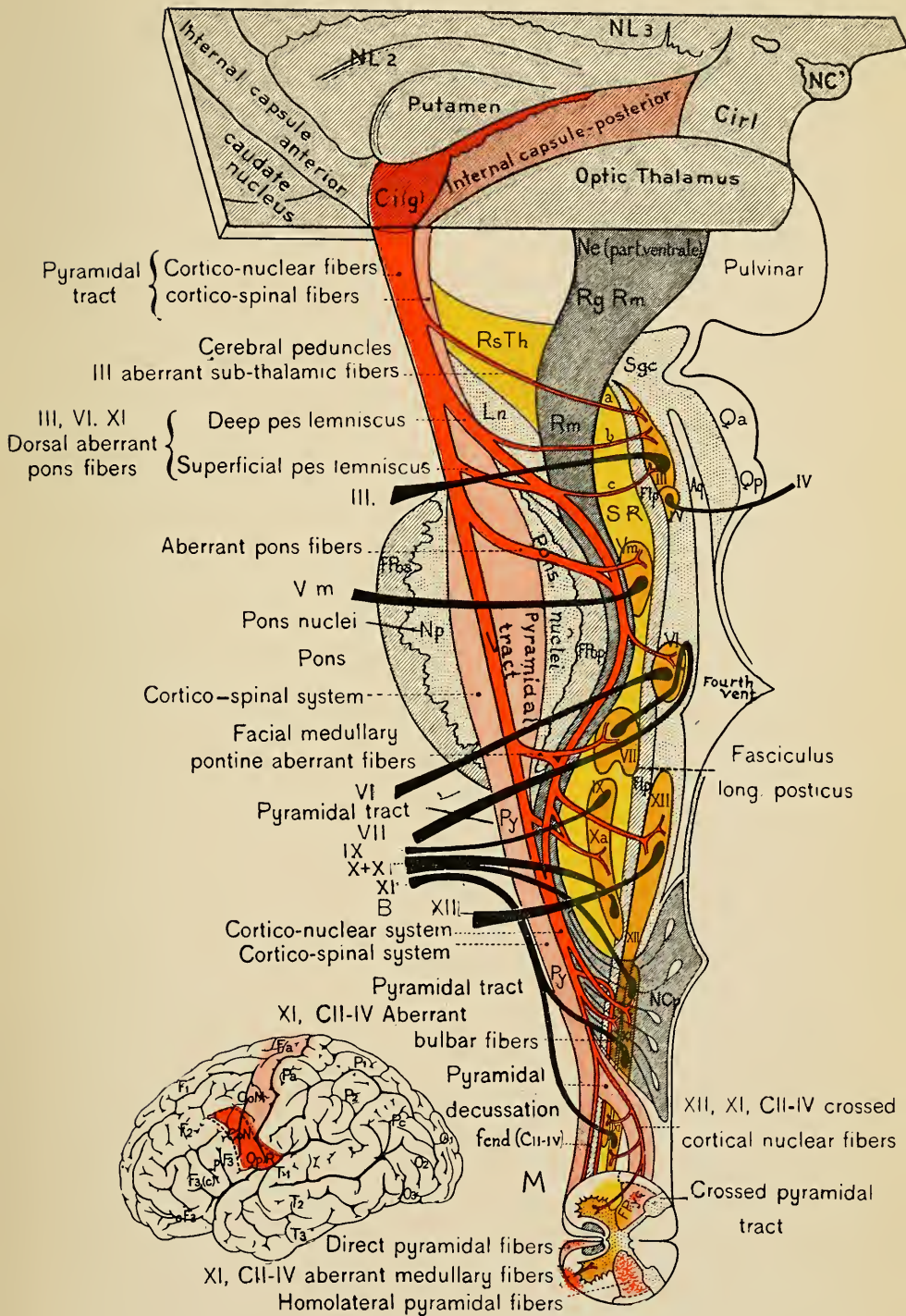
The corticomedullary (corticospinal) *Co.M*, is colored light red; the corticonuclear portion, *Co.N*, darker red. The reticular substance (*S.R.*) of the tegmentum in yellow and the motor nuclei of the cranial nerves orange. The corticopontine portions and the cerebellar paths of the tegmentum are omitted.

Abbreviations: *Aq*, aqueduct of Sylvius; *Cia*, anterior segment; *Ci(g)*, knee; *Cip*, posterior segment; *CirP*, retrolenticular segment of the internal capsule; *CoM*, corticospinal system (pink); *CON*, corticonuclear system (red) of the pyramidal tract, *VP*; *Flp*, posterior longitudinal fasciculus; *FPba*, *FPop*, anterior and posterior pontine fibers; *FPyc*, crossed pyramidal tract; *FPyd*, direct pyramidal tract; *fPyh*, homolateral pyramidal fibers; *fab*, medullary aberrant fibers; *fap*, pontine aberrant fibers; *fabp*, medullary, pontine aberrant fibers; *fasth*, subthalamic or superior pontine aberrant fibers; *fcnc*, *fcnd*, crossed and direct cervical corticonuclear fibers; *Ln*, locus niger; *NC*, caudate nucleus; *NC'*, tail of caudate nucleus; *NCp*, nuclei of posterior columns (Goll and Burdach); *NL₁*, *NL₂*, *NL₃*, the three segments of the lenticular nucleus; *Np*, pontine nuclei; *PLp*, deep pes lemniscus; *PLs*, superficial pes lemniscus or aberrant pontine fibers; *a*, *b*, *c*, the three modes by which the pontine aberrant fibers enter the third nerve nuclei: *Pul*, pulvinar; *Qa*, *Qp*, anterior and posterior corpora quadrigemina; *RgRm*, region of the median lemniscus; *Rm*, the median lemniscus; *Sgc*, subependymal gray substance; *SR*, reticular formation, colored yellow; *Th*, thalamus; *V₄*, fourth ventricle: *VP*, pyramidal tract; *III*, *IV*, nuclei and root fibers of the oculomotorius and trochlearis nerves; *Vm*, nucleus and root fibers of the trigeminus (masticators); *VII*, *IX*, *XI*, *XII*, nuclei and root fibers of the facial (*VII*), glossopharyngeal (*IX*), spinal accessory (*XI*), and hypoglossal (*XII*), nerves; *Xa*, anterior root of the spinal vagus; *X-XI*, root fibers of the spinal vagus.

The encephalic trunk and its three great divisions: Cerebral peduncles, *P*; the pons *PO*, the medulla, *B*, are shown in sagittal section with its connections with the internal capsule (*Cia*, *Cig*, *Cip*, *Cirl*) and the thalamus (*Th*) in part; and with the spinal cord (*M*) for the other part. The anterior segment contains the pyramidal tract (*V.P*) and is made up from above below of the foot composed of the fibers passing through the knee (*Cig*) and the posterior segment (*Cip*) of the internal capsule, the anterior segment of the pons with the pontine nuclei (*Np*) and the anterior (*FPoa*) and posterior (*FPop*) pontine fibers and the anterior pyramid of the medulla (*Py*); and the posterior segment or tegmentum, is separated above by the locus niger (*LN*), below it abuts the posterior pontine fibers (*FPop*), and the pyramid of the medulla (*Py*).

The tegmentum is made up of (1) a gray substance the reticular formation (*S.R.*)—colored yellow—which extends from the subthalamic region (*RsTh*) to the lateral columns of the cord and contains the motor nuclei of the cranial nerves—colored orange—disposed in two longitudinal columns; the anterior column includes the motor nuclei of the trigeminus (*Vm*), the facial (*VII*), the nucleus ambiguus or the anterior spinal vagus nucleus, (*Xa*); the posterior column includes the nuclei of the (*III*) and (*IV*) pair, the nuclei of the (*VI*), the long nucleus of the hypoglossal (*XII*) and the long inferior nuclei or spinal accessory (*XI*); (2) of the longitudinal fibers of which a part group themselves in fascicles more or less compact to form the posterior longitudinal fasciculus (*Flp*) and the median lemniscus (*Rm*). The median lemniscus, represents an important sensory pathway which terminates in the thalamus (*Th*) and takes part of its origin from the posterior column nuclei (Goll and Burdach). The posterior longitudinal fasciculus consists of an important association fiber system between the nuclei of the oculomotorius, the spinal

PLATE I



nuclei, the facial, to serve the important functions of lateral movements of the head, eyes, and trunk for maintaining the static equilibrium of the body. (See Plate VIII.)

Pyramidal Tract.—The corticospinal portion (*CoM*) of the pyramidal tract (pink) takes its origin in the ascending frontal convolutions (*Fa*), descends in the posterior segment of the internal capsule (*Cip*) and in the anterior portion of the cerebral peduncle. The corticonuclear (*CON*) portion (red) arises from the Rolandic operculum (*OpR*) at the foot of the ascending frontal convolution and the second frontal, descends through the knee (*Cig*) of the internal capsule and along the lenticular border of the posterior limb of the internal capsule (*Cip*) then to the cerebral peduncle, where it divides. One portion of the corticonuclear contingent follows the course of the corticospinal fibers to the anterior segments of the pons and the anterior pyramidal region of the medulla. Another portion, the system of the *aberrant fibers of the pyramidal tract*, break up at different levels, penetrating the tegmentum, descending in the median lemniscus (*Rm*) and the interolivary portion of the medulla to make up the fibers entering the motor nuclei of the cranial nerves.

In the medulla portions of the corticonuclear fibers fuse more or less with the aberrant fibers, pass by the inferior hypoglossal and spinal accessory, and behave like the corticospinal fibers. One part descends in the direct pyramidal (*FPyd*), another traverses the middle line with the crossed pyramidal (*FPyc*), a third descends as the homolateral pyramidal (*FPyh*) and the homolateral lateral cord. All of these fibers end finally in the motor nuclei of the anterior horns of the cord, functioning the movements of rotation, flexion, and bending of the head on the neck (*C_{II}* to *C_{IV}*).

The *aberrant fibers of the pyramidal tract* which detach themselves from the corticonuclear contingent are: (1) some inconstant fibers in the subthalamic region, the *subthalamic aberrant fibers* (*fasth*) destined for the oculomotor nuclei (*IIIa*) and to the anterior corpora quadrigemina. (2) In the region of the cerebral peduncles the *aberrant pyramidal fibers proper* deep pes lemnisci (*PLp*), and superficial (*PLs*) which leave the nuclei of the oculomotor nerves (*b* and *e*), then descend in the median lemniscus (*Rm*) and contain fibers for the (*VI*) and (*XI*) nuclei-cortical *oculo-rotary* and cortical *cephalo-rotary* fibers. (3) In the pons region, the *pontine aberrant fibers* (*fap*) which reinforce the preceding in the median lemniscus to enter the motor nuclei of the trigeminus (*Vm*) the hypoglossal (*XII*) and the spinal vagus (*Xa*). (4) In the neighborhood of the ponto-medullary sulcus, the *medullopontine aberrant fibers*, which are constant and well developed, reinforcing the preceding and destined to the nuclei of the facial (*VII*), the mixed nuclei of the glossopharyngeal (*IX*) and the spinal vagus (*Xa*), and the hypoglossus nucleus (*XII*). (5) In the medulla, *medullary aberrant fibers*, homologues of the homolateral pyramidal fibers, in constant fibers which descend the length of the periphery of the homolateral antero-lateral cord and terminate in the superior cervical cord nuclei of the rotators and flexors of the head and of the neck.

These aberrant fibers present great individual variation in their development—as seen in the different degeneration pictures studied by the Marchi method. Their chief characteristic is their descending pathways in the tegmentum, particularly in the median lemniscus and the anterolivary space in the medulla. This disposition explains why these fibers can be affected or spared by peduncular lesions, according to the site above or below, or whether it involves the anterior or posterior (tegmental) regions. The mode of termination of these corticonuclear fibers, possibly direct, possibly by intercalated neurones, in the motor nuclei of the cranial nerves has not yet been elucidated (Dejerine).

1. Epicritic sensibility is that which recognizes light touch, distinguishes small differences between the points of a compass, and recognizes small variations in the temperature of objects.

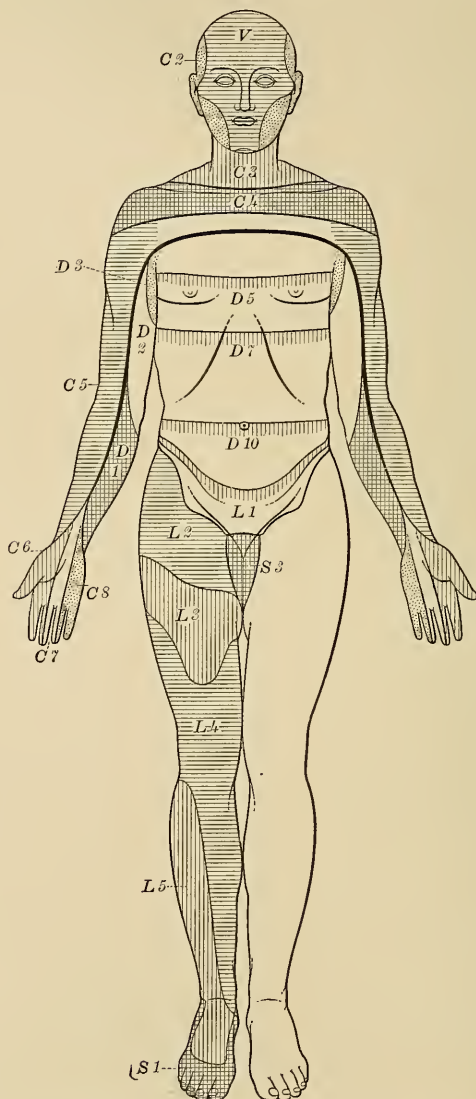


FIG. 19.—Illustrating the segmental spinal sensory areas. Front view.

2. Protopathic sensibility recognizes pain and extremes of heat and cold.

3. Deep sensibility recognizes deep pain and muscle and joint sense. Bony sensibility is included here.

To test epicritic sensibility one first tries the method of *light touch*. Touching the skin with the end of the finger is not a light touch test. It is a test for deep sensibility. Such a test is too coarse. Either a

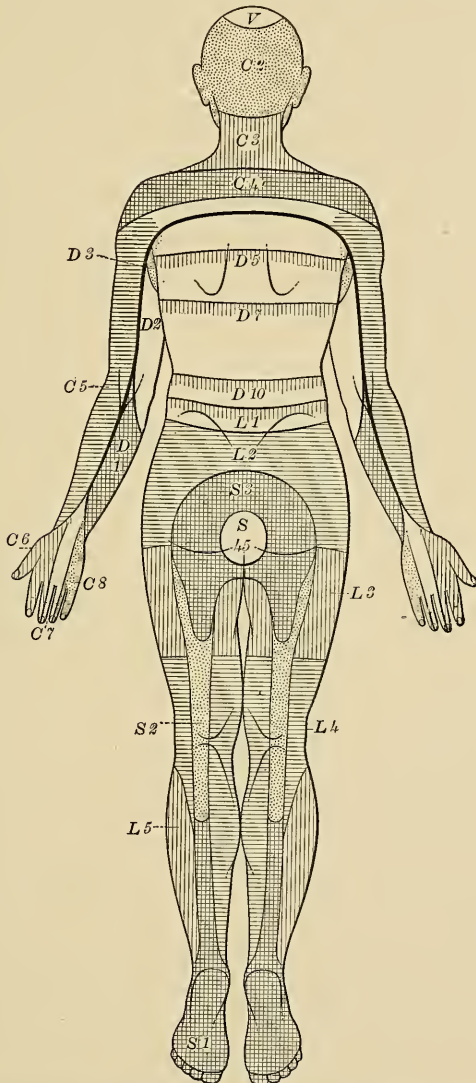


FIG. 20.—Illustrating the segmental spinal sensory areas. Rear view. These or similar sensory charts are useful in plotting sensory changes.

wisp of cotton-wool should be used or a fine camel's-hair brush. For most purposes the cotton-wool is to be preferred. In accurate testing, special esthesiometers are indispensable. Those mostly in use are Van Frey's hairs or Franz's simple esthesiometer.

The patient's body is systematically explored. He is asked to say "Yes" every time he is touched, and then asked to localize the spot touched. The testing should be made with the patient first lying down, and due attention should be given to the character (thickness, etc.) of the individual patient's skin in drawing conclusions from the tests. In going over the extremities, care should be taken to circle the limb with the touches as well as going up and down. It is specially desirable to avoid suggestive questions, such as, Do you feel this? What do you feel? etc. In certain cases, usually hysterical, one will get the steady response "No" to each touch over the so-called anesthetic area. This is a highly suggestive reaction. Modifications of light touch should be charted and marked on the skin with an anilin pencil. Such indications are very useful as landmarks for localization. There may be anesthesia to light touch or hyperesthesia, the patient feeling very acutely. In all hairy parts the skin should be shaved for accurate testing. (See Plates X and XI for the pathways involved.)

A Weber compass is useful for testing the individual capacity for recognizing one or two points. There is a great variability in individuals and in different regions. Some of the available figures for the minimum separation distance recognized as two points are as follows:

Tongue	1.5 mm.
Finger tips	2.3 mm.
Lips	3.4 mm.
Dorsum of fingers	5.6 mm.
Forearm	9.10 mm.
Forehead	10 to 15 mm.
Neck	23 to 30 mm.
Leg: back of foot	30 to 40 mm.
Back	50 to 60 mm.
Arms and thigh	70 to 80 mm.

Further tests may be made of epicritic light touch by placing variously shaped objects on the skin. Such tests are very valuable in special cases.

Epicritic thermal sensibility. This is most easily tested by use of the back of the finger for warm, and the metal head of a percussion hammer for cool.

Epicritic thermal sensibility recognizes differences as small as two to five degrees of temperature, while protopathic sensibility is unable to recognize differences between 40° and 20° C. Loss of epicritic sensibility for heat, with preservation of protopathic thermal sensibility, is not uncommon. The reverse, while rare, is occasionally found.

In making careful thermal tests an electrical thermometer, as contrived by Mills, is useful. In ordinary routine work test-tubes with ice water and hot water may be used, or metal tubes which have been plunged in cold or hot water employed.

It is not sufficient to test patients for extremes of heat and cold alone; minute differences should be tested as well.

Protopathic Sensibility.—*Pain.*—This is quickest tested by pinching the skin between the nail and the finger. A sharp-pointed pin with a round glass head is also useful. The patient is asked to distinguish between head and point. Absence of pain (analgesia) should be carefully charted as well as increased pain sensibility (hyperalgesia). The limbs should always be tested in their circumference as well as in their length, care being taken not to overlook thin strips of analgesia from root lesions. Hair sensibility should also be tested by pulling the hair.

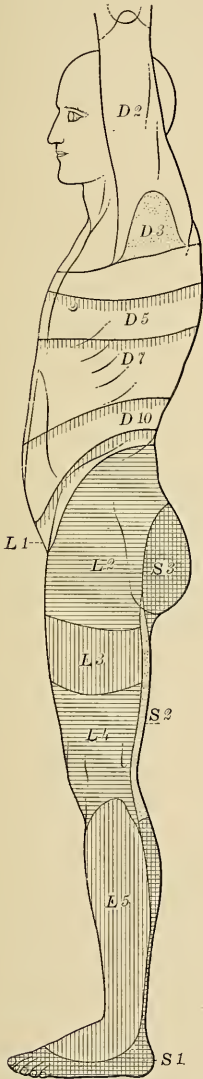


FIG. 21.—Illustrating the segmental spinal sensory areas. Side view.

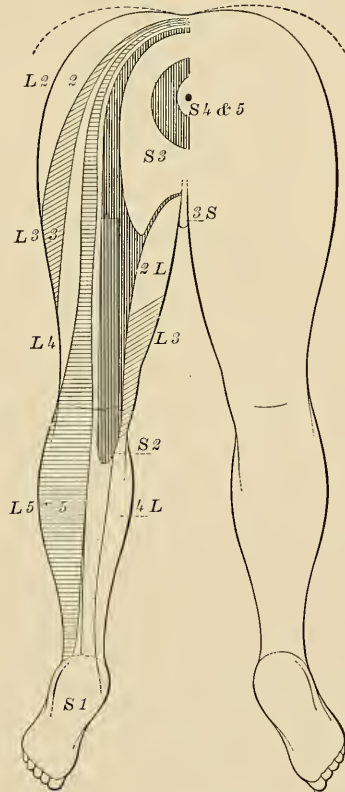


FIG. 22.—Illustrating the segmental spinal sensory areas of the lower extremities.

Painful faradic stimulation is at times of value in determining the value of an existing analgesia.

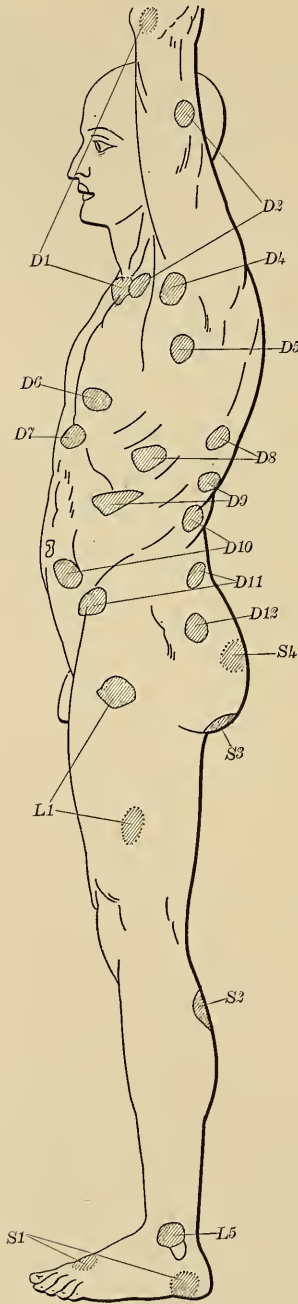


FIG. 23.—Cutaneous reflex zones of hyperalgesia, showing their relations with the spinal root segments and their vegetative nervous system connections. The dotted areas are to be referred to the internal surfaces. (After Dejerine.)

Deep Sensibility.—Here deep pressure pain, muscle and joint sense and bony sensibility are to be tested. Deep pressure with the thumb and fingers, or a special instrument (barethesiometer), is used. The pressure should be sufficient to cause pain.

Muscle and joint sense are tested by first showing the patient that one moves the thumb and big toe up or down—and then repeating movements up or down while the eyes of the patient are closed. Further, weights may be used and the ability to estimate differences

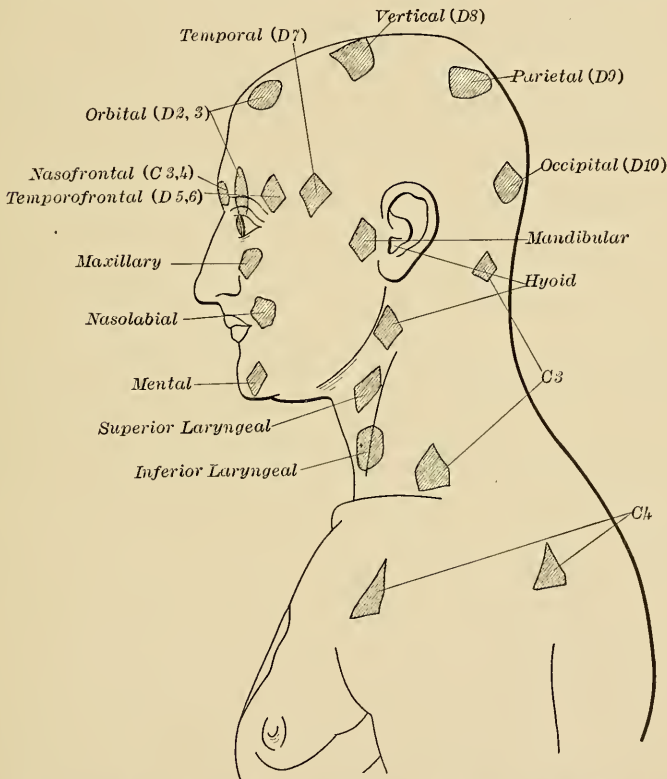


FIG. 24.—Cutaneous reflex zones of hyperalgesia of the head, neck, and shoulders in their relations to vegetative nerve (somatic) disturbances. (After Dejerine.)

observed; or the patient is requested to imitate with one hand, a definite position of the other hand.

Bony sensibility is tested by a tuning fork of low vibrating capacity.

Sensibility of the nerve trunks to direct pressure should then be tested. In the upper arm the brachial plexus branches in the neck and under the arm are palpable and along the inner arm and elbow-joint one may reach the median, radial, and ulnar. Anesthesia of the ulnar (Biernacki) is frequently a tabetic symptom.

The radicular and peripheral sensory distributions are shown in Figs. 11, 12, 13, 14, 15, 16.

In the lower limb the sciatic, anterior crural, cutaneous femoris, tibialis, and superficial peroneus are palpable.

Lasègue's Test.—This consists in flexing the extended leg on the abdomen, holding the patellar, when in neuritic processes a sharp pain (subpatellar space) is brought out. It is an indispensable test in the presence of suspected alcoholism.

The distribution of pain in neuralgic or neuritic affections should be carefully charted.

With the sensory examination, gnostic and praxic tests should be carried out.

Stereognosis signifies the ability to recognize objects by touch. Astereognosis is its absence. Objects should not only be named but their qualities described—shape, margins, density, etc. A lump of sugar, thimble, match box, marble, knife, pencil, scissors, etc., are useful test objects.

Apraxia consists in the loss of ability to carry out a purposeful movement, not dependent on a palsy. The most useful tests have been referred to. The examiner is specially referred to Plates X, XI for the interpretation of his findings and their anatomical foundations.

Vasomotor and Trophic Disturbances.—The presence of dermographia, of blushing, of redness, or blanching of the skin should be looked for. Ulcers, thickness of skin, dryness, or other trophic disorders should be charted. Reflex hyperalgesias (referred pains) should always be inquired for. Patients refer to them chiefly as "sore spots." (See Figs. 23 and 24.)

Scheme for Testing Sensibility.—The following scheme for testing sensibility is advised:

A. *Spontaneous Sensations:* Pain, numbness, tingling, position of the limb, idea of the limb, hallucinations or illusions.

B. *Loss of Sensation:*

1. Touch.

(a) Light touch, cotton-wool on hairless and shaved hair-clad parts; threshold with von Frey's hairs.

(b) Pressure touch, threshold with pressure esthesiometer.

2. Localization: Naming the part touched. Henri's or Head's method, target, etc.

3. Roughness: Threshold with Graham-Brown's esthesiometer. Sand-paper tests, discrimination of relative roughness.

4. Tickling and scraping: Tickling on soles and palms. Cotton-wool rubbed over hair-clad parts. Light scraping with finger nails.

5. Vibration, tuning fork: Loss or diminution of sensibility. Alteration in the character of the sensation evoked.

6. Compass points: Points simultaneously applied. Points successively applied.

7. Pain:
 - (a) Superficial pain: pin prick; threshold with algometer; reaction to measured painful stimuli.
 - (b) Pressure pain: threshold with the algometer; reaction to painful pressure.
8. Temperature: Thresholds for heat and cold. Effect of adaptation on threshold. Discrimination of different degrees of heat and cold. Affective reactions: (a) to extreme degrees, (b) to warmth.
9. Position: By imitating with the sound limb the position of the affected limb; by pointing with the sound limb; measurement of defect by Horsley's method.
10. Passive movement: Appreciation of movement. Recognition of the directions of movement. Measurement of the angle of the smallest movement which can be appreciated; falling away of the unsupported limb when the eyes are closed.
11. Active movement: Imitation of movement by the sound limb; ability to touch a known spot; measurement of the defect by Horsley's method.
12. Weight:
 - (a) With hand supported: Recognition of differences in weights applied successively to one hand. Appreciation of increase or decrease of weight. Comparison of two weights placed one in each hand.
 - (b) With hand unsupported: Comparison of two weights placed one in each hand. Recognition of differences in weights applied successively to one hand.
13. Size: Difference; threshold. Distinction of the head from the point of the pin.
14. Shape (two dimensional).
15. Form (three dimensional): Recognition of common objects by their form.
16. Textures.
17. Dominoes: Ability to count points by touch.
18. Consistence.
19. Testicular sensibility:
 - (a) Light pressure.
 - (b) Painful pressure.
20. Sensibility of glans penis to measured prick.

Status Corporis.—A systematic physical examination is a *sine qua non*. The main facts to be noted in the questionnaire are the condition of the heart, the presence of murmurs, the character of the arteries (hard, tortuous), blood-pressure, the lungs, presence of tumor in abdomen, enlargement of liver, and the condition of the urine, the blood, and the cerebrospinal fluid.¹

¹ Further neurological data will be taken up in the mental examination.

CHAPTER II.

MENTAL EXAMINATION METHODS.

IN no department of medicine is a complete examination of the patient more important than in the department of psychiatry. This examination must not only include the symptoms that the patient may present when seen, but must also include the most detailed obtainable anamnesis. It must be borne in mind that a psychosis is a condition of an individual who was previously well and that above all it is not a something that comes from without, attacks and seizes on the patient like, for example, a pathogenic microorganism, but is rather to be considered as a type of reaction of the individual to certain inimical conditions. In order, therefore, to understand a particular case it is of the highest importance to have, as fully as possible, a conception of the individual before he became afflicted, so that the symptoms which are the expressions of this reaction may be understood.

The scheme of examination which follows is directed primarily to elucidating the mental state. It is taken for granted that the student is familiar with the various methods of physical examination. The omission of specific directions as to the physical examination is not, however, to be taken as an indication that it is considered unimportant. On the contrary a physical examination in minute detail is of the utmost importance and unless it is made the risk is bound to be run that the key to the whole situation will be overlooked.

Mental disorders at best are obscure phenomena and no pains should be spared to illuminate them from every quarter. It is not, of course, expected that every possible physical test will be applied to each case. For example, it would be quite foolish to stain for the malarial parasite unless there was some clinical evidence of malarial infection. The usual examination of heart, lungs and urine should, however, be made in each instance. Similarly with the neurological examination, Trousseau's or Chvostek's signs would hardly be thought of unless tetany were suspected, while in every case the reaction of the pupils to light and accommodation and the patellar tendon reflex should be recorded and in patients suspected of organic brain disease or paresis the Babinski reflex and Biernacki's sign should be examined for.

The principal value that a scheme of examination may have, however, is in formulating tests that call for an actual record of the patient's reaction and not the conclusions of the examiner. Hospital records are filled with such remarks as "the patient shows lack of judgment"

or is "disoriented" or has "failure of memory." All of these are conclusions and by no means records of facts. Such histories are useless to any one except perhaps the persons who wrote them. The reader of a history is entitled to a statement of the facts on which the conclusion is based and then he is at liberty to form his own conclusion from the identical premises. How much better and more accurate than the statement "defective memory" would be this test: The patient in the course of the examination is given the address 375 Oxford St. After five minutes he is asked to recall it. He gives the number 176, but cannot give the name of the street at all. Here is a definite fact. A multiplicity of such facts gives one a basis for conclusions about the patient. Of such statements should the record of an examination be composed.

The examination will be considered under the following heads:¹

- I. Family History.
- II. History of Patient.
- III. Present Illness.
- IV. General Observations.
- V. Physical Examination.
- VI. Neurological Examination.
- VII. Mental Examination.

I. **Family History.**—*Informant* (name, relationship to patient, address).

Grandparents; parents (uncles and aunts); *Siblings*.

Children (with abortions and miscarriages).

NOTE.—In securing the family history it must be remembered that it is equally important to get a record of all the normal members of the family as well as the abnormal and not stop with securing the latter, as is often done. The patient's relation to hereditary tendencies can only be determined by securing the fullest information about his ancestors.

II. **History of Patient.**—*Full name; address; occupation.*

Birth; childhood diseases; when learned to walk and talk.

Diseases (especially convulsions, delirium, head-injury, gonorrhea, syphilis, rheumatism, neuritis).

Habits (alcohol, drugs, and sexual).

Marriage; Menstruation; gynecological.

Previous attacks (special attention to so-called hysterical, to nervous breakdown, and to melancholic periods).

Crimes and misdemeanors.

Mental make-up.

NOTE.—The history of the patient is especially important for getting a comprehensive idea of the sort of person the patient was before becoming ill. A given mental disorder cannot be fully understood without understanding, not only the circumstances that gave

¹ Many of these details have been covered in the neurological questionnaire, and hence are here briefly recapitulated only.

rise to it, but the other and more important factor, the make-up of the individual in whom the disorder occurs.

III. **Present Illness.**—*Onset; cause; physical; mental and moral changes; emotional condition; hallucinations and delusions; judgment; memory; suicide and homicide; insight.*

NOTE.—Under this head an inquiry is made into all the circumstances surrounding and conditioning the onset of the psychosis and the patient's attitude toward them and his insight. He should be asked frankly whether he believes himself insane; if not, how is it that he has been sent to a hospital for the insane; what he may have done or said to lead others to think him insane; what is his explanation of the whole situation and how it all came about.

IV. **General Observations.**—*Facial expression; appearance and demeanor; movements; speech; mental.*

NOTE.—The general observation of the patient is, of course, always important: whether he appears silly, resentful, indifferent; whether he has mannerisms, etc. It is particularly important, however, in stuporous and delirious patients who either will not speak or are not responsive and, therefore, not accessible. These patients should be observed particularly as to their general attitude of body and limbs, the expression of the face, the reflexes, and the reactions—volitional, emotional, and organic (hunger, sexual, responding to calls of nature, etc.).

V. **Physical Examination.**—*Form; nutrition; weight; height; skin; bones and joints.*

Decubitus; scars (especially penis and mouth).

Respiratory system.

Circulatory system; heart-position, size and sounds; blood-pressure.

Genito-urinary system.

Gastro-intestinal tract; stomach content (if indicated); glands; abdomen.

Sputum (if indicated).

Blood composition (if indicated).

Cerebrospinal fluid (if indicated).

Urine (always).

NOTE.—It is hardly necessary to insist upon the necessity for a thorough physical examination in every case. It is especially important in the deliria in which the mental disorder may be the expression of an obscure physical condition.

VI. **Neurological Examination.**—(See Chapter I.) *Atrophy; hypertrophy.*

Movements, voluntary: activity; rapidity, accuracy, force (especially paresis); limitations.

Movements, involuntary: rigidity; tremor (at rest, intention); spasms; convulsions.

Reflexes: KK; TA,¹ contralat. add.; plantar; cremasteric (inguinal); bladder; anal; epigastric; triceps; ulnar; radial; jaw; clonus (ankle, patellar and wrist).

¹ KK = knee-kick; TA = tendo-Achillis.

Nerve-trunk sensitiveness; tender areas (especially vertebræ, breast, ovarian).

*Coördination: FN; FF; FT; KH;*¹ station (eyes open and closed, one and both feet); gait.

Sensations: touch; pain; organic; parasthesia; hypesthesia; hyperesthesia; anesthesia; analgesia; sense of position; feeling of reality.

Cranial nerves:

1. Smell: solutions and subjective.
2. Hemiopia: fundus; hallucinations.
- 3, 4, 6. Eye movements (all directions): squint; diplopia; ptosis; nystagmus (horizontal, vertical, rotary). Pupils: size; outline; direct light; consensual light; accommodation; sympathetic.
5. Corneal reflex: chewing movements; taste; solutions and subjective.
7. Facial symmetry (whistling); tremors; test-phrases.
8. Hearing: objective and subjective vertigo.
- 9, 10, 11. Swallowing: pharyngeal reflex.
12. Protrusion of tongue.

NOTE.—In a condition which admittedly involves the central nervous organs, particularly the brain, the neurological examination becomes of the greatest importance. Especial importance should be paid to the cranial nerve distributions and to the presence of paralyses or anesthetics, which might have localizing significance.

VII. **Mental Examination.**—*Orientation:* time; place; persons.

General memory: family; school; occupation; marriage; children; diseases.

Emotional status: insight; sleep; dreams.

Hallucinations: auditory; visual; other senses.

Speech: voluntary; writing (name, date, the United States of America, the Commonwealth of Massachusetts); auditory; visual; test-phrases. (Statistical, perturbation, Third Riding Artillery Brigade.)

Stories ("Cowboy," "Gilded Boy," "Polar Bear," "Shark," "Good Girl").

Special memory: Civil War; names of two generals; three European countries; capital of native State; President; 45319628; 35984271; 487631; 955217; 7368; 487; 352; 375 Oxford Street (after 3 or 5 minutes).

Masselon (hunter, dog, gun, forest, rabbit; man, wood, coal, stove, dinner; needle, thread, button, vest; pipe, match, smoke; pen, ink, letter).

Ziehen (horse and ox; dwarf and child; lie and mistake; water and ice). 7 x 6; 56—18; 23—14; 81—9; x—5=17; x—8=13; have 50c; buy cherries 12c, butter 7c, bread 10c; how much change?

Forward and backward associations (month; days of week; 752186, 25729, 6418, 265, 497).

¹ FN = finger-nose; FF = finger-finger; FT = finger-thumb; KH = knee-heel.

General information: cost of postage; color of stamps; holidays and meaning (Christmas, Easter, Fourth of July).

Finnch ("The early bird catches the worm;" "Lies have short legs;" "Set a thief to catch a thief;" "Burn a candle at both ends").

Ethical questions:

Drawing diagram (after five seconds' exposure).

NOTE.—Here especial caution is needed to avoid recording conclusions. For example: Under orientation the patient's actual answers to such questions as, When were you born? How old are you? What day is this? etc., should be put down.

The stories which are named are as follows:

"Cowboy Story."—A cowboy from Arizona went to San Francisco with his dog, which he left at a dealer's while he purchased a new suit of clothes. Dressed finely, he went to the dog, whistled to him, called him by name and patted him. But the dog would have nothing to do with him in his new hat and coat but gave a mournful howl. Coaxing was of no effect, so the cowboy went away and donned his old garments, whereupon the dog immediately showed his wild joy on seeing his master as he thought he ought to be.

"Gilded Boy Story."—It is related that at the coronation of one of the popes, about three hundred years ago, a little boy was chosen to act the part of an angel; and in order that his appearance might be as gorgeous as possible he was covered from head to foot with a coating of gold foil. He was soon taken sick, and although every known means was employed for his recovery, except the removal of his fatal golden covering, he died within a few hours.

"Polar Bear Story."—A female polar bear with two cubs was pursued by sailors over an ice field. She urged her cubs forward by running before them, and, as it were, begging them to come on. At last in dread of their capture she pushed, then carried and pitched each before her, until they actually escaped. The polar bear is a wonderful swimmer and diver. In the capture of seals lying on the ice, it dives some distance off and swimming underneath the water, suddenly comes up close to the seals, shutting off their retreat to the sea.

"Shark Story."—The son of a governor of Indiana was first officer on an Oriental steamer. When in the Indian Ocean the boat was overtaken by a typhoon and was violently tossed about. The officer was suddenly thrown overboard. A life preserver was thrown to him, but, on account of the heavy sea, difficulty was encountered in launching the boat. The crew, however, rushed to the side of the vessel to keep him in sight, but before their shuddering eyes the unlucky young man was grasped by one of the sharks encircling the steamer and was drawn under the water, leaving only a dark streak of blood. (Adapted from Ziehen.)

"Good Girl Story."—Once upon a time there was a girl, whose father and mother were dead, and who was so poor that finally she had nothing but the clothes on her back and a little piece of bread in

her hand. She was deserted by everybody, but since she was good and honest she went into the world with confidence in God. As she went along she was met by a poor old man who said, "Give me something to eat, I am hungry." The girl gave him the piece of bread and went on farther. Soon afterward she encountered a little girl freezing and almost naked, who begged for her clothes. The good girl gave the poor child the warmest of her garments. Night came on, the good girl was tired, cold, and hungry. She traveled into the woods, and, wandering off the road, she knelt and prayed to God. As she knelt she saw the stars falling all about her, and when she looked she found they were many bright gold dollars. (Adapted from Ziehen.)

These stories which are used have been selected with great care. They are especially valuable. It is remarkable the amount of information that one can obtain from getting a patient to repeat one or two. Defects of memory and attention show immediately, while the manic tendency to elaborate is characteristic. They should never be omitted. The cowboy story is usually the easiest, while the good girl story is hard, because of the great amount of detail. The emotional feature of the "streak of blood" in the shark story is particularly impressive and may be about the only feature of the story reproduced.

In the special memory test, of course, different people will have to be treated differently. A Polish immigrant just landed would hardly know about the Civil War. The important thing, however, is to record actual question and answer.

In the Masselon tests the patient is asked to incorporate such words as pen, ink, letter, into a sentence.

In the Ziehen test the patient is asked to tell the difference between horse and ox, dwarf and child, etc.

The problem of calculating the change left from 50c after making certain purchases is an excellent example of the usefulness of standard questions. Every one on the hospital staff knows that the answer is 21c; and although this is a little thing, when multiplied many times it makes a great deal of difference in the ease with which one can go over a history or appreciate it when read.

The forward and backward associations are valuable as roughly quantitative. The average person should be able to give six numbers forward and five numbers backward. This test will disclose just how many the patient can give and is one of the valuable tests for repeating from time to time during the course of the psychosis. It is also very valuable in detecting the malingerer. A definite intention to blunder is usually readily distinguishable from a natural blunder.

In the Finckh test the patient is asked the meaning of the several sayings such as "The early bird catches the worm."

Such ethical questions can be asked as "What would you do if you saw a man drop a \$10 bill?"

In addition to the tests given in the table frequent use is made of the Ebbinghaus test which consists of having the patient complete a

sentence in which certain words have been left out, such as: I got up in the . . . , and after washing my . . . went to . . . Or better often is Ziehen's modification of this test. The patient is asked to complete such a sentence as this: If it rains . . . because . . . in spite of . . . The Bourdon test is very valuable as a measure of attention. It consists of getting the patient to strike out certain recurring letters or numbers in a standard page and timing the result. A similar test is the tapping test—timing the number of taps that can be made in a given time, say thirty seconds.

Of course the cases will be numerous in which it will be found desirable or necessary to pursue the examination further in some direction. No scheme can cover all possibilities and would be useless if it did, because impossible to carry out. Much must of necessity be left to the judgment of the examiner. By following this plan, however, it is believed that the general and important features necessary for a case record will be covered in the large majority of cases.

It is useful, after completing the examination, to accent the significant features in a short summary, which might include a provisional diagnosis if the facts warranted.

The tests described above are for the most part intelligence tests. Even such questions as might be propounded under the head of "ethical questions" may very easily have only the value of intelligence tests because the patient will quite likely answer in accordance with the conventional ideas with which he is perfectly familiar rather than answer in accordance with the way in which he feels. While the intelligence tests are important and while by taking them in a routine manner one can get a good deal of information from the patient, often information of matters that lie deeper than mere questions of intelligence, still they are by no means all sufficient. The intellect is after all only superficial as a guide to conduct. The deeper motives that move men to action come from the realm of feeling, and if the symptoms are to be explained or understood the emotional springs of conduct must be fathomed.

PSYCHOANALYSIS.

Psychoanalysis is the method by which the human mind is, so to speak, dissected, and by means of which the hidden motives of conduct are sought. If anything like a complete understanding of patients is to be had the methods of psychoanalysis must be used. This is hardly the place to discuss these methods at length. It would require more space than a text-book of this character could properly give it. The student is referred to special works. Here only will be given the briefest suggestions.¹

The Complex.—The mind cannot be conceived of as consisting of or containing ideas which are deposited here and there, helter skelter,

¹ Hirschmann, *Freud's Theories of the Neuroses*, New York; Jung, *Theory of Psychoanalysis*, New York; *Psychoanalytic Review*, New York.

without order, as the scraps of paper that are thrown carelessly into a waste basket. Quite the contrary. Ideas are grouped about central experiences, constellated one may say, built into coherent and harmonious structures not unlike the way in which bricks and stones are brought together to form buildings and these buildings are again grouped to form the larger whole—the city. The significant fact in this connection is that the cement that holds the bricks and stones together, the binding substance, is *feeling*.

This orderly arrangement of ideas upon a background of feeling which serves to unite them is what gives character, individuality to the personality. The creating of the proper feeling-tone about things and events is one of the main functions of education.

Now it so happens that in certain types of individuals a constellation of ideas, grouped about a central event that conditions a highly painful emotional state, is crowded out of clear consciousness—*repressed*—into the unconscious and so tends to lead an existence which is relatively independent and in so doing gives origin to various symptoms. Such a constellation is a “complex.”

The complex, crowded out of relation with the personal consciousness, seeks for expression and because it is not synthesized with the rest of consciousness, because the individual is not aware of its existence, its expression cannot be controlled and guided into the usual channels and so creates symptoms.

The extreme difficulty in locating and uncovering the complex is due to the symbolic forms in which it usually manifests itself. The painful memories of disagreeable experiences, unethical, unconventional, and otherwise impossible and hateful wishes while crowded out of mind by what Freud has so aptly termed the “censor of consciousness” nevertheless struggle to find expression. The complex cries for recognition, the censor will have none of it—the fight is on, the conflict wages, until finally a sort of compromise is reached by permitting the complex to come into clear consciousness but only on pain of not disclosing its true self, under the cloak of a complete disguise.

For example Freud's case of Elizabeth. She was engaged in nursing her sick father who afterward died. One evening, spent away from home at the solicitation of the family, she met a young man of whom she was very fond and he accompanied her back home. On the walk home she quite gave herself up to the happiness of the occasion and walked along oblivious of her duties. On reaching home she found her father much worse and bitterly reproached herself for forgetting him in her own pleasure. She immediately repressed this disagreeable thought from her consciousness. Now she had, each morning, to change the dressings on her father's swollen leg. To do this she took his leg upon her right thigh. The suppressed complex seized upon the feeling of weight and pain of her father's leg upon her thigh as a handy and efficient means of expression and so the repressed erotic wish comes into consciousness under the disguise of a painful area of the

right thigh corresponding in extent and location to the place upon which she rested her father's leg.

This is the sort of mechanism that accounts for many unusual and strange experiences that otherwise appear to be without reason. Unexplained forgetting, slips of the tongue, certain mental attitudes, moods, and even the dominant traits of character are due to the activity of submerged complexes while the phenomena of dreams are explained in the same way.

The unconscious methods are very logical. As already described the complex often expresses itself symbolically (*symbolism*), often by the transfer of an emotion from a painful event to a less painful or indifferent event (*displacement*), often, as in hysteria, by the conversion of the conflict into a physical symptom (*conversion*).

Dreams.—The analysis of dreams is for the purpose of determining the presence and nature of complexes which are exercising a controlling effect upon the patient's conduct and feelings. The dream appears as a quite senseless experience to the patient and upon the face of it it would appear also to be senseless. A very little effort, however, will show that there is a certain rough meaning to the dream. For example, the scenes of the dream will be representations, usually more or less fragmentary, of things which have happened in the life of the individual during the previous twenty-four hours and may easily be associated in his mind with events of some moment to him. As soon as this is pointed out the patient will acknowledge it, if he does not know it first himself, and will think that the dream has been explained. This, however, is only the *manifest content* of the dream. Behind this manifest content there lies a deeper, a more profound, a more important meaning or series of meanings. The superficial experiences only serve to hide the *latent content*, which contains the real material of importance in the dream.

The latent content will almost invariably show that the dream in its deeper meanings is dealing with repressed material, with material that the patient is not willing to acknowledge to himself. The whole object of the dream is primarily to conserve sleep, and to that end it does not permit the real dream thoughts to become known, and so builds up a structure that upon its surface appears utterly without meaning and nonsensical, and therefore is hardly deserving of attention. Therefore the dreamer usually passes the dream by without any particular notice, and in the course of the day it is generally forgotten.

The mechanisms by which this disguise is effected have already been spoken of in discussing the complex. The mechanisms are those of *distortion*. These mechanisms of distortion are first the mechanism of *displacement*, which effectuates the transfer of the principal affect from the situation where it belongs to some other situation. The feeling of anxiety, or fear, or disgust, if it were retained attached to that portion of the dream to which it is normally attached, would disclose to the patient the real thing about which he was anxious, fearful, or

disgusted, but if displaced upon some indifferent part of the dream serves the purpose of distortion. *Overdetermination* is a mechanism which secures the appearance in the dream of a particular figure, for example because of its likeness not to any one experience in the patient's life, but to many. All of these experiences, as it were, focus in one direction and produce the result; for example the face of a dreamed-of person may appear to be entirely unknown to the dreamer, but when it is, so to speak, dissected it is discovered that each portion of the face, the nose, the lips, the eyes, the hair, each of them, have well defined associations in the mind of the dreamer and that they have all condensed into this composite to serve the purpose of the dream formation. *Symbolism* is another mechanism which aids distortion so that instead of things being dreamed of as they are in reality, they are dreamed of as being represented symbolically, and so the dream becomes increasingly difficult to read, and to the dreamer quite unintelligible. Further, the mechanism of *secondary elaboration* is the mechanism by which the dreamer, after he is awake, goes on, as it were, with the dream material and explains to himself by a process of rationalization what certain features of the dream mean, and still further distorts it and places it beyond the pale of his power to recognize it.

Here at work in the dream is seen one of the most exquisite of defense mechanisms of the human being. One is accustomed to think of the human animal biologically as defending itself from all sorts of attacks upon its integrity. The various infections by micro-organisms are met by vital processes of defense which are frequently fully successful, and the whole theory of immunity is the theory of a defense mechanism of the human animal. The dream is just such a mechanism, and if what is going on in the mind of the patient would be known, what the patient is defending himself from, what are the disintegrating factors at work at the psychological level, the easiest access to the knowledge of these factors can be found if the meanings of the dream can be learned. Dream analysis is a most important tool for the unravelling and treatment of all of the neuroses and psychoneuroses and for the understanding of the psychoses.

Technique.—The technique of psychoanalysis is an art more successful in some hands than in others. However, the general methods of procedure may be briefly outlined.

In the first instance the physician must be fully imbued with a profound belief that mental symptoms have, each and every one of them, a meaning and a meaning which can be brought to light and will show them to be logical and understandable in each instance. He must then have patience to listen to the story of his patient, and not only listen to it, but listen to it attentively for the purpose of trying to find the meaning in it, for the purpose of trying to find out where the vital points are which can be attacked to best advantage.

It is true that the dream analysis is the main avenue to the understanding of the unconscious motives of action, but all sorts of hints

may come from other sources. For example one of the authors was recently listening to the story of a patient. In the course of that story the patient mis-spoke and said quinine when he intended to say calomel. Realizing that a "slip of the tongue" of this sort must have its meaning and is not an accidental occurrence, because nothing mental is accidental, the slip of the tongue was analyzed and led directly back to one of the most important emotional events in the life of the patient, an event which threw a flood of light upon his psychoneurosis.

Nothing is too trivial to be worthy of analysis, nothing but may throw light upon the situation. All the little slips of the tongue, forgotten incidents, points at which two recitals of an occurrence do not agree, even witticisms are necessary to trace out besides the analysis of the dream life, and offer an abundance of material in the course of the analysis.

The method of procedure is the method of free association, whether it be the analysis of some component of a dream or of a slip of the tongue, or what not, the method of free association is the one employed. The patient should be alone with the physician. It is practically impossible to conduct an analysis, at least beyond the surface, in any other way. Under circumstances of quiet and freedom from interruption, as far as possible, the different points which are to be analyzed are taken up. The patient is instructed to take a certain element of the dream which he has just recounted, for example, and hold it in his mind, and then tell freely all of the ideas that come to him. He is told to tell all of the ideas without any effort on his part of selection, no matter whether the ideas appear to him to have any relationship with the portion of the dream that he has been told to keep in mind or not, and no matter whether they appear ridiculous or have other qualities that incline him to lay them aside. He must tell them all just as a man might sit at the window of a railroad train and jot down, as far as possible, everything that he sees pass the windows as the train speeds on.

The theory of this procedure is that if the patient does not direct the thought in any way every idea that comes must of necessity have some relation to the event held before the mind about which enlightenment is sought. This is the method of unraveling the tangled network of the mental life and while it may be supplemented by word association or other means, still it would seem as our experience increases, that no other method is needed, that this answers all the purposes. It takes a long time, as a rule, however, to effect an analysis, —weeks, months, perhaps longer. It must be remembered that what has taken years to form cannot be unraveled in an hour.

It will probably occur to many to wonder how it is that one can expect to find memories reaching back for years sufficiently well preserved to be helpful. As a matter of fact the memories of all repressed experiences are perfectly clear no matter how old. The explanation for this is that being repressed they are dissociated from

the everyday events of life, they are kept in their original form, they have not been subjected to the attrition and amalgamation with the intricacies of associational life. They do not fade out by this process of absorption as do the memories of indifferent events, but remain where ever after they may be brought to light by analysis and used as helps for cure.

It will be seen from this short description what a far-reaching method this is. A method of analysis from which no event of life, no matter how apparently trivial, is free. A method that in its results lays bare not only the immediate antecedents and causes of the symptoms, but the whole innermost life of the patient, reaching back even to the period of early childhood. This of course takes time. A case of any complexity and difficulty quite generally takes several months, of at least three seances each week, to reach a final result.

PART I.

PHYSICO-CHEMICAL SYSTEMS.

CHAPTER III.

VEGETATIVE OR VISCERAL NEUROLOGY.

THE AUTONOMIC AND SYMPATHETIC NERVOUS SYSTEMS— THE INTERNAL SECRETIONS.

A THOROUGHLY consistent plotting of this enormous chapter in neurology is not yet possible. All of the disorders here brought together may not ultimately be found best grouped here. The general assumption followed, however, is that they all belong to disorders of a particular group of organs, partly nervous, partly glandular, but which are all governed by a homogeneous series of structures, the so-called vegetative nervous system.

Certain of these organs are probably closely related to nervous structures, hypophysis, pituitary, parathyroids, suprarenals, blood glands, etc., and have been variously brought together as the chromaffine system or the endocrinous or blood glands—while others are distinctly non-nervous, liver, pancreas, ovaries, uterus, lungs, stomach, heart, etc., but their functions are automatically regulated by one or other of the two portions of the vegetative system, the *sympathetic proper*, and the *parasympathetic* or *autonomic*. This twofold division of the vegetative system will be here adopted.¹

The vegetative nervous system consists of those nervous structures which supply, by afferent and efferent pathways, impulses to the special sense organs, smooth muscle fibers, and all those automatically working organs such as the heart, lungs, intestines, genital apparatus, blood-vessels, excretory glands, skin and organs of external and internal secretion, such as the liver, stomach, pancreas, intestinal glands, and the thyroid, thymus, adrenals, parathyroid, hypophysis and epiphysis, etc., respectively. All of these structures are constantly in function, and their disturbances are manifold; either through affect activities as

¹ See Higier, *Vegetative oder Viscerale Neurologie*. *Ergebnisse der Neurologie und Psychiatrie*, vol. 2, No. 1, 1912. For a complete discussion of this generalization see Eppinger and Hess, *Vagotonia*. *Nervous and Mental Disease Monograph*. 20. New York, 1915.

seen in many neuroses and psychoneuroses as palpitation of the heart, anorexia, fainting, crying, diarrhea, mydriasis, etc., or in infections or intoxications as reddening, swelling, gooseflesh, tachycardia, dryness of the mouth, stenocardia, gastric or visceral crises, Argyll-Robertson pupil, etc.

The vegetative nervous system in its essence is the primitive, archaic rest of the ganglionic or metameric system of the lower vertebrates. Its chief switchboard is in the midbrain. In the evolution of higher animals its development has been left behind, as it were, by the relatively more important (*i. e.*, for purposes of civilization and culture) neopallium or cortex with its rich cortical-association system, but with which it has remained in close relationship, since these structures producing consciousness and intelligence have grown out of the phylogenetically older system. Thus it comes about that the ganglionic system which in man serves the vegetative functions of

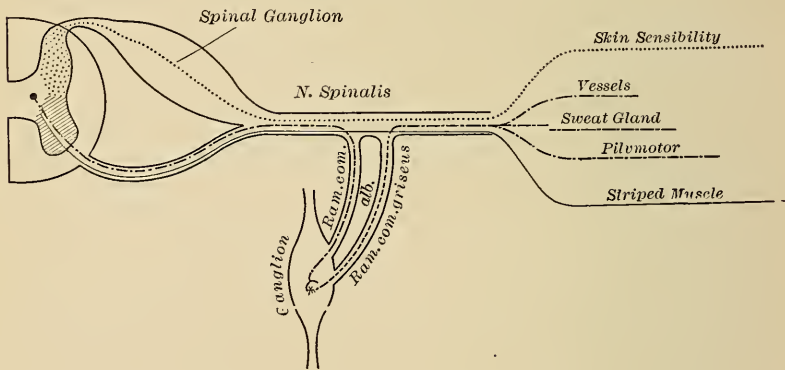


FIG. 25.—Diagram of the central connections and peripheral distribution of the vegetative system, — · — ·; the motor, —, and the sensory,, fibers. (Higier.)

the body is represented in the primary metameres, the spinal cord, again in the brain stem, central gray matter and midbrain, lenticular nucleus and optic thalamus, and finally in the cortex.

The central or spinal synapses are probably located in Clark's columns and in the lateral gray of the spinal cord (Jacobsohn). From here the centrifugal fibers pass through the anterior roots to and from the sympathetic vertebral ganglion as rami communicans albus and rami communicans griseus respectively to be distributed to the vessels, glands, unstriped muscles, etc., after the sympathetic break in the ganglion. This is the original type for each metamere, the symmetry of which, however, has been much distorted, either showing as an irregular or inconstant localization of the ganglia, or synapses, inconstancy of the communicating branches, irregular distribution of the centers in the cerebrospinal axis or incongruity of the embryonal metameres with the spinal and cranial segments (Figs. 25-27).

The chief anatomical results may be recapitulated as follows: In the skull the distortion is greatest as many metameres are anatomical conglomerates. Many of the ganglia are modified intervertebral ganglia, such as the geniculate and the Gasserian; others are compound ganglia due to the amalgamation of a spinal with a sympathetic ganglion as the jugular and vagus ganglia; other pure intervertebral ganglia are the ciliary, otic, sphenopalatine, submaxillary and sublingual which supply the smooth muscle of the eye, the vessels, the tear, salivary and mucous glands respectively. Some of the chief anatomical features for the head ganglia may be seen in the superb charts from Müller.¹ The upper ganglion, ganglion cervicale supremum, obtains its precellular fibers from the last cervical (CS) and upper dorsal (D 1-3) segments and innervates the vessels, hair muscles and

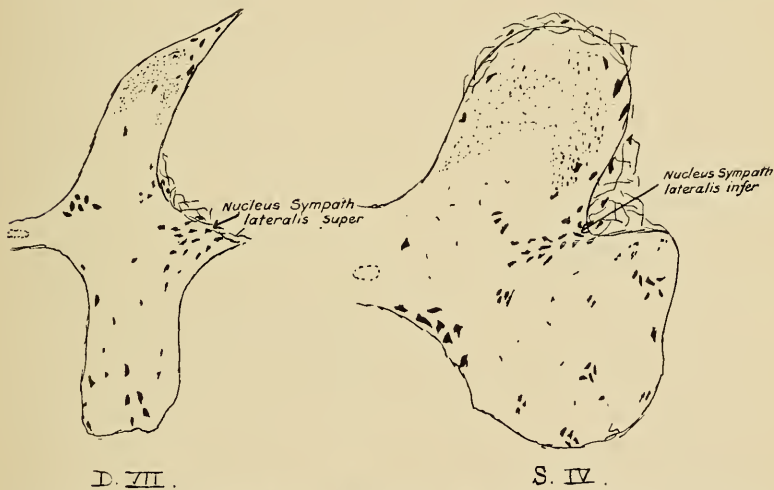


FIG. 26.—Sympathetic nuclei at the seventh dorsal and fourth sacral levels of the spinal cord. (Timme, *Jour. Nerv. and Ment. Dis.*, 1914.)

skin glands of the head, the dilator pupillæ and Müller's orbital muscle. The inferior cervical ganglion with its closely related stellate ganglion derives its preganglionic fibers from the D 1-5, and gives rise to the accelerator nerve of the heart and probably vasoconstrictor fibers of the pulmonary vessels.

The largest ganglion of the abdomen, the celiac, has its chief root in the celiac plexus in the major and minor splanchnic nerves, the former of which comes from D 4-9, the latter from D 10-12. As the mesenteric nerves they innervate the stomach glands, liver, pancreas, spleen, kidneys, adrenals, and intestine as far as the ascending colon. The inferior mesenteric ganglion receives its precellular fibers from L 1-3 and sends its postganglionic fibers to the colon and as the hypogastric, in part, to the anus, bladder, sphincter of bladder and genitals.

¹ Higier, loc. cit.

A series of blood glands, chromaffine cell containing structures (paraganglia) have of late been regarded as closely related to these clearly recognized ganglia of the vegetative system. The most important of these are: (a) Paraganglion caroticum, (b) paraganglion coccygeus, (c) paraganglion aorticum, and (d) paraganglion suprarenalis or adrenals.

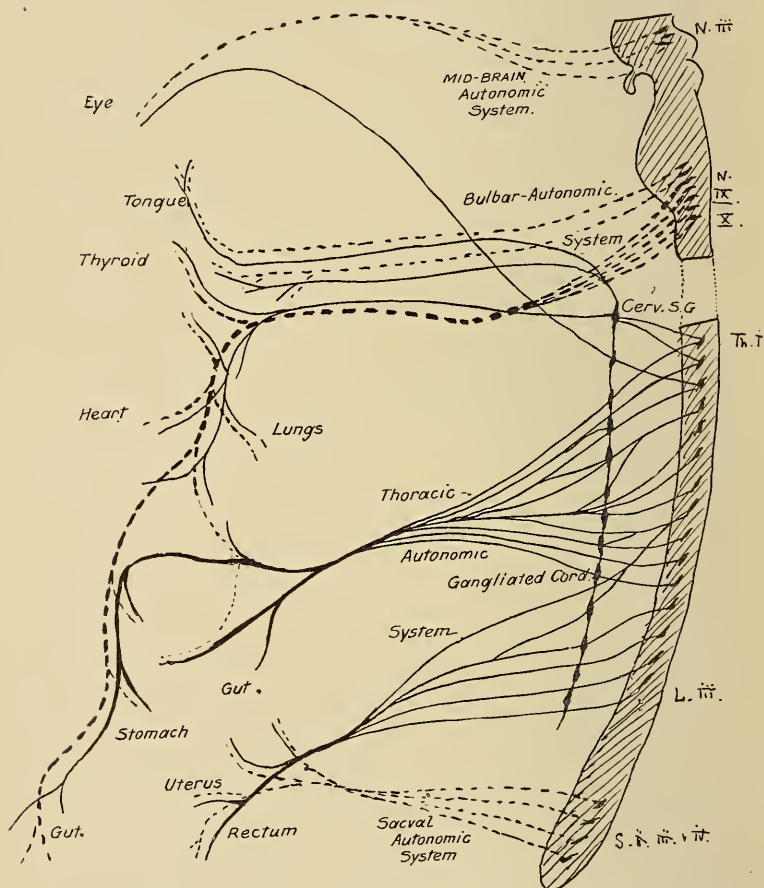


FIG. 27.—Distribution of the various divisions of the vegetative system. Sympathetic fibers are indicated by unbroken lines; parasympathetic (autonomic) by dotted lines. (Timme, loc. cit.)

Sympathetic and Autonomic Division.—Anatomically as well as pharmacologically it appears that two types of physiological activity are present in the vegetative nervous system. These are the sympathetic and the autonomic. All those non-voluntarily influenced organs, smooth muscle structures, heart muscle, glands, whose nerve fibers are derived from the spinal cord from the first dorsal above to

the fourth lumbar segment below, belong in the sympathetic system in the narrower sense. All others are controlled through the parasympathetic or autonomic. The uppermost come from the midbrain, enter the ciliary ganglion, and are distributed to the smooth internal muscle of the eye. A second or bulbar autonomic system comes through the facial and goes as the tensor tympani to the salivary glands. The glossopharyngeus and vagus belong to this bulbar autonomic system. A sacral autonomic system supplies the organs of the pelvis and genitals.

How this division will prove out in clinical work is yet to be tested, but it has seemed a necessary hypothesis in codifying the results of pharmacological experimentation. This is particularly to be seen in the reactions of the vegetative nervous system to certain products of the endocrinous glands, *i. e.*, hormones, and to certain toxic substances, notably nicotine. Inasmuch as the vagus constitutes the chief representative of the autonomic system, the terminology is applied to it more particularly.

Thus atropin and the nitrites paralyze the vagus (vagoparalytic). The former paralyzing the positive or stimulating element of the autonomic system, causing mydriasis, loss of secretions; the latter paralyzing the negative or depressing element and giving rise to vasodilatation. Vagospastic drugs, such as muscarin, pilocarpin, picrotoxin, and physostigmin, cause lowering of blood-pressure, weakening of the heart action, bradycardia, myosis, increased secretions, and increased peristalsis.

The chief contrasting activities of these two systems are here shown in tabular form as taken from the studies of Fröhlich, Eppinger, Hess, Loewi and others (see page 84).

A number of the products of the endocrinous system act as vagotonics; among these is cholin, from the cortex of the suprarenals. Whereas iodothylin, hypophysin, and adrenalin act partly as sympathicotonic, partly as autonomotonic or vagotonic. Iodothylin, or the related hormone from the thyroid, acts upon both systems to produce the characteristic signs of exophthalmic goitre (see later).

The active principle of the hypophysis acts upon both systems to produce pictures of hypo- or hyperpituitarism or a mixture of both, dyspituitarism. Adrenalin, from the inner part of the adrenal, causes tachycardia, increase in heart action, increase of blood-pressure from contraction of the bloodvessels, mydriasis and exophthalmos, paresis and anemia of the respiratory and stomach tracts, increase of sphincter tonus and of certain glands, mobilization in the carbohydrate depots in the liver and muscles, and increase in its oxidation. The antagonistic action of the sympathicotropic adrenalin and the vagotropic pilocarpin shows itself in that adrenalin can counteract a pilocarpin eosinophilia and pilocarpin an adrenal glycosuria. Other remarkable opposing reactions are known showing the striking antagonistic physiological possibilities of the vegetative system.

Action of stimulation of the sympathetic system.	Action of		Organ.	Action of		Action of Autonomic system.
	Atropin.	Adrenalin.		Pilocarpin.	Ergotoxin	
Stimulation Th. I-II	Paralysis	Stimulation	Sphincter iridis	Stimulation	Stimulation N. III.
Stimulation Th. I-III	Paralysis	Stimulation	Dilator iridis	Stimulation	Stimulation N. III.
Stimulation Th. II-IV	Paralysis	Stimulation I?	Ciliary muscle	Paralysis	Ch. tympani secretion.
Constriction Th. II-IV	Constriction	Constriction	Orbital muscle	Stimulation	Dilatation N. X.
Constriction Th. II-IV	Constriction	Dilatation	Salivary glands	Constriction N. IX.
Constriction Th. II-IV	Constriction	Constriction	Cortical bloodvessels	
Constriction Th. II-IV	Constriction	Constriction	Buccal bloodvessels	
Constriction Th. II-IV	Constriction	Constriction	Skin bloodvessels, head region	
Constriction Th. II-IV	Constriction	Constriction	Coronary bloodvessels	
Stimulation Th. II-L, IV	Inhibition	Constriction	Intestinal bloodvessels	Dilatation	Dilatation N. pelvic.
Stimulation Th. IV-VII	Inhibition	Inhibition	Genital bloodvessels	
Stimulation Th. I-V	Stimulation	Stimulation	Sweat glands	
Relaxation Th. I-V	Relaxation	Relaxation	Pilomotor muscles of the face	
Paralysis Th. II-L, IV	Paralysis	Paralysis	Heart muscle	Inhibition	Inhibition N. X.
Diminished Th. II-L, IV	Diminished	Diminished	Esophagus	Stimulation	Stimulation N. X.
Diminished ?	Diminished	Diminished ?	Cardia	Stimulation	Stimulation N. X.
Inhibition Th. II-L, IV	Paralysis	Paralysis	Gastric tone	Increase	Increases N. X.
Relaxation Th. II-L, IV	Paralysis	Paralysis	Gastric peristalsis	Increase	Increases N. X.
Relaxation L. I-IV	Relaxation	Relaxation	Gastric secretion	Increase	Stimulation N. X.
Relaxation Th. II-L, IV	Relaxation	Relaxation	Small intestine peristalsis	Stimulation	Stimulation N. X.
Inhibition ?	Inhibition	Inhibition	Colon	Stimulation	Stimulation N. pelvicus
Contraction L. I-IV	Inhibition	Contraction	Sphincter ani (muscle)	Spasm	Spasm N. pelvicus.
Relaxation L. I-IV	Inhibition	Relaxation	Gall-bladder	Contraction	Contraction N. X.
.....	Pancreatic secretion	Stimulation	Stimulation N. X.
.....	Bronchial muscle	Stimulation	Stimulation N. X.
.....	Sphincter vesicæ	Relaxation N. pelvicus.
.....	Detrusor vesicæ	Contraction N. pelvicus
.....	Uterus (pregnant)	Relaxation	
.....	Uterus (gravid)	Contraction	
.....	M. retractor penis	
Sugar puncture	Carbohydrate tonus	Diminished	Relaxation N. pelvicus.
Heat puncture	Heat balance	
Contraction.	Contraction	Pigment cells	Dilatation	

Th. = Dorsal, thoracic nerves. L = Lumbar nerves. N = Nerves.

Inasmuch as this system is very markedly under psychical influences, particularly of the affects, its relations to what is known as affectivity and ambivalence in psychoanalytic literature is of far-reaching importance. (See chapters on Psychoneuroses and Psychoses.)

The accompanying table shows most strikingly the material which has in part led to the hypothesis of an autonomic and a sympathetic partition of the vegetative nervous system.

Special Pathology.—Eye Sympathetic.—The ciliary, pupillary sphincter and dilator muscles, Müller's orbital muscles, and the tear glands are all vegetative organs of the eye which are innervated in part by autonomic and in part by sympathetic fibers. The pupillary innervation is of special moment. The nucleus of the dilator sympathetic fibers is the cilio-spinal center in D 1-3. These fibers pass through to the superior cervical ganglion where a synapsis is made. Here fibers pass to the Gasserian ganglion, join with the trigeminus (1), and in the long ciliary nerves pass to the vessels, dilator pupillæ, and to Müller's muscle, which pushes the eyeball forward. An autonomic pathway (sphincter) passes by means of the oculomotorius and ciliary ganglion. Connections with the cerebrospinal axis are many. The central origin of the precellular fibers of the smooth ciliary muscle of accommodation of the iris sphincter is not certainly fixed. (See Oculomotor.)

Several important clinical conditions depend upon the complicated pupillary innervation, the chief of which are: (1) absolutely stiff pupils, (2) Argyll-Robertson pupil, (3) sympathetic paralysis, (4) variations and deformities in pupils.

1. In the first all automatic stimuli to the iris muscle are inoperative with the exception of the sympathetic, which of itself has a minimal action. The pupils are dilated and distorted. Since the ciliary ganglion serves for autonomic tonic activity, any disturbance of the ganglion gives rise to great dilatation, which is more marked than is produced by nuclear or peripheral lesions of the oculomotor. Absolute rigidity is seen particularly in fainting, high grades of anxiety or fear, frequently in hysteria, in most epileptic convulsive attacks, and in central cerebrospinal syphilis. Pupillary inequalities are frequent in the psychoneuroses and such anomalies have special significance in the study of the repressions of unconscious material.

2. The Argyll-Robertson pupil is a complicated phenomenon. It has already been described. (See Examination.) It is an extremely common accident in cerebral syphilis, as seen in tabes and paresis particularly. It is occasionally found in extreme alcoholism (Korsakow particularly), and occurs from rare and isolated lesions of the corpora quadrigemina. In apes, Karplus and Kreidl have shown that a severance of commissural association fibers passing in the arm of the anterior corpora quadrigemina to the anterolateral border of the anterior corpus will cause a bilateral reflex pupillary rigidity with retention of pupillary activity for accommodation, convergence and psychical stimuli. Chronic meningeal exudates in syphilis pressing

upon these fibers may account for the frequency of this symptom in tabes and paresis. Explanations are numerous, however, and may be consulted in the literature. (Willbrand and Saenger, *Die Neurologie des Auges*.)

3. Sympathetic paralysis, or Horner's syndrome, is characterized by retraction of the bulb, narrowing of the palpebral fissure, dropping of the upper and raising of the lower lid and myosis, with conservation of the psychical and light reflexes of the pupil.

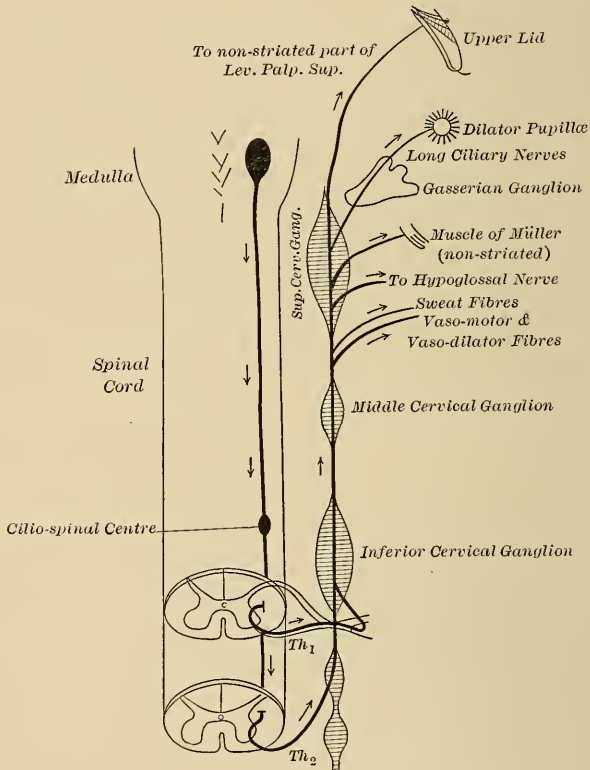


FIG. 28.—Diagram of course of oculo-pupillary fibers of cervical sympathetic. (Stewart.)

Topographically the picture results from pressure on the sympathetic fibers (goitre), a lesion of the cervicodorsal cord (hematomyelia), gliosis (syringomyelia), myelitis, especially of the upper dorsal region (Dejerine, Klumpke—Budge's centers), thrombosis of the posterior inferior cerebellar artery, cervicodorsal radiculitis, and in certain hysterical conversions, compulsive ties, and psychotic projections.

Tests with cocain and adrenalin are of value in determining the sympathicotropic activity, a 2 per cent. cocain solution stimulating the

dilator fibers. A failure to cause mydriasis is evidence of weakness of the sympathetic. Where such a paresis-producing lesion may lie, pre- or postganglionic, above or below the superior cervical ganglion, can be determined by the use of a 1 per cent. solution of the sympathico-tonic hormone adrenalin. Six drops in five minutes normally causes no action. If after fifteen minutes, however, there is a marked dilatation the lesion is postganglionic. Adrenalin mydriasis is frequently present in anterior and middle fossa disturbances (orbital disease, fracture of base). This is through the activity of the sympathetic fibers of the carotid plexus which joins with the trigeminus to the Gasserian ganglion. Thus a combination of disturbances of the supraorbital, with adrenalin mydriasis of the postganglionic sympathetic paralysis, may give important evidence as to the localization of a fissure, tumor, or fracture of the base of the skull. Double-sided adrenalin mydriasis (Löwi's reaction) is also seen in hyperthyroidism, pancreatic diabetes, and in increased irritability of the sympathetic nervous system in general.

Vagotonic reactions give rise to accommodation cramps with lachrymation which may be diminished by atropin. In youth when vagotonia is more pronounced, atropin acts less protractedly than in older people, and pilocarpin in the eye may cause von Graefe's symptom as a sign of an increase in the tonus of the autonomic levator palpebræ.

Tear Glands.—These are autonomically innervated through the superior cervical ganglion and sympathetically through the sphenopalatine ganglion. Irritation of the neck sympathetics causes increase, paresis of the same, diminution in the secretions. The postcellular branches of the neck ganglia, secretory or vasomotor fibers, pass in the internal carotid plexus reaching the glands either by the way of the ophthalmic plexus or through the cavernous plexus and the lachrymal sensory branch of the trigeminus.

Mucous and Salivary Glands.—The vegetative control of these is exercised through the sphenopalatine, otic, submaxillary, and sublingual glands. The sphenopalatine sends only autonomic vasodilator fibers through the posterior nasal nerves to the mucous membrane of the nose; sympathetic vasoconstrictor fibers come from the cervical sympathetics.

The parotid gland has both a sympathetic and autonomic supply, the former from the cervical sympathetic, the latter through the otic ganglion. The small superficial petrosal is its viator or precellular root, the auriculotemporalis of the trigeminus is its postcellular branch. The autonomic bulbar center is Kohnstamm's nucleus salivatorius inferior. Autonomic stimulation delivers a different type of secretion from sympathetic stimulation. Lesions of the tympanic in the mastoid operation give rise to parotid disturbance, and may be looked for in middle-ear disease. The submaxillary and sublingual glands have a sympathetic and an autonomic supply. The latter which causes vasodilator and secretory stimulating effects has its

autonomic bulbar center in Kohnstamm's superior salivatory nucleus, its rami communicans albi in the chorda tympani and through the lingual to the gland. The sympathetic vasoconstriction and secretory stimulating fibers are derived from the cervical sympathetic. Autonomic stimulation causes the full, thin, watery, salty secretion, cut off by atropin; the sympathetic the scanty, viscous (organic constituent) secretion acted upon by cocain, cholin, adrenalin. Xerostomia (Hadden), xerostomia senilis, xerophobia, dryness of stage and other types of

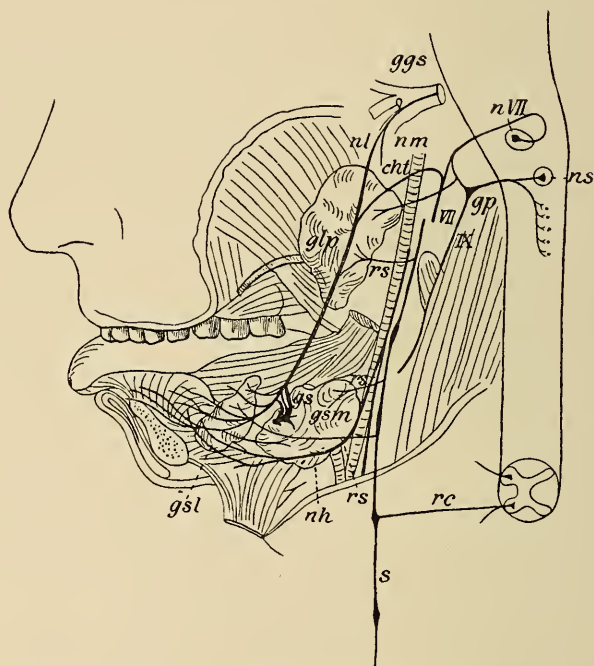


FIG. 29.—The innervation of the salivary glands: *gtp*, parotid; *gsm*, sub-maxillary; *gsl*, sublingual; *ggs*, Gasserian ganglion; *nl*, lingual nerve; *nm*, mandibular nerve; *nVII*, facial nerve nucleus; *cht*, chorda tympani; *VII*, facial nerve; *IX*, glossopharyngeal nerve; *ns*, nucleus salivatorius; *gp*, petrosal ganglion; *s*, sympathetic; *rs*, sympathetic branches; *gs*, submaxillary ganglion; *nh*, hypoglossal nerve; *rc*, ramus communicans. (Bechterew.)

fright, as in marked depressions, etc., are among the disorders of the secretions of these glands of neurological and psychiatric interest. Cortical, glossopharyngeal, and trigeminal associations are the basis for reflex stimulation of the glands.

Neck Sympathetic.—The superior cervical sympathetic supplies, through the internal carotid nerve and the internal carotid plexus, the dilator of the pupils, Müller's muscle, tear, parotid, and maxillary and lingual glands, the pilomotor, vasoconstrictors, and sweat glands of the face.

The clinical picture of loss of function of the cervical sympathetic through pressure or trauma are myosis of homolateral pupil with retained reflexes, narrowing of the homolateral palpebral fissure, enophthalmos, hyperemia of the homolateral side of the skin of the face and head, and transitory anidrosis of the same side. The motor lesions occur in the minor, the others in addition in deeper seated lesions.

The *vagus* like the oculomotor, glossopharyngeal and facial is a mixed nerve and contains motor, sensory and autonomic fibers. The somatomotor nucleus is the nucleus ambiguus; the nucleus solitarius the sensory nucleus; the visceral nucleus for the heart, lung, and digestive systems is the nucleus dorsalis vagi. All three sets of fibers travel through the jugular and nodosus ganglia to form the vagus. The two ganglia point to two nerves phylogenetically: (a) The pure motor branches are the rami pharyngeus; (b) the pure sensory branches are the meningeal and superior laryngeal; (c) the mixed motor-sensory-visceral is the recurrent laryngeal sending motor fibers to the larynx, receiving sensory fibers from the trachea and the visceral fibers supplying the heart, aorta, and vessels of the larynx; (d) the purely visceral branches pass to the digestive tract, the heart, the liver, and the lungs.

Only the visceral branches will be taken up here, the motor and sensory being discussed later under the cranial nerves.

Esophagus.—The entire digestive tract is served by the sympathetic (narrow sense), whereas the vagus (autonomic) only supplies the lower two-thirds of the esophagus, the stomach, and the intestines to the descending colon. The combined action is stimulating (autonomic) and depressing (sympathetic), which actions are apparently reversed in the case of the heart muscle. Local ganglion cells seem to regulate the motor functions. Tactile, thermal, and chemical stimuli are apparently unresponded to. Deep pressure sensibility is present, but the pathways are not definitely located.

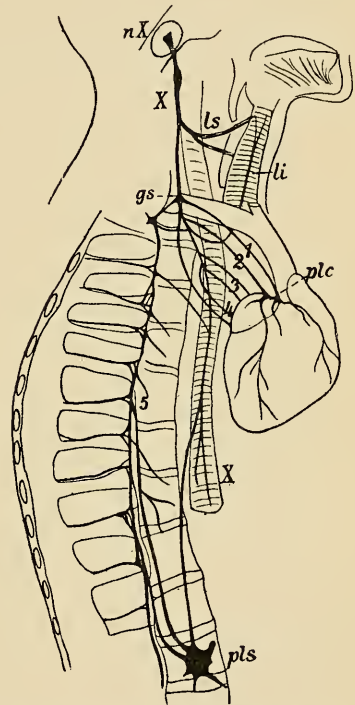


FIG. 30.—Schematic arrangement of cardiac nerves; *nx*, chief motor nucleus; *x*, vagus nerve; *gs*, stellate ganglion; *ls*, superior laryngeal nerve; *s*, sympathetic; *pls*, solar plexus; *plc*, cardiac plexus; 1, upper inner branch to heart; 2, accelerator; 3, internal inferior branch; 4, upper and inferior external branch; 5, Ansa Vieussensii. (Bechterew.)

Stomach and Intestines.—Local ganglion cells are very frequent and are to be sharply distinguished from the sympathetic structures. They serve largely for the motor functions. The stomach is strongly under associative relations with the sight, hearing and smell areas and its affect (psychical) reactivity is extremely intimate. Ordinary sensibility to tactile, chemical, and thermal stimuli are also lacking here, but deep sensibility fibers are present and carry pressure stimuli (pain, colic, crises). The pathways used for these are not known. It has been thought that the pathways here involved passed through the splanchnics (sympathetic). Eppinger and Hess have seemed to

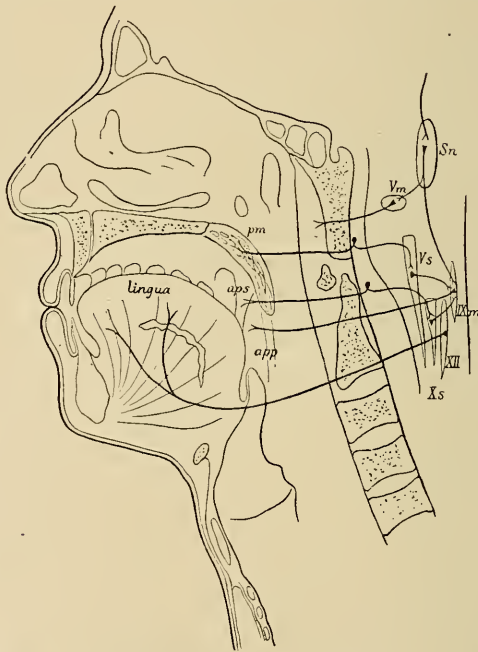


FIG. 31.—Innervation of the mechanism of swallowing: *Sn*, substantia nigra; *Vm*, motor nucleus of the trigeminus; *Vs*, sensory root of the trigeminus; *IXm*, motor nucleus of the glossopharyngeus; *XII*, nucleus of the hypoglossus; *Xs*, sensory nucleus of the vagus; *pm*, soft palate; *aps*, palatal vault; *app*, pharyngeal vault. (Bechterew.)

show that in laryngeal, bronchial, esophageal, gastric and intestinal, genital and rectal crises, the autonomic system only is involved; the vagus bulbar autonomic for the upper types, the pelvic autonomic series for the lower ones. Seen from another angle, vagus and splanchnic (?) crises are to be distinguished. The former without pain, but with nausea, hypersecretion, and vomiting, the latter with pain and hyperesthetic skin zones, and increased epigastric and abdominal reflexes.

The local topographical diagnosis and the physiological understanding of nervous dyspepsias, the motility and secretory anomalies

(achylia, hypersecretion, hyperacidity), changes induced in Addison's disease and in exophthalmic goitre and in all of which sympathetic (psychical) influences play a large role is as yet not well grasped.

Individual and social adjustment to spiritual, *i. e.*, psychical factors, seems to influence them much more effectually than measures addressed

FIG. 32

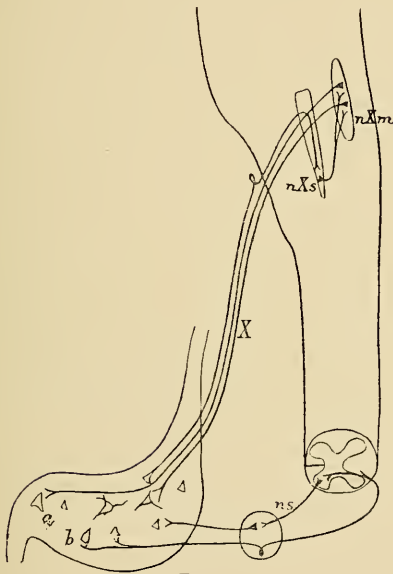


FIG. 33

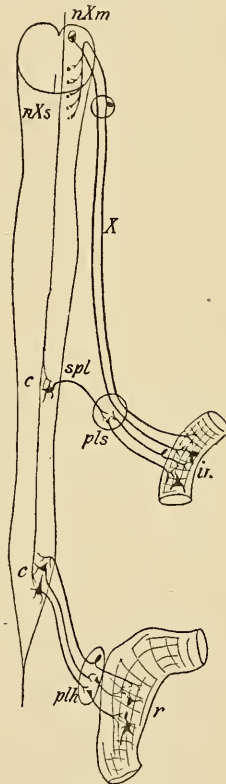


FIG. 32.—Scheme of stomach innervation: *a, b*, ganglia in walls of the stomach; *nXs*, sensory nucleus of the vagus; *nXm*, motor root of the vagus; *ns*, splanchnic. (Bechterew.)

FIG. 33.—Scheme of intestinal innervation: *in*, small intestine; *r*, lower end of the large intestine; *pls*, celiac plexus; *plh*, hypogastric plexus; *spl*, splanchnic; *c.c.*, spinal center of intestinal movements; *X*, vagus; *nXm*, motor nucleus of the vagus; *nXs*, sensory nucleus of the vagus. (Bechterew.)

to modify the perverted chemisms and motility, especially at the beginning of these disorders. There is little doubt that long continued psychical disturbances which cause very pronounced secretory and motor anomalies may ultimately induce definite organic changes. Many visceroptoses are of this type. The relaxation is due to irregularities in the reciprocal innervation of the sympathetic and

autonomic fibers, induced in many instances through psychical depression. Possibly toxic factors are important.

The vagus, by way of the solar ganglion, stimulating the sympathetic, depresses the peristalsis and secretions of the intestines. The intestinal movements, however, may take place independent of either. The tactile-mechanism-reflexes are continuously active; chemical reflexes are operative during the passage of absorptive material. Each have their sympathetic and autonomic pathways—working independently one of the other. The chief psycho-reflex pathways seem to act through the vagus; thus at the upper end such psycho-reflex activities show increase of secretion with appetite, loss of secretion with worry, fear, and, acting on the autonomic pelvic arc, the various constipations and diarrheas so frequently of psychic origin; the anal erotic and anxiety neurosis phenomena so well elaborated by Freud being among them. Purely sympathetic disturbances with increased peristalsis and serous fluid may result from loss of function of the splanchnics, either as a vital reaction to cutting, trauma, etc., or to psychical influences, as from shock, emotion, fear, and desire.

The great importance of the autonomic and sympathetic control factors on the vessels of the abdominal cavity and organs cannot be more than mentioned. Here the peripheral vascular regulations are in direct contrast with the abdominal ones, and hence the purely mechanical and vital process of adjustment of blood-pressure regulation takes place. The interpretation of the phenomena of shock must come about through a study of these factors, but such cannot be taken up here.¹

Another feature of activity of the vegetative nervous system concerns itself with the gastro-intestinal ferments, and the specific secretions or hormones (gastrins, gastrosecretine, enterokinases of the various authors). Many hormones of the endocrinous glands are thought to influence the gastro-intestinal functions. The diarrhea of exophthalmic goitre (thyreoglobulin) is adduced as a classical example of this influence. The stomach mucous membrane contains a hypothetical hormone which acts upon the activity of peristalsis (peristalsis hormone), through the sympathetic pathways. Hormonol as a definite substance has entered the therapeutic field of neurology and promises much material for speculation and interpretation at least. Direct indications are slowly crystallizing.

Rectum.—The chief innervation here is through the hemorrhoidal plexus and the inferior mesenteric. Here both autonomic and cerebrospinal influences are active. Voluntary muscle activities play a large role in defecation, the grade of tension in the rectum, however, is registered by the autonomic system, which is responsible for the original impulses, after which voluntary and involuntary activities are operative. The spinal autonomic center is located in the lumbo-

¹ Consult Crile, *Study of the Emotions*, Philadelphia, 1915.

sacral spinal segments. The cortical association connections are thought by Bechterew to be in the sigmoid gyrus. Frontal association pathways are also present, interference with which causes involuntary defecation, as with frontal tumor, general paresis, epileptiform convulsion, profound stupor, emotional loss of control, etc.

Interruption of spinal pathways may cause obstipation or diarrhea (tabes, poliomyelitis, multiple sclerosis, tumor, syphilis of cord,

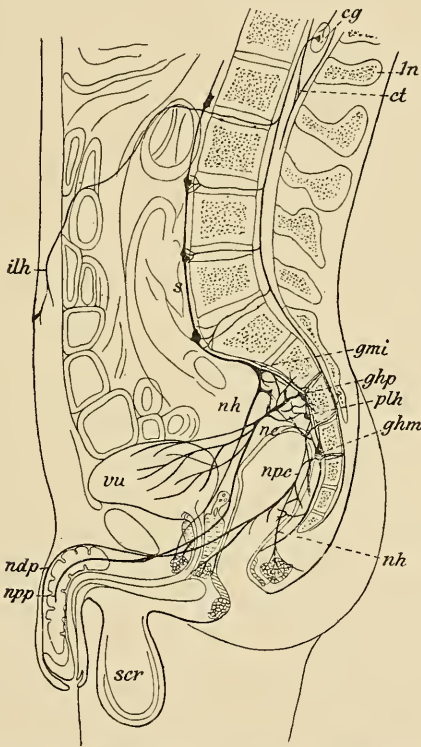


FIG. 34.—Scheme of pelvic innervation: *cg*, spinal genital center; *1n*, first lumbar; *s*, sympathetic; *ct*, conus; *ilh*, ileohypogastric; *gmi*, inferior mesenteric ganglion; *ghp*, hypogastric ganglion; *plh*, hypogastric plexus; *ghm*, hemorrhoidal ganglion; *nh* (above), hypogastric nerve; *ne*, sacral nerve; *s*, erigens; *npc*, common pudendal; *nh* (below), hemorrhoidal nerve; *ndp*, dorsalis penis; *npp*, deep perineal; *vu*, bladder; *scr*, scrotum. (Bechterew.)

hematomyelia, syringomyelia, etc.). Here deep sensibility conducting fibers—autonomic and cerebrospinal—are interfered with and the autonomic reflexes fail to establish the psychical connections either for compulsion (discharge) or control of sphincter (retention). The anal reflex here is of great localizing value, its positive appearance ruling out disease of the lower sacral and coccygeal segments. Lesions of the cervical or dorsal cord interfere with the voluntary activities of the abdominal muscles in defecation, while lesions of the lower lumbar

cord cause changes in the voluntary sphincters. In sacral lesions, with involvement of the external sphincter nucleus, the anus remains wider open, not so sharply corrugated, not as vigorous in closing and there is loss of the anal reflex. Notwithstanding the loss of the voluntary sphincter, autonomic closure is possible. There is therefore no single defecation center in the lower cord.

Severe constipation may be of purely autonomic origin. Reactions occur at physico-chemical levels, such as loss of water; at vital levels, *i. e.*, pain from kidneys, gall-bladder, peritonitis, chronic appendicitis, hemorrhoids; or at purely psychical levels, where phantasy, infantile pleasure motives, may play a large role, *i. e.*, anal erotic in displacement of affects, birth phantasies, etc. Birth phantasies which deal with feces, and which are concealed behind constipations and diarrheas are very frequent among psychoneurotics and psychotics, particularly in schizophrenics, *q. v.* The anatomical pathways which make such relationships comprehensible exist in the autonomic fibers. The pelvic autonomic and the cervical autonomic, principally the vagus, are essentially the same system, and having intimate anatomic relation, they thus afford an anatomical substratum for the general deductions of the close relation of the reproductive instincts with the vagus, anxiety states thereby coming to represent repressed libido strivings. Constipation as a correlate of miserliness is an instance in point, and is more fully discussed in the chapters on the psycho-neuroses.

Vagotonic manifestations within the gastro-intestinal tract are of considerable importance, although as yet far from being definitely analyzed. Pilocarpin and physostigmin increase them, whereas adrenalin and atropin diminish them. In vagotonic individuals there are increased esophageal cardiac spasms, tendency to increased salivation and to increased secretions from the nose and eyes. There is slowness in the peristalsis, as shown by radiosopic examination, due to increased muscular tonus. This latter causes the stomach form of hyperkinetic motility gastroneurosis. It may arise from disorder at the physico-chemical or psychical levels. Hypersecretion and hyperacidity are accompaniments with pylorospasm. Certain cerebrospinal levels seem to be involved as shown by the Head hyper-sensitive skin areas. Membranous enteritis or colitis with mucus and many eosinophile cells in the blood and mucous secretions is associated with this condition summarized as vagotonia. Here psychical influences are of great moment. The constipation just spoken of may be arranged in this vagotonic group. Renal and biliary colics, spasmodic icterus, reflex anuria, eosinophilia, and increased glucose tolerance are to be found in this vagotonic group.

Genito-urinary System.—Here autonomic and cerebrospinal controls are in evidence. The former act through the mesenteric, hypogastric, and hemorrhoidal autonomic sacral ganglia, supplying with non-medullated fibers the involuntary muscles and the mucous membranes,

N. hypogastricus to muscles of colon and bladder (sphincters), the plexus cavernosus, and nervus erigens to the genital vasomotors. The latter act through medullated fibers to the voluntary muscles and adjacent skin areas. The nervus pudendus communis supplies the external sphincter ani, external sphincter vesicæ, compressor urethræ, deep perineî, etc.

A series of autonomic reflexes are here met with, the most important being:

1. Scrotal reflex: Stroking of perineum or femoral skin; contraction of dartos.

2. Bladder reflex: Stretching or stimulus (mechanical, psychical) of bladder wall; contraction of bladder (mechanical, psychical).

3. Rectal reflex: Stretching or stimulus of rectum; contraction of rectum.

4. Genital reflex: Psychical or mechanical stimulus; erection and hyperemia; corpus cavernosus.

5. Uterus reflex: Stretching or irritation of uterus; contraction.

6. Anal reflex: Stretching of anus; psychical; contracture of sphincter ani.

All of these reflexes act through psychical levels as well as through peripheral, *i. e.*, somatic ones.

Bladder.—The general mechanisms of the bladder pattern after those of the rectum and quite homologous symptoms follow disturbances of homologous relationships of the autonomic and spinocerebral pathways. The chief autonomic series travel in the sacral vesical nerves to and from the inferior mesenteric and hypogastric ganglia. Sympathetic fibers are also functioning through the hypogastric to and from the inferior mesenteric ganglia. Thus the bladder has a vegetative mechanism comparable to that of the pupils.

Emptying of the bladder follows similar lines to that of emptying of the rectum. Section of the cord to above the mid-dorsal region brings about automatic emptying. Psychical influences are here active as in the case of the rectum—urethral erotic with retention and incontinence of purely psychical character. These are discussed under psychoneuroses, whereas the more mechanical, neurological features are taken up under diseases of the spinal cord.

Sexual Organs.—Autonomic and sympathetic supplies are present. The former carry stimuli through the nervi erigentes from the sacral cord producing vasodilatation and erection, turgor in the female, nipple erection, etc. The latter carry stimuli through the hypogastric nerve to cause vasoconstriction and contraction of the unstriped musculature of the sexual glands and discharge channels. In the sexual act, orgasm, the sexual desire, erection and ejaculation may show separate mechanisms. At the physico-chemical level the concretization of sexual desire usually reaches an active adult stage with the onset of puberty. It is assumed that chemical stimuli—hormones—act at this level to cause tension—tumescence—within the organs them-

selves, and also possibly working upon higher level nervous structures cause an increase in vital and psychical tension, thereby causing increased sensitization to sensory contacts and to mental stimuli. Thus the love impulse springs up at a touch or under the influence of a symbolic expression, as in poetry, or other artistic creation.

The whole impulse of life and of the principle of race-preservation, *i. e.*, immortality, is bound up in the instinct of reproduction. The energy of this instinct has been termed libido by various writers, by others the word is used in a wider sense, as synonymous with the life energy wherein one can distinguish a nutritive and a sexual component.

Certain hints obtained from the study of the processes of reproduction in lower organisms—protozoa, protophyta—tend to show that continuance of the life of the individual, and of the species, has been obtained through a sacrifice of the ego, *i. e.*, purely individual reproduction gave way to gametic reproduction, *i. e.*, to the principle of fertilization by sexual processes. A complete discussion of this principle cannot be given here, but it is assumed that the human species shows the same general trends with many more complicated reactions within all three levels of the nervous system. Hence the hypothesis which assumes that both vital and psychical sexual manifestations may be stimulated by purely physico-chemical determinants, such as tension within the glands, or by the activity of specific chemical substances, sexual hormones—from both testicles and ovaries—which in the ontogenesis of the individual may serve as stimuli to the development of structure, and also, given such structures, may aid in their developed functioning. Inasmuch, however, as vital energy acting solely through physico-chemical processes does not afford any adequate explanation for all and least of all for the most important of the phenomena of evolution, an adequate hypothesis must also include similar activities at higher levels, *i. e.*, vital and psychical. The out-and-out materialist stops at the lowest levels, the vitalist midway, the evolutionist argues for the leadership of the psychical, but needs the interrelationship of all. Psychical impotence with intact organs, for instance, is inexplicable on materialistic or vitalistic hypotheses.

Seen from another angle this vexed subject of interrelationships is well illustrated in the large disease group of schizophrenia (dementia precox). From the psychical side alone some have endeavored to explain it as a series of reactions to repressed and unconscious sexual activities—repressed and unconscious because of higher cultural demands and inability on the part of the patient to sublimate *i. e.*, employ his libido in its numerous useful transformations arrived at in the course of cultural development. A compromise situation adopts the interrelatory hypothesis granting a somatic inferiority of brain structure (defect or degeneration) of physico-chemical origin (dyspituitarism or other glandular defect) with a symptomatology determined largely by symbolizations of the sexuality. The Abderhalden pregnancy reaction—ovarian, testicular, hormone changes—shows a pecu-

liar activity with schizophrenics pointing to some disturbance at the physico-chemical level. This whole matter is still speculative, but affords an excellent illustration of the possible value of thinking in all of the terms proposed.

In the phenomenon of *erection* one sees these principles at work. The cerebral or psychical is the most frequent source of origin for vasodilation. The pathways are by means of the cord to the upper lumbar segments and by way of the erector nerves. In severe spinal injuries psychical erection may remain intact. Severe continuous priapism is not infrequently of purely cerebral origin, either organic as in encephalitis, non-purulent or purulent, syphilitic (paresis), or possibly purely psychical as in some manic states, some schizophrenics.

Vital levels respond to the sensory stimuli of the skin of the penis or adjacent organs, and the reflex pathways are made up of the spinal sensory nerves, the second sacral segment and the dorsalis penis and pudendis communis nerves acting through synaptic junctures in the sympathetic ganglion. Transverse lesions of the cervical dorsal cord may also induce priapism. Certain cases of encephalitis just mentioned show cervical cord lesions as well.

The physico-chemical levels respond to the tension stimuli from the bladder, seminal glands, etc., acting through the hypogastric plexus.

In ejaculation, sympathetic and cerebrospinal pathways are utilized. A summation of stimuli, acting through the sympathetic, forces the threshold, setting free a peristaltic contracture of the vasa deferentia with the accumulation of sexual secretions in the prostatic portion of the urethra. A spinal reflex causes the contraction of the bulbo- and ischio-cavernous muscles with the ejaculation of the semen.

Sympathetic disturbances are rare, spinal ones not infrequent in conus lesions, either being traumatic or due to new growth or infiltrating disease, tumor, syphilis, etc. Ejaculation in coitus, in masturbation, or in pollution dreams is usually accompanied by other autonomic signs, such as mydriasis, hyperidrosis, and cardiac palpitation. Pollution dreams have determinants at all the levels mentioned. They are usually not harmful. When frequent and evidently pathological they may arise from lower level stimuli (prostatic disease, etc.), but are more often of psychical origin—usually accomplishing the repressed and unconscious wish for culturally forbidden sexual activities (masturbation, homosexuality, Edipus phantasy). Hence their great frequency in the psychoneuroses, in schizophrenia, or in compulsive states, unless some other type of symbol carries out the forbidden and repressed wish.

Respiratory Apparatus.—Complete data are not available for definite plotting of the vegetative nerve physiology in this region. Autonomic vagus fibers, acting through the superior laryngeal, tracheal, and bronchial nerves, induce reflex coughing, inflammatory reactions with increase of mucus, etc. Somatic fibers are concerned as well. Hysterical coughing utilizes the autonomic pathways. Asthmatic attacks,

with spasm of the bronchi, difficulty in breathing, slowing of respiratory phases, emphysema, and eosinophilic sputum, are illustrations of increased vagotonia, hence relieved in part by adrenalin. Here the exciting causes may also lie at any of the three levels. Physico-chemical (parathyroid with tetany, calcium metabolism), vital (from pressure phenomena on laryngeal and bronchial nerves, reflexes from nose), or psychological (emotions, sexual excitement, repressed sexuality). The problem in treating asthma is, therefore, to find which nerve system level is chiefly implicated. Psychoanalysis would be folly for those asthmatic attacks which are due, for instance, to cheesy, tuberculous

deposits pressing upon nerve structures in the posterior mediastinum, while it alone would remedy those asthmas that are of psychological origin solely. Combined therapy—interrelational—is of greatest value.¹

Vascular Apparatus.—Only a brief outline is possible.

Heart.—Sympathetic, autonomic, and intraganglionic mechanisms are present. The sympathetic pathways arise from D 1-5. Wrisberg's ganglion is the first synapsis, the postganglionic fibers passing to the heart musculature. The vagus is active through three main branches, one arising below the superior laryngeal, a second from the recurrens, a third from the thoracic part of the vagus. The deeper layers of the heart are supplied through the right vagus, the superficial cardiac plexus supplying through the left. The sympathetic fibers which accelerate the heart's action are in relation with the extra cardiac ganglia, the end branches of the vagus in connection with the intracardial ganglion, *i. e.*, cells. These

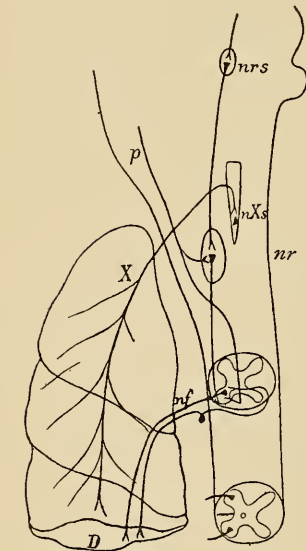


FIG. 35.—Scheme of innervation of breathing: *D*, diaphragm; *nf*, phrenic nerve; *X*, sensory vagus branches to the lungs; *nr*, respiratory nucleus in medulla; *nXs*, sensory nucleus of the vagus; *nrs*, respiratory center in midbrain region. (Bechterew.)

intracardial cells are here assumed to be visceromotor, and are thickest at the origin of His' bundle, Tawara's nodes and at the origin of the Keith-Flack bundle. The activity of the vagus upon the muscles seems to manifest itself chiefly through the ganglion cells.

The gray matter of the midbrain in the neighborhood of the floor

¹ Cheyne-Stokes respiration is found in a great variety of pathological states such as high cervical myelitis, hemorrhage of the medulla, hemorrhage of the base, tumors of the midbrain region, and occasionally in certain cortical atrophies or hemorrhages. It is rarely present in certain hysterics. Snorting, barking, coughing, sneezing, hiccupping and yawning are frequent respiratory affections. They are for the most part psychical, but not always.

of the third ventricle is thought to be a higher coördinating switch-board—the nucleus dorsalis vagi, an end station. Through this portion of the mechanism, psychical influences are switched in, modifying the tonus through emotions, pain, and local stimuli.

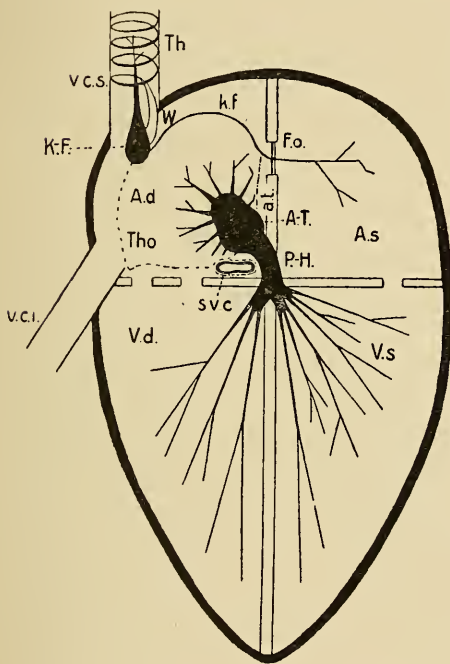


FIG. 36.—Scheme of cardiac innervation: *A.d.*, right auricle; *A.s.*, left auricle; *V.d.*, right ventricle; *V.s.*, left ventricle; *v.c.s.*, and *v.c.i.*, superior and inferior vena cava; *s.v.c.*, sinus venosus; *F.o.*, foramen ovale; *K.F.*, Keith-Flack sinus node; *A.T.*, Aschoff-Tawara auriculoventricular node; *Th*, Thorel's superior vena cava bundle; *Tho*, Thorel's intermediary node bundle; *W*, Wenckebach's auriculoventricular bundle; *k.f.*, Keith-Flack fibers between *K.F.* and *F.o.*; *a.t.*, Aschoff-Tawara bundle between *A.T.* and *F.o.*; *P.H.*, Paladino-His bundle. (Janowski.)

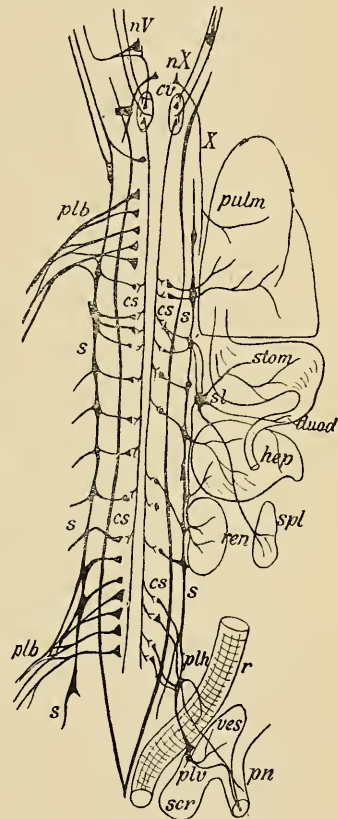


FIG. 37.—Scheme of innervation of the vasomotors: *cv*, main center of vasomotors in the medulla; *cs*, spinal vasomotor centers; *s*, sympathetic; *nX*, vagus nucleus; *X*, vagus; *nV*, trigeminal nucleus; *plb*, brachial plexus; *plv*, vesical plexus; *pulm*, lungs; *stom*, stomach; *duod*, duodenum; *hep*, liver; *spl*, spleen; *ren*, kidney; *r*, rectum; *ves*, bladder; *scr*, scrotum; *pn*, penis. (After Langley.)

Bradycardia appears through a number of influences, chiefly following acute infectious intoxications, by increased intracranial pressure, in hypothyroidism, digitalis and allied glucosidal actions. Trigeminal reflexes through the nose, eyes (pressure) may also cause brady-

cardia. The various arrhythmias, dislocations, and blocks cannot be discussed here.

The relation of changes in or due to His' bundle cannot be entered into here, although they may properly be discussed in a text-book on neurology.

Angina pectoris, in some of its forms at least, is due to autonomic overstimulation whereby vascular cramp states are brought about; vagus paralyzing and vasodilator drugs, therefore, aid in overcoming the condition, particularly in the vasomotor types of angina. Cardiac discomfort so frequent in visceral heart disease, as well as in psychical disorders is carried to consciousness chiefly through communicating sympathetic branches through the spinal ganglia, or directly to the spinal systems. Head's hyperalgesias are explained in this manner. The vagus (autonomic) fibers are not implicated.

Bloodvessels: Vasomotor Neuroses.—The anatomy, physiology, and clinical disturbances of the bloodvessels make a large chapter in contemporary neurology. Cassirer has devoted a monograph of 1000 pages alone to their consideration. Only the briefest sketch is offered here.

The bloodvessels of the face are innervated from the upper cervical sympathetic fibers passing over the internal carotid plexus to the Gasserian ganglion, and with the pathways to the sweat glands pass with the sensory fibers of the face. Those of the upper extremities are supplied chiefly from C5 to D7, mostly leaving by way of the D3 to D7 root segments. Those of the lower extremities arise from D12 to L3. In the spinal axis are located only the local segmental functions. Bulbar centers are present in the nucleus dorsalis vagi, which is an autonomic center for peripheral vessels as well as those of the intestines. Stimuli in the bulbar centers tend to cause contraction of the peripheral vessels and dilatation of the visceral ones. Intracranial bloodvessels have vasodilator and vasoconstrictor pathways, possibly conveyed through the cervical sympathetics.

Cortical centers have been placed in the frontal areas (Lewandowsky, Weber; denied by Müller and Glaser, who claim the midbrain as the highest center), from which the pathways pass through the internal capsule, caudate nucleus, thalamus, hypothalamus, pons, central gray of fourth ventricle, oblongata, Helweg's triangular bundle, anterolateral bundle to lateral horns—the fibers crossing in the posterior commissure (Helweg). The autonomic and sympathetic fibers apparently follow different pathways from the cord, the vasodilator autonomic pathways following the course of the sensory roots, the vasoconstrictor sympathetic by way of the anterior roots, the motor nerves, and the sympathetic ganglion. Thus irritation of the posterior roots causes hyperemia (vasodilatation) with pain; paralysis of the same causes anemia with anesthesia.

Within the bloodvessels themselves ganglion cells are found, save perhaps in those whose vasomotors run in the spinal nerves (Müller

PLATE II



Erythromelalgia. Foot after hanging down for twenty minutes. (Mitchell.)

(Case of Dr. J. M. Taylor).

and Glaser), and reflexes occur here exactly as in all of the skin and tendon reflexes from terminal stimuli. Hence an analysis of vascular disturbances must include a study of the sensory, motor and central portions of the reflex arc—the last including both medullary and corticospinal reflex pathways.

VASOMOTOR NEUROSES.

Disturbances of the peripheral mechanisms of the vasomotor pathways have been more completely analyzed than those occurring in the spinal, bulbar, thalamic, or cortical portions of the same. Of these, more detailed mention may be made of the: (1) tonic hyperemias (erythromelalgia), (2) spastic anemias (pseudosclerosis, Raynaud's disease, migraine, intermittent claudication), and (3) vasomotor irritability, as in acute angioneurotic edema, multiple gangrene of the skin, etc.

Physiological alterations in the tonus of the peripheral vessels are seen in sleep, emotional states, active digestion, overexercise, overheating, in collapse, and in fatigue states.

1. Tonic Hyperemias.

These consist in prolonged irritability of the peripheral vasomotors. In certain individuals (sympathicotonic) a diminished alkalinity of the blood is thought to bring about such a stimulation of the sympathetics. These tonic hyperemias are seen more particularly in neuralgias, neuritides, in infections, or toxic erythemas, and reach a pronounced grade in the syndrome known as erythromelalgia.

Erythromelalgia.—Two main trends may be distinguished—those with pain as described by Weir Mitchell, and those without pain but with hyperidrosis and hyperalgesia (Hess).

Weir Mitchell, in 1878, described a paroxysmal disorder of the extremities which was marked by a painful redness and swelling of the feet. Lannois, in 1880, wrote an important monograph on the subject, and Cassirer, in the second edition of his *Vasomotorisch-trophischen Neurosen*, 1912, has given a complete description of the general group to which the name erythromelalgias may be given. He was able to gather reports of about 130 cases. One may conclude it to be rare. Only 2 in Oppenheim's 25,000 dispensary patients are reported, while in Jelliffe's statistics of Starr's dispensary service of 18,000 patients 21 were observed, 15 in males and 6 in females. It is more often observed in the later years of life, although six-to-ten-year-old (Baginsky) patients are recorded.

Causes are difficult to run down. Thermic influences apparently play some role as exciting agents at most. Psychological factors may determine an attack.

Hypothetically erythromelalgia is a pure sympathetic affection, an angioneurosis, due to prolonged sympathetic stimulation. Prac-

tically it shows itself in combined forms, being an accompaniment of spinal disorder (involving the sympathetic cell groups) in multiple sclerosis, and in tabes; it may occasionally be seen in cerebral disorders, hemiplegia, thalamic involvement, or may be a part of a peripheral nerve disorder, accompanying a neuritis, or it may be a cause of or a part of a chronic vascular disease of an obliterating or spasmodic type. In each of these the chief action is directed upon the sympathetics. Thus a number of gradations and variants exist which are discussed in the works already cited, particularly in Cassirer and Oppenheim.

Symptoms.—The chief symptoms are heat, redness, and pain in the extremities, either localized along a definite nerve distribution, often following a root area, or peripheral. It is usually intermittent, worse at night, and the patient suffers tortures. Heat usually makes it worse, so also does movement, especially walking, whereas any position reducing passive congestion, thus overcoming the tonic hyperemia, affords relief. Severe grades of the disorder show a purple cyanotic skin, with erythema, usually due to transudation following stasis from slowed circulation in the area of vasodilatation. Hyperidrosis may be present.

Accessory symptoms, such as headache, palpitation, and fainting, are reactions to the pain, and in part to the fear, or may be another facet of a psychoneurosis in which the erythromelalgia is also a symptom. Trophic changes in the skin, hair and nails may take place, which are either a part of the sympathetic disturbance itself or are results common to the angioneurosis, and a producing or accompanying lesion—tabes, multiple sclerosis, paresis, etc.

Course and Therapy.—The outcome depends much upon the causation. An erythromelalgia due to spinal changes may get better if these do (syphilis) or not (tumor, multiple sclerosis). The therapy will be determined by the cause. Palliatives, such as the use of high frequency current, violet rays, cold, antipyrin, are valuable as well.

A neuritic erythromelalgia will improve or not as the neuritis does; similarly an arteriosclerotic one; but it usually gets worse. The therapy is for the more fundamental condition. A psychically determined erythromelalgia, possibly a hysteria, needs psychoanalysis.

2. Spastic Anemic Group.

Here the chief results are due to persistent or intermittent vasoconstriction. The syndromes are numerous and confusing, but among them a few are sufficiently distinct or constant to be given diagnostic titles such as Raynaud's disease, intermittent claudication, migraine, pseudosclerosis, asphygmia alternans; etc. Only the chief types can be taken up; the purely tentative nature of the classification must be emphasized.

Raynaud's Disease.—Raynaud's disease is also known as symmetrical gangrene, local asphyxia. This syndrome, like the preceding one, may be of many origins. It may be psychological (shock, hysteria, schizophrenia), cerebral, or spinal organic (capsular (thalamic) hemorrhage,

PLATE III

Fig. 1



Fig. 2



Raynaud's Disease, showing superficial gangrene. (Osler.)

trauma, paresis, multiple sclerosis, tabes, syringomyelia, tumors), or peripheral in nerve or bloodvessels, neuritis of all various etiologies, arteriosclerosis either peripheral or of the large vessels (aorta).

It may readily be seen that from such a polyetiological viewpoint there is no true Raynaud's disorder. Hence, Cassirer's attempt to make true sympathetic types and those due to complicating disorders, such as the local gangrenes due to diabetic neuritis, or to arteriosclerosis, etc. Even this is difficult to accomplish. Thus a spinal gliosis (syringomyelobulbia) may invade the sympathetic cells in a given segment and bring about a local paroxysmal gangrene. This is truly a sympathetic affection, but it is to be considered a part of the syringomyelia just as much as it is to be considered Raynaud's disease, even if it happens to be the very beginning symptom of the syringomyelia.

Symptoms.—The attacks are paroxysmal. The fingers or toes begin to get cold, and have the feeling of prickling and of going asleep. They become pale and waxy from the vasoconstriction. Pain is frequently felt and local coldness is present. An attack of this kind may come and go in a few hours.

More persistent attacks lead to more marked grades of local asphyxia, with cyanosis, or bluish-red discoloration of the extremities. Pain is extreme. Vesicles may form—the fingers may even get bluish-black—and all gradually disappear after a few days, or leave slowly healing broken vesicles, or more deeply lying trophic ulcers (protopathic nerve fiber injury). Other types of sensibility also suffer. Epicritic touch and thermal as well as protopathic pain, thermal and deep sensibility, may also be involved. Gangrene is a severe grade with loss of fingers or finger tips.

Accessory symptoms (such as trophic changes in the nails, in hair, in the bones, etc.), which are due to the different etiological factors, syringomyelia, neuritis, arteriosclerosis, etc., need not be entered into. Attacks, with recovery, may persist as long as three to four months.

Treatment.—The therapy is often without avail, as the underlying condition is unmodifiable (syringomyelia, multiple sclerosis, etc.). As a rule, however, the attack subsides, although to appear again. Then attention should be addressed to the general health of the patient, especially to emotional features which produce vascular instability. Mild massage, local warmth and Bier's hyperemic treatment are of value during the attack, strong analgesics being necessary for the pain at the time. In psychogenic cases psychotherapy is alone available.

Intermittent Claudication.—This is an angiospastic syndrome and rests upon a number of foundations. Clinically it consists of a spastic vascular state with weakness, pain, and coldness in the affected region. In the majority of cases it appears in the leg or legs. After the patient has walked, perhaps rapidly, the leg or legs begin to be fatigued, and commence to feel numb and painful until it is impossible to keep up the pace or walk at all. After a rest, the patient may resume his walk for a time free from distress, but the state of pain and fatigue recurs to be again relieved following rest. There is a later tendency for the

state to recur when the limbs are at rest. Cyanosis, coldness, paleness are accompanying phenomena. There is mild hyperesthesia of the affected part but no other sensory signs. The chief vessels may be pulseless. These should be tested by touch and the eye aided by the sphygmograph.

The chief sites are the vessels of the legs but the arms may be involved. Any muscular group may show the symptoms. Lumbago-like forms occur in the back muscles. The vessels of the intestines, internal organs, brain, and spinal cord may be involved.

The chief lesion is arteriosclerosis, but others are operative. The arteriosclerosis itself may be secondary to syphilis, alcoholism, to chronic nicotine poisoning.¹

Oppenheim has called attention to the frequency with which these arterial changes are found in Russian Jews. This disorder is mostly confined to the men of this people. Here flat foot probably plays a role—excess in walking (peddlers) may aid. Psychoneurotic factors also may play a part in the causation of these arterial cramps independent of any definite arteriosclerosis. The complicated question of altered chemism within the vessel walls cannot be entered into. Hereditarily inferior vascular systems are factors.

Therapy.—Rest, warm applications to the parts, and high frequency current application are of value in treating the attack in its acute stage. Treatment of the condition rests upon the proper conception of the individual provocative disorder. Arteriosclerotic cases need treatment for this; psychoneurotics require psychotherapy. Of the more fundamental therapy of the vegetative system which permits the spasticities as well as modifies the calcium metabolism in the vascular walls nothing as yet can be laid down.

Ophthalmic Migraine.—This is also known as sick headache; megrims; hemicrania; bilious headache.

This protean affection is difficult to define. It may be a simple or an extremely complex condition. Migraine may, however, be defined as a periodical abnormal state in which the patient suffers from a peculiar oppressive pain in the head, unilateral or bilateral, localized or general, which develops very gradually from heaviness to dulness, to pain that is splitting, and is accompanied or more often preceded by characteristic visual signs, such as scotomata, flying specks, or partial blindness. Chilliness, depression, and sensory disturbances, particularly in the stomach, and which may lead to nausea or vomiting, are also usually present. An attack may be terminated, after a few minutes, by vomiting, or it may persist hours or even days. After a variable length of time, usually following a heavy sleep, the patient regains his previous condition of well-being. Nearly everyone has an attack or attacks of migraine during a life-time, hence its extended description here.

¹ Frankl-Hochwart, Deut. Zeit. f. Nervenheilk, 1913, vol. xlvii and xlviii.

History.—A heritage of the rich and the poor, the great and the small alike, it has numbered among its sufferers many of the master minds of all times, and no disorder can vie with it in richness of description from medical writers who have been themselves subject to its vagaries.¹ Aretaeus is credited with having given the first description of migraine. Celsus gave a description which, while not corresponding in many details with what is now understood to be migraine, is nevertheless very suggestive. Caelius Aurelianus noted for the first time that the Greeks called it hemicrania. Lepois, in the seventeenth century, gave his personal experiences through fourteen years, and called attention to the fact that the usual after-effects of vomiting and sopor might come on without the presence of the headache. Wepfer in the same century seems to have more clearly appreciated the eye symptoms.

Tissot's description, 1784, remained authoritative up to the appearance of Liveing's monograph, "On Megrin, Sick Headache, and Some Allied Disorders" (1873), although in the interim the symptomatology was becoming richer and the case analyses more exhaustive. Thus, Vater, Henricke, and Heberden made observations upon the scotomata. Plenck, Parry, Wollaston drew from personal experiences the picture of half-sided blindness. Schönlein and Romberg introduced the neuralgic theories, while Dubois-Reymond, influenced by the newer work of Claude Bernard, developed the hypothesis of arterial spasm which Möllendorf controverted, and postulated a sympathetic paralysis, both of which views were conciliated by Jaccoud and by Eulenberg (1867), who described angiotonic and angioparalytic conditions.

Etiology.—The general hypothesis of a vasomotor disturbance acting through the vegetative nervous system, seems to account for most of the facts, and the general position here taken is that such disturbance may be conditioned by a host of causes. The view advocated then admits that a certain amount of fact exists in practically all of the hypotheses but maintains that a one-sided mode of interpretation is inadmissible.

Abortive Attacks.—Incomplete or abortive attacks may be said to be the rule rather than the exception and attempts to classify the disorder according to the number of symptoms present offer no help in the understanding of the complete picture.

Möbius suggests that the parents of patients suffering from migraine with scotomata often have suffered from migraine without scotomata, but he also speaks of the reverse as happening. The extreme prevalence of migraine makes many of the conceptions regarding its necessary hereditary nature very dubious, and the extreme variability of the individual attacks in the same patient makes general hereditary features extremely improbable. It is by no means infrequent to find patients that show at one time or another almost every symptom mentioned in the voluminous literature of migraine. Thus, one patient

¹ See article by Jelliffe: Osler, Modern Medicine.

under personal observation, had about two attacks weekly for a year. He then went two years without a single attack, and he then had several severe ones with aphasia and psychical symptoms, interspersed with abortive attacks, with hardly any two alike. He was a veritable museum of migraine attacks in the fifteen years that he was under observation.

Many families are known in which both parents have been sufferers from chronic migraine for years, and yet none of the children, now in some instances over forty years of age, have ever had more than one or two attacks. The high percentage of incidence makes it almost impossible to calculate an hereditary factor. Again, it may be borne in mind, that as there are many kinds of epilepsies, so also there are undoubtedly many migraines. Some are due to hereditary anomalies, while others have nothing to do with anything of an hereditary character. Thus, one can speak of migraines that are possibly hereditary and others that are not.

The commonest abortive attacks are those that begin in the classical manner, with chilliness, perhaps with pinched face, and cold extremities. The patient then has the scotomata and wretchedness, depression and apprehension, and then while waiting for the headache the patient notices that it does not come, and, although he may still have heaviness and a sense of discomfort, the feeling of relief is sufficient to make him feel well.

Others have added the sensation of prickling in the fingers, numbness in the hand or arm, or other sensory disturbances without the headache. In some the entire attack will consist of a disturbed painful sense of discomfort, without sensory symptoms, scotomata, or headache, but they feel sick at the stomach, and have an attack of what they term "biliousness," which clears up after vomiting. This feeling will recur with sufficient frequency, and at times be combined with such other symptoms of a migraine attack, in its varying aspects, as to stamp the whole process as a variant of a true attack. Isolated attacks of vomiting as the sole expression of a migraine are known.

Attacks of scotomata occur alone, without antecedent distress, and no after-attacks are noted. These are most uncommon. Historically it may be noted that Parry and Airy had such attacks. It is highly probable that the majority of patients who have had many migraine attacks will have had some of this nature. Attacks of scotomata and vomiting occur without headache. In many on the contrary headache is the only symptom.

Some patients have attacks of hemiparesthesia with no other symptoms of migraine. These generally occur at night, and usually follow severe mental exertion; in one patient under observation a severe ordeal in playing a difficult piece of music will bring on such an attack without other signs. This patient's severe attacks are very extreme, being associated with hemiedema, hemiparesis, hemianesthesia, and marked hysteromaniacal outbursts.

Under the heading of equivalents, Liveing speaks of stomach attacks associated with some of the vascular phenomena of migraine; glossal spasms are also mentioned by him. Attacks of giddiness, vertigo, intestinal colic, mental anxiety and depression which occur periodically in partial association with migraine symptoms, are also noted as equivalents. There is need of further study of these isolated phenomena associated with vasomotor disturbances.

Attempts have been made to determine the relative frequency of migraine attacks with and without the visual signs. These are not over-reliable, because of the vast preponderance of abortive attacks over those of the complete classical type.

Möbius expresses the opinion that the percentage of visual accompaniments of the attacks is usually overstated. His statistics show 130 cases, with 14 visual aura. In Liveing's 60 patients, 37 suffered from scotomata. Gowers says that the cases are about half and half, with and without eye signs. Galezowski maintains that the visual aura migraines appear later in life, thirty to fifty years, than ordinary migraines.

It is difficult to state an individual position, the results of personal inquiries having been so diverse. Close questioning has revealed the fact that at some time or other in the course the majority have had visual symptoms, and it is not improbable that the usual statistics are largely derived from studies of too few attacks, *i. e.*, largely from the severer attacks only. Some notes on individual histories are of interest. Several patients have kept fairly accurate records of their migraine attacks for several years. One shows 168 attacks in a period of about ten years; of these, about 100 were abortive attacks, the vast majority of which, 60 per cent., consisted of scotomata alone. Of the 68 remaining attacks, about 50 per cent. were ordinary hemicrania, lateral or bilateral, without scotomata, the others ophthalmic migraine, usually unilateral and with scotomata. Not one of the attacks was ever accompanied by vomiting. Two were associated with aphasia, fifteen with sensory tactile associations; there were five or six attacks of hemiparesis, one in the day-time, the rest at night. Spasms of the orbicularis were a common accompaniment. Every attack sufficiently severe to require an analgesic was promptly relieved by from 5 to 10 grains of either antipyrin, acetanilid, or phenacetin.

Classical Migraine.—*Early Symptoms.*—These may be termed precursors of a full attack of migraine, or they may constitute the symptoms of an abortive attack. The most striking are a sense of heaviness, with yawning, chilliness, dizziness, or depression, motor twitching, even sharp spasmodic closure of the eyelids, sensory phenomena, chiefly paresthesiæ, occasionally anesthesia, and affections of the eyes or other sensory organs, ringing in the ears, blowing, whistling, modifications of taste, of smell, of touch, etc. There may be failure of appetite, constipation, diarrhea, vascular instability, hot flashes chasing here and there over the body, throbbing in the carotids, etc.

The temporal arteries are often smaller, the saliva diminished, and the pupils narrowed.

The premonitory signs which show a great deal of variability in different individuals, and also in different attacks in the same individual, may be felt several minutes before the attack, in some rare instances even days. This is frequently the case in women in whom the onset of the menstrual function seems to bear some relation to the attack. The ordinary depression felt at this time is a thing apart from this special type of depression that pervades them. At times such attacks of depression and anxiety, combined with a sense of chilliness and dizziness, will constitute the entire picture of the abortive attack. Many attacks come apparently without the slightest warning.

Many patients having attacks at night find themselves heavy, and tired, with sore spots on the scalp in the morning. Möbius relates a case in which the patient dreamed of having swallowed a rabbit, which ate its way out through the stomach wall. After this unpleasant dream the patient had a severe migraine on awakening.

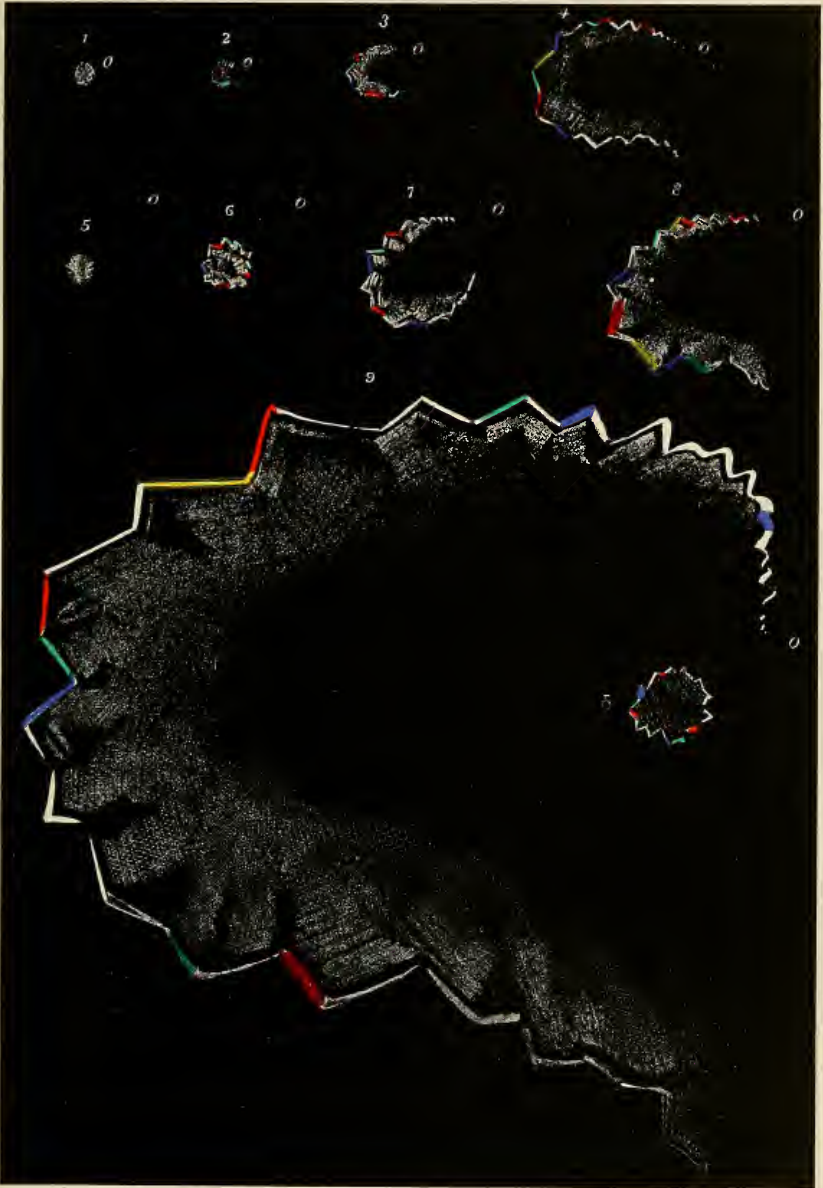
In all probability premonitory symptoms of some type are invariably present; when thought to be absent it is because the patient has overlooked them, either by reason of their mild character, or because of naturally poor powers of observation. Many patients, who have had headaches for years, have never noticed their one-sided localization, or the well known fortification spectra, until their attention has been directed specifically to them. Many patients will deny ever having had zig-zags of light, etc., until shown Airy's pictures, when they remember having seen such phenomena. It is because of such poor observation that many cases of true migraine are overlooked, which fact lends further support to the belief that this disorder is very much more prevalent than is usually supposed.

Sensory Symptoms.—In the more classical attacks the patient has preliminary sensory symptoms. These are spoken of by Möbius in the sense of an aura. If the term aura be used as, for instance, the term "fever" is used, there can be no objection, but if by an aura is meant a restricted phenomenon essentially related to an epileptic aura, the term should be eliminated.

A sense of coldness and chilliness is one of the commonest sensations. This is usually general, and is associated with a pale countenance, gooseflesh, perhaps clammy hands, and a sense of misery. Cases are known, and are by no means uncommon, in which the chilliness has been one-sided, and is accompanied by other phenomena involving one-half of the body, including the face, of the same side. Yawning is a common early sign.

Unilateral paresthesia is not an uncommon early sign. Many patients note a tingling or numbness in the fingers of one hand; this may spread up the arm, and in rare instances general unilateral pares-

PLATE IV



From Dr. Hubert Airy's Paper, On a Distinct Form of Transient Hemipopsia, *Philosophical Transactions*, 1870, p. 247.

Figs. 1 to 4.—Early stages of Fortification Spectra as seen in dark. O=sight point.

Figs. 5 to 8.—Similar series, beginning lower.

Fig. 9—Fully developed. ψ =secondary attack within.

thesia of a very uncomfortable nature may be present. In some instances such unilateral paresthesiæ have constituted the sole symptom of an attack, save for the heaviness and usual discomfort. Occurring at night, such attacks are often extremely wearing, keeping the patient awake. Photophobia, flow of tears, strange sounds, tinnitus, peculiar odors, queer peppery or flat tastes, may be noted.

Anesthesia is less often observed, largely because of the negative character of the symptom. When involving the face or mouth it is complained of. Anesthesia frequently follows the tingling of the early paresthetic disturbances. Franz¹ has shown that there is a very evident decrease in the pain threshold, especially after the headache has set in.

The *visual phenomena* are the most striking, and hence held to be of the most frequent occurrence. The ease of observation in part accounts for the usually accepted opinion that they are the commonest of the early symptoms. Very few individuals have been subjected to a careful sensory examination. If more were investigated, it is probable that other slight sensory signs would be found to be equally prevalent and as evanescent. The visual signs have been described by many writers, and many illustrations have been made showing their chief characteristics. The extreme uniformity of their general character is striking, as well as the variations of the same pattern.

As a rule, the patient notices a slight blurring of his vision if reading, or a slight flicker of light located in one eye, to one side of the center. Closer observation reveals either a slight cloudy spot, which seems to follow the eye in reading, cutting out the after-images, a letter or so from the center of clear vision. The slight subjective sense of difficulty in reading may precede the discovery of a scintillating spot which becomes visible on closing the eyes. Little by little this spot spreads out, usually in a crescent-like fashion. General statistics are thus far unavailable, but a special study has shown that the majority of these scotomata have begun in the left eye, are situated to the left of the middle line, with the convexity of the crescentic border to the left. As the crescent gradually grows larger, the difficulty in seeing clearly becomes more marked, especially on the periphery of the visual field. For most, the scotomata is in constant motion, flashing in its spectral zig-zag fashion, thus causing the classical name "fortification spectrum" from the play of colors, and the fortress-like "ins and outs" of the outline.

After a variable time, from five to twenty minutes, the scotomata gradually subsides, or suddenly disappears, to be followed by the headache. Not infrequently the headache never comes, and the preliminary sensory phenomena of chilliness, heaviness, and scotomata constitute an abortive attack. A description of the scotomata of migraine might fill a volume. The classic of Living reproduces the excellent illustration of Airy's, which is here reproduced.

¹ Amer. Jour. of Physiology, 1905.

Occasionally the right half of the field is involved. Sometimes it is the upper half, one of Möbius' patients saying that everybody seemed

THE DEVELOPMENT OF SCOTOMATA IN MIGRAINE.

have not studied immigrants into which immigrants desire for larger opportunities always been the prime force. This has proved any regulation of immigration for the selection of immigrants desirable it is to appreciate colonization and to examine their place in the causes at

There has been no such the United States as they longing for liberty and for large immigration at settlement of America; Island in the present time and the frontiersman of that a very large number lured by purely economic rights, many immigrants

FIG. 38.—Stage of blurring three minutes.

questions is usually introduced were immigrants and the children following the Colonial period, their kinsmen that immigrated the West and that we are indeed industrial and commercial elements are and it is difficult owes to immigrants. It is, however, of present immigration as still inspired by intense longing to find, it even at the cost of the part of the civilization of the period and in the years following

FIG. 40.—Five to ten minutes growing scotoma.

of arrivals at Ellis Island in a single month. The addition of these 125,000 aliens the racial or economic changes. Nearly those who had come before and many of ship as well as those with whom they the next twenty years there was a steady of immigrants until the latter part of in Ireland in 1840 suddenly caused a large illustration of an important new social in Europe and a definite relation to the America. It was many years later than as developed by immigration—that economic definite relation to the volume of immigration the number of immigrants in this country who in Europe. The exodus from Ireland was on account of the failure of the potato considerable extent by philanthropic individuals it was influenced, too, by the same motive

FIG. 42.—Fifteen to twenty minutes.

headless; occasionally, it is the lower. In rare instances the patients complain of total blindness, *i. e.*, central scotomata. Berbez reports

FIG. 39.—First outlines of scintillations.

Discussion of immigration statements that our forefathers if immigrants, that the century new generations coming to join the back the Indians and people to immigrate for our present For the most part these state the debt which this country a very serious one to conceive continuation of that movement, al liberty and determination to d privation, which transferred a world to the New in the Colonial Revolutionary War. Such a con-

FIG. 41.—Ten to fifteen minutes.

Commons has pointed out, the English, Dutch who constituted practically all arrivals in the Revolutionary War were close, they had been one Germanic race in the North America. They colonized the Atlantic and helped form the thin line of civilization were of the same race. The "immigrants" many years after the Revolutionists than those who had preceded them which we have considered as that of continuation after the Civil War. During that period about Europe. It is not possible to learn the date to this country before 1820 for in that time we commenced to record their number and to as them. We know, however, that the population was not materially increased by immigration following the Revolutionary War. During

FIG. 43.—Just before disappearing, twenty to thirty minutes, and beginning of headache.

an interesting case of a ring-like scotomata—the patient, on looking at his watch, could see only the central pin where the hands were united; the figures on the dial were all obscured by the scintillating scotoma. These scotomata are usually bilateral phenomena. They may begin in one eye before appearing in the other, and be somewhat different in the two eyes, and may disappear in one eye sooner than the other. Scotomata limited to one eye are probably rare.

The relationship of these scotomata to psychical symbolizations has not yet been investigated. In the few cases thus far analyzed, left-sided symptoms are apt to symbolize the love conflicts, right-handed ones, the nutritive. Pains in the back of the head are frequently associated with hate complexes, as are also jaw pains.

The retinal occurrences during the time of these scotomata are uncertain. Blanching of the papillæ has been observed by some (Galezowski); pulsation of the retinal arteries, with dilatation by others. Personal experience has shown similar dilatation in a few cases, but, as a rule, a normal fundus is found. The picture seen will depend upon the stage of the attack and its severity.

Pupillary dilatation occurs late. Slight irregularity of the pupils during a severe attack of an ophthalmic migraine, dilatation being usual on the affected side, is not unusual. Bilateral pupillary contraction is the rule in the headache stage.

During the onset of the fortification spectra it not infrequently happens that mild motor phenomena occur in the eyelid of the side to be affected. The eyelid droops a particle, and Gowers and others report double vision, interpretable as a sign of paresis in an ocular muscle.

Motor Disturbances.—Speech.—This may be considered as both a motor and sensory phenomenon, for the most frequent type of change is a transitory sensory aphasia. Anarthrias are known, especially in the ophthalmoplegic variety, but for ophthalmic migraine the type of aphasia found is very characteristic. As described by Charcot, it is an intermittent, halting aphasia. At one moment the patient can get the right word, at the next he cannot. He stumbles on a word; uses madame for monsieur, etc. In Liveing's cases, 15 out of 20 had speech disturbances; one on hearing clock bells was unable to inquire what they were. Féré cites the case of a coachman who forgot where he was going to drive his passengers; Berbez a like case in which a pedestrian lost his way, as he could not read the street signs understandingly. Gowers speaks of a case of word deafness. Cases of agraphia are also known. Möbius reports a case with typical scintillating scotomata at one time on the right side, at another on the left. When the patient suffered from a right-sided scotoma he had sensory aphasic signs, but they were not present when the scotoma was on the left side.

Other observers have noted the same phenomena, while contradictory observations are also recorded. The speech disturbance

sometimes resembles a paraphasia, the patient using a jumble of words. In a personal case the patient could not sing a well-known tune correctly, his sense of musical values having been interfered with.

The onset of the aphasic disturbance may vary greatly. It is usually temporary, persisting at times for only a few minutes, again persisting a few hours. It frequently antedates the headache, or is coincident with it. In a case reported by Meige the aphasia persisted as long as the headache, and disappeared, as a rule, when that disappeared. The patient showed a loss of ability to say certain words and a tendency to the employment of incorrect words. There was no anarthria.

Cerebellar Symptoms.—Oppenheim has called attention to a cerebellar hemicrania in a patient in whom every attack of migraine was accompanied by typical cerebellar symptoms. The patient was uncertain in his gait, walked like a drunken man, was dizzy, and had the sensation that his body, or individual parts of it were doubled. The sense of equilibrium was disturbed in each attack. Dizziness and loss of the sense of equilibrium are not infrequent but such a complete syndrome has been described only by Oppenheim.

Paralytic Phenomena.—Attention has already been called to the rare occurrence of hemiparesis, which may even involve the facial muscles. Up to the present time no instances of crossed hemiplegic types have been found in the literature. This is of interest in connection with the hypothesis of the bulbar origin of migraine, especially of the ophthalmoplegic variety.

Headache.—This is the most common feature and exhibits a great amount of variability as to location, quality, intensity, and duration. In the more classical attacks the headache begins on the average about fifteen to thirty minutes after the appearance of the scotomata or other sensory phenomena. It frequently begins on one side, and may remain so or become bilateral. As a rule, it is frontal, or occupies the vertex, but may involve the temporal regions, the occiput, sometimes as low down as the neck. Gowers' experience points to the parietal region as being oftenest affected, and usually over a small area. Henschen, in 123 patients, shows the pain to have been located 110 times in the forehead, 100 times in the parietal region, and 54 times in the occiput. There is usually pain over the eyes, and the eyeballs are usually painful to pressure. In a few instances pressure over the malar bones is painful, and occasionally there is a well-marked jaw ache.

Statistics of the percentage of different locations are uncertain since the individual will have all the different varieties. Thus, in a case already cited, in which the abortive attacks were so frequent, the headaches comparatively rare, the strictly unilateral headaches were only 5 per cent. of the entire number. In others the hemicranic type runs much higher. In Henschen's records of 123 cases, 56 had one-sided attacks, in 67 both sides were involved. In Liveing's

61 patients, 17 had one-sided attacks, in 7 the attacks were variable, while in 34 both sides were involved. Möbius and others note that the headache often appears on the side opposite to that affected by the sensory aura. Personal studies do not confirm Möbius' statement. It does seem, however, as first noted by Liveing, that one-sided sensory symptoms are oftener accompanied by one-sided than by bilateral headaches. With bilateral sensory phenomena, scotomata, etc., bilateral pains are the commonest.

In many attacks the pains are limited to the eyes, the feeling of soreness of the eyeballs being so very marked that it is painful to move them. Pain in the neck may also cause the desire to hold the neck rigid.

The character of the pain defies analysis, since descriptive phrases are used in such various ways by different observers. In some attacks, the head simply feels slightly sore, or heavy, or dull, or thick; "like a block of wood," is a frequent expression. "Filled with sawdust," one patient says. Again, the pain is agonizing, impossible to describe. Some patients shriek with the pain, become hysterical, and roll about the floor, grasping the head between the hands, wishing to beat their brains out. Between these extremes numberless variants are found among different individuals, and in different attacks in the same individual. Nearly all patients will say that the severe pains are throbbing or thumping, usually indicating great pressure from within or without; as Möbius has said, "some patients think the head will burst, others that it is being squeezed in a vise." Descriptions of bursting are more common. The pain is an all-pervading one, gradually mounting to a maximum, then running along continuously without any let up, with, at all times, sudden accessions, especially on movement, if one leans over, or is forced to sudden exertion. In but the rarest instances is it described as lancinating in quality. It is the type of pain apparently seen in cerebral tumor, in acute hydrocephalus, in cerebrospinal meningitis, and is allied to the pain of opium poisoning, or of sea-sickness; all pointing in the direction of a modification of intracerebral pressure, at times an increase, or it may be a decrease, either of which may cause severe pain. Occasionally the phenomenon of a bilateral headache with marked predominance of one-sided pain will be observed.

The severity of the pain may be conditioned by a number of factors. Movement uniformly increases it. Bending over becomes impossible. The first movement on lying down is usually accompanied by a sudden rise in severity, but this gradually subsides. The taking of alcohol usually increases the severity of the pain, as does also the use of tobacco. Eating, if possible, may help somewhat, but usually augments the pain, and is avoided. Strong sensory impressions invariably increase the pain. Noises of various kinds often aggravate the pain tremendously and cause certain patients marked distress. The "Fourth of July" invariably drives many migrainous patients to some quiet

spot in the country, free from crackers and bombs. Möbius notes that the rage of migrainous parents directed toward their noisy children often resembles a pathological hatred. Strong light is invariably avoided, because of its tendency to increase the pain. The movements of the eyeball and attempts at visual accommodation cause an increase in the pain.

Psychical effort is often impossible; in milder attacks the awakening of a strong mental stimulus may make one forget the pain. Möbius says that his attacks, usually light ones, are frequently forgotten during an interesting visit to the Policlinik, to be once more prominent afterward. One of us has frequently begun a lecture with a severe migraine to find it almost forgotten until the close, when it reappears, usually with renewed vigor.

The movements of straining at stool, and vomiting, coughing, etc., invariably cause a rapid and sharp rise in the severity of the pain. Sensory stimuli may have an unpleasant effect on the psyche. Thus, certain odors cause distress; the smell of cooking acts much as it does on shipboard; it accelerates vomiting. Certain skin phenomena, such as sore spots, are frequent after the headaches.

In certain personal experiments with drugs the following have invariably increased the headache within a few minutes: A few whiffs of chloroform or of ether, adrenalin by mouth, digitalis, strophanthin, and ergot. Drugs that raise the blood-pressure, in general, increase the pain when taken, especially at the beginning of the headache. The headache may clear away very suddenly after an attack of vomiting, or it may pass without vomiting; in some it fades away gradually. It may last a few minutes, a few hours, or a few days. Some cases of what Möbius chooses to call status hemicranicus are recorded.

Vasomotor Disturbances.—Practically all attacks of migraine are accompanied by visible vasomotor disturbances. In most cases vasoconstrictor phenomena (coldness, paleness, gooseflesh, etc.) precede, to be followed later by vasodilator changes. Thomas and Cornu both point this out as a result of their experiences. Thomas contributes a statistical study of 107 cases in support of the early pallor, small pulse and coldness, which pass over to the phenomena of warm, red, flushed face and skin, and full pulse. The period of initial constriction may be unnoticed by reason of its transitory character. In some instances this initial vasoconstriction may be very marked and give rise to the phenomena of localized cyanosis, even advancing to the picture of the constriction phase of the Raynaud disease type.

In the same manner the secondary vasomotor dilatation may pass the bounds ordinarily observed and lead to localized edema, to the erythromelalgic type, or, exceptionally, to hemorrhagic phenomena in the conjunctiva, ocular tissues, or even in the walls of the stomach.

Secretions.—Alteration in secretory functions are frequently observed early or late in the attacks. Reference has been made to the excess of secretion of tears as a frequent precursor. Vomiting of frothy mucus, serous diarrhea, increase of sweat, coryza (Calmeil), or incessant salivation (Liveing, Tissot) are common phenomena.

The changes in urinary secretion have attracted careful attention. The early vasoconstriction of the periphery, coldness, lack of secretion, of perspiration, etc., account in a purely mechanical way for the increase of urinary secretion in the early stages. Metabolic studies show no fundamental disturbances. Bioglio was unable to show constant changes. Although it is not possible to exclude metabolic disturbance as causing changes in vegetative control, probably it is more true that psychical influences cause the metabolic disturbance.

Trophic Disturbances.—These have been reported by several observers. Cornu says that nearly all of his cases of migraine show facial asymmetry and facial atrophy is recorded. These instances are nearly always coincidences and are not necessarily attributes of the migraine. A facial atrophy which can be interpreted only on the basis of a migrainous disturbance of the vasomotor apparatus is very problematical, and certainly Cornu's results are not confirmed by others. Loss of weight in the severe rapidly recurrent cases is due to disturbance in general nutrition due to gastric, rather than to other causes. Herpes is a not infrequent accompaniment of some cases, but the recognition of its infectious nature has successfully disposed of its essential relationship to migraine.

Psychical Disturbances.—These have been noted by many observers, Liveing being one of the first to point out the relationship of disturbed psychical states to the attacks of migraine.

In the vast majority of migraine attacks there are no mental changes, either before, during, or after the attacks. Mild depression, hopelessness, despondency with clear consciousness, are frequent mental states. With very severe pains Möbius admits clouding of consciousness, and is not sure that severe stuporous states are not due to pain as well. Mingazzini, on the other hand, believes there is justification for erecting a special group, which he has termed the hemicranic dysphrenias, and distinguishes a transitory and a more permanent variety. Recent observers are practically in accord, in showing that severe mental disturbances varying in character and intensity may be part of a migraine attack.

Guidi has amplified these observations by reporting the history of a number of cases in which the patients suffered during the day before the onset, in a much more decided manner than by feelings of anxiety or depression as described by Liveing. Thus Guidi calls attention to grave alterations in the psychical state of a number of his patients. In one the entire character of the personality would change preceding the attack. A patient who had always been calm, reserved, quiet, and modest, suddenly became much agitated, was forward,

noisy, and loquacious, and told salacious stories, which was far from his usual behavior. While in health a spare eater, preceding an attack he suddenly became very hungry, and hankered especially for starchy foods. During the attack the patient had glycosuria, which disappeared later.

With the onset of pain the picture is less clear, yet there is little doubt that many patients suffer from profound psychical disturbances, which arise independently of the pain. One such case, under personal observation, would be interpreted by Möbius, and rightly so, as one in which the pain is the first link in a hysterical reaction. But there are other cases which do not belong to this group. Mingazzini's hemicranic dysphrenias may be cited as examples, in part, at least. In others severe disturbances have occurred, such as states of anxiety, rising to actual anguish (Charcot); phobias of inability to perform acts (Cornu-Charcot); terror (Liveing, Féré, Kraft-Ebing); hallucinations of sight (phosphenes, colored lights, animals) and hearing with mental confusion (Forli, Mingazzini); maniacal excitement (Mingazzini, Jelliffe) and stupor; unconsciousness (many authors).

Liveing reports that 25 per cent. of his cases showed psychical symptoms. The Italian observers record fewer, but it appears that at least from 10 to 15 per cent. of the cases of grave hemicrania show some distinct mental disturbance in some one or more of their attacks which is more significant than the usual depression which is so universal.

Symptomatic Migraines.—The occurrence of migraine-like attacks accompanying, or due to, definite disease conditions, notably organic disease of the brain, is well known. The association of migraine with gout and malarial affections has been noted. So far as gout as an etiological factor is concerned, Möbius is inclined to see nothing more than a coincidence; while, as for malaria, he holds it to cause an orbital neuralgia, not a migraine. As for the latter, it seems clear that the well-known effects of malarial infection on bloodvessel tonus are entirely sufficient to cause a typical migraine attack. It is known that attacks of migraine may be very frequent during the continuance of a malarial infection. Such may disappear for months after quinine therapy, and then reappear at the time of a later malarial infection.

Migraine-like attacks are not infrequent in cerebral tumor; they may appear periodically, as in cases fully reported by Abercrombie and Möbius, or they may be continuous and distinguishable with great difficulty from the pain of tumor, as in cases reported by Wernicke, who has said that such attacks may be quite readily confused with those more typical of tumor. In tumors, however, vomiting brings little or no relief; quiet gives less relief, and the fluctuation in the intensity of the pain is less prominent. A primary onset of migraine-like attacks in adult life should always awaken the suspicion of an organic brain lesion.

Oppenheim has called particular attention to the occurrence of migraine-like attacks at the onset of tabes; Möbius is inclined to think it a rare combination, and regards it either as a pure coincidence or a migraine-like neuralgia. In general paresis, migraine-like attacks may be an initial symptom. Migraine attacks are not infrequent throughout the early stages of the disease, but the anatomical correlations are still hypothetical.

Diagnosis.—The difficulties appear in the consideration of ordinary headaches and in neurasthenic headaches; in distinguishing between the scotomata of migraine and other scotomata; the paresthesia of migraine and other paresthesias; the aphasia, the vomiting, etc., as seen in migraine, and the same as due to other causes. In most individuals abortive and incomplete attacks are the rule, and it is often extremely difficult to determine their precise significance.

Möbius has suggested that the problem is not only whether the case is one of migraine or not, but whether it is migraine alone, and not something additional. This author's contention that migraine is hereditary and begins in youth, would seem to make it a simple matter, but clinical experience shows that real migraines do appear in later years, apart from other affections, and as for the hereditary factor, the extreme prevalence of the affection makes it hard to accurately weigh this factor. The periodic recurrence is a difficult criterion. There is usually no difficulty in diagnosing the classical attacks from simple headache, but at times such differentiation is impossible. Many chronic sufferers from migraine know well their real attacks, are able to distinguish abortive attacks, and also have headaches of an entirely different nature. The simplest test in separating abortive migraines from simple headaches is the occurrence of sensory phenomena, other than pain, which have their main origin in vasomotor disturbances. It is on this account we would ally the severe headaches following the use of alcohol, ether, chloroform, opium, or related drugs to the migraines rather than to simple headaches. The headaches of neurasthenia, anemia, syphilis, lead poisoning, nasal sinus involvement, supra-orbital neuralgia, nephritis, eye strain, glaucoma, etc., should present little difficulty.

Treatment.—The treatment of the migraine attack is, for the most part, fairly satisfactory. There are few patients for whom some relief cannot be obtained, both with reference to the diminution in the number of attacks, and to the mitigation of the severity of the attacks themselves. The migraine habit, constitution, or liability—call it what one will—exists in very varying degrees; in some a very slight disturbance is sufficient to set free those forces which culminate in an attack; for others it requires a very much greater maladjustment. If the general reflex vascular hypothesis be taken as a tentative explanation it is very readily understood why the taking away of various forms of peripheral irritation may result in eliminating one or more, and in certain instances all, of the causes which set the migraine reaction in operation.

It is folly to shut one's eyes to the very evident clinical fact that a few migraines are relieved, if not entirely wiped away, by the correction of some peripheral disorder, sometimes more than one, which has had definite effect on the nervous system. Just what the interrelation may be between the severity of the irritant and the mildness of an attack it is impossible to judge, but certainly the relief from eye strain, from diseased turbinates, from adenoids, from constipation, from dysmenorrhea, from a number of minor yet definite peripheral irritations, will relieve a certain number of patients. Perhaps they are the very slight migraines, perhaps not; one is not yet in a position to say. One should, therefore, eliminate at the onset such of these structural defects as are shown to have some influence on the nervous system. In denying any possibility to these influences in the causation of a migraine attack, one errs as badly as when maintaining some one of them to be the *only* and *invariable* element in the case, as faddists are doing and always have done.

Gastro-intestinal factors are closely analogous to those just mentioned. In the minds of most clinicians, and certainly as generalized in the feelings of those most affected, it is in the stomach, liver, or intestines that the main seat of the trouble is to be sought. The gastro-intestinal factor is undoubted in many cases; it may be exclusively gastric or colonic; perverted chemism, perverted bacterial action (primary or secondary factors, no one can yet say). As to the significance of chemical features, resulting from altered gastric secretions or from toxic bacterial products, we are entirely in the dark. It is certain that none of the products which have been held responsible as auto-intoxicants are universal causes. At any rate, the general features of gastro-intestinal hygiene should be carried out. Constipation is to be avoided, and such diet taken as experience has shown is individually applicable. Excesses in certain articles of diet are held by many as exciting causes; such empirical feelings should be respected; the patient often knows himself better than does the physician.

In some, excessive carbohydrate intake acts disastrously; in others wine, whisky, or gin. The history of inability to eat fatty food, particularly sausages, is not infrequent.

In rarer instances, one notes that certain auditory stimuli may bring on a migraine. To attend certain fatiguing and thrilling operas is followed in some by migraine attacks. Here psychological mechanisms are at work.

If the varying elements mentioned have any real relation, it is evident why such a variety of measures will be of help to a few, and why so many more will be worthless for many but useful for some. Medication between attacks is largely useless, save naturally in the symptomatic migraines. General medication, for no definite purpose but just in the hope that it may do good, as iodides, bromides, strychnine, etc., is senseless. If definite factors are found that need

correction, and can be so modified by drugs in the desired direction, then they will prove useful. Thus iodides will undoubtedly help many presenile arteriosclerotic migraines; bromides are useful for sleepless and irritable conditions which provide a good foundation for the nervous instability that permits an attack; laxatives are called for if persistent constipation bears any causal relationship.

Complicated systems of diet have been devised. Usually such are more prolific in engendering semi-invalidism than useful for migraine. Here and there a patient derives benefit from a strict dietary régime, but unless there are real reasons why a patient should not eat red meat, or tomatoes, or sundry other articles, as determined by actual experience and under repeated experimental trials, in order to eliminate faddist's errors, the patient is better off without a diet card. The reasons sought for are not those contained in many treatises on dietetics, in which primitive notions concerning differences in red meat and white meat, vegetables growing under the ground and those above the ground, are foolishly perpetuated. The only satisfactory manner to attack the metabolic problem is to carry out a complete metabolism analysis. Haphazard attacks here and there lead only to premature and insecure judgments.

Complete formulas for attacking excessive bacterial putrefaction are applicable only when it is proved that such excessive bacterial action exists and has a relation to the migraine. The hypothesis cannot be excluded *ex cathedra*, but it remains unproved for most cases, and of doubtful applicability in a few. The belief that the presence of indicanuria is an infallible index of harmful putrefactive products is not well founded.

The avoidance of alcohol and tobacco, while advisable, is so only relatively. The individual's reaction to all influences should be rigidly estimated before those usually self-evident restrictions are imposed in the name of health.

In certain individuals a change of occupation may be absolutely necessary, but here again one must be wisely conservative, and not consign all migraine patients to an outdoor life, especially when outdoor workers are by no means exempt and ought to be clerks. The character of the work is to be borne in mind. The elements of haste, of pressure, and of lack of leisure are to be thought of in this connection.

Psychoanalysis should be advised for severe recurrent migraines in neurotic individuals. Chronic headaches are usually psychogenic in origin and need psychoanalysis.

For the treatment of the attack itself, one finds that a like fitting of remedies to the individual is called for. In the initial phase of vasoconstriction a number of vasodilators are of service, although their action is extremely unequal. The nitrites and nitrates have been employed for years, and usually with a fair degree of success if the dosage and individual member of the group be correctly chosen with reference to the severity of the attack. A mixture is of greatest value;

nitroglycerin and erythrol tetranitrate give the best combination, for following the very evanescent and powerful action of the former, the more prolonged and steady action of the latter maintains the effect. The slowly acting nitrites are practically useless. Nature's readjustment, vasodilatation by vomiting, etc., has already reduced the cerebral pressure, and the stage has passed when the dilating remedies might be useful. It is practically only in the vasoconstriction stage that the nitrites are worth much; and in many they are inefficient, the reasons for this being as yet unappreciated. Given too late, they overdo the dilatation and increase the difficulty.

The analgesic vasodilators have come to occupy the front rank. The precise significance of each must be appreciated in order to obtain the best results. Solubility, time of absorption, slight differences in the chemical formula and in action, continuance of effect with minimum by-effects, are all to be studied. The list is a long one and is constantly on the increase. Antipyrin, acetanilid, phenacetin, and the related salicylic acid (aspirin, etc.) compounds are the chief members. It is to be remembered that while their general action is closely related, there are specific differences in the working of each, and the measure of success that one has in mastering the majority of migraines depends upon a knowledge of these factors. Antipyrin, by reason of its rapid solubility and quick action, occupies an important place, but is not always applicable. Acetanilid, alone or in combination with other analgesics of related type (salicylic acid derivatives), bromides, and caffeine, are also valuable. The dosage should be graded according to the usual severity of the attacks. Tolerance is established in the quickly recurring attacks, and changes must be made. It is not yet certain what part is played by the respective analgesic and vasodilatation actions of this group. They have robbed migraine of most of its terrors, and tended to diminish the use of morphine and its derivatives very markedly.

Caffeine is a much overrated drug. In the abortive attacks and in the morning remains of a migraine it is useful; but for a full-fledged attack it is not efficient. Similarly, bromides alone, chloral, and other widely used drugs are valuable only in mild attacks. They should be used in preference to other more potent remedies, which should be reserved for the severer attacks, in order that one's therapeutic measures may more correctly approximate the needs of each individual occasion.

The use of aconite and *Cannabis indica* is more restricted now that really efficient analgesics are known. Aconite is rarely called for, while *Cannabis indica* or *Cannabis americana* has a limited, though no less definite, place. In attacks associated with much mental depression the addition of cannabis is often useful. The often experienced inefficacy of this latter remedy is largely due to its extreme variability. Great care is therefore to be exercised in the selection of a

proper preparation. Tablet preparations are usually worthless. This is equally true of the volatile nitrite preparations. Opium, or its main derivative morphine, should be used only as a last resort. It is rarely really needed.

Lying down in a quiet, darkened room—a brisk saline laxative taken as early as possible, the patient being undressed and well covered—these are essential in the severe exhausting attacks. A very hot bath often aids very materially in restoring the patient to comparative freshness. Cold is to be avoided.

The greatest folly of all is to treat all patients and every attack alike.

Periodic Palsies: Ophthalmoplegic Migraine.—It is known that in the ordinary attack of ophthalmic migraine there may occur various sensory or motor phenomena, among which anesthasias or paralyses are the most marked. These sensory and motor changes are extremely diverse when the entire range of the migraine symptomatology is brought into review, but there is one symptom grouping which, by reason of its comparative frequency and close similarity, was set apart from others occurring in this affection and named by Charcot ophthalmoplegic migraine, in order to distinguish it from its more classical relation. It consists in a paresis or a paralysis of one or more muscles of the eye, innervated chiefly by the oculomotorius, which comes on either following or during a migraine attack.

Inasmuch as oculomotor pareses or paralyses may occur from a great diversity of causes, apart from a migraine, and may appear periodically, it has been held by many that the term ophthalmoplegic migraine has no particular right to exist, but the evidence is too great to eliminate migraine as a competent producing cause for these periodic oculomotor paralyses.

It is purposed also to enlarge the group and include the so-called *periodic palsies*, familial as well as those in which the hereditary factors are not known to be present.

Etiology.—Whether heredity plays any greater part here than in migraine in general is difficult to decide. Certain periodic palsies show marked heredity.

There has been much speculation concerning the central or peripheral nature of this third nerve palsy. The present view taken for migraine in general, that it is due to a disturbance in cerebral pressure, secondary to vascular modifications, is sufficient to account for the oculomotor palsies as well, in view of the location of the peripheral branches of the third nerve in regard to the cerebral vascular plexuses. In fact, the occurrence of the ophthalmoplegic type is one of the strong arguments for the general pressure hypothesis, as Spitzer has well argued. If, as has been shown by several autopsies, to these considerations additional local causes be added, which increase or permanently maintain

such pressure effects, the interpretation is comparatively simple. Thus, exudates, fibrous processes, swelling in the cavernous sinuses, swelling of the hypophysis, tumor formation, gummata, etc., have been found in patients suffering from periodic oculomotor paralyses associated with migraine.

It is true that some of these are to be interpreted as symptomatic migraines, in which the foreign body acts primarily as an irritant to cause the vascular disturbance, which sets free the migraine reaction, and secondarily serves as an additional cause of pressure to bring about the palsy. In a personally observed case, with basal gummata, the periodic oculomotor palsy and migraine attacks have occurred for a period extending over four or five years, usually with every menstrual period. Here were three interplaying factors, and the exact part played by each can only be inferred. The slight disturbance of menstruation, usually adjusted, in this case by reason of the exudate was not. A migraine was set up, the acute pressure of which, added to that of the exudate, caused the ophthalmoplegia. This ophthalmoplegia has become fairly persistent in the intermigrainous interval in recent years.

Symptoms.—Leaving aside for the moment the atypical and symptomatic periodic oculomotor paralyses due to other causes than migraine, one finds in these patients, usually during or after a severe attack of unilateral migraine, with headache, nausea, vomiting, etc., a ptosis of the eyelid on the same side, and a loss, partial or complete, of the upward, downward, and inward movements of the eye of the same side. This eye is usually directed outward and downward, and the patient sees double. This may or may not be accompanied by sensory disturbances in the superior branch of the trigeminus, just as may be observed in ordinary ophthalmic migraine.

After a variable length of time, a few days, a week or more, the paralysis disappears, usually gradually, and the patient suffers no inconvenience from the ocular palsies or the ptosis. In some individuals such palsies accompanying a migraine have come on comparatively young in life, almost with the beginning of the migraine attacks; for the majority, however, they follow several years after the establishment of a migraine, in some instances as late as thirty years. In some only a very severe attack will be accompanied by the oculomotor signs, or only slight palsies; transitory ptosis may occur frequently. But in others the palsies develop with each attack of migraine and often in increasing severity. The effects may persist longer and longer between the attacks, until in a few they become permanent palsies. This type, however, often permits of other interpretations.

A double lesion can be understood, although it rarely occurs. Isolated abducens palsy has been described, also isolated trochlearis; and complete ophthalmoplegia is reported in a single case, but in view of the many possible contributory factors it perhaps is preferable to view such a case from another standpoint.

Some *periodic palsies* belong in this group. Those palsies occurring in cranial nerves become associated intellectually with the migraines because of the accompanying headaches, but the cause is a vasomotor disturbance. A similar vasomotor disorder in the cord, however, will produce a periodic paralysis in other muscle groups. It is therefore largely a matter of terminology just how to consider the periodic palsies. A more extended discussion of them will be found in the vasomotor-irritability group.

Diagnosis.—Every patient should be regarded as one suffering from something more than the migraine, until all accessory causes are excluded. What these may be have been mentioned already.

Treatment.—Little needs to be added to the therapy outlined under migraine. Syphilis as a cause for both a migraine and an exudate should be treated, and the Wassermann reaction utilized to clear up the diagnosis and therapeutic indications.

3. Vasomotor Irritability Group.

Angioneurotic Edema.—This condition is better described as Acute Circumscribed Edema (Quincke), since such a name does not commit one to its being a vascular neurosis, although this is probable.

These most striking skin edemas were described as early as 1778 by Salpertus. Erichton in 1801 also observed them, and Graves, who gave such an excellent outline of exophthalmic goitre, in 1848, described a patient with localized swelling of the face, forehead, and eyes, in whom the edema persisted only a few hours. Various aberrant localizations have been described often under different names. Naturally hysteria bulked large in the diagnosis in the earlier days. Other synonyms indicate under what different rubrics it was grouped: urticaria, urticaria œdematosa, epidermolysis bullosa, urticaria tuberosa, urticaria gangrenosa, giant urticaria, all indicate where one should search the early literature. Further, one finds rheumatic edema, arthritic edema, repeating rheumatic edema, hydrops articulorum, intermittent rheumatic edema, neuroarthritic edema, in the period when the cases were grouped among the "rheumatisms." *Gastrosuccorrhœa periodica* is a stomach localization. Probably there are others of obscure nature. Acute brain swelling, meningitis serosa, spinal swelling, local transient edema, intermittent edema, and periodic paralysis are among them.

Quincke, in 1882, described it as acute circumscribed skin edema, while in a Kiel dissertation, one of his students, Dinkelacker, brought together many of the older descriptions, and showed the unity of several apparently dissimilar processes. He termed it acute edema.

Occurrence.—The disorder is not frequent, yet it is not rare. Men and women appear about equally involved. It may be present in young children—one and a half months (Crozier, Griffith); three months (Dinkelacker). After forty it appears very rarely, as an

initial development, although in affected individuals it may persist until late in life. Cassirer reported cases of seventy-nine and sixty-nine, in which the disease appeared comparatively late in life. Raven reports a case in a woman of eighty-six.

Occupation apparently plays no role. Heredity on the other hand is conspicuous. Many authors have mentioned this feature. Osler's family tree has been freely cited, and is here reproduced in slightly changed form. Ensor reports a family of eighty members, with thirty-three affected individuals twelve of whom died of edema of the glottis. Similar hereditary features are reported by several observers. The question of its transmission has not been completely cleared up. In Apert and Delille's families only the males were affected, but this does not seem to be the rule.

INHERITANCE IN ANGIO-NEUROTIC ŒDEMA
"T" FAMILY

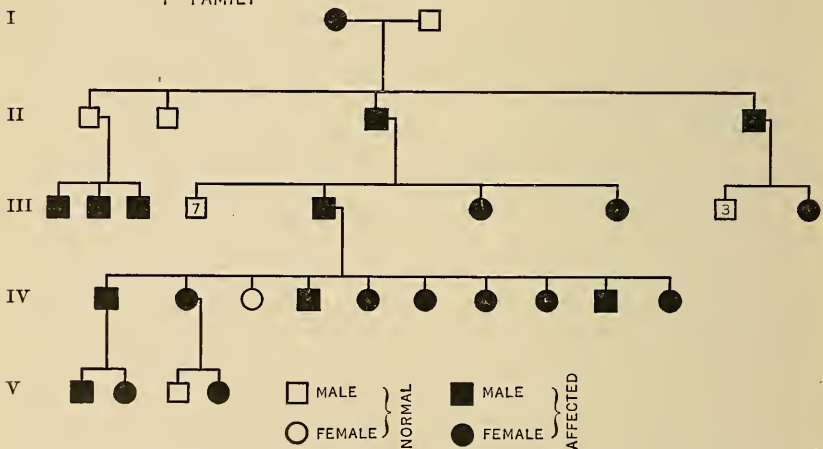


FIG. 44.—Chart showing heredity in an angioneurotic edema family. (Osler.)

In many families, similar types of localized edema prevail in the members, while in others, apparently more often, all of the possible variants disappear. Other nervous-system involvements appear associated with many of the families; how much of this is largely coincidence, how much general neuropathic causal relationship is difficult to determine from the studies at hand.

The syndrome is associated infrequently with tabes, myasthenia gravis, spinal cord tumor, exophthalmic goitre, myxedema, periodic palsies, while it seems very frequently associated with many so-called functional neuropathic states—hysteria, neurasthenia, tics, compulsion neuroses, migraine, etc.—and in certain psychotic individuals with schizophrenia, manic-depressive psychosis, feeble-mindedness.

Local traumata play a role at times, particularly in determining the location of the swelling. Emotional shock bulks large as a direct

etiological factor, as does also the action of thermal influences. Cold is very frequently an exciting factor in the reaction. Menstrual factors seem to enter into the etiology of certain cases.

A moment's reflection, therefore, will show that under the term Acute Circumscribed Edema, one is dealing with phenomena of great variability and multiform genetic pathogeny. In discussing the pathology, a return will be made to this many-sided etiology.

Symptoms.—The original conception of Quincke has been much employed, and Cassirer in his large monograph shows the present-day trend to include a large number of acute edematous swellings within the nosological group. Thus one distinguishes localized edema of the skin, edemas of the mucous membrane, of the eyelids, mouth, glottis, esophagus, stomach, intestines, respiratory tract; edemas of the joints, the meninges, the tendinous aponeuroses, of the spinal cord, of the brain, of the kidneys, with polyuria, albuminuria, hemoglobinuria, diminished secretions, and edema of other structures.

The *onset* is usually acute, with some initial prodromal signs of malaise, fatigue, chilliness, anorexia, nausea, and slight rise in temperature. The symptoms that develop will depend upon the localization of the process.

In the *skin* there are isolated swellings. These are localized, variable in size, at times small, resembling urticarial blotches (intermediary forms) but usually as distinct swellings, with an elastic feel, and due to local accumulations of clear serum within the skin. The color of the swelling is usually that of the skin, or paler, rarely red or reddish. The swelling comes on with great rapidity, a few moments only, and remains a few hours, mostly a few days, and then disappears without leaving any trace. They are as a rule non-irritating, painless, and only cause discomfort as a result of the tension. Certain cases show burning, itching, and intense pain.

The *size* of the edematous patches varies greatly. At times very small—one-half inch—they are more apt to be three to four inches in diameter, or at times involve the larger part of a limb. The scrotum may at times swell up to the size of a foot-ball. The penis, in cases reported by Borner, has swollen to double its diameter. The entire body was swollen also in a remarkable case reported by Diethelm. At times the swellings are numerous, polymorphous, semi-confluent. They rarely rise more than one-quarter to one-half centimeter, but swellings two to four inches above the skin occur. The margins of the swellings are usually sharply circumscribed, but at times may shade off imperceptibly into normal areas. The usual descriptions of the swellings are circular or sausage-shaped. The swellings invade almost any layer in the skin, the musculature, or they may even invade the periosteum. Some have been termed pseudolipomata.

The *consistency* is semi-solid, non-pitting, or slightly so. The *color* as stated is usually that of the normal skin, or it may be paler, or have a cadaveric hue. Again it is pinkish to red, or even deep red. Often

the color disappears on pressure. The color may change during the rise of the swelling.

Local *temperature* varies. At times the skin is colder, again it is warmer than that of the non-affected parts. Exact studies are wanting. It seems not unlikely that there is an initial increase in the local temperature.

Sensory changes are not present as a rule. Certain cases have shown preliminary neuralgic twinges, no definite sensory defect has been noted, but refined methods of examination, such as those demanded by Head, have not yet been made. There is frequently the subjective sense of great discomfort, especially in marked swellings about the face.

There are rarely any *residuals*, although occasionally scaling or peeling has been observed, probably for the more superficially lying edemas.

Secretory symptoms have not been carefully recorded. Local hyperidrosis, dermatographia, increased tear secretion have been noted.

The location of the swelling may be almost anywhere, it cannot be said that one place more than another is a favorite site (statistically). Exposed portions of the body seem to be more often involved, but when on the hand or hands, the distribution is not of the glove type, nor are the swellings apt to be symmetrical, nor do they seem to follow radicular or peripheral distributions. There is a distinct tendency for a recurring edema to occupy the position involved during a former attack.

Periarticular swelling constitutes a peculiar type, so do also parotid and salivary gland edemas.

Mucous Membranes.—These are frequently involved. The lips, mouth, soft palate, tongue, pharyngeal pillars, nasal membrane, larynx are all sites of election. The last is particularly frequent and is dangerous to life. In these cases, other structures than the larynx are implicated, especially the epiglottis and closely associated structures. In the larynx the mucous membrane is swollen and tense; the edema infiltrates throughout.

When the larynx is involved, the symptoms are apt to be very marked. There is beginning tickling, and rapidly oncoming difficulty in breathing, until marked dyspnea may supervene, with death, unless intubation or tracheotomy is performed. Some of these patients die within a few hours. Many cases, on the other hand, clear up in an hour, after severe dyspneic symptoms. Acute conjunctival edema is not infrequent.

Edemas within the bronchi occur in perhaps twenty per cent. of the cases. They make up a certain percentage of the cases of asthma. Certain hay fevers possibly belong in this group. Lung edemas have been described.

In edemas of the *stomach* (gastrosuccorrhea periodica) external signs

are also usually present. There may be intermittent vomiting, or sudden acute pains and anorexia. The attack may last a few hours with severe pain, and finally more or less continuous vomiting of clear or bile-colored watery masses, marked thirst, and gradual disappearance of all of the symptoms. Bits of gastric mucosa have been accidentally dislodged which showed marked edematous swelling.

In *intestinal localizations* profuse diarrheas are present, with colicky pains, meteorism, tenderness of the abdomen, diminished urination, great thirst, and collapse. The diarrheas are purely nervous diarrheas, so-called, and occur in association with other signs of a circumscribed edema.

Rarer localizations present in the tendons have been described, particularly by Schlesinger. Muscle edemas are also rarely described, although it is probable that they are of frequent occurrence. Articular edemas have been mentioned. They are frequently of psychological origin. Compare gout and anger.

Optic-nerve edema is one of the rarer localizations, as is also an edema in the labyrinth leading to a Ménière syndrome.

The bladder, kidney, and heart structures are among the rarest localizations. Meningitis serosa, aphasia, are among some of the more problematical occurrences reported and periodic paralysis and myasthenia gravis are included here as well as in the previous group allied to the migraines.

Prognosis.—In general this is not good. The tendency to laryngeal localization must always be viewed with gravity. A great many individuals have died from edema of the glottis. Remissions are to be expected. Some patients suffer many years, others, but the minority it would appear, have but few attacks. There is some general tendency for the disorder to become milder as the affected individual grows older.

Transition forms are common, especially urticaria-like eruptions. Acroparesthesias, Raynaud-like attacks, local asphyxias of the extremities, paroxysmal hemoglobinuria, acroasphyxia chronica, erythromelalgia, periodic paralysis, epidermolysis bullosa hereditaria, synovial serositis, fibrous serositis, herpes zoster are all affections with which attacks have been combined, singly or in groups of two or three. Occasionally edema, acroparesthesia, and erythromelalgia may alternate in one and the same patient.

Pathogenesis.—Our conceptions concerning edema are undergoing such vital modifications that it is practically impossible to interpret the findings here outlined along those present-day lines that regard all edemas as cell phenomena and independent of the mechanical conceptions of stasis, pressure, osmotic tension of the vascular and lymph vessels, etc. The studies of edema made by Fischer and others emphasize the purely physico-chemical side of the problem. They neglect the role of the vegetative system in regulating tissue tension and cellular chemism. The simple statement that the disorder is

an angioneurosis by no means clears the situation, although it is certain that the sympathetics are media from cause to effect. The study of anaphylactic phenomena, especially as seen in the so-called anaphylactic serum reactions, or serum diseases, has offered suggestive glimpses indicating certain analogies with the series of changes here outlined. Wherein are the proteids supposed to cause these related to the endocrinous hormones? It can only be stated that precisely similar processes and appearances are found in the serum reactions, and that it is not without profit to enquire more into the mechanism of their production in an attempt to understand acute circumscribed edema. Unfortunately the mechanisms of the changes in the anaphylactic reactions are still much in the dark. There is a distinct tendency to include the anaphylactic reactions under the phenomena regulated by the vegetative nervous system.¹

Acute circumscribed edema, has also been interpreted as a modified colloid absorption reaction, due to toxic influences brought to the cells of the deeper layers of skin, muscle or mucous membrane. The view here tentatively adopted is that it is a neural reaction brought about through the vegetative nervous system, which controls reciprocal tension relations, or cellular chemical composition relations.

It is not improbable that there are a series of reactions represented in the acute circumscribed edemas. It is no unicum, and analysis will show that a number of different pathological processes may underly precisely similar phenomena, be they in any vascular area of the body.

Cassirer adopts this viewpoint, but consents to make only two groups of cases—(a) a toxic, autotoxic group, in which the poison works in some mysterious way, which a wealth of language can conceal, better than it can reveal, and (b) a heredofamilial or constitutional neuropathic group, which he regards as intimately associated with instability in certain parts of the vegetative nervous system. This may be, he says, associated in some manner with modifications in the internal gland secretions. Here we enter another dark portal. At all events, Cassirer is loath to permit so-called angioneurotic edema to wander from the neurological fold, and concludes that the disease is conditioned—at least his group (b)—by the lability of the vegetative nervous system.

Treatment.—This is purely empirical. It consists first in avoiding all those things which experience has shown to be liable to bring on an attack.

If one has one of the more pronounced toxic-anaphylaxis-like reacting types, careful study must be made of all of the patient's protein reactions, and attempts made calculated to regulate the diet accordingly. It seems plausible that it is through the gastro-intestinal canal that such products gain entry, particularly in food, yet some may

¹ Roncoroni, Ergebnisse d. Neurologie u. Psychiatrie, vol. ii, No. 1.

enter the respiratory tract, as seems to be the case in the related hay-fever reactions which are known to follow certain contacts, variously ascribed to ragweed, rose, hay, and other pollens, or even the emanations from cattle.

From specific exclusion of certain proteins one passes to the general hygiene of the intestine. This means a sort of search in the dark for effective agencies by chemical means. One is justified notwithstanding in trying to bring about altered bowel conditions, which empirically may do some good, when a *laissez-faire* attitude seems to perpetuate the disturbance. Naturally one should avoid intestinal therapy, should the patient be of an entirely different type, say, the intensely neurotic forms with familial hereditary burdens, and emotional shock reactions. These patients need a psychoanalysis.

Of the gastro-intestinal antiseptics so-called, few are such. Menthol, saline laxatives, carbonated waters, careful dieting (?) may be found among the conventional remedies in the books. The taking of a milk-vegetable diet has been coincident with betterment in some individuals and coincident with getting worse in others.

In certain cases with associated toxemias, such as malaria, etc., a specific therapy is indicated.

On the supposition that the bloodvessels needed bracing up to prevent transudation through their walls, also a hypothetical postulate, apparently inadequate, such drugs as strychnine, ergot, arsenic, atropine, morphine have been recommended. While all of these will bring about vasoconstriction it is not apparent whether they can alter a hypothetical transudability or not. Calcium lactate is the modern weapon for this latter. The authors have not seen it recommended, but it may be of service in preventing transudates, as such are thought to be conditioned by a diminution in the calcium content of the body plasma. Cassirer mentions calcium chlorate. At all events the vasoconstricting drugs have not been of any particular service clinically. Now and then they seem of service; none have been proven of prophylactic value which is a stricter test of their usefulness, since the disease is so self-limited. Atropin is of great value at times.

In many cases associated with laryngeal symptoms, intubation is often necessary—even tracheotomy. There are records of certain patients condemned to the persistent use of the tracheotomy tube.

In the more strictly neurotic type—Cassirer's group (*b*)—it is important that the patient be taught a healthy morale. The substitution of reasonable and intelligent actions for purely instinctive and emotional reactions must be acquired by them, if they can hope to in any way control their hair-trigger vegetative nervous system. Perhaps it was so given to them, defective and badly coördinated; even then a rational pedagogy will prove of service. Many will be helped by the methods outlined by Dubois or Dejerine;¹ others will need a psycho-

¹ Dubois, *Psychic Treatment of Nervous Disorders*; Dejerine, *Psychoneuroses and Psychotherapy*, Philadelphia, 1915.

analysis. Steckel has reported some extremely interesting and severe asthmatic cases, with pronounced symptoms of circumscribed edemas with psychoneurotic combinations or complications. Just how the psychological pathways become involved in their complex neurobiochemical relations has been shown in the pages preceding also in the chapters on the neuroses and psychoneuroses, under which groups also many of these patients are classified.

Multiple Neurotic Gangrene of the Skin.

—The condition described by this title may be mentioned. It is an analogous process in which the vasomotor constriction is not limited solely to the extremities, but to apparently unrelated, isolated patches of the skin.

While exaggerations in neuropaths (hysteria), or in psychoses (schizophrenia) may bring these changes about by artefact, malingering explains only a small number of them, granting that the term is any explanation at all.

The disorder sets in with a burning and prickling of the skin. Then after a variable length of time (a few minutes to forty-eight hours) swellings occur, blisters form, with later necrosis. At times only a circumscribed edematous bleb forms.

Sweat Secretory Mechanisms. — These are closely related to, yet independent of, the vasomotor mechanisms. They probably have independent ganglion cell representation in the lateral horns. In general those pharmacological agents which increase vasodilation (autonomic) increase sweat secretions, yet vasodilation may occur without sweating, and the latter may occur with anemia and vasoconstriction (cold sweat of fear, sweating of face in migraine, epileptic aura).

Cervical sympathetic and bulbar-autonomic innervation is probable, and cortical connections, though still topographically unanalyzed are certain (unilateral sweating in thalamic lesions, hemiplegia, hysteria, compulsion neuroses, schizophrenia). Certain anatomists place the cortical pathways

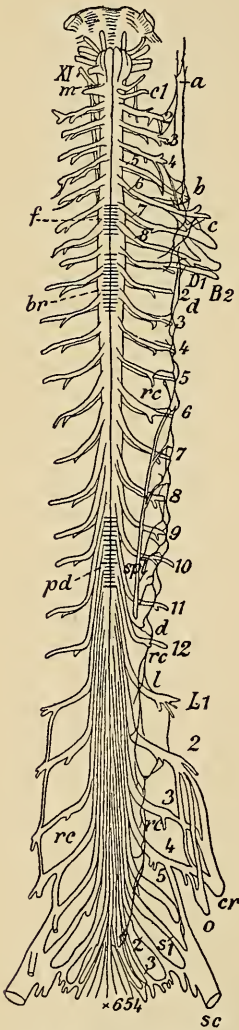


FIG. 45.—Scheme of spinal centers of the sweat secretions: *f*, center for the face and neck; *br*, center of the upper extremities; *pd*, center for lower extremities; *a*, superior cervical ganglion; *b*, middle cervical ganglion; *c*, inferior cervical ganglion; *m*, medulla. (Bechterew.)

among the motor tracts in the internal capsule. The hypothalamus is made a midbrain center by some.¹

Clinically, sweating is increased in certain hemiplegias, and in zoster. Diminution of the sweat is seen in certain cases of poliomyelitis, multiple sclerosis, syringomyelia, myelitis and tumor of the spinal cord. Great variability in the sweat activity is seen in many psychopathic individuals, in vagotonic types and in the psychoneuroses, hysteria, anxiety states, compulsive states.

The reactions of the sweat secretion mechanisms are exquisitely sensitive, as is seen by the response to pain, gastro-intestinal cramp states, action of carminatives, to nicotine, to anxiety and to joy. Veraguth's psychogalvanic reflex experiments show that a close relationship exists between the skin secretions and psychical processes (Jung). The fineness of registration and the extreme complexity of the phenomena, however, militate against the practical utility of the galvanometer tests.

Pilomotor System.—The smooth muscle fibers of the skin are under sympathetic innervation. The pilomotor fibers run with the sensory fibers (Higier), each sensory nerve carrying fibers from about five sympathetic ganglia (Higier), and have similar topographical (segmental) distributions. Mechanical, thermal, and electrical stimuli cause contractions. The erector pilæ reflexes (best observed by side light) are particularly responsive to cold. The frequently felt paresthesiæ, acroparesthesiæ, etc., of psychoneurotics depends upon these sympathetic reactions, and their exact observation is of much diagnostic importance. Mackenzie's observations should be consulted by the interested student.² The feeling of cold over the abdomen after taking cold water in the stomach is an example of the relationship of the sympathetic innervation of an internal organ and a skin area.³ A large number of analogous phenomena are known. Thus among them, mechanical stimuli of the plexus pudendi, as in cystitis, rectal exploration, prostatic massage, prostatitis, causes distinct gooseflesh or paresthesiæ in the region of the motor lumbar plexus. Erector pilæ crises are known to occur in tabes; they are at times migraine equivalents. Psychological stimuli may lead to localized, or more often to generalized reactions. A great variety of extremely important skin hallucinatory experiences are known to occur in dementia precox, and in the psychoneuroses, hysteria, anxiety states, compulsive states. (pathological blushing, etc.). Their correlation is only just being understood through the results of psychoanalytic research.

¹ See Müller and Gläser, *Deut. Zeit. f. Nerven.*, vol. 47-48, p. 365 for literature.

² Mackenzie, *The Signs of Disease.*

³ Head's Zones, Mackenzie.

CHAPTER IV.

THE ENDOCRINOPATHIES.

INTERNAL SECRETIONS.

MEDICAL biology has not yet arrived at an hypothesis sufficiently comprehensive to permit a unitary scheme which can explain the relations of the vegetative nervous system to the control of the viscera. Some suggestions have been offered concerning the action of the vegetative nervous system upon the gastro-intestinal tract, and also some ideas relative to the intricate adjustments of internal and external organs brought about through the bloodvessels have been reviewed. The problems of cellular adjustment now demand attention. These concern the vital phenomena of anabolism and katabolism, and the adjustment of all of the organs of the body involved in the elaboration of special substances which are of importance to the metabolism of the rest of the organism.

Thus, what part is played by the nervous system in the carbohydrate oxidations of the body, acting chiefly through the lungs, the liver, the pancreas, and suprarenals? The substance of the hypophysis, its hormones or active substances, what are they; and what is the interrelationship between them and growth as seen in the clinical phenomena of infantile dystrophies, of acromegaly, and a number of related conditions? Similar questions arise for discussion concerning the thyroid, the thymus, and the adrenals. The pineal gland is also a chromaffin formation, likewise the parathyroids, with their problem of regulating the calcium metabolism not yet certainly disposed of. What of the other constituents of the body plasma; what keeps them all in a state of equilibrium, so that all types of functioning may go on, physico-chemical, vital, and psychical?

This entire group of questions cannot even be asked here. It can only be said that in their consideration, one sees an entirely new country opening up which promises to greatly modify the geography of our present neurological schemes. Sequard in 1889, reached out for immortality by his use of testicular substances. So far as space permits the more essential interrelationships and correlations will be brought out in the discussion of the various diseases. They are at present grouped under their respective glands. A shift in the point of view, is apt to come at almost any time.

The chief available literature summaries are Biedl,¹ Sajous,² Falta,³

¹ Internal Secretions.

² The Internal Secretions.

³ Die Erkrankungen der Blutdrüsen, exhaustive and suggestive.

Lewandowsky,¹ Parhon, and Goldstein, and special monographs to be mentioned under the separate diseases, such as Cushing on the *Pituitary*, Klose and Vogt on the *Thymus*, Morel on the *Parathyroid*, Sattler on *Basedow's Disease*, etc.

Among the earlier attempts at correlation of the group of disorders of the blood glands or internal secretory glands were those of Claude and Gougerot, and Laignél-Lavastine. The latter proposed the following:

1. Uniglandular syndromes, with pluriglandular lesions, as myxedema.
2. Pauciglandular syndromes, *i. e.*, predominance of one blood gland syndrome with changes occurring in others.
3. Pluriglandular insufficiencies without predominance of one or another. Dercum's disease—ovariothyroid.
4. Pluriglandular hyperfunction. Acromegaly.
5. Pluriglandular balancing, where a hyperfunction in one direction is a compensation for a lack in another. Thus, exophthalmic goitre following ovarian removal.
6. Pluriglandular disharmony, as seen in exophthalmic goitre with myxedema.
7. Abortive types.

The French school has carried these hypothetical deductions to great lengths, and have developed an organotherapy of startling complexity. As yet no systematic presentation is possible. Here we purpose to discuss those disorders of the internal secretions with pronounced disturbance of the nervous system, beginning with those best known.

We shall therefore take up: (1) the hypo-, hyper-, and dysthyreoses, (2) the hypo-, hyper-, and dyspituitarisms, and (3) the disorders of the parathyroid, thymus, and adrenals. Finally, some suggestive relationships between diseases of certain viscera, liver, kidneys, spleen, etc., and the nervous system.

THE THYREOPATHIES; THYREOSES.

Hypothyreoses: Myxedema.—The chief *hypothyreoses* are grouped under the symbols *myxedema* and *cretinism*. Three main types of the former, congenital, idiopathic, and operative, are described, while sporadic, endemic, and irregular types of cretinism are distinguished. The whole group may be considered as one, *i. e.*, hypo- and athyreoses. Clinically the various subgroups have grown up since Gull, in 1873, first called attention to myxedema. They are still in need of clearer differentiation and description.

¹ Handbuch der Neurologie, vol. iv, Special Neurology, a collection of valuable monographs.

Operative myxedema (cachexia thyreopriva) has been the best studied type since Kocher, in 1883, called attention to it, one year after Haddon had shown the relationship between myxedema and the thyroid.

Symptoms.—The chief symptoms are present in the skin, nervous system, thyroid, circulatory apparatus, temperature, digestive tract, blood, urine, bones, and general metabolism.

Skin.—Here there occurs a general gradually increasing swelling, most marked in the hands and head. It seems edematous and yet does not pit on pressure. The thickness of the lips causes an eversion or hanging down of the same. The folds of the skin, especially of the forehead, are more distinct than is usual. Irregular, flattish, fatty-like deposits are present in different parts of the body, often being most marked in the supraclavicular region.

The hands are apt to be clumsy and fat, the skin of the back of the hand being much thickened. The feet may show similar changes. The whole skin is whitish and dry; it scales readily and rarely shows any perspiration. Diaphoretic drugs even are unable to bring about any marked perspiration, and the mucous membranes are apt to be dry and not easily irritated to cause exudates. Yellowish pigmentation may occur. The hair breaks easily and is apt to be badly and sparsely developed. The nails are brittle, develop slowly, and show irregular markings. The teeth also develop badly.

There is a sense of tension in the skin, and coldness of the extremities seems universal, and is made much worse in winter, with marked tendency to chapping and frost bites.

The nervous system shows a number of defects varying with the grade of hypothyreosis. Any of the cranial nerves may show defective development. The cerebrospinal nerves may be deficient. The motility as well as the sensibility is diminished both as a result of peripheral and central maldevelopment. The changes in sensibility are furthermore augmented by the localized skin changes. The reflexes are not markedly changed. The gait is usually wide based and clumsy and dependent upon the mental substratum. Defective thyroid substance seems to hinder the regeneration of cut or injured peripheral nerves.

Mentally a great variety of changes may be observed. They are usually in the nature of defect. There is defect of memory, attention is diminished, thinking goes on more slowly, but may be of fair capacity. There is usually a loss of initiative, and emotional dulness goes along with the sensory losses and motor reluctance. The speech is apt to be slow as are other motor acts. It is monotonous and the thickened lips further contribute to make it at times unintelligible. The whole appearance of the patient is one of gradually advancing stupidity which, if there is no relief, goes on to more profound defect states—dementia.

The *thyroid* itself is usually much diminished in size, or not at all

palpable. Though palpable its active secretory substance is usually defective.

The *circulatory* apparatus shows little abnormality. Unlike the hyperthyreoses there is no contrasting bradycardia. The heart action is usually normal—the larger vessels may be felt. Vasoconstrictor action is prominent and is responsible for the cold extremities and possibly some of the dryness of the skin.

The *cold* sensations are not subjective alone, as there seems to be a fairly constant diminution in the bodily heat, as is also seen in hypopituitarism. Digestive discomforts from dry mouth and enlarged tongue are frequent. There is not infrequently diminished muscular tone and deficient secretions in the entire digestive apparatus with obstinate constipation.

Genital anomalies are frequent, consisting of irregular or depressed menstruation or diminished potency. The organs themselves—testes, ovaries—may be diminished in size and infantile defective hairy development is present.

The *blood* shows fairly constant eosinophilia, and the clotting time is distinctly increased and the fibrous content above normal.

The *urine* is not characteristically altered, save that its quantity is usually very low.

Metabolism is slowed down in many directions. Oxygen exchange is reduced, the calories consumed being markedly diminished. The nitrogen output is less, as well as that of the purin derivatives. The calcium-magnesium metabolism is not modified save in the operative individuals, in whom the parathyroids are also disturbed. Carbohydrate tolerance is high.

The *bony* system is variously altered. Here the grade and complexity of change depends largely upon the age of the patient at the onset of the disorder. The long bones fail to develop normally and those with late ossification centers fail to undergo complete development. The skull is apt to be macrocephalic, but the thickness of the bone may diminish the interior capacity a great deal. The fontanelle in young patients is apt to remain open. The changes in myxedema are not those seen in rachitis or chondroostrophia.

Types.—The *idiopathic* form usually begins with changes in the skin, and is often accompanied by neuralgic pains. A patient recently seen was diagnosed as a tabetic. The skin of the face is often first affected and usually very gradually the extremities are implicated. The symptoms may all come on within a few weeks, but usually their development occupies months. Women are much more frequently observed, and usually about the menopause period, when the hypothyreosis may be diagnosed as a so-called “menopause neurosis.”

Operative myxedema is now comparatively rare, since the essential relationships have been pointed out. The tetany symptoms often seen in the earlier and badly operated cases were due to the parathyroid removal.

Congenital forms, thyreoaplasias, occur in children usually of normal birth, and usual development up to about the time of weaning—if not breast-fed usually earlier (thyroid in mother's milk). The symptoms then develop rapidly and, as a rule, are very extreme. The irregular, imperfectly developed, cretinoid pictures are not the usual ones in congenital myxedema; as Eppinger has remarked there are few "half-way" congenital thyreoaplasias. The female sex preponderates and there are no geographical limitations as in endemic cretinism. Neither is there, as a rule, any goitrous family history as is often found in cretinism.

These little patients forget to suckle and to swallow. The skin becomes folded, the nose broad, the eyes deeply sunken, the nasal wings widely spread apart. They are mouth-breathers, with swollen, not infrequently protruding, cyanotic tongues. Salivary increase is often present. The hair is badly developed, the face that of an old man. The head grows in size, but the rest of the body stays behind with marked disproportion in length and breadth throughout. The epiphyses do not ossify, and the ossificatory nuclei, especially in the hands, fail to develop. The fontanelles remain open perhaps to the twelfth or fifteenth year, and the teeth are slow in appearance. The body is apt to be fat and the abdomen especially swollen, in part from gas and obstinate fecal accumulations. Umbilical hernia is frequent. Other defects are often present in heart, palate,



FIG. 46.—Myxedema showing failure of ossification in epiphyses of the bones of the hand. (Siegert.)

and other structures, and they die young with the general mental symptoms in part described.

Diagnosis.—Nephritic edema and other skin edemas must at first be ruled out, especially ovarian pseudoedema of the menopause and rare forms of syphilitic or familial neurotrophic edema. Chondro-strophia must be separated from the cretinoid complications. The relations to cretinism are close. There are differences in the skin and perspiration. Deaf-mutism is rare in myxedema, frequent in cretinism.

This whole problem is too complex to discuss in a text-book. (See Eppinger in Lewandowsky, and Falta, *Erkrankungen der Blutdrüsen*.)

Prognosis and Therapy.—See *Cretinism*.

Cretinism.—This is a broad, general term applied to a combination of physical and mental changes which, in the young, result from loss or diminution of the thyroid functions. Such a loss may occur sporadically, *sporadic cretinism*, from causes to be enumerated, where the picture is analogous to that seen in the adult from removal of the thyroid, *cachexia thyreopriva adultorum*, or it may occur as a localized or *endemic* degeneration, affecting the thyroids of a large number of individuals, causing a *hypothyreosis* which may show a number of tendencies. The chief of these are *goitre*, *goitrous heart*, and *endemic cretinism*.

These three fairly well-separated conditions may be discussed to advantage under the head of cretinism. In the first place to call cretinism a type of idiocy is misleading. There are numerous very intelligent cretins. Cretinism, as here used, is solely a series of different conditions due to a lack of development of one or more elements in the body and due to defect or loss of the thyroid hormones.

The historical chapters on cretinism are full of interest. The disorders were known in early days. Pliny has left indubitable evidence of their presence in early Roman times. Vogt in his admirable monograph in the Lewandowsky *Handbuch der Neurologie*, tells of Marco Polo's descriptions of certain types he had seen in his Asiatic travels. During the past two centuries the disorders here included under this term have been observed throughout the world. In certain lands the disease is very widely distributed, certain mountainous districts of Switzerland, Northern Italy, etc.—endemic cretinism—while in other regions it occurs rarely—sporadic cases. In the United States it is not frequent. It has been observed in California, in Vermont, and such patients have been seen in New York State (Adirondacks). In certain regions it has been a veritable plague. Thus in Switzerland between the years 1875–1884, 7 per cent. of the recruits in the army showed some form of cretinoid degeneration. In ten years 2500 men were lost to the Swiss army from this cause alone. Certain valleys, especially those of Berne and Wallis, are over-thickly populated with individuals showing cretinoid degeneration. In the school years 1899–1904, of 336,000 children fit for school, 15,000 had one or another type of cretinism. Similar conditions existed in Styria, Austria, and in certain Italian provinces. F. Bircher has contributed an important study to the distribution of cretinoid degeneration.

So far as the etiology is concerned, it seems certain that the conditions are due to a defect of the thyroid substance—the thyroid hormones. There are certain limitations which must be discussed in their respective paragraphs.

Sporadic Cretinism.—*Infantile Myxedema of some Authors.*—The clinical picture in an extreme case—*i. e.*, fully developed—in contrast to the many irregular or incomplete forms—is that of a normally born child who about the end of the first or the beginning of the second year begins to show the characteristic changes in development.

The little patients fall behind in their normal bony development. This is due to a defect in the development of the long bones. The epiphyses fail to lay down bone even after twenty, thirty-five years, and in twenty-year-old cretins the anterior fontanelle may still remain open. There is a proportionate loss in bone substance throughout; thus a characteristic dwarfism results save perhaps in the development of the skull, which develops larger in proportion to the rest of the body. The sphenoid, however, fails to develop and therefore gives the peculiar characteristic nose to the cretin. The bony defect is in the nature of a sclerosis and the bony tissue is unusually hard which is the reverse of that seen in rachitis.



FIG. 47.—Cretinism. Woman, aged thirty-four years; mentally, seven years by Binet-Simon test; height, 49½ inches; protuberant abdomen, typical facies, supraclavicular pads of fat.

Dental deficiencies go hand in hand with the bony defect. In severe athyroid cretins the teeth do not develop for a number of years, and the first or milk teeth may persist far beyond the normal period. Other defects appear in a high palatine arch, with large adenoids and tonsils and a chronic hypertrophic rhinitis causes the child to snore and sniffle, often with copious excretions from the nose.

A fairly constant finding is that of umbilical hernia. The abdomen is usually puffy, the navel sunken. The skin is myxedematous in the young, but becomes atrophic in later years, the supraclavicular and facial swelling remaining for many years.

The facial habitus is characteristic. The hair line begins low. The nose is sunken, the zygomatic arches prominent, the eyelids swollen, the face puffy, the tongue enlarged and often protruding between the swollen lips,

in the mild cases giving one the impression of a child whose whole countenance is puffed up with crying.

There is usually an enlargement of the liver. Respiration is unusually slow in the severe athyroses. The genital organs show

marked changes. The labia are small, the external not covering the internal ones. The uterus and ovaries are usually small, and the mammary glands are atrophic or hypoplastic. The penis is apt to be small, the testicles undescended and small. Genital hair and that in the arm-pits is absent or scanty. In boys the pubertal changes in the voice are lacking.

Blood changes are present. The hemoglobin is reduced and is out of proportion to the erythrocytes. The leukocytes are increased, the polymorphic neutrophiles being markedly diminished and the lymphocytes correspondingly increased. Large numbers of granular cells are observed. These changes appear as a result of defective thyroid substance.

The metabolism of calcium is markedly diminished (one-third of its normal amount in the studies made by Haugardy and Langstein) and the required calories are far below that of the normal child's need. The assimilation of carbohydrates seems high, and adrenalin injections, without increased sugar intake, do not result in a glycosuria. There is a definite hypothermia. In many cretins there is a widening of the sella turcica. Hypoplasia of the thymus is also not infrequent.

Nervous system defects are present with the others and apparently conditioned by the endocrinous gland insufficiencies. These show at vital levels in defects of sensory and motor nerve structures, and at psychical levels in various grades of stupidity, mental weakness (moron), imbecility or even idiocy. These words are here used in accordance with the arbitrary scale of the Binet-Simon tests.

Thus smell is at times defective. The eyesight poor; hearing is frequently disturbed, and with it speech, so that many patients are deaf and dumb. The vestibular function is frequently involved, so that these patients balance badly, often showing unsteady gait, with wobbling of the head, and nystagmus.

Some sporadic cretins may show little disease of the nervous system.

Cretinoid Degeneration.—Mention has been made of the widespread character of this type of degeneration related to defective or absent thyroid secretions. The statistical study of the conditions, particularly in Switzerland, in France and in Italy (Bircher) has shown that goitre, goitrous heart, endemic cretinism, endemic deaf and dumbness, and endemic feeble-mindedness are closely allied. The cretins are almost all goitrous, or nearly always have goitrous parents. Exophthalmic goitre (hyperfunction) is rare with cretinism, but very frequent with goitrous heart conditions.

The causes of the hypothyroidism are not definitely settled but there seems to be a constant relation between it and certain elements in the water supply, and goitrous springs are known. Just what the noxious element may be is still conjectural but it apparently is related to mineral constituents found in certain geological formations, notably in the Trias and Tertiary. The disease is absent in regions fed from waters of crystalline formation. Through Bircher's sugges-

tion of supplying a goitrous region in Rupperwill from Jura water coming from an adjacent valley, the disease disappeared. Similar results followed in the town of Asp. Animals may be made goitrous from drinking water from certain springs. They also develop goitrous hearts, and are delayed in their development. The thyroid shows degenerative changes. The agent passes through a Berkefeld filter, but is modified and made non-active by being heated to 70° C. It does not dialyze and is thought not to be an organized plant or animal substance, but is of a colloidal nature. An hypothesis which had the authority of Bircher behind it was that the disease was of an infectious nature.

Goitre.—Here Falta describes those enlarged hyperplastic, non-inflammatory thyroid formations, with degenerative changes in the struma. The hyperplasia invades the parenchyma and the vessels. Notwithstanding histological hyperplasia there is physiological diminution in function. Functionating parenchyma, however, does not exist. Histologically one finds parenchymatous, vascular, or fibrous hyperplasias, with circumscribed or diffuse goitres. A relationship between goitre and uterine myomata exists, and with a diminution of one there is a decrease of the other at the menopause.

Goitre Heart.—See Exophthalmic Goitre.

Endemic Cretinism.—Here there is a richer and much more variable picture than obtains for sporadic cretinism. Whereas a typical habitus is described, there are many anomalies and variations. The head is usually broad, but may be small and flat, instead of large and broad, at times very large. The nose is usually wide spreading and flat, the eyes wide apart. The neck is short and thick, the features swollen, the early impression, especially of the prognathism, one of moroseness or stolidity. The bones are shortened, various anomalies as scoliosis, ankyloses, etc., being present. Great variation in dwarfism is observed. Certain cretins are under three feet six inches, but full cretins have been observed of seven feet in height. As a rule they die young, but Kocher reports cretins seventy and even one hundred years of age.

The general coördination of these patients is poor. They are usually short, clumsy, inelastic with badly developed musculature. The skin is loose, lax, anemic, marked with folds and wrinkles, giving a peculiar appearance of old age. The lips are swollen, the tongue enlarged, and not infrequently protruding. The breasts are flat or badly developed, the abdomen flat or pendulous. Short, stumpy fingers and toes give an ugly appearance to the extremities and contribute to clumsiness. The entire activity is apt to be heavy and awkward, although a few athletes and acrobats may be found among cretins.

The changes in the bones have been mentioned in the paragraphs on sporadic cretinism. Here, however, the variations are more marked and Weygandt's study of Virchow's material shows that many

bony anomalies exist among cretins not mentioned in Virchow's classic which has remained a standard for writers for many years.

The skin has a peculiar cachexia. It is swollen and flabby, whitish or yellowish, folded and soggy. The general appearance of old age is striking. The hair and nails are badly developed, both breaking easily. Thick, underlying, fatty masses are unevenly distributed, usually in the neck, back, upper chest regions, occasionally over the hands. Variable states of tension occur in these fatty masses; at times they are hard, again like empty sacks. The mucous membranes are also pale and gray, often folded but look different from a typical anemia.

The sexual organ changes have been touched upon in the description of sporadic cretinism. They are characteristically infantile. Menstruation is scanty, wanting, or develops very late. Fecundation may take place, but the results are either miscarriages, dead children, monsters, etc. The secondary sexual characters are all delayed in their development.

The majority (63 per cent., Ewald) of cretins show a swollen thyroid, but it is not an overfunctioning one, nor do they all show athyreosis, or hypothyrosis. Schonemann has reported the findings of strumous changes in the glandular portion of the hypophysis. In 112 autopsies on endemic cretins he found a normal hypophysis in only twenty-seven instances. These individuals had no goitre. He states that in individuals with struma of the thyroid almost invariably there was an increase in the size of the hypophysis, and chiefly consisting of increase in the connective tissue, also the chromaffin cells, struma of the vessels, hyaline degeneration and swelling of the cell strands and finally goitres with colloid formation. It is highly probable therefore that the goitre poison works deleteriously upon the hypophysis (Falta). The parathyroids show no changes.

Most of the internal organs show reduction in activity. Digestion is usually slow, constipation is marked. The metabolism is modified as already indicated. The urinary secretions are apt to be diminished, and of high specific gravity.

Mentally cretins show marked variability. A few are practically normal, but most show a characteristic combination of mental traits, which is in marked contrast with many other defective mental states. (See chapters on Feeble-mindedness.)

As noted, the great majority suffer from impairment of the chief sensory tracts. Hearing seems to suffer most. The defect in hearing is associated with speech defects. Taste and smell are also defective. They take little interest in their food or drink. The feeble-mindedness is accompanied by great slowness of all reactions, with marked retardation of motion, with apathy, and indolence. This indolence is a marked feature. Many cretins will lie in the sun all day long, and in the hospital or other institution will sit around and do nothing for weeks or months. In the milder grades there is often great shyness

which makes them unapproachable and serves to make them appear more feeble-minded than they really are. It is with the greatest difficulty that they can be trained to the simplest of performances. With many, in spite of the marked general stolidity of their average mood, they may show great excitement and emotional outbreaks.

The sense of sight is frequently diminished. It is highly probable that the receptors and conduction paths are less involved than the perception areas in this diminution in sensory intake. The hearing seems to be affected both as to its receptors and to the conduction paths. Pain, touch and thermal sensibilities are all dulled. Motility is extremely retarded. The reflexes are active (50 per cent.). The field of vision is reduced in many, although the fundus is usually normal (Hitschmann).

Aberrant and abortive types are to be expected. In the former one may find patients with striking development of one or more features, in the latter a very great shading off to almost normal states, *i. e.*, endemic goitre with mild mental signs.

Endemic Deaf-mutism.—This combination is extremely frequent where endemic cretinism is present (29 per cent., Scholz). It may constitute one of the aberrant types just mentioned with striking development of single features, or it may be associated with all the grades of a complete cretin picture. According to the studies of Kocher, the loss of hearing is due to a bony defect which has destroyed the possibility of normal cochlear development. Bad hearing is reported at 32 per cent. among cretins in Scholz's investigations.

The changes found in the brain which may account for the feeble-mindedness have been variable. Meningeal inflammation and mild grades of hydrocephalus have been found by Scholz and Zingerle. The brain is often asymmetrical, small, or single lobes are diminished in size. Often the brain's development is arrested at an infantile stage, the pallium or the ganglia being involved alone or together. The cerebellum is often imperfectly developed, which fact stands in correlation with the marked incoördination and possibly in relation with defective labyrinthine development.

The ear difficulties are numerous. Peripheral, conducting and central mechanisms are found to be at fault, but at all events seem secondary to the developmental anomalies induced by the action of the poisonous substance on the thyroid. The speech defects usually go hand in hand with those of hearing, but this is not universal. The cortical developmental defect is sufficiently explanatory for most of the cases.

Therapy.—Many contradictions may be found in the literature concerning the use of thyroid substance in various types of myxedema and cretinoid degeneration. This is to be expected since so many observers use their diagnostic terms so lightly. Age differences are not recorded—stage and intensity of the disease—and grade of defect

are overlooked, and hence no uniform basis for comparisons have existed.

Among the best reported results are those of v. Wagner who obtained a diminution in the myxedematous swelling of the skin; the genitals developed rapidly, the tongue diminished in size, there was loss of the umbilical hernia, development of new hair, dentition was hastened, closure of the fontanelles occurred, and there was an increase in bony development. The psyche was less hopefully modified, but there was a diminution in the apathy, and slight increase in the intellectual capacity was obtained.

Early therapy is naturally the main feature. According to v. Wagner, small doses of iodine in addition seemed to stimulate the thyroid activities still further. Magnus Levy, v. Eysselt, and others also report excellent results, complete cure resulting in some patients still in their teens.

A widespread state experiment carried out by v. Kutscheras in Styria treated 1011 cretins. A large number were neglected by the parents *i. e.*, treatment was not kept up. In 2.4 per cent. the thyroid tablets could not be well borne. All idiots and severe grades of deafness and dumbness were left alone. Of 440 of the cases 10.2 per cent. showed slight increase in bony development, 4 per cent. showed definite change, 85 per cent. showed an increase well above the average. The increase in bony growth was marked with the younger individuals, but also persisted into the third decade.

A careful revision of 677 cases showed 42.8 per cent. marked improvement, 48 per cent. some definite improvement, 8.6 per cent. no improvement.

Scholz's experiences with 100 cretins in an institution were disappointing. He used as many as eight tablets a day. V. Wagner claims the doses were too extreme, hence the bad results. Emaciation, weakness, loss of appetite, vomiting and diarrhea, and other symptoms of hyperthyroidism developed. v. Wagner recommends the use of only one-half or one tablet (thyroidinum siccum, Merck=gram 0.1); 0.4 gram corresponds to the activity of an entire gland. (Burroughs Welcome Co., 0.1-0.3 gram=gr. iss-ij.)

Iodothyrene has also been utilized. One gram contains three milligrammes of active substance representing the iodine content of one gram of fresh sheep's thyroid. It would seem that the iodine content is not the only factor in the activity of the thyroid substance and it is not as yet definitely demonstrated what the combination is that is effective. Surgical implanting of the thyroid gland itself would be the ideal therapy, and experiments directed to this end have been carried out since 1889 when Bircher was one of the first to attempt it. The gland has been implanted in different portions of the body—the neck, under the breast, in the spleen, even in the bony substance. As a rule, however, the implantation has not been as successful as was hoped, the gland itself undergoing retrograde changes. Further-

more it would appear from the studies by Enderlen and Borst that thyroids from other animals possibly are not the best things to use as the biochemical composition of the human and animal types varies so widely as to render degeneration of the implanted gland likely. Implantation of human glands has not been successfully brought about as yet, but with the newer work on organ transplantation as inaugurated through the researches of Carrel it would seem that this technical difficulty might be overcome in the very near future.

One is compelled, therefore, to resort in most cases to the dried or liquid preparations of the thyroid itself or to such biochemical products as laboratory research has provided through the utilization of the glandular substance itself or that portion of it which presents its chief hormone activity.

The general results of thyroid medication in typical cases is fairly constant. Especially is it of value in the aberrant and minor forms of the disease of which one of the most chronic of symptoms is the persistent anemia. This may be in part overcome by the simultaneous use of small doses of arsenic, which have been recommended by a number of experimenters. Alcohol and morphine work disadvantageously, and should be carefully avoided. The use of small doses of sodium bicarbonate and bismuth work advantageously in diarrheal states.

Thyroid medication for the sporadic cases varies somewhat from its use in the endemic cases. In the sporadic cases of the light or mild type the action is quite similar to that seen in the endemic ones, but as a rule, sporadic cases by reason of their longer involvement and the less rapid development of the symptoms, their more hidden or obscure nature with their great mixture of syndromes make such cases less responsive to the therapy. Nevertheless, many of them respond very kindly to it, the same dosage being utilized.

Hyperthyreoses: Exophthalmic Goitre.—Graves described the condition in 1835, Basedow in 1840. Möbius in 1886 insisted on the relation of the disorder to changes in the thyroid gland.

Exophthalmic goitre is a disorder due to a modification of the activity of thyroid gland substance which in turn leads to an increased activity of the vegetative nervous system with a series of cardiovascular signs, tachycardia, exophthalmos, tremor and increased metabolic activity.

The disorder is widespread and presents many variations. Indeed abortive forms are among the commonest of the manifestations of hyperthyroidism. Women much more often than men show this particular type of disturbance. In Sattler's great monograph 3200 of 3800 cases reported were females. Hereditary cases are known.

Symptoms.—The chief symptoms are found in the thyroid, eye, heart and bloodvessels, skin, and muscles. Psychical, gastro-intestinal, respiratory, genital, and metabolic changes occur as well.

An enlarged thyroid is fairly constant although occasionally absent.

It is soft and elastic, rich in new bloodvessels, pulsates, and varies in volume (often very rapidly) fairly commensurate with the intensity of the symptoms. Auscultation of the enlarged thyroid often gives a marked bruit.

The heart action is rapid (tachycardia), and the pulse is very variable, reacting excessively, particularly to psychological influences. The heart sounds are increased in force, the beating being felt in the neck, and the whole chest wall is at times moved by the cardiac tumult. The blood-pressure is rarely raised and the radial and other vessels show marked hypotonus with reddening of the face, ears, and finger extremities.



FIG. 48.—Exophthalmic goitre, showing marked exophthalmos and enlarged thyroid. (Courtesy of Dr. George W. Crile.)



FIG. 49.—Same patient four months after operation (extirpation). Greatly diminished exophthalmos and change of facial expression. (Courtesy of Dr. George W. Crile.)

The *eye* symptoms consist of a marked and variable protrusion of the eyeball, with widened orbital fissure, sometimes greater on one side than the other, and felt as a disagreeable pressure and tension by the patient. The eyelids are at times swollen, and the upper, and possibly the lower lids largely retracted (Dalrymple, Stellwag) independently of and often preceding the protrusion of the eyeballs. The upper lid also does not move synchronously with the lowering or the raising of the eyeball (von Graefe's symptom), following more slowly or receding more rapidly (spasticity) than the moving eyeball, in the presence or absence of protrusion. Both signs may be unequally present. The relative infrequency of winking (Stellwag) is a frequent

symptom. Eppinger shows in tabular form the relative frequency of these ocular phenomena.

Symptom.	Wilbrand and Sänger. Per cent.	Sattler. Per cent.	Eppinger. Per cent.
Protrusion, wide, Graefe	23.0	37.3	35.6
Protrusion, Graefe	36.0	19.8	16.4
Protrusion	10.4	17.6	19.2
No eye symptoms	15.3	13.3	4.8
Wide, protrusion	2.2	6.7
Graefe, wide	2.6	5.4	9.6
Wide	5.1	1.1	3.8
Graefe	7.6	3.3	3.8
Number of cases	39.0	91.0	104.0

Löwi's symptom (dilatation from adrenalin) is frequent. Occasionally mydriasis is present, less frequently miosis. Anisocoria, or stiff pupils, may be observed as well as loss of the accommodation reflexes. Optic atrophy is an infrequent occurrence. Increased tear secretion is often observed early, dryness late in the disease. Mechanical complications—pus, ulcerations—are met with.

Insufficiency of convergence, without double vision (Möbius sign), is frequent, and is independent of protrusion.

Cardiovascular symptoms are among the most constant and early signs of exophthalmic goitre, and are due to the action of the thyroid secretion. Tachycardia (occasionally paroxysmal) is the most prominent single sign. The pulse may beat from 100 to 160 times a minute—even a 200 beat has been recorded. Undoubted cases may show no rise above 100, and great variability is the rule, especially in response to psychical stimuli. During sleep, and also on lying down, the pulse frequency drops.

Angina-like attacks, with hyperalgesia in the left ulnar region are not infrequent, and in most patients the feeling of distress and anxiety over an increased sense of heart oppression (apart from the tachycardia) is one of the most annoying symptoms. Cardiac dilatation, with later hypertrophy, without valvular defect may or may not (50 per cent.) accompany the disease, and disappear at its termination. On auscultation the first sound is usually accentuated, and systolic murmurs at the base are frequent. Valvular insufficiencies occur under special circumstances, and are often of serious consideration in operative cases.

Strong pulsation of the carotids is frequent, and though the large vessels are often prominent, and apparently arteriosclerotic, the walls are usually soft and yielding. Vasomotor instability is frequent. Marked reddening alternates with paleness. Irregular erythemata

also are not infrequent and many patients complain of surface heat, seek cold places and light clothing, even in winter, and yet show no temperature anomalies. Dermographism is also a frequent vasomotor phenomenon, as well as not infrequent nasal bleeding. Rarer urticariæ, irregular circumscribed edemas, pruritis, etc., are to be expected.

Skin symptoms are frequent. Increased perspiration is not rare and the skin is always moist with some patients, possibly one-sided, or in isolated (head) areas or smaller spots.

The electrical resistance of the skin (Veraguth-Vigoureaux) is diminished as a result of this increased secretory activity. Gooseflesh develops readily with these patients, and changing pigmentary anomalies, chiefly chloasma spots, are present on the skin, not on mucous membranes, more especially on the eyelids, neck, nipples, armpits, and genitals. In many patients (23 per cent.—Sattler), there is a tendency for the hair to fall out (sometimes unilaterally) with the development of the disease, and usually there is renewed growth of the hair with improvement.

Tremor is an early and frequent symptom, and may involve the entire body. It is extremely fine, varies from seven to forty vibrations per second, and occasionally is intermingled with wider irregular choreic-like movements. Psychological rather than physical stimuli increases the tremor greatly; lying down tends to diminish it. It is more apparent in the upper than in the lower extremities and is marked in the eyelids (Rosenbach), in the tongue, and when in the vocal cords produces a peculiar staccato breathing (Minor).

Digestive disturbances are frequent. Dry mouth may alternate with excessive salivation. There is a marked tendency to chronic alvine discharges (30 per cent.) and to vomiting without anorexia (15 per cent.). Both occur in paroxysms, somewhat resembling tabetic crises. Hour-glass contraction of the stomach may be demonstrated by the *x*-rays. Both the vomiting and diarrhea are obstinate, are accompanied by mucus or colloid, at times bloody material and the diarrhea may occur as often as four or five times a day. Fatty stools without diarrhea may occur. With both diarrhea and vomiting the patients are in grave danger. Obstipation of spastic type may also occur. In many patients there are enlarged lymphatics, tonsils, tongue follicles, thymus, and lymphatics of the intestines.

Respiratory symptoms, dyspneic in character are usual. Normal breathing is frequently irregular in depth and rhythm, and seems strained. The swollen gland may cause relative stenosis. Asthmatic tendencies are present, and the general sense of air hunger is striking, with nervous pseudohysterical coughing.

Menstrual irregularities are common. The flow is usually small in amount and infrequent in occurrence, with occasionally the direct reverse condition. Thinning of the breasts, and other atrophies (testicles) have been recorded, and seem to be coördinated with thymus anomalies.

Metabolic anomalies are characteristic. The patients become markedly emaciated and get very weak. This is related to a definite nitrogen loss, and also to a marked overoxidation of carbohydrates and fats (see fatty stools). This sudden loss of flesh and strength may come on in attacks, and then a distinct improvement takes place. Eppinger speaks of these patients as individuals who, not doing any work, need all the calories of a hard working individual. Thus in mild cases an increase in calories keeps the patient at a normal weight. The increased oxidation also shows in a mild hyperthermia. Alimentary glycosuria is frequent, and gradually disappears on re-



FIG. 50.—Exophthalmic goitre.
(Hammond.)



FIG. 51.—Exophthalmic goitre.
(Hammond.)

covery; true diabetes is an infrequent complication. Polyuria is frequent (13 per cent., Sattler), less so albuminuria (11 per cent., Sattler).

The *blood* shows anemic changes frequently at the very outset. The number of red cells is not markedly lowered as a rule save in those patients with marked cachexia. The leukocyte count is usually low; the percentage of cells is altered. Lymphocytosis is marked (60 per cent.). The polynuclear neutrophils are markedly diminished. The eosinophile cells are increased (8–20 per cent.). The large mononuclears are normal. Thyroidectomy changes the entire blood picture more towards normal, as does also ligation of the thyroid arteries and according to Kocher the blood picture is a valuable prognostic

index. The coagulation time is increased. In the young, hyperthyreosis leads to increased growth of the bones and young exophthalmic patients are apt to be very large.

The mental symptomatology of hyperthyreosis is of great importance, since from the studies of Parhon and others it seems possible that like others of the symptoms the mental signs may develop almost exclusively. In many cases the psychical signs are mild. The tendency is to both psychomotor and emotional irritability. Moodiness and sudden changes are frequent. In marked stages distinctly manic phases may develop; again acute and deep depressions (often suicidal) take their place. Thus the picture approaches very closely at times to the type of Kraepelin's mixed manic-depressives, or the more typical circular forms of this psychosis. Toxic epiphenomena may take place with ideas of reference, of persecution, even hallucinations, principally of sight. The general picture of an acute delirium is a grave sign.

The analysis of the psychical pictures in exophthalmic goitre is far from complete. Sattler advocates a catholic attitude, saying there is no one psychotic picture, a typical hyperthyroid psychosis, but the present tendency is to ally the mental phenomena of the hyperthyreoses with the manic-depressive group.

General Etiology and Pathology.—General considerations relative to the athyreoses, dysthyreoses, and hyperthyreoses as seen in the clinical pictures of congenital and acquired hypofunctioning as in cretinism and myxedema, and in hyperfunctioning as seen in exophthalmic goitre point to paralytic and irritative phenomena of the sympathetic and parasympathetic (autonomic) nervous systems.

The blood changes in exophthalmic goitre and in myxedema are similar, *i. e.*, there is a relative lymphocytosis with diminution of the neutrophile leukocytes. In exophthalmic goitre the coagulation time is increased, in myxedema diminished. In exophthalmic goitre the sympathetic irritation explains the exophthalmos, tachycardia, loss of weight, and the alimentary glycosuria. Autonomic irritability causes the von Graefe, the lymphocytosis, the diarrhea, the increased secretions. The influence of the thyroids on the carbohydrate metabolism, as seen in the rapid emaciation and alimentary glycosuria, possibly acts through the pancreatic retardation or through a relative increase in adrenalin action. That the thymus is involved in the blood picture formation seems certain.

Thus one comes to a combined neurochemical theory in that exophthalmic goitre is dependent upon hyperactivity of the thyroid secretions, which increased secretions act through the visceral or vegetative nervous system. Both autonomic and sympathetic systems are thus in a state of hyperexcitability—a condition the anatomical foundations for which are found in a certain type of individual termed vagotonic by Eppinger.

The detoxication hypothesis of Blum, Ostwald, Kocher, Klose and others, in which a dysthyreosis is assumed and that the disease

is really a type of iodine poisoning from insufficient detoxication is ingenious.

The full etiology is still very dark. Many apparently healthy individuals suddenly develop the disorder following a shock. This shock frequently involves the complex of the fear of death, loss of money. Minute analyses from the psychoanalytic school are not yet available to permit generalization, but the psychical import of shock is undoubted.

An infectious disease type is also recognized—acute thyroiditis, and also a type of iodine intoxication in individuals who have taken potassium or other iodides.

The changes in the gland itself are of little moment for the interpretation of the disorders. A great variety of variations from the normal have been described, chiefly of hyperplastic type. Apparently perfectly normal glands are at times associated with severe types of the disease. The gland is usually enlarged, elastic, the vessels dilated, and new proliferating bloodvessels are found. Kocher has described the goitrous type as parenchymatous hyperplastic struma, poor in colloid and in iodine; other changes are largely due to complications.

Forms and Diagnosis.—If the classical triad, exophthalmos, tachycardia, and goitre be present, there is little question as to diagnosis, but still all may be absent and yet the patient be suffering from severe hyperthyroidism. Hence great variability may be expected. Eppinger and Hess distinguish two chief groups according to the predominance of the sympathetic or autonomic irritative phenomena. These deserve more detailed study. The autonomic group in particular is often overlooked. These show von Graefe's sign, diarrhea, lymphocytosis and increased perspiration and marked anxiety. They are not infrequently taken for cases of anxiety neurosis or other neurasthenoid hybrids. Severe gastric or enteric crises have led to a mistaken diagnosis of tabes. Undeveloped forms may be readily overlooked, especially when the more classical symptoms just noted are not present.

Particular attention should be focussed on the thyroid itself. Its rich and increased vascularity tends to give it a peculiar consistency, even when not markedly enlarged, which is very characteristic. Kocher has compared it to the general fulness of the breast of a pregnant or nursing woman.

In a very large number of patients, especially those showing the sympathetic irritation (vagotonic) signs already discussed, hypertrophy of other lymphatics is to be observed. These are chiefly to be sought in the thymus, tonsils, tongue and rectal lymphatics. There is a tendency to elongated extremities, scanty beards in men and badly developed genitals in women. Marked lymphocytosis is also present. This relative lymphatism possibly plays a very important compensatory role in the problem of the disease and points to another subdivision of the malady. The patients with marked

psychical signs are apt to show both autonomic and sympathetic symptoms. Certain patients show only cardiovascular signs. These are those described as goitrous heart cases. They show tachycardia, dilated heart, some respiratory arrhythmia. The eyes are often shiny, pupils dilated, and striking even if not protruded. Dermographia is frequent and dizziness is often complained of. Other closely related forms suffer from dyspnea and bronchial catarrh, bleeding from the nose and congestion of the upper air passages. Neurotic goitrous heart from pressure is another special type often overlooked. There is also one-sided, unilateral mydriasis, at times tachycardia, and protrusion of the eye on the pressure side comes and goes. Rarer cases are disguised under mild diabetics, and F. Müller has described a group of pseudosclerosis cases of hyperthyroidism.

The iodine toxic cases form another group. Running from the nose, bronchial catarrh, salivation, stomachic distress, nausea, diarrhea, sleeplessness, headache, and skin eruptions are the more frequently found symptoms which may develop with but small doses of iodides.

That the blood of patients with exophthalmic goitre contains an increased adrenalin content which in some cases may be experimentally demonstrated in animals, is a point of hypothetical diagnostic value.

Occurrence and Course.—The disease is comparatively rare. The proportion of men to women is 1 to 6 on the average. Between fifteen and thirty are the most frequent years of incidence.

The course is usually chronic with ups and downs and many variations. Emotional shocks almost invariably bring on an attack or increase an existing one. Acute infections frequently bring on exacerbations, while pregnancy frequently acts advantageously. The length of time that the disease persists is extremely variable—from three months to thirty years. The prognosis also varies with the severity of the hyperthyroidism. With healthy individuals the prognosis is relatively good, with distinctly nervous (especially vagotonic) individuals it is less hopeful.

Lymphocytosis, with normal numbers of white cells, is a better prognostic sign than lymphocytosis with leukopenia.

Treatment.—This may be surgical or by internal remedies. Surgical treatment is the most radical. The statistics of various operators has shown improvement in from 6 to 76 per cent., death in from 2 to 22 per cent.¹ Kocher has reported 76 per cent. good results. These figures are probably high, if ultimate results are meant. The operation of choice is the successive elimination of thyroid substance, with minimal handling of the gland. This is advocated particularly by Kocher, whose lethal results have been from 3 to 7 per cent. Death frequently results in narcosis, with a very characteristic symptom-complex. The face gets red, the whole body becomes tremulous and breaks out in perspiration. Diarrhea supervenes, the temperature rises and

¹ Eppinger, *loc. cit.*, p. 70.

the heart action becomes excessively rapid, and death with cyanosis and dyspnea takes place. Status thyreolympathicus is possibly responsible for these results.

The sympathetic operations are not to be recommended. They help the eye symptoms possibly, but the disease is not one of the cervical sympathetics alone.

Internal therapy is still unsatisfactory. Rest in bed is primary and essential. Any remedy increasing the thyroid secretion is bad, hence thyroids and iodine are to be avoided. Thymus has been tried, with best results in the sympathetic types. The fresh gland is given by mouth. α -ray treatment as at present developed is justified in a limited number of carefully chosen cases. The chronic infectious, hyperplastic goitres do best with light therapy.

Pharmacological agents which act to diminish the thyroid secretion have been used. Chief of these is belladonna. It is the most reliable of the internal remedies and can be given in fairly large doses. Adrenalin in solution by rectum is of service at times. Vagotonic cases react best to its influence, the tachycardia and diarrhea being well influenced by it.

The digitalis group of glucosides are not to be recommended, neither is iron of any service.

Arsenic and bromides may be of passing service, especially the latter in aiding sleep.

Psychotherapy is above all of great value, especially as applied toward an education of the patient toward his fears in the sense of Dubois. Psychoanalysis is of the most signal service in a large group of cases.

Serum treatments aiming to exert a lytic action upon the secretory cells of the thyroid have been devised. The most promising are those of Rogers and Beebe.

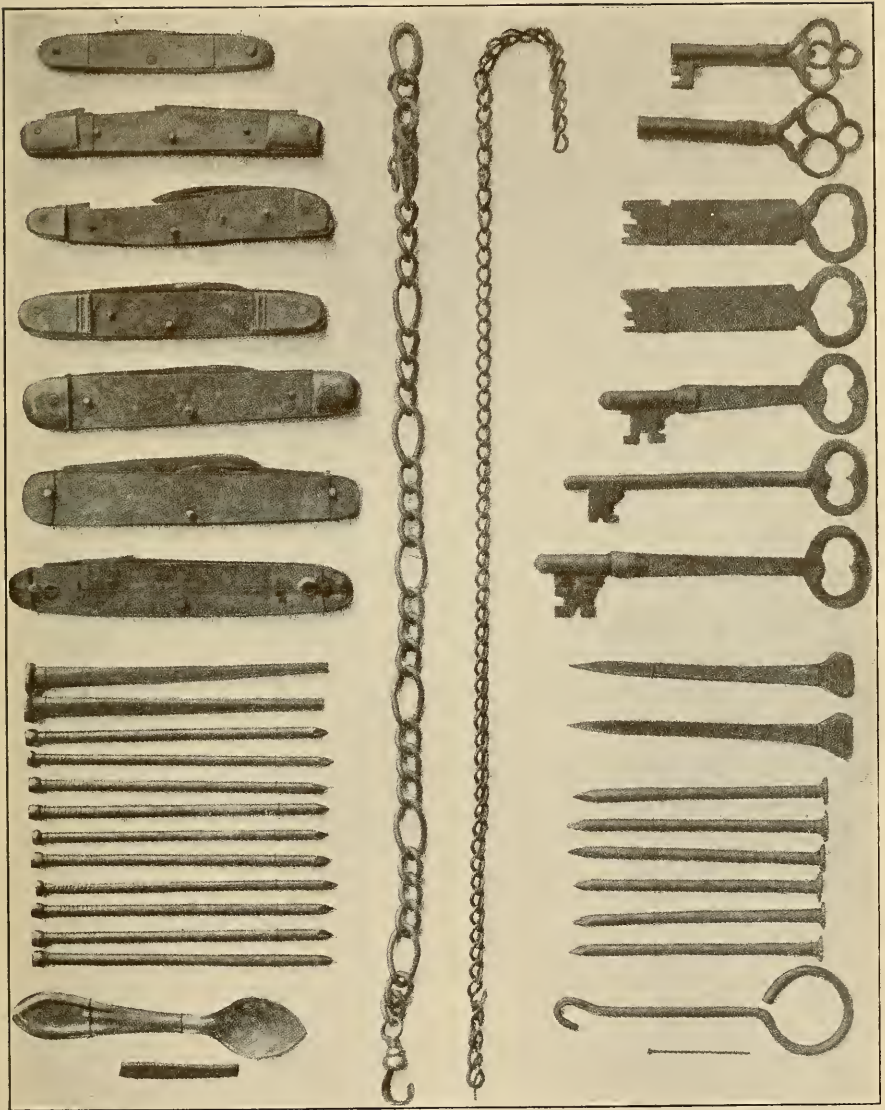
• PARATHYROID SYNDROMES.

Tetany.—Corvisart first termed this "tetanie" in 1852. Frankl-Hochwart, in 1887, clarified this conception, and since his striking description the term tetany has had universal recognition.

It is highly probable that tetany must be regarded as an example of hyperfunctioning of certain peripheral parts of the motor mechanism in which altered muscular excitability is secondary, while special vegetative neurological features, namely, parathyroid metabolism, plays the primary role.

Symptoms.—Tetany has, as its main symptoms, tonic, intermittent, bilateral, often painful cramps, which, without, for the most part, any loss of consciousness, involve the muscles of the upper extremities, particularly the hand, which is held in the obstetrical position. The muscles of the lower extremities may also be involved, those of the larynx, of the face, and of the jaw, seldom those of the chest, abdomen,

PLATE V



Objects Removed from Stomach of Patient Suffering from Gastric Tetany. (Warbasse.)

neck, diaphragm, or tongue. In rare cases the eyeball muscles are implicated, as is also the bladder. In the sensory sphere paresthesia and pains are present, while hyperesthesia occurs now and then. Pressure upon the brachial plexus may give rise to an attack (Trousseau); hyperexcitability to electrical currents is present (Erb); mechanical hyperexcitability of the muscles and motor nerves is observed (Chvostek), while the sensory hyperactivity to mechanical and electrical stimuli is also present (Hoffmann). The psyche is rarely uninvolved, and following operative removal there has developed extreme anxiety with the sense of impending dissolution.

In chronic and repeating forms secretory and trophic disturbances occur, such as increased perspiration, reddening of the skin, swellings of the joints, mild edema, falling out of the hair and nails, discoloration of the skin, urticaria, and herpes. Dyspnea may intervene; polyuria and glycosuria are rare accompanying symptoms. Abortive and incomplete forms have been designated "tetanoid" by Frankl-Hochwart.

For didactic purposes, Frankl-Hochwart divides tetany into simple and acute forms and chronic recurring forms. A further division of forms occurring in children and in adults is made. Tetany of the adult he groups into seven classes: (1) Tetany idiopathica—tetany of otherwise healthy individuals—workman's tetany. This is the form which seems to occur epidemically as an acute, or acutely recurring affection in certain cities, notably Vienna, Heidelberg, etc., principally in the early spring months, and among certain handworkers—tailors, shoemakers, etc. (2) The tetany of gastric and intestinal affections. (3) The tetanies of acute or infectious diseases, typhoid fever, cholera, measles, scarlet fever, etc. (4) The tetanies of acute poisoning, chloroform, morphine, ergot, phosphorus, renal, and genital substances. (5) The tetanies of maternity (pregnancy, parturition and nursing). (6) The tetanies of parathyroid involvement. (7) The tetanies of other nervous diseases, exophthalmic goitre, brain tumors, cysticerci, syringomyelia, etc.

Incidence.—Tetany in its different manifestations is undoubtedly rarely seen, and is even less frequently reported. In undeveloped phases the tetanoid reaction is comparatively frequent in children. The instances of gastric tetany are probably the most frequent, while the pure epidemic form has not been encountered in the United States. In Griffith's study only 77 cases were found recorded, while Howard's later collection brings the American cases to 154 in 1907. The incidence in the Johns Hopkins Hospital has been 0.05 per cent. Thus, so far as clinicians in English-speaking countries are concerned, tetany may be considered as being infrequently seen, but even in countries in which it is thought to be epidemic, it is rarely observed. It is undoubtedly often overlooked, and at times confused with hysteria.

Etiology.—It is unnecessary to go through all the steps which have led to the knowledge that the parathyroids play an important role in the functions of the body, and that the peculiar muscular hyper-

excitability seen more particularly in tetany is in some measure related to these glands, especially as a reaction to their defect or deficiency. It is possible that other anomalies of neuromuscular activities are correlated with these glands. Lundborg has advocated wide hypothetical possibilities; even the peculiar motor manifestations of a group of mental disorders—katatonias and their allies—may have some light thrown on them through this avenue.

More recent workers have not rested on the apparently certain foundation that tetany is essentially a manifestation of perverted parathyroid activity, but have sought to bring the anomalous forms into conformity with this conception, and further, to obtain a more fundamental insight into the essential features of the disturbed neuromuscular reaction. Whether the work of MacCallum and Voegtlin¹ has definitely solved this problem is to be determined, but it would appear that the essential factor has been found in the relation of the parathyroid to the calcium metabolism of the body, the hyperexcitability of the neuromuscular apparatus is primarily due to a lack of calcium in the blood, and this is thought to be due to a relative or absolute insufficiency of the parathyroid glands. Just how the calcium exchange of the body is controlled by the parathyroids, and what role in such control is played by other glands, is not known.

The second series of factors still remains to be solved. Why is it, if the facts just quoted bear an essential relation to tetany, that the disease reaction seems to be so strictly localized? Is this simply bad observation on the part of the clinicians, or are there local conditions of diet, water supply, etc., that bring about changes in the intake of calcium? Such conditions are known to prevail for certain infants fed on cow's milk, and have been extensively studied.²

Why do certain workers—tailors, shoemakers, etc.—suffer and not others? Is Frankl-Hochwart's suggestion regarding the peculiar position adopted by these workers of value in relation to the blood supply of the organ? What have gastric dilatation, gastro-intestinal disorders, etc., to do with the calcium intake? Are there perversions of gastric chemism that favor gastric exclusion? This must be determined in order to explain these cases, and why should certain gravid women develop a parathyroid insufficiency?

Pathology.—Concerning the histological changes, the present view excludes a specific pathology. The insufficiency of the parathyroids, be it relative or complete, may be brought about by a great variety of lesions. Such have been described by various observers, and each, in turn, has been considered of specific significance. These in reality offer evidence in favor of the parathyroid insufficiency hypothesis, but go no further. In the minor grades of tetany in children, particularly in so-called spasmophiles which Frankl-Hochwart regards as tetany, the findings of Yanase of Escherisch's clinic are

¹ American Journal of Insanity, 1909.

² See Escherisch's Monograph, *Tetanie der Kinder*, 1909, for most recent summaries.

illuminating. Here hemorrhages in the parathyroid seemed fairly constant findings, and offer an explanation of the galvanic hyperexcitability. At the other extreme one finds the absolute insufficiency tetanies in experimental parathyreopriva and in the rarer cases, such as Pool's in man. Here the parathyroid insufficiency is established, and the calcium treatment restorative. In acute epidemic forms thyroid (and probably parathyroid) involvements are known. Tumors, tuberculosis and a host of other changes in the thyroids have been described. It will probably be found that in most of these the parathyroids are likewise implicated. Thus, in exophthalmic goitre a combination of thyroid and parathyroid symptoms is often present. In many tetanies pure thyroid symptoms appear.

Symptoms.—Considerable variation is to be found, but in general four types of symptoms are observable in the fully developed attack. These are the muscular spasms, which may go on to an exhaustion paralysis, or paresis; the Trousseau phenomenon; increased electrical excitability, or the Erb symptom; and mechanical hyperexcitability of the muscles—Chvostek's sign. In some patients one or more of these may be missing. Abortive forms, so-called, may present even fewer signs. On the other hand, a richer combination of symptoms, apparently closely related to the general disorder, may be encountered. Sensory disturbances, anomalies of circulation with edema, of respiration with cyanosis, and of temperature are sometimes found. True psychoses, perhaps indistinguishable from the hysterical confusions, are found. Trophic disorders of the skin, hair, and nails occur. In some rare instances, widely confused phenomena resembling epileptic seizures occur.

Muscle Cramps.—The most persistent feature consists in a characteristic form of muscular spasm. It occurs chiefly in the extremities, mostly involving the flexors. The spasm is tonic, generally bilateral, and is usually induced by some irritation, mechanical or electrical. Overexertion, exhaustion, changes in temperature, acute diarrhea, or emotional excitement may precipitate an attack. The small muscles of the hand are usually first implicated, perhaps after tingling-like prodromata. There is marked adduction in the interossei and the thumb. The hand usually takes a very significant position—that of the "obstetrical hand." It is also described as a "penholding" position. In many mild attacks only the thumb may be involved, and in others the hand alone. Sometimes the hands are closed, making a fist. Flexion at the wrist may follow, the arms then being folded across the chest, or they may be held up in the air or down at the side. Such wider movements occur in the more severe attacks only. A somewhat similar series of flexor cramp-like movements may occur alone or in combination in the lower extremities. Talipes equinovarus, inversion of the foot, and the plantar flexion may be present. The legs may be flexed on the thigh, and the thigh on the pelvis in the most severe instances.

Considerable emphasis may be laid on the bilateral character of these muscular movements. It is extremely rare in the true tetany reaction to find one side alone involved.



FIG. 52.—Tetanic spasm, showing flexion of wrist. (Pool.)

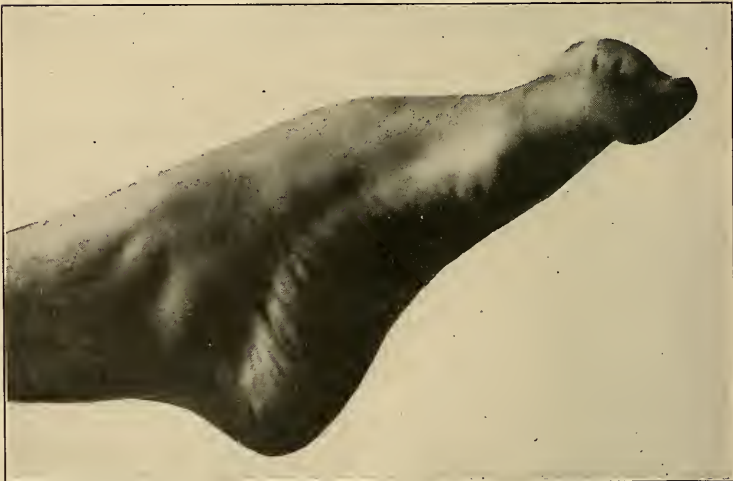


FIG. 53.—Tetanic spasm, showing plantar flexion of foot and toes. (Pool.)

These muscular cramps persist for a very variable length of time. Not only do they vary in different individuals, but the same patient in different attacks, or at different times in the same attack may show

marked variation. In the majority of cases reported the spasms persist for from fifteen minutes to an hour, and two or three hours is not an excessive period. Hoffmann has reported a persistent cramp which lasted for ten days. In fatal cases the contractions pass over into a lethal continuous spasm.

Clonic spasms are rare, but are known as blepharospasm, spasms of the tongue, etc. Postconvulsive paralysis or paresis is an uncommon outcome. Excessive muscular tire, however, is not rare.

Course.—Clinicians have recognized arbitrarily three groups of cases in adults, and most modern authors are inclined to follow Trousseau in his classical description. In the *benign* form the sensory phenomena, such as formication or a simple sensation of heat, may precede the spasms. These are confined for the most part to the hands or occasion-



FIG. 54.—Method of producing tetanic spasm of hand by stretching the brachial plexus by forcible abduction of the arm. Note "obstetrical" hand. (Pool.)

ally to the feet. The contractures may be fleeting, persisting for from five to fifteen minutes, or they may persist for an hour or more. Often the attack terminates by a recurrence of the sensory symptoms. A period of repose lasting for a quarter of an hour to two or three hours supervenes, and the spasms recur. In some instances two or three attacks a day may persist for several months. These mild attacks may recur at yearly intervals, or, as in some of Frankl-Hochwart's cases, several years may elapse, and then they will recur. These benign cases are usually unattended with much pain. Consciousness is not disturbed, and there are usually no sensory, trophic, or temperature disturbances. Again, more *severe* attacks are observed. These occur more often in young adults, the benign forms having been mostly observed among children. Here the contractions are more

violent and more painful. The preceding sensory phenomena are usually more pronounced, and with the increase in the severity of the condition other symptoms may be noted. Headache, malaise, and a rise in temperature of 1° to 3° may be noted. The affected muscles may show signs of congestion, and localized edema of the hands and feet may be observed. Other muscles than those of the extremities may be involved. The muscles of the abdomen may be frequently contracted or the sternocleidomastoid and the pectorals; strabismus may be noted, either the internal, or the external rectus, particularly the latter, being involved. Trousseau first noted the spasms of the larynx, the pharynx, the bladder, and the muscles of respiration, a severe dyspnea supervening in the event of the latter muscles being affected.

These severe attacks are rarer than the benign ones. Frankl-Hochwart has shown that there is a distinct tendency for the well marked lighter cases in many instances to become graver, and the good prognosis which most writers have given is now seriously doubted by this observer.

In the *grave form* there is no addition of symptoms. The attacks occur with greater and greater frequency and become more and more intense, and the patients die as a direct result.

Diagnosis.—The diagnosis of a classical case offers few difficulties. In English speaking countries it is apt to be overlooked, although the more frequent reports of recent years point to the fact that it is being recognized more often, especially in its milder forms. The presence of cramps in the upper extremities, alone or in conjunction with the upper limbs, with the classical obstetrical hand and the additional evidence supplied by the Chvostek, Trousseau, and Erb signs, is usually sufficient to determine a diagnosis. Frankl-Hochwart would separate the different forms, saying that gastric tetany often offers particular difficulties, especially as a group of individuals exists in whom with gastro-intestinal disturbances there is a tendency to cramp-like contractures with the typical hand position. He groups them with the hysterias, rather than with the tetanies, however.

Tetany strumipriva, or, better, *parathyreopriva*, as suggested by Erdheim,¹ offers the most classical manifestations of the disorder, throwing, as well, considerable light upon some of the possible underlying and fundamental features of this peculiar reaction type. Insufficiency of the parathyroids results in convulsive phenomena of the tetany type.

Prognosis.—The point of view here maintained precludes the possibility of the statement of a general prognosis. Very little is known definitely of the prognosis in infants and children. Most authors agree in giving a fairly good prognosis, although Frankl-Hochwart says that healthy children rarely acquire convulsions, and that the

¹ Mitt. a. d. Grenzgebiet. d. Med. u. Chir., 1906, vol. xvi.

prognosis is not good. In many of these children only one tetany-like spasm has been noted. In others the spasms may persist for weeks and even months. In simple cases the prognosis is much better than in those complicated especially with gastric or intestinal affections. Bronchitis, pneumonia, and occasionally an ascaris infection also determine a less favorable prognosis. Dangerous signs appear with glossal cramps, which may cause death. Recurrences are frequent in those who recover.

Tetany coming on during pregnancy and childbirth usually has a good prognosis. The hyperexcitability of the nervous system may persist for weeks after delivery. In succeeding pregnancies the recurrence of the phenomenon may be looked for. Frankl-Hochwart notes that in succeeding pregnancies the attacks are apt to be milder. It should be borne in mind, however, that sudden death may occur in these cases. The classical cases of Trousseau and Szukitz are cited.

In the cases apparently due to disturbances of the stomach surgical interference has brought about distinct amelioration. Sudden death may occur, and apart from surgical intervention the prognosis is admittedly bad (70 to 80 per cent.). The cases are comparatively rare, however. In severe cases associated with marked gastric dilatation, operation, if only exploratory, is advisable. The mortality after operation in some dozen or more cases now reported is as low as 30 per cent. The subsequent history of these patients remains to be reported.

Tetany following infectious diseases and acute or chronic poisoning seems to present a favorable prognosis, perhaps the best of the various forms.

The prognosis of tetany thyreopriva depends upon the amount of thyroid gland removed and whether the parathyroids are included. Total extirpation of the entire thyroid and parathyroid tissue is recognized to have a fatal outcome. Tetany appears after total removal of the parathyroids, not only in man, but in lower animals. What the outcome in man may be of complete parathyroidectomy with the retention of thyroid tissue is not yet satisfactorily determined.

The most important class numerically is the so-called epidemic tetanias. Frankl-Hochwart, in his earlier papers, gave a fairly good prognosis in this form, but in later contributions he presents the further histories of these patients, and finds that the prognosis is far from being good.

Treatment.—From the standpoint here outlined it may be readily deduced that a general treatment is not a rational mode of approach. The parathyroidectomized individual would not be benefited by a gastric operation. The essential feature in the general treatment is the search for the irritant, and this cannot always be successful.

In approaching any specific instance the first question is as to the validity of the phenomenon. This is placed first, because it should be the simplest factor to exclude. If no reason, remote or apparent,

can be found which would account for simulation, aggravation, or suggestion, it may be assumed that the excitation of the nervous system has other than a purely psychogenic origin. In rare instances, simulation has been a factor; thus, patients have simulated tetanoid contractures in order to obtain the pleasure of chloroform semimarcosis, and continue a habit once acquired. Hospital parasitism counts among its devotees a number who find the tetanoid spasm a source of interest and advantage; but these cases are not common.

With a positive diagnosis established, the organ involved should come into review. Inasmuch as parathyroid insufficiency is the most general cause, it is rational to treat those cases, many in children, the whole group of so-called idiopathic tetanies, many tetanies of pregnancy and of thyroid disease, by thyroid and parathyroid preparations. Parathyroid preparations seem to fulfill most of the conditions, yet occasionally the combined thyroid and parathyroid involvement renders the giving of the combined products of more service.

The use of foodstuffs rich in calcium and of calcium salts follows as a natural corollary from the studies enumerated. For the most part, it would seem that such medication might entirely replace the use of the glandular substances themselves. In experimental tetanies the successful effect of the calcium salts has been very striking, and in tetanies in children calcium therapy has given almost uniformly good results. Such therapy apparently renders the older means unnecessary, such as curare, opium, hyoscyamus, the bromides, chloral, belladonna, chloroform, galvanism, sweat baths, etc. Up to the present time therapeutic experience is not sufficient to definitely prove the durability of calcium medication in the cases in which it seems needed.

The surgical expedient of transplanting parathyroid tissue has proven successful in animal work;¹ its successful application in persistent chronic tetanies in man is clearly foreshadowed by the experimental work on dogs. The technical difficulties do not seem insuperable in view of the ready transplantation of these structures in different parts of the body.

Inasmuch as comparatively definite light has been thrown on the whole subject of the tetany reaction, by the researches of MacCallum and Voigtlein in particular, the stomach tetanies, heretofore a particularly obstinate group, offer opportunity for a combined treatment, radical as well as conservative. Useless gastric operations need not be performed, yet at the same time the lines are laid down that point to the time when such operations may be of life saving service. When obvious gastro-intestinal conditions point not only to such irritants in the cycle, it were folly to persist in a line of medication which, notwithstanding its theoretical possibilities, does not give results. Gastrostomy, gastrectomy, gastroduodenostomy, etc., accord-

¹Leischner, Arch. f. klin. Chir., 1907, lxxxiv. 1, 20S.

ing to the gastric lesion, may be demanded, since the gastric intestinal mucosa seems to be an important factor in the possibility of gastric intake.

The best methods of administration of calcium salts are matters of individual experience. Foodstuffs rich in calcium, milk, eggs, whey, etc., are clearly useful forms. Injections of calcium chloride, which have been useful in animal work, may be adopted in man. Calcium hypophosphites or other salts to be taken by mouth are available, and intestinal enemata are indicated if other avenues of medication are contra-indicated. The exact dosage remains to be determined.

DISEASES OF THE HYPOPHYSIS—PITUITARY.

Clinically three main trends may be recognized which are due to increased, diminished or irregular functioning of different parts of the pituitary structures. The analyses of these syndromes has only just begun, but in view of Tilney's fundamental studies on the hypophyseal structures, a definite classification will probably develop: (1) *Hyperpituitarism* is associated with gigantism and with acromegaly; (2) *hypopituitarism* with various grades of infantilism, physical and mental, with adiposity and genital dystrophies; (3) *dyspituitarism* shows many mixed syndromes.

Hyperpituitarism: Acromegaly and Gigantism.—These conditions are apparently closely related and intermingled, showing overgrowth in the skeleton and particularly in the long bones in gigantism; changes in the toes, fingers and bones of the face, more prominent in the acromegalic tendency. In general gigantism occurs when the disorder begins prior to epiphyseal union, acromegaly when the changes occur after the union of the epiphyses. Prodromata such as fatigue, muscular pains, apathy and sleepiness are frequent.

The growth in acromegaly is very gradual. It includes changes in the skin and hair as well as in the bones. These are all hypertrophied, causing marked variation from the usual. The eyebrows are heavy and overhanging, with coarse hair; the lips are thickened and protrude, with marked projection of the lower jaw. The mucous membranes share in the hypertrophy. The hands and feet are notably widened, the fingers and toes stumpy and thick. The skin and hair throughout, inclusive of the genitals, show the same hypertrophies, as do practically all of the bones of the skeleton. Amenorrhea is frequent in women and loss of potency in men. Glycosuria is frequent. Carbohydrate tolerance may be high, however, and an increased fondness for sweets is frequent.

Symptoms.—In addition to the essential metabolic disturbance, symptoms due to the nature of the producing lesions—tumor, hyperplasia, *i. e.*, neighborhood symptoms, are frequently found, but these are not invariable.

Severe bitemporal headaches are frequent. This is an intracranial pressure sign. The sella turcica is usually enlarged from tumor formation, and this is readily seen by *x*-ray.

Pressure upon the optic nerves at the chiasm is usual, leading to various types of hemianopsia. Distorted fields are the rule. Pupillary phenomena are frequent, and visual defects are often serious and progressive.

Mental symptoms ranging from sluggishness to complete deterioration occur, but are not invariable.

Other of the endocrinous glands may show changes.



FIG. 55.—Characteristic hand of acromegaly. Note heaping of tissues about nails, "type en large" of Marie. Compare with Fig. 56. (From Cushing's "Pituitary Body.")

Prognosis.—This is always grave. The disorder is progressive, usually very gradual, five to twenty years, but the advance in symptoms may be arrested.

Hypopituitarism.—Deficiency of the hypothetical pituitary substance gives rise to a group of syndromes the most classical of which is Fröhlich's *dystrophia adiposogenitalis*. This is characterized by a progressive accumulation of fat throughout the body, chiefly showing about the buttocks and breasts, and by a lack of development of the primary and secondary sexual characters.

Thus there is a stunting of the growth physical and mental, a hypoplasia genitalis, and a failure to develop the voice, the mammaræ and the scrotum or testicle. As a rule there is a fairly persistent

though slight subnormal temperature, a marked degree of sugar tolerance, slowed pulse and a tendency to sluggishness or even sleepiness. Taper fingers are a contrasting picture to the pudgy ones of acromegaly.

Symptoms.—Neighborhood symptoms may also be observed as with the acromegalic symptoms. The optic nerve changes, bitemporal hemianopsia, are among the most important. Other symptoms of a general nature, as headache, nausea, vomiting, changes in the sella turcica, etc., often occur, especially from tumors which destroy the hypophysis.



FIG. 56.—Typical tapering hand of adolescent hypopituitarism. Compare with Fig. 55. (From Cushing's "Pituitary Body.")

It is by no means clear as yet whether the theory of a hormone is an adequate explanation. The theory of action through the vegetative nervous system is probably more satisfactory.

Dyspituitarism.—Under this head, the majority of the anomalies due to disturbed pituitary structures may be gathered. These are incomplete forms of acromegaly and gigantism, cases of adiposity, alone or with genital atrophies, or genital anomalies, showing hyperfunction or hypofunction. Various epilepsies, probably conditioned by hydrocephalic changes are examples of dyspituitarism. Variations in mental capacity are frequent, as well as a variety of anomalies such as increased sugar tolerance or glycosuria; slightly sub- or supra-normal temperatures, polyuria, wakefulness, irritability and a group of character anomalies as well. The skin is usually smooth

and soft and free from moisture, and the hair is apt to be thin, fine and scanty.

Treatment.—Acromegalic patients, or those showing pituitary syndromes due to evident tumor of the hypophysis region, need surgical intervention, whether the signs of hypo- or hyperpituitarism be present. Hypopituitary and dyspituitary cases without neighboring symptoms



FIG. 57.—Case of post-traumatic hypopituitarism in a child, with extreme adiposity, high sugar tolerance, and epilepsy. Marked improvement with whole gland feeding (pituitary). (From Cushing's "Pituitary Body.")

of tumor may be fed pituitary extract, sometimes to advantage. Certain stationary acromegalic cases are benefited by this treatment and others apparently are rendered stationary.

Radiotherapy is in general inefficient.

The chief surgical modes of relief are: (1) sellar decompression, for hypophyseal headaches, or to permit a tumor mass to expand outside

of the cranial cavity; (2) partial removal of a hyperplastic and over-acting gland; (3) partial removal for sake of saving eyesight; (4) subtemporal decompression to relieve general brain pressure symptoms; (5) combined operations;¹ (6) operations for glandular transplantation.

POLYGLANDULAR SYNDROMES.

Under this broad grouping, a large number of syndromes, due to various glandular insufficiencies or hyperactivities, may be conveniently placed. French clinicians have studied them extensively



FIG. 58.—Adipose genital dystrophy. Tumor of pineal. (Bailey and Jelliffe.)

as has already been indicated. Some of the more striking of these syndromes are here briefly mentioned. Their more extended consideration may be found in the works already cited, notably those of Falta, Lewandowsky, Cushing and Biedl. Clinically speaking they make a large and variable medley.

Pineal Syndrome.—Here teratomata are chiefly represented. The patients, usually young, show the signs of: (1) general pressure, especially with quadrigeminal signs; (2) metabolic anomalies—adiposity, sexual precocity and abnormal hair development.²

¹ See Cushing, *Pituitary Body*, Lippincott, 1912.

² Bailey and Jelliffe, *Tumors of the Pineal Body*, *Arch. of Int. Med.*, December, 1912.

Adiposis Dolorosa.—Dercum first named one of these syndromes. The patients slowly develop fatty deposits, often enormous in amount. This adiposity may show as nodular deposits (symmetrical lipomatosis), varying in size from a bean to an apple. They may be circumscribed or the adipose deposits may be generally diffused throughout the entire body. The hips, shoulders, upper arm and abdomen are predilection sites. The skin is tense. The fatty deposits are painful to pressure, especially at nodular points, and even at times before there is much fatty infiltration. Hyperesthesiæ and paresthesiæ in the form of tingling, burning, numbness, etc., are frequent. Spontaneous pains occur with some. They are sharp and intermittent, localized in the skin or more deeply, and often increase with motion. Asthenia is a marked sign and psychological alterations are the rule. These latter are mostly in the nature of depressions with, at times, suicidal ideas, irritability, capriciousness and other signs closely resembling

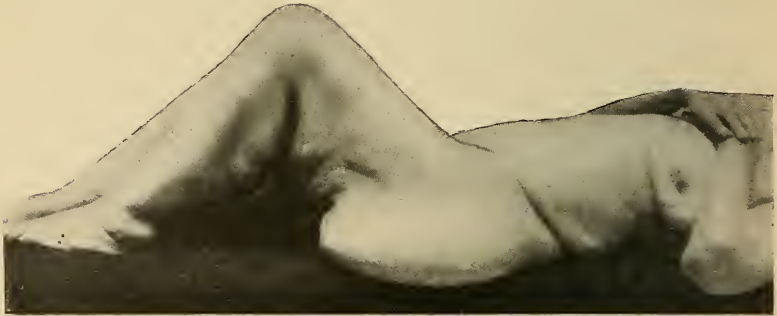


FIG. 59.—Adiposis dolorosa. (Dercum.)

manic-depressive states (*q. v.*) with flight of ideas and confusions. Other patients show marked deteriorations.

Various anomalous conditions have been observed with different patients, such as vasomotor signs with edemas, ecchymoses, hemorrhages, and pigmentation. Hyperidrosis, anidrosis, and trophic changes also are recorded. Various complications with other members of this vast collection of chemical metabolism anomalies are too numerous to mention.

The disorders usually progress slowly and vary greatly in intensity from year to year. The patients recover spontaneously or finally die of intercurrent disease.

The organs involved have been the hypophysis and the thyroid chiefly, but the exact relationships are still uncertain. Hypophyseal tumors are not infrequently found, ovarian disease is present in some, vascular neurotrophic disorders (blood gland disease) are present in still others.

Therapy.—Some patients have been helped by thyroid feeding, others not. Surgery is needed in the tumor cases. Other symptoms are met on their own indication.

Genital Syndromes: Agenitalism, Hypergenitalism and Hypogenitalism.—Testicular and ovarian syndromes are frequent and give rise to a motley group. Aplasias of the genital glands, hermaphroditism, eunuchism, eunuchoidism (artificial and natural, early and late), hypergenitalism, chlorosis, menopause irregularities, osteomalacia, infantilism, achondroplasia are the more frequently utilized symbols now current.

Inter-renal syndromes may be considered here, since the cells of the inter-renal (suprarenal) and the sperm cells (gonads) have a common origin. The chief syndrome of interest is that called *Addison's disease*. Here the chief signs are great muscular asthenia, gastrointestinal disturbances, and pigmentation—bronzing of the skin. The loss of adrenalin is responsible and this is under nervous (vegetative) control.

Status Thymolymphaticus.—The thymus has very close relationships to the gonads, and it is thought that the thymus and testes are reciprocally acting organs. This does not seem to be true for the thymus and the ovaries.

Certain individuals have excess of thymus lymph tissue throughout the body. In recent years the researches of Bartel,¹ Wiesel and A. Paltauf have shown that this condition is very frequent. Notwithstanding the fact that a pathological diagnosis postmortem is easily arrived at the clinical diagnosis during life presents many difficulties. This latter is largely due to the circumstance that the diseased organs are difficult to examine and, furthermore, the signs of defective development which result from the condition are often very slight.

The recognition of status thymolymphaticus often requires exhaustive chemical, physical, x-rays, and other forms of examination. Such recognition is highly important, however, since these individuals, if they may be grouped together, are prone to react very markedly to anesthetics, bodily shocks, infectious diseases, and to drugs, particularly salvarsan, sera, and mercury. A large medley of conditions accompanying and partly due to status thymolymphaticus, have been described. These may be summarized as follows:

1. *General Signs.*—As a rule the length of the body exceeds the stretch between extended finger tips. The length of the upper part of the body and of the extremities is above the average. The arrangement of fatty tissue tends to make the male resemble the female type, and *vice versa*. The mental status is inclined toward the infantile. Alcohol resistance is very slight.

2. *Face.*—The under jaw and the mastoid processes are underdeveloped and the former results in faulty bite, anomalies of dentition

¹ Status thymicolymphaticus, Deuticke, 1912.

by displacement and by crowding. The palatal arch is high; the tonsils and tongue papillæ are increased in size. The epiglottis is inclined to be infantile in type. Epicanthus, eccentric pupils, irregularly pigmented irides, adherent ear lobules and narrow external auditory meatus may be present.

3. *Neck.*—The thyroid, cervical, and other glands are enlarged.

4. *Skeleton.*—The thorax is long and narrow. Cervical floating ribs are present. Compensatory lordosis of the spine is lacking. The scapulæ are wing-shaped. The pelvis develops heterosexually; the sacrum is small, the pulse high. Hyperdactyly, flat-foot, and hyperextension of the elbows may be looked for.

5. *Hair.*—Axillary and pubic hair are diminished; the extremities may be hairy.

6. The thymus is enlarged, the breasts resemble those of the opposite sex; polymastia may be observed. The aorta is narrow, the heart small, the blood-pressure low. Palpitation is frequent and there is cardiac dilatation with weakness.

7. In the abdomen ptoses are frequent. The jugulopubic distance is increased, the abdominal circumference diminished. The spleen is enlarged, the kidneys prolapsed. There is a tendency to orthostatic albuminuria and to alimentary glycosuria.

8. The blood picture shows a neutropenia, lymphocytosis, and eosinophilia.

9. The genital anomalies are in the nature of cryptorchis, hypoplasia, disturbances of menstruation and secondary sexual characters of the opposite sex.

10. There is a marked disposition to other disease and usually a tendency to an increase in the severity of the disorder. Thus, tuberculosis shows more often in other organs than the lungs; infectious diseases of childhood are severe; there is a tendency to tetany, gliomata, syringoses, hydrocephalus, tabes, paresis, myasthenia. Diabetes, excessive fat and gout occur. Pernicious anemia, leukemia and chlorosis, exophthalmic goitre, Addison's disease, osteomalacia, nephritis, eclampsia, asthma, infantile emphysema, eczema, hemangiomas, appendicitis and tumor formation are among other accompanying disorders.

Observation of many cases of status thymolymphticus shows that little weight is to be given to the occurrence of isolated symptoms. The diagnosis consists in the accumulation of the anomalies. The differences in body dimensions are of universal importance, whereas the increase in the tongue follicles and the infantile character of the epiglottis and its frequent omega shape, are more characteristic. Genital hypoplasias are frequently associated with eosinophilia and lymphocytosis is to be expected.

The inter-relationships of the various endocrinous glands may be illustrated by the reproduction of the series of charts or diagrams in Paton's admirable treatise on the *Nervous and Chemical Regulators*

of *Metabolism*, London, 1913. As Paton well remarks, these may well be a grotesque parody of what will ultimately be found to be the relationship of the activities of these organs. "They are probably as near the truth as those quaint ancient maps of the Indies with their 'here be much gold' scrawled across them, which served as the charts of our forefathers, but if, like them, they merely indicate the direction which further investigation should take and suggest lines of attack, they will have served their purpose."

The direct and profound action of the secretions of the sexual glands (gonads) upon the body is seen in every tissue of the body. How far their action is facilitated and how far checked by other endocrinous organs is not yet entirely worked out. The thymus supplements the action of the testes secretion. Its relations to the ovaries is not so certain. It exercises a checking action on the male gonad activity which in its turn acts reciprocally on the thymus. (See Figs. 60 to 63.)

The removal of the thyroid checks the growth of the gonads. Castration acts less on the thyroid, although menstruation, childbirth, and the menopause cause marked thyroid activity. (See Thyroid, Figs. 60 and 63.)

The destruction of the pituitary leads to gonad atrophy and reciprocally castration causes hypertrophy of the pituitary. The secretion of both stimulate the growth of the long bones, the uncontrolled activity of the former leading to gigantism and acromegaly. The action of the gonad secretions is to check the pituitary activity and the increase in size of the eunuch is possibly a response to this unchecked hypophyseal activity. The gonads are not alone in hindering the pituitary action.

Suprarenal and gonad activity are closely related and suprarenal loss is usually accompanied by genital aplasias or anomalies. Paton has suggested the identity of certain elements of these tissues and that the suprarenals constitute a sort of bridge or intermediary between the bodily and the sexual cells.

The thyroids and pituitary are closely related. Removal of one causes hypertrophy of the other. (Fig. 60.) They thus mutually check each other in part and are also coöperative, the pituitary needing the thyroid to complete its activities. Hyperthyroid activity does not lead to hyperplasias of connective or bony tissues as does hyperpituitary action; the reciprocal autonomic and sympathetic nerve activity is not exactly similar; although diminished activity of both substances may lead to diminished bony growth—atrophy. The vegetative mechanism of this, however, has not yet been elucidated.

The action of thymus on thyroid is far from clear, but the tendency is to show a reciprocal checking action especially on the neuromuscular apparatus. The problem of myasthenia gravis has been thought to lie behind this reaction.

The thyroids and parathyroids have distinctly different and even

antagonistic activities: The former seems to be related more distinctly to the iodine, the latter to the calcium metabolism of the body. Just how they are regulated through the vegetative nervous system is unknown. Calcium is of pronounced value in neuromuscular activity

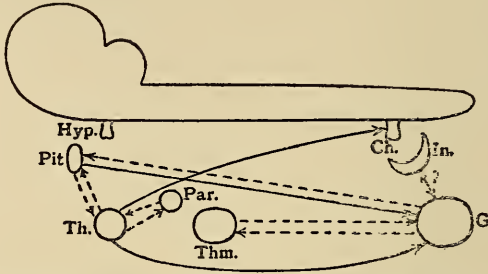


FIG. 60.—To show the probable influence of the various endocrinous structures on one another. The following explanations apply to this and to the three succeeding figures. (Paton.) ———stimulation; - - - - -inhibition. The arrow indicates the direction of action. *Hyp.*, hypophysis; *Par.*, parathyroid; *Ch.*, Chromaffin system; *Art.*, artery; *Pit.*, pituitary; *Thm.*, Thymus; *Th.*, thyroid; *G.*, glands; *B.*, bone; *In.*, inter-renal; *Pan.*, pancreas; *M.*, muscle. (Paton.)

as the phenomena of tetany show. Myasthenic states in general and myasthenia gravis in particular are more directly related to disordered thyroid and thymus activities. Lundborg has shown that the parathyroid function plays some part in the reaction.

The thyroid acts on the pancreas chiefly through its action on the liver sympathetic fibers. Sugar mobilization and release are brought

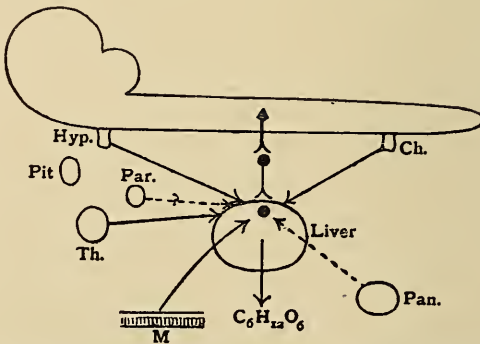


FIG. 61.—To show the probable mode of action of the various internal secretions on the mobilization of sugar in the liver. (Paton.)

about through modified thyroid and pancreatic action, which latter prevents the mobilization of sugar in the liver. Thus glycosuria is frequent in hyperthyroid states. (See Fig. 61.)

The complicated inter-relationships cannot be entered into more

fully. The chief available literature has been indicated. One point, however, should be emphasized and that is that the activities of the internal secretion organs are all under vegetative nervous system control. The active substances, hormones, if one wishes, are not

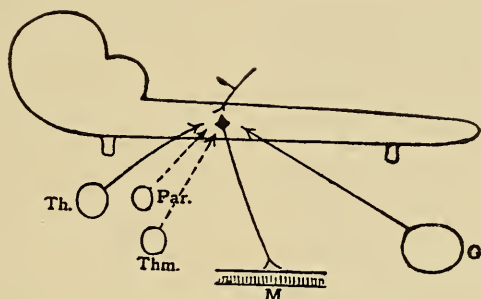


FIG. 62.—To show the probable mode of action of certain of the internal secretions upon the spinal reflex arc. (Paton.)

independent chemical activators, they are under sympathetic and parasympathetic (autonomic) control. The output of iodine, of calcium, of adrenalin, of hypophysin and of all of the substances thus far known or named is controlled almost exclusively by the nervous system. The internal secretions act through the nervous system. While it may be shown that within an organ itself primary

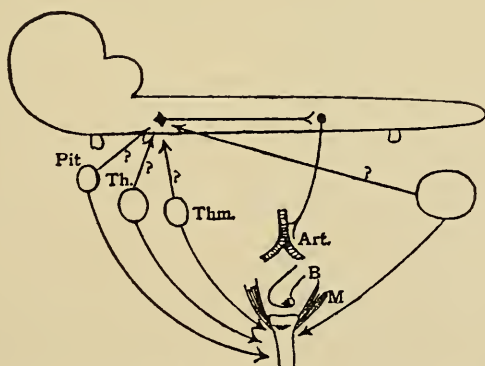


FIG. 63.—To show the probable mode of action of the internal secretions on the growth of muscle and of bone and other connective tissues. The possibility of this being a vasomotor reflex mechanism is indicated by lines marked? (Paton.)

chemical regulators may be effective—thus one must explain the positive and negative tropisms within the cells of an organ in its initial response to a disturbance of cellular adjustment—yet the chief activities of the internal secretions are brought about by neurochemical regulators, as Paton terms them.

Just as the complicated sensori-motor integrations are effective in governing the muscular activities of the human body, so the integration of neurochemical regulators, taking place at the physico-chemical level, is effective in adjusting the metabolism of the body cells. Hormones are not the activators primarily; they are the servants of the vegetative nervous system. All of the endocrinopathies are really polyglandular syndromes and under psychical control.

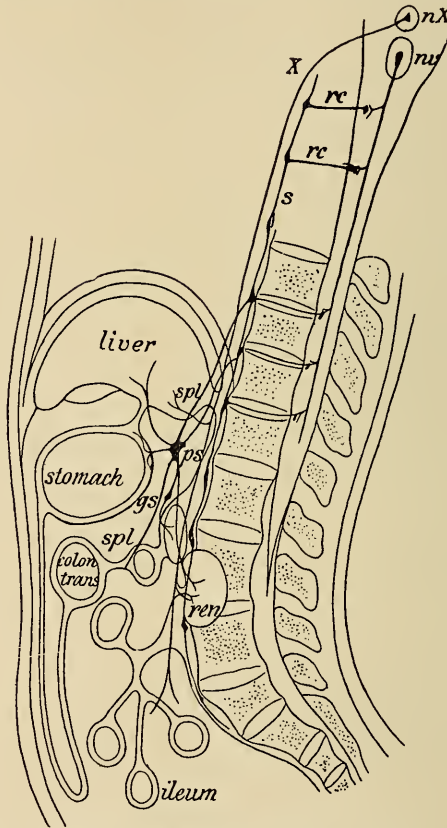


FIG. 64.—Scheme of innervation of the liver, spleen, and kidney. *nX*, nucleus of the vagus; *X*, vagus; *nu*, vasomotor nucleus in medulla; *s*, sympathetic; *rc*, rami communicans; *spl*, splanchnic nerve; *ps*, solar plexus; *gs*, semilunar ganglion; *spl*, spleen. (Bechterew.)

Pancreatic Syndromes.—Falta holds that the chief activity of the pancreas is subserved through an assimilatory hormone, which controls the glycogenesis of the liver and muscles. In mild grades of pancreatic insufficiency disturbances of carbohydrate metabolism appear only when great demands are made upon the glycogenic function of the liver through excessive alimentary carbohydrate intake. In graver disturbances in addition to the modification of anabolism a high

grade of catabolic destruction takes place with a failure to form higher and lower fatty acids (ketonuria).

Pancreatic syndromes occur as a result of gross anatomical disorder, acute pancreatic hemorrhage, and chronic pancreatitis, syphilitic pancreatitis, etc.; all of which are discussed fully in works on internal medicine. Those of interest here, however, are diabetes mellitus, true diabetes, and pancreatic infantilism, all closely related to disorder of the chromaffin tissues of the pancreas—its internal secretory part. Although the pancreas seems primarily a digestive gland it also produces an internal secretion which holds in check the mobilization of sugar, thus acting in a balanced relation with the thyroid and hypophyseal secretions which tend to facilitate the use of sugar as an energizing material by the muscles. This mobilization may be considered to be made effective by the terminals of the vegetative nervous system in the liver cells; just how it is not known. Pancreatic infantilism, shows polyglandular disturbances, through arrested bodily growth and arrested sexual development. Vagotonic symptoms such as excessive diarrhea and flatulent distention are also present.

Liver Syndromes.—Progressive Lenticular Degeneration.—Wilson¹ has described this syndrome, to which he applies the term progressive lenticular degeneration.

The disease is familial, in the sense that frequently more than one member of a family is affected with it, but it is not hereditary; it may also occur sporadically. It occurs in young people, either in an acute or a chronic form. As far as is known, it is progressive and invariably fatal, its duration ranging from six months or a year to as long as four or five years.



FIG. 65.—Progressive lenticular degeneration. (Tilney.)

¹ Progressive Lenticular Degeneration. A Familial Nervous Disease, Associated with Cirrhosis of the Liver. Brain, 1913.

Symptoms.—The clinical symptoms consist of involuntary movements, nearly always a bilateral tremor of both upper and lower extremities, the head and trunk also being sometimes involved. The tremor is usually rhythmical but occasionally irregular, and increasing with volitional movement; there is pronounced spasticity of the limbs and of the face, the latter being usually set in a spastic smile, while in the latter stages contractures of the limbs develop; there is dysphagia and dysarthria, the latter eventually degenerating into complete anarthria; there is also spasmodic laughing and emotionalism. As a result of the extraordinary degree of stiffness of the musculature there is considerable difficulty in maintaining equilibrium. Little or no true paresis or paralysis occurs, however, inasmuch as most ordinary movements can be executed, although, it may be, slowly and feebly. In spite of the great degree of motor weakness and helplessness, in a pure case the abdominal reflexes are present and a double flexor response is obtained. In other words, this affection, where it occurs in an uncomplicated form, is an extra pyramidal motor disease, the importance of which is apparent by reason of the light it sheds on such a process as paralysis agitans.

The chief pathological feature of the disease is bilateral symmetrical degeneration of the putamen and globus pallidus, in particular the former. This degeneration is a sequel to the selective operation of some morbid agent on the cells and fibers of the putamen and lenticular nucleus generally. The caudate nucleus is often somewhat degenerated, but never to the same extent, while other large collections of gray matter in the immediate neighborhood of the lenticular nucleus—*e. g.*, the optic thalamus, which has partially the same blood supply—is not affected at all in a pure case unless it be indirectly, and to a very slight extent. The morbid agent is possibly some form of toxin. A constant, essential, and in all probability primary feature of the pathology of the disease is cirrhosis of the liver, not syphilitic or alcoholic; it is multilobular or mixed in type, always pronounced, but presenting a varying pathological picture of necrosis, fatty degeneration, and regeneration.

It is probable that the toxin is associated with the hepatic cirrhosis, and may be generated in connection therewith. An important analogy may be drawn from the occurrence of "Kernikterus" in certain cases of familial icterus gravis neonatorum, where in spite of the universal bile staining of the tissues of the body certain collections only of gray matter in the brain show a marked avidity for the circulating poison, while others do not. The parts that are stained deeply are in particular the nucleus lenticularis and the corpus Luysii (among others), while the optic thalamus, for instance, is scarcely stained at all.

Muscle Syndromes.—**Myasthenia Gravis.**—The clinical position of this disorder is very uncertain. By some it is to be regarded as a contrast picture to tetany and due to vegetative nervous disturbance conditioned in part by diminished parathyroid activity. It has of

late been shown that the striped muscular system is provided with vegetative fibers which undoubtedly regulate the muscular metabolism. By others it is grouped with the muscular atrophies.

The disorder is infrequent. It was separated from the progressive bulbar palsies of organic nature by Erb (1878) and later studied by Oppenheim (1887), who termed it a myasthenic paralysis without anatomical foundation. In 1891 Jolly described the characteristic electrical reactions occurring in the muscles, termed the myasthenic reaction.¹

The early symptoms which usually came on between fifteen to thirty years of age, usually involve the facial muscles, particularly those of the upper lid, causing ptosis. Diplopia from paresis of an ocular muscle also may be an initial symptom. The two often occur together (Asthenic ophthalmoplegia). The patients note the beginning fatigue of the muscles, which perhaps intact in the morning on awakening, show fatigue signs at night. This muscular asthenia then progresses slowly to distinct paresis. Other cranial nerve innervations then show a similar asthenia. Difficulties in chewing develop, or of swallowing, or of speaking. The muscles of the neck may also be involved. Whatever group is involved the chief feature is the great fatigue which develops very rapidly after the use of the muscle.

Any muscle or muscle group of the body may be involved. Dyspnea and tachycardia are among the rarities of implication of the respiratory and cardiac muscles. Sensory disturbances are not characteristic. Pains may occur.

Leukocytosis is usually present. The reflexes are not implicated. In some instances fatigue of the tendon reflexes is recorded.

The chief feature is the rapidly developing fatigue of the muscle. This is best demonstrated by faradic stimuli. These cause a rapid loss in the excitability of the muscle until it no longer reacts to the intermittent faradic current. Hoffmann has shown that this is largely influenced by the rate of the interruptions. With seventy interruptions per second the myasthenic reaction develops promptly, with fifteen it does not. Continuous faradic stimulation produces a similar myasthenic fatigue curve. This myasthenic reaction seems to separate the disorder from other forms of muscular fatigue such as occur in bulbar palsy, medullary syphilis, multiple sclerosis, Addison's disease, exophthalmic goitre and the fatigue of intermittent claudication. There are certain analogies with this last disorder which are not yet cleared up.

Atrophies develop in the affected muscles, but there are no definite indications of the reaction of degeneration. Certain transitional cases which show relationships to distinct organic (nuclear) cases may evidence electrical changes approaching R. D. Fibrillary twitches in the affected muscles are not the rule, but they have been observed.

¹ Oppenheim, *Die myasthenische Paralyse*, 1901.

Myasthenia gravis runs a chronic course with at times marked remissions. It has been known to develop rapidly in three to four months with fatal issue in from one to three years and on the other hand it has been known to extend over fifteen to twenty years. The outcome is usually fatal, but certain cases cease to progress.

Little is known of the underlying causes. Status thymicolymphaticus is frequent. Many cases are associated with disorder of other endocrinous glands, chiefly with hyperthyroid states. Constitutional anomalies, also often regarded as of lymphogenic origin are described. Nothing is known concerning the psychical states.

The pathological lesions are not constant. In the greater number of cases the muscles are swollen, edematous and infiltrated with lymphoid cells. These changes have not been interpreted. It is possible that they are edemas due to disturbance of the vegetative nervous system control, in which case myasthenia gravis is to be allied with the circumscribed edemas. To know this does not help very much, but it does indicate that search must be directed toward all causes for vegetative nervous system disturbance, toxic and psychic.

The first important therapeutic agent is rest; absolute and prolonged. The second is psychotherapy. Organotherapy has been tried, with as yet little results, but it probably has been entirely too empirically applied. Careful attention should be given to a complete survey of the functions of all of the endocrinous glands, and if a lack of balance be found an attempt should be made to restore the balance. Artificial feeding is at times necessary. Atropin has been of service occasionally. Alcoholic preparations are to be avoided as are also mechanical forms of stimuli, particularly severe massage.

Thomsen's Disease.¹—**Myotonia Congenita.**—This is a very rare disorder. Its relationships to other nervous diseases is very obscure. It is hereditary, and is probably conditioned by a constitutionally inferior thoracic autonomic control of the muscle metabolism.² This is often associated in the affected families with other signs of inferiority; neuroses, psychoses, tetany, etc.

The chief anomaly is one affecting the muscles. At the beginning of any voluntary movement, the patient finds it difficult to overcome a muscle hypertonus. This makes the muscles stiff and unyielding. After repeated efforts the resistance gradually disappears and in a few minutes or more the muscular activity becomes normal. This limbering up effect is lost after a cessation of the movements. Any group of muscles may be affected, but the lower extremities are oftenest involved. This makes the beginning of walking difficult. In the upper extremities a similar condition makes manual movements difficult. A patient cannot readily loosen his grasp of an object. Talking and eating, etc., may be similarly affected. Changing the tempo of a movement increases the difficulty and emotional stimuli

¹ Thomsen, Archiv f. Psychiatrie, 1892.

² S. de Boer, Zeitschrift f. Biologie, 1914, vol. lxx.

invariably augment the stiffness and awkwardness. Mechanical stimuli cause welts to appear which subside slowly. Atypical cases are reported, in some of which the disorder appears intermittently. (Compare with periodic paralysis.)

The pathological changes are slight. Muscle-cell hypertrophy, analogous to that seen in myasthenia gravis, is described.

The disorder begins early, is very chronic, is not fatal itself, nor does it seem to get well spontaneously.

No therapy has been shown to be effective. If the present hypothesis is of value some results should follow from polyglandular therapy.

PART II.
SENSORI-MOTOR SYSTEMS.

CHAPTER V.

SENSORI-MOTOR NEUROLOGY—CRANIAL NERVES.

DISEASES OF THE OLFACTORY TRACT.

Olfactory.—In man the olfactory apparatus has lost much of the importance it possesses in a number of the lower animals. (For the



FIG. 66.—Extent of true olfactory receptors on the mucous membrane. (v. Brunn.)

best recent discussion of the anatomy see Edinger's *Lessons*, 8th edition, 1911-12.) Disease of, or pressure upon, the olfactory nerve

in its peripheral, thalamic, or cortical portions results in either diminution (hyposmia) or loss (anosmia) of smell; hallucinations, illusions (parosmia, ismosmia or cacosmia), or hyperesthesiæ, causing excessive sneezing. Odor influences taste directly and by association involve the entire vegetative nervous system. The different parts of the olfactory pathways need to be taken into consideration. The study of olfactory hallucinations, particularly in certain psychoses, and in certain tumors in or about the frontal lobes, renders this of value.

The receptors for smell are located in a limited portion of the Schneiderian mucous membranes. Changes in that portion of the membrane, such as occur in any acute inflammatory disease, coryza, influenza, diphtheria, etc., cause diminution or loss of ability to smell. Albinism is usually associated with loss of smell. Chronic inflammatory processes, often accompanied by fetid odors, polyps, frontal or maxillary sinusitis, lead poisoning, usually bring about unilateral or bilateral loss of smell. Most of the causes for this mostly peripheral loss of smell may be estimated by direct inspection. Certain directly acting drugs, cocaine, etc., influence smell.

From the receptors, the pathways traverse the cribriform plate and enter the olfactory bulb, forming synapses with the mitral cells. Lesions in and about the cribriform plate, fractures, meningitis, syphilis, pressure of frontal tumor may determine a diminution or loss of smell; possibly lesions in this portion of the olfactory pathway may cause hallucinatory odors, but this is still debatable. Certain tumors lying upon the orbital plate of the sphenoid and compressing the lobus olfactorius have seemed to give rise to unilateral and bilateral hallucinations of smell. (See Fig. 67.)

It is known that certain sneezing crises have been determined by tabetic lesions. Just which parts of the olfactory tracts are involved is not certain.¹

Tertiary neurones pass to the thalamus and to the cortex. (See Fig. 68.)

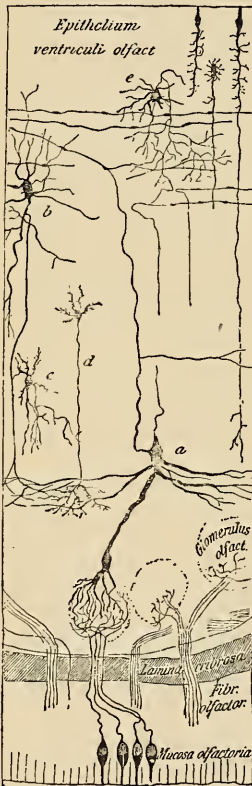


FIG. 67.—Illustration of first and second neurones of the olfactorius. The first synapse takes place in the glomeruli (mitral cells). (Elinger.)

¹ Klippel and L'Hermitte. Sem. Méd., February 17, 1909.

Clinical correlations with disorder of this portion of the olfactory pathway are not certain. Certain overaffective reactions to odors, disgusto, nausea, even vomitings from odors need to be more carefully sifted in this connection, especially in relation to brain tumor localizations. They should not be viewed as whims or fancies of hysterical patients. Loss of smell may result from thalamic lesions, usually homolateral. The crossing of the olfactory pathways is incomplete, and takes place principally in the anterior cerebral commissure (Fig. 68).

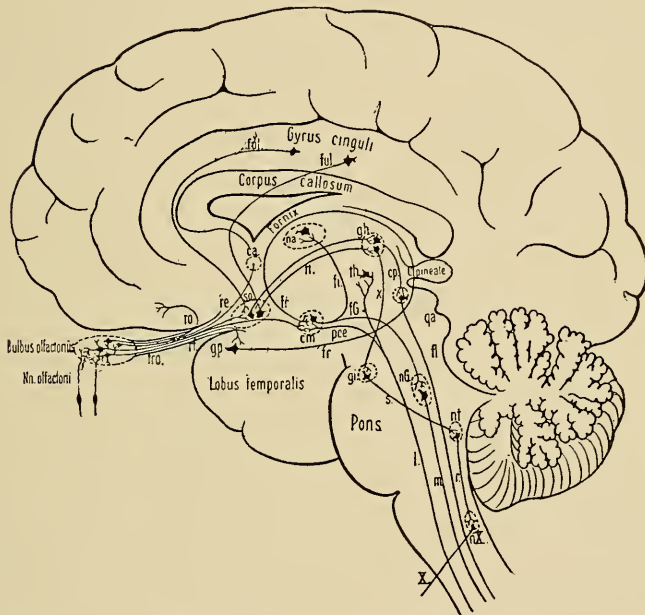


FIG. 68.—Scheme of olfactory paths. *X*, vagus root fibers; *ca*, anterior commissure; *cm*, mammillary body; *cp*, fibers from nucleus habenulæ to posterior commissure; *fG*, tract from mammillary body to Gudden's nucleus; *fi*, fasciculus mamillio-thalamicus; *fl*, fasciculus long, medianus; *fr*, fornix; *ful*, fibers of fornix; *gli*, nucleus habenulæ; *gi*, interpeduncular ganglion; *gp*, gyrus pyriformis; *l*, median lemniscus; *m*, fibers from Gudden's nucleus to substantia reticularis; *na*, anterior thalamic nucleus; *nG*, Gudden's nucleus; *nt*, tegmental nucleus; *nX*, vagus motor nucleus; *peE*, ped. corp. mammilaris from fillet; *qa*, quadrigemina; *r*, fibers from n-tegmenti to cranial nerve nuclei; *re*, radix lateralis tractus olfactorii; *rf*, fibers of olfactory tract to trigonum olfactorii; *ro*, median olfactory tract root; *s*, fibers from interpeduncular ganglion to tegmental nuclei; *so*, olfactory trigone; *th*, optic thalamus; *tro*, olfactory tract; *tt*, tenia thalami; *x*, fasciculus retroflexus. (Bechterew.)

The cortical neurones end in the cornu ammonis, which is a large olfactory association field. (See Edinger, Ramón y Cajal, Van Gehuchten.) Lesions here result in peculiar olfactory auras, as seen in certain hippocampal epilepsies (uncinate fits of Hughlings Jackson). Such fits occur from temporosphenoidal tumors also. Olfactory agnosias also result from lesions in this general region. Some of congenital origin, with agenesis of the cornu ammonis have been described.

Anosmias or olfactory agnosias are frequent in general paresis, and in abscess and tumor.

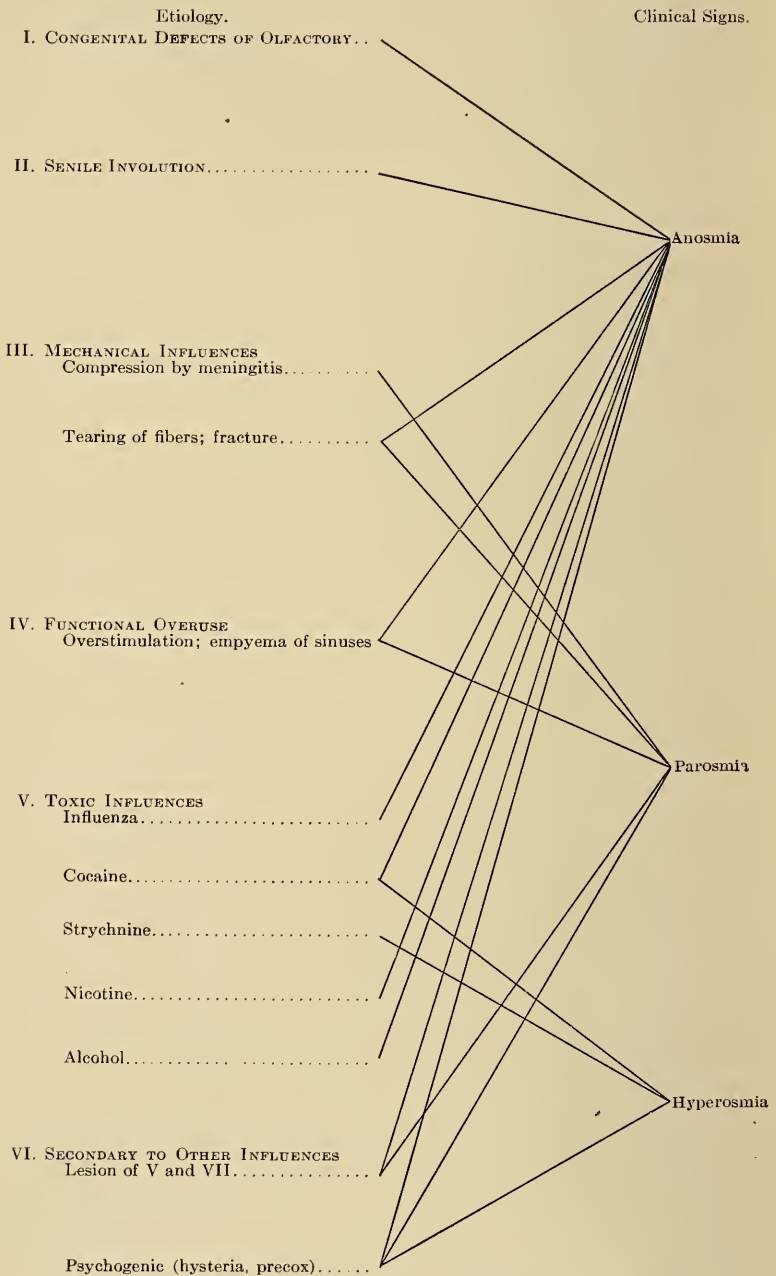


FIG. 69.—General summary of olfactory disturbances. (Veraguth.)

The efferent pathways and synaptic reflex paths of the olfactory are many. The most common motor reflex is that of sniffing, with dilatation of the nostrils. This is occasionally seen as the result of a central lesion (dementia precox, paresis, compulsion neurosis). The relation between odors and the vomiting reflex is to be borne in mind in hysterical vomiting, furthermore the very primitive associations between odor and sexual complexes. Hysterical anosmia may be closely associated with general hysterical hemianesthesia, which latter, however, is more often due to a thalamic lesion than to an hysteria, or it may be an isolated phenomenon and is usually classical of the mechanism of conversion in a narrow Freudian sense. The psychology of smell and its complicated relationships to infantile phantasies of disgust, to childbirth from intestinal canal, to the identification of feces and money, make the study of odor phenomena of great import in the psychoneuroses and psychoses. In certain psychogenic epileptics the olfactory symbolisms are highly developed. What relations these have, either as cause or result, to the cornu ammonis lesions found in these epileptics (Alzheimer) has not yet been determined.^{1 2}

Treatment.—The underlying cause of the changed olfactory state needs treatment, not the state. Local applications of cocaine, morphine, etc., are usually unjustified.

DISEASES OF THE OPTIC PATHWAYS.

The course of the light pathways and its synapses is extremely complex, since sight has become one of, if not the principal tool of advance in the evolution of man's mental powers. The symptomatology by implication of the pathways is likewise rich and varied.

The retina, the mostly decussating optic nerve, ending in the external geniculate, the pulvinar of the thalamus and the anterior corpora quadrigemina, and finally the optic radiations ending in the calcarine region of the cortex make up the primary, secondary and tertiary incoming neurones of this pathway. (See Fig. 86.)

The intricate symptomatology is dependent upon, and will be discussed in accordance with, these anatomical divisions. The chief signs to be considered are night blindness, color blindness, dimness of vision, blindness in one or both eyes, temporary or complete, scotomata, temporary or permanent hemianopsia, hemichromatopsia, hemiopia, mind blindness or optical agnosia, photophobia, hallucinations and illusions of sight, hysterical blindness, malingering of blindness, unilateral or bilateral.

Retinitis.—Involvement of the first neurone is termed a retinitis. It may result from the extension of an inflammation or may be due

¹ Bailey, P., Flaubert's Epilepsy, Proceedings of Charaka Society, New York, vol. iii.

² Clark, L. P., The Epilepsy of Dostoiewsky, Medical Record, New York, 1915.

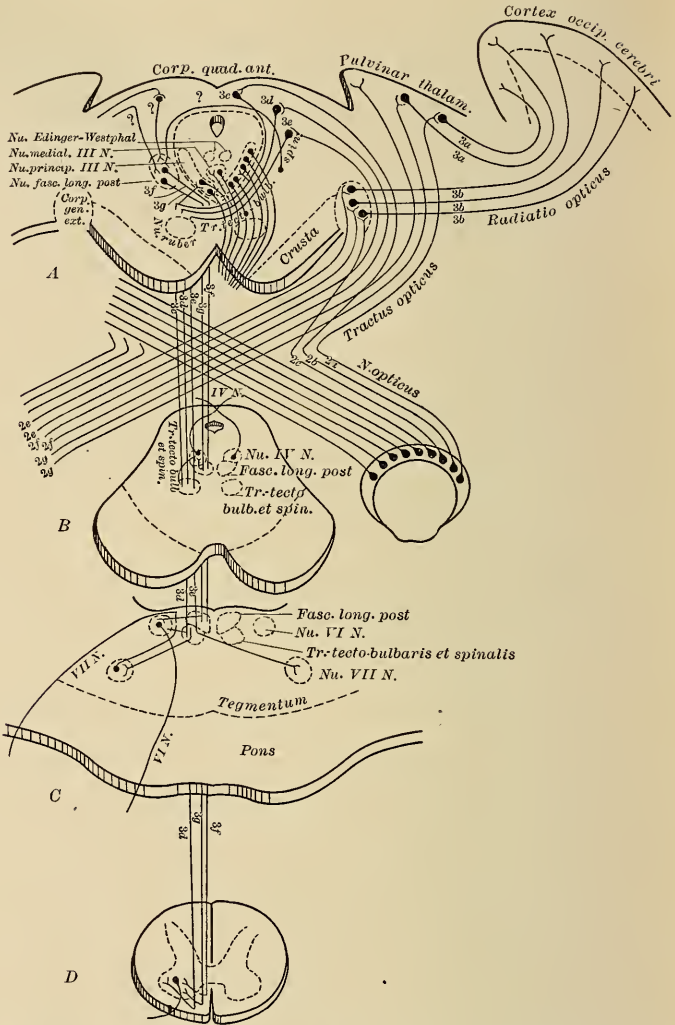


FIG. 70.—Diagrammatic scheme of optic paths and chief connections at four levels. A, level of II and III nerves; B, level of IV nerve; C, level of VI and VII nerves, tegmentum of pons; D, spinal cord. *Neurone 1.* Receptors in rods and cones of retina are not indicated in the diagram. *Neurone 2.* 2a, axones passing to pulvinar of same side; 2b, axones passing to corpus quadrigeminum same side; 2c, axones passing to external geniculate of same side, all from temporal side of retina; from nasal side 2a, axones crossing in chiasm going to opposite external geniculate; 2f, axones crossing in chiasm to go to opposite anterior corpus quadrigeminum; 2g, axones crossing in chiasm to opposite pulvinar. Papillomacular bundle fibers partly crossed, partly uncrossed (see Fig. 75). *Neurone 3.* Pulvinar axones to occipital cortex; 3b, external geniculate axones to occipital lobes; 3e, d, e, corpora quadrigemina fibers, middle layer decussating (Meynert) to median longitudinal fasciculus and forming tractus tecto-bulbaris et spinalis to go to medulla and anterior columns, forming synapses with third, fourth, sixth, and seventh nerves and motor nuclei of spinal nerves (space orientation); 3f, g, fibers from interstitial nucleus (Cajal) of fasciculus longitudinalis posticus forming part of longitudinal fasciculus, passing to anterior columns, forming synapses with III, IV, VI cranial nerves and motor spinal nerves. *Neurone 4.* Axones from oculo-motor, facial, and spinal nuclei. (Strong.)

to toxic or to hidden constitutional factors. The chief indications are ocular discomfort or photophobia, diminution of the visual acuity, appearance of scotomata, general contraction of the visual fields, micropsia, megalopsia or metamorphopsia.

Different grades of retinitis are distinguished ophthalmoscopically. The chief types are simple, albuminuric, syphilitic, diabetic, hemorrhagic and anemic retinitis.

In *simple retinitis* there is clouding particularly of the superficial layers, in patches or in larger portions at the posterior pole. The veins are dull and dark and full, and seem imbedded in the swollen or hazy retina. Sight is dim and worse in spots (scotomata). The disorder usually involves first one eye and then the other.

Albuminuric retinitis is frequent in Brights (25-40 per cent.). Headache and loss of vision in a middle aged to older person are the usual signs. There are characteristic changes in the retina and albumin and casts in the urine. Cirrhotic kidney is the most frequent lesion. The chief change is an arteriosclerosis of the retinal vessels. They are unduly tortuous and show contractions and widenings, often being beaded. There is also a translucency in the retina, white stripes accompany the vessels. The veins are likewise tortuous, and disturbances of circulation show particularly at venous-arterial crossings. Retinal edema with grayish opacity shows. Hemorrhages are frequent. The margins of the disk become obscured, the nerve expanding into the retina without sharp lines of demarcation. The disk may be much swollen, wooley in appearance, and much extravasated. Fatty degenerations with "snow bank" appearances occur.

Blindness, scotomata, dimness of vision appear as in simple neuritis, but chronic cases of albuminuric retinitis may be present with little loss of visual acuity in the early stages. Permanent impairment of vision is the rule. Albuminuric retinitis accompanied by hemorrhages, and fatty degeneration of the retina, in a patient over 35 to 40 usually portends a fatal issue within comparatively few years.

Syphilitic retinitis is probably much more frequent than has been supposed. It may result from hereditary or acquired syphilis, in the latter case appearing soon after infection. Clinically there is contraction of visual fields, dimness of vision, may be night blindness, or marked dimness of vision with poor illumination. Shimmering lights which are persistent and annoying occur, with micropsia and at times metamorphopsia. Central, partial or complete scotomata are fairly constant.

The ophthalmoscope shows hyperemia with serous exudation much resembling the albuminuric variety but in milder degree. Hemorrhages are much rarer, and the "snow bank" glistenings much less pronounced. Opacity about the disk is a variant feature, with inflammation of the uveal tract.

Hemorrhagic retinitis is of import, but must be referred to works on ophthalmology with the other types.

Retinitis pigmentosa is an hereditary variety of primary retinal degeneration showing night blindness with striking frequency. Nettleship's famous study of a French family showed this to be a striking instance of Mendelian dominance. (Plate VI.)

Optic Nerve.—The disorders affecting the second optic neurons fall into two groups: those affecting (a) the optic nerve, (b) its terminations in the midbrain structures.

(a) *Diseases of the Optic Nerve.*—Here three situations need to be distinguished: (1) whether the affection lies anterior to the chiasm, (2) whether it involves the chiasm, or (3) lies behind the chiasm in the path of the second optic neurone.

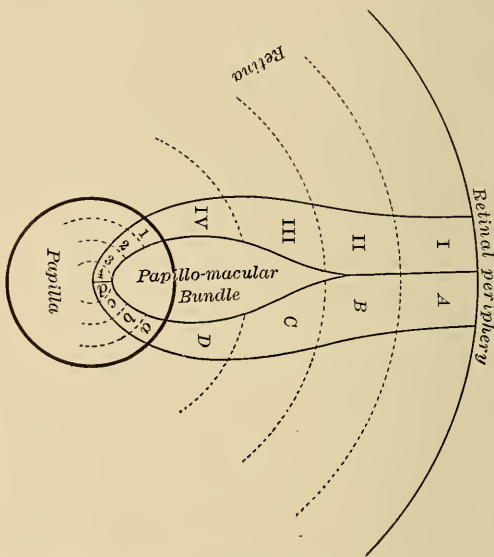
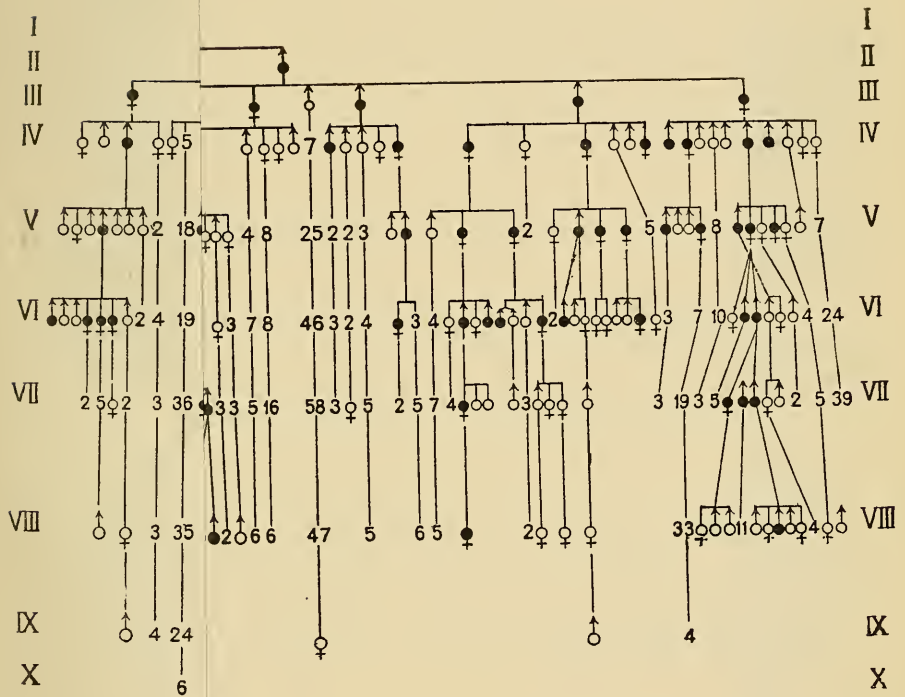


FIG. 71.—Scheme of papillomacular bundle. (Wilbrand and Sanger.)

(1) Diseases of the Optic Nerve before reaching the chiasm: *Optic Neuritis*, in general sense. Three types are distinguishable with pronounced symptomatology. They are: (a) Axial Neuritis, (b) Interstitial Peripheral Neuritis, and (c) Diffuse Neuritis.

A. Axial Neuritis.—This is a system-disease of the papillomacular bundle, involving the nerve in front of the chiasm. It may be acute or chronic.

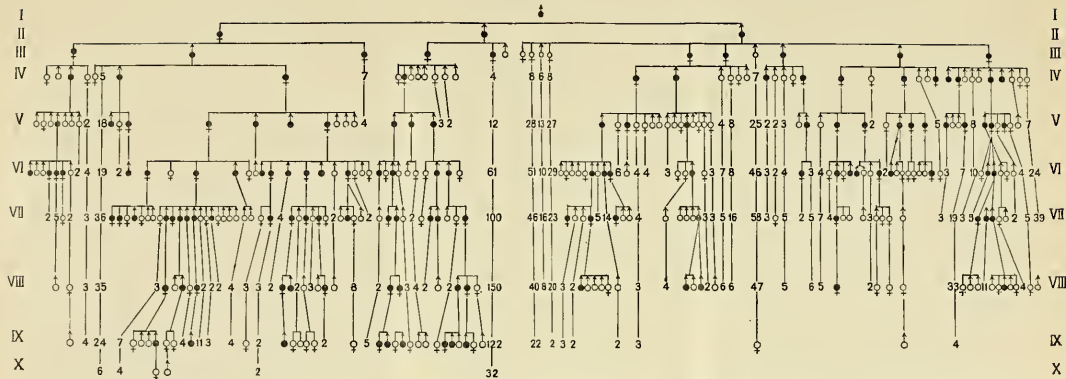
Acute Axial Neuritis.—Occurs usually in young adults, 12 to 24, more particularly women. There appears sudden clouding or dimness of vision, and occasional photopsias. A frontal or temporal headache, or deep pain in the orbit, made worse by pressure or movement of the eyeballs is present. The loss of sight is rapid, reaching a maximum usually in five days, and often is so severe that the patient can



Descent of a Factor (DR) in a Family (Based on Cunier's Records with Later Additions.) Detail. The Affected Man (DR) Who Married the Affected Woman in Generation VII.

Black symbols show descent to a dominant factor. According to the records there is a great excess of normals, the table gives a remarkable illustration of the permanence and mode of descent.

PLATE VI



Descent of a Form of Stationary Night Blindness. (Condensed from the Chart Published by Nettleship, Based on Cunier's Records with Later Additions.) Only those Families Which Contain Affected Members are Here Set Out in Detail. The Affected Man (DR) Who Married the Affected Woman (DR) is Represented by the Sixteenth Black Symbol from the Left in Generation VII.

Black symbols show the night-blind individuals. The descent is always through the affected, showing that the condition is due to a dominant factor. According to the records there is a great excess of normals over the affected. It cannot be said that the responsible factor is a simple allelomorph. Nevertheless, the table gives a remarkable illustration of the permanence and mode of descent of a dominant variety. (Bateson.)

just count fingers at 15 feet, or is blind. With the loss of sight the headache lets up. There may be no retinal changes. The pupil of the affected eye is larger, and is usually sluggish to direct light stimulation, but shows no consensual light reflex loss.

After the acute stage is over there is gradual recovery of the sight in the periphery with various degrees of persistent central scotomata. The loss may be unilateral or bilateral and absolute, or unilateral or bilateral to color only, or various gradations of these paracentral scotomata, etc. The scotomata gradually diminish and after six to eight weeks, with proper therapy, may entirely disappear (Fig. 72).

The fundus picture may remain normal throughout or show a papillitis. This will depend upon how far back of the optic disk the lesion, which is usually a vascular one, occurred. When there is a papillitis it shows slight paling of the temporal half or halves of the fundi (Fig. 72).

Etiology.—The most frequently ascribed cause is exposure to cold. This is probably only an incident to other real causes such as infec-

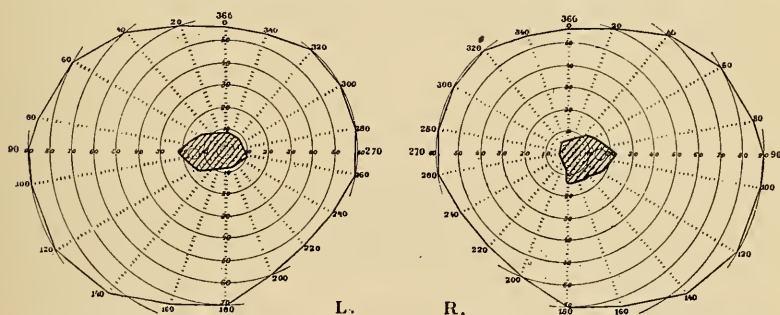


FIG. 72.—Central scotomata in acute axial neuritis. (Wilbrand and Sanger.)

tious disease—syphilis, tuberculosis, typhoid, erysipelas, sinusitis, influenza, mumps, pneumonia, tonsillitis, cerebrospinal meningitis, malaria, beri beri, etc., or toxemias, such as those of pregnancy, nephritis, burns, CO poisoning, methyl alcohol, quinine, flix mas, morphine, etc.

Treatment.—Hot baths, and treatment of cause, as antisyphilitic in syphilis, etc.

The *Chronic Form* is much more frequent. It is the classical situation in chronic alcohol or nicotine poisoning, and affects males more often. Here the course is a chronic one, spreading over several months or years. The blindness appears slowly, and at first consists of a central scotoma for colors, or of a hemeralopia, the patient seeing better in the dusk than in the bright light (fatigue). The scotomata become more marked if the poisoning continues (Fig. 72).

The type of scotoma varies widely. Bilateral, fairly symmetrical, oval scotomata for red and green, lying between the blind spot and the fixation point is the early picture. It usually starts as a defect

for red, stretching toward the blind spot (Fig. 73). The chief defect usually lies about 2° to 8° from the fixation point. Absolute central scotomata are rare.

Vision is usually diminished, and more on one side than the other. In monocular reading the type to the right of the fixation point is not clear for the right eye, while for the left eye the defect lies to the

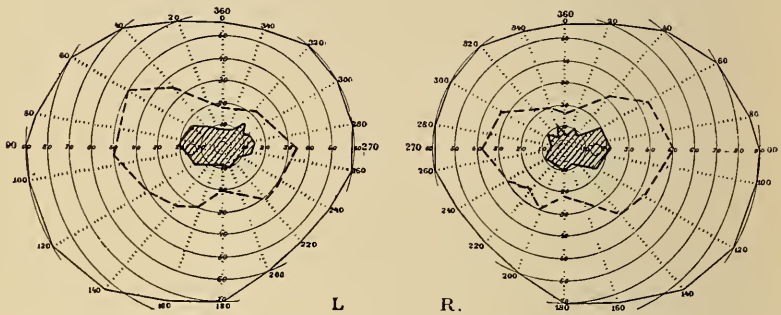


FIG. 73.—Scotoma for red and green in tobacco axial neuritis. (Wilbrand and Sanger.)

left. The defect in vision bears little direct relation to the size of the scotomata. Pupillary anomalies, diminution of both light and accommodation reflexes and pseudotabetic pictures are to be found.

The fundus picture may be normal with gross defect in vision and large scotomata, or there may be hyperemia, a mild neuritis, with some temporal pallor. If marked pallor is present it speaks in general for a more severe process.

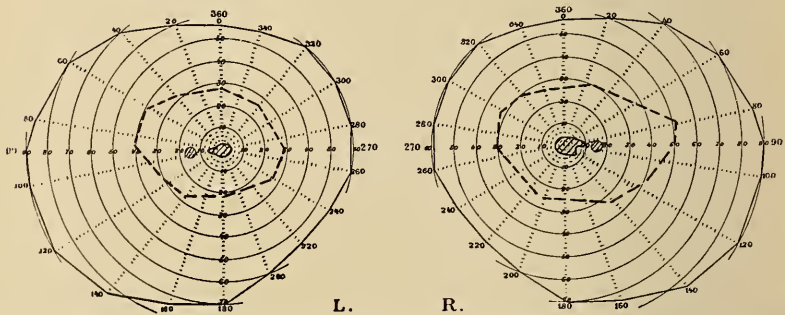


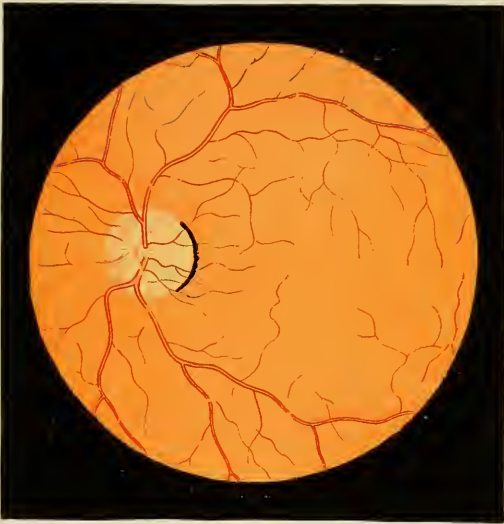
FIG. 74.—Beginning scotoma for red at the onset of a tobacco or alcoholic axial neuritis. (Wilbrand and Sanger.)

The course is chronic. Removal of the cause may save the sight, but slight scotomata may persist. Atrophy is the end in the severe cases.

Pathology.—The changes in both acute and chronic types occur in the papillomacular bundle. They are degenerative rather than inflammatory changes, with greatest severity in the optic canal, because of the richer vascular supply of the nerve trunk at this point.

PLATE VII

Fig. 1



Normal Eye-ground (average tint). (Norris and Oliver.)

Fig. 2



Ophthalmoscopic Appearances in Early Stage of Optic Neuritis.
(Norris and Oliver.)

Etiology.—The chief causes are alcohol and tobacco. Other less frequent poisonings are by carbon bisulphide, in the rubber industries, arsenic, lead, sulphuric acid, anemia (anchoylostoma), diabetes, aniline, gasoline.

Hereditary Forms.—These occupy intermediary positions. One type affects usually the males, passing through unaffected females (Knight's move in heredity). These develop acute or subacute headaches about the age of 20, with gradually diminishing vision. Unequal bilateral scotomata appear and gradually increasing central blindness. There is usually a subacute and progressive stage for about six months, one eye lagging behind the other in its deterioration.

Gradually increasing central scotomata show. Nyctalopia is frequent. The marginal vision is retained, save for colors. Total blindness develops in about two-thirds of the cases. One-third remain more or less stationary or even improve. In mild cases central color scotomata alone appear, or there is diffuse loss of color sense without scotomata.

The fundus picture is variable, but the presence of atrophic changes, usually temporal, stands in marked contrast with any other changes in the nervous system.

Hereditary syphilis plays a role in some cases. In others there is an anomaly in the germ plasm.

B. Interstitial Peripheral Neuritis.

—Here the disease involves the periphery of the optic nerve rather than the central or eccentrically lying papillomacular bundle. Concentric limitation of the field of vision, for white and colors, is the chief finding rather than central scotomata. Here there is a peripheral inflammation of the nerve trunk, starting in the pia and proceeding inward in the septa.

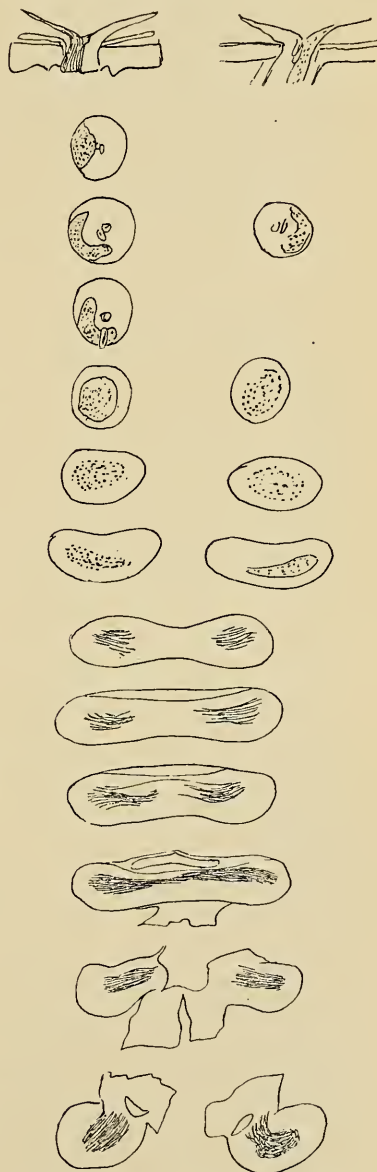


FIG. 75.—Course of the papillomacular bundle at different points in the optic tract. (Wilbrand and Sanger.)

The concentric limitation of vision is rarely observed in the beginning. As it slowly advances the patients become uncertain of space localization and need to turn the eyes frequently to get clear pictures of where they are going. Central vision is usually sharp even for color.

The fundi show simple or neuritic atrophy, occasionally choked disks. Very variable fields are observed (Figs. 75, 76, 77, 78).

Hysterical limitation of the field is to be considered here. Normal fundi are consistent with a true interstitial neuritis, since lesions lying far back in the nerve trunk may cause little or no disk changes. A psychoanalytic anamnesis usually will clear up a possible hysteria. Tabes with neuritis may begin as an interstitial neuritis. The cytobiological findings will clear up the diagnosis.

Etiology.—Syphilitic meningitis of the base is the most frequent cause. A negative Wassermann is not a just criterion to deny specific medication. Other etiological factors are measles, diphtheria, influ-

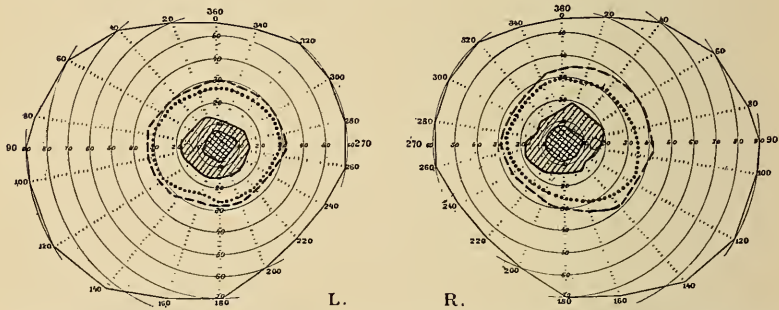


FIG. 76.—Visual fields in a patient with hereditary axial neuritis. Fields for white normal, for blue ----, and for red . . . , concentrically contracted. Absolute central scotoma with larger bordering scotoma for blue and red. (Wilbrand and Sanger.)

enza, myelitis, gonorrhoea, sinusitis, typhoid, lead, diabetes, leptomeningitis, cerebrospinal and tuberculous meningitis.

The *therapy* is causal, usually specific. Salvarsan is less to be feared than an active syphilis. Most so-called neurorecidives causing blindness are due more to the syphilis than to the arsenic.

C. Diffuse Neuritis.—Here the inflammatory process involves the entire nerve stem resulting in marked amblyopia, or blindness. It may be implicated locally or throughout its entire course from the retina to the chiasm. In acute myelitis this severe inflammation at times occurs. Infectious diseases are important. Influenza here plays almost as large a role as syphilis.

The infections and toxemias mentioned in the preceding paragraphs may also induce a total optic neuritis. Malaria, scarlet fever, yellow fever, erysipelas may be added to the causes. Orbital sinus disease is important, and also multiple sclerosis (Fig. 81).

Other atrophic states, double, one sided, total or partial, occur,

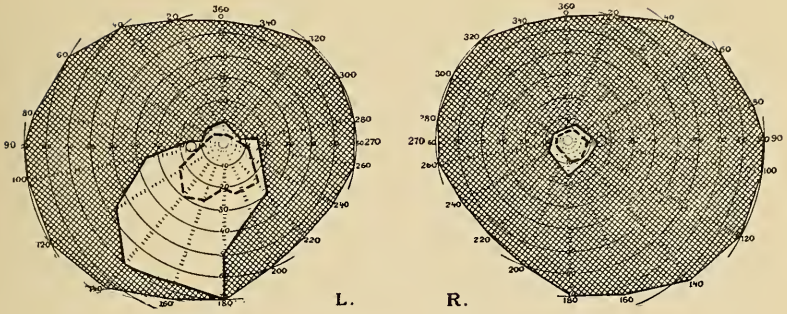


FIG. 77

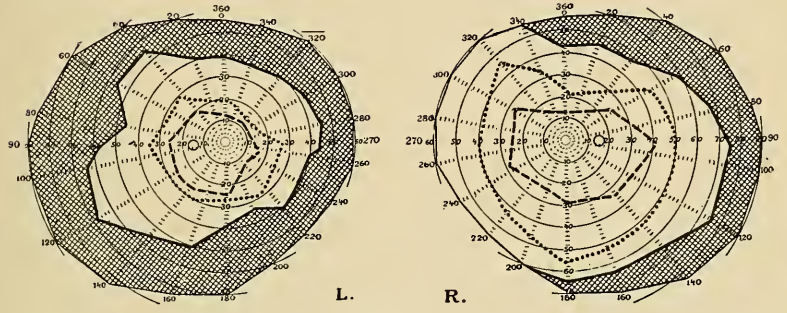


FIG. 78

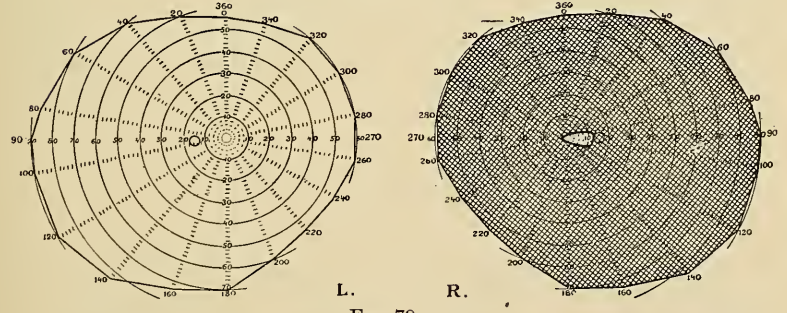


FIG. 79

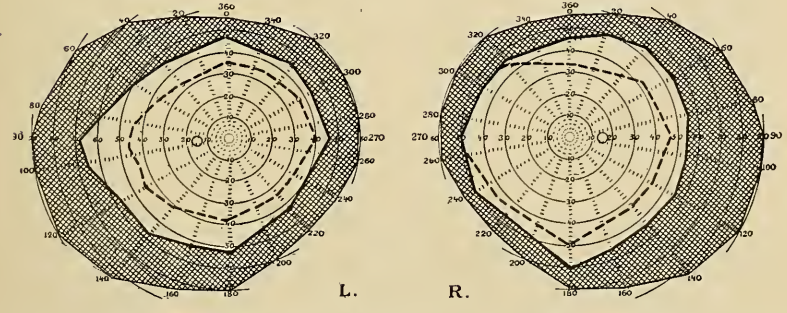


FIG. 80

FIGS. 77, 78, 79, and 80.—Visual fields in cases of interstitial peripheral optic neuritis in cases of syphilis. (Wilbrand and Sanger.)

either in the papillæ, from pressure of a glaucoma, or may be descending atrophies from higher lying causes such as brain tumor, hydrocephalus. Primary progressive atrophy, arising by itself, probably does not

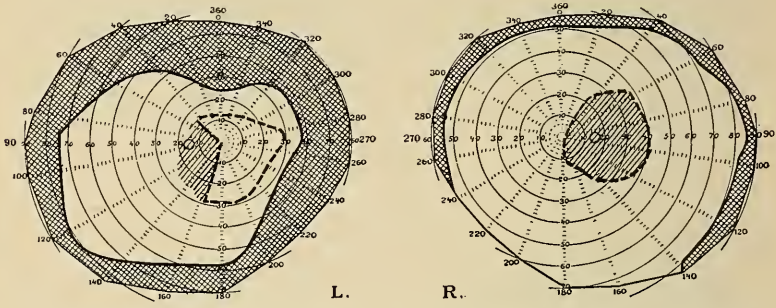


FIG. 81.—Visual field with diffuse neuritis due to multiple sclerosis. On right side a temporal hemianopic color scotoma, on the left intermittently appearing hemianopic uncertainty scotoma. (Wilbrand and Sanger.)

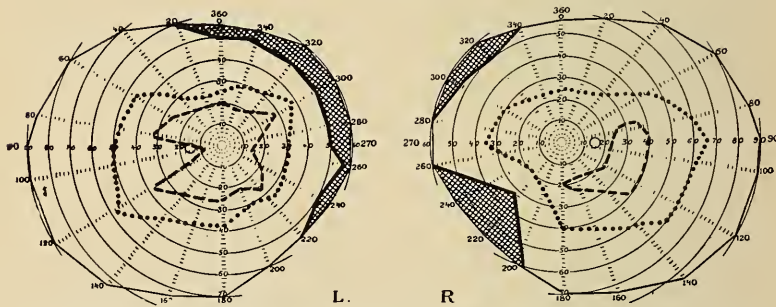


FIG. 82.—Visual field showing atrophy in tabes. Field for blue, for red - - - - . (Wilbrand and Sanger.)

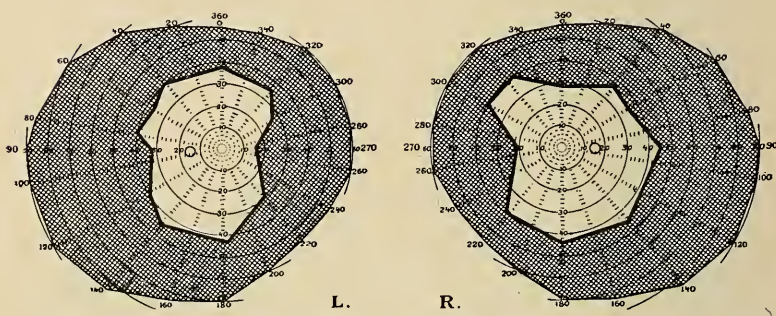


FIG. 83.—Optic atrophy in tabes with loss of color discrimination. (Wilbrand and Sanger.)

exist. The most suggestive cause of an isolated, bilateral, progressive optic atrophy without other tangible neurological signs is tabes. Cytobiological tests will complete the diagnosis (Figs. 82 and 83).

Disease at or about the Chiasm.—The anatomical peculiarities, due to the crossing of the fibers at the chiasm, introduces certain definite signs which are of value. Scotomata and concentric limitation are replaced by hemianopsias of varying type.

In lesions in front of the chiasm bitemporal hemianopsia will be present. This is rare. A lesion behind the chiasm, usually in the sella turcica, and not infrequent, as in pituitary disease, causes a binasal hemianopsia, partial or complete. Lesions to the right or left of the chiasm will cause incomplete homonymous hemianopsias—whereas lesions in the tract back of the chiasm—*i. e.*, in the midbrain or optic radiations or occipital lobes will cause a usually more complete homonymous hemianopsia.

Horizontal hemianopsias, either superior or inferior, occur in chiasm lesions from pressure above or below. They are readily

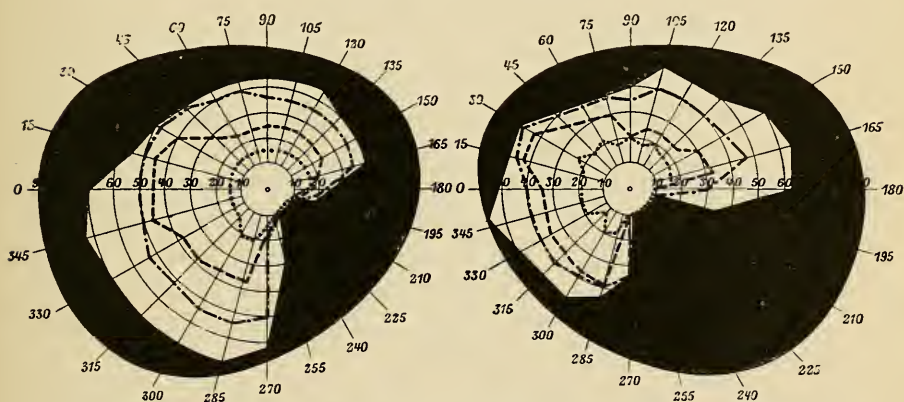


FIG. 84.—Quadrant hemianopsia of lower right segment due to hemorrhagic destruction of the external geniculate.

explained from the position of the crossing fibers in the chiasm. Such hemianopsias may rarely occur from retinal causes.

A common cause for chiasm changes is hypophyseal tumor. Syphilis, however, is specially frequent in just this situation, most basal syphilitic meningitic processes beginning here. The process spreading forward to the optic stem produces a multiplicity of field changes. Thus one may have partial scotomata, monocular temporal hemianopsia, bitemporal hemianopsia (the most frequent), temporal hemianopsia with blindness of one eye, blindness in one eye and nasal hemianopsia of the other, blindness in both eyes. This very great irregularity and changeability, advancing or receding under treatment, is of much importance in excluding a hypophyseal tumor. A loss of the hemianopic pupillary reaction is of importance in making a definite localizing diagnosis.

The pupillary changes are variable. Other signs of basal syphilitic meningitis are discussed in the chapter on Cerebral Syphilis (*q. v.*).

Affections of the chiasm are more rarely encountered as a result of trauma, brain tumor with general pressure, cavernous sinus disease, cerebrospinal and tuberculous meningitis, bone disease, aneurism and arteriosclerosis of the carotids.

Thalamus Disease.—In lesions of the optic tract posterior to the pulvinar, *i. e.*, in the third optic neurone, pupillary disturbances are absent. Thus Wernicke has shown that by careful illumination of the blind side of the eye one can distinguish between an hemianopsia in the second optic neurone (by loss of pupillary light reflex) and a hemianopsia of the third optic neurone (intact hemiopic pupillary reflex). As a matter of fact this test is extremely difficult to perform, but fortunately lesions in the midbrain—end station of second optic

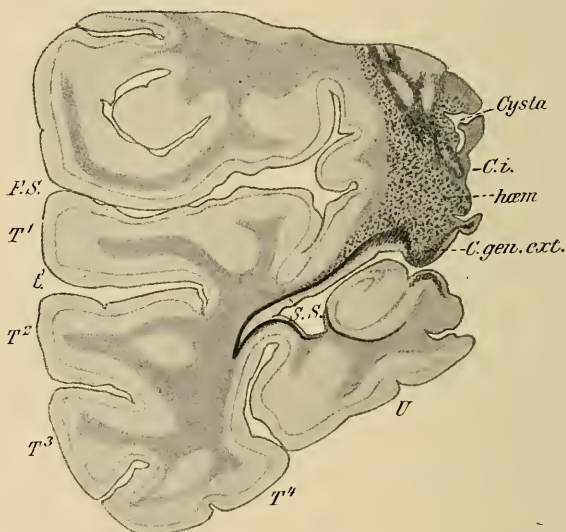


FIG. 85.—Site of lesion in external geniculate giving rise to quadrant hemianopsia seen in Fig. 84 and the secondary degenerations in Fig. 86. *C. gen. ext.*, external geniculate; *hæm*, hemorrhage; *C.i.*, internal capsule; *F.S.*, fissure of Sylvius; *S.S.*, optic radiations.

neurone—are almost invariably accompanied by other sensory signs (usually a complete or incomplete thalamic syndrome (*q. v.*), thus aiding in the diagnosis.

The distributions of the second optic neurone are multiform. Some fibers end in the corpora quadrigemina. Their implication causes pupillary changes; others end in the thalamus (pulvinar), and their involvement causes no disturbances of vision. The majority of the fibers end in the external geniculate bodies. These are in close relation to the auditory tract, the sensory tracts, the oculomotor nuclei and pyramidal tracts. Hence lesions of the optic tract in the geniculate region cause not only homonymous hemianopsias, often

only quadrant, but they are also liable to be complicated by the involvement of these other near lying structures. Isolated, quadrant hemianopsias may mean small hemorrhages, thrombi, emboli, tumors, or encephalitis in the external geniculate (poliomyelitis—rare) as well as from lesions farther back in the tractus (Figs. 84, 85, and 86).

Cortex Disease.—Lesions of the end stations of the optic tract or its associated areas in the occipital lobe may cause mind blindness, *i. e.*, optic agnosia. Here the patient may have no disturbance of sight, or he may have partial hemianopsias, but is unable to recognize words or objects previously known, speech being intact.

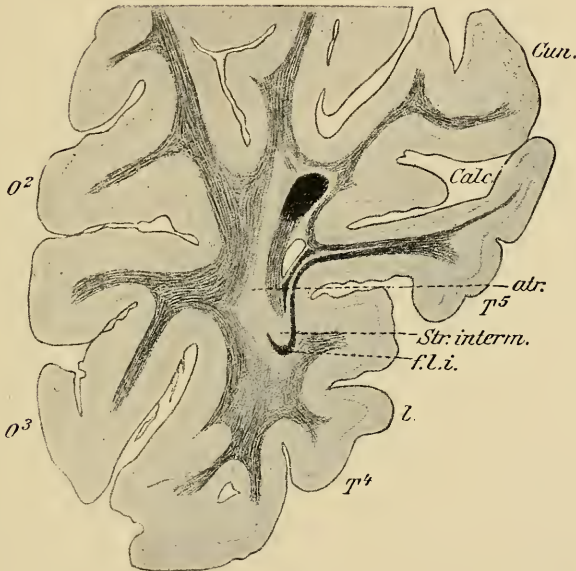


FIG. 86.—Showing atrophic degenerations in optic radiations (*atr.*) from hemorrhage in external geniculate (Fig. 85), giving rise to quadrant hemianopsia of Fig. 84. *Cun.*, cuneus; *Calc.*, calcarine fissure; *f.l.i.*, inferior longitudinal fasciculus; *l.*, left hemisphere; *atr.*, atrophy. (Henschen.)

Optic hallucinations are present in disorder of the optical end stations in the occipital lobe. When they show definite projections in space one can make an accurate localization of the portion of the lobe involved. This may be of great value in determining the site of a tumor or abscess formation.

The chief arterial supply of the posterior neurones is drawn from the calcarine branch of the posterior cerebral. The most occipital portion is supplied by the median cerebral. The anterior cerebral sends branches which innervate the optic radiations just posterior to the corpus callosum, but lesions of this artery at this place cause no definitely recognizable lesions.

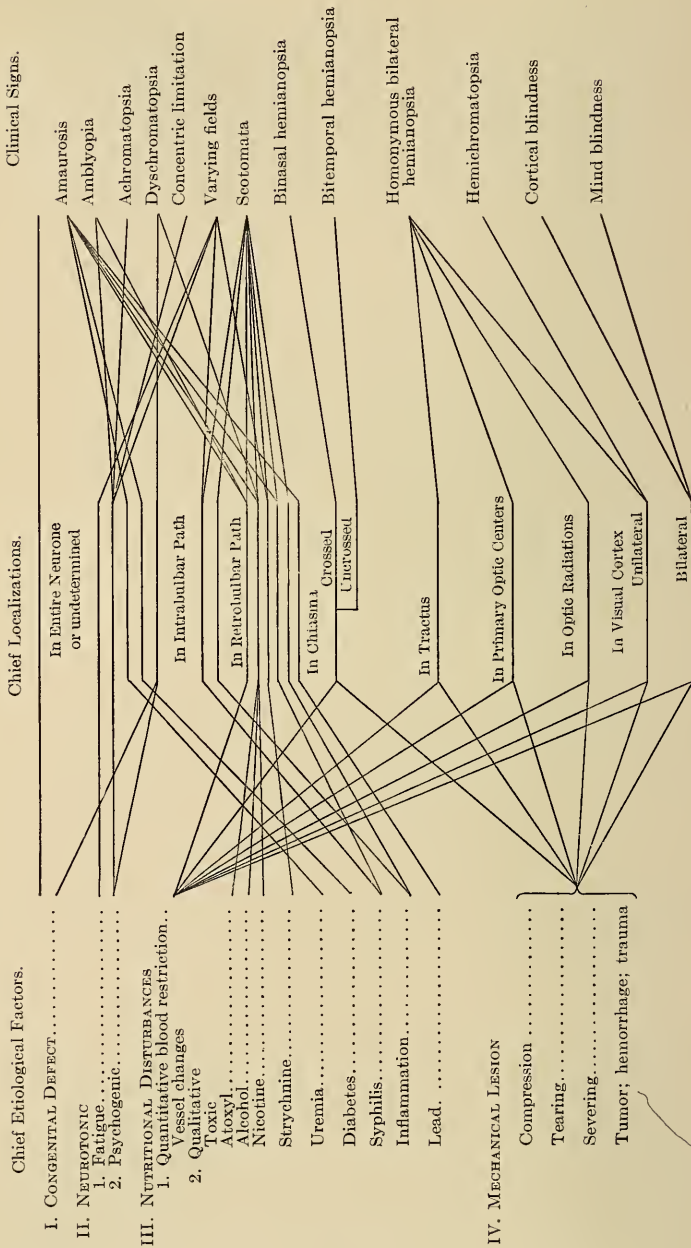


FIG. 87.—Summary of optical disturbances. (Veraguth.)

The cortical disturbances of vision from lesions of the temporal or occipital lobes cannot be entered into more fully here. See chapters on Aphasia, Syphilis of the Brain (Paresis), Brain Tumor, Hemiplegia, Thrombosis, Arteriosclerosis, etc.¹

DISEASES OF THE OCULOMOTOR NERVES.

Ocular Nerves: Third, Fourth, Sixth.—Disorders of the functions of these nerves are best discussed under a general head, since the usual ocular palsies are often complex syndromes in which one or more of these nerves are involved.

The third nerve is a motor nerve for all of the muscles of the eyeball, save the external rectus, and the superior oblique, which latter receive their motor fibers from the sixth and fourth nerves respectively. The third nerve also supplies the levator palpebræ, the ciliary muscle and the contracting fibers of the pupil. The dilating fibers of the pupil receive a branch from the sympathetic. Deep sensibility fibers also pass in the motor roots (Sherrington).²

Third Nerve Palsies.—These are often very complicated and may be central or peripheral, complete or partial. Complete paralysis of both third nerves is rare, partial palsies are the rule. Unilateral palsy of all of the external muscles governed by the third nerve (often termed ophthalmoplegia externa) is due usually only to a lesion involving the second or peripheral motor neurones of the third nerve. Bilateral external ophthalmoplegia may occur also from lesions of the mesencephalon and cortical oculomotor paths. The chief causes for central palsies are various types of encephalitis, polioencephalitis, either infectious as in Heine-Medin's disease, or toxic as in alcoholism. (Wernicke's polioencephalitis superior.) Syphilitic thrombosis may cut off the blood supply of the nuclei. Pressure from the aqueduct above, or third ventricle may cause pressure palsies, usually of irregular distribution. (Nothnagel's Syndrome. Pineal Syndrome.)

Peripheral palsies are more frequently due to disease at the base, usually basal syphilitic meningitis, tumor, tuberculosis, hemorrhage, traumas (rarely) or are occasioned by involvement of the fibers as they pass through and about the red nucleus by tumor, multiple sclerosis, or when implicated in a thrombotic or hemorrhagic softening of the cerebral peduncle—Millard-Gübler, Benedict, Fovilles' syndromes, red nucleus syndromes. Infectious disease neuritis may also occasion peripheral palsies. Pressure from aneurism of the internal carotid, and thrombosis of the cerebral sinuses (sinus cavernosus) may also cause peripheral palsies. A special herpes zoster ophthalmicus is known. Exophthalmic goitre and diabetes are special causes.

¹ See Wilbrand and Sängner, and Henschen in Lewandowsky Handbuch, vol. iii.

² Sherrington and Tozer, Proc. Royal Soc., 1910.

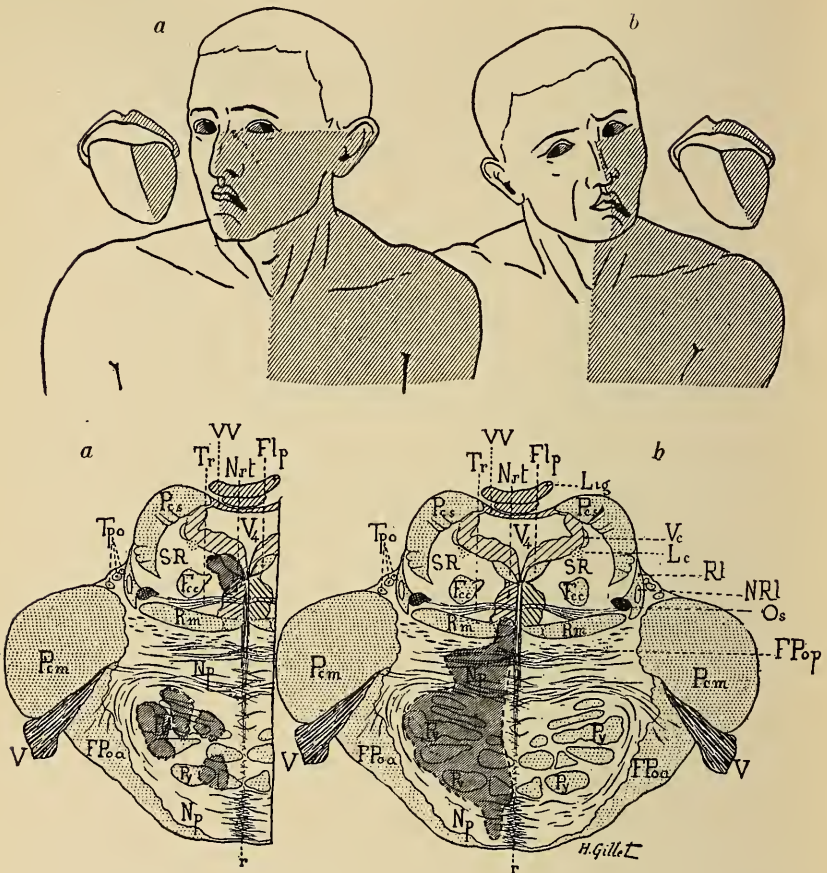


FIG. 88.—Foville's syndromes, with anterior and posterior pontine syndromes. Hemiplegia, cerebral type, with (a) conjugate deviation of the head and eyes, (b) by lesions of the upper portion of the pons, right side, involving the anterior portion of the pons and the region of the tegmentum. On the left side there is a contralateral hemiplegia of the limbs, of the lower part of the face and of the tongue, because of the involvement of the pontine pyramidal fibers *Py* (cortico spinal pyramidal fibers, cortico nuclear facial and lymphoglossal fibers. In c, right-hand figure, there is a single lesion which involves the tegmentum at its antero-internal angle and destroys the head-turning (cephalogyric) and eye turning (oculogyric) fibers of the right side which at this level are situated in the pes lemniscus and the internal portion of the median fillet giving rise to conjugate deviation of the head and of the eyes. By reason of the predominant action of the antagonists the head is inclined to the right and the eyes look to the right, the patient looks to the side of the lesion. In a there are multiple isolated lesions. Four large foci in the anterior portion destroy the pontine pyramidal fibers with a resulting crossed contralateral hemiplegia of the extremities, the face, and the tongue. Another focus occupies the posterior internal portion of the tegmentum and destroys the internuclear oculogyric fibers of the posterior longitudinal bundle which directly unites the nuclei of the sixth and third nerves and *vice versa*. There results a paralysis of the eyeballs by which they cannot turn sideways toward the right—right oculorotary paralysis—by reason of the predominance of the antagonists the patient looks to the left. The patient looks away from the lesion toward the paralyzed members. The cortical oculorotary fibers and the pes lemniscus are intact. For details of structure and abbreviations, see chapter on Midbrain Lesions. (Dejerine.)

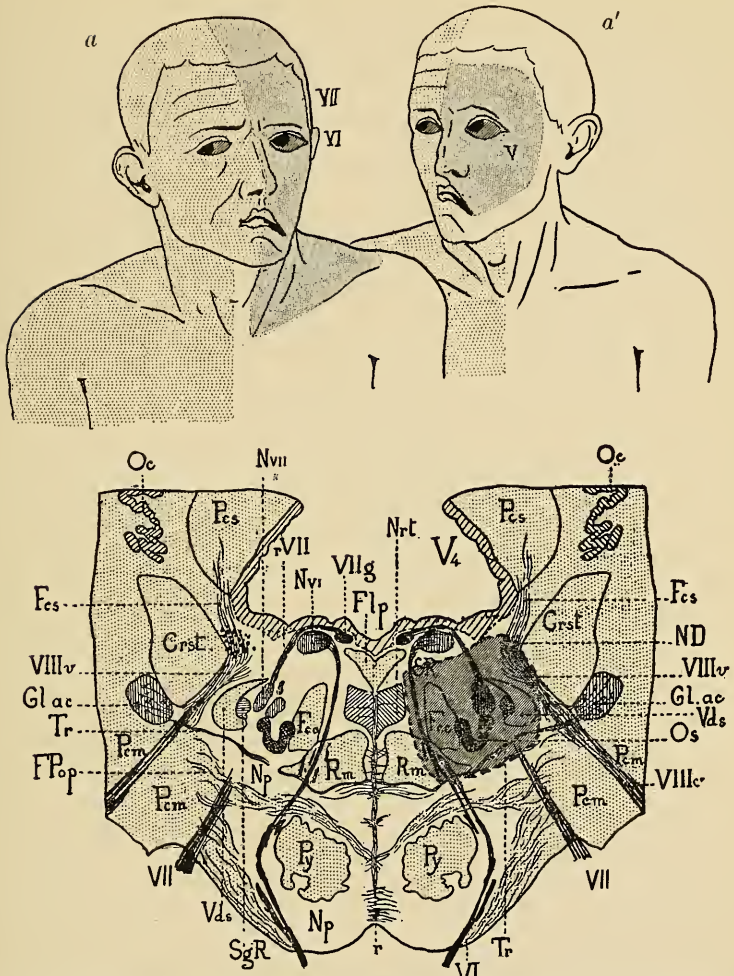


FIG. 89.—Pontine syndrome, with eye palsies of central origin and syringomyelic dissociation. There is here a crossed hemianesthesia with alternating paralysis of the VI and VII cranial nerves, anesthesia of the V nerve due to hemorrhage in the lateral and lower portion of the pontine tegmentum of the left side. The *right-hand figure* shows the hemianesthesia, dissociated as in *syringomyelia* (hemianalgesia and hemithermanesthesia due to lesion of the crossed sensory pathways of the lateral portion of the reticular formation). There is preservation of the tactile and postural sensibilities and of the stereognostic sense, because of the incomplete extension of the lesion to the median lemniscus (*Rm*). The *left-hand figure* shows (1) atrophic paralysis of the VII nerve with reaction of degeneration, lagophthalmia, drooping of the lips, loss of facial mimicry, paralysis of the entire left facial (VII) indicated (Fig. *a*); (2) anesthesia of the face, following involvement of the descending root of the trigeminal (see *V* on *a'*); (3) paralysis of the external rectus with convergent strabismus by reason of the overaction of the antagonists. Furthermore, there is a paralysis of the lateral movements of the eyeballs toward the left notwithstanding the integrity of the posterior longitudinal fasciculus (*Flp.*), of the nucleus of the VI and of the adjacent reticular formation. The lesion of Deiters' nucleus, and of the labyrinthine oculo-rotatory fibers which unite Deiters' nucleus (*ND*) to the nuclei of the III and VI causes this. By reason of the overaction of the antagonists the patient looks to the right. (After Dejerine.) For abbreviations of the anatomical sketch see section on Midbrain.

Transitory third nerve palsies occur in the disorder known as ophthalmoplegic migraine (*q. v.*).

Syndromes.—The compound character of the nuclei and the loosely arranged bundles making up the nerve explain the great range in symptomatology. Obersteiner (5th edition, 1912) follows Bernheimer chiefly in his teaching regarding the complicated question of the localization of the brain stem nuclei.¹ Thus it will be seen that from before backwards the nuclei are arranged as follows: Levator palpebræ, rectus superior, rectus inferior, obliquus inferior, rectus inferior, trochlearis.

A complete unilateral palsy, probably nuclear (ophthalmoplegia completa), would then cause ptosis, wrinkling of forehead on same

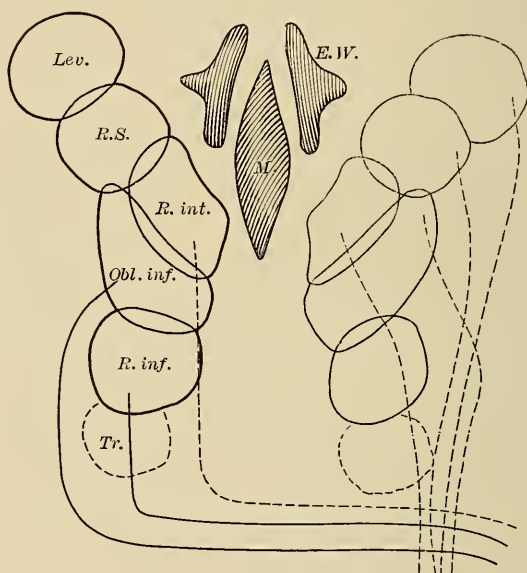


FIG. 90.—Scheme of oculomotor nuclei modified from Bernheimer. Basal projection. *M*, median nucleus; *E.W.*, Edinger Westphal sympathetic nucleus. (Obersteiner.)

side, wide pupil, irresponsive to light and accommodation, eye turned outward and slightly downward. Double vision is present and some dizziness in the early stages. A variety of individual muscle palsies may also result from either nuclear or peripheral involvement as indicated—ophthalmoplegia externa, when the pupil is not involved; ophthalmoplegia interna when only the internal muscles are involved—a rare condition.

The distinction of nuclear from peripheral palsies is usually made on the basis of accompanying symptoms—sensory or motor, due to implication of the red nucleus, or of the cerebral peduncles. In

¹ See Kidd, *Rev. Neu. and Psych.*, xi, 507.

the absence of these accessory symptoms (Weber-Gübler, Benedict syndromes, rubrospinal syndrome) the distinction may be impossible. There is no single disease process to which the term ophthalmoplegia may be rigidly applied. Hence there is no general course and no general treatment. The various palsies must be interpreted on the basis of the dynamic factors, and the treatment must be founded upon the causation. Syphilis is responsible for the majority of these palsies, and calls for verification by the cytobiological tests and prompt antisyphilitic treatment, best by salvarsan and hypodermic injection of mercury. (See chapter on Syphilis of the Nervous System.)

Isolated involvement of the pupillary apparatus may be discussed here. Contracted pupils, irregular pupils, unequal pupils, dilated

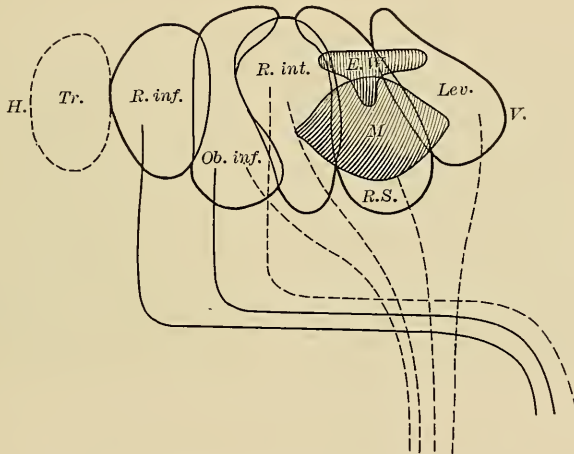


FIG. 91.—Scheme of oculomotor nuclei, modified after Bernheimer. Sagittal projection. Lettering as before. (Obersteiner.)

pupils, etc., have been discussed (see Symptomatology). A dilated pupil with loss of accommodation reflex is frequently seen in severe alcoholism (Korsakow's syndrome). It may be present also in optic nerve disease—combined with loss of light reflex as well. A loss of light reflex with preservation of the accommodation reflex (reflex iridoplegia, Argyll-Robertson pupil) is a frequent sign of syphilis. Its mechanism has been discussed. It is often unilateral in the beginning of a tabes or paresis, or other type of cerebrospinal syphilis and may occur in a number of other conditions, though rarely. In cerebral syphilis it usually becomes double.

Isolated ptosis may also be seen. This may be due to oculomotor invasion (tabes), or it may be hysterical. In the former case there is usually a compensatory overaction of the corrugators, in the latter, this is less likely to take place. Sympathetic disease,

ptosis from palsy of Müller's muscle, is usually accompanied by pupillary disturbances and the eyeball lies somewhat retracted in the eyeball cavity. The cervical sympathetic is here implicated.

Chronic Progressive Eye Palsies.—These make up a special group, occasionally congenital, more often they are a part of a progressive anterior poliomyelitis. (Bulbar palsy, *q. v.*)

Fourth Nerve Palsy.—The fourth (trochlearis) nerve supplies the superior oblique muscle with its motor fibers. Afferent fibers carry deep sensibility fibers from the muscle. The fibers are crossed and uncrossed, the latter being phylogenetically the first to appear, but later are overshadowed by the crossed fibers. Isolated palsy causes a marked diplopia, and some dizziness when the patient looks downward and outward. The false image stands lower and nearer than the true one, its upper end inclined toward the true image.



FIG. 92.—Paralysis of the fourth nerve. Inability to look down. It will be noted that as the eyeball does not turn downward the eyelid does not descend, at the same time the patient can close his eyes when told to do so. (Russel).



FIG. 93.—Paralysis of the fourth nerve. The same patient closing his eyes to command. (Russell.)

Looking upward or downward causes no diplopia. These patients have difficulty in descending stairs, and they incline the head forward and toward the sound side to adjust to their diplopia.

The fourth nerve is frequently involved with the third and sixth in basal inflammations or new growths, or may be involved independently from pressure in the posterior fossa (cerebellar tumor). It also is involved at its nuclear origin from encephalitis, poliomyelitis, toxemias, etc. (Figs. 92 and 93.)

Sixth Nerve Palsy.—This is perhaps the most frequent of the eye palsies. The peripheral motor neuron is exposed for three-fourths of an inch or more on the base of the skull, and is therefore subjected to greater possibility of local pressure than any other cranial nerve. When the external rectus muscle is paralyzed there is an internal strabismus and a slightly smaller pupil in the affected eye. There is also a diplopia.

Sixth nerve palsy more usually results from basilar disease, fracture of base, meningitis (syphilitic and other types), tumors not only of the base but also of the brain substance itself. Nuclear involvement is seen in encephalitis, poliomyelitis, toxemias, etc. External rectus palsy as a result of a myositis is not unknown.

Central Motor Neurons.—Isolated eye palsies are due to nuclear or to peripheral involvement of the third, fourth and sixth nerves. Supranuclear disease, involving the oculomotor paths, does not result in the loss of function of a single eye, much less of a single eye muscle. Such lesions between the oculomotor cortex and the nuclei in the midbrain cause complicated disorders of the associated movements of the eyes. The most frequent of these are: (a) conjugate deviation, (b) lateral associated palsy, (c) vertical associated palsy, (d) loss of convergence, (e) central nystagmus, (f) irregular types. (See Plate VIII.)



FIG. 94.—Paralysis of upward movement of the eyes, showing the excessive wrinkling of the forehead in the attempt to look up. Skew deviation. (Holmes.)

(a) *Conjugate Deviation.*—Here both of the eyes are directed to the side of the lesion, and cannot be voluntarily moved in an opposite direction. Yet, if the eyes are fixed upon an object and the head is turned away from the lesion, the eyes will turn in the direction which voluntarily is impossible. This is termed conjugate deviation of the eyes and head. The eye axes may not be truly parallel, but may diverge slightly. In acute apoplexies this symptom is occasionally seen,—*i. e.*, forced deviation of the head to the side of the lesion.

Lesions of the inferior parietal, angular gyrus, and possibly the foot

The Oculorotary Paths and in Particular the Innervation of the Muscles which Turn the Head.

Abbreviations.—*BrQa*, arms of anterior corpora quadrigemina; *C*, the cochlea with spiral ganglion, the cochlear branch of the *VIII* pair; *Ca*, anterior horn of the spinal cord; *Cge*, external geniculate; *Cj*, juxta restiform body; *Cio(Rm)* interolivary region of the medulla containing the median lemniscus; *Cirl*, retrolenticular segment of the internal capsule; *CoN*, corticonuclear contingent of the pyramidal tract, *CSgt*, sagittal view of the occipitotemporal lobes; *CV*, visual area of the internal face of the hemisphere, transparent view; *CI-II*, first and second cervical pair; *DC*, center of conjugate deviation of the head and of the eyes; *Dext*, right external rectus muscle; *Dint*, right internal rectus muscle; *Fa*, ascending frontal convolution; *F₁, F₂, F₃*, three frontal convolutions; *fap*, posterior and internal arcuate fibers of the medulla; *Flp*, posterior longitudinal fasciculus; *fls*, tectospinal fibers; *xV₂*, central or secondary trigeminal pathway; *GG*, Gasserian ganglion; *G.Sc*, Scarpas ganglion; *NBe*, Bechterew's nucleus; *ND*, Deiters' nucleus; *NVs*, sensory trigeminus nucleus; *NIII*, oculomotor nucleus; *III* pair; *NVI*, nucleus external rectus; *NVIIIc*, anterior terminal nucleus of the cochlear; *NVIIIv*, triangular nucleus of the vestibular; *NXI*, spinal accessory nucleus (trapezius-sternocleidomastoid); *Os*, superior olive; *P₁, P₂*, superior and inferior parietal lobe; *Pa*, ascending parietal convolution; *Pc*, angular gyrus; *Pci*, inferior cerebellar peduncle; *Pcm*, middle cerebellar peduncle; *Pul*, pulvinar; *Qa, Qp*, anterior and posterior corpora quadrigemina; *R*, fissure of Rolando; *RgRm*, region of median lemniscus; *RI*, lateral lemniscus; *Rm*, median lemniscus; *SgR*, gelatinous substance of Rolando; *SR*, reticular substance; *SRg*, gray reticular substance; *T₁, T₂, T₃*, temporal convolutions; *Tr*, trapezoid body; *V*, vestibular nerve; *V₁V₂V₃*, three branches of the trigeminus, ophthalmic, superior and inferior maxillary; *Vsd*, descending root of the trigeminus; *xM*, tegmental crossing of Meynert; *XII*, optic chiasm; *III*, oculomotor; *VI*, external rectus; *VIIIc*, cochlear branches of the auditory; *VIIIv*, vestibular; *XI*, spinal accessory.

The tegmentum in its medullary, pontine, peduncular portions seen in projection at the level of the aqueduct of Sylvius and the fourth ventricle, with the reticular formation (*SR*), the posterior longitudinal fasciculus (*Flp*) and the median lemniscus (*Rm*). It is limited laterally by the lateral lemniscus (*RI*), colored in yellow, and the long sensory nuclei of the trigeminus (*V*) and of the auditory (*VIII*) nerves (*NVs, SgR*) colored green, and in yellow (*NVIIIc, NBe, NVIIIv, ND*) and showing each side of the median line: (1) above the nuclei of the *III* pair (*NIII*) which innervates by crossed and by direct fibers the internal rectus of the eye; in the center, the nuclei of the *VI* pair (*NVI*) which innervates the external rectus of the eye, and (3) below, the cephalorotary nuclei which act to rotate and incline the head and neck; spinal nuclei and spinal accessory *NXI*, and motor centers of the cervical cord (*Ca*).

Myelinated early are the fibers which unite the nuclei of the sixth and of the third pair and of their associated fibers to enable the lateral movements of the eye to take place in the early stages of life. These *internuclear* fibers, colored in red, take their origin from small cells in the nuclei in the oculomotor, *III* and abducens *VI*, and pass by means of the posterior longitudinal fasciculus; the small ganglion cells of the nucleus of the left *VI* pair for example, can put into action the crossed and direct root fibers going to the left internal rectus; and at the same time the ganglion cells of the nucleus of the *III* left pair, can put into action the root fibers of the homolateral external rectus of the same side (left). Thus there is established a strict physiological association, permitting the action of a dextrorotary system, turning the eyes toward the right, or a levorotary system, turning the eyes to the left, an association which can be incited and activated may be by the cortex, or by various sensory or sensorial paths, labyrinthine, tactile or optic.

1. The cortical oculorotary pathway (colored dark red in the right, pale red on the left) belongs to the corticonuclear path (*CoN*) (see Plate I, *O*) and takes its

origin from the center of deviate conjugation of the head and eyes (*Dc*) situated at the juxtaposition of the second and ascending frontal regions. It passes by way of the knee of the internal capsule, internal portion of the cerebral peduncles; descends with the aberrant fibers of the pyramidal tracts (see Plate I, *D*) in the median lemniscus and passes to the nuclei of the *III* pair, to the nuclei of the *VI* pair of the opposite sides, and to the cephalorotary nuclei of the two sides.

2. The labyrinthine oculorotary pathway, colored yellow, contains a vestibular oculorotary path, for static equilibrium, and a cochlear, or acoustic, oculorotary pathway. The vestibular oculorotary pathway takes its origin in the cells of the terminal nuclei of the vestibular root (*NBe, NVIIIv, ND*). Its fibers belong to the posterior and anterior arcuate fibers of the medullopontine tegmentum (*fap*). They actionate the two *VI* nuclei and give to the posterior longitudinal fasciculus (*Flp*) direct and crossed, ascending and descending fibers. The ascending crossed and the descending direct fibers are the most numerous. The ascending fibers actionate the nuclei of both *III* pairs; the descending fibers actionate the head turning nuclei.

The acoustic rotary pathway is represented by the cells of the anterior nucleus of the acoustic (*NVIIIc*) the trapezoid body (*Tr*) the fibers destined to the superior olive of the same side (*Os*) and of the crossed side and the fibers which go in the lateral lemniscus (*Rl*). This pathway can incite the oculorotary system by the fibers of the hilum of the superior olive which actionate (a) the motor cells of the external rectus (*VI*), (b) the motor cells of the oculomotor (*III*) acting on the internal rectus of the opposite side by the intermediation of small cells of the intranuclear oculorotary system. The labyrinthine oculorotary pathways can be activated not only by peripheral sensations—equilibrium (vestibular), auditory (cochlear), but also by the cerebellum, by reason of the system of internal fibers of the semicircular canals and cerebellovestibular fibers which terminate in Bechterew's and Deiters' nuclei. These connections are not figured in the plate.

3. Lateral movements of the eyeballs can be induced by sensory stimuli acting on the skin of the head and body. The central trigeminal oculorotary pathway (green) takes its origin in the nuclei of the *V* pair (*NVs*), passes by the arcuate fibers of the medullopontine tegmentum and the posterior longitudinal fasciculus, and then is divided into ascending and descending fibers which actionate the oculomotor nuclei (*III* and *VI*) and the cephalorotary nuclei (*XI*, ant-horn cells *Ca*). The central sensory pathway (black) can actionate the oculorotary and cephalorotary nuclei by fibers which originating in the reticular formation (*cSR*) ascend and descend in the posterior longitudinal fasciculus.

4. The visual oculorotary pathway, tecto-spinal, (*fts*) (light blue) leaves the anterior corpora quadrigemina, crosses the middle line at the level of the dorsal decussation of Meynert (*xM*) descends in the prelongitudinal fascicle, giving off terminal and collateral fibers to the nuclei of the *III* pair of the homolateral side, to the nuclei of the *VI* pair and to the cephalorotary nuclei (*NXI, Ca*) of the opposite side.

It is actionated in part by the peripheral visual pathway (retina, nerve, chiasm) (*xII, II*) (dark blue) and in part by the central visual pathway in the calcarine fissure (visual center) and the anterior corpora quadrigemina. It can also be indirectly stimulated by the efferent visual pathway, which originating in the primary optic centers (*Pul, Cge*) irradiates in the calcarine cortex passing by way of the retrolenticular segment of the internal capsule (*Cirl*) and the optic radiation of the occipitotemporal lobes.

A destruction of the cortical oculorotary pathway (right) for example, determines a paralysis of the levo-oculorotary and levocephalorotary systems; that is to say, a conjugate deviation of the head and of the eyes of the opposite side. A lesion of the labyrinthine oculorotary pathway, left, will also determine a paralysis of the levo-oculorotary system by abolition of the lateral movements of looking of the direct, homolateral side. In both instances the patient looks to the right because of the predominance of the antagonists (Dejerine).

of the second frontal gyrus may produce or occasion this type of forced position of the eyeball.

Lesion of the centrum ovale, and of the internal capsule involving the projection fibers of the oculomotor may cause conjugate deviations, here associated with hemiplegia as a rule.

The chief lesions causing conjugate deviation are: hemorrhage or softening, abscess, encephalitis, occasionally tumor.

(b) *Lateral Associated Palsy*.—Here the eyes are unable to pass the middle line. The altered position of the head as seen in conjugate deviation is absent and movements of the head are unavailing in bringing the eyes past the middle line. Convergence, however, may remain intact. Certain incomplete conjugate deviations are found here.



FIG. 95.—Cerebral syphilis. External rectus palsy, right eye.



FIG. 96.—Trochlearis palsy in cerebrospinal syphilis.

Lateral conjugate palsy is usually due to a pontine lesion on the side of the palsy, and which implicates the abducens fibers near the nucleus, and the synapses of the posterior longitudinal bundle, possibly Lewandowsky's tractus pontis ascendens. Pressure at a distance may also occasionally cause a lateral conjugate palsy.

Fractures, pontine tumors, multiple sclerosis, softening (after labyrinthine infection) are among the causes of this comparatively rare condition.

(c) *Vertical Associated Palsy*.—Here the motion of both eyes is hindered only on looking up or down—all other associated movements

are possible. When there is loss of ability to look down usually the eyelids do not descend as they normally do. In some patients the palsy is not symmetrical, one eye moving up or down more than the other.

Certain forced positions of the eye, one being higher than the other—(Magendie-Hertwig position) may be mentioned here. These are present as a cerebellar sign (middle cerebellar peduncle). The side involved is indicated usually by the lower lying eye. There is also nystagmus.

The cause for the failure of the lid to respond, analogous to von Graefe's symptom in exophthalmic goitre, is not thoroughly understood.



FIG. 97.—Inequality of pupils. Left pupil larger than right. Cerebral tumor. Immobile.

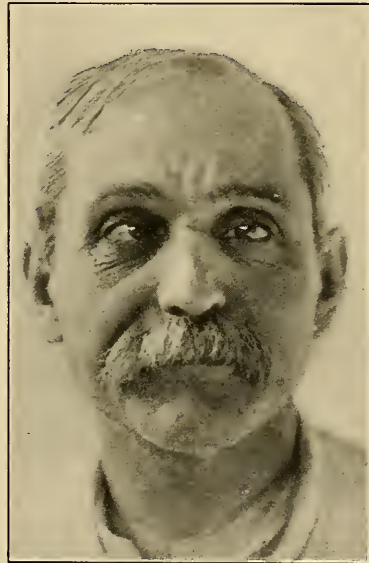


FIG. 98.—Tabes. External rectus palsy.

In the majority of the cases lesions have been found implicating the corpora quadrigemina (Pineal case) either directly or by tumor, or by direct pressure. So-called hysterical cases are usually mistakes in diagnosis. One such of Lewandowsky's proved to be a cysticercus of the corpora quadrigemina. A personal case developed a sarcoma of the third ventricle, pressing upon the anterior corpora quadrigemina.

(d) *Paralysis of Convergence*.—As an isolated symptom this is rare, it is usually accompanied by other associated palsies. It is found most frequently in multiple sclerosis. A closely related phenomenon—weakness of the internus muscles, Möbius' symptom in exophthalmic goitre, is thus far difficult of explanation.

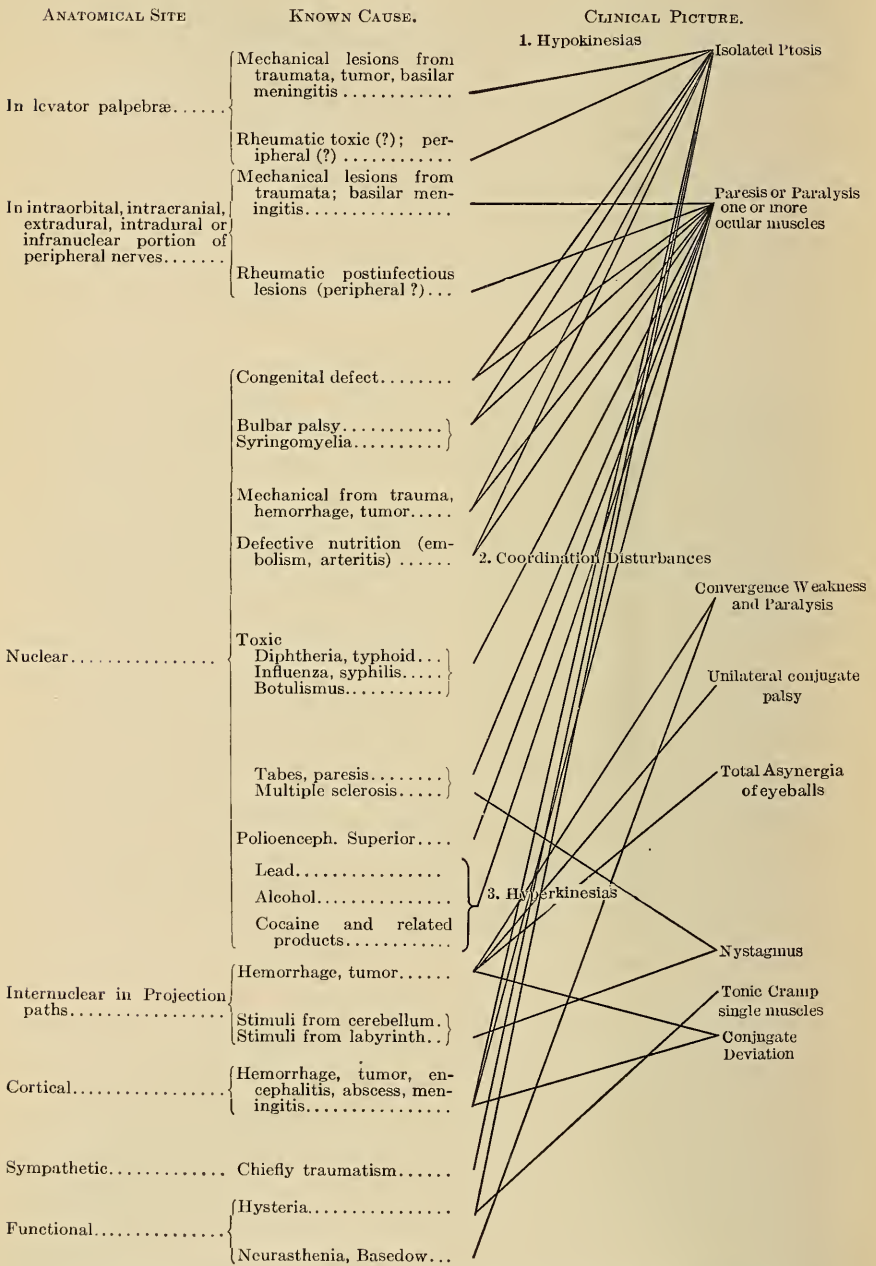


FIG. 99.—Summary of disturbances of external ocular nerves. (Veraguth.)

(e) *Central Nystagmus*.—The extremely complicated subject of central nystagmus is more fully discussed in the section on vestibular disease. When rhythmic, *i. e.*, possessing a quick and a slow excursion, it is usually vestibular. Undulating nystagmus, *i. e.*, with uniform backward and forward movements, is more apt to be due to involvement of the central or peripheral eye muscle nervous pathways. Possibly vestibular associations must always be involved. Undulating nystagmus is occasionally seen in severe fatigue, in myasthenia, in progressive muscular atrophy, alcoholism, hydrocephalus, etc. Undulating nystagmus as well as dissociated eye movements is normal in infants, and is frequently seen in congenital defects—(idiots, imbeciles, congenital blindness).

(f) *Irregular Palsies*.—Dissociated eye movements in which the eyes move irregularly, each according to its wish, as in crustacea, in the very young infant, in congenital defective developments, is seen coming on in adults from destruction of the associative mechanism of the eye movements, more particularly from separation of the nuclei of the oculomotorius. Lesions which cut the nuclei apart one from another (multiple sclerosis, tumor) will cause this asynergia or ataxia of the eye.

Cycloplegia is a condition of absolute paralysis of both eyes, the gaze being fixed and immobile, and there being no movement of the iris either. A double-sided lesion of the corticopontine eye muscle projection fibers causes such a lesion.

Skew deviations are conditions in which one eye is directed outward and downward, the other inward and upward. Such a compulsory eye position is usually either due to a middle cerebellar peduncle affection or to a cerebellar lesion elsewhere.

Eyeball apraxias, so-called, or ideomotor dissociated movements offer certain complex analogies with similar disturbances of the tongue muscles in speech, the facial muscles in mimicry, or the arm muscles in expression. They are usually due to lesion of the projection fibers in the centrum ovale or internal capsule.

DISEASES OF THE TRIGEMINAL NERVE.

Fifth or Trigeminal Nerve.—The symptomatology of lesions of the fifth nerve is diverse, as it has both a sensory and a motor part, and has many synaptic junctions with cranial, spinal and sympathetic nerves.¹

Motor Part.—The cortical origin of the motor part is bilateral, and is located in the lower third of the central convolution. From here the fibers pass through the corona radiata, enter the internal capsule with the pyramidal tract, and make their first synapsis with the chief motor nuclei, in the dorsolateral part of the tegumentum of the pons. Most of the fibers cross about the level of the posterior corpora quadrigemina. From here the second motor neurone passes with the

¹ Map scheme of the Sensory Distribution of the V. Nerve. L. H. Pegler, 1914.

inferior maxillary branch through the foramen ovale, and is distributed to the masseter, temporal, pterygoids, tensor tympani, tensor veli palatini, mylohyoid and the anterior belly of the digastric.

Affection of the cortical motor neurons occurs in pseudobulbar palsy. Here the lesion is bilateral also. Unilateral interruption of the tract causes little disorder in mastication. (Hirt claims that a left-sided lesion may cause bilateral palsy.) Bilateral disorder is nearly always associated with the other features of pseudobulbar palsy (*q. v.*). The paralytic signs are the half-open mouth, with inability to close the teeth. The jaw cannot be protruded, and the lateral movements are impaired. The food is apt to fall out of the mouth, cannot be held by the lips, and cheeks or tongue, and has to be manipulated by the fingers. Food often is pushed up to the pharynx and nose. There is no atrophy of the muscles of the jaw, and no reaction of degeneration. The jaw jerk is increased.

Cortical foci may give rise to chattering movements of the jaw. Grinding movements of the jaw, so frequent in paresis and occasionally present in senility, are due to cortical irritation. The champing movements of the jaw in paralysis agitans are possibly to be interpreted similarly to the general tremor of the other muscles; namely, as an interruption of the tonic impulses passing through the midbrain structures. (See Paralysis Agitans.) Grinding of the jaw is not infrequent as a reflex in children, and it occasionally is seen as a result of basal meningeal irritation of the motor root, as in tuberculosis, syphilis, or even tumor formation.

Prolonged spasm of the muscles of mastication is seen in certain toxemias, such as strychnine poisoning, tetanus, tetany. Here the interpretation is not simple. It is a result possibly of the marked lowering of the synaptic threshold in the pontine motor nuclei, causing over response to the cerebral or reflex motor impulses. The violent convulsive movements of the epileptic discharge are cortical in origin.

Irregular or anomalous spasmodic movements of the jaws occur in multiple sclerosis, usually from midbrain or pontine localizations of the plaques, or they may be reflex or psychogenic (hysteria or dementia precox). In the latter instances the biting symbolizes, in a few cases analyzed, hate or sadistic complexes. The negativistic clenching of the jaw in food refusal as in dementia precox, depressed manic-depressives, fever deliria or confusion, expresses various symbolizations. Fear of being poisoned is here a frequent motive.

Nuclear disease of the motor neuron of the trigeminus may be unilateral or bilateral, partial or complete. In unilateral monoplegia masticatoria, the lateral movement of the jaw takes place to the paralyzed side. Bilateral lesion causes the jaw to fall, and abolishes all lateral movements. The floor of the mouth is flaccid from the mylohyoid and digastric palsy, and there is difficulty of hearing notes of low-pitched tuning forks. The muscles show atrophy, reaction of degeneration and the jaw-jerk is absent.

Peripheral trigeminal motor palsy is usually associated with sensory, sympathetic and taste phenomena.

Nuclear disease of the motor neurons is comparatively rare. It may occur in multiple sclerosis, in syphilis of the pons, hemorrhage,

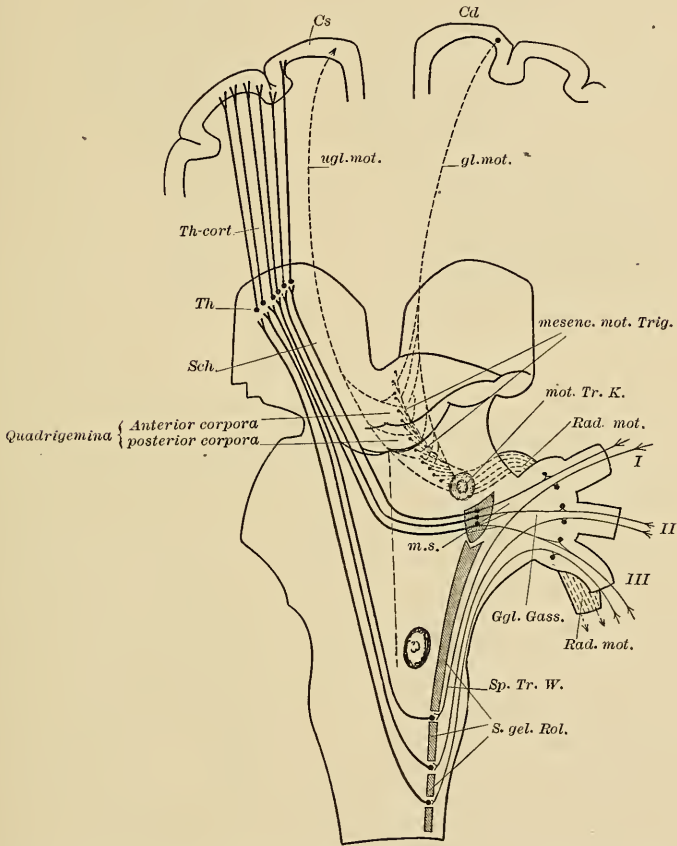


FIG. 100.—Scheme of intracerebral trigeminal pathways. *Cs*, left hemisphere; *Cd*, right hemisphere; *Th-cort.*, thalamocortical trigeminal tracts; *Th*, synapses of trigeminal in thalamus; *Sch*, mesencephalic lemniscus; *mot. Trig.*, motor trigeminal root nuclei in midbrain; *mot. Tr. K.*, motor trigeminal nucleus in locus ceruleus; *Rad. mot.*, motor roots; *Ggl. Gass.*, Gasserian ganglion; *I, II, III*, three trigeminal branches; *m.s.*, mesencephalic sensory trigeminal roots; *sp. Tr. W.*, spinal trigeminal roots; *S. gel. Rol.*, substantia gelatinosa Rolando. Dotted line, motor, solid line, sensory. (Veraguth, Bing.)

poliomyelitis, syringomyelia. Peripheral palsies are more frequent, and are due to trauma, to pressure of carotid aneurisms, tumors, chronic meningitis, rarely to an interstitial neuritis.

Sensory Part.—Affections here are much more intricate and complex, and are often combined with motor symptoms. The sensory receptors

of the trigeminus are widely distributed over the face, the mucous membranes of the superior and anterior nasal fossa, the frontal and

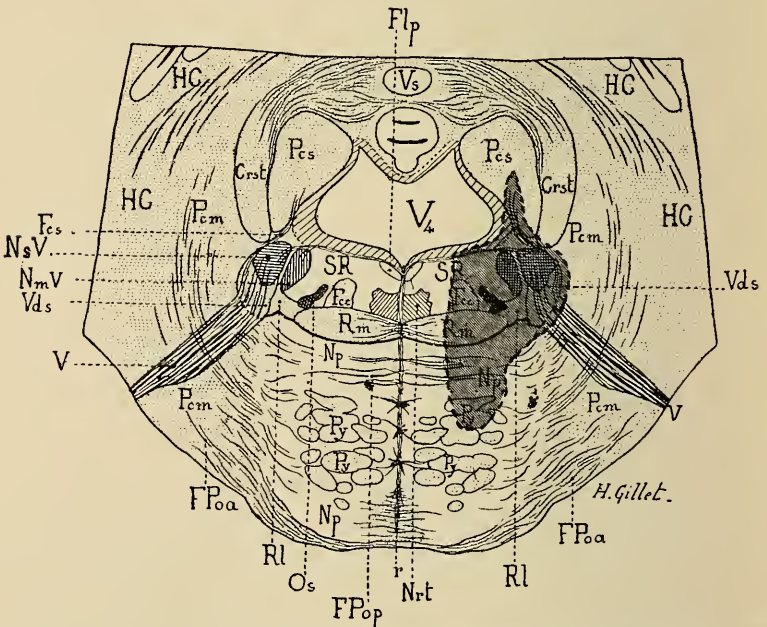


FIG. 101.—Pontine syndrome. Alternate hemianesthesia of the trigeminus by lesion of the median portion of the left pontine tegmentum involving the motor and sensory roots of the trigeminus, their root fibers, the external portion of the median lemniscus, the crossed sensory paths of the tegmentum and extending as far as the superior cerebellar peduncle behind and in front as far as the pons implicating peduncular pyramidal fibers. On the *right* side there is hemiparesis of the extremities and of the inferior facial (oblique cross hatchings), hemianesthesia of the extremities, neck and head especially of pain and temperature sense (vertical lines). On the *left* side there is anesthesia of the trigeminus; paralysis of the masticators, the pterygoids especially, the masseter and the temporal. There are choreo-athetoid movements of the body due to lesion of the superior cerebellar peduncle (*Pcs*). (Dejerine.) See Midbrain Section.

ethmoid sinuses, tentorium cerebelli, teeth, mucosa of posterior inferior nares, the sinuses of the jaw, the dura mater, the mucous membranes of the lips, cheeks, posterior and inferior portion of the mouth, and anterior two-thirds of the tongue as taste buds. The sensory ganglion is the Gasserian.

Collateral synapses occur with the ciliary ganglion for the passage of impulses from the corneal and sclera receptors. Impulses from the ethmoid and sphenoid sinuses; from the pharynx, posterior nares, hard and soft palates, maxillary sinus, uvula, pharyngeal walls, tonsils and related mucous parts pass by way of the sphenopalatine ganglion and come into relation with the glossopharyngeal. The submaxillary ganglia connections are involved and probably pass with the vegetative nervous system fibers.

The chief reflexes arising from these connections are:

1. Winking reflex.
2. Pupillary (sympathetic reflex)—pinching the cheek or neck causes a dilatation of the pupil on the same side.
3. Jaw reflex.
4. Sneezing reflex.
5. Pharyngeal reflex—(gagging and swallowing).

The centripetal pathways from the Gasserian ganglion join to form a large sensory root which is distributed to two main end stations: a mesencephalic and a spinal one with numerous collaterals. From these roots the second sensory neurone passes through the median lemniscus to the opposite side to end in the thalamus. A further neurone then passes to the sensory brain area.

The chief sensory symptoms of fifth nerve involvement are hyperæsthesiæ and neuralgia, hypæsthesiæ, anesthesiæ, loss of taste, trophic and secretory.

The most frequent of the syndromes is *trigeminal neuritis* or *tic douloureux* (*q. v.*). Here the Gasserian ganglion is often involved or the neuralgia may be due to pressure upon one or all of the roots. The distribution of the hyperæsthesia is of value in determining the branch or branches involved. It is comparatively rare to find reflex neuritic pains from disease of the teeth, hence the hope that removal of healthy teeth will cure a *tic douloureux* is usually doomed to disappointment. This is an extremely common error and needs to be emphasized.

Central or thalamic trigeminal pains are possible. Clinically little is known of them. Trigeminal agnosia is a curiosity merely. Anesthesia may be due to interruption of peripheral, pontine, thalamic or cortical pathways. The diagnosis as to localization must be made on the basis of the accompanying symptoms, sensory, secretory, trophic, and motor.

An inflammation of the Gasserian ganglion causes a trigeminal herpes zoster. Dryness of the eyes, with hyperæsthesia or anesthesia is due to a peripheral lesion of the superior or first branch of the nerve.

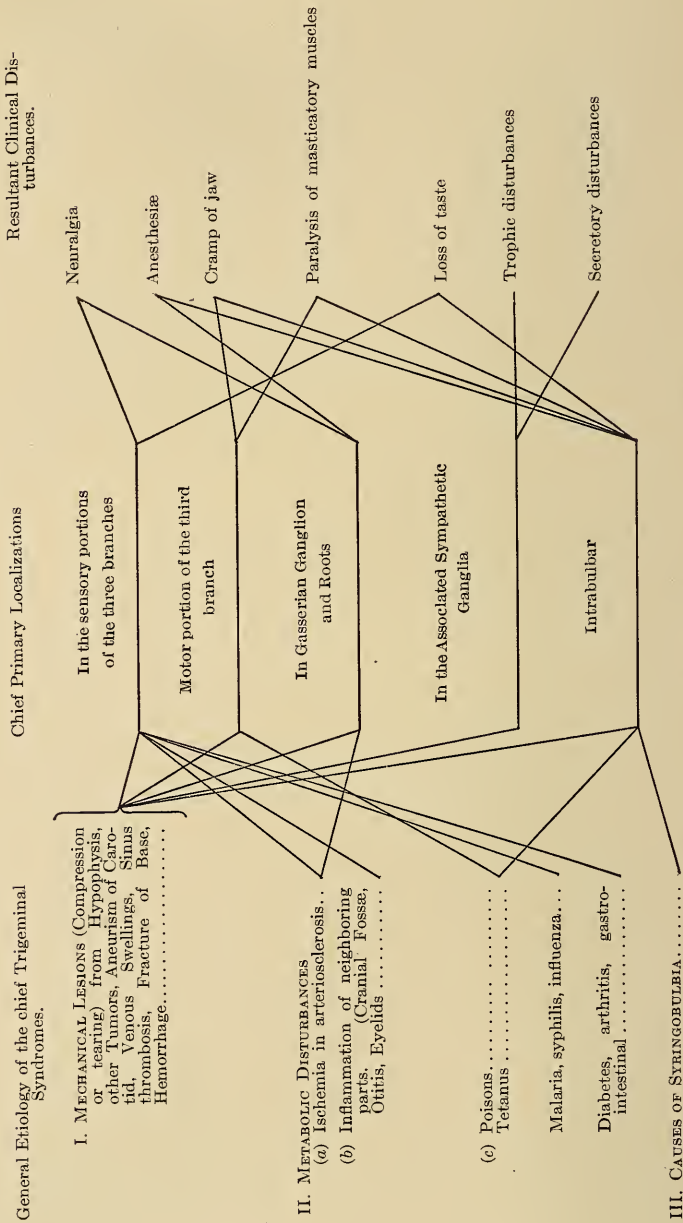
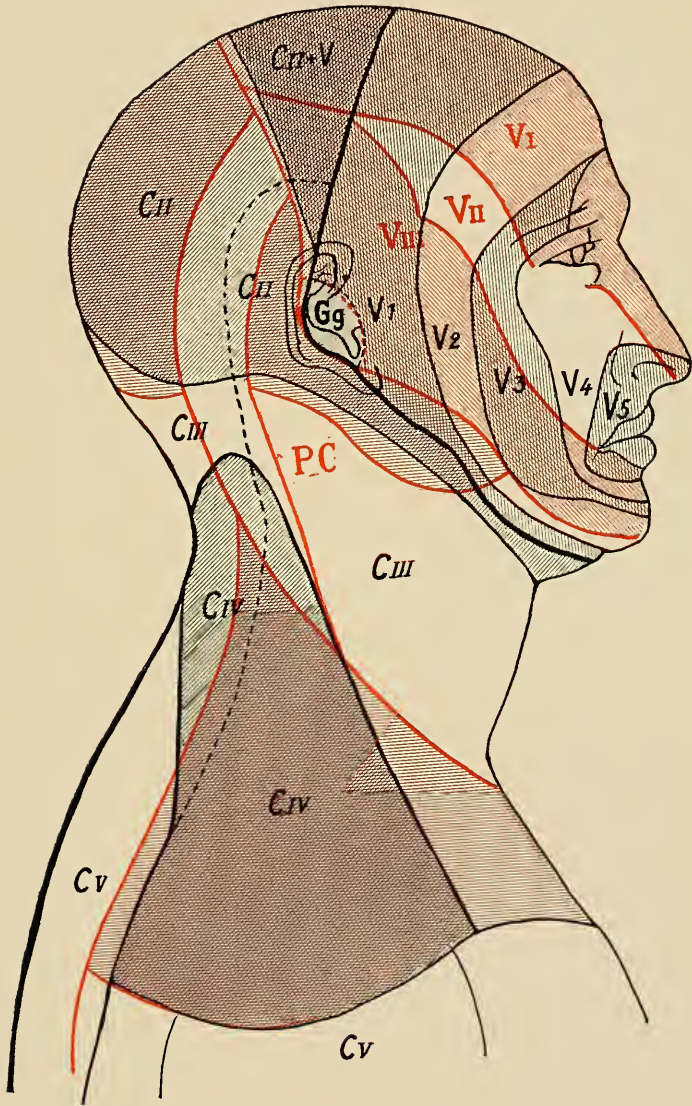


Fig. 102.—Summary of trigeminal disturbance. (Veraguth.)

PLATE IX



Superposition of the Peripheral Sensory Distribution (in red) and of the Nuclear (in black) of the Trigeminus.

V1-5, shows gradual involvement of the area supplied by the trigeminus in a case of syringobulbia; V1, V2, V3, V4, V5, the three branches of the trigeminus; C1-v, distribution of the cervical nerves; Gg, the territory of the geniculate ganglion (Hunt's zoster zone of the facial). (Dejerine.)

Unilateral myosis may also point to trigeminal irritation here. Related dryness of the mucous membranes of the nose, lips and cheeks, with anesthesiæ, usually point to peripheral disease of the second branch, while taste impairment of the anterior two-thirds of the tongue may be, but not invariably, associated with lesions of both second and third branches.

In root lesions, the epicritic loss is usually less than the protopathic loss, while the reverse is usually true for peripheral lesions. Pontine lesions show a more general loss of epicritic sensibility on the side of the lesion with mono- or hemi-hypesthesiæ or anesthesiæ on the opposite side of the body, while thalamic lesions are associated often with anesthesia and analgesia to pin prick, central pain and affective over-response.

Trophic disturbances, usually due to peripheral disease (?), cause changes in the gums and mucous membranes, ulcerations, herpetic eruptions. Corneal ulceration and loosening of the teeth are often present, but whether trophic or not is not certain.

Dissociation of pain and temperature from epicritic touch sensibility may take place in the trigeminal distribution. For lack of space here a complete analysis of sensibility disturbances of the trigeminal should be sought in special monographs. (See Lewandowsky, *Handbuch der Neurologie*, for complete literature—1910–1912.)

Progressive Facial Hemiatrophy.—

This rare condition, which shows at its onset a gradual thinning, with wrinkling of the skin about the orbit or jaws, with later progressive atrophy of the bones, cartilages and muscles, also of the tongue and soft palate, without sensory signs or reaction of degeneration is at times a result of peripheral or pontine (nuclear) disease of the fifth nerve.



FIG. 103.—Syringomyelia, beginning as hemifacial atrophy, then developing "Morvan's disease," and finally showing classical syringomyelic end lesions. (Hammond.)

DISEASES OF THE FACIAL NERVE.

Seventh Nerve.—The seventh nerve is a mixed nerve. The cortical origin of the motor neurone occupies the lower third of the precentral convolution, from here the fibers pass through the knee of the internal capsule, through the middle third of the peduncle and make their first junction (possibly by means of intercalated neurones) with the

homo- and contralateral seventh nerve nuclei in the tegmentum of the pons, just ventrolateral to the abducens nerve nucleus. From these nuclei, four in number are usually described, the second motor

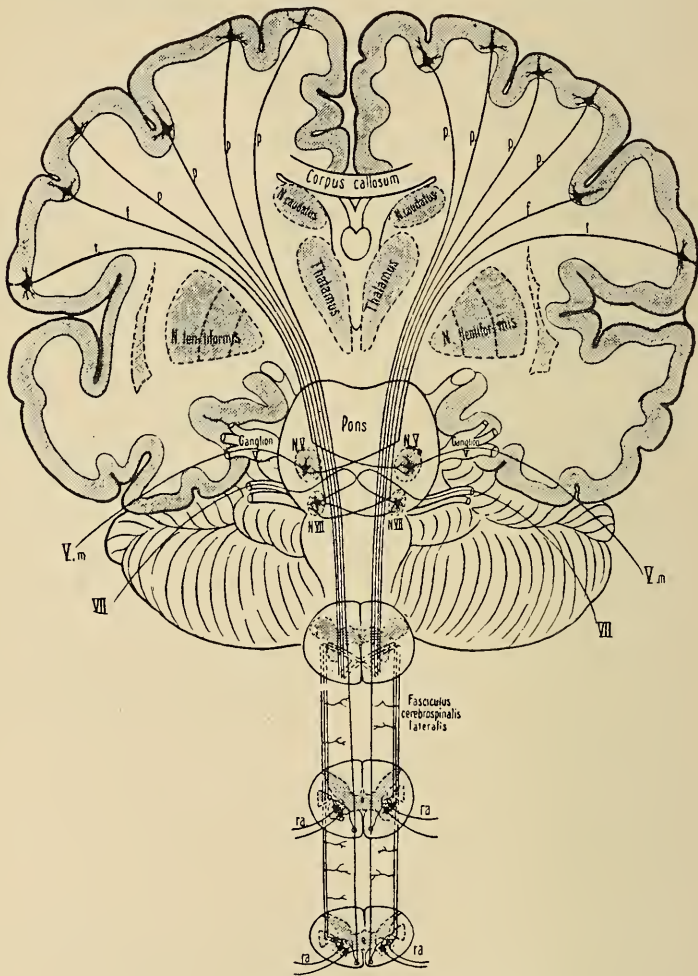


FIG. 104—General scheme of the course of the pyramidal fibers and the central paths of the motor trigeminal and facial. *Vm.*, under portion of the trigeminal; *VII*, root fibers of the facial; *f*, central facial fibers; *NV.*, motor fifth nucleus; *NVII*, facial nucleus; *p*, corticospinal fibers, lateral and anterior; *ra*, anterior roots; *t*, central tract of the motor fifth. (Bechterew.)

neurone fibers make a dorsal upward curve (genu facialis) around the abducens nucleus, then pass ventrally and emerge at the posterior border of the pons, lateral to the olive, where they lie in close relation to the fifth and eighth nerves in the cerebellopontine angles. They

are finally distributed (three ventral nuclei) to the muscles of expression of the face, to the muscles of the external ear, and of the stapedius, the posterior belly of the digastric and the stylohyoid. The frontalis, corrugator supercili, and orbicularis palpebrarum are innervated by fibers coming from the dorsal group.

In its peripheral distribution the nerve passes through the facial canal in the temporal bone (acqueduct of Fallopius), coming into intimate relations with other cranial nerves, eighth, pars intermedia,

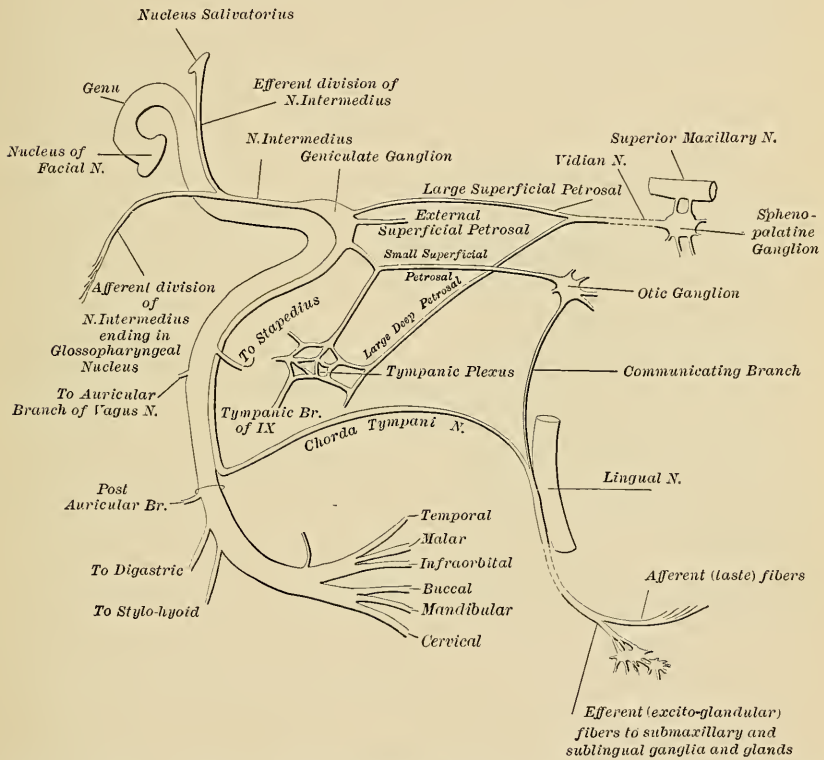


FIG. 105.—Plan of the facial and intermedius nerves and their communications with other nerves. (Gray.)

and also forming sympathetic nerve associations of more than usual complexity, a study of which is of value in the local diagnosis of lesions of this nerve and contiguous parts. (See Fig. 104.)

The anatomy of the possible sensory portion of the nerve has not been definitely homologized. The comparative studies of Herrick, Johnson fail as yet to show sensory components in forms higher than the amphibia.¹

¹ Jour. Comp. Neurol, 1914.

By some its chief ganglion is considered to be the geniculate, which is thought to contain the efferent fibers from the receptors located in the auricle of the ear, the floor of the external auditory canal, the tympanum, and from certain soft parts of the internal ear. The nerve of Wrisberg is considered to be the sensory portion of the nerve. Hunt's syndrome is a clinical picture thought to uphold this interpretation. Again the nerve of Wrisberg has been described as a mixed (thirteenth) nerve. From the geniculate two branches are described, one the chorda tympani, and a second entering the tract of the nucleus solitarius to form part of the gustatory path.

Cortical Palsies.—Cortical or capsular facial palsy is usually unilateral, and most characteristically affects only the lower facial distribution. The face is drawn to the sound side, the angle of the mouth

droops and the nasolabial fold is flattened, but the eyes can be closed and the forehead wrinkled. There is paresis or paralysis of the lower muscles varying with the severity of the lesion. The soft palate may show palsy, pulling to the paralyzed side on phonation. Babinski further described a loss of the contractions of the platysma of the affected side on forcing the mouth open against slight resistance.

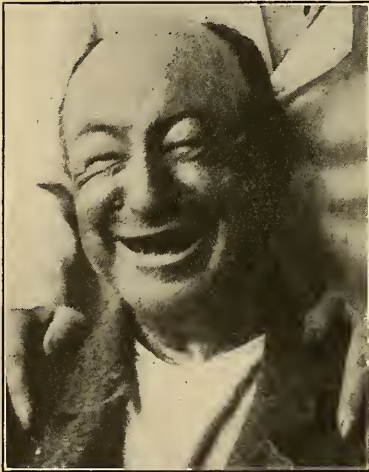


FIG. 106.—Pseudobulbar palsy.
(Tilney.)

In certain widespread cortical neurone palsies, however, the upper branches may be involved, with narrowing (at times widening) of the palpebral fissure, and a drooping of the outer angle of the eyebrows on the affected side. Pontine syndromes frequently show these signs. See Midbrain Section.

Apraxia of the facial musculature is met with in cortical, or corpus callosum lesions. Here the patient loses the power to make proper mimetic movements. He may not be able to close the eye on the paralyzed side, independently of the other. Furthermore, in cortical neurone palsy the tongue protrudes to the paralyzed side, or may be unable of protrusion at all. Speech disturbances are frequent.

In cortical facial monoplegias—or hemiplegias with facial involvement—there are no atrophies, the electrical reactions are not involved, and secretory and taste modifications are absent.

Cortical and subcortical irritation may give rise to facial convulsive movements—spontaneous laughing or crying movements.

The facial mimetic movements of purely psychogenic origin, tics, etc., are numerous and often closely resemble choreic movements.

Pontine Facial Lesions.—Here the nuclei of the peripheral neurone being involved one expects to find all of the branches affected, but inasmuch as there are different groups of nuclei, occasionally, as in poliomyelitis for example, certain muscles are involved and others are not. A general lesion here will cause a total palsy of the muscles with atrophy and loss of electrical reactions. There are no changes in taste, nor hearing in the pure nuclear cases.

Lesions here are apt also to involve the third nerve, also the pyramidal tract fibers, and the sensory fibers of the fillet, hence a variety of hemiplegic or hemianesthetic syndromes—crossed or lower alternate hemiplegias. (See Section on Midbrain.)

Supranuclear pontine lesions may rarely be double (Pseudobulbar palsy types).

Peripheral Facial Palsies.—Bell's Palsy more properly speaking. Here a variety of syndromes may occur depending on the exact site of the lesion. These may be conveniently divided into five syndromes: (1) Most peripheral, due to disease or pressure at or outside of the stylomastoid foramen. This results in a complete paralysis of the muscles of the side of the face. At rest the asymmetry is marked in proportion to the severity of the palsy—all grades are found. The muscles of the forehead cannot be contracted horizontally or vertically, the eye remains partly or widely open on attempts at closure, closing at night in sleep; the nasal orifice is narrowed, the nasolabial fold is obliterated, the angle of the mouth droops and shows the teeth, and there is pulling of the mouth to the sound side. Puffing the cheek is impossible, holding food and saliva are difficult, and on attempting to whistle the air comes out on the paralyzed side. Tears run down the cheek but the reddening of the eye is secondary. There is less sweating on the paralyzed side. Pressure-pain sensibility is unimpaired. The palate and tongue may be apparently involved, but careful scrutiny shows otherwise. Reaction of degeneration sets in as a rule in the severe cases. Slight speech disturbance is apparent, especially in the beginning, and is very marked with a (rarely occurring) double peripheral palsy (see Figs. 107, 108, 109 and 110).

These palsies are due to trauma or pressure from a tumor, possibly a perichondritis of or swelling about the stylomastoid foramen (called rheumatic or refrigeration palsy). The effect of cold upon the facial nerve itself, *i. e.*, by exposure in riding with one side exposed to open windows, etc., looms large in statistical enquiries.

2. Just within the stylomastoid foramen, and within the Fallopian canal, the chorda tympani travels with the facial nerve. A lesion here will cause all of the symptoms just enumerated, and in addition there will be an impairment or loss of taste of the anterior two-thirds of the tongue. There will also be a diminution of the salivary secretions. Pressure-pain sensibility is not impaired.

3. If to this last syndrome hyperacusis and tinnitus alone be added the lesion is slightly further back in the canal involving the branch given off to the stapedius muscle (see Fig. 104).

4. Lesions lying between the geniculate and the stapedius within or at the internal entrance to the Fallopian canal, cause a bewildering variety of additional symptoms, whose exact anatomical relations are still somewhat obscure.

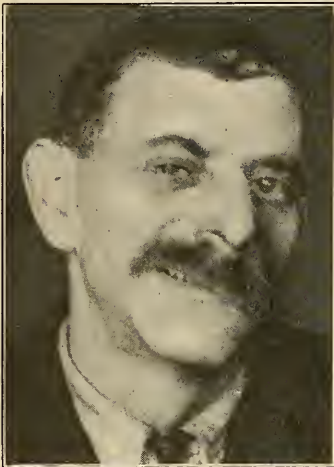


FIG. 107.—Facial palsy. Smiling.



FIG. 108.—Facial palsy. Whistling.

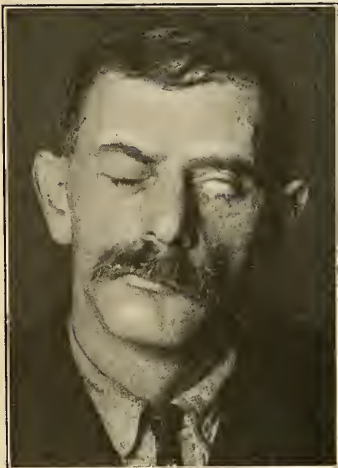


FIG. 109.—Facial palsy. Closing the eyes.

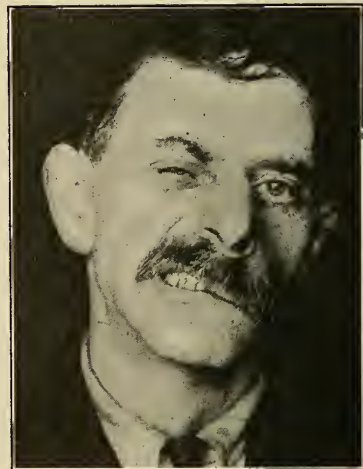


FIG. 110.—Facial palsy. Showing the teeth.

(a) *Geniculate Syndrome. Hunt's Syndrome.*¹—Here one meets with a herpes of the auricle and the external auditory canal. This,

¹ J. Ramsey Hunt, *Journ. of Nerv. and Ment. Dis.*, 1909. Kidd, *Rev. Neur. Psychiatry*, September, 1914.

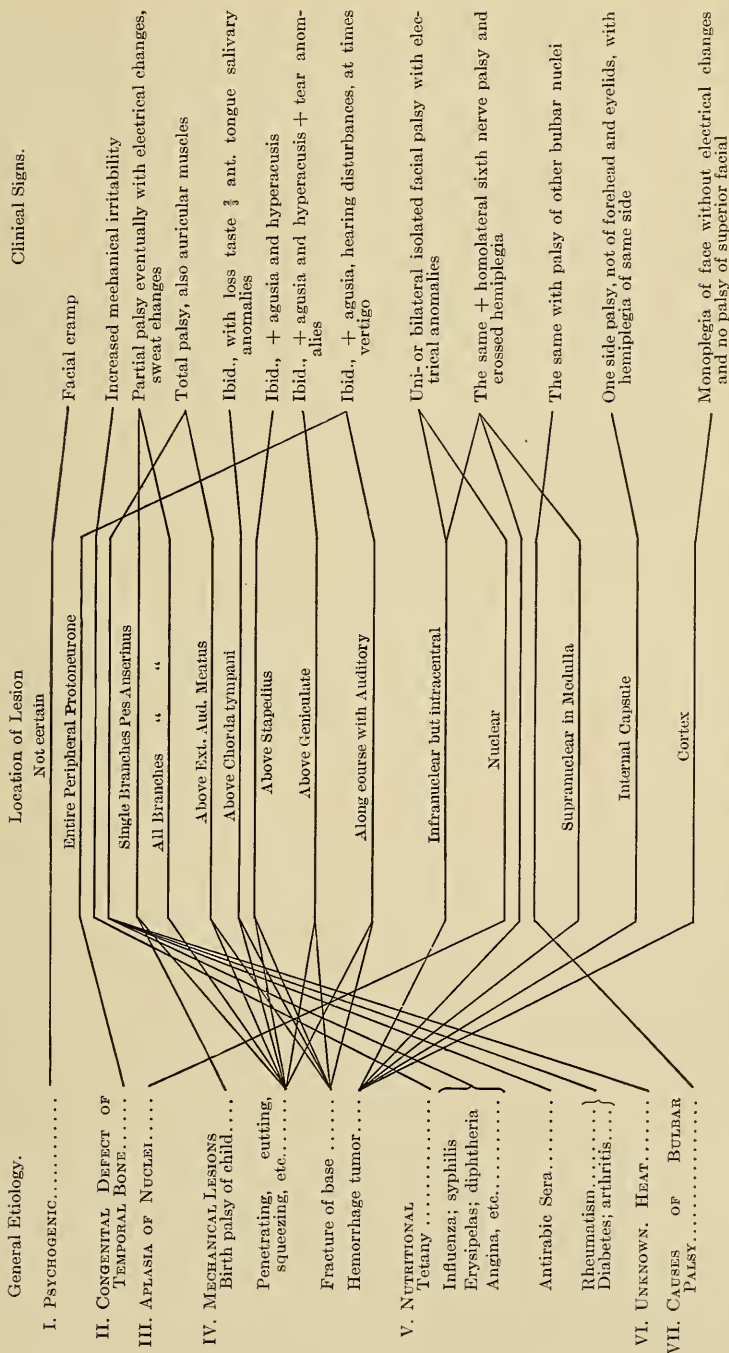


FIG. 111.—Summary of the chief seventh nerve syndromes. (Veraguth.)

according to Hunt, is the zoster zone of the geniculate. Extension of the inflammation, or pressure, causes a facial palsy plus the herpes. In a more extensive process auditory symptoms, tinnitus, diminution or loss of hearing are added. In rare instances, from involvement of the vestibularis, nausea, vomiting, nystagmus and dizziness are present. The chief causing lesion is an inflammation of the geniculate ganglion. Occasionally occipitocollaris herpes is an associated phenomenon. Severe otalgias with or without tympanic herpes are also at times the expression of a geniculate involvement.

Facial palsy of non-geniculate origin from lesions in the same region may or may not be accompanied by loss of hearing. The chief additional diagnostic feature of lesions here is the lowering of the threshold of deep sensibility (Maloney).

Lesions of the seventh nerve at its emergence from the pons usually implicate other structures, notably the fifth or eighth nerves, at times the sixth, eleventh, twelfth. The facial palsy is of the peripheral type with no loss of taste, changes in the secretions, or suppression of the lachrymal secretions. Basal symptoms such as anorexia, nausea, headache, disk changes are often present. The chief pathological processes are basal meningitis, usually syphilitic, or tumor formation.

DISEASES IN THE AUDITORY AND VESTIBULAR PATHWAYS.

The Eighth Pair.—The eighth cranial nerve is in reality two separate nerves, with distinctly different structures, pathways and functions. It is not a single nerve with two parts. The two nerves are the cochlear or auditory proper, and the vestibular—a portion of the cerebellar apparatus.

The former handles sounds, the latter serves to orient the body in space. Their chief receptors lie closely related in the sphenoid bone. By reason of this close topographical relationship infections of the middle ear are apt to involve both structures, and by reason of the close associations with intracranial structures, brain involvements, meningitis, abscess, etc., may result. Their central stations are wide apart in the temporal cortex and cerebellum respectively.

Auditory Nerve.—The receptors for sound stimuli lie in the organ of Corti in the cochlea. They respond to sound stimuli of 11 octaves, *i. e.*, from 10 to 7840 double vibrations. Ordinary conversational or musical sounds usually lie within 16 to 4032 vibrations. Space orientation through sound is purely associative. The chief avenues for sound conduction to the receptors is the auditory canal, but sound conduction is also possible by way of the bones, chiefly of the skull, in part by other bony structures. From the cochlea the branches coalesce to form the acoustic nerve which, passing in the auditory canal with the facial, enters the medulla at about the cerebellopontile angle. The sensory ganglion is the tuberculum acousticum. The further course of the pathways is illustrated in Figs. 112, 113, also see Plate VIII.

The chief disturbances of the auditory nerve are: (1) various forms of deafness. These vary considerably and may be absolute or partial. Certain tones may be cut out and not others, upper or lower tones,

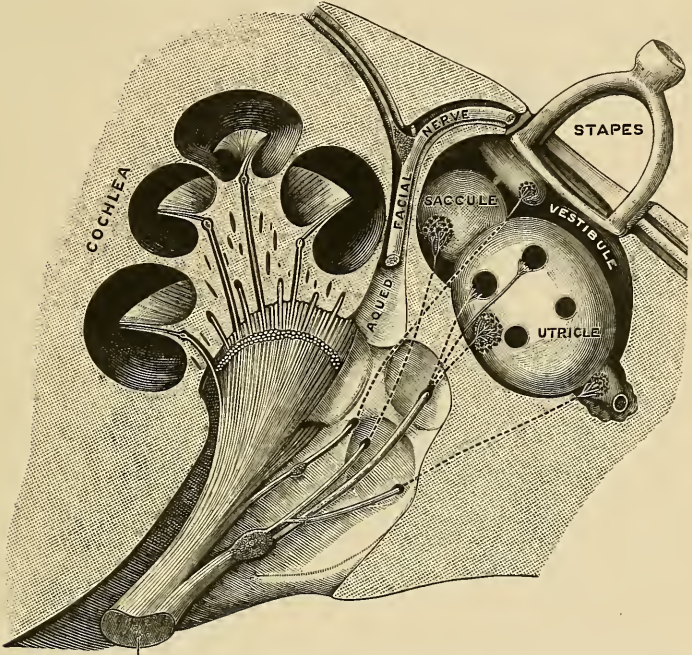


FIG. 112.—Distribution of the acoustic nerve. (Semidiagrammatic.) (Testut.)

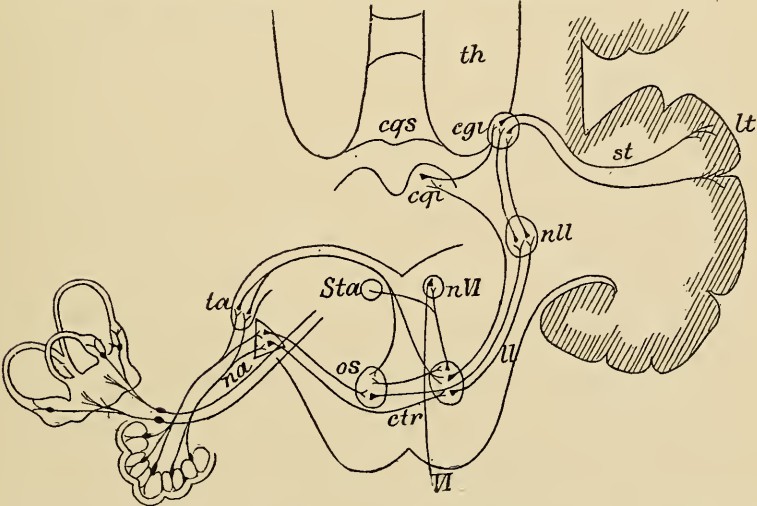


FIG. 113.—Central acoustic paths. *na*, anterior acoustic nucleus; *ta*, acoustic tubercle; *Sta*, acoustic stria; *os*, superior olive; *ctr*, trapezoid body; *nVI*, abducens nucleus; *nll*, lateral lemniscus; *cgq*, *cgq*, posterior and anterior corpora quadrigemina; *cgq*, external geniculate body; *st*, acoustic path to cortex; *lt*, temporal lobes; *th*, thalamus. (Bechterew.)

sometimes intermediate tones drop out (hearing scotomata, analogous to optic scotomata, are not infrequent in hysterical reactions, dementia precox, in multiple sclerosis, paresis, tabes, etc.). These anomalies of hearing are chiefly peripheral, either in the primary receptors or occasionally in the ganglion. Paracusia (buzzing, whistling, crackling), are also peripheral for the most part, but may also be central as in psychotic or psychoneurotic syndromes.

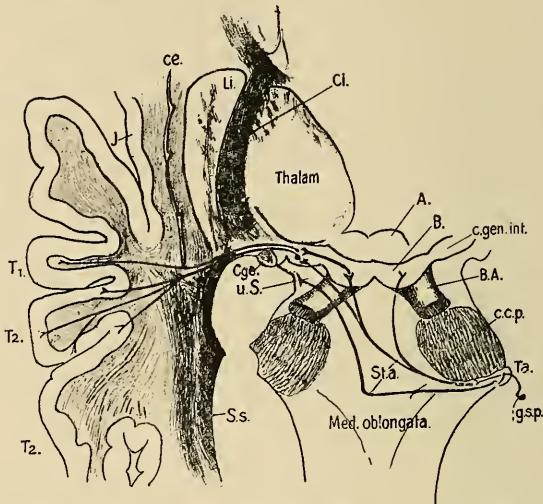


FIG. 114.—Scheme of the central acoustic pathways. *T*₁, *T*₂, first and second temporal lobes; *J*, island of Reil; *ce*, claustrum; *Li*, lenticular nucleus; *Cl*, internal capsule; *Thal.*, thalamus; *u.S.*, median lemniscus; *S.s.*, optic radiations; hint. *V.L.*, posterior quadrigemina; *B.a.*, middle cerebellar peduncle; *c.c.p.*, crus cerebelli ad pontem; *c. gen. int.*, internal geniculate; *Cge.*, external geniculate; *Sta.*, striæ acoustical; *gsp*, ganglion spirale; *Ta.*, acoustic tubercle. (v. Monakow.)

Complete deafness from medullary lesions is rare for anatomical reasons already indicated.

Cortical or sub-cortical involvement of the auditory pathways cause various forms of amnesic aphasia. (See under Speech Mechanism.)

CHART FOR DIFFERENTIATIONS OF PERIPHERAL AND CENTRAL LESIONS.

	Disease of sound-conducting apparatus.	Disease of sound-perception apparatus.
Speech test.	Deep tones heard worse than higher ones.	Deep tones heard worse or better than higher ones.
Air Conduction for forks C. cc ¹ , c ² , c ³ , c ⁴ and for Galton's whistle.	Deep tones heard worse than higher ones. Lower tone threshold raised.	High tones heard worse than deep ones. Upper tone threshold lowered.
Weber (c ¹).	Median or localized in worse ear.	Median or localized in better ear.
Schwabach (c ¹).	Lengthened.	Rarely normal, mostly shortened.
Rinné (C. cc ¹ , c ²).	Negative. According to grade of retardation only for C. or for C. and the higher tones to c ² .	Positive.

Vestibular.—It has been established, almost beyond question, that the labyrinth is the chief organ of the body connected with the receiving of impressions of its position in space, particularly for the

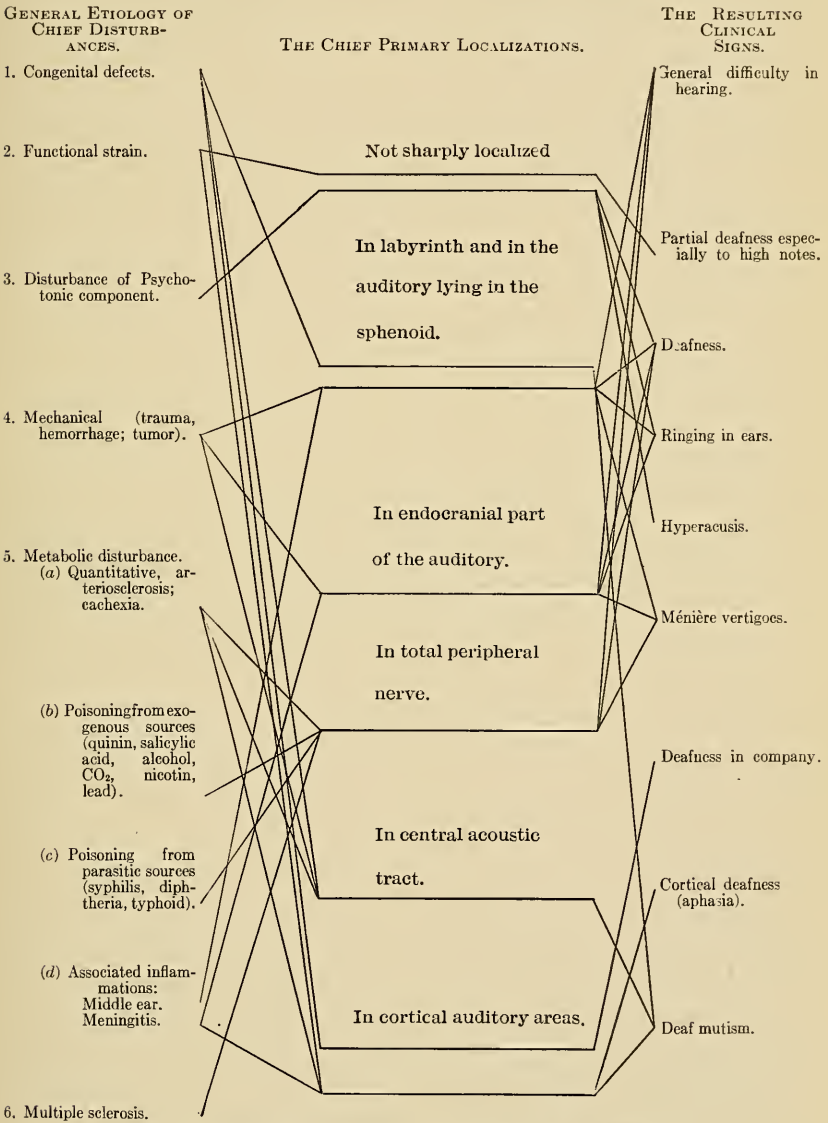


FIG. 115.—Summary of auditory disturbances. (Veraguth.)

head. The meaning for the human body of the physical laws of gravity is its chief concern. It is adapted to the mechanical stimuli of gravity, acting largely through the otolith organ, which react to

changes in the incidence and degree of pressure upon its sensory end organs, due to changes in the specific gravity of its surrounding fluids; and also through the semicircular canals which react to changes in position in the three planes of space. The slightest change of the body in space is felt by this apparatus, and in the healthy central nervous system any such change is automatically reacted to by appropriate motor response.

This motor response, however, is a complicated mechanism, and all of its elements are not thoroughly analyzed. One of its parts is that of a reflex muscular tonus, by which the ordinary posture of the body is maintained. It is this function that has entitled it to the appellation of the labyrinthine tonus. Sherrington¹ has analyzed the complicated interrelations between the proprioceptors of the limbs, muscles, joints, etc., which carry impressions of movements, strains, tensions, etc., and the receptors in the labyrinth. It is impossible to enter into them here. Suffice it to say that the result is the reflex maintenance of the posture of the body, including the compensatory reflexes of the head, and those muscles of the head capable of changing the sense of consciousness of position, particularly the muscles of the eyeballs.

The labyrinth belongs to a series of organs that work in response to gravity. It is a part of a great system of connections—which Sherrington has designated as the proprioceptive system—which gives the animal, human as well as others, a definite attitude toward the external world of space. It is the most important of these organs. It is connected in a system with other nervous structures performing their part in the same general function, and each segment of the body is caught up in the chain of connections from the lower end of the spinal cord to the frontal area of the cortex.

This whole complicated system of end organs, fiber connections, long and short fiber tracts, has its chief center, just as every other reflex system has its center. The chief center or head ganglion of this whole proprioceptive system is the cerebellum. The cerebellar connections of the vestibular system, the vestibulospinal, vestibulobulbar, vestibulocerebellar, and, finally, the cerebellorubro-cortical components which carry those fibers whose functioning is recognized in the consciousness of space relations, are now fairly well known, not in their entirety, but in their main tracts and connections. Hence, disease or disorder which shows any perturbation of the function of orientation in space may be more or less accurately localized along the fiber tracts, carrying the necessary impulses underlying these functions, and an appropriate therapy adopted (see Plates VIII, X, and XI).

Nervus Vestibularis.—The fibers of the median acoustic root (Lewandowsky—mixed) constitute the central prolongation of the bipolar ganglion cells which make up the vestibular or Scarpa's ganglion. The peripheral prolongations of the cells originate in the walls of the

¹ The Integrative Action of the Nervous System.

semicircular canals. The thick bundle of the median root pushes its way between the spinal trigeminus root and the corpus restiform (inferior cerebellar peduncle) lying at first close to the median edge of the spinal accessory nucleus, and reaches dorsally like the tines of a fork toward the end nuclei. These end nuclei of the vestibular are the triangular and the large cell nucleus, which latter consists of: (1) spinal acoustic; (2) Deiters' and (3) Bechterew's nucleus. Cajal describes also a crossed root of the vestibularis whose bundles can be traced along the dorsal border of the spinal trigeminus root through the raphe and can be followed to the other side (Bechterew).

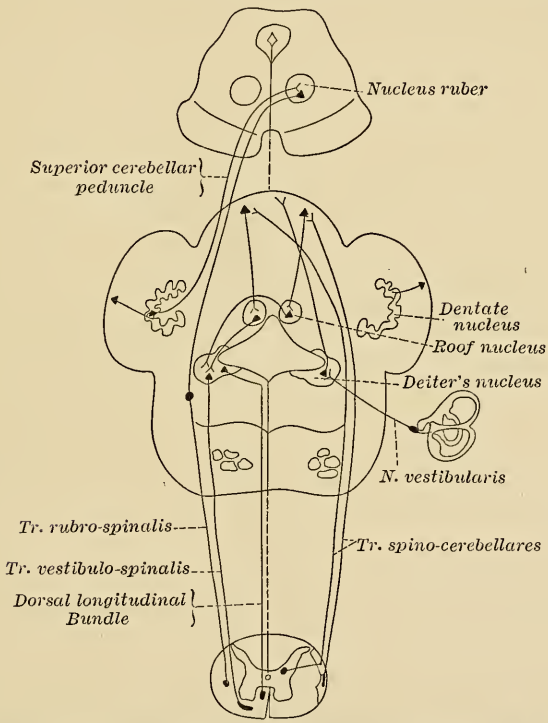


FIG. 116.—Diagram to illustrate the spinal connections of the cerebellum. On the right the afferent tracts are represented, on the left the efferent cerebellar tracts.

Of the connections of the end nuclei of the vestibularis those of the cerebellum are the plainest. Strong, somewhat swollen bundles of nerve fibers go from the Deiter and Bechterew nuclei dorsally in the cerebellum. Fibers from the nuclei triangularis also join them. The acoustic cerebellar tract lies on the medial side of the inferior cerebellar peduncle, in the medial lateral portion from the superior cerebellar peduncle, in which a portion also goes. The majority of the bundles go to the cerebellar worm and end, mostly crossed, in the

nuclei of the roof (tectalis), probably also in the nucleus globosus and nucleus emboliformis.

Within the superior cerebellar peduncle portions it may be said that, according to Bechterew and Flechsig, the Bechterew nuclei are connected by means of commissural fibers which pass out with the superior cerebellar peduncle from the cerebellum and bend arcwise in the posterior angle of the crossing of the superior cerebellar peduncles.

Vestibular Vertigoes.—At one time loosely grouped together under the name Ménière's disease, the analyses of later years have shown a great variety of these affections depending upon the anatomical sites of the lesions. One must distinguish between:

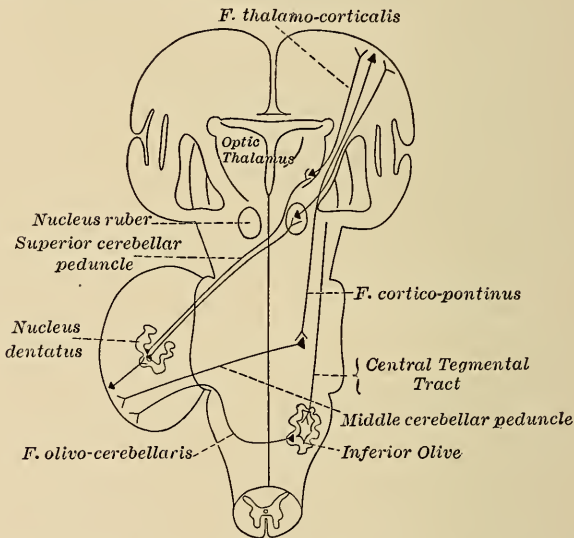


FIG. 117.—Diagram to illustrate the afferent and efferent connections of the cerebellum with the forebrain.

1. Disease of the peripheral end organ, (*a*) partial, or (*b*) complete; these are the vertigoes of partial or complete labyrinthine disease.

2. Disease of the first neurone, (*a*) paresis, (*b*) paralysis of the vestibularis.

3. Disease of the primary end nuclei in the medulla and of Deiters' nucleus. The latter gives a special symptomatology termed Bonnier's syndrome.

4. Disease in the region of the posterior longitudinal bundle associated with eye movement vertigoes.

5. Disease of the nuclear region of the eye muscles in the corpora quadrigemina.

6. Disease of the pontine eye nuclei.

7. Disease of central eye paths.

8. Disease of cerebellum.

In disease of all these regions vertigoes are to be expected by implication of the vestibular nerve; the character of the accompanying phenomena, especially the nystagmus, determines the location.

In partial or circumscribed disturbance of the vestibular end organs in the labyrinth the vertigo is associated with nystagmus movements. The nystagmus is spontaneous and shows a long slow, due to the vestibular, and a quick return movement due to the tegmental nuclei, the direction of the quick movement naming the nystagmus. Vestibular nystagmus usually increases when the eyes are directed in the direction of the quick movement, and usually diminishes or ceases on looking in the opposite direction. There is almost always a combination of horizontal and of rotary nystagmus. Barany states that every other form of spontaneous nystagmus is of intracranial origin. If the nystagmus movement is rotary and horizontal it must be determined whether it is peripheral or central. A peripheral nystagmus to the right should show on caloric, pressure, and rotation tests that the right vestibule is functionally active. Should such tests show an inactive right vestibular then the nystagmus must be of central origin. If the right vestibular is active, then continued observation of the nystagmus will alone determine. Should the nystagmus continue uninterruptedly for twenty-four hours or more it is of intracranial origin. If it lasts a shorter interval and is uninterrupted by quiet intervals, it may be either peripheral or central. When there is also nystagmus of the well side, which lasts about two weeks, gradually decreasing, then a peripheral disturbance seems certain. Intracranial nystagmus is not so apt to diminish.

The Ménière-like attacks are either mild or marked. Buzzing in the ears is rare in mild attacks. There is no impairment of hearing. In the severer attacks there is little buzzing, but hearing is apt to be impaired. In free intervals the nystagmus diminishes or disappears, the Barany caloric reaction is diminished on the affected side.

Total destruction may be acute or chronic; the latter may show no symptoms. The former sets in with violent vertigo, nausea, vomiting. There is marked horizontal and rotary nystagmus of the sound side. The slightest movement of the head increases the vertigo and nystagmus during the first forty-eight hours; the latter gradually disappears in three to four weeks. There is marked loss of coördination, with tendency to fall to one or the other side. After the period of quiescence of the nystagmus, caloric and rotation tests show the defective function. The galvanic reaction is not usually affected.

Disease of the vestibular nerve, usually due to tumor of base (acoustic, cerebellopontine angle), leads to similar reactions. Here, however, there seems to be a difference in that Neumann has found that the galvanic reaction is reduced or lost, according to a partial or complete destruction of the vestibular ganglion. Other cranial nerves are here involved as a rule. The cochlearis is frequently involved. Complete deafness does not result. The trigeminus is also

often involved and pain, paresthesiæ, or motor defects appear. Cerebellar symptoms may also complicate the picture. The nystagmus is apt to continue in intensity with tumors, and may be on the sound as well as the affected side.

Involvement of the nuclei (encephalitis, abscess, syphilis, tumor) brings about similar attacks of nausea, vomiting, vertigo, and nystagmus. The symptoms continue and increase, as a rule, beyond the three weeks ordinarily seen in labyrinthine disease.

The method of continuous observation aids in locating the diseased focus.

Bonnier's Syndrome, due to implication of Deiters' nucleus and contiguous structures, usually causes a marked attack of nausea, vomiting, vertigo, and nystagmus with buzzing in ears and deafness (Ménière's syndrome), with irradiations to the ninth and tenth nerves causing anxiety, tachycardia, and hemiplegic weakness. The trigeminal and oculomotor are also apt to be involved. Bonnier has also described peculiar somnolent attacks accompanying his syndrome. Little can be done for these cases unless the focus is of syphilitic origin.

Here vertigo and nystagmus are associated in various ways, but the vertigo disappears on closing the eyes. Here forced movements conjugate deviations, and various skew deviations afford a clew to diagnosis. Caloric and other tests determine the integrity of the labyrinthine functions.

Cerebellar vertigoes have a number of special features. So far as the vertigo is concerned they may not be separable from the labyrinthine or vestibular vertigoes. Hearing symptoms are usually absent. The nystagmus is less apt to be horizontal and rotary, but may be up or down or oblique, and is usually directed toward the affected side.

There are usually also symptoms of a tumbling gait toward the side of the lesion; there is asynergia and usually adiadokokinesia. No real distinction as to the side of the lesion affected can be gained from the fact as to the subjective or objective motion of the objects during a vertiginous attack. Closure of the eyes has no marked affect upon the vertigo, nor upon the gait. Caloric and other tests determine a normal labyrinth.

Treatment.—Here there comes into consideration the surgery of the ear and the surgery of the cerebellum and the cerebellopontine angle. The ear specialist should treat the labyrinthine cases, not the neurologist. Rest in bed, quinin, and the usual medical treatment which shuts one's eyes to the danger of a suppurative labyrinthitis, brain abscess, etc., is folly.

In the apoplectic form of Ménière's syndrome (hemorrhagic labyrinthitis) often mistaken for a cerebral, or cerebellar hemorrhage, the patient must be kept absolutely quiet, the eyes should be kept closed, the room darkened, and all noises excluded as far as possible—telephone, house bell, etc., shut off. Ice should be applied to the mastoid. Leeches are at times of value. The continuous vomiting

may be in part relieved by swallowing cracked ice. Surgical interference may be called for.

In syphilitic cases mercurial injections, salvarsan, or inunctions are called for. It may be noted that the acute labyrinthine disturbance which has been known to occur after the use of salvarsan is probably due to the syphilis and not to the arsenic (Benario).

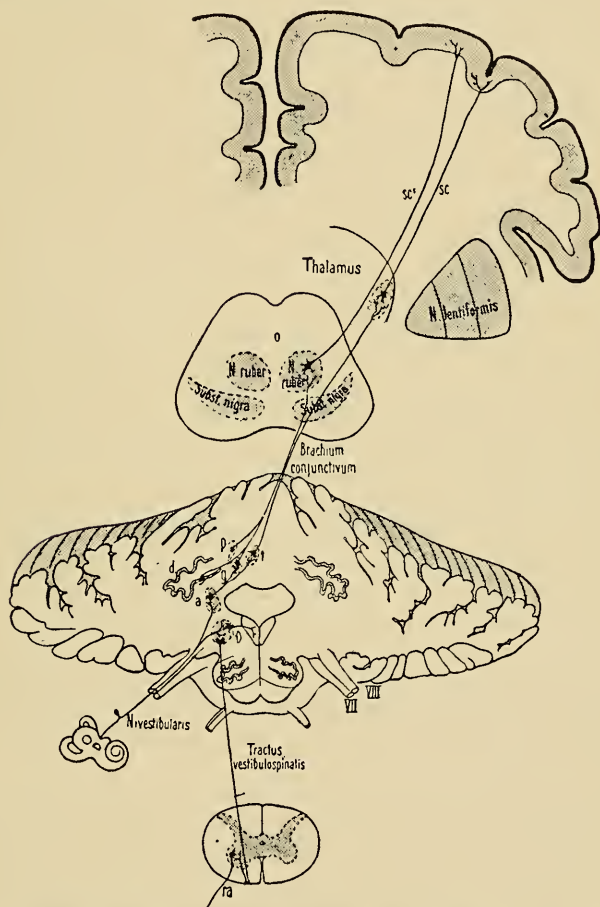


FIG. 118.—General scheme of the cerebral paths of the vestibular. VII, facial nerve; VIII, acoustic nerve; *a*, Bechterew's nucleus; *D*, Deiters' nucleus; *d*, dentate nucleus; *g*, nucleus globosus; *p*, nucleus emboliformis; *ra*, anterior root fibers; *sc*, *sc*¹, subcortical fibers of the red nucleus and of the thalamus to the cortex; *t*, tegmental nucleus. (Bechterew.)

Treatment of Seasickness.—Seasickness is a special form of disturbance of the labyrinth. As the stomach has little or nothing to do with seasickness, diet has little or no effect upon this malady, and the ocean traveller need pay no more attention to the question of food than that dictated by common sense. Eat one must, for there is

nothing worse than continued retching on an empty stomach. The oftener one vomits, the oftener one should eat or take liquid food.

If one is predisposed to seasickness, morning walks on deck before breakfast should be dispensed with. One should try to breakfast immediately upon rising, and a little fruit or other light food eaten before rising may be found helpful. What one eats is of small moment; the great thing is to eat; but one should avoid food which one does not like. There is no potency in any particular food in the prevention of seasickness.

Nor is alcohol of any use, unless enough be taken to anesthetize the stomach. Indeed it is far more likely to prove an irritant, especially if the individual be unaccustomed to its use. The much vaunted value of champagne is a myth kept alive by the suggestion of dealers.

One should not go to dinner until it is just about to be served, thus avoiding the discomfort of waiting in a stuffy and perhaps overheated dining room. When the meal is over it is well to lie down, rather than go for a tramp on deck in the hope that it will aid digestion.

Warm clothing and wraps should be taken on a sea voyage even in very warm weather. Cold, damp, and foggy weather is apt to be met with on the ocean at any time of the year, and the consequent chilliness, added to that of an unstable vasomotor control, through the labyrinth, is a great cause of discomfort, which may be removed or alleviated by wearing warm garments.

If the sea be at all rough and the motion of the vessel appreciable, the sensitive traveller should lie down at once, as it is easier to accustom one's self to the labyrinthine hyperstimulation in a recumbent position, especially if one adopts the position in which the motion is least felt in the superior canals, *i. e.*, one should lie down as flat as possible—semireclining does not so place the plane of the semicircular canal as to cause the least possible flow of fluid within it. One flat pillow is all that one should use since half sitting up is as bad as standing up. Chairs should be shifted, if possible, according to the pitch or roll of the vessel.

As soon as the first symptoms of seasickness are felt the patient should lie down, if possible, on deck. As eye movements aid in causing seasickness, one should close the eyes if there is much motion of the ship, so as to relieve the muscles from the constant adjustment necessary in watching a wavering horizon, and in very bright weather, colored glasses should be worn to subdue the glare.

Reading continuously is rather to be avoided, therefore books should be chosen which will allow one to close one's eyes and meditate. Cards or other games which divert the attention are very helpful.

In making choice of rooms, one should give preference to those in the middle of the boat where the motion is less felt. To overcome the smells and stuffiness incident to ocean travel, one should keep plenty of air circulating in one's state room, unmindful of drafts, which are of much less consequence than one is prone to think them.

Eating fruits and salads, drinking plenty of liquids, and occasionally taking a pill of aloes, aloes and mastiche, or similar laxative, is generally sufficient to counteract the constipation which is a frequent consequence of the unusual change of habits, especially when one eats very little.

The headache of seasickness is best combated by eating, by coffee, and by small doses of bromids and phenacetin. The widely used headache mixtures incorporating caffen and antipyrin in the elixir of sodium bromid are useful. The sodium salt of veronal in doses of from 8 to 10 grains, given by rectum in suppository, is a very useful remedy in causing sleep and in relieving excessive irritability of the labyrinth.

TABLE OF DIFFERENTIAL DIAGNOSIS OF LABYRINTHINE AND CEREBELLAR DISTURBANCES.

Tests.	I. Circumscribed labyrinthitis.	II. Diffuse labyrinthitis.			III. Diffuse purulent labyrinthitis and cerebellar abscess.	
	Fistula formation.	(a) Serous labyrinthitis.	(b) Acute purulent labyrinthitis	(c) Chronic purulent labyrinthitis.	Type A.	Type B.
Spontaneous nystagmus	May be absent on sound or diseased side	Mostly on sound side also both sides	Rotatory and horizontal strong on the well side	Weak on well side, or on both sides	Strong rotatory and horizontal in sick side	Rotatory and horizontal on well side
Turning nystagmus	Produced both sides same intensity	Both sides with same intensity	Cannot be tested because of excessive reaction	Residual; on well side, normal time; on sick side, shortened	Produced both sides same intensity	sides same intensity
Caloric nystagmus	Diminished or not present	Absent	Absent	Absent	Absent	
Compression Hearing	Present Hears with difficulty or deaf	Absent Hears with difficulty or deaf	Absent Deaf	Absent Deaf	Absent Deaf	
Other symptoms	Vertigo and nystagmus on rapid movement of head, nausea and	Marked vertigo, nausea, fever	Vertigo, nausea, vomiting	Slight vertigo, also slight equilibrium disturbances	Changeable vertigo and equilibrium disturbance; finger test positive, eye grounds positive	
Treatment	Radical operation	No operation	Radical and labyrinthine operation	Radical and labyrinthine operation	Radical, labyrinth and cerebellar operation	Radical and labyrinth operation. If nystagmus continues, cerebellar operation

DISEASES OF THE SPINAL ACCESSORY AND HYPOGLOSSAL NERVES.

Accessorius.—The spinal accessory nerve innervates the sternocleidomastoid and the trapezius. Branches to the vagus are known. Its cortical origin is not definitely localized. The supranuclear pathways pass through the internal capsule to end in part in the medulla near the olive and vagus nuclei, in part in the anterior horns of the six upper cervical "spinal" segments. The combined branch passes

by way of the jugular foramen to its muscle distribution, being combined with sympathetic fibers from the cervical plexus.

Clinical.—Cortical disturbances (first motor neuron) cause irregular and spasmodic actions. These are seen in certain epilepsies, usually conditioned by cerebral syphilis, multiple sclerosis, or other brain disorder.

The various tics (wry-neck, etc.) are mostly of cortical, *i. e.*, psychological origin. They represent compulsion neuroses for the most part. (See Psychoneuroses.)

Treatment of these tics, torticollis, when mental, by peripheral operations is folly.

Nuclear affections of the accessorius are rare.

Peripheral palsies are not infrequent and are due to various injuries, direct, through disease of the base of the skull, syphilis, osteitis. They cause degrees of loss of power to pull the face to one side, with tendency to contraction of the opposite side (*caput obstipum*). Electrical changes, R. D., atrophy, loss of reflexes, are present in the nuclear and peripheral palsies, but are absent in the central palsies or centrally induced torticollis. Trapezius palsy causes the characteristic rotation of the scapula.

Treatment will vary with cause. It can only be emphasized that the surgical treatment of spasmodic torticollis, which in the overwhelming majority of cases is a psychological reaction, usually a compulsion neurosis, is useless. Psychoanalysis and reëducation have been much more valuable.

Hypoglossus.—The hypoglossal nerves, twelfth pair, are the chief motor nerves of the tongue. Through collaterals they also send motor fibers to the sternohyoid and sternothyroid muscles. The chief physiological functions by symmetrical innervation are as follows: The genioglossus muscle moves the tongue forward and down, the hypoglossus muscle moves the tongue back and up. The styloglossus moves the base of the tongue up and back. In a symmetrical innervation—that is, loss on one side—the combined action of these three muscles causes the tongue to deviate *in toto* to the paralyzed side.

Affection of the longitudinal muscle by symmetrical innervation causes shortening of the tongue, either pulling the top of the tongue up or down. By asymmetrical innervation the anterior portion of the tongue is pushed to the paralyzed side. Any loss of the function of the branch which innervates the transverse muscles brings about a narrowing of the tongue, whereas symmetrical affection of the vertical muscles produces a flattening of the tongue. The geniohyoid raises the hyoid bone, pulling it forward when the lower jaw is fixed, or pulls the lower jaw down. When the hyoid bone is fixed the sternohyoid and the thyrohyoid pull on the hyoid bone.

The nucleus of the hypoglossus lies in the lower two-thirds of the medulla, stretching as far down as the pyramidal crossing, ventrally from the central canal to the midline. At least ten to fifteen root

bundles pass from the hypoglossal nuclei between the pyramidal tracts and the olive, and join together for a short distance within the hypoglossal canal, at the orifice of which the hypoglossal vein, which is in

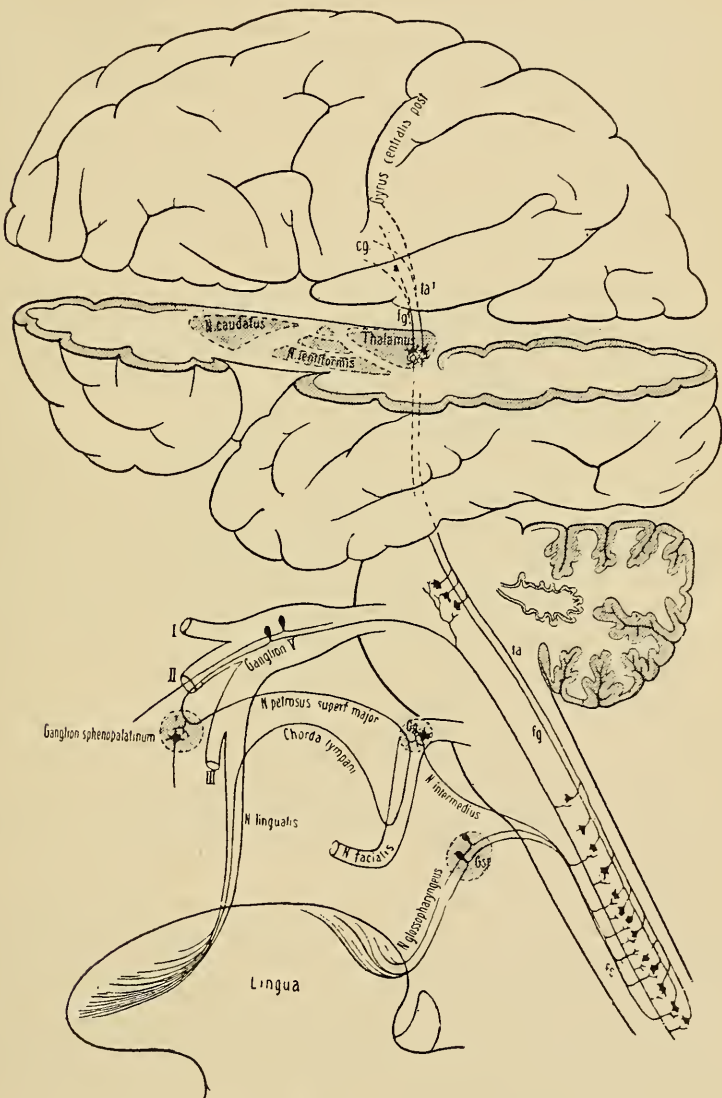


FIG. 119.—Pathways of the taste fibers. *I*, ophthalmic branch of *V*, *II*, maxillary branch; *III*, mandibular branch; *cg*, cortical taste area; *fg*, central ascending taste fibers in median lemniscus; *fg*, subcortical paths; *Gg*, geniculate; *Gsp*, jugular and petrosal ganglia of the glossopharyngeus; *ta'*, subcortical connections of the thalamus with the inferior posterior central gyrus. (Bechterew.)

connection with the occipital sinus, surrounds it. The canal is narrow and short—less than half an inch—lying close to the occipito atlantic articulation, at a place where fracture of the base of the skull is very apt to affect it.

At its exit from the skull the hypoglossal lies median to and dorsal of the vagus and of the internal jugular vein, proceeds laterally, passes beneath the stylohyoid muscle and the posterior belly of the digastric, splits into its various branches, and innervates the muscles already mentioned.

Anastomoses with the ganglion nodosum, superior cervical ganglion, the lingual and first and second cervical nerves take place. A special branch, the ansa hypoglossal is formed by anastomoses of the descending branch and the hypoglossal, and branches from the second and third cervical nerves.

The supranuclear pathways are followed with considerable difficulty. In the cerebral peduncles they lie in the center somewhat more median than the facial; within the internal capsule they lie at the knee. The cortical stations lie in the lower portion of the central convolutions. The supranuclear pathways decussate freely.

Peripheral lesions of the hypoglossal are the result, usually, of mechanical causes, resulting either from fracture of the base of the skull, from tumors, direct injury or tuberculosis, or dislocation of the upper cervical vertebræ. Cerebrospinal syphilis, particularly of long standing, is a not infrequent cause of peripheral palsies, while poisoning from lead, arsenic, alcohol, carbon monoxide may cause peripheral lesions.

Nuclear and supranuclear affections of the hypoglossal are due to hemorrhage within the medulla. Poliomyelitis, tumors, syphilis, and multiple sclerosis—these are the most frequent cause of nuclear or supranuclear lesions of these nerves.

Clinical.—The most frequent lesion of the hypoglossal is unilateral. There is atony of the longitudinal muscles of the paralyzed side, and when the tongue lies quiet in the mouth its apex deviates slightly to the non-paralyzed side. The base of the tongue usually rises higher on the paralyzed side than on the sound side as a result of atony of the hypoglossal muscle.

Movements of the tongue are diminished; it becomes difficult to remove food which lies between the teeth and the cheek, and it becomes difficult for the patient to direct the tongue to the teeth on the paralyzed side. On thrusting the tongue out it deviates to the sound side. Speech disturbances are present, especially the labials and linguals.

In long-standing disease atrophies develop with fibrillary twitchings and tremors and pronounced irregularities, and electrical stimulation shows reaction of degeneration.

In bilateral paralyses speech disturbances are very marked. Chewing and swallowing are rendered difficult, and the movements of the tongue are markedly diminished in all directions.

In hysterical tongue paralyses, which are by no means uncommon,

resistance to passive motion of the tongue is seen. There are no electrical changes and speech disorder is apt to be obvious.

In nuclear palsies, atrophy and fibrillary twitching are marked, the speech disturbances are pronounced, the chief characteristic being what is termed "hot-potato speech." The patient speaks as though he had a hot morsel in his mouth. Reaction of degeneration is also present. Supranuclear palsies, such as occur in hemiplegia, involve the muscles as a whole, cause deviation of the tongue to the paralyzed side, and other signs of hemiplegia are present. Isolated cortical spasm of the hypoglossal may be present.



FIG. 120.—Atrophy of right half of tongue. Cerebral syphilis and injury.

Psychogenetic hypoglossal disturbances are by no means rare. These consist of tongue tics, of constant tongue movements, such as are seen in hysterias, in patients with dementia precox, compulsion neurosis, and in various paranoid types of thinking.

Treatment of hypoglossal disturbances varies according to the cause and is usually purely symptomatic.

Speech Disturbances.—Human expression, articulatory, mimetic, tactile, or by writing, is a highly complicated mechanism. It includes all of those movements resulting from optical, auditory, or tactile contacts by which communication between individuals is brought about for social purposes. Language as it fully develops is, therefore, a tool with which one may cut into reality and utilize the facts of

nature for ultimate adaptation. Thus, all speech disturbances may be divided into those in which the receptive (sensory) side of the pathways are involved and into those in which the productive (or purely motor) part of the arc is implicated.

On the sensory side one finds the gradual accumulation of experience, chiefly with auditory stimuli (with the gradual evolution of speech), symbols (language) which stand in the developing psyche for the images of things, ideas or feeling values. Thought is symbolic action.

Optical stimuli—objects, signs, various glyphs, words, letters—unite to more complex types of expression in writing (psychically developed to conquer spatial limitations). While tactile stimuli are an integral part of language from the more restricted side of tactile reading of blind and tactile speech of the deaf and dumb to

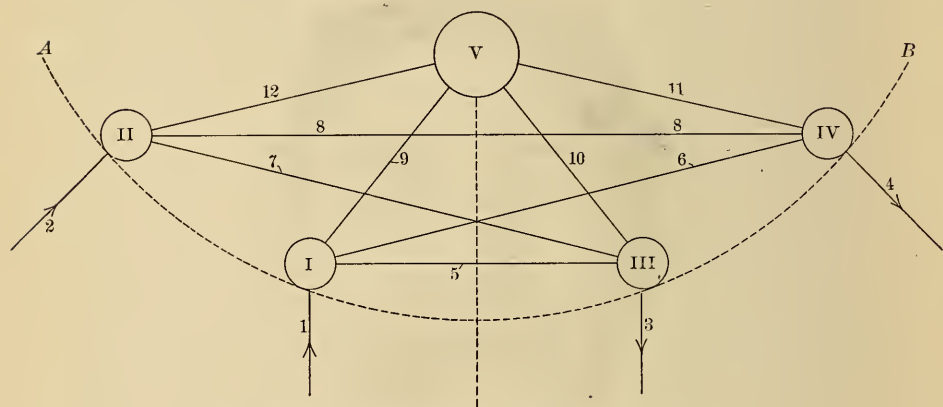


FIG. 121.—Scheme of speech connections. (Veraguth.)

the sensory stimuli of the movements of the muscles and tongue and lips in speech. A complete analysis of the great complex of sensory factors which ultimately find outlet in speech symbolism with its infinite psychical implication is not possible here. It would involve the entire problem of the evolution of civilization and culture.¹

The productive side of the speech mechanism is less complicated. Originally showing itself in the child as a noisy symbol of crying, there is gradually shaped by the libido more and more accurate sounds of expression to meet the needs of hunger and of love. Grunts, laughs, gurgles evolve into more precise formulations, until the rich symbolizations of speech are gained, with all their advantages of precision and efficiency. Here the muscles of the mouth, lips, larynx, chest, abdomen, arms, and pelvis all come into a gradually refining and orderly series of coordinated activities.

¹ Gutzmann. Sprach-Störungen. Various monographs on Aphasia.

Many schemes have been devised to set forth graphically some of the phases of these speech disturbances. One of Veraguth's is here utilized.

Here 1 represents the incoming auditory stimuli (tone, sound, words) with their more or less sharply defined sensorial perceptions gained gradually through experience. They constitute in their totality the various heard components of speech and are constituents of organic, auditory memories. The circle I represents such a psychophysiological combination, rather than an anatomical hearing center, which latter is roughly outlined in the first and second temporal convolutions.

Pathway 2 represents the optic as well as the tactile, and kinesthetic neuron chain which convey to the brain centrals graphic symbols (pictures, diagrams, graphs, letters, etc.). The general assembly place of these is represented by the circle. It represents not an anatomically circumscribed area, but rather a functional capacity, but not unrelated to an optical sensory area in the cuneus and precuneus of the occipital lobes.

Outgoing pathways 3 and 4 represent the motor side of the arcs of expression by all those motions by which the act of articulation with infinite variation, shades, and nuances, and those of graphic representation are carried out. Both pathways are related to cortical, bulbar, spinal localizations, which make functional unity possible as speech and writing (in widest sense). These are symbolized by circles III and IV. All of these are brought together in an enlarged concept (circle V), which symbolizes the heard, read, spoken, or written mode of expression (words, acts, representation, mimic, etc.).

The lines which bind these various centers represent, therefore, a series of possibilities. Whereas an anatomical substratum underlies these possibilities, no attempt will be made to represent them here.

1. Repetition of words without comprehension—Pathway 1, 5, 3.
2. Reading aloud without comprehension—Pathway 2, 7, 3.
3. Writing to dictation—Pathway 1, 6, 4.
4. Writing to dictation without sense—2, 8, 4.
5. When heard word is comprehended—1, 9.
6. When read word is comprehended—2, 12.
7. Spontaneous speech of an idea—10, 3.
8. Spontaneous graphic expression of an idea—11, 4.
9. When heard word is comprehended and reproduced by speech—1, 9, 10, 3.
10. When heard word is comprehended and reproduced graphically—1, 9, 11, 4.
11. When read word is comprehended and reproduced by speech—2, 12, 10, 3.
12. When read word is comprehended and reproduced graphically—2, 12, 11, 4.

The scheme also attempts to show an internal and external speech. At the present time exact correlation between all types of speech

disturbance and definite pathways cannot be made. But in the main certain broad facts have accumulated to permit certain fairly exact generalizations. In the first place the general speech mechanisms are located predominantly in the left hemisphere in right-handed individuals. In the left-handed the localization is predominantly in the right hemisphere. Ambidextrous brains are known and educable opposite speech areas are known.

While one speaks of speech areas, auditory (temporal) and optic (occipital), motor (Broca's convolution) and attempts to localize them, the fact is that the architecture of the brain is so complex, the pathways utilized in the speech mechanisms so widespread, that it is better to speak of aphasia areas. These are areas of special predilection for the occurrence of speech disturbances which are more or less stereotyped. There are areas which are supplied more particularly by the 1, 2, 3, 4 branches of the Sylvian artery, distributed to the third

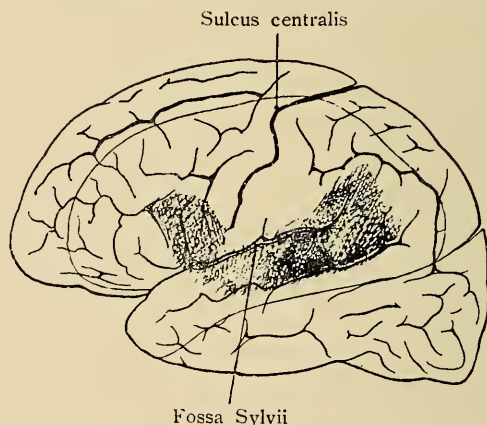


FIG. 122.—The aphasia regions in the left hemisphere. (Veraguth.)

frontal convolution and operculum of the central convolution, Broca's area (motor speech aphasias), the insula (Heschl convolution), the posterior part of the first temporal, auditory centers, amnesic aphasia, and the angular gyrus (Cuneus, optic alexias).

These areas are well shown in v. Monakow's diagram here reproduced.

Clinical Forms.—These may be subdivided into external and internal speech disturbances.

I. Deafness brings about a special form of speech disturbance (deaf mutism). Even though the speech apparatus be intact, it lacks the dynamic stimuli to be utilized. When speech is acquired, it has a peculiar monotonous quality. Certain forms of mutism from faulty hearing are to be distinguished.

II. Dysarthrias.—Used in a broad sense, these include disturbances in speech due to defect in the productive pathway.

(a) They may be of purely psychogenic origin, *i. e.*, compulsive ideas, hysterical conversions, psychotic symbol distortion, such as are evidenced by stammering, stuttering, hysterical speech, katatonic speech.

(b) Peripheral motor palsies; facial, palate (rhinolalia).

(c) Bulbar palsies (mouthful speech), as seen in progressive muscular atrophy (Aran-Duchenne type, often syphilitic) in amyotrophic lateral sclerosis, also at times of same etiology, in acute poliomyelitis, in multiple sclerosis, syringomyelia, in tumors of the medulla and pons, and in general paresis. In this latter case almost specific speech

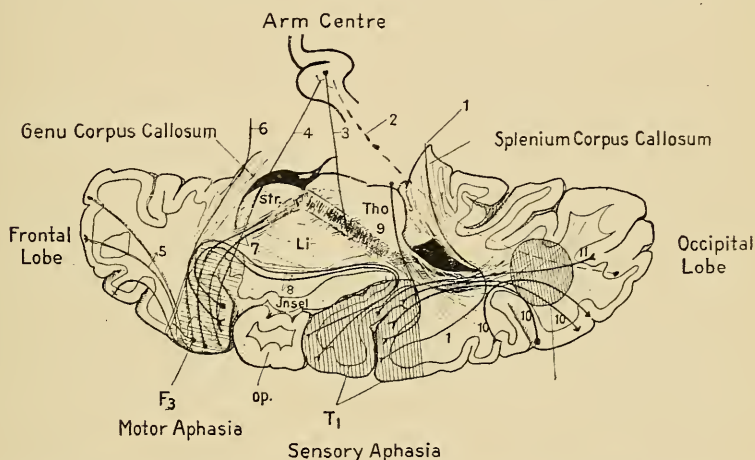


FIG. 123.—Scheme of the chief areas and pathways involved in aphasic disturbances. Nos. 3, 4, 5, 6, 7, sensory pathways; Nos. 1, 2, 8, 9, 10, 11, motor pathways. *Str.*, corpus striatum; *Li.*, lenticular nucleus; *Tho.*, optic thalamus; *op.*, operculum; 1, path from left temporal lobe to the arm region; 2, path from occipital lobe to the arm region; 3, path from the arm region to the internal capsule and peripherally; 4, path from temporal lobe to arm region; 5, paths from the prefrontals to F_3 ; 6, connecting path with the right hemisphere by means of the corpus callosum; 7, paths from F_3 through the internal capsule downward; 8, connecting paths from F_3 to T_1 , operating both ways; 9, path from the internal geniculate to T_1 ; 10, connections between angular and supramarginal gyri and T_1 ; 11, path between occipital and T_1 . (Veraguth after v. Monakow.)

disturbances appear which are described under the chapter on Syphilis of the Nervous System.

(d) In disorders of the static equilibrium mechanism, as in chorea, paralysis agitans, multiple sclerosis, one finds incoördination or scanning, jerky, or monotonous speech.

The disturbances of inner speech are termed *aphasias*. They are here divided into (A) Receptor, and (B) Productive, which are practically synonymous with sensory and motor.

Aphasias.—Although the separation of all aphasias into two groups, receptive and productive, is theoretically possible, practically this is not the case. The actual clinical pictures seen vary enormously.

So great is this variation that a text-book description of aphasia is almost impossible. It may be accepted, however, that lesions in the frontal section of the speech area, which includes the anterior portion of the island of Reil (Heschl's Convolution) will occasion the motor speech types of aphasia, here called productive; whereas lesions in the temporal, parietal, and occipital regions will give rise to sensory, or receptive, aphasias. (See Fig. 122.)

Without attempting to go into minute details the lesion in the motor speech area gives rise to a loss of the motor components of words, that is to say, the patient is unable to assemble his motor memory images of words. In motor aphasia then he is more or less dumb, and is able to say only a few words or parts of words or fragments of sentences. The patient is unable to repeat words on request and cannot read aloud spontaneously. For the most part he is also unable to write spontaneously or to write to dictation, but he may be able to copy. This is the usual picture of what is known as *complete motor aphasia*.

In the contrasting picture of *complete sensory aphasia* there is no difficulty in speaking *per se*, but the patient cannot talk comprehendingly and is apt to babble. He hears sounds and tones but does not know that they are words and cannot use them in their proper way. He does not recall the meaning of these sounds, either as to their letters, nor in the letter combinations, as syllables or words. If this sensory aphasia involves chiefly his memory of sounds he is word-deaf and suffers from auditory sensory aphasia. He may also make mistakes in writing just as he does in speaking; and paraphasia, or its severe grades, known as jargon aphasia, in speech is paralleled by similar mistakes in writing, paraphraphia. The patient may also make foolish mistakes in reading, paralexia, or at times he is unable to read at all, alexia. If he does not understand what he sees one speaks of him as being word-blind.

These are the two chief forms of aphasia.

A *total aphasia*, which frequently follows a rupture of the Sylvian artery, involving both the frontal and the temporoparietal regions of the speech domain is even more frequent than motor aphasia or sensory aphasia. Most aphasias partake of this dual nature. In the majority of cases the motor defect masks the sensory one.

In addition to these main divisions, *partial aphasias* have been described. A complete summary of these would fill a large volume. A few of the more characteristic, however, are worth picking out. Students should recall, however, that the aphasia problem is highly complicated, and every case presents marked variations.

Isolated hemorrhages or tumors which involve the island of Reil, give rise to what is known as an island aphasia. Here association fibers between the auditory and motor-memory centers are cut off. The clinical symptoms closely resemble motor aphasia. Spontaneous speech is much impaired and the patients are better able to repeat on

dictation. There is apt to be a certain amount of paraphasia, patients talking a more or less senseless jargon. Paragraphia is also usually found.

Pure word-deafness is a type in which the patient cannot understand spoken speech. He therefore cannot write to dictation nor can he repeat the words spoken to him; on the other hand he is able to speak spontaneously, can read aloud, can write and can copy. He has no paragraphia, paralexia or paraphasia. He is simply word-deaf, being unable to distinguish word sounds from other sounds. He behaves a good deal like a patient who seems to be deaf, and in testing for this type of aphasia it is important to exclude deafness. In this form of aphasia the lesion is usually located beneath the cortex of the first and second temporal convolutions.

GENERAL SCHEME OF DIFFERENT APHASIA TYPES. (EICHORST.)

Type of aphasia.	Location of lesion.	Loss of function.	Intact function.
1. Cortical motor aphasia. (Word-dumbness.)	Broca's center, posterior two-thirds of the third left convolution, pars triangularis and operculum.	Spontaneous speech. Repetition of words. Reading, writing (to dictation or spontaneous). Ability to repeat syllables.	Comprehension of words. Comprehension of writing. Copying.
2. Transcortical motor aphasia.	Hypothetical. Cutting of paths between (1) with ideational center in frontal region.	Spontaneous speech. Spontaneous writing.	As 1, also can repeat spoken words. Write to dictation. Read aloud.
3. Subcortical motor aphasia.	Connection between Broca's center and the bulbar nuclei of the muscles of speech.	Spontaneous speech. Repeat spoken words. Reading aloud.	Understands speech. Understands writing. Spontaneous writing. Copy and copy to dictation.
4. Cortical sensory aphasia. (Complete word-deafness.)	Wernicke's center; posterior third of the superior temporal gyrus and gyrus supramarginalis.	Comprehension of speech. Repeat spoken word. Reading. Writing to dictation.	Spontaneous speech. Spontaneous writing. Copying. Paraphasia. Paragraphia. Senseless copying.
5. Transcortical sensory aphasia.	Connections between 4 and ideational center, otherwise as in 2.	Comprehension of speech. Comprehension of writing.	Repeat words, and read aloud without comprehension. Copy to dictation without comprehension.
6. Subcortical sensory aphasia. (Pure word-deafness.)	Connections of Wernicke's center mostly lesions in the left temporal lobes or of corpus callosum between the temporal lobes.	Comprehension of speech. Repeating of words. Copying to dictation.	Spontaneous speech. Reading aloud. Comprehension of writing. Spontaneous copying writing.
7. Conduction aphasia.	Lesions in cortex and gray matter of the island of Reil.	Repeating words. Reading aloud. Writing to dictation.	Paraphasia. Paragraphia. Comprehension of writing. Comprehension of speech. Copying.

In addition to these forms there are described pure, or subcortical, motor aphasia, and pure, or subcortical word-deafness. Cortical motor aphasia, cortical sensory aphasia and isolated alexias, agraphias, etc., these must be looked for in larger works on the subject, already referred to.

A short summary of these characteristic forms is given on page 243.

Writing Disturbances.—A great variety of disturbances in writing, quite analogous to speech disturbances, are known. Thus, writer's cramps are analogous to stuttering and stammering. They are probably psychogenic in origin. In the psychoses, very characteristic writing features and failures are present. The paretic may write just as he speaks, slurring, leaving out syllables or words, etc. The katonie may show stilted writing just as he shows a stilted affected speech. The haste of the manic is seen in writing as in speech. In time a true science of chirography may be built up on a psychical basis, just as a true science of phonetics.

Paralysis agitans, multiple sclerosis, shows analogous features in writing and in speech.

CHAPTER VI.

AFFECTIONS OF THE PERIPHERAL NERVES: SENSORY AND MOTOR.

NEURALGIAS.

THE most characteristic feature of disordered peripheral sensory nerves is pain. In the presence of pain alone, one speaks of neuralgia; pain with trophic disorders, tender nerve trunks and altered muscular function when peripheral is usually termed neuritis; radiculitis if in the roots or the plexus, whereas pain associated with peculiar skin eruptions due to ganglionic root involvement is called herpes zoster or zona. The psychic pain of hysteria, and the central pains of thalamic lesions are not now considered.

The boundaries between these affections are largely artificial. Thus a very mild neuritis presents only its neuralgic features; and a zona may be so slight as to cause no eruption. From a clinical standpoint separation of these processes may be impossible. It is not always necessary. Pathologically speaking, one locates the lesion of zoster in the sensory ganglion, yet tic douloureux—or trigeminal neuralgia—is preëminently a disease of the sensory ganglion, the Gasserian, and yet there is rarely any zoster eruption. One invokes the etiological factor of an acute infection element in herpes zoster yet there are zoster cases due to other than bacterial causes. Sciatic neuralgia or sciatic neuritis indicates a fluctuating point in the causation and severity of sciatic pains. The differentiation between a radiculitis and a neuritis is often solely a question of terminology.

Too much weight, therefore, is not to be laid upon the classifications given. For practical purposes these affections are treated under three heads, but their fluctuating separations should not be forgotten. It is useless to call neuralgia a functional disorder. This is tantamount to saying it is a disorder of unknown causation, and hence serves as a cloak for careless investigation.

Like many other conditions in nature, these affections, when seen in an accentuated and pure form, for practical purposes, represent different entities, yet the partial and intermediary forms are so many that the description of the clear cut, classic types does not do justice to the forms found in nature.

Definition.—A painful affection of the nerve trunk or its branches, characterized by remittent or intermittent flashes of acute pain, with free intervals, not usually accompanied by trophic disturbances of the muscles, unless its severity limits the activities of an organ, occasionally associated with painful nerve trunks and with disturbances in the skin structures.

Neuralgias are but the expression of many diverse lesions which may involve the body in general, as toxic and infectious states, a nerve trunk itself, the sensory ganglia, contiguous structures, or they may be the reflex expression of a disorder in a viscus remote from the site of the pain. They may be of purely psychogenic origin, mostly hysterical conversions, occasionally delusional projections. Neuralgia therefore is to be considered solely as a symptom, a syndrome, or a purely reflex condition. There are no idiopathic neuralgias.

Etiology.—An extraordinarily wide range of causative factors may determine mild or severe neuralgias in very diverse regions of the body. The most frequent causative factors are:

(a) Anemias due to hemorrhages, chlorosis, pernicious anemia, and parasites.

(b) Toxins of exogenous origin, inorganic and organic, or purely endogenous toxins: thus poisoning by lead, mercury, arsenic, and copper. Alcohol and tobacco are frequent causes. Morphinism causes neuralgia as an abstinence symptom. The toxins of many infectious disorders are especially prone to bring about neuralgias. Tonsillitis and malaria are examples. Typhoid fever, measles, gonorrhoea, possibly syphilis, and streptococcic infections are frequently accompanied by neuralgias. The endogenous toxemias of diabetes and latent nephritis are further examples.

(c) Inflammation of the sensory ganglia, which may be either of infectious or non-infectious nature, gives rise to some of the severest forms, as seen in herpes zoster. These zoster occur from involvement of any ganglion, from the uppermost to those furthest caudad. They are usually dealt with in books on dermatology, but they are essential nervous disorders. Ganglion involvements of non-infectious types give rise to neuralgias, such as tic douloureux, while tumors of the sensory ganglia may condition persistent and obstinate neuralgias in the affected sensory nerves.

(d) Involvements of the nerve trunks themselves, either by mild neuritic processes, perineuritis, pressure from anatomical structures, pressure from lesions, cuts, bullets, wounds, tears, tumors, aneurism, exostoses, fractures, or displacements may cause severe neuralgias. If the nerves degenerate neuritis results.

(e) Reflex neuralgias are numerous and puzzling. Pulmonary cardiac, gastric, hepatic, renal, ureteric, intestinal, vesical, uterine, ovarian, prostatic, testicular, and affections of other viscera give rise to herpetic eruptions, with painful, sensitive skin areas and neuralgias; in many instances the neuralgia is not accompanied by herpes. Head's¹ complete analysis of this class of cases is of paramount importance. Thus a persistent sciatica may be the reflex of a prostatic disturbance. An anemic woman may not suffer from pain, but on menstruation her referred neuralgic pains may become very severe over the tenth dorsal

¹ Brain, xvi, 1; xvii, 339; xix, 153.

nerve, and pain and tenderness are frequent over the areas of the sixth dorsal (heart), seventh dorsal (stomach), and there may be occipital and midorbital neuralgia (Head). (See Figs. 124 and 125.)

(f) Organic disease of the nervous system. General paresis, tabes dorsalis, spinal or cerebral disease, thalamus disease, syphilitic meningomyelitis, etc., are often accompanied by neuralgic pains.

(g) Constitutional Factors: The arthritic, gouty, rheumatic, and scrofulous may be said to be predisposed. Unknown factors thought to be related to atmospheric pressure, humidity, high electrical tension, etc., play a role in many of these cases.

(h) Chronic vascular disease, and especially arteriosclerosis, is a frequent cause, particularly in the aged, the senile, and the presenile. Syphilitic vascular disease is a cause.

(i) Exposure to cold is an important factor. It is not certain that all neuralgias caused by cold are not really mild types of neuritis or perineuritis; discussion of the distinction is fruitless. The older writers found colds a predisposing cause in from 25 to 40 per cent. of the cases. In damp, cold countries this is particularly noticeable.

(j) Psychogenic Factors: These play a large part in practical medicine in determining neuralgic pains, so-called.

Symptoms.—Pain is the main factor in neuralgia. For the most part it is the only expression of the nerve disturbance. The character of the pain varies considerably, but in general it may be described as unilateral and paroxysmal. It is characteristic of most neuralgias that they are not primarily localized in the periphery. The pain seems to begin beneath the surface, and may then shoot out to the periphery. It may be described as biting, boring, tearing, darting, cutting, like an electrical shock, like a hot iron, etc., each patient having his own pet expression. It may come and go in lightning-like flashes, or throbbing pulsations, persisting for a shorter or longer time, then stopping for minutes, hours, or days, then recurring. When continuous, the pain varies considerably in its intensity.

The painful area usually conforms to the peripheral distribution of the sensory nerves. In the herpetic and referred neuralgias the root zone area is involved.

Certain *points* seem to be foci from which the pains start. These are usually situated along the nerve trunks, and pressure upon them is often sufficient to cause an exacerbation of a mild attack, or to provoke an attack in a period of intermission. Valleix attached considerable importance to these points. They are found, according to him: (1) at the point of emergence of the nerve trunks; (2) at such situations where a nerve trunk transverses a muscle to reach the skin; (3) at points where the nerve fiber breaks up into branches; (4) at points where the nerve becomes very superficial; (5) at Trousseau's apophyseal points. Valleix's points are of diagnostic importance particularly in separating the neuritic from the neuralgic types.

Accompanying phenomena are frequent. In some patients a sense

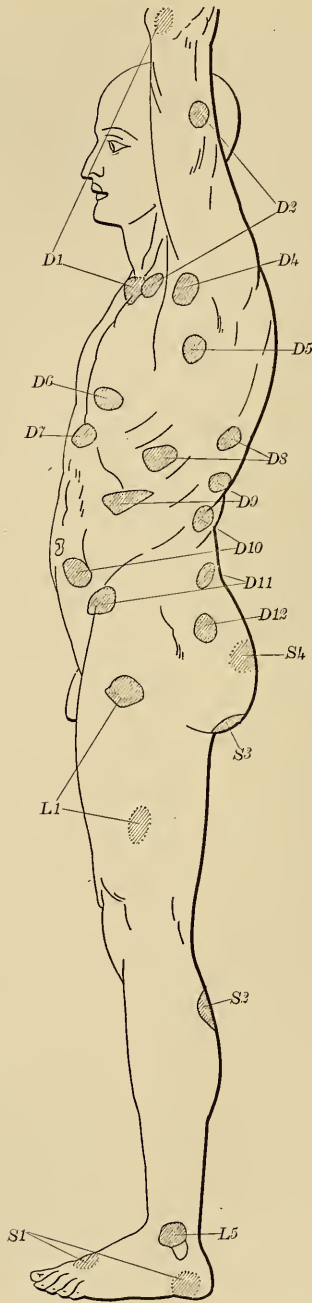


FIG. 124.—Cutaneous reflex zones of hyperalgesia, showing their relations with the spinal root segments and their vegetative nervous system connections. The dotted areas are to be referred to the internal surfaces. (After Dejerine.)

of apprehension may precede the coming on of an attack; vague sensations of discomfort often antedate the neuralgic outbreak. Ripples of pain, like pin-pricks, short twinges, etc., announce the advent of a more serious attack, or may be the sole evidence of an abortive one. Such mild phenomena are extremely frequent in certain of the so-called predisposed or neuralgic individuals, and they feel that they cannot live at high altitudes; others fear rain, or an east wind; a thunder storm causes others to have twinges; while, again,

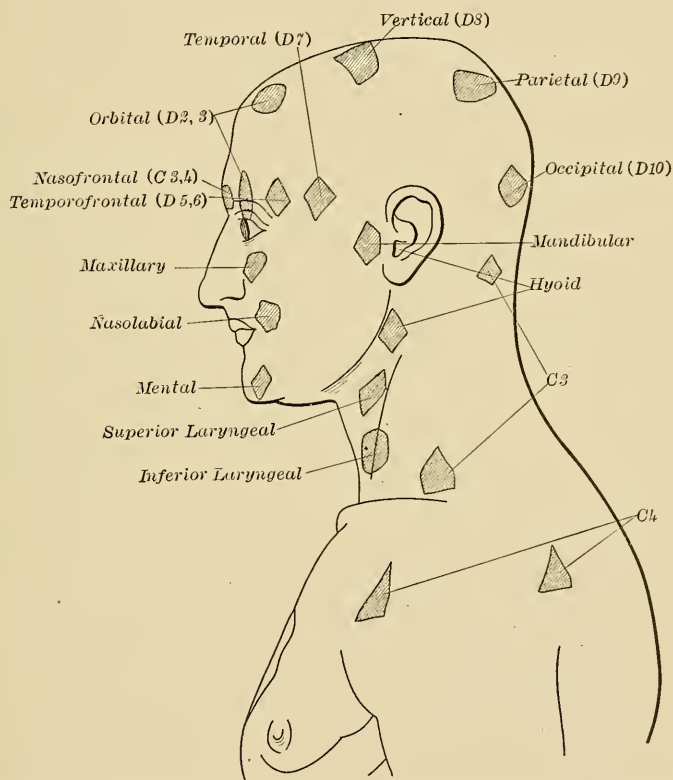


FIG. 125.—Cutaneous reflex zones of hyperalgesia of the head, neck, and shoulders in their relations to vegetative nerve (somatic) disturbances. (After Dejerine.)

certain dietary indiscretions make others complain of painful twinges for days. Just what conditions are at the basis of these features may be difficult to run down. They are none the less real.

Skin hypersensitiveness is frequent. It may precede or accompany an attack, and persist after the pain has ceased. Epicritic sensibility is mostly implicated. Light touch, a pin-prick, or slight degrees of heat or cold are magnified. Deep pressure and extremes of heat and cold are usually palliative.

Anesthesia is not infrequent following an attack of pain, and the exact topographical distribution of the sensory modifications on the skin throw considerable light on the possible causation of a neuralgia (Head).

Paresthesia is very frequent, and certain distributions seem to show it more than others. Thus, in the cutaneous branches of the femoral, they are not infrequent. Here they take on the character of a "meralgia paresthetica."

Motor disturbances, either as cramp-like contractions or as paralyses, are not infrequent in accompanying conditions. The painful contractions of tic douloureux and the oculomotor paresis of ophthalmoplegic migraine are familiar examples of this.

Vasomotor and secretory symptoms are frequent. The blood-vessels are frequently contracted in the early stages of a neuralgic attack, with resulting blanching and cooling of the skin. Following this a period of warmth, of redness, of free perspiration may result from the secondary dilatation of the vessels. In many cases of trigeminal neuralgia other secretions may be modified. Crying, coryza, or salivation are not infrequent, while in widespread neuralgic attacks an increase in the amount of urine and of milk secreted is frequently found.

Premature graying of the hair, loss of hair, thickening of the skin, erythemata, eczema, pemphigus, herpes, thickening of the bones, and, occasionally, muscle atrophy are among the rarer trophic by-products.

During an attack, irregularities of the pulse are not unusual; slowing is the rule. The pupils are frequently dilated.

The general physical and psychological reactions are extremely important. Loss of sleep and anorexia cause the patient to lose strength and flesh; and anxiety, irritability, and petulance are almost inevitable. Mental depression, sufficient to lead the patient to make suicidal attempts, is not infrequent, especially in severe cases of trigeminal and sciatic neuralgia. The contracting of a drug habit is not unusual.

Course.—This depends naturally upon the underlying condition. Many are acute and transitory, persist three or four days and never reappear. Such are the herpetic types. Many reflex neuralgias run an acute recoverable course, but show a marked tendency to recurrence. The neuralgias which accompany the chronic cachexias of nephritis, carcinoma, brain or spinal cord disease, usually progress in a markedly chronic manner. In those hereditarily disposed individuals the tendency to chronicity with longer and shorter periods is proverbial. Earlier French writers attempted to distinguish benign and severe forms. Most neuralgias in which the causative factor is undiscoverable (the so-called idiopathic or primary neuralgias) run a benign course, while the neuritic types are less amenable to treatment.

The subdivision of neuralgic neuroses, subacute neuritic neuralgia, and chronic neuritic neuralgia offer a grouping referable to course which has only clinical convenience to warrant it.

In the first type one finds the disorder more or less limited to the neuropath. The attacks come with appreciable cause, or follow a nervous shock. Exposure to cold, or dietary indiscretions are frequently claimed as causes, but are not. The pain comes on with great suddenness and usually goes without great violence; it comes and goes apparently without rhyme or reason, and is not accompanied by painful nerve trunk nor trophic disturbances. It recovers at times, to recur at intervals of a year or years.

In the subacute neuritic neuralgic type, exposure to cold or pressure, especially in arthritic patients, determines an attack. The attack develops gradually; the pain, at first mild and intermittent, gets worse and worse and more continuous. Finally, after a day or more, the paroxysms become extreme, the intervals being marked by dull pain; Valleix's points are characteristic findings.

When a mixed nerve is involved, muscular atrophy or other trophic signs appear, signaling the occurrence of a neuritic process. Local edema and herpes zoster are frequent accompaniments. This type usually commences to recover in from two to three weeks, and an ultimate recovery is to be expected. Recurrences occur, however, and a leap to the third type of chronic neuritic neuralgia is made. This form is frequent in the aged. The history is usually that of several subacute attacks with increasing tendency to chronicity. Here the trophic disturbances in muscle and in skin are more marked. The paroxysms run a remittent course.

Diagnosis.—Enough has been said to emphasize the need for a searching analysis of the causative factors of every neuralgia. They are many, and presumably the most widespread diagnostic error is the overlooking of an early tabes dorsalis in young to middle-aged adults.

Children are not prone to neuralgias—in the narrower sense—and a neuralgic affection in childhood calls for close scrutiny.

Since unilateral pain, of special localized type, occurring in irregular attacks, is almost the sole criterion of neuralgia, it is very frequent that organic disease of a viscus will show precisely similar accompanying features. In the majority of cases the underlying organic lesion may be detected—occasionally it remains difficult to locate. Not infrequently the diagnosis of a persistent neuralgia may be cleared up by the finding of malarial organisms in the blood, or more rarely the presence of a marked eosinophilia will call attention to trichina as the cause of an obstinate neuralgia; or the eggs of an intestinal parasite in the feces (uncinaria) may direct attention to an anemia which underlies a severe neuralgia. Syphilitic neuralgias, either toxic or vascular, are by no means infrequent.

The diagnosis of myalgia from true neuralgia is not often difficult, but occasionally, especially in the intercostal and lumbar regions, the diagnosis becomes uncertain. These neuralgic-like myalgias are usually isolated in their location, are not, as a rule, accompanied

by acute exacerbations, nor are the regions usually painful on pressure. Motion, on the contrary, usually aggravates myalgias.

Neuritis of a mild grade offers a specially difficult problem. As already stated, mild neuritis shows itself as a neuralgia. The question to be solved concerns the likelihood of a more severe degree of neuritis. In this case the usual signs of neuritis are painful, swollen nerve trunks, trophic disturbances, more continuous pain, Lasègue's phenomenon, weak, flabby muscle fibers, and electrical changes. New growths, pressing upon or involving the nerve trunks, within or without the spinal canal, in the early stages particularly, begin as pure neuralgic syndromes. Minute analysis of the sensory phenomena will usually clear up the diagnosis early, although at times it may be impossible in the earliest stages.

A neuralgic affection may be one of the earliest signs of a multiple sclerosis. Oppenheim has found a severe *tic douloureux* to have been the earliest sign of this disorder. Syringomyelia may begin as a localized neuralgia. Minute hemorrhagic lesions of the spinal cord of traumatic origin give rise to neuralgias.

In the diagnosis of hysterical neuralgia, great caution should be exercised. Hysterical neuralgias partaking of the nature of a pseudo-neuralgia are extremely diffuse, and react very rapidly and markedly to suggestive influences. Hysterical neuralgias are almost invariably accompanied by other conversion signs.

Neurasthenic pains need to be differentiated if not almost entirely ruled out as a common diagnostic pitfall. The many mixed forms of neurasthenic, hypochondriacal, and hysterical neuralgic pains should bear their characteristic sidelights. The diagnosis of these types of neuralgia should not be lightly made, for it is not to be forgotten that these syndromes of themselves may be the reaction on the part of the nervous system to some more fundamental organic lesion. Thus patients suffering from severe so-called neurasthenia with cachexia, and severe intercostal neuralgic pains may have an undiscovered carcinoma of the stomach, mediastinum, etc.

In *tabes dorsalis* the neuralgic pains have a wide range, are rarely localized in a peripheral nerve distribution, and are apt to be radicular in their distribution. Pain on pressure of the nerve trunk is usually absent. The objective findings in the pupils and cerebrospinal fluid establishes the diagnosis.

The pains, cramps, and muscular weakness of *intermittent claudication* sometimes gives rise to severe neuralgias. Aortic aneurism gives rise to reflex neuralgic pains, which are usually very severe, burning or boring in character. Aneurisms in other regions are to be carefully excluded.

In reflex neuralgias the use of cocain or other local anesthetic may determine, by exclusion, the site of the original lesion. An orthoform suppository pressed well against the prostate has been known to relieve a severe sciatic neuralgia. Tumors of the pelvis frequently give rise

to sciatic and crural neuralgias, and persistent neuralgic pains of the knee are often a reflex from hip-joint disorder.

Neuralgic pains are a frequent indication of disturbed nerve fiber metabolism, with a hyper- or a hypothyroid affection behind it.

For the precise localization of the areas involved consult Figs. 11, 12, 13, 14, 15 and 16, where both the peripheral and radicular nerve distribution are figured.

Prognosis.—This is conditioned by the pathological process that is responsible. The more chronic of the neuralgias, which in years gone by tended to bring about chronic invalidism or inveterate drug habits, have ceased to have such a sinister import by means of a better understanding of the underlying conditions, and by a much more resourceful therapy. The younger and stronger the individual, and the less the tendency to hereditary disposition, the better the prognosis in those neuralgias which apparently are idiopathic, as well as those due to alcohol, lead, or other toxic agent. In the more chronic forms which are not due to removable condition the prognosis is bad. With increasing insight, however, into the many intricate disturbances of nerve metabolism many of the intractable forms may be conquered.

Treatment.—The chief indications are to quiet the pain and ascertain the cause. A painstaking study of the history, and an exhaustive physical examination, are necessary in all cases. The therapy will vary, therefore, widely if the cause be ascertained; quinin will cure one patient, a surgical operation may be called for in another. General rules, therefore, are largely illusory. One should never treat a *neuralgia per se*, as it is solely a general result of many causes.

Taking up the general therapeutic indications, the analgesics which have proved useful may be discussed first. Phenacetin, acetanilid, antipyrin, aspirin, pyramidon, lactophenin, and phenocoll are among those that have been valuable. New ones are constantly being added, and among them some are certain to be of value. The salicylic acid group combinations are at times useful, especially in the milder cases and in patients with arthritic tendencies. In influenza and tonsillitis neuralgias the salicylates are useful. Combinations of these with soporifics, such as chloral, paraldehyde, sulphonal, trional, or veronal, are useful in procuring sleep, and thus prevent the reduction of the patient's resistance.

If any of the opium group be necessary it is better to give such in sufficient doses. Usually smaller doses may be given when combined with the analgesics mentioned. Aspirin, gr. vij (0.5 gram), codein, gr. $\frac{1}{3}$ (0.02 gram), and trional, gr. vij (0.5 gram), for instance, is a useful combination to be taken at night. Other combinations are equally effective. In the chronic neuralgic pains morphin is to be avoided as long as possible. This does not apply to a very old patient, or one in whom the neuralgia is simply the expression of some chronic incurable disorder, carcinoma for example. The gradually acquired immunity,

with the need for larger doses, and the pernicious effects of a habit apply to all the members of the opium group.

All analgesics are purely provisionally used. They are meant to give the patient ease while looking for the real cause of the pains.

If one's inquiry is satisfied by the relief of pain the use of analgesics alone is bad therapy. Other drugs are quinin, which in combination with the salicylates is specially valuable; arsenic which is serviceable in the neuralgias due to anemia, especially in combination with iron. Atropin and aconitin were used widely before the days of the antipyretic analgesics. Their definitely poisonous qualities have driven them into the background. The unreliability of *cannabis indica* has done the same for this otherwise useful analgesic. The iodides are called for in the syphilitic neuralgias and are useful in many neuritic neuralgias.

Counter-irritation is of great service in many cases of severe neuralgia, especially after the acute onset is over. The Paquelin cautery is the best means; mustard paste, cantharides, turpentine, chloroform, ether, and acupuncture all have their place. Local freezing may be carried out by ethyl chloride, methyl chloride, ether, or other volatile substances. Menthol, or other similar derivatives may be used for mild neuralgic pains to advantage.

Direct applications of local analgesics, either to the nerve trunk or within the spinal canal, are valuable in many deep-seated neuralgias, especially of medullary origin. Cocain, tropococain, eucain are all useful given by the Corning or Quincke method.

Local applications of heat are grateful and valuable. Hot water bags, hot sand, electrical pads, etc., may be utilized. General or local hot-water baths or hot-air baths (baking) are at times desirable.

General hygienic treatment is imperative. A generous diet, full sleep, healthful occupation, and freedom from mental worry are essential. Cod-liver oil, nitrogenous diet, with iron, arsenic, strychnin, calcium salts, are indicated. Faddy dietaries should be avoided. Even in arthritic neuralgias it is doubtful if meat does any particular harm when not taken to excess. Alcoholic beverages are to be denied.

An alkaline therapy often helps many fugacious, persistent neuralgic pains. Fruits containing the citrates seem to give relief.

Preparations of the internal secretions, particularly thyroid and pituitary clear up some intractable neuralgias of unknown origin. They may be given in doses of from $\frac{1}{10}$ to $\frac{1}{2}$ grams twice or thrice daily.

Climatic changes are rarely advisable. Low-lying, damp and humid atmospheric conditions seem least desirable for certain patients. The general stimulus that comes from a dryer, higher atmosphere, even if colder, works to the general advantage, even if not directly valuable for the relief of pain.

Electrotherapy when well managed and properly selected is of great value in some neuralgias. It cannot be said that it is clearly recognized just what forms of current are best utilizable in what types of neuralgia, hence most efforts must follow the method of trial and error.

In general, however, Leduc's modifications of d'Arsonval's rapidly interrupted current offer the readiest and most widely applicable form of electrical current for the relief of neuralgic pain. It is doubtful if any other form of electrical application is known at the present time that is as valuable as this. It is, in fact, a type of electrical anesthesia, solely palliative, but very grateful. Newer applications are being brought out, and other forms may replace the Leduc currents, but at present these seem to give the most reliable results.

Faradic currents, as heretofore employed, act for the most part simply as counter-irritants, and seem to possess little superiority over the actual cautery. Galvanism with mild currents is useful for many topalgias.

Psychotherapy is the only rational treatment for the psychogenic neuralgias. They make up nearly 25 per cent. of the neuralgias.

Surgical intervention is called for in all cases in which pressure is demonstrable and the cause removable. Tumors and new growths, involving or pressing upon nerve structures, if removable should be taken away. Surgical interference may be of radical service in many of the reflex neuralgias of obscure origin, probably related to visceral ptoses. Such surgical interference is justifiable only in chronic cases where these visceral ptoses have resulted from long-continued psychical causes. Psychotherapy is preferable in the early stages. Nerve stretching needs mention mostly to be condemned.

SPECIAL LOCALIZED FORMS OF NEURALGIAS.

While any sensory nerve in the body may become painful, there are certain regions which show a greater tendency to involvement than others. Bernhardt has collected the statistics of localized distribution in some 685 cases, with the following results: trigeminal, 124; occipital, 42; brachial, 108; intercostal, 45; lumbo-abdominal, 12; crural, 25; obturator, 2; sciatic, 303; anterior femoral, 11; Achilles, 3; tarsalgia, 4; metatarsalgia, 4; and coccygeal, 2. In 616 cases collected by one of us (J.) during four years (1902 to 1906) the distribution was as follows: trigeminal 315, occipital, 28; brachial, 31; ulnar, 1; intercostal, 19; lumbo-abdominal, 19; crural, 2; sciatic, 194; coccygeal, 1; peroneal, 2; and plantar 1.

Trigeminal Neuralgia.—Simple neuralgia of the branches of the fifth nerve are among the commonest of all the neuralgias. Fothergill's studies on *A Painful Affection of the Face*, published in 1773, is a classic. The inferior and superior branches preponderate in frequency of involvement. Most frequently these neuralgic pains are due to some affection of one of the branches. Inflamed teeth play a predominant role. Affections of the ears, the eyes, iritis, cyclitis, iridocyclitis, the skin of the face or head, inflammation within the accessory sinuses of the nose, forehead, antrum, mastoid, all of these may produce

diffuse neuralgic pains, at times clearly separable from a neuritic neuralgia of the fifth, at other times not.

Cold and wet are important agents in facial neuralgia. In certain countries, notably England and the north of Germany, trigeminal neuralgias from this cause are extremely common; they seem to be much less frequent in the United States, and notably so in southern countries.

Neuralgia of the superior branch is seen more commonly by physicians although the dental branches are involved much more frequently. These patients go to dentists and therefore do not enter into medical statistics. This is a reason why it is incorrectly stated by most writers, that the superior branches of the fifth are most often involved. For the most part the milder types of neuralgia are induced by irritation of some of the terminal filaments, while in the neuritic form, *tic douloureux*, which is the more classic, a lesion of the Gasserian ganglion is usually present. Mild cases of *tic douloureux* may be indistinguishable clinically from other types of neuralgic pain.

Tic Douloureux.—Enough has been said on neuralgia in general to indicate the character of the simpler form of neuralgia of the fifth. One type, however, by reason of its severity and its fairly definite pathological anatomy, needs more extended consideration. Avicenna knew *tic douloureux* and described it with great accuracy. It would be desirable to restrict the term *tic douloureux* to a definite and, if possible, limited type of neuritis of the fifth nerve, particularly to the form due to changes of a chronic degenerative nature occurring in the Gasserian ganglion. This is not yet possible, and clinically the neuritic, and peripheral neuralgic cases are either not at all separable from the ganglion cases, or with considerable difficulty.

Tic douloureux usually affects one side of the face. In the majority of cases some selection occurs among the branches, one or two being involved, rarely all three; the ophthalmic branch the oftenest, the inferior maxillary the least often.

The more classical *tic douloureux* neuralgias are characterized by the extreme severity of the pain, usually preceded by paresthetic prodromata, and widely accompanied by sympathetic or irradiating pains in other branches than the one chiefly involved, or in other nerves. The pain may be paroxysmal or continuous, with marked exacerbations. Patients compare them to the piercing pains of a sharp knife or the burning of a red-hot wire. The patient remains for a shorter or longer period, a few minutes to several hours, under the grip of the pain, unable to move a muscle of the face or fearful of stirring, lest a spasm more fearful than the others should occur; even the air pressure of a suddenly closed door may bring on an exacerbation. The longer attacks are rarely as vicious as the shorter ones.

Valleix's points are relatively constant. In ophthalmic involvement the sore points are found above the supraorbital notch, at the external angle of the upper lid, the upper outer aspect of the nose, and

the globe of the eye; in the superior maxillary branch the inferior orbital notch is the chief point of pain; the malar bone, and opposite the last upper molar are other less frequently found points, while the outer angle of the mouth, and the roof of the mouth are rarely their site. In the inferior maxillary distribution the points are chiefly just in front of the auditory canal, the side of the tongue, the border of the chin, and Trousseau's points over the first and second cervical vertebræ.

Vasomotor and secretory disturbances are usual. The skin is, as a rule, hot and swollen, occasionally pale and frigid; tears, nasal secretions, and saliva flow in abundance. The eyelids may be swollen, the conjunctiva reddened to the point of ulceration at times; within the nose and mouth extravasations occur, and ulcers are not uncommon. Herpetic attacks are also not infrequent, and in some of these attacks grave injury to the eye structures may take place. Glaucoma is one of the severe complications. Other trophic disturbances are skin eruptions, acne, erysipelatous reddening, graying of hair, and blackening of the tongue. In long-continued cases hemiatrophy may occur. Changes in the sense of taste, of touch, of hearing, are at times present. Photophobia is frequent, while diminution in the visual fields and accommodation cramps are noted.

Severe mental disturbance, amounting at times to hallucinatory confusion may be present. Suicidal attempts are to be guarded against in these excruciating cases.

The motor disturbances consist in convulsive movements of the facial muscles (convulsive tics, spasmodic epileptiform neuralgia, Trousseau), sudden forced closing of the eyelids, drawing of the mouth to one side, or sudden turning of the head. At times the convulsive movements extend to the arms. Paralytic phenomena in the third nerve are noted. The general psychical disturbances noted are prone to occur in this type.

Course.—In the majority of cases the attacks appear in series and attain a periodicity which comes to be dreaded by the sufferer. The free intervals usually become shorter and shorter; but many patients may have only one attack a year, especially in cold weather, or even at longer intervals. A single attack may last a few days, or in the severe forms several weeks, the patient not being free from pain day or night, save under the influence of morphin. Some patients have a few attacks in a lifetime, others are not free from the disease for years. The severer convulsive forms are prone to occur late in life.

Diagnosis.—Ordinarily the classical form of tic douloureux is recognized without difficulty. Patients have all their teeth extracted, however, under the mistaken diagnosis of a dental disease, while some intractable trigeminal neuralgias have been cured by proper attention to diseased teeth. Aneurism of the carotid, tumors pressing upon the nerve or upon the Gasserian ganglion, may be difficult to determine as the exciting cause. These, however, are usually accompanied by accessory symptoms, palsies, eye-ground changes, aneuris-

mal murmurs, pain within the head, cerebellar syndromes, ear pains, etc. The otalgias (tympanic neuralgias) usually considered in this connection are possibly due to geniculate ganglion disorder, and have been referred to by Hunt as neuralgias of the seventh nerve.

Multiple sclerosis has started as a trigeminal neuralgia.

Treatment.—It is as essential to endeavor to find and treat the cause for a facial neuralgia as for neuralgia in general. The various remedies given under the heading of neuralgia may be tried, and as malarial neuralgias are very frequently trigeminal, energetic quinin therapy should be given; the absence of blood findings is not contra-indicative, especially in non-malarial neuralgias. Gelsemium, the tincture in 10 minim doses, gradually ascending, aconite in doses of $\frac{1}{500}$ grain, cannabis indica (fresh), in doses of from $\frac{1}{4}$ to $\frac{1}{2}$ grain, is reputed as specially valuable in the facial cases. Any of the analgesic antipyretics may suit individual cases, and avoid the use of morphin, which alone is reliable in many severe cases. Local applications of cocaine to the conjunctiva, nasal mucous membranes, buccal surfaces are sufficient to repress some mild attacks.

Injection methods have been tried for years. In the beginning the peripheral branches were injected by various analgesic drugs, in early days chloroform, and in later times particularly cocaine and its allies or derivatives. The effects were valuable, but temporary. Osmic acid was used later, but regeneration took place. Pitres and Vaillard, in 1887, and Schlösser,¹ in 1900, took up a series of experiments with alcohol, and the latter has perfected a method of injecting alcohol within the substance of the Gasserian ganglion, which has given excellent results. The chief features in the deep alcohol injection method is the introduction of a long, dull, cutting needle into the foramen ovale and there injecting in situ the branches of the trigeminus. Special methods have been devised. Narcosis is not necessary; 80 per cent. alcohol is used. In three to four hours following the injection the pain is relieved, and two or three more injections are given within a week to complete the treatment. Immediately following the injections, which should be done only after extended practice on the cadaver, there is a marked anesthesia on one side of the anterior part of the head, including the nostril, palate, and one-half of the tongue; a slight paralysis of the muscles of mastication, which may persist for some time, but usually disappears in a few hours; a degenerative process is set up in the nerve trunk, which is recoverable, and general sensibility usually returns, but the pain returns. Relief extending over a year in a number of cases is reported by numerous observers. Some patients have been relieved for four or five years. Edema of the posterior eye structures and hemorrhage are among the discomforts and even dangers of the operation, especially in the use of the intra-orbital methods devised by French operators. Relapses are apt to occur.

¹ Münch. med. Woch., April 30, 1897.

Three surgical procedures have been seriously advocated. The first and earliest consisted of peripheral section, first said to have been done by J. C. Warren of Boston. Section of the fifth may be employed to advantage in those cases in which the disease is undoubtedly peripheral. As modified by more recent procedures, the older objection that regeneration takes place is partly done away with.

Rose, MacEwen, Horsley, Hartley, and Krause perfected the operation of excision of the ganglion, and the modified Hartley-Krause operation by the temporal route has been largely the method of choice. Cushing's more recent modifications are of lasting value. The operation still remains one of much difficulty and seriousness. Recurrences are known even with this method, and the efficiency of the newer devices for preventing this by capping the ends of the divided nerve trunks with metallic laminæ is too recent to pronounce upon.

Van Gehuchten, in 1903, suggested tearing. Spiller, in 1898, had suggested the surgical expedient of cutting the sensory root, which he claims is safer than the operation of tearing, a procedure tried in 1881. The method of division of the sensory root, as reported by Frazer and Spiller, promises to be one of the most valuable surgical procedures thus far devised.

Cervico-occipital Neuralgia.—This occurs in the distribution of the sensory nerves of the cervical plexus, consisting chiefly of the occipitalis major, the occipitalis minor, auricularis magnus, cervicalis superior, supraclavicularis and phrenic. Neuralgia in this general region seems to be rare. In Remak's summary of 15,000 cases only 50 were in the cervico-occipital regions. Valleix has given one of the most complete monographs on neuralgia in this area and little has been added to his description, save in the finding of rare etiological factors.

Etiology.—The several causes of neuralgia are operative here and need not be repeated. Special determining features seem to be the carrying of heavy weights on the shoulders (a more frequent cause seen in brachial neuralgias), arthritis deformans of the upper cervical vertebræ, caries, syphilis, tuberculosis, tumors, cervical pachymeningitis, falls and blows wrenching the cervical vertebræ, enlargement of the cervical lymphatics, and aneurisms of the vertebral artery. Oppenheim refers to the great frequency of hysterical neuralgia in this region and psychogenic neuralgias of the back of the neck and occiput are extremely common. They are often found in individuals who are either under great strain or those who are constantly forcing themselves.

The pains occupy the regions mentioned, being particularly localized in the neck, below the occiput, and running up to the vertex, occasionally behind the ears. The Valleix point found most frequently is the occipital point between the mastoid apophysis and the first cervical vertebra; points between the sternomastoid and trapezius

(cervical), the anterior border of the mastoid, and the middle of the ear are of less frequent occurrence.

The pain is frequently bilateral. Dull pain on pressure, with tender skin, is usual as a paroxysmal occurrence. This tends to make the sufferer hold his head in a stiff position, which in time may cause a characteristic attitude. This tenderness may be so acute that ruffling of the hair will start a paroxysm. Graying of the hair, loss of hair, with other trophic signs may be present. Sudden pulling back of the head, or other muscular involvement, is an occasional symptom.

Diaphragmatic Neuralgia.—This form of neuralgia, also known as phrenic neuralgia, is of rare occurrence. Falot and Peter have written upon it. The pain is usually present near the free border of the ribs, occasionally as high as the chin and in the neck, beneath the clavicle, and in the scalenus anticus muscle. Trousseau's points are located over the second to the fifth cervical vertebra. The pain frequently runs down the arm, especially in certain complex cases of mixed brachial neuralgia.

Breathing may be seriously interfered with, the breath coming fast and short; longer excursions of the diaphragm are impossible. It is a common experience to have a short, sharp stitch in the side, with inability to breathe for fear of pain. This is the type of distress encountered in phrenic neuralgia. In the majority of cases the pain is in the left side.

Anemia, affections of the mediastinum, heart and pericardium, and aneurism of the aorta are the most frequent attending features. An intractable phrenic neuralgia may complicate an exophthalmic goitre, or be present in carcinoma of the neck region.

Idiopathic or pure phrenic neuralgias seem to be unusual, whereas temporary or more permanent types are seen as symptoms of the affections named. In the latter case the prognosis depends on the initial difficulty.

Brachial Neuralgia.—In this general form the components of the brachial plexus, from the four lower cervical, or some of its filaments, and first dorsal roots, are those involved. The chief nerves carrying sensations from the skin area of the arms and shoulders are the circumflex, radial, internal cutaneous, and musculocutaneous. These enter, for the most part, the upper and middle cords of the plexus. In the majority of cases the pains of brachial neuralgia are located in the upper arm and about the shoulder, *i. e.*, in the area of the circumflex, radial, musculocutaneous, and internal cutaneous nerves.

Bernhardt's statistics show that men are more frequently affected than women, but the reverse shows true in the figures of other observers (Romberg, Erb). More women have brachial neuralgia than men, and in most instances it seems that excessive sweeping is the attributed cause. In piano-players, neuralgias in this area are frequent. Perhaps these should be relegated to the occupation neuroses with the pains of hair-dressing, skirt-carrying, telegraphy, writing, etc. At any rate,

arm and shoulder pains are frequent, in their mild grades at least, and very variable. (See Fig. 126 and Figs. 11, 12, 13, 14, 15, and 16.)

The usual causative factors come into play here. The neuropathic constitution is put in the foreground by Oppenheim; Bernhardt lays considerable stress upon the importance of bone injuries with callus formation in the causation of many arm neuralgias. Small punctured wounds about the forearm, wrist, and arm are responsible for many symptomatic neuralgias, as Weir Mitchell has so well shown. More remote cases are found in vertebral disease, tumor formation, aneurisms, syringomyelia, multiple sclerosis, and tabes. The frank neuritic processes in their beginnings must be borne in mind, and cervical rib should not be overlooked.

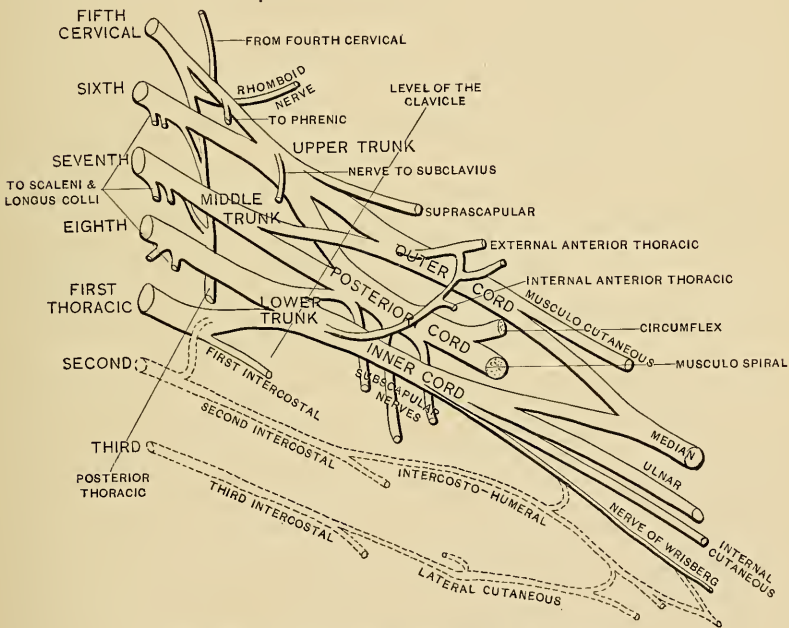


FIG. 126.—Plan of the brachial plexus. (Gerrish.)

Symptoms.—Cervicobrachial neuralgias are extremely variable in distribution, extent, and severity. The onset is usually sudden, especially in those patients in whom an antecedent history of exposure to cold and to wet is obtainable (motormen, policemen, etc.); at times the beginning is preceded by twinges and slight distress. On awakening in the morning sharp pain is felt in the shoulder and arm. The pains in brachial neuralgia are less apt to be the sharp, shooting variety so dreaded in tic douloureux, but sudden accessions of sharp pains, varying in their intensity, are frequent. As with most neuralgic pains, movement increases them. Toward evening the pains are apt to in-

crease and the patient, although obtaining relief by lying down, rarely sleeps well. Soreness of the skin, slight swelling, and general reduction in tone are the usual accompaniments. With increasing disuse slight atrophy is common, and swelling is usual. The tendon reflexes are usually more irritable and active. More atrophy paresis with vasomotor-trophic symptoms and altered tendon reflexes indicate a definite neuritic process. Herpetic eruptions occur with non-infectious, as well as with infectious involvement of the sensory ganglia.

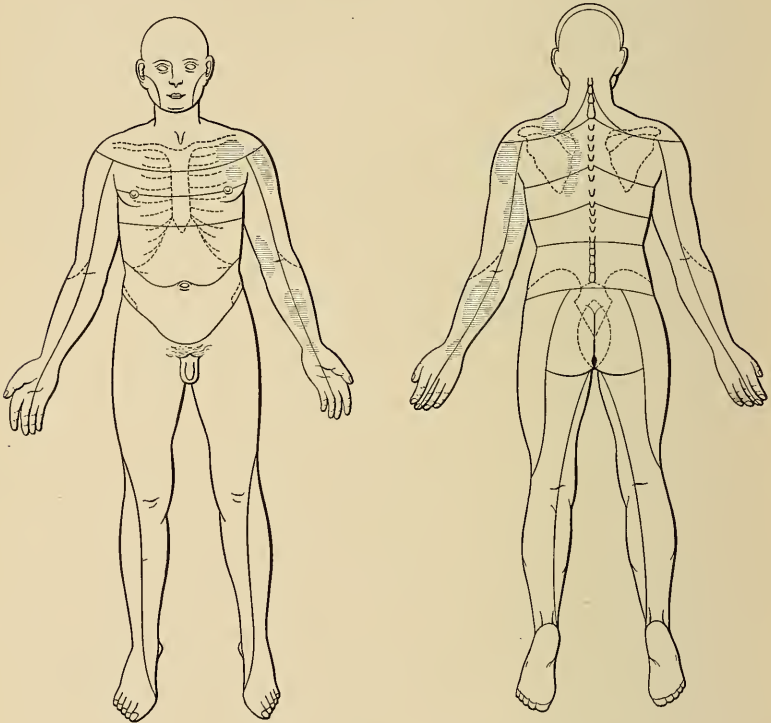


FIG. 127.—Painful points in brachial neuralgia.

Tender points are very variable. They are most frequently in the middle of the back; about the level of the second or third dorsal there is usually a sore Trousseau point. Gowers notes that the inferior ulnar point in front of the wrist is the commonest sore point. Babinski has called particular attention to a radial neuralgia due to a mild or severe neuritis of the radial. The pains occupy the posterior portion of the arm, and are unusually severe. Neuritic changes are not infrequent. The chief causes seem to be exposure to cold and disturbances of the reciprocal action of the ovaries and thyroids or testicles and other endocrinous glands. The menopause is a frequent period of onset.

Diagnosis.—In the diagnosis particular care is needed in excluding affections of the spinal cord, meninges, and vertebrae, as well as angina pectoris and pseudo-angina. Disease of the joints and bones should be excluded at the outset, although it may be impossible in some cases of periostitis. In tumors and other organic affections of the cord the painful points are usually absent, but the earliest and only symptom of spinal cord tumor, intramedullary or extramedullary, may be a brachial neuralgia. In tabes the pains are apt to be bilateral. The exhaustion neuralgias are also apt to be bilateral. Valleix's and Trousseau's points are usually absent. The general indefinite features of a myalgia, plus the muscular, rather than the nerve soreness, are usually sufficient to exclude it.

The occupation neuroses involving the arm and shoulder are many. The history of protracted exercise of certain groups of muscles is usually sufficient to identify the proper cause for the neuralgic pains. Occupation neuralgias, like neuritic neuralgias, are neuralgias none the less, the sole diagnostic question arising as to the cause, and through this the proper mode of therapeutic attack and the probable outcome. Alcoholic neuritis in its mild grade offers particular embarrassments. Lead poisoning neuralgias are to be borne especially in mind, while diabetes is of prime importance. Brachial psychalgia is a possibility, but the diagnosis must be made with extreme caution after a rigid exclusion particularly of organic factors. Hysterical and neurasthenic neuralgias occur in this distribution as well.

Treatment.—Rest is a necessity, and is primarily insured by means of a sling. The diagnosis of a cause being assured, treatment should be begun to remove it, either by medical or surgical means. Nerve suturing for injury has come to occupy an important place, and is usually attended with good results, even after long periods of loss of function. In the early stages active mechanical treatment is to be avoided. Hot applications are useful in most acute neuralgias. In the later phases massage, particularly the Nægeli movements, are valuable. Galvanism, 3 to 6 milliamperes, is well adapted to these neuralgias, but usually much better results are obtained by the Leduc rapidly alternating currents. Salicylates (especially in analgesic combinations), iodides, quinin, arsenic, and large doses of strychnin are of value at times. The internal secretions are curative for some. Psychoanalysis is to be used in hysterical cases.

Intercostal Neuralgia.—The twelve dorsal nerves constitute the plexus involved, although the upper series, especially of the left side, are most frequently concerned. Bernhardt says that the site of election is mostly from the fifth to the ninth. Since the dorsal nerves divide into internal and external branches the site of the neuralgia may be on the surface or within (pleurodynia, etc.). The two upper nerves send branches to the internal surface of the arm, and pain is occasionally felt there. The abdominal involvements are rarer and may extend down to the genitals. (See Figs. 11 to 16.)

Women more often show this form of neuralgia than men, and the disorder is much more common in cold weather.

The pains are usually less severe than in other regions, although their sharp, sticking character distresses the chest movements, especially since all movement tends to aggravate them. Tender points are found at the site of perforations near the spine. Skin hyperesthesia is extreme at times. Herpetic neuritic neuralgias are relatively common in this distribution.

Among the causes to be diagnosed may be costal caries, affections of the spinal cord and meninges, disorders of the pleura, particularly carcinoma and tuberculosis, aortic aneurism, dilatation of the stomach, carcinoma of the liver, angina pectoris, pericarditis, local trauma, fractures, etc.

Mammary neuralgia or *mastodynia*, which is frequent in the later stages of nursing, and in some women at the menstrual epoch, is a special form. The pain is usually deep within the gland, and may be accompanied by a slightly increased secretion. The whole skin may be sensitive, especially the nipple, when the superficial nerves are mostly involved. Local glandular induration occasionally occurs. This has led to the mistaken diagnosis of carcinoma, but a neuralgia may occur due to a carcinoma of the breast. *Tabes* may give rise to an intercostal neuralgia.

Treatment.—Local applications are useful, especially the ethyl chloride spray. Blisters are efficacious. Bandaging affords marked relief. General measures already described call for no further mention.

Lumbar Plexus Neuralgias.—These are most conveniently arranged as (1) lumbo-abdominal; (2) ilio-crotal or testicular, (3) crural (sciatica), (4) femoral, and (5) obturator, involving in each case certain of the branches of this plexus. Mixed and indeterminate forms are not infrequent.

The Sciaticas (sciatic neuralgias, sciatic neuritides).—It has already been indicated that it is largely indifferent whether one regards this as a neuralgia or a neuritis, since transition forms are very frequent. It consists of pain in the distribution of the nerves of the sacral plexus, the sciatic and its branches.

History.—Cotugni, in 1764, gave so clear a description of sciatica that the malady is often given his name. Valleix, in 1841, described the painful points with great minuteness. Lasègue, in 1864,¹ described his well-known symptoms of neuritis of the sciatic, since which time many monographs have appeared, the most important of which are those of Brühl, Lago, Vulpian, and Bernhardt.

Etiology.—In the same manner that one finds for the other neuralgias and neuritides, similar causes are at work for sciatic pains. It is unnecessary to amplify these causative factors. Any of those general causes found on the previous page may cause a sciatica, but special

¹ Arch. Gén. de Méd. (1864). Oppenheim.

emphasis should be laid upon two or three. Trauma is responsible in many cases for the development of sciatica. Syphilitic osteoarthritis, and syphilitic meningitis of mild grade are responsible for the development of intractable sciaticas. Certain French authors claim as high as 90 per cent. of all sciaticas to be due to this syphilitic factor. In this respect then sciatica stands in sharp contrast to the neuralgic neuritides of the upper extremities.

Gout is an infrequent causative factor, while diabetes is more common, especially for double-sided sciaticas. Double sciaticas may also be the expression of a tumor of the pelvis, of pressure due to a gravid uterus, of venous stasis, spinal cord tumor, or new growths of the pelvis. Occasionally prostatic enlargement of tuberculous or

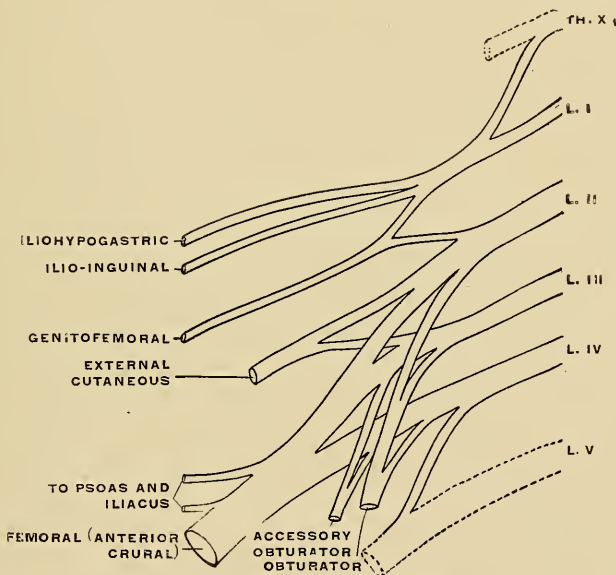


FIG. 128.—Diagram of the lumbar plexus.

gonorrhoeal origin have given rise to sciatic pains. Exposure to cold, with prolonged standing is frequently met with in the histories, and occasionally in those who sit a great deal. Prolonged walking or marching may occasion an attack, and bicycling predisposes to mechanical injury of the nerve.

It is a comparatively common affection, particularly in men, being one of the most frequent neuralgias met with in dispensary practice. Chronic constipation as a cause should not be overlooked. The etiological factor in some cases is impossible to find; this is a result of our insufficient methods of examination.

Symptoms.—There is no one sciatica, there are many, and it is advisable at the outset to separate those cases in which the principal

symptoms are pain and inability to use the limb from those in which there is added nerve tenderness, with motor, sensory and trophic phenomena.

The pains rarely commence abruptly, but beginning more or less gradually from a sense of soreness to uneasiness with occasional twinges, gradually develop into well marked severe pains, usually at first more intense just beneath the sciatic notch, gradually extending from above downward to the entire distribution of the sciatic and some of the branches. There is usually considerable variation in the

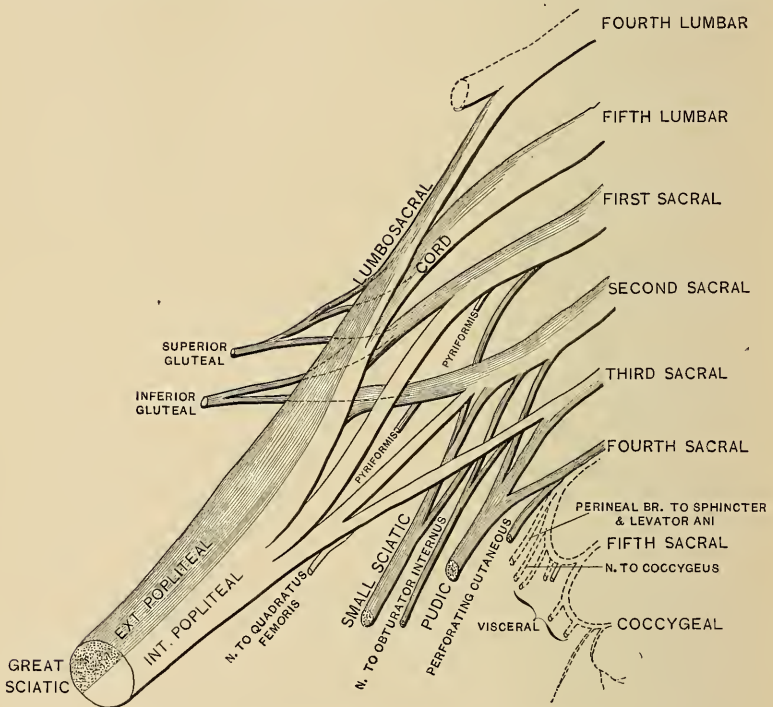


FIG. 129.—Plan of sacral plexus with the pudendal plexus. (Gerrish.)

character of the pain during an attack. Some patients suffer for some time simply from boring, dull pains, while others have excruciatingly sharp, stabbing twinges that make the slightest movements impossible. Rarely continuous, the pains come in attacks, sparing almost no region of the distribution of the plexus either en masse or picking out special branches.

The pain in the proximal portions of the leg is usually deep seated, but becomes more superficial distally. In some patients an extensive series of involuntary adaptive positions take place with slight scoliosis in order to seek the most comfortable position, not only of the thigh

and leg, but of the pelvis, or even the vertebral column. In sitting, similar adaptive positions are taken to try to avoid pressure.

Painful pressure points were pointed out as fairly constant by Valleix. The most important of these are situated at the sacroiliac joint, the sciatic notch, or the gluteal point on the gluteal fold over the nerve, and the peroneal point at the head of the peroneus. In some cases, often mild, pressure points are lacking. Lasègue's phenomenon is of considerable diagnostic significance. It is brought out either in the lying or sitting position. The patient's foot is grasped with one hand, the other placed upon the knee, and keeping the leg stiffly extended the thigh is flexed upon the hip, when a severe pain develops beneath the knee or higher up in the course of the nerve. In the sitting position the pain is more apt to be beneath the knee, since the extension on the pelvis cannot be made so extreme.

With the general extension of the neuritic process—as in many alcoholic cases—the entire nerve may be sensitive to pressure.

Minor¹ has described some interesting findings on having the patients arise from a prone posture. Patients with well-marked sciatica can rarely get up—without excessive pain—with the arms crossed. They put their two hands behind them, push the hips backward, between the arms, flex the knees slowly beneath the buttocks, then gradually with one hand on the hips, and then on the knee, the other balancing in the air gradually come to a standing position. The procedure is not invariable, but is useful in gaining some idea as to simulation, and as to the diagnosis of a lumbago. The untrained simulator gets up in a variety of ways, the lumbago patient usually rises on all fours, the arms in front, somewhat as does the pseudohypertrophic dystrophy patient.

In those groups of patients with more serious neuritic involvements one has added the signs of muscular atrophy, of circumscribed paralyses, of fibrillary contractions, of contractures, of sensory disorders, of trophic disturbances.

The atrophies may be true atrophies, but are usually position or disuse atrophies. They are found by palpation, or by measurements.

Moreover the atrophies may be segmentary, or radicular, in either of which instances a localizing diagnosis is aided. The electrical examination of the muscles in sciatic neuritis is usually contradictory. In positive cases with partial R. D. the nature of the process becomes clearer. Fibrillary contractions may occur in the parietic muscles.

The patellar reflex, in the beginning of the process, is usually increased on the affected side, while if marked neuritis be present it may be diminished or absent. The Achilles tendon reflex shows a similar reaction. Oppenheim has called attention to a mild degree of flabbiness or diminution in the size of the Achilles tendon on the

¹ Deut. med. Woch., 1898.

affected side. A pseudo-Babinski is described, largely a plantar flexion of the smaller toes, with immobility of the great toe.

Sensibility is often unaffected, but careful testing, following Head's methods, may show a hypesthesia to touch, pain, and temperature. Occasionally the disturbance of sensibility shows a marked radicular distribution, which speaks for a more or less localized process in the plexus, frequently of a syphilitic, meningomyelitic, or osteo-arthritic nature.

Trophic and vasomotor phenomena are not infrequent, consisting either of acroparesthesiæ, erythemas, local cyanoses, diminished or increased perspiration, changes in the growth and character of the hair or even the nails. True herpes is rare. Glycosuria, polyuria, azoturia are rare complications.

Course.—In the early attacks in healthy adults, the prognosis is good. The patient usually recovers in from six to eight weeks under proper treatment. A failure to respond should excite suspicion as to the diagnosis, especially with reference to tabes, to a spinal growth or a radiculitis. Recurrent cases usually develop a tormenting chronicity, which exhausts one's therapeutic resources, and occasionally drives the patient into a mental state which suicide or chronic morphinism alone terminates. Fortunately such cases are becoming rarer with better methods of diagnosis and enlarged therapeutic resources.

Clinical Forms.—Certain variants—largely based on etiological concepts—may be met with. Fournier's gonorrhœal sciatica with an acute onset, slight temperature, with prostatic and articular complications is one. Brissaud has described a spasmodic type with increased tendon reflexes, contractures in the periarticular muscles of the hip, and trepidation or pseudoclonus. Quenu's varicose sciatica, which has certain analogies to the intermittent claudication arteriosclerotic type, is characterized by deep-seated pain, and a very protracted onset and chronic course. Hysterical sciaticas may always be expected, but they are extremely rare.

Diagnosis.—The increased knowledge afforded by lumbar puncture, *x*-rays, and finer modes of testing for sensory disturbances is dismembering the old sciatica group fairly rapidly. Of the more common diagnostic errors, tabes dorsalis and lumbago call for special mention.

Tabes lacks the pressure points, the Lasègue phenomenon, and usually shows the lost knee-jerks, lost Achilles jerk, and possible Argyll-Robertson phenomenon. The pains are usually bilateral.

Lumbago is usually much relieved by the recumbent posture, and is increased by the movements of the trunk; the site of the pain is as a rule higher.

Lumbosacral radiculitis calls for special mention since many of the classical chronic sciaticas fall under this disorder.

Muscular rheumatism lacks the pressure points, Lasègue's sign, and the pains are more diffuse.

Spinal cord tumors in their initial symptomatology cause sciatic

pains, usually bilateral, occasionally unilateral, but soon careful sensory examination shows anomalies, special localizing anomalies, and pareses and trophic symptoms point to a severe lesion of the cord.

Hip-joint disease lacks the classical situation of the pains. Arthritis deformans cases with sciatic pains show diminished power of abduction and adduction and the joints are painful.

Intermittent claudication occasionally gives rise to diagnostic difficulties. Its arteriosclerotic nature is revealed by palpation of the bloodvessels, and by x-ray examinations which show the tortuous modified bloodvessels.

Achillodynia, Morton's tarsalgia, and flat-foot are occasional difficulties.

Therapy.—Rest and quiet are the first essentials. Then an etiological therapy becomes imperative. Mercury for syphilitic cases, quinin for malarial cases, surgical intervention for pressure cases, diet for diabetic cases, etc.

While one is waiting to obtain a clear notion as to etiology, general treatment may be necessary. Such treatment should always be regarded as provisional, not final.

For the treatment of the pain, analgesics are imperative. These are numerous, and different patients will respond to different members of the group. Even during an attack it may be found that one analgesic has lost its value, and another must be substituted. It is of value to bear the chemical structure of the various analgesics in mind in one's therapeutic endeavors. Antipyrin, or its related products, aspirin, salipyrin, pyramidon; the amido-phenol series, with phenacetin, lactophenin, exalgin, apolysin, citrophen, phenosal, phenocol, and salocol, as representatives are often of signal service, but require considerable testing. Acetanilid itself, with its compounds, is in wide use. They have active analgesic properties, but one runs the risk of cheap and impure products, since acetanilid is so extensively manufactured largely as a by-product. These analgesics can be used in combination, when smaller doses of the two or three in use are more efficient than large doses which often have marked toxic action, either on the blood cells (acetanilid, amido-phenol series) or on the vasomotors (antipyrin derivatives).

In the presence of anemia, iron and arsenic should be added.

Counter-irritation is very useful especially. It is best practised by means of the actual cautery (Paquelin) but mustard plasters, cantharides plaster, etc., are useful adjuvants. Applications should be made along the nerve trunks.

Hydrotherapy is often extremely valuable but must be employed with reason. A too energetic hydrotherapy with massage, often aggravates a sciatic pain, especially in the initial stages when rest is so imperative. Later hot packs, mud baths, spray douches, with mild massage are indicated. In many patients the treatments carried out in bath resorts is especially indicated. Hot air treatment is not

well borne in the initial stages, but later is grateful and of therapeutic value.

Direct nerve injections of substances having a degenerative action in nerve fibers, osmic acid, carbolic acid, etc., are to be condemned. Infiltration methods, using water or cocaine, or allied substances, or various mixtures have more to recommend them. Schlosser has reported excellent results but has also had permanent palsies follow his injections.

At times it may be deemed necessary, by reason of the severe pain, to practise injections of stovaine, cocaine or allied substances into the region of the cauda or into the spinal cord (Corning). Such injections are useful, but their action is temporary as a rule. Nerve stretching is to be condemned.

The opium derivatives should be used only as a last resort.

Electrotherapy.—The older methods of galvanization and faradization are useful in a few cases, but on the whole are unsatisfactory. Sinusoidal currents are more valuable, while the Leduc rapidly alternating currents are almost always of some service in relieving pain but not in curing. High frequency currents with the use of the ultra-violet rays at times give extremely satisfactory results from the same stand-point.

Lumbo-abdominal.—These occupy the lower half of the trunk, and are extremely variable. The chief nerves involved are the iliohypogastric and its branches, the inguinal, and genitocrural. Strict localization to one trunk is rare, and men are more frequently affected than women. The chief causes in addition to those of general moment are local inflammatory conditions or new growths involving the plexus or some of its branches. The pains are usually unilateral, occasionally bilateral, involve the region of the back below the ribs, the gluteal region, the abdominal and inguinal areas, the scrotum, or the labia. The chief Valleix's points are over the lumbar vertebræ, the hip or iliac point, hypogastric point, and the scrotal point. Lumbo-abdominal pains are usually accompanied by intercostal pains above or thigh pains below.

Testicular Neuralgia.—Astley Cooper termed this neuralgia the "irritable testicle." The pains are usually unilateral and pass into the testicle which may be swollen and tender to the touch. The pain not infrequently passes into the leg and back, and the patient may have an attack of vomiting. Bernhardt notes that the pain may be so intense as to cause the patient to seek castration. The affection is an obstinate one, and is not helped, as a rule, by removal of the testicle. Diagnosis involves a rigid exclusion of organic disorder of the testicle although many affections (gonorrhœa, tuberculosis, chronic prostatitis, etc.) are not infrequently accompanied by persistent neuralgic pains.

Crural Neuralgia.—The crural or femoral nerve is here implicated. The pain extends in the upper front and inner side of the thigh, to the knee, and further through the saphenous distribution to the ankle

and inner aspect of foot, extending as far as the big toe. It is almost entirely confined to men, and shows considerable variability as to the branch involved. It not infrequently accompanies a sciatica. Special etiological features are found in fecal impaction, or even chronic constipation, disease of the hip or knee bones, enlargement of the inguinal glands, aneurism of the iliac artery. Charcot called attention to the frequent association of crural neuralgia and diabetes. Spinal arthritis is an obscure cause.

Movements of the thigh usually are painful and the patient comes to bend his body forward in a strained position. The painful points of greatest frequency are just below Poupart's ligament, just within the inner condyle, over the malleolus, inner side of the instep, and one over the great toe. Neuro-atrophic changes usually occur in the quadriceps, but the patellar reflex is rarely affected, save when a definite neuritis is present. Herpes, reddening, hyperesthesia are not infrequent. In the diagnosis, disease of the inguinal vessels is to be looked for, as well as intrapelvic disorders, new growths, etc. Crural neuralgias have a fairly good prognosis.

Femoral Neuralgia.—Here the cutaneous femoris lateralis, arising higher up in the pelvis, is involved. The pain is felt in the upper and outer aspects of the thigh, extending to the knee. A painful point over the anterior spinous process of the ilium is usual. Paresthesia in the distribution of this nerve has been extensively studied (*Meralgia paresthetica*). The relation of the pressure of corsets in the causation of this type of neuralgia has been pointed out by Freud, and much sitting in adipose individuals is frequently associated with this neuralgia. The prognosis is favorable.

Obturator Neuralgia.—Lesions of this nerve are fairly constant as a result of the pressure of the intestinal loops of a hernia. The pain is located in the inner side of the thigh, and is accompanied by a feeling of stiffness, creepy, crawly feelings of the skin, and inability to bring the thigh toward the middle line of the body.

Neuralgias of the Pudendal Plexus.—A large number of neuralgias of the genital plexus are recorded. The median hemorrhoidal branches, distributed to the rectum, bladder, and vagina, the inferior branches to the anus, and the pudendal nerve supplying the testicular sac, the labia, penis, urethra, and clitoris, are the chief nerves involved. The general terms, spermatic neuralgia, anal neuralgia, perineal neuralgia, rectal neuralgia, vesical neuralgia or cystalgia, urethralgia, prostatic neuralgia, penis neuralgia, irritable uterus, ovarian neuralgia, are utilized to describe these different affections. These neuralgias are very rare, but often very obstinate. Spermatic neuralgias are among the most frequent, and are not infrequently accompanied by painful priapism, perhaps ejaculation.

Since the advent of bicycle riding neuralgias of this general region have been on the increase. The ovarian neuralgias are complex, and more often come within the domain of the gynecologist, as structural

defects are usually the underlying causes. Localized herpetic eruptions accompany neuralgias of this plexus. Lesions of the cauda equina are to be carefully excluded in neuralgias of this region.

Neuralgias of Coccygeal Plexus.—Coccygodynia, painful coccyx, is a not infrequent disorder in women, especially in multiparæ and in the badly constipated. Trauma and caries are frequent causes. The hysterical coccyx is not infrequent, and referred coccygeal pains are common. The pain is so intense at times that defecation is rendered impossible; the patient cannot sit, and a grave neurasthenic condition supervenes. The medicolegal significance of coccygodynia is real, appearing frequently as a local symptom of a general traumatic neurosis. Surgeons frequently lay considerable stress on a freely movable coccyx in accident litigation. A just estimate of the true bearing of an injury to the coccyx can only be arrived at by a careful survey of all of the factors of the particular case.

Local treatment is seldom efficacious save in the truly neuralgic types. Resection is rarely a justifiable procedure.

HERPES ZOSTER: SHINGLES: ZONA. RADICULOGANGLIONIC SYNDROME (ACUTE POSTERIOR POLIOMYELITIS).

In a broad sense zoster consists of a special type of painful erythematous eruption with formation of vesicles following the radicular distribution of the segment involved, due to disease of the posterior roots and the sensory ganglion. In this sense it may be due to an acute or chronic meningitis, tabes, Pott's disease, carcinoma of the vertebræ, acute infectious diseases, intoxications or other lesions implicating the posterior roots and the ganglion (Symptomatic Zoster).

In a narrower sense it may be conceived of as a specific infectious disease affecting the ganglion cells in the posterior spinal ganglia and the adjacent fibrillary structures (essential zoster or posterior poliomyelitis).

History.—Zona was first well described by Rayer in 1835, although notes on its occurrence date from Hippocratic times. Baerensprung, in 1861, gave the first important monograph, and pointed out the implication of the ganglia as an essential feature of the disease, while Head and Campbell (1900) called particular attention to the specific infectious type. Rosenow and Oftedal¹ have isolated streptococci from the ganglia.

Etiology.—Nearly all of the general causes which give rise to a neuralgia or a neuritis may by an extension or an intensification of the pathological process involve the posterior ganglia and thus develop a herpes. In poisoning by arsenic and carbon monoxide these ganglia seem to be specially affected, and the acute gastro-intestinal affections, pneumonia, and tuberculosis are not infrequently contributory factors.

¹ Jour. Amer. Med. Assoc., June 12, 1915.

Trousseau first called attention to the zoster, which was a specific infection, which type has been so extensively studied by Head and Campbell. Epidemics of zoster point to the truth of this position.

Symptoms.—Neuralgic pains and a skin eruption constitute the main symptoms. The disorder shows a slightly different order of development according to the etiological factors. In the pure or essential zoster (acute posterior poliomyelitis) there is usually a feeling of malaise, a slight temperature, and gastro-intestinal disturbances, then the patient has neuralgic pains which may be mild and superficial, burning or pricking, or deep and extremely severe, and in from three to four days an eruption develops. There is marked



FIG. 130.—Herpes zoster. Typical thoracic location. (Knowles.)

hyperesthesia of the skin along the affected segment, with redness, and suddenly or gradually there appears a group of vesicles varying in size from a few millimeters to a few centimeters. These vesicles are rarely confluent, and the fluid, which is at first serous, sometimes tinged with blood, may later become purulent. The vesicles gradually dry, leaving a scaly, yellowish-brown, stained scar which persists for a long period. Ulceration or gangrene occasionally occurs. The cycle occupies about four to eight days. There is usually some anesthesia to both epicritic and protopathic sensibility after the acute stage has passed. One attack seems to confer immunity.

In the symptomatic zoster there is rarely fever or gastro-intestinal disturbance, the development of the eruption is usually irregular, and

it often shows a chronic character. Symptomatic zona may involve both sides, whereas the infectious type is practically invariably one-sided.

In the dorsal types only is the girdle distribution maintained (intercostal herpes zoster), whereas involvement of the Gasserian, cervical, lumbar or sacral roots gives rise to irregular eruption appearances by reason of the segmental complexities of these regions.

Ophthalmic zoster is an especially severe type, occurring in individuals above middle life, and often accompanied by alcoholic and arteriosclerotic factors. It may develop apparently like an erysipelas of the face, with severe neuralgia, and then a widespread vesicular eruption, even involving the mucous surface, develops. Ocular complications, conjunctivitis, keratitis, iritis, of a severe nature, are not infrequent. Facial palsy may be present in zoster of the Gasserian ganglion or of the geniculate. A symptomatic ophthalmic zoster due to lesions in the region of the pons occurs.

Pathology.—In essential zona there is an acute, often hemorrhagic inflammation in the sensory ganglia. These are swollen, the capsule notably thickened, with marked infiltration of leukocytes. The ganglion cells are in part destroyed or damaged, and the contiguous fibrillary structures, both central and peripheral, are also involved in the inflammatory reaction. The inflammation is usually limited to a few ganglia. In the spinal cord secondary degenerations have been observed, and occasionally there is an extension of the general process to the cord. In certain cases of what appears to be essential zona the ganglia have been free, the only lesions found being those of a neuritis. There is an inconstant lymphocytosis of the cerebrospinal fluid in the infectious zonas.

Treatment.—For the symptomatic cases, the cause must be found. Otherwise the treatment is purely symptomatic. Local applications of zinc oxide ointment for protection and the use of mild antiseptic to prevent suppuration are advisable. For the pains the analgesic remedies already spoken of in the treatment of neuralgia, are useful. Gastro-intestinal therapy relieves the discomfort and itching somewhat, and may possibly limit the accumulation of a possible secondary irritant.

RADICULITIS.

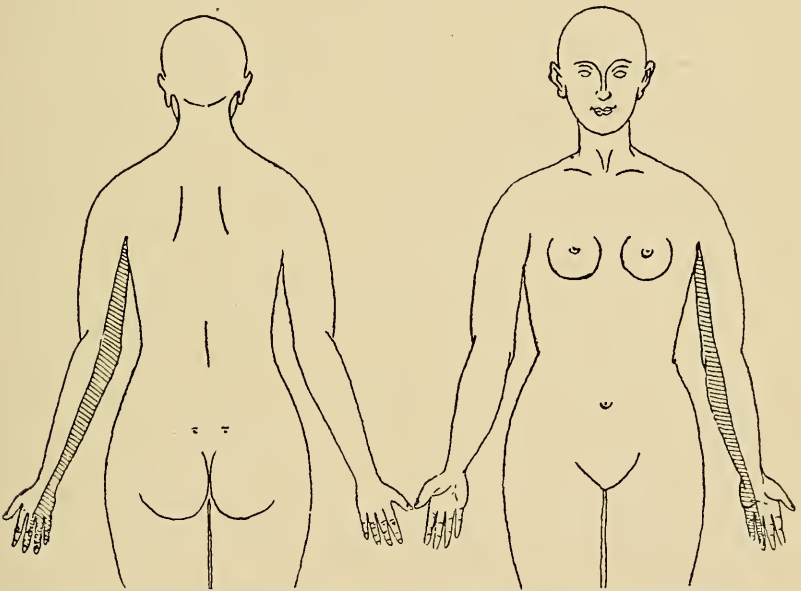
The radicular syndrome, often confused with neuralgia and neuritis, is due to an inflammation of the sensory nerve roots, usually of the brachial, and of the lumbosacral plexuses.

Attention has been given to it chiefly by French neurologists, notably Dejerine (1905) and his pupils.¹

Symptoms.—These are neuralgic pains, which are usually severe, yet very variable. They usually occur in crises, and are sticking

¹ Dejerine et Thomas. *Maladies de la moelle épinière*, 1909.

and lancinating in character, at times extremely intense; not infrequently resembling the pains of tabes. The pains have a tendency to remit and then to recur at shorter intervals, leaving a certain soreness behind. The nerve trunks are usually not markedly painful. There is usually a marked hyperesthesia over the radicular segment, It does not follow the peripheral distribution as in a non-radicular neuralgia. This hyperesthesia is usually followed by an anesthesia to both epicritic and protopathic sensibility, and occasionally bony sensibility is involved as well if the inflammatory reaction is intense. Deep sensibility may be so involved as to cause astereognosis. Paresthesiæ and acroparesthesiæ are common.



FIGS. 131 and 132.—Topography of the sensory disturbance in a syphilitic radiculitis (type Klumpke). The C_8 and $D_{1,2}$ distribution are involved. (Dejerine.)

It is essential that these features, which may be found in other affections, be radicular in their distribution. They are not segmentary, *i. e.*, involving the hand, the forearm, or the arm; nor do they follow the peripheral nerve distribution. They are distributed in long bands down the arm or the leg, corresponding to the root segments involved. (See Figs. 133, 134, and also Figs. 11 to 16, and Plates X and XI).

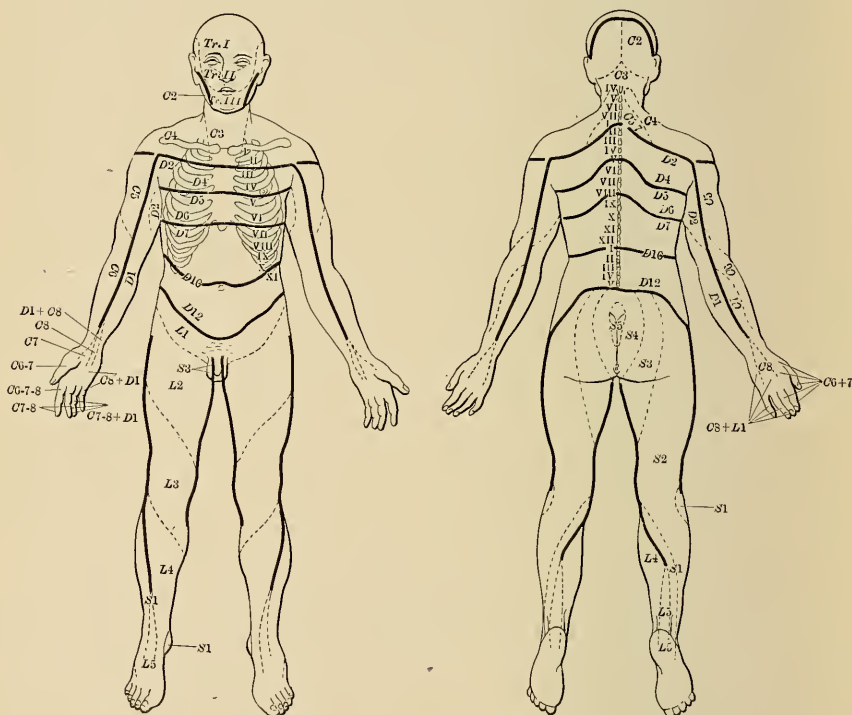
It is a striking fact that sneezing or coughing may bring on a paroxysm of pain in the cervicodorsal plexus, and coughing and straining at stool may bring on pain in a sciatic radiculitis.

The knee-jerks are primarily exaggerated, later diminished or lost. In the upper extremity it is rare to find a pure radiculitis, *i. e.*, one

without some motor involvement, while for the lower limb the great majority of the classical cases of sciatica are due to a radiculitis.

In the mixed cases, involvement of the anterior roots causes muscular atrophies, likewise radicular and not peripheral in their distribution. The atrophy is rarely accompanied by fibrillary twitchings, nor by spasmodic contractions.

Ataxias may be met with. Lasègue's sign is commonly found (Rousellier, Paris Thèse 1907). Vallieux's points are not constant.



FIGS. 133 and 134.—Scheme of root (radicular) segment distribution. (Flatau.)
(Compare with Figs. 19 to 22.)

Trophic disturbances, particularly in the joints, and vasomotor signs (cyanosis) are found in long-standing cases. In rare instances the anterior roots are involved primarily.

Diagnosis.—Lumbar puncture may reveal a lymphocytosis. As noted, radiculitis of the lumbosacral plexus has for the most part been grouped with the sciatic neuralgias; both under the so-called true and the symptomatic neuralgias.

With extremely careful sensory tests, following the procedures of Head and Dejerine, the radicular distribution of the hyperesthesiæ, the anesthesiæ or the atrophies is sufficient to determine a diagnostic

picture. The crural nerve is more often involved in the radicular process than in the peripheral sciaticas.¹

The presence of ataxia in the lower limbs, occasionally in the upper, Romberg's sign, and the frequent loss of the knee-jerks often leads to the mistaken diagnosis of tabes. From one point of view tabes begins as a radiculitis, and the diagnostic difficulty centers about the etiological element. Since so much of radiculitis of the lower extremity is due to syphilis, the ordinary lumbar puncture and Wassermann tests are essential to make the diagnosis positive. In tabes one must also take into consideration the involvements of the cranial nerves, Argyll-Robertson pupil, etc.

Cervical radiculitis naturally travels under the guise of a cervico-brachial neuralgia. Here the greater implication of the sensory system is enough to make a diagnosis. In mild early cases the diagnosis of a radiculitis is impossible. Muscular atrophy is a frequent complication of cervical radiculitis. Furthermore, pupillary phenomena (Klumpke) are common in this type.

Brachial radiculitis gives rise at times to a typical Aran-Duchenne atrophy—the biceps, anterior brachial, supinator longus and deltoid—the fifth and sixth cervical roots being chiefly involved. The Klumpke oculopupillary phenomena, *i. e.*, diminution in size of the palpebral fissure, slight retrogression of the eyeball and miosis are present if the last cervical and first dorsal roots are involved.

Disseminated types are described by Dejerine² with practically all the signs of a tabes.

Occasionally cerebral tumors cause the symptoms of a radiculitis with alterations in the posterior columns and radicular lesions (Collier, 1899; Nageotte, 1909; Raymond, 1907). Both limbs may be involved.

Acroparesthesia was originally described by Putnam (1882), named by Schultze, and then shown by Pick to be due to a radicular or intraspinal involvement, chiefly in the lower half of the cervical region.

It consists, in the periodic cases, of pains or burning, or cold sensations, usually in the fingers, accompanied by blanching and coldness of the skin with the sensation of engorgement and extreme heaviness of the hand or the fingers.

Treatment.—Spontaneous radiculitis seems to be preëminently syphilitic. Hence an antisiphilitic treatment is indicated in every case of suspected radiculitis. Other forms of meningitis may produce it, however. The prognosis is good in the syphilitic forms, but less so for the others. Many patients with cervical and brachial radiculitis recover spontaneously, after from six to ten months. Treatment seems to alleviate but not cure. The general treatment for a neuritic-neuralgia is indicated. Violet-ray exposure should be tried in the intractable cases.

¹ Dejerine, *Semiologie du système nerveux*, 1914.

² *Rev. Neurol.*, 1904, p. 524.

NEURITIS.

Neuritis is a generalized inflammation of the peripheral nerves involving in varying degrees of completeness the motor, sensory and trophic elements. In old-standing cases the spinal portions of the neurones are implicated.

The inflammatory changes may proceed either from the perineurium, from the endoneurium or involve the axis cylinder. These may be poisoned and degenerate (parenchymatous inflammation). The pathological nature of the lesion bears little relation to the general symptomatology, and pathologically considered there is considerable interplay of the various processes.

Etiology.—A vast variety of causes may bring about a neuritis, seen either as the result of acute toxic parenchymatous changes, acute degeneration due to actual pressure or injury, or some acute or chronic inflammatory changes following a variety of noxa. The most important of these causes are as follows:

Infections.—Neuritis may result from the toxins of microorganisms, as those of diphtheria, tuberculosis, syphilis, influenza, smallpox, dysentery, typhoid fever, pneumonia, streptococcus, occasionally measles, scarlet fever, influenza, whooping-cough, etc. Practically there is no infectious disease that has not produced a toxic, usually parenchymatous, neuritis. A second group of infections, such as leprosy, beri beri, malaria, rabies, bring about neuritis, but here the mechanism is different.

Intoxications.—Next in general importance are the intoxications, either exogenous or endogenous. Alcohol plays the chief role, while arsenic, lead, carbon monoxide, sulphur, and anilin compounds are rarer exciting causes. Of the auto-intoxications diabetes is perhaps the most important. Others are gout, leukemia, and anemia.

Trauma.—A third important cause for the neuritic process is trauma to the nerve, either as the result of accident or injury, or from the pressure of new growths, dislocations, false positions or other anomalies of structure combined with the practice of certain occupations (professional neuritis).

Less important causes, numerically speaking, are extensions of inflammatory processes (ascending neuritis), inflammations above joints, inflammations of adjacent organs, etc.

Symptoms.—The symptomatology of neuritis varies enormously, not only so far as the etiological factors which determine general trends of reaction are concerned, but also with reference to location, acuteness of onset, etc.

Only the symptoms of the generalized process will be considered here, reserving for the sections on Pareses or Paralysis, both of the plexuses and of the peripheral nerves a more detailed description of the various isolated types.

It has already been indicated that the conceptions neuralgia, radicu-

litis and neuritis are very flexible—it is only for the sake of description that one draws more or less arbitrary lines between them. Such do not exist in nature.

There is a generalized type of neuritis which of and by itself constitutes a fairly definite syndrome. This is so-called multiple neuritis, or polyneuritis. It is largely due to toxemias, either of organic or inorganic nature.

Under the general caption of peripheral neuritis, one considers a large number of peripheral palsies, while as localized neuritis one has to consider a number of the professional neuritides.

Polyneuritis, Multiple Neuritis.—This is a general, widely distributed, diffuse, parenchymatous neuritis in which the entire peripheral neuron is involved. Secondary degenerations of the cerebral neurons take place.

Etiology.—Multiple neuritis is almost invariably due to some toxemia. Such toxemias may be due to (1) alcohol, lead, arsenic, carbon monoxide, bisulphide of carbon, sulphuric acid, alcohol, and some of the rarer metals: mercury, copper, phosphorus, etc., (2) or to the toxins of acute or chronic infectious diseases, such as smallpox, typhoid fever, grippe, measles, scarlet fever, diphtheria, pneumonia, dysenteries, streptococcemias, leprosy, tuberculosis and syphilis, or the inflammation may result from (3) auto-intoxications such as diabetes, leukemias, severe anemias, etc. Acute chilling of the body is held to be responsible for certain cases, particularly in the presence of some infectious diseases, or acute toxemias, notably in alcoholic cases, cases of rabies, influenza, etc.

Occurrence.—No general laws can be made with reference to occurrence because of the wide range of etiological factors. Thus, factors of age, of social state, of occupation, etc., which may co-exist, for instance, with the occurrence of diphtheria, have nothing whatever to do with the same class of facts in cases of alcoholic polyneuritis.

Symptoms.—Clinically considered, one meets with subacute and acute cases. In the subacute cases, which are in general milder, the patient usually begins to have a rapid progressive enfeeblement of the muscles, as a rule, of the lower extremities. There is rarely any fever at the onset, and the loss of power gradually extends from the peripheral segments toward the trunk. Thus, the extensors of the leg and of the foot first show weakness, and later those of the thigh and hip. At the same time, or closely following, the upper extremities may be involved, in accordance with the same general law, the muscles of the hand, wrist and forearm usually being primarily involved. There are occasional exceptions to this general law of progression, but they are comparatively rare.

It is further characteristic that the weakness and paralysis are more or less symmetrically distributed. Although one leg or one arm may show a greater amount of weakness than the other there is almost invariably quadrilateral involvement. In the milder cases quantita-

tive variations in the severity occur, and in the mild subacute cases the cranial nerves are less often diseased. Still the muscles of the abdomen, the diaphragm, the face, eyeballs or tongue may all suffer.

In the more severe cases, the implication of the pneumogastric is shown by tachycardia, dyspnea and feebleness of the pulse.

The superficial reflexes may first be exaggerated, but later become lost as a rule, and the tendon reflexes usually exhibit the same phenomenon. Disturbances of sensibility are usually more marked. Initial pain is more or less universal, but the sensations of actual pain are often preceded by tingling or creeping sensations, and the skin, muscles, nerve trunks and joints may all show hyperæsthesiæ. The Lasègue phenomenon is universally present.

Careful testing of epicritic sensibility may show no loss, although as a rule the sense of localization to light touch and the ability to distinguish between two points of a compass soon becomes somewhat diminished. A certain amount of loss of epicritic temperature sense may also be met with. In the milder cases the atrophy gradually disappears, and there is no tendency to the development of contractures, but in other cases contractures may follow, and the limbs become fixed and immobile.

In acute generalized polyneuritis the attack begins very abruptly, usually with high temperature and chill, headache, malaise, suppression of urine, albuminuria, and the general signs of an acute illness. Paralysis develops very rapidly, usually involving the lower extremities first, and gradually ascending the trunk and the arms, closely resembling the ascending type of acute anterior poliomyelitis (Landry). There is great tenderness on pressure over the nerve trunks, sharp, shooting pains, marked hyperæsthesia of the muscles and the Lasègue phenomenon. The reflexes are rapidly abolished, the patient loses epicritic sensibility, is unable to localize touch, cannot distinguish points of the compass, but rarely loses sense of pain or of deep pressure. The sphincters are not usually involved, except *in extremis*. Atrophies, contractures, trophic disturbances of the skin, such as glossy skin, pemphigus-like eruptions, perforating ulcers, fragile nails, thick and fragile hairs, etc., develop. Then ocular palsies are met with, occasionally facial palsy, tinnitus frequently results from cochlear disturbance, and neuritic vertigo from vestibular disorder is found. Pupillary inequalities are frequent in the severe cases. Sluggish light and accommodation reflexes are fairly constant and occasionally a true Argyll-Robertson pupil is found. Loss of accommodation with retained light reflexes is met with occasionally. Amaurosis, complete or partial, is not infrequent. (See Interstitial Optic Neuritis, p. 189.)

The cranial nerve nuclei are not infrequently involved. In the fatal cases the implication of the pneumogastric causes death. The symptoms are those of asphyxiation, or with cardiac irregularity and paralysis of that organ.

Course.—**Diagnosis.**—**Treatment.**—Inasmuch as multiple neuritis varies not only with reference to its symptomatology and course, but offers special diagnostic problems according to the etiological factors, and since the treatment must depend upon a due consideration of the etiological factors, it is best to discuss these problems under special heads.

Alcoholic Multiple Neuritis.—This is the most common of all the types of multiple neuritis. Any form of alcohol-containing drink can cause it, including beer. Cologne, ether and other substances of the marsh-gas series are among the etiological curiosities as causing neuritis.

It is usually subacute in development, although occasionally, especially if the patient has been subjected to severe cold, the disease may begin in a very acute manner, and present the picture of a Landry syndrome. In a few instances an apoplectiform onset has been noted. Intercurrent infectious disease in an alcoholic may constitute the point of departure for a polyneuritis; this is especially true of influenza.

These patients usually complain of tingling sensations; of formication over the hands or down the legs, with occasional twinges of pain, particularly in the legs. The skin is usually hyperesthetic throughout the entire body, and the muscles become very sore. Such symptoms may be present for weeks or even months. The patient commences to notice difficulty in walking, inability to go up and down stairs and occasional falling when called upon for certain muscular exertion.

Examination of the muscular power at this stage shows marked weakness, particularly in the extensors of the feet, and of the hands. In walking there is a tendency to "drop-foot," and the patients lift the legs a little higher, and may show a characteristic flopping step superficially resembling that of the tabetic.

In this stage, before patients are confined to bed by reason of the muscular weakness, an exaggeration of the tendon reflexes is occasionally found, although diminution and loss becomes the rule. Atrophy, flabbiness of the muscles, and the trophic signs may then appear.

A number of these patients are able to be about, and are often mistaken for cases of beginning tabes, especially as there is very frequently a certain amount of ataxia, definitely marked in the lower extremities, less so in the upper. The sphincters are as a rule intact.

In some severe cases, in addition to the physical signs, a very definite psychosis develops. This is treated under the head of polyneuritic psychosis—chronic alcoholic delirium, Korsakow's psychosis. (See chapter on Toxic Psychoses.)

Course.—Alcoholic polyneuritis shows an innumerable number of varieties, but in the main it runs a subacute course. The patients go through the usual symptoms of chronic alcoholism, with tremor, sleeplessness, gastric disturbance and malnutrition. In the severe cases which develop marked mental signs—Korsakow's syndrome—

they usually go through one or more periods of acute delirium (delirium tremens).

Then the neuritic symptoms commence to appear, usually with fornication or other paresthesiæ in the arms and legs. Sudden twinges of pain, particularly in the lower limbs, are frequent, and an unusual sense of muscular fatigue develops. Dynamometer readings show this loss of power and excessive fatigability early in the disorder. The average case, if drinking continues, and the malnutrition and insomnia are not overcome, takes three or four to eight weeks for the development of definite palsies. The patients note great difficulty in going up and down stairs, with much palpitation and shortness of breath. Then on some sudden exertion they fall, or their legs shut up like a jack-knife under them. They totter in their walk, or show a steppage or ataxic gait. The disease may be arrested at this stage, and recovery



FIG. 135.—Alcoholic neuritis in chronic stage with contractures.

takes place with proper care. In advancing cases, locomotion becomes impossible. Foot-drop, wrist-drop, and ptosis may develop, and the atrophies, contractures, and trophic disturbances come on rapidly. The pains have increased in severity and frequency, and are apt to be excruciating. The hyperesthesia, which has been excessive, may now be gradually and irregularly supplanted by hypesthesia or anesthesia, hypalgesia, or analgesia, and extension to the cranial nerve nuclei may be looked for. Sensitive nerve trunks and Lasègue's phenomenon are invariably present.

The patient who has advanced to the stage of paralysis, atrophies, and trophic changes is usually confined to bed for several months, and then commences to make a slow and irregular recovery. It may be complete, but there is apt to be some local persistent impairment which may require treatment for years, especially if fibrotendinous

contractures have developed. In the Korsakow cases certain grades of residual mental impairment are extremely common.

No two cases of alcoholic polyneuritis are alike. There is a general tendency for the disorder to involve all four extremities in the pronounced cases, but the lower extremities are more severely implicated. Hyperesthesiæ and paresthesiæ are frequent. Special predilection is shown for the extensors of the foot and wrist. Pains are extremely severe and are universal. Blindness (amaurosis) is frequent; with methyl (wood) alcohol it usually comes on in advance of any other neuritic symptoms.

Treatment.—Treatment of alcoholic neuritis involves the absolute withdrawal of alcohol in any form, complete rest, forced feeding, particularly with foods rich in fat—milk, eggs, butter. Pain is best relieved by hot applications; continuous hot baths are very grateful if there is marked hyperesthesia of skin, muscles, and nerve trunks; the temperature should not exceed 96° to 97° F., if the bath is to be continued for any great length of time. Active catharsis and diaphoresis are essential in the early stages. After the stage of acute hyperesthesia is passed the forced feeding should be continued and the use of strychnine and electricity commenced. Great care should be taken in the selection of the hypnotics used to give sleep, since so many of them contain alcohol, and a few are directly poisonous to the nerve trunks. Bromides, hyoscin, or occasionally the opium derivatives may be employed. If an alcohol hypnotic seems necessary paraldehyde and chloral are indicated.

Glycerophosphites with calcium are valuable, preferably not given in an alcoholic medium. Massage and muscular movements of various types are indicated in the chronic stages, and most cases will repay constant working upon them. In the presence of contractures, surgical intervention may be necessary, but should be deferred until persistent massage and muscular therapy have been exhausted.

Lead Neuritis.—*Lead Palsy.*—In neuritis from lead poisoning which occurs from the gradual intake of lead in some form—drugs, hair dyes, cosmetics, contaminated water, certain occupations—plumbers, workers in type foundries, printers, glaze workers, potters, etc.—the picture is very different from that seen in alcoholism, although the terminal stages may be similar. The histological alterations are practically identical. Many cases are complicated with alcoholism.

Symptoms.—There are the initial gastro-intestinal symptoms of lead poisoning, furred tongue, constipation, attacks of colic, headaches, anemia, painful joints, and perhaps the signs of a nephritis. The gum lead line is frequently present. After a few months, or even a year or so of exposure, the neuritis develops, often after an attack of colic. It usually attacks the upper extremities, by preference, although there is always some slight involvement of the lower limbs. Definite lower limb palsies are rare, and usually occur only in children. The pareses predominate particularly in the extensors of the index finger and thumb

—the sensory symptoms, hyperesthesiæ, pains, nerve tenderness, Lasègue's phenomenon, paresthesiæ, are usually much less than in alcoholic neuritis. The supinator longus is very frequently spared. The paralyses are usually symmetrical, but may be quite irregular; the proximal trunk muscles may be involved—the distal ones free. This occasionally happens in alcoholic neuritis as well. Reactions of degeneration appear in the paretic muscles. Anesthesia, atrophies, trophic disturbances, and contractures are met with, but may be considered exceptional. Oculomotor palsies also occur, and optic nerve atrophy is not infrequent. Other cranial nerves, those of the larynx, pharynx, and face are also implicated, though rarely. Lead encephalopathies resembling those of alcohol are known.

Course and Treatment.—The course of lead polyneuritis is essentially chronic, lasting from several months to a year. The prognosis is usually favorable. The treatment is the same as for neuritis in general, with the addition of excessive diuresis, and the use of such remedies that may hasten lead elimination, such as potassium iodid.

Arsenical Neuritis.—Dejerine,¹ in 1883, first insisted on the essential similarities of neuritis of alcoholic and arsenical origin, which viewpoint has been amply verified in the extensive studies following a severe epidemic of arsenical polyneuritis in England, in 1899–1900.² The most frequent source of arsenical poisoning has been shown to come from impure glucose products; the sulphuric acid used to convert the starch containing arsenic. Other sources are wall-papers, certain manufactures (dye-stuffs), artificial flowers, cosmetics, beauty pastes and powders, hair dyes, and arsenic used in medicines. The pathological alterations (a parenchymatous neuritis) do not differ from those seen in alcoholic neuritis.

Symptoms.—Acute polyneuritis from arsenical poisoning is rare. It sets in shortly after the gastro-intestinal symptoms of acute toxemia have passed.

In the chronic cases the general symptoms of chronic arsenic intoxication are first observed. These are the anorexias, congestions of the upper respiratory tract (nasal catarrh, cough) or more frank diarrheas of gastro-intestinal irritation. The neuritis develops simultaneously with the symptoms of chronic intoxication.

As with alcoholic neuritis, sensory symptoms, paresthesiæ, hyperesthesiæ, numbness, shooting pains, sweating, develop first. A pigmented condition of the skin, most marked about normally pigmented areas, is found in the majority of the cases. The pigmentation may become very general and very dark, and is associated with herpetic, eczematous, or scaly, papular eruptions. Certain of the newer preparations of arsenic which have been extensively advocated for the treatment of syphilis, notably atoxyl and arsacetin, are reported to have caused optic nerve atrophy with blindness.

¹ Comptes Rendus, October, 1883, vol. xeviii, No. 17.

² Lancet, 1900, vol. i, p. 1610.

The signs of sore nerve trunks, Lasègue's phenomenon, loss of motor power in both extremities, are present in arsenical cases. Ataxias occur, and cases of arsenical neuritis have been confounded with tabes.

The prognosis is usually good, but the blindness has been permanent.

Other Intoxications.—Carbon monoxide, and illuminating gas poisoning, if severe and not lethal, frequently develop a severe grade of multiple neuritis not differing in any marked degree from alcoholic polyneuritis. The gases found in natural gas, and in many artificial gases contain the same chemical radicals as alcohol, and the toxic action is identical. In the very severe cases, polioencephalitis develops with multiple softenings, not entirely confined to the thalamus or corpora striata.

Carbon bisulphide, which is extensively used in rubber industries, may give rise to a multiple neuritis. The toxic ion is not definitely known. Similar poisoning results from sulphonal and trional, two sulphuric acid-alcohol hypnotics. A number of the nitrobenzol series can produce localized or general neuritis.

Phosphorus, mercury, copper, and silver can produce poisoning with the development of multiple neuritis.

Infectious Disease Types.—Mild or severe general neuritis has been observed to have occasionally followed practically every known infectious disease.

Diphtheria.—Here the neuritis is rarely generalized, and the cranial nerves bear the chief brunt of the poison. In mild forms the soft palate and pharyngeal muscles are first or alone implicated. Oculomotor palsies are also frequent. Some degree of facial palsy is also met with. In the severe types the larynx, tongue and the pneumogastric innervations become invaded.

Generalized forms occur, differing in no essential particular from the types already described.

Diphtheritic palsies may come on soon in the disease or they may follow a month or six weeks after the subsidence of the disease proper. For those patients who develop polyneuritis the diagnosis is usually grave. The cranial nerve types are usually less severe, although occasionally one finds pneumogastric palsies which are fatal.

Influenza.—The toxins of the influenza organism seem to have a special attraction for sensory nerve structures. Neuralgias, localized neuritides, are extremely common, and polyneuritis not a rarity. The polyneuritis is of a parenchymatous type, is usually mild, quite irregular, and differs in no essential manner from other types described. Its course is rarely over a few months and the prognosis is usually good.

Polyneuritis of a mild parenchymatous type occurs as a sequel of typhoid fever, smallpox, erysipelas, pneumonia, pleurisy, acute articular rheumatism, parotitis, gonorrhoea, dysentery, measles, Pasteur rabies treatment, whooping cough, and peripheral septicemia.

In chronic tuberculosis mild grades of neuritis are frequent, and

severe polyneuritis is occasionally met with in the marantic type. Syphilis rarely causes a polyneuritis, but it is known. Malaria is also a rare cause. Leprosy causes a specialized form.

PLEXUS PALSIES.

Plexus or root palsies are comparatively rare. They occur more often in the upper extremity. Brachial plexus palsy, as Erb's birth palsy, is the type. Lumbar plexus palsies rarely occur alone uncomplicated by cord lesions, since they are usually produced by compression, either resulting from tumor, fracture, Pott's disease, etc. Sacral plexus palsies, however, are not infrequent. They make up the classical cauda equina lesions, arising from the pressure of a tumor,

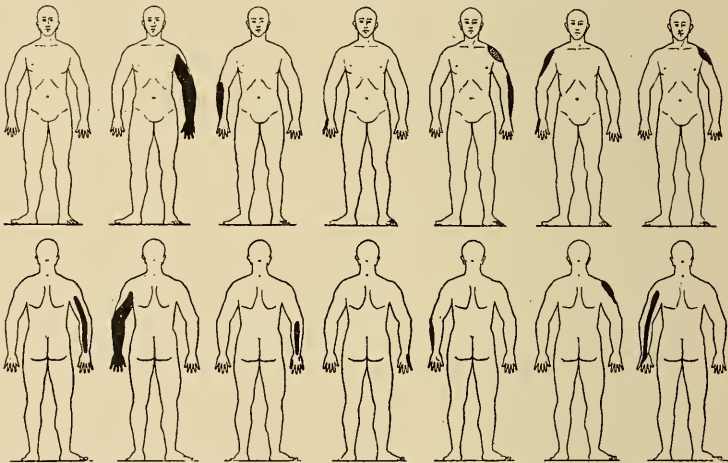


FIG. 136.—Sensory disturbances in seven cases of gunshot wounds of the brachial plexus. (Edinger.)

from hemorrhage, fracture of the sacrum, bony disease of the lumbar vertebræ, pelvic tumor, abscess, etc.

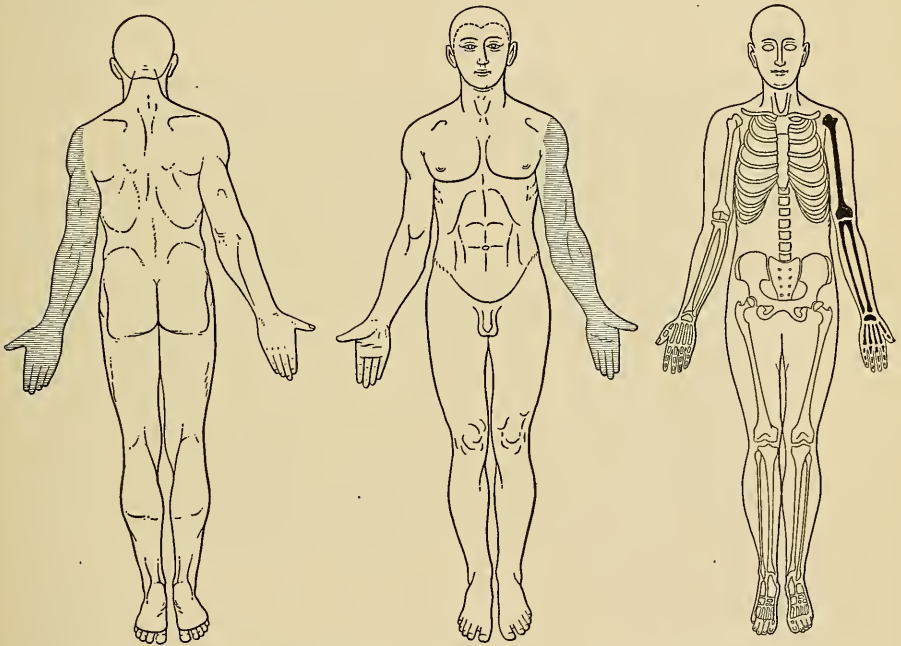
Brachial Plexus Palsies.—These most frequently arise from pulling or wrenching of the arms from accidents. Dislocation of the shoulder-joint can cause a plexus injury. Cervical rib is a rare cause, aneurism of the subclavian also. Gunshot wounds occasion them. (See Fig. 136.)

The plexus is made up of the lower four cervical and the upper dorsal root. Extraspinal as well as intraspinal lesions go to make up the symptom picture. This picture is rarely complete, but as the fibers making up the plexus have a fairly definite arrangement so far as muscular distribution is concerned, a study of the muscles involved gives a key to the roots injured.

The fifth and sixth cervical roots contain the fibers going to the

deltoid, biceps, brachialis anticus, supinator longus, supra- and subscapularis, the clavicular fascia of the pectoralis major, and the serratus magnus. The seventh cervical root contains the fibers distributed to the triceps, the sternal portion of the pectoralis major, to the dorsalis magnus, to the extension of the wrist, and also some filaments to the median and ulnar nerves.

The eighth cervical and the eighth dorsal form the brachial, internal cutaneous, ulnar, median and parts of the radial with their muscular innervations, as seen in the illustrations. (See Figs. 11 to 16, also Fig. 126.)



FIGS. 137, 138, and 139.—Distribution of radicular sensory disturbances in case of traumatic brachial palsy of the left side. There was total paralysis, atrophy, and loss of tendon reflexes, edema, and cyanosis of left arm. Abolition of all types of sensibility of the distribution of the *C₇* to *D₁*. Myosis and enophthalmus in left eye. (Dejerine-Klumpke.)

The clinical pictures seen are those of a total brachial plexus palsy; a superior and inferior type.

Total Brachial Palsy.—Here all of the muscles of the hand are paralyzed, those of the forearm, the arm and the shoulder. The arm hangs limp like a flail. In the early stages the skin is cyanosed, there are severe pains (in the accidental cases), and a loss of the secretion of sweat. Atrophy comes on quickly and is extreme, with loss of electrical responses. Trophic disturbances are usually present.

Sensory disturbances are present. There is loss to all forms of sensibility, including the sense of position in the hands and the forearm anteriorly and posteriorly. Anteriorly the upper border of anesthesia ceases just above the internal condyle; externally it extends to the insertion of the deltoid.

Furthermore, since cervical sympathetic fibers are represented in the communicating branch of the first dorsal, one finds oculopupillary signs first demonstrated by Madame Dejerine-Klumpke (1885). They consist of a contraction of the pupil on the paralyzed side, a diminution in the size of the palpebral fissure (pseudoptosis) and a retraction of the globe of the eye within the orbit (enophthalmus).

Furthermore, lesions here frequently cause changes in the sweat secretion. These are sharply delimited to the head, neck, face, to the



FIG. 140.—Erb's birth palsy. (Frauenthal.)

level of the third rib in front and the spine of the scapula behind, including the upper extremity, all confined to the side injured.

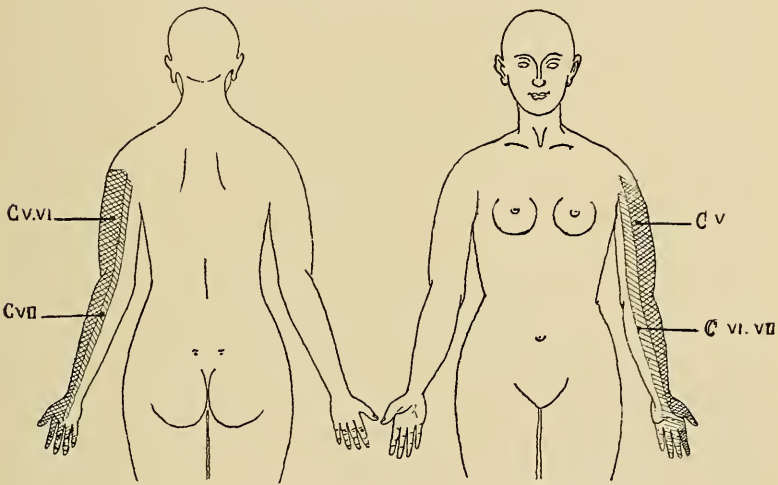
Inferior Root Type.—This has also been designated the Klumpke palsy. It follows a less complete lesion of the plexus, involving the eighth cervical and the first dorsal. It may result from injury, birth palsy (Erb's palsy), from cervical rib, or from syphilitic deposits. The small muscles of the hand are involved. The atrophy is rapid. There is edema of the skin, cyanosis, perhaps trophic changes in the nails. The anesthesia is less extensive, being limited to the distribution of the radial and internal cutaneous nerves. The oculopupillary signs are evident.

Superior Brachial Plexus Palsy.—Here the upper branches, fifth and sixth cervical, are involved. Duchenne described the earlier cases minutely. The palsied muscles have been mentioned. The sensory

disturbances are more extensive than those observed in the inferior type. The pupillary symptoms are absent.

Mixed Types.—These are more frequent. The more common form of so-called Erb's palsies belong here. Many of these are bilateral, the two arms, however, being dissimilarly involved. The study of the muscles involved in the atrophy, the electrical changes, the sensory changes, to light, touch, heat, cold, pain, bony sensibility, and to position, the presence of trophic disturbances (protopathic system changes), and the oculosupillary phenomena determine the roots involved. (See Figs. 11 to 16.)

There are a number of bony conditions which can determine brachial plexus palsies. Tuberculous, syphilitic, carcinomatous, sarcomatous,



FIGS. 141 and 142.—Topography of tactile, pain, and thermal sensory disturbances in a superior brachial palsy type due to injury to the shoulder. The fifth, sixth, and seventh cervical roots are involved. (Dejerine.)

arthritic infiltrations about the vertebral canal impinging upon the cords of the plexus can give rise to palsies of this type. Similar changes not infrequently also cause pictures which are often confused with brachial neuritis. Some of the severe arm pains with mild atrophies are forms of brachial radiculitis (*q. v.*), either inflammatory or traumatic in origin. In the course of a rheumatoid arthritis one not infrequently encounters these radicular disturbances which are undoubtedly referable to a vertebral arthritis.

Course.—General statements regarding the course are misleading. The majority of Erb's palsies due to obstetrical accidents recover, especially when only a few roots of the plexus are involved. Total separation from the cord, as in severe dislocations, falls, etc.; with

complete plexus palsy, usually means an incurable affection, not remediable by surgery.

The underlying etiological factor determines the course in a number of others. Palsies caused by cervical rib, or subclavian aneurism do not get well spontaneously, nor does iodide help them. Proper surgery, as indicated by the *x*-ray findings, may be of service.

Diagnosis.—The rarer nuclear and neuritic atrophies and dystrophies have been mistaken for these radicular palsies, but the study of the sensory changes should at once establish the differences. Spinal gliosis of the upper arm type will show dissociation symptoms, indicating the intramedullary nature of the lesion. Such a dissociation is a

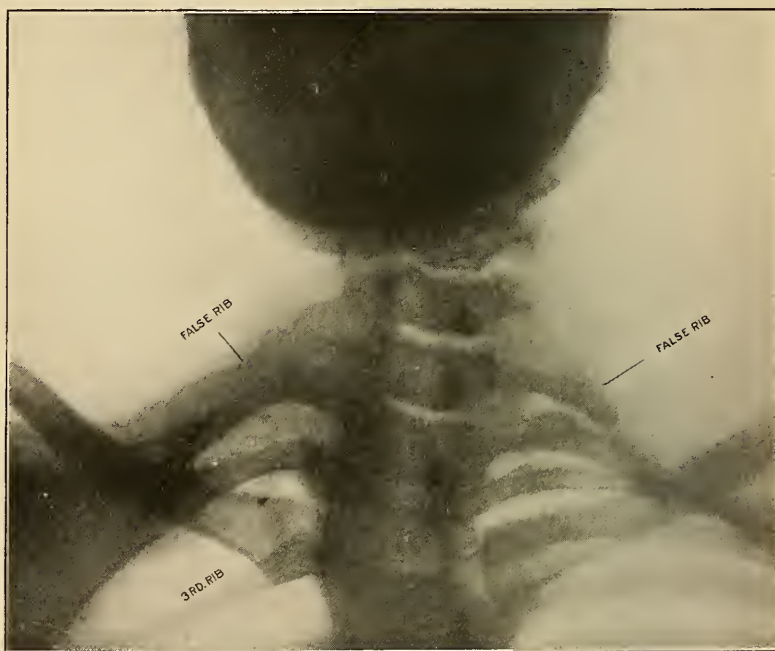


FIG. 143.—Double cervical rib, causing mixed type of brachial palsy. (Goodhart.)

retention of tactile sensibility with loss of pain and thermal sensibility. Various neuritides, lead, diabetes, have a peripheral rather than a root distribution. Certain exceptional cases of tabes, with atrophy, can be separated, but may require a cerebrospinal fluid examination. Syphilitic cervical spondylitis cases at times show these symptoms.

Treatment.—There is little treatment for the severe, tearing lesions of the plexus. Whether they can be looped up with other roots to advantage has to be answered in the future.

The cause of the palsies, other than tearing, can often be removed. Thus surgical treatment of neck glands, subclavian aneurisms, cervical

rib, spondylitis, and osteo-arthritis is efficacious. The treatment of a cervicodorsal Pott's by proper fixation, and the antisiphilitic treatment of a luetic spondylitis are satisfactory. Rare cases, often bilateral, due to profound secondary anemia, are often very refractory. True radicular neuritis is also stubborn.

The pains are best relieved by analgesics; antipyrin, aspirin, pyramidon, or other combinations are valuable. Heat is usually harmful, and massage contra-indicated in the early stages. Osteopathic manipulation is a dangerous procedure in the early stages. In certain osteo-arthritic cases it proves valuable later.

Simple counter-irritation over the site of the plexus, above the clavicle, is invaluable in many mild neuritic attacks; while for the severer attacks high-frequency currents, violet light therapy, Leduc current at times cause great relief.

Dietetic and general management in the neuritic cases is not to be overlooked. They need fats in ample proportion. This is best supplied through taking large quantities of milk.

Lumbosacral Plexus.—The attention of the neurologist is often focussed about the process of delivery. Whereas it is the child that occasionally has a birth palsy which is brachial, it is the mother who has a lumbosacral palsy due to long-continued pressure of the head upon the plexus. Here the palsy may be partial, or it may be fully developed, resembling a palsy due to a lesion of the cauda equina.

Intra-abdominal pressure may also arise from bony tumors, from pelvic inflammations, and pus collections of the pelvis due to old appendicitis, salpingitis, etc. Gunshot wounds occasion sacral plexus palsies.

Intramedullary causes for lumbosacral plexus palsies are more frequent than for brachial palsies, because of the arrangement of the nerves.

The cauda equina comprises the entire group of coccygeal, sacral and three last lumbar roots. The second lumbar is practically included within the canal. (See Figs. 15 and 16.)

Symptoms.—In the fully developed picture one finds complete flaccid palsy of the lower extremities. There is marked toe-drop, and limitation of all of the motions of the legs. Atrophy of the muscles takes place rapidly, especially of the lower extremities. The muscles of the anterior portion of the thigh innervated in part by the second lumbar, remain normal and active. Fibrillary twitchings are frequent in the atrophied muscles, and reaction of degeneration is present.

The cutaneous reflexes are usually absent, the Achilles reflex is absent and that of the patellar as well unless the lesion strikes below the third lumbar root.

Pains are usually very marked and persistent. They occur in paroxysms, and are usually extreme. The chief path is that of the sciatic, but they may be located almost anywhere about the thigh. They may be bilateral or unilateral and shift considerably.

Sensory examination shows typical diminution of all forms of sensibility following the neuritic type. Lesions higher up involving the conus or the cord show dissociated sensory phenomena to be discussed later.

The touch anesthesia extends to the limit of the second dorsal, *i. e.*, at the upper level of the sacrum, including the anus, perineum, and genito-urinary organs. (See Plates X and XI.)

Vasomotor phenomena, cold, somewhat cyanotic skin, and hand edema may be present. The bladder, rectum, and genital controls are involved. This is the usual picture in a total lesion. In practice this is less often seen than the many irregular types.

Course.—Prognosis.—Limiting the discussion to the cases of pressure of parturition one finds that these patients often suffer from irregular sciatic pains in the later stages of their pregnancy. A few days after a protracted delivery the pains often increase very markedly and then a paralysis of the lower extremities develops. This is usually more

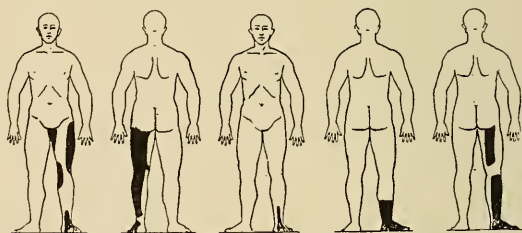


FIG. 144.—Sensory disturbances in five cases of sacral plexus injury due to gunshot wounds. In the first case there was a total palsy in the peroneus and tibialis nerve distribution; in the second the peroneus alone was involved; in the third the peroneus, tibialis, ilio-inguinalis and obturator; in the fourth the peroneus and tibialis; in the fifth the crural nerve distribution alone suffered. (Edinger.)

marked on one side. Bladder and rectal difficulties arise and the patient shows the picture usually of an incomplete plexus palsy of the lumbosacral region. After a month or so—sometimes several months in the severe cases—a complete recovery usually takes place.

The results of sacral plexus injury by gunshot wounds are less promising.

Treatment.—Is that for a neuritis in general (*q. v.*).

PERIPHERAL PALSIES.

Numerous forms of peripheral nerve palsy are found, varying according to the particular fibers involved. The branches that arise from the brachial plexus have already been described. Any of these may be injured or inflamed with a resulting partial or complete palsy. The various neuritides will not be reëxamined and the following sections will deal with those peripheral palsies largely due to defect or injury.

Defects, Congenital and Acquired.—Muscular defects in the upper and lower extremities are by no means uncommon. They have been described for some centuries. Their precise formulation began with Ziemssen's work in 1857. The later literature is summed up by Bing,¹ Lorenz,² and by Hirschfeld.³ The causes for these muscular defects are extremely complicated.

The occurrence is very manifold. Bing was the first to collect the entire group, and Abromeit, in 1909, completed the study which shows that an absence of any muscle of the body may be expected. One in 10,000 shows such defects.

In the majority of the cases the defect is unilateral, and they are more frequent in men.

Abromeit collected reports of 186 cases of defect of the pectoral muscles, the sternocostal portion being most affected.⁴ This seems the most frequent of such anomalies. The trapezius was absent in 33 cases, the serratus magnus in 22, quadratus 26, omohyoid 16, semimembranosis 7, rectus muscles 11, pyramidalis, often found absent postmortem, diaphragm 3 times, deltoid 5, sternocleidomastoid 8, etc. The smaller muscles of the hand are not infrequently absent.

Certain combinations of muscles may be absent, constituting a group complex. Atrophy or loss of other parts may be combined and gross anomalies of structure may be combined with marked muscular defects; various monsters, syndactylia, etc. The hereditary nature of syndactylous anomalies has given occasion for some important studies.

The symptoms involve the physiological loss of the special muscle function or the resultant of function from the muscles present in a combination.

These patients rarely show a marked loss of efficiency, especially with one-sided defect, as do those who acquire a defect. Acquired defects are usually bilateral. They usually involve a group of muscles; the disease rarely causes a total loss of muscle substance, and anomalies of accompanying structures are missing.

Fibrillary twitchings are often diagnostic of the acquired muscular defects.

The prognosis and treatment require little discussion. Gymnastic exercise of the residual muscular combinations directed to the acquisition of greater efficiency by skilful application of mechanical principles is always an individual goal that cannot be more than mentioned here.

Peripheral Palsies Due to Nerve Injuries.—This section discusses peripheral palsies due to nerve injuries rather than to those due to general neuritic processes. An injury may affect a nerve either in its continuity, or in one of its terminal branches, thus giving rise to different symptoms, and requiring a diverse therapy.

¹ Virch. Arch., 170, 1902.

² Die Muskel Erkrankungen Nothnagel, 1904.

³ Kongenitale Muskeldefekte, Lewandowsky Handbuch, 1911.

⁴ See Lewandowsky, II, p. 253.

Injury in *continuity* may result from penetrating wounds, traction, pressure, blows or by operation. They may give rise to complete or incomplete division.

Symptoms.—In complete division Sherren and Head have shown that the afferent peripheral fibers may be divided into three systems:

1. Those which subserve deep sensibility and conduct the impulses produced by pressure. The fibers of this system run mainly with the motor nerves and are not destroyed by division of all the sensory nerves of the skin.

In a part innervated only by this system gradual increase in pressure can be recognized and pain experienced when such pressure becomes excessive. The patient can also appreciate the extent and direction of movements produced passively in all the joints within the affected area.

2. Those which subserve protopathic sensibility. This system of fibers and end-organs respond to painful cutaneous stimuli and to the extremes of heat and cold; it also endows the hairs with power to react to painful stimuli. The distribution of the protopathic fibers usually overlaps greatly the area supplied by similar fibers from adjacent nerves.

3. Those which subserve epicritic sensibility. The nerve fibers and end-organs of this system endow the part with the power of responding to light touch with a well-localized sensation. The existence of this system enables one to discriminate two points and to appreciate the difference between cold and heat. The distribution of these fibers in large peripheral nerves, such as the median and ulnar, has very little overlap compared with the greater overlapping of the protopathic supply.

These investigations were carried further, particularly with regard to deep sensibility and the distribution of heat and cold spots by Head and Rivers after voluntary section of the radial and external cutaneous nerves in the former's arm.

To illustrate these changes in sensibility after division of a mixed nerve the ulnar is an excellent example (Fig. 149). After complete division of this nerve at the wrist, if no tendons have been divided at the same time, the patient is able to appreciate those stimuli commonly called tactile. A touch with anything which deforms the skin may be readily appreciated and correctly localized. When pricked with a pin the patient knows that he has been touched but fails to perceive the sharpness of the stimulus (deep sensibility). But if tendons are divided at the same time or the section involves the nerve above the point at which its muscular branches are given off, deep touch may be unperceived. These characteristics are of the utmost importance, many cases of nerve injury have been overlooked from failure to recognize these facts.

The point of a pin and all temperatures are unappreciated within an area which varies somewhat in each case (loss of protopathic

sensibility). Surrounding this area and corresponding closely to the distribution of the nerve as figured in Plates X and XI is a territory within which the patient is unable to appreciate light touches with cotton-wool and temperatures between about 22° and 40° C. (minor degrees of temperature), and fails to discriminate the points of a pair of compasses when separated to many times the distance necessary over the corresponding portion of the sound limb or the unaffected portion of the injured one (loss of epicritic sensibility). Within this area of loss of sensibility to light touch, to which we gave the name of "intermediate zone" the patient is able to appreciate the sharpness of a pin-prick and to differentiate temperatures below 20° C. and above 45° C., naming them correctly.

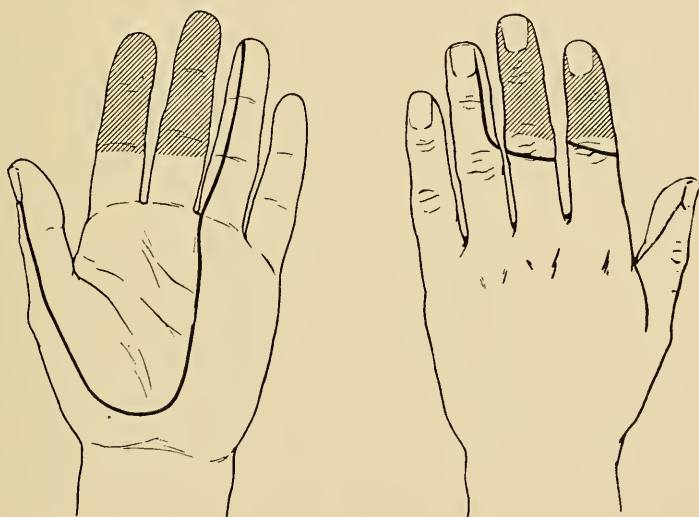


FIG. 145.—To illustrate the changes in sensibility met with after complete division of a peripheral nerve. The area inclosed by a line is that in which epicritic sensibility is lost. The shaded area is that of loss of epicritic and protopathic sensation. The unshaded portion is the "intermediate zone." (Sherren.)

Division of a peripheral nerve produces a well-defined loss of epicritic sensibility, a smaller loss of protopathic sensibility with, as a rule, ill-defined limits. In many cases there is no loss of deep sensibility.

Complete division of certain nerve branches produces no objective change in sensibility, these are the musculospiral below the point at which its external cutaneous branches are given off, the radial, and certain cervical anterior primary divisions.

Symptoms in Incomplete Division.—The sensory symptoms may be *nil*. The patient is conscious of an area altered in sensibility, and it is often possible to demonstrate this by the changed sensibility produced at its borders when a piece of cotton-wool or the point of a needle is dragged lightly across the skin from sound to affected portions (line

of change). If the area of changed sensibility is well marked, response to the compass test will be defective.

In cases of greater severity the loss of sensibility to cotton-wool may be absolute, with borders as well defined as after complete division.

When the injury is more severe, impairment or loss of protopathic sensibility results and the sensory loss may resemble exactly that seen after complete division.

Sherren has shown that, contrary to the usual teaching, the motor loss in incomplete division is not more than the sensory loss. Paralysis of muscles of the injured nerve distribution may result after eight to fourteen days. The usual reaction on or about the tenth day is that the muscles do not respond to the interrupted current but do react

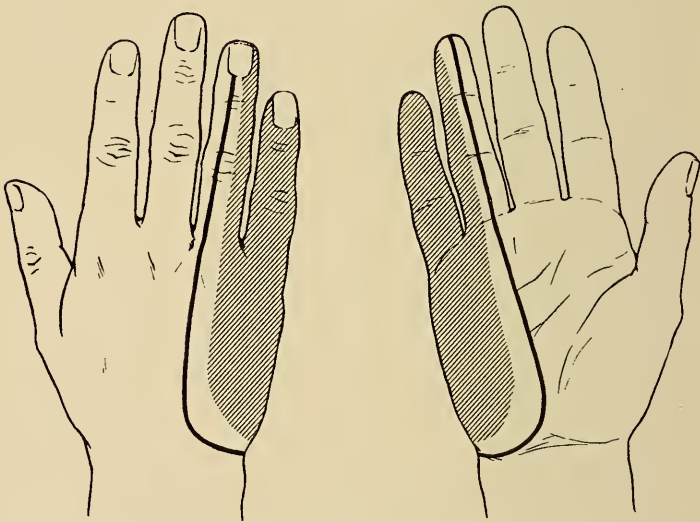


FIG. 146.—Loss of sensibility after complete division of ulnar nerve. (Sherren.)

to the constant current. The strength of the current needed to cause a contraction is less on the sound side; the contraction is brisk in comparison to the R. D. and polar reversal is absent.

Pain is a frequent after-result in incomplete division; there may also be tender and glossy skin and changes in the nails.

Stages of Recovery.—Following complete division, in from six to sixteen weeks, the restoration of protopathic sensibility commences and is completed in from four to twelve months after suture of divided nerves. Blisters may occur early, but later all ulcers heal; blisters no longer appear. Epicritic touch is unchanged throughout, but in from twelve to fifteen months the whole area is sensitive to light touch and intermediate degrees of temperature. Improvement in the power of accurate localization is the third stage, and is tested out by the com-

pass. The motor recovery is gradual, the electrical reactions of incomplete division first appearing.

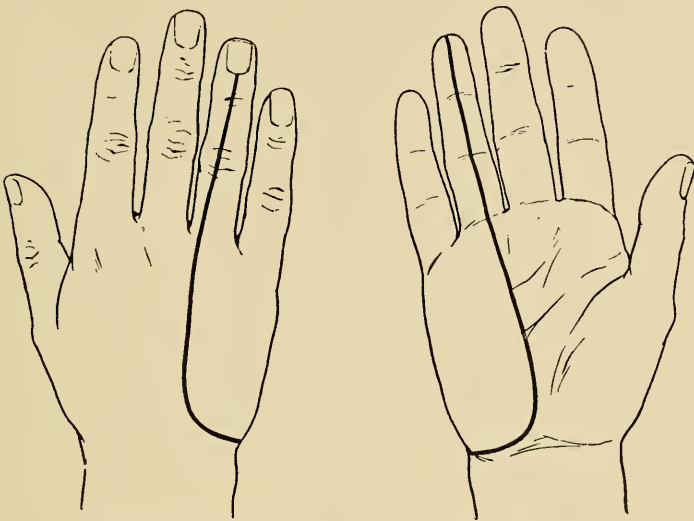


FIG. 147.—End of first stage of recovery. (Sherren.)

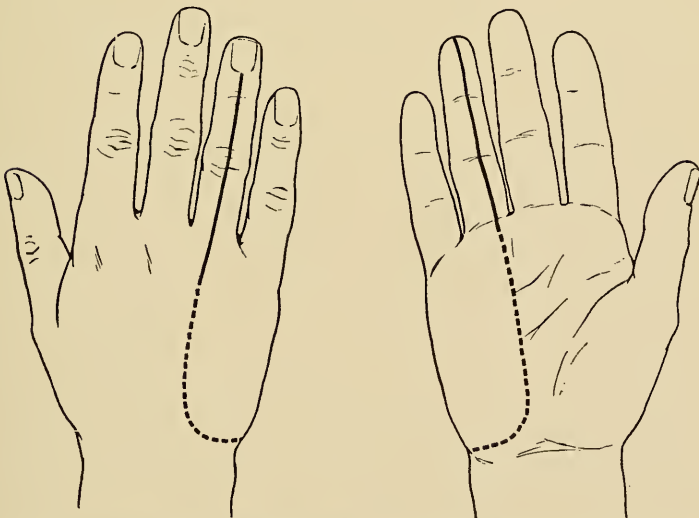


FIG. 148.—Commencement of second stage of recovery. The dotted line marks the area gaining sensibility to cotton-wool. (Sherren.)

It seems certain that no regeneration takes place in the peripheral end of a divided nerve without union with the central nervous system. After incomplete division of a mixed nerve the loss of sensation and

motion may at first resemble that which follows complete division, but the method of recovery is entirely different.

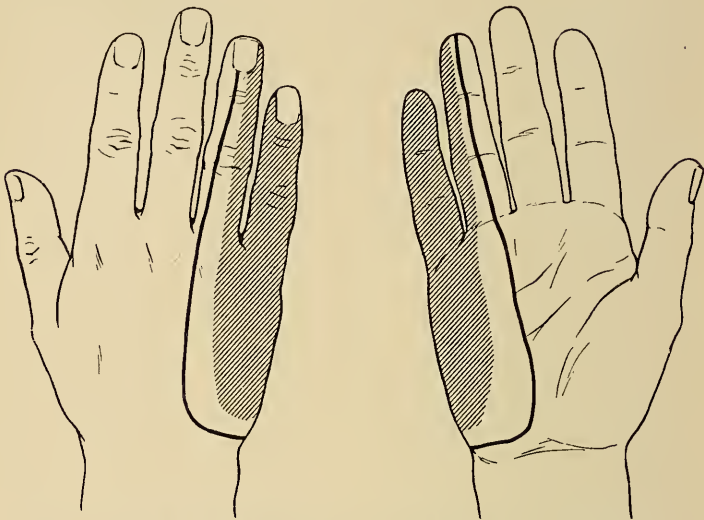


FIG. 149.—Loss of sensibility after complete division of ulnar nerve.

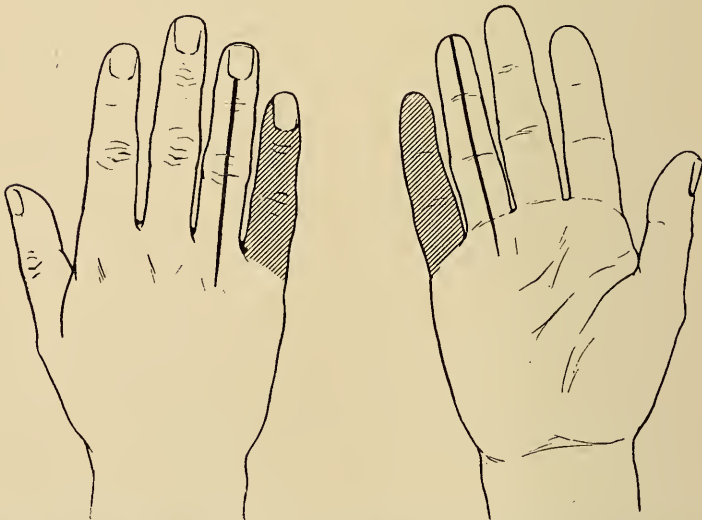


FIG. 150.—Showing method of recovery after incomplete division. (Sherren.)

After complete division of a nerve and suture, sensibility to prick is restored before the commencement of recovery of sensibility to light touch. Complete sensory recovery often occupies several years.

But after incomplete division sensibility to light touch and to prick are restored together and, unless nerve fibers have been anatomically divided in considerable number, the power of appreciating two points (the compass test) is soon regained. This is an extremely important point, for upon the recovery of this power of localizing depends the utility of the part for fine work. It is important to recognize that in injuries of nerves without interruption of their anatomical continuity the power of localization returns quickly, unless the injury has been sufficient to cause complete division; in this case the usual three stages are present, but the time of the third stage is much shortened.

Knowledge of this method of sensory recovery, first described by Head and Sherren, is a valuable addition to our powers of diagnosis. If both forms of sensibility are recovering together, it is certain that the injury has not been severe enough to produce complete interruption of conduction in the injured nerve, with degeneration of the whole peripheral end.

Motor recovery after incomplete division follows the same march as after complete division; the muscles nearest the seat of the injury first regain voluntary power and excitability to the interrupted current.

In the cases in which the reactions typical of incomplete division are present, voluntary power usually returns before the reestablishment of excitability to the interrupted current.

Sensory recovery usually begins in about three weeks, and is complete in about six months. Motor recovery in from a few days to ten weeks.

These times of motor and sensory recovery are approximate only, and vary with the severity of the injury and its distance from the periphery. When epicritic sensibility alone is lost recovery is much more rapid than when both forms of sensibility are affected. When the injury affects the brachial plexus, considerably longer time is necessary for the commencement and progress of recovery.

To sum up, after incomplete division of a mixed nerve, both forms of sensibility (epicritic and protopathic), if lost, return at the same time, commencing at a date which varies with the distance of the injury from the periphery from about three weeks at the wrist to six months in the plexus, and also with the degree of the injury. Complete recovery, as a rule, rapidly ensues. Muscular recovery commences at a time which varies in the same way. In cases in which the muscles, though paralyzed, retain their irritability to the interrupted current, recovery commences in three or four weeks, sometimes earlier, and soon becomes perfect. This degree of injury is seen most often as the result of compression of the musculospiral nerve, producing sleep, anesthetic or crutch paralysis. If the reactions typical of incomplete division are present a much longer time is necessary.

After neurolysis, or when the nerve has been relieved from any form of pressure, recovery follows exactly the same lines.

Injuries to Terminal Branches.—Facial paralysis may result from injury to the nerve (*a*) above the geniculate ganglion, (*b*) between the ganglion and the point at which the chorda tympani is given off, and (*c*) below this point. When injured at (*b*) taste is affected over the anterior two-thirds of the corresponding side of the tongue.

Apart from the so-called "rheumatic" affections of the nerve (Bell's palsy), interference with the function of the nerve in the middle ear as a result of disease or operation is the usual cause. The nerve may suffer in fractures of the skull primarily, or more often from involvement in callus. Outside the skull it may be injured during operations in the parotid region or in the removal of tuberculous glands, or from forceps pressure during childbirth; in most of these cases the "division" is incomplete and spontaneous recovery ensues.

In cases of incomplete division the usual treatment directed toward maintaining the nutrition of the paralyzed muscles must be adopted. When due to middle-ear disease it is an indication for the complete mastoid operation.

When the reaction of degeneration is present, showing that complete division, either anatomical or physiological, has occurred, the treatment to be adopted depends on the cause. If it follows a radical mastoid operation, the sooner operation is carried out after the wound has healed the better; in cases of Bell's palsy it is justifiable to wait for six months. If the nerve is divided during the course of a mastoid operation, the ends should be adjusted in the aqueduct, when restoration of function may be expected unless neuritis is set up as the result of sepsis. If discovered after the operation the electrical reactions should be tested at the end of a fortnight; if the reaction of degeneration is present, the wound should be opened up and an attempt made to adjust the ends. If this fails, nerve anastomosis must be undertaken. It must be remembered that the injury during mastoid operation is in most cases incomplete, and that spontaneous recovery follows the usual non-operative treatment.

Spinal Accessory Nerve.—The external or spinal portion of this nerve is not infrequently divided during the course of operation upon the neck, particularly during the removal of tuberculous glands. In many of these cases the branches of the cervical nerves to the trapezius are affected at the same time producing its complete paralysis. The extent of supply of the spinal accessory and the cervical nerves to the trapezius varies; as a rule the upper portion of the trapezius is paralyzed by division of the spinal accessory alone.

Cervical Rib.—Symptoms usually appear in early adult life and are due in most cases to pressure upon the lowest trunk or nerve entering into the plexus. In many cases they consist of wasting of the intrinsic muscles of the hand, most marked and starting in those of the thenar eminence; if of long standing the reaction of degeneration may supervene. In other cases the principle complaint is pain along the ulnar border of the forearm and hand, or a general weakness or

heaviness of the whole limb noticed at the end of the day. Sensory changes are unusual and when present rarely exceed epicritic loss.

This condition should be thought of in all cases of "brachial neuralgia," or of wasting of the thenar muscles. The ribs can usually be felt but occasionally can only be demonstrated by x-ray examination.

Although a cervical rib be present it does not necessarily mean that it is the cause of the symptoms. Several cases have been observed in which a cervical rib was removed from a patient suffering from syringomyelia to which the symptoms were due. Thomas Murphy has recorded a case in which the symptoms were due to the pressure of the first rib. A similar case is reported by Sherren.

Treatment.—Treatment consists in removal of the rib together with the periosteum covering it, followed by careful after-treatment.

The Long Thoracic Nerve (*Nerve of Bell*).—This nerve is most often injured in males between the ages of twenty-five and forty, usually on the right side. Generally caused by prolonged pressure in the supraclavicular region, it occasionally follows violent muscular efforts and direct violence applied to the shoulder.

Paralysis of the serratus magnus rarely occurs alone; it is usually combined with paralysis of the lower trapezius.

The Circumflex Nerve.—Injury to the circumflex nerve is by no means so common as is usually supposed. It has been said to follow direct blows on the point of the shoulder, but in most cases the injury is to the anterior primary division of the fifth cervical nerve, and careful examination will show that the spinati also are affected. In other cases the wasting of the deltoid in common with the muscles around the joint on which the circumflex injury was diagnosed has been found to be the result of a traumatic arthritis.

Careful examination is necessary before coming to a decision with regard to treatment; testing must be carried out for all forms of sensibility. If there is no loss of sensibility, and there is paralysis of the deltoid with the reaction of degeneration, it is extremely improbable that the circumflex nerve is injured. If the signs are those of complete section of the nerve, the age of the patient and his occupation must be taken into consideration; in some cases operation can be avoided by training the neighboring muscles to take the place of the deltoid.

Ulnar Nerve.—This nerve may be injured at the elbow, or at the wrist above or below the point at which the dorsal branch is given off. While it may suffer in any part of its course from injury and the result of penetrating wounds, these are more usually at the wrist. At the elbow the injury is due in most cases to fracture or deformity, recent or old, usually the latter, and to dislocation of the nerve.

If complete recovery is to be obtained the paralyzed muscles must be kept relaxed. Sherren has devised for this purpose an aluminum splint to be worn on the dorsum of the hand, keeping the fingers slightly flexed at the metacarpophalangeal and extended at the interphalangeal

joints. If this is worn the onset of "claw-hand" may be prevented and function be completely restored.

The condition of dislocation of the ulnar occurs most often in males between the ages of twenty and thirty. The symptoms usually come on immediately after the injury; occasionally some time elapses, the frequent injuries to which the nerve is subject in passing over the internal condyle causing fibrosis. In a few instances the condition originates without injury; in these patients the fascia which keeps the nerve in place is gradually stretched.

Musculospiral Nerve.—The special interest in connection with this nerve lies in the fact of its frequent injury in association with fractures of the humerus, particularly of its lower or middle third.

In addition, "crutch," "sleep," "bridegroom," and "Saturday night" paralysis are not uncommon as the result of direct pressure upon the nerve.

In every case the paralyzed muscles must be kept relaxed by suitable splints, and in no case must "wrist-drop" be allowed. Operation is rarely indicated except in the cases complicated by fracture, as the injury is incomplete and the recovery is usually rapid.

The prognosis is better than after injury of any other nerve. In cases in which neurolysis is necessary, restoration of function often commences within a few weeks. Even after secondary suture, motor power usually returns within a year.

External Popliteal Nerve.—This nerve suffers most often as the result of direct violence as it passes around the neck of the fibula. When primarily injured in association with fracture, operation should be undertaken at once, for the nerve not infrequently passes between two fragments.

However caused, the division is usually incomplete, but recovery is always slow and, unless great care be taken, often imperfect. It is essential to keep the foot at right angles and to prevent foot-drop.

External Cutaneous Nerve.—Injury to this nerve is important in that it is occasionally the precursor of Bernhardt's disease. The nerve is usually involved at its exit from the deep fascia and in many cases a spindle-shaped swelling may be felt here. The injury causing it may be long continued as by a badly fitting truss or in other cases it follows a sudden strain.

CHAPTER VII.

LESIONS OF THE SPINAL CORD.

THE anatomy, histology, and physiology of the spinal cord will be discussed only in so far as problems of localization and pathology are concerned.

A large group of disorders, chiefly limited to the cord, come under review. One may present them as a series of syndromes which affect:

1. Chiefly the peripheral motor neuron (anterior horn cells).
2. Combinations of anterior horn cells and pyramidal tracts.
3. Chiefly pyramidal tracts.
4. Chiefly posterior columns.
5. Sympathetic cell groups.
6. Central gray.
7. Combined and diffuse lesions.

These subdivisions are largely arbitrary; at times they correspond to clinical entities, so-called, again they are fortuitous combinations. Thus a poliomyelitis may clearly delimit, *i. e.*, in its end-results, a group of motor neurons, anterior horn cells, while, on the other hand, spinal syphilis may show any of the localizations just tabulated. For pedagogic purposes the following general scheme may be of value:

AREAS INVOLVED.	CHIEF SYNDROME.	GENERAL DIAGNOSTIC SYMBOL.
1. Anterior horn group No. 7. (Fig. 151).	{ Varying with metameres involved, paresis or paralysis of muscles, atrophy, atony, loss of reflex in muscle group, R. D.	{ 1. Acute anterior poliomyelitis. 2. Chronic anterior poliomyelitis. 3. Muscular atrophies combined with some dystrophies.
2. Combined (1) and (2) pyramidal tract groups Nos. 6 and 7.	{ From (1) muscular atrophies, R. D., from (2) increased reflexes, increased tonus, contractural tendencies	{ 4. Amytrophic lateral sclerosis. 5. Some multiple sclerosis.
3. Pyramidal tract lesions No. 6.	{ Paresis or paralysis, more usually entire side, with spasticity, increased tonus, increased reflexes, no atrophy, no R. D., associated movements of opposite side.	{ 6. Lateral sclerosis. 7. Compression, tumor, Pott's disease, bony growths, traumatic lesions. 8. Most multiple sclerosis. 9. Toxic (pellagra, anemia, arterio-sclerotic and senile syndromes.
4. Posterior column lesions Nos. 1 and 2.	{ If unilateral, Burdach's column only, with deep anesthesia, hyperesthesia, ataxia of limbs lying above lesion. Diminished or lost reflexes; when including Goll's columns, deep anesthesia, superficial hyperesthesia, below site of lesion.	{ 10. Tabetic syndromes. 11. Toxic (lathyric, alcoholic). 12. Rare compressions. 13. Friedreich's syndromes. 14. Agenetic (cerebellar ataxias) syndromes.
. Sympathetic groups No. 9.	{ Trophic disturbances; vascular disturbances, changes in secretions, hair, skin, etc.	{ 15. Trophoneuroses { See 16. Angioneuroses { Vegetative 17. Dermatoses { Nerv. System Disease.
6. Central group No. 4.	{ Dissociation of thermal and pain senses from tactile discriminations.	{ 18. Hematomyelic syndromes. 19. Syringomyelic syndromes.
7. Combined, general and indiscriminate groups.	} Showing various combinations of preceding.	{ 20. Tumor, destruction or compression. 21. Diffuse myelitis. 22. Multiple sclerosis. 23. Syphilitic meningomyelitis.

A study of the cross-section of the cord will bring these localizing factors more closely into view. The localizing features are brought out in Figs. 11-16 and 19-22 (see pp. 42, 43, 46, 47, 50, 51).

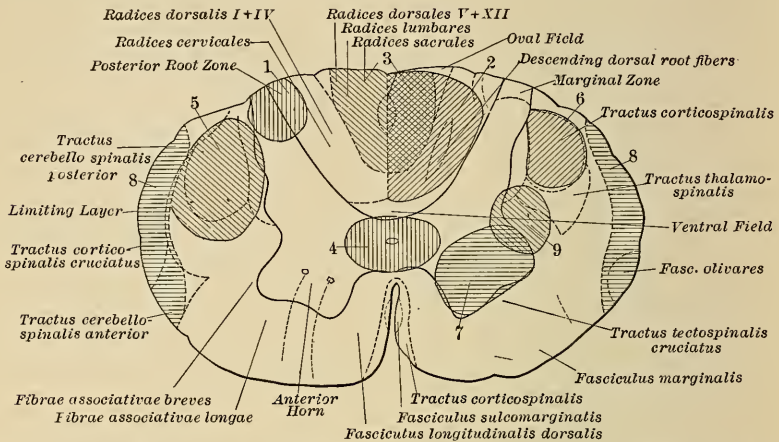


FIG. 151.—Cross-section of spinal cord showing localization of chief structures with lesions. (Veraguth.)

LOCATION OF LESION.

1. In the posterior root zone.
2. In posterior column of one side.
3. In Goll's columns of both sides.
4. In central gray, especially of anterior commissure.
5. Posterior portion of the lateral columns with integrity of limiting layer.
6. Pyramidal tracts.
7. Anterior horns.
8. Spinocerebellar paths.
9. Lateral recess.

CHIEF SYMPTOMS.

- Irritation causes hyperesthesiæ. Destruction causes loss of superficial sensibility in the root distribution spreading over at least three roots. Ataxia and eventually astereognosis in the extremity involved.
- Anesthesia to deep sensibility and to touch. Ataxia of metameres below the lesion.
- Anesthesia to deep sensibility and hypesthesia of the lower extremities only, even in high lesions.
- Dissociated sensibility (thermanesthesia and analgesia in the affected metamere as indicated in the skin distribution).
- Crossed hemihypesthesia plus the symptoms of 6.
- Spastic paralysis of the caudal metameres below the lesion without reaction of degeneration, often crossed movements, no atrophy and with increased reflexes.
- Flaccid paralysis of the muscles of a number of root zones, atropia and atrophy of muscles of involved metameres; R. D., loss of reflexes.
- Bilateral involvement causes cerebellar ataxia.
- Sympathetic disturbances metamERICALLY distributed.

The majority of these cord syndromes are considered here, some are discussed under their etiological groupings: syphilis of the nervous system, for instance, taking over tabes, spinal cord gummata, syphilitic meningomyelitis.

ACUTE POLIOENCEPHALOMYELITIS.

Historical Note.—This disease has affected mankind for centuries. Mitchell reports shortening of the femur, presumably due to this disease, in an Egyptian mummy. Jonathan's son (II Samuel, iv, 4.) possibly had this disease. Numerous drawings and paintings of the fourteenth and fifteenth centuries (Hieronymus Bosch) attest to its prevalence in these times. Throughout the period of the later Latin writers it was usually included under paralysees, hemiplegias, etc. It was Underwood who, in 1784, described a new disease "Debility of the Lower Extremity," that gave the first impetus which led to its definite separation as a type by Jacob v. Heine in 1840. In 1810 Jörg gave an excellent case history, and in 1843, independent of Heine, Rilliet and Barthiez contributed an important article to French literature. They called the disease an Essential Paralysis, and thought it had little spinal pathology.

Although much was written previous to Duchenne's time, his work in 1855 marked the beginning of a new era in the study of this disease and in Heine's second edition, 1860, the status of the disease at that time is well reflected. In 1865 attention was first called to the possible relation of infantile to adult poliomyelitis by M. Meyer, which study was followed by a large number of further contributions from Charcot's students.

The anatomical era may be said to have opened with Cornil in 1863, and there then grew up the Charcot thesis of a primary affection of the ganglion cells of the anterior horns, which has had to give way to the more recent studies of Wickmann, Harbitz and Scheel, Flexner, Strauss, and others (1807-1910).

Seeligmüller's masterly monograph in 1880 practically contained the standard teachings up to the appearance of Medin's work in 1896, when the epidemiological features of the disease were brought out. In later years Lövegren (1904), Wickmann (1905-1907), Harbitz and Scheel (1905) have still further refined the clinical and pathological aspects, while Flexner, Landsteiner, Popper, and others have been able to convey the disease from the human to monkeys, and thence to other monkeys (1909-1911).

The most recent monographic treatment of the subject is found in Wickmann's very able contribution in the *Handbuch der Neurologie*, 1911, and Müller's equally valuable monograph (1911), one by Römer¹ (1911), and by Peabody and Draper (1913).

Etiology.—The disease is an acute infectious disease. The organism is probably a living one; it can be conveyed to human beings, to monkeys and to rabbits. A small amount of the emulsion of the spinal cord of humans injected into the brain of a monkey has caused the disease, which Flexner has transmitted from monkey to monkey for

¹ Translated in Nervous and Mental Disease Monograph Series, New York.

twenty-five generations. The organism is thought to be a small organism, capable of passing through a Berkefeld filter, and one whose virus is destroyed by heat and weak disinfectants, but not by cold nor dryness. In many respects the virus resembles that of rabies. It has been conveyed by direct injection into the brain, through the uninjured and scarified nasal mucous membrane, and through injection of the intestinal lymph glands of affected animals (Flexner).

An immunity of yet unknown length seems to be established by one attack. The disease seems to be conveyed by direct contact, through indirect contact, and through nasal and gastro-intestinal secretions. It does not seem to be highly contagious.

Epidemic extensions have now been studied for nearly sixty years, and Cordier first expressed a belief in its contagiousness. Medin first definitely proved it. Some eighty or more epidemics have been reported to 1912. The most recent pan-epidemic apparently started in Norway and Sweden in 1903-1904, spread to the United States in 1907-1912, to Germany and France in 1908-1911, with isolated far-lying outbreaks in Cuba, Australia, etc.

Rural districts have suffered greatly, and density of population has not seemed to play a large role; cold climates seem more favorable, and the season is usually in the warm, dry months.

The majority of the cases occur in children from one to five years of age, with lower limits at two weeks, and over sixty years as outlying rarities. Certain epidemics have shown marked variability in the matter of age incidence, the Swedish epidemic of 1905 having as high as 10 per cent. adults. In large epidemics adults seem more often affected. The male sex has seemed to be more often involved, but the differences are not very marked. Nationality seems to play little role, although it has been assumed that Scandinavians are peculiarly susceptible. This may only be an indication of the more careful study given by these authors. Neuropathic heredity seems to play no role.

The incubation period varies from one to ten days, the majority showing a period of from one to five days. In experimental poliomyelitis of monkeys the incubation time is about six to nine days.

Pathology.—The older concepts of a primary intoxication or of an inflammation of the motor horn cells exclusively must be abandoned. Acute poliomyelitis is due to an acute interstitial inflammation (a meningomyelitis), involving all parts of the cord. There is a congestion, infiltration, and edema of the leptomeninges, cord, medulla, pons, cerebellum, and cerebrum. The dura is not markedly involved; the pia is congested and infiltrated with round mononuclear cells (lymphocytes), particularly in the sacral and lumbar region. The vessels are congested and their sheaths infiltrated, the progression of the inflammatory reaction apparently following the vessels from the periphery within the cord. The cerebrospinal fluid is increased in quantity, almost opalescent early in the disease, with markedly increased lymphocytes, in some of which Lafora and Hough have

found pictures resembling the Leishman-Donovan bodies. The fluid later becomes clearer, but still shows a pathological increase in leukocytes.

Within the cord the inflammatory process follows the pial processes into the depths of the anterior fissure and along the sheaths of the central vessels. The posterior root fibers and the spinal ganglia are also infiltrated.

The vascular lesions are particularly noticeable, and the interstitial and ganglionic changes depend largely upon them. The vessels throughout are dilated and engorged, the capillaries often being enormously distended. This marked hyperemia is found throughout,

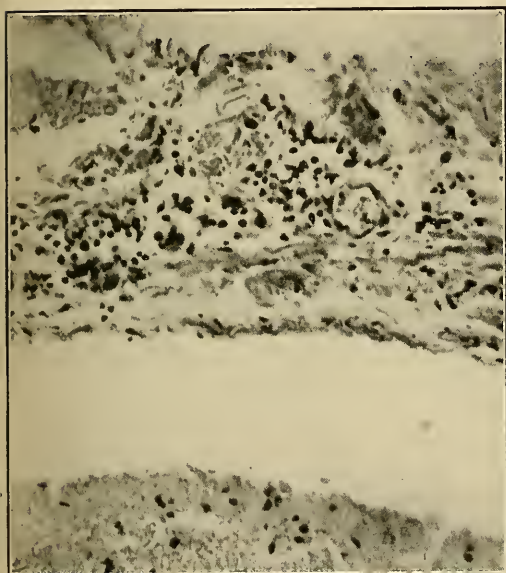


FIG. 152.—Acute poliomyelitis, showing *A*, associated meningitis; *B*, edge of cord.

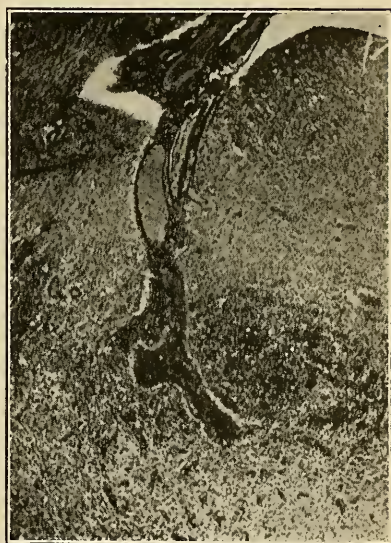


FIG. 153.—Acute poliomyelitis, showing the vascular congestion and the surrounding infiltration. The anterior spinal artery.

and vascular hemorrhagic lytic changes are frequent but small. As in rabies, and to some extent in syphilis, there is a marked perivascular or intra-adventitial infiltration, apparently of lymphocytes, not plasma cells. Interstitial changes in the gray and white matter, chiefly of an infiltration of cells, and proliferation of glia cells, occur. By reason of the rich blood supply the gray matter of the anterior horns bears the brunt of the inflammatory edema and hyperemia, with destruction of many of its motor cells. This is a secondary process. Harbitz and Scheel believe that small abscesses take place, but this is an exception if it does occur.

The ganglion cells undergo varying degrees of degeneration, somewhat proportioned to the infiltration, the axis-cylinder finally breaking

down. Amid the degenerated cells many normal ones may be found, and the distribution of degenerated areas is very variable, the sacro-lumbar cord being more seriously affected, although any part of the cerebrospinal axis may be involved. All classes of ganglion cells go under, but the lateral dorsal cord regions are much less involved; however, many vegetative system cells are injured.

As a rule the functional involvement is, by reason of the edema, hyperemia, and infiltration, far in excess of the permanent anatomical loss, hence the widespread character of the paralysis in the early stages and the marked degree of recovery possible. Only a small proportion of the primarily involved ganglion cells degenerate entirely.

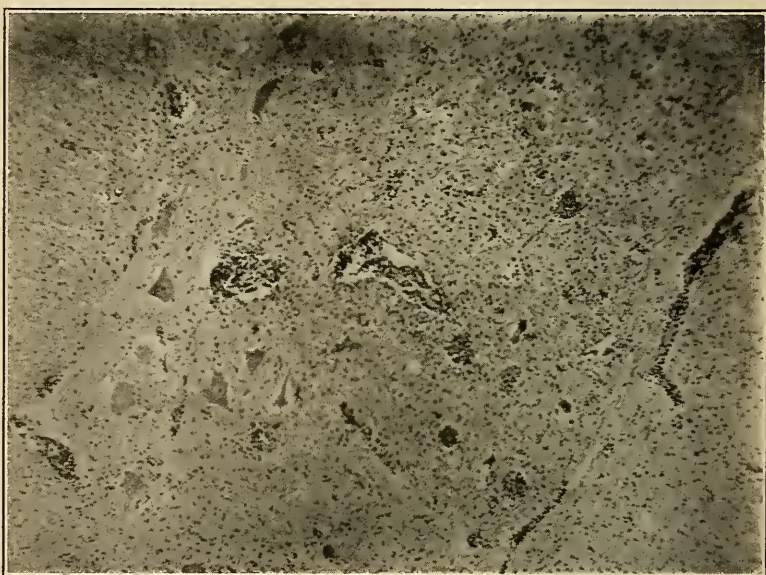


FIG. 154.—Acute poliomyelitis. The various stages of destruction of the anterior horn cells.

The fiber pathways may show permanent infiltration changes with the production of anomalous spastic phenomena.

The spinal nerves are involved usually at their junction with the cord, and some polyneuritis may be present early in the disease.

The medulla, pons, cerebellum, and cerebrum all are involved to a greater or lesser degree. Certain cases show that the main lesion is in one or more of these regions, rather than in the spinal cord. True encephalitis, with varying degrees of weak-mindedness, is the frequent result of these higher lying types.

The other organs of the body do not escape. There are evidences of an acute general infection everywhere.¹

¹ Walter, Deut. Zeits., vol. xlv, No. 2.

Symptoms.—The study of the recent epidemics has shown a great variability in the affection, but practically all of the cases show the effects of an acute infection as prodromal and early signs. The later course of the disease, dependent upon the locations chiefly involved, permits a separation into several types of which Wickmann recognizes eight classical pictures.

Prodromata are usual. They vary in the different epidemics. They consist of fatigability, loss of appetite, slight digestive disturbances, with nausea, looseness of the bowels, coryza or bronchial irritation (bronchitis, bronchopneumonia) with slight elevation in temperature. Conjunctivitis may occur; lymphatic swellings are usual. The patient may thus suffer for twenty-four to seventy-two hours before the acute prostrating effects of the disease become manifest. Some few cases show almost complete recovery after such prodromata, and then are again taken ill suddenly. Careful observation will probably reduce the number of cases reported as coming on without prodromata.

The cerebrospinal fluid in the prodromal stages may show opalescence with very marked lymphocytosis. The blood changes are apparently not constant. La Fetra has reported a leukocytosis of from 13,000 to 20,000; whereas Müller has found a leukopenia of from 3000 to 5000, and also finds leukopenia in experimental monkey poliomyelitis. The lymphocytes are increased. No parasites have been found in the blood up to the present time (Dickson).

After twenty-four to seventy-two or more hours the temperature suddenly rises. It varies from 99° F. to 102.5° F. or even 105° F. to 106° F., and bears little relation to the severity of the disease. Abortive cases have shown high temperatures, and severe cases little. Chills and convulsions occasionally attend the rise in temperature. The temperature curve is not characteristic, being either remittent or continuous. Subnormal temperatures occur, and indicate marked cervical involvement.

Headache is a frequent symptom. It is often severe and usually frontal; it may be occipital, resembling a meningeal headache. Prostration is marked, diarrhea is extremely common, vomiting is frequent, and constipation is not unusual. Respiratory symptoms are not marked, save when respiratory palsies occur. Here dyspneas are the rule. The kidneys show little, and the heart's action is that of an acute infection in general, with a tendency to show nervous tachycardia, especially in the cases with medullary involvement. Weakness is the rule.

Early profuse sweating is a frequent symptom and skin eruptions are occasionally seen, although herpes is rare in contrast with cerebrospinal meningitis. Other exanthemata occasionally appear.

Nervous Symptoms.—As a rule there is considerable restlessness, especially in older patients. The children are peevish, petulant, or very fretty. Sleep is often disturbed, with frequent crying out.

Some children lie drowsy or apathetic, and may, though rarely, show coma, delirium, or convulsions. Pain is a frequent early symptom, and may be very severe. Marked hyperesthesia is usual (90 per cent.), and is brought on by the least attempt at motion of the limbs, pressure on the nerve trunks, touching of the skin. Heine called attention to this in 1840. Movements of the head and spine are particularly painful, and some patients are extremely anxious and fearful, whimpering continuously, closely resembling, in the early stages, meningeal cases. There is frequent photophobia and hyperacusis.

The infiltration in the cord causes other sensory symptoms, such as paresthesiæ and anesthesiæ. Stiffness is not infrequent, with slight though not marked retraction of the head (Kernig's sign) in some and varying degrees of rigidity of the limbs, with contracted position of the lower limbs particularly, are frequent.

Twitching and jerking of the limbs are also frequent.

The stage of paralysis soon sets in and, according to the predominant localization, following Wickmann, eight types may be distinguished. These types represent general tendencies rather than hard-and-fast differences. The further symptomatology will be given in accordance with these divisions. These types are: (1) spinal poliomyelitis forms (the commonest type); (2) acute ascending types (Landry's paralysis); (3) bulbar or pontine forms; (4) encephalitic forms; (5) ataxic forms; (6) meningitic forms; (7) neuritic forms; (8) abortive forms.

1. *Spinal Forms*.—There is usually an early prodromal weakness, or even a paresis which is very widespread. This develops to a relatively marked paresis, sometimes within twelve to twenty-four hours, but more often in from two to five days, occasionally after a week. Careful observation has shown that the paralysis is a gradual, rather than an abrupt one. It begins with weakness, advances to paresis, and finally becomes a definite paralysis, whereas the weakness, however, is very widespread, the palsies are less so, and the paralyzes even more restricted. When the ultimate stages are reached, the weaknesses clear up, the palsies gradually disappear, and the residual paralyzes often represent but a small part of what appeared to be a wholesale devastation. This is in strict accordance with the pathological features.

The distribution of paralyzes is due to factors concerning which there is little definite information. From a purely statistical study of the cases it has been found that the lower limbs are involved twice as often as the upper; in some epidemics four times as often. The entire limb is rarely involved permanently, but special muscle groups are picked out. Thus in the lower extremities the quadriceps, the peronei, and the tibialis anticus are the oftenest affected; in the upper extremities the scapular and deltoid muscles. Asymmetry in the final picture is the rule. The muscles of the trunk are involved next most frequently, while the arms are least frequently involved. The trunk muscle palsies are often overlooked, however.

In young children it is almost impossible to localize the paralyses in the early stages, especially as many such patients go through the initial stages of the disease without there being a suspicion of the real difficulty. Here the loss of the reflexes, the hypotonus, the careful scrutiny of the position of the limbs, the behavior to passive motion and resistance movements and the tickling responses lead to a correct appreciation of the difficulties. They also permit a diagnosis of rudimentary and of mild cases. Babies in arms behave as though they were lumps of dough, and the mother notices the hypotonus.



FIG. 155.—Abdominal muscle palsy—poliomyelitis. (Frauenthal.)

Weakness of the muscles of the abdomen is an important early diagnostic feature, especially in the differentiation from meningitis. The involvement is usually bilateral and diffuse. The muscles are hypotonic, and swell out as though the intestines were inflated with gas. There is often a loss of the epigastric and abdominal reflexes—symmetrical or assymetrical. The patients are further unable to come from a horizontal to a sitting position. In some patients the abdominal muscles are alone involved. Obstinate constipation usually accompanies the abdominal palsies.

Of the back muscles the latissimus dorsi are the oftenest involved. The glutei are also somewhat implicated. Children with these palsies

waddle when they walk. They behave like children with muscular dystrophy on rising from the floor.

Only rarely is the diaphragm implicated. They are usually among the fatal cases. Unilateral diaphragmatic palsies have been observed. The muscles of the hips are involved in at least two-thirds of the cases.

The following table from Wickmann will serve to indicate the general run of the cases. The figures represent the study of 868 cases in the Swedish epidemic of 1905:

1. Paralysis of one or both legs	353
2. Paralysis of one or both arms	75
3. Combined paralysees of arms and legs	152
4. Combined leg and thigh paralysees	85
5. Combined arm and thigh paralysees	10
6. Isolated thigh paralysis	9
7. Paralysis of the entire body	23
8. Ascending paralysis	32
9. Descending paralysis	13
10. Combination of spinal and cranial nerve paralysees	34
11. Isolated cranial nerve palsies	22
12. Localization uncertain	60
	<hr/>
	868

The figures of the Committee of the New York Neurological and New York Pediatric Societies give similar results.¹

The bladder is frequently involved in the early stages. Urinary retention is frequent, incontinence is rare. As a rule, the disturbance is transitory, hence overlooked. It is frequently thought to be a symptom of the general infection, but Wickmann regards it as due to central nervous lesions.

Constipation is not unusual, but marked disturbances of the intestinal functions are rare.

Sensory Disturbances.—The older teachings that sensory disturbances are uniformly absent is not true. Almost invariably there is in the beginning of the disease a marked hyperesthesia. The slightest touch causes marked reaction. Loss or diminution of sensation is also not uncommon. Complete anesthesia is rare, but has been observed. Hypesthesiae are common, and may include both thermal and pain sensibility. In young children the difficulties in observation cause one to overlook these sensory anomalies.

Atrophic Stages.—For a variable length of time, a year at least, the palsied or paralyzed muscles gradually regain their form and their function, but sooner or later, depending on the grade of central involvement, a more or less permanent state of inactivity is reached—a residual period in which the final account of stock may be taken. In this stage one meets with the definite atrophies, the beginning of the various deformities, changes in the bones and joints, and the residual secretory and trophic anomalies.

¹ Nervous and Mental Disease Monograph Series No. 7, New York, 1910.

The various deformities belong more in the domain of orthopedic surgery and cannot be taken up here. Pes equinus, pes calcaneus, pes valgus, pes varus, hyperextension of the knee-joint, genu valgum, genu varum, scolioses, kyphoses, lordoses, torticollis, flail arm, etc., are among the more common residual deformities. Seeligmüller's monograph, already cited, treats of them at great length.

The permanent vasomotor disturbances are chiefly cold extremities and cyanosis. Dryness of the skin is frequent.

2. *Acute Ascending Form—Landry's Paralysis.*—Landry, in 1859, described an acute ascending paralysis, which later study has shown to be, for the most part at least, a true polioencephalomyelitis. Here the palsy shows itself usually first in the lower extremities, then the muscles of the hip, the abdomen, the thorax, and the cranial nerves, are involved and death generally occurs through implication of the cardiac and respiratory centers. Death takes place in from four to five days, usually with clear consciousness, or slight coma. The sensibility is usually intact, or only slightly dulled.

Occasionally the patients recover, and then show the residuals of a severe spinal poliomyelitic type, with mixture of bulbar or pontine features.

A descending form is also to be distinguished. This is much rarer. Here the bulbar symptoms develop early, and the spinal extension shows later.

The majority of the fatal cases of epidemic polioencephalomyelitis show the type of a Landry paralysis.

3. *Bulbar and Pontine Forms.*—Here the features that stand in the foreground, either as initial or as residual conditions, are the cranial nerve palsies. A large number of the patients with poliomyelitis show some cranial nerve complications, but when these are the chief features, and the spinal palsies are the minor complicating factors, then one speaks of the bulbar and pontine types.

Many of these patients show isolated palsies—others have two or more cranial nerve involvements. The facial is oftenest implicated (10 per cent.), next the hypoglossal. These are usually unilateral, although Medin has described a bilateral facial palsy. Eye muscle palsies are infrequent. The third and fourth less often than the sixth (Wickmann, Müller).

Ptosis, ophthalmoplegia interna, externa, nystagmus are among the rarities. Pupillary disturbances are not infrequent, according to Lundborg. Here both light and accommodation reflexes are interfered with. Very rarely one observes amaurosis with optic nerve involvement. The presence of choked disk in meningitis is an important differential. The IXth, Xth, XIth are involved, usually in the fatal cases, occasionally unilateral pharyngeal and laryngeal palsies are residual conditions. Speech disturbances are present, but infrequent. Midbrain involvements with peculiar tremors, vertigoes, rotatory movements, ataxias, etc., are among the curiosities.

4. *Encephalitic or Cerebral Form.*—Strümpell called particular attention to the possibility of a pure encephalic form of the disease, thus widening the conception from a poliomyelitis to a polioencephalomyelitis. Medin's valuable study confirmed his teaching, and the investigations of recent years have still further amplified the findings. Here the inflammatory reaction spreads throughout the entire cortex, as in the cord. The central and frontal gyri, the basal ganglia, the internal capsule, and centrum ovale are most frequently involved.

Here, in addition to headache, stupor, and convulsions one encounters spastic palsies, hemiplegic or diplegic in type, usually associated with bulbar palsies.



FIG. 156.—Eye palsies, poliomyelitis.
(Frauenthal.)



FIG. 157.—Facial nerve palsy, poliomyelitis.
(Frauenthal.)

These cerebral forms are probably rare, and often fatal. They are also rare in experimental monkey poliomyelitis. Müller is inclined to regard the spastic palsies that occasionally occur as due to pontine rather than to motor cortex involvements, whereas Wickmann lays particular stress upon the probabilities of their cortical origin.

5. *Ataxic Forms.*—Medin described forms in which the patients showed ataxia in walking, with staggering or ataxic gaits, and others behaving like Friedreich's disease of acute onset. These forms are closely allied to the bulbar and pontine types, and also may be occasioned by extension of the disease to the cerebellum. The latter structure is almost always involved to some extent in this disease.

6. *Meningitic Forms.*—Here meningeal symptoms occupy the foreground. Headache, vomiting, pain in the neck with stiffness, Kernig's sign, stiffness of the back, opisthotonos, convulsions, strabismus, somnolence, and unconsciousness are present. These cases either then

develop marked spinal and bulbar symptoms of the ordinary type, or the symptoms recede with either minor residuals, eye palsies, etc., or go on to complete recovery.

7. *Polyneuritic Forms.*—The study of recent epidemics has shown the great frequency with which pain is found in the initial history. In many cases there are painful nerve trunks, with Lasègue's phenomenon, and great hyperalgesia over the entire body, resembling polyneuritis. Anatomically, however, marked neuritic changes are wanting. These cases Wickmann prefers to call neuritis-like.

8. *Abortive Forms.*—The study of the epidemics of 1909–1912 has shown that in a number of patients the illness began with the characteristic symptoms of poliomyelitis, and then went on to recovery without any palsies. In others again, widespread, though mild, palsies with hypotonia developed and total recoveries occurred within a short time. Wickmann brought these facts into prominence, and showed that these were to be regarded as abortive cases. The most frequent forms under which these cases develop are (1) that of a mild meningitic type with the usual prodromal signs, associated with the neck symptoms, stiffness, pains, sometimes opisthotonos and the like; (2) cases with the symptoms of a general infection only; (3) cases running a course like an influenza; (4) cases with marked gastro-intestinal signs.

Wickmann has estimated that at least 15 per cent. of all the cases can be grouped under this rubric, while Müller believes them to occur much oftener, indeed, more often than the usual type. Their significance in the epidemiology of the disease is great, because it is highly probable that its spread may be conditioned by these ambulatory abortive cases. They are more frequent in children, but may also be present in adults. If Müller's stand be correct, viz., that they occur oftener than the well-developed forms, their importance from this stand-point is enormous. The question of rarity of the disease after puberty may be solely conditioned by the fact of previous abortive attacks in many individuals. Müller states that in the study of small epidemics he has found that in the abortive cases there is a marked tendency to show the same symptoms in their epidemic extension. Thus gastro-intestinal cases give rise to gastro-intestinal cases, respiratory to respiratory, meningeal to meningeal, etc.

Diagnosis.—Sporadic and epidemic polioencephalomyelitis are conditioned by the same etiological factors. If an epidemic is in progress, particular attention should be given to all acute infections, whether they show marked palsies or not, and careful examination be made of nerve tenderness, hypotonias, tendon reflexes, and limb motility. Most cases develop either in the guise of general infections with temperature, or with distinct local symptoms, either in the respiratory or gastro-intestinal tracts, or in the meninges. Influenza, polyneuritis, anginas, bronchitis, gastro-enteritis, typhoid and epidemic cerebrospinal meningitis come into review. A marked general hyper-

esthesia, and a distinct pathological tendency to perspiration is significant. Leukopenia, according to Müller, with fever is also of importance. Sleepiness, to drowsiness of the children, during the day, wakefulness and fretfulness at night, easy fatigability, weakness of the extremities, loss of muscular tonus, especially in the abdominal muscles, with meteorism and loss of the abdominal reflexes, point to poliomyelitis. An early lumbar puncture will resolve many of the difficulties. Influenza is separated with considerable difficulty in the early stages, so much so that certain observers (Borström) hold that poliomyelitis is nothing but a severe neural type of influenza. Monkey experimentation has disposed of this hypothesis.

Polynuritis also offers particular difficulties. This is rare in children, apart from diphtheritic neuritis, and is usually quite symmetrical in its development. The time needed for development of the palsies is longer, the pains are more persistent, there are usually more objective sensory disturbances, particularly deep sensibility, and early edemas are more frequent.

In diphtheritic neuritis, cardiac irregularities are the rule, in poliomyelitis the exception; the palsies of the palate are further characteristic in the former.

Pure neuritic-like forms of poliomyelitis are sometimes present, but there is here more tendency for a mild dissociation syndrome, diminution of pain and temperature sensibility. Neuritis is more apt to include all the forms of sensibility, or gives a diminution in touch, (epicritic) with an increase in pain sensibility. In very small children these differences are difficult to bring out. Williamson lays stress upon the loss of bony sensibility in neuritis, whereas in poliomyelitis it is rare.

Further differentials from Wernicke's polioencephalitis, superior myelitis, hematomyelia, myatonia congenita, hysteria, Parrot's palsy in hereditary syphilites, etc., must be sought for in monographs.

In the meningitic forms, and in many of the ordinary spinal cases the separation from spinal or tuberculous meningitis is very difficult. The lumbar puncture here usually clears up the diagnosis. Clinically, the more marked mental symptoms, the high degree of stiffness of the spinal column, Kernig's sign, and at times papillary edema, ear complications, and herpes, all speak for meningitis. In tuberculous meningitis the spinal fluid reactions and the longer course usually establishes the diagnosis.

Prognosis.—The older teachings that the disease is rarely fatal, and always shows persistent palsies, must be modified in both directions. Many fatal cases do occur, and total recoveries are frequent. In certain epidemics the mortality is very high (42 per cent.), in others only 10 per cent., counting only those patients with evident palsies. In the New York epidemic of 1907 the mortality was approximated as 5 per cent.

If the abortive cases are included the percentage falls markedly.

The mortality is worse in the older patients, as high as 50 per cent. in one epidemic (Lindner and Malley). The period of danger lies usually in the fourth and fifth day of the disease. In the second and third week, bronchopneumonia is a dangerous complication.

As for total recoveries, they have varied from 10 to 50 per cent. in the various epidemics, and are more common in younger children than in those over fourteen years of age. (See Wickmann.)

From the stand-point of electrical prognosis, the older views are certainly false. The whole subject is in need of entire revision. A total loss of faradic excitability after a week is no certain criterion of permanent palsy, as has been taught by Oppenheim and others.

Reparation takes place most rapidly in the first six months, but continues throughout a year or more, and with continuous, rational treatment weak and paralyzed muscles will continue to improve for many years.

Treatment.—Prophylaxis, treatment of the acute stage, and of the chronic stages are to be distinguished.

Prophylaxis.—Isolation and disinfection are as yet unprecise in their application. The mode of transmission of the virus is as yet unknown, and whereas there is little difficulty in isolating the severely attacked, the abortive cases are not, and rarely can be, properly regulated. That abortive cases do carry the disease seems definitely proved. The proper length of time for isolation has not been determined. Wickmann regards three weeks as sufficient, Müller eight weeks. As yet the data do not permit definite counsels.

The virus in monkey poliomyelitis seems to have been found in the nasal mucous membranes and in the feces. Hydrogen peroxide, 1 per cent. solution, and menthol solutions can be used for the former, formaldehyde or carbolic acid for the latter. Special attention should be directed toward the pocket handkerchief.

Rooms may be disinfected with formaldehyde. Preventive inoculation, analogous to Pasteur rabies treatment, may become practicable in time.

Acute Stage.—Absolute rest in bed is to be enforced, and if an epidemic be in progress, children with mysterious diarrheas, pseudo-influenzas, mild neuritic pains, etc., with fever, should be kept in bed, even after they appear to have recovered. The patients who have been sick for a couple of days, and who get up and around, and are then suddenly stricken down, are in reality very numerous. Foresight here cannot be overvalued.

Counter-irritation to the spine, by mustard plaster or other means; prompt catharsis, first by enema and then by saline cathartics, should be employed. The motor restlessness and pain are best controlled by analgesics, and the various salicylate preparations. Codein may be employed, but its inhibitory action upon the bladder should not be overlooked.

Diaphoresis by hot packs is helpful for the pains, muscular soreness,

and, possibly, in aiding elimination. Frequent treatment by free use of deep, warm baths, 102° to 104° F. is highly desirable. The warm water relieves the sense of pain greatly, and the irritability, and is greatly appreciated by the patient. The baths can be repeated every three or four hours; the time in the bath is from ten to twenty minutes. On taking the child from the bath, he should be rolled in a blanket and dried in bed. Continuous warm baths are worth trying.

In the later stages the bath treatment is invaluable for straightening out the contracted limbs and aiding in active motion.

Urotropin may be administered. It is thought to reduce to formaldehyde in the cerebrospinal fluid in sufficient amounts to act as an antiseptic. The hypothesis lacks definite experimental confirmation.

Chronic Stages.—After the acute stage has passed there is the long, hard stage, lasting for weeks or months or years, of partial or total paralysis, great weakness, extreme tenderness, nervous irritability, and muscular pains, with gradually developing deformities.

The diet should be full and regularly administered. A careful inventory should be made of every affected muscle, both in terms of its functional capacity, and its electrical excitability, the milliampères necessary for bringing about contraction being carefully noted. Full measurements of the limbs should be taken. Careful note must be taken of the position of the limb in bed, and appropriate support given to relieve pain, avoid stretching, and diminish contraction. Bed-sores, sore heels, elbows, etc., need very careful treatment.

Chief reliance is now laid upon massage, passive motions and resistance motions. Here the deep, warm bath is invaluable, serving to help the motions of the limbs, and to straighten out the contractions. Movements in water are more easily performed, and small children can play in the deep tub for hours. Special exercises should be planned, both for the water and in bed. Special calisthenics must now be developed, depending upon the muscle group involved.

Particular attention must be directed to the mental life of the child. He is apt to be morose, reticent, shy, and resentful, becomes very sensitive if his deformity be marked, and selfishness and exactingness develop easily if pampered because of his weakness.

In the later stages, and for those old enough, swimming is the best exercise.

The correction of the deformities is a matter for the orthopedic surgeon. Mechanical aid should be given as soon as possible, if it carries out a progressive therapeutic principle. Operative procedures should be conservatively considered, and not used too early. These patients make wonderful recoveries unaided, or by the persistent use of massage and mechanotherapy. Many operative procedures are, however, imperative. Nerve splicing, and tendon splicing have their special indications.

Electrical therapy is of use only as a bridge to gap the period after

the first onset until such time as definite willed movements can be started.

Drug therapy is of purely symptomatic value. Iron, strychnine, calcium are the most useful remedies, and meet special indications.

PRIMARY PROGRESSIVE MUSCULAR ATROPHIES.

The spinal motor neuron is differentiated into three structures, termed the anterior horn cell, the motor nerve fiber, and the muscle plate. It would be a great advance toward the simplification of the vexed questions regarding this group if a clinical division could be made, in strict accordance with the pathological alterations of these structures. To a certain extent this is justified. For categorical purposes then one can distinguish:¹

1. The progressive nuclear atrophies, or myelogenous or myelopathic types: *Etiology*: Heredity, trauma, cold, toxins, infections. *Anatomy*: Primary degeneration of the anterior horn cells, with secondary fiber or muscle changes. *Forms*: Infantile, Hoffmann-Werdnig; adult, Aran-Duchenne; mixed forms.

2. The neural, neuritic, or spinal neuritic atrophies: *Etiology*: Heredity. *Anatomy*: Primary degeneration of the motor nerve fibers with secondary ganglion, cord, and muscle changes. *Forms*: Peroneal-arm type (Charcot-Marie-Tooth); tabetic type (Dejerine-Sottas); peroneal type and arm type (Sainton and Haenel).

3. The muscular dystrophies or myogenic types. *Etiology*: Heredity. *Anatomy*: Primary changes in muscles. *Forms*: Hereditary (Leyden-Möbius); juvenile pseudohypertrophy (Landouzy-Dejerine); and mixed forms (Erb-Zimmerlin).

The separation of these forms has occupied the attention of neurologists since about 1849 when Duchenne first attacked the problem of dismemberment of this large group with muscular wasting.

Clinicians for many centuries had described progressive muscular wasting. Pictures and images in stone and wood of the muscular atrophies and muscular dystrophies dating from the fifteenth, sixteenth and seventeenth centuries are in existence. Van Sweiten, Abercrombie, and others gave general descriptions. This group was first broken into by Duchenne, in 1849, by the loose description of a special type, which a year later Aran (1850) supplemented. Cruveilhier, in 1853, and Luys, in 1860, sharpened the picture somewhat by their demonstration of the exclusive implication of the anterior horns. In 1865 Charcot eliminated the amyotrophic lateral sclerosis from the group. Duchenne himself had, in 1853, also set aside the pseudohypertrophies, the muscular features and varieties of which were later demonstrated by Eulenberg (1866), Charcot, Leyden, and Dejerine. Wachsmuth, in 1864, recognized the bulbar forms. Finally, Duchenne also called

¹ Kügelgen, Beitrag. zur neuralen progressiven Muskelatrophien, Arch. f. Psych., 1909, xlv, 944.

attention to the presence of sensory anomalies in certain of his 1853 studies on the atrophies; these were for the most part syringomyelias, which Kahler and Schultze separated definitely in 1882. Another small group were the neuritic atrophies, first clearly recognized by Dumesnil (1864). Thus it took thirty years for the sorting out of this medley of muscular atrophies; the groups as they are at present recognized, at least two of which, syringomyelia and amyotrophic lateral sclerosis, being entirely set outside of the muscular atrophies *per se*. One result was that the original disorder of Aran-Duchenne was so much reduced that finally Marie, in 1894, tried to sweep it away entirely, but his iconoclasm has not been justified.

Group 1. The Progressive Nuclear Atrophies: (1) Spinal, (2) Bulbar, and (3) Mesencephalic Forms.

Three spinal main forms are here to be recognized. (a) Chronic poliomyelitis *per se*, with lesions limited primarily to the motor ganglion cells; (b) Aran-Duchenne's disease, progressive spinal muscular atrophy and (c) infantile hereditary—Werdnig-Hoffmann.

The former two forms are much alike, and many authors maintain their identity. They are here considered separately, since such a course seems justified clinically if not pathologically.¹

(a) **Chronic Poliomyelitis.**—Here are grouped those rare subacute or chronic muscular atrophies usually occurring in the late years of life, in which certain toxic agents seem to account for the disorder. Such toxic factors are lead, diabetes, syphilis, and other unknown toxemias. Heredity is sometimes present.

The anatomical lesions are a progressive destruction of the motor ganglion cells, with chronic vascular alterations.

Symptoms.—There is a progressively developing flaccid paralysis, with antecedent weakness, without sensory or trophic signs, and diminution or loss of tendon reflexes. The nerve trunks are not tender or swollen. Fibrillary twitches appear, the myotatic irritability is increased, reaction of degeneration is present. There are no disturbances of the rectal, vesical, or sexual functions, and the medullary nuclei are rarely involved.

The localization may vary; the legs or arms first showing weakness or atrophy, sometimes one arm or both, one arm and one leg, or both legs. The atrophy and palsies take place usually slowly, with at times periods of more rapid development, and also stationary periods during which no advance is made for years, or the patient slowly recovers.

The distribution of the atrophic muscles is characteristic. Certain muscle groups are spared. The arrangement is segmental, not radicular. The triceps may escape with all the other muscles of the arm attacked, or the flexors of the fingers may be intact. In the legs the

¹ Marburg, *Handbuch. d. Neurologie*, 1911, vol. ii, p. 280, for complete literature; Lövegreen, *Zur Kenntnis der Pol. acuta und chronica*, Karger, 1904.

tibialis anticus may stand out unimpaired. The course is usually very chronic, lasting years. Subacute onsets are more favorable. Some patients recover, especially those with suspicion of a radiculitis or a neuritis. In the progressive cases the patients at times develop bulbar symptoms, and die of aspiration pneumonia, or weakness.

Treatment.—Rest, hydrotherapy, gentle massage, electricity, over-feeding are the chief indications. Syphilitic cases need specific therapy.

(b) *Aran-Duchenne Type.*—*Progressive Muscular Atrophy.*—**Etiology.**—Here heredity may play a part in the development of a familial form, seen in infancy, and also in adults (Bernhardt). It may follow acute poliomyelitis. Other factors, as toxemias, trauma, cold and wet,



FIG. 158.—Chronic poliomyelitis showing atrophy of hands.

overexertion, are not definitely established. Occupation atrophies are at times incorrectly included here. Pathologically there is degeneration of the motor ganglion cells and fibers, with some secondary degenerations in the anterior lateral columns.

It is a rare disorder. The disease begins very slowly, the upper extremities are attacked, and rarely the lower. The smaller muscles of the hand are initially affected. Weakness, atrophy, and flaccid paralysis is the sequence. Fibrillary twitchings accompany the atrophy and there is reaction of degeneration in the muscles involved. Sensory and trophic disturbances are lacking and the tendon reflexes are diminished or lost. Early over-excitability of the tendon reflexes may be noted in both of these forms.

Oppenheim has called attention to the tendency for these atrophies to jump from one muscle group to another. The atrophy is very gradual; the disease advances slowly for years. Remissions occur, but recoveries are very unusual.

The distribution of the affected muscles varies considerably. Death usually results from paralysis of the muscles of respiration.

Diagnosis.—The separation of these two forms is at times impossible. There is a tendency for the former to advance more rapidly, to involve the lower limbs, to advance to the medulla, to show longer remissions,

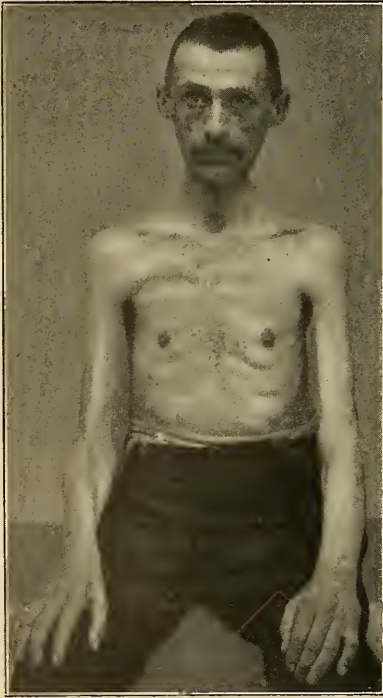


FIG. 159.—Primary nuclear atrophy.
(G. M. Hammond.)



FIG. 160.—Primary nuclear atrophy.
(G. M. Hammond.)

and to recover. Further, there is a greater tendency to segmental distribution in the muscle groups affected in the former type, with marked loss of power followed by atrophy. In the latter type the atrophy seems to precede the palsies, and the patients are often very adept with their residual muscles.

The sensory disturbances of occupation atrophies, of neuritis, of syringomyelia should exclude these, while the increased tendon reflexes of amyotrophic lateral sclerosis eliminates that disorder. Radiculitis needs separation. Here the atrophies are apt to be unilateral, and are

radicular. There is a low-grade neuritis, and the Dejerine-Klumpke syndrome appears in the cervical type.

Treatment.—This may be treated as the preceding affection, but such therapy seems of little avail. Strychnin, arsenic, and fats are indicated. Newer ideas must be gained if any effective control of this disorder is to be hoped for; the older methods are useless.

(c) **Infantile Hereditary Forms** (Werdnig¹-Hoffmann²).—These are rare hereditary types, beginning in infancy, first described in 1891. The disease begins gradually, usually in the pelvic girdle and thighs (ileopsoas, quadriceps femoris). Later the back, neck, and shoulder girdle is involved. Finally the distal extremities are involved. The intercostals and diaphragm are often affected. Occasionally the bulbar muscles atrophy. The atrophies are usually symmetrical, often associated with apparent hypertrophy (adiposis).

There is gradually increasing paresis and loss of skin and tendon reflexes. Fibrillary twitching seems absent and a peculiar trembling of the fingers is described. Scolioses, talipes, and other contracture states develop. These little patients often acquire unique modes of locomotion. Faradic currents are borne better than galvanic currents, but reaction to both is diminished or lost.

The course is usually progressive, rarely stationary.³ Mental reduction is not usual.

(2) **Bulbo-pontine Types. Chronic Progressive Bulbar Palsies.**—In these forms the progressive atrophy is limited to the muscles of the face, tongue, palate, and larynx.

Etiology.—Nothing is known of the causation factors. Certain toxic factors—lead, syphilis—have seemed to play a role at times; constantly recurring electrical shocks was a factor in one patient personally observed; the majority give no clue as to etiology. A few cases are observed in childhood, still fewer in adult manhood; the majority occur after thirty-five years.

Symptoms.—There is a slow progressive weakness of the tongue, and muscles of the cheeks and lips, followed by fibrillary twitchings and slowly progressive atrophy. Speaking becomes fatiguing, and slight changes in the voice become apparent. In eating the patients find they must use their fingers in dislodging food from behind the teeth in the cheek. Gradually increasing difficulty in swallowing is observed, and difficulties in breathing appear. Finally the speech becomes lalling, dysarthric, the lingual letters *d, t, l, r, n, s*, are first slurred over, then the labial letters *b, p, f, m, w, o, v, e*. The laryngeal weakness causes hoarseness, monotony, and finally aphonia. The pharyngeal muscles and those of the tongue do not act in concert, and liquid food regurgitates through the nose.

¹ Archiv f. Psychiatrie, 22, 26.

² Deutsche Zeitschrift f. Nervenheilkunde, 3, 10, 18.

³ Late literature Batten. Lancet, June 3, 1911; Senator, Charité Annalen, 1902; Lange, Deutsche Zeitschrift f. Nervenheilkunde, 1910, 40.

The face becomes atrophied below, the lips thin and folded. The patients cannot protrude the lips, nor whistle. The temporals and masseters also become affected, and the movements of the jaw become impossible. The masseter, pharyngeal, and vomiting reflexes are absent. The sensibility is intact. The upper face region, including the levator palpebræ, is rarely affected.

Vasomotor disturbances are occasionally observed. Vagus involvement causes cardiac irregularity.

The usual progression is from the tongue to the lips, then to the other facial muscles, and finally to the fifth, and hypoglossal and vagus muscles. An apparent increase in saliva, an annoying symptom, is largely dependent upon the inability to swallow.

The usual course extends over four or five years, death taking place most frequently from pneumonia or from inanition.

Pathology.—Here one finds changes in the medullary nuclei, precisely analogous to those found in progressive spinal nuclear atrophy. The corticobulbar tract may be involved somewhat, but rarely to the degree found in amyotrophic lateral sclerosis, yet intermediary forms are to be expected.

Diagnosis.—Unusual types of gliosis, of multiple sclerosis, tumors, gummata, arteriosclerosis, general paresis may cause somewhat similar pictures at first, but these also soon show other symptoms, indicating that the lesion is not confined to the nuclear structures. Pseudobulbar palsy shows palsied muscles, still electrically reflexly excitable, without atrophies. Myasthenia gravis shows the typical electrical myasthenic reaction.

Prognosis.—Absolutely bad, save in syphilitic cases.

Treatment.—Thus far none is known. One should always be on the lookout for the other causes of the syndrome, however, in the initial stages. Here a nihilistic therapy will be recognized too late for recovery.

Galvanization has seemed to help the swallowing of some patients. Atropin, hyoscyamus, etc., or other related drugs can be used to control the salivation, and the pains and discomforts of coughing, dyspnea, etc., relieved by narcotics. Specific therapy is valuable in the Wassermann positive cases.

3. Pontomesencephalic Forms.—Chronic Progressive Ophthalmoplegia.—Chronic progressive ophthalmoplegias as a part of an amyotrophic lateral sclerosis, of tabes, of general paresis, multiple sclerosis, tumor of corpora quadrigemina, or other organic disease are not rare. As pure, chronic nuclear affections they are infrequent. Uthoff credits them as high as 14 per cent. of the chronic ophthalmoplegias.

There is beginning weakness of the eye muscles, usually the external rectus, with slight internal strabismus, frequently worse at night. Diplopia is not usually present, because of the gradual development of the disorder, and its symmetry. In hereditary types, in infants, binocular vision may not have developed.

The internal eye muscles are usually intact, although occasionally irregular pupils occur; light-immobile pupils are not present although loss of accommodation movements have occurred. Ptosis is not infrequent; usually worse on one side.

In a completely developed case the Hutchinson face develops bilateral ptosis with inability to move the eyeballs.

The disorder is usually progressive. Starr has described a stationary condition, and some patients recover entirely.

Treatment.—When a Wassermann test has revealed a positive reaction, mercury and iodides are indicated; otherwise general tonics, strychnin, iron, arsenic, and general dietetic treatment directed to the reduction of arteriosclerotic changes.

Group 2. The Neural, Neuritic, or Spinal Neuritic Atrophies.

Hard and fast lines cannot as yet be drawn between the neuritic atrophies from certain nuclear atrophies on the one hand, nor certain dystrophies on the other. Jendrassik, in a recent monograph (1911), speaks of them as “dystrophy forms with degeneration of the peripheral nerves.”

Many forms of neural atrophy have been described. The most characteristic are: (1) the peroneal, forearm type—Charcot-Marie-Tooth; (2) the tabetic, or hypertrophic interstitial neuritic type—Dejerine-Sottas, Marie’s familial form; (3) the peroneal type—Sainton; (4) the arm type—Haenel.

1. **Peroneal-forearm Type.**—Charcot, Marie and Tooth described this form which is characterized by muscular wasting in the distal parts of the extremities, from knee and elbow outward.¹

Etiology.—Eichorst found thirteen cases in six generations and Haenel thirty-two in four, while others have found only one.

Symptoms.—The feet are usually first involved. There is early equinus position from loss of power of extension of the great toe. Then the leg, from the knee down, thins and atrophies. The gait thereby becomes either widespread or steppage. A similar process begins in the forearms, usually advancing from the muscles of the hand. Monkey hand, or *main en griffe* develops. Rarely the upper extremity atrophy precedes. The shoulder, arm, neck, back, hips, and thigh muscles remain intact.

Sensory changes are usually present, and slight pains, intermittent and lancinating in character, coming and going, hyperesthesia, parasthesia, hypesthesia, especially to cold, are present. Painful nerve trunks are occasionally found. Vasomotor disturbances are frequent. Cold marble-like skin with increased secretion is observed.

The knee-jerks are often initially increased; later they are diminished

¹ 1881, *Rev. de Méd.*, 1886; *Brain*, x, 243; Spiller, *Journal of Nervous and Mental Disease*, 1907.

or absent. The Achilles reflex is usually absent, as is the radius-periosteal reflex.

Reaction of degeneration in the regions affected is the rule. Fibrillary contractures and muscle unrest are constant, often persisting during sleep. Tremors and choreic-like movements are not uncommon.

Course.—The disease usually begins in childhood, advances slowly, usually progressively, sometimes halting for long periods.

Pathology.—Since Hoffmann's description (1889) of a neuritis in the distal peripheral nerves, this disorder has passed as a mixed neu-



FIG. 161.—The neuritic form of muscular atrophy. (Spiller.)

ritic atrophy, but later Siemerling, Gierlich,¹ Kugelgen,² Spiller³, and others have shown changes, not only in the peripheral nerves and muscles, but that there were extensive changes in the other parts of the nervous system, in the eighteen to twenty cases thus far autopsied (1910). These show parenchymatous and fatty degeneration of the muscles, ascending degeneration of the peripheral motor nerves, especially intermuscular branches, with chronic interstitial neuritis,

¹ Arch. f. Psychiatrie, 1909.

² Ibid.

³ Loc. cit.

degeneration of spinal ganglia, atrophy and loss of anterior horn cells, sclerosis of columns of Goll, and Burdach, with changes in lateral columns and posterior roots.

2. **Tabetic Type** (Dejerine-Sottas¹).—This is apparently an hereditary form, the symptoms beginning in childhood. The muscular changes are similar to those seen in the Charcot-Marie forms, but the sensory nervous system is much more markedly involved. There are present pupillary changes—myosis, or even Argyll-Robertson, beginning or



FIG. 162.—The neuritic form of muscular atrophy. (Spiller.)

complete Romberg, nystagmus, and ataxia. Pain is present. The peripheral nerves are enlarged and palpable. Reaction of degeneration was stated to be absent by Dejerine and Sottas.

It may easily be confounded with a juvenile tabes. Dejerine maintains that it has no relation to the Charcot-Marie atrophies.

Other forms are described in which the atrophies are limited to the

¹ Arch. d. Neuro; II Ser., vol. xvii, No. 91. Rev. de Med., 1897; Rev. Neur., 1902, 1906.

lower extremity (Sainton), to the upper extremity (Haenel), to the femorotibial region (Eichorst). The transition types are many.

Diagnosis.—A complete separation of all the forms is not possible in the present state of our knowledge. The Wassermann reaction and cerebrospinal fluid examination will probably throw considerable light on the hypertrophic neuritic types of Dejerine, and may remove them from this group entirely; also cases described by Marie under a similar name.

For the classical cases of neuritic muscular atrophy there is little ground for confusion, but the numerous aberrant forms introduce difficulties.

The separation from the dystrophies is usually made on the ground of pseudohypertrophy and the absence of reaction of degeneration in these cases. Certain transition forms are undifferentiable.

Chronic polyneuritis is rarely hereditary (unless one follows Oppenheim in classing the neuritis atrophies here under consideration as hereditary chronic multiple neuritis). In polyneuritis there is rarely a club-foot, the progress is more rapid, and recovery is apt to take place. From tabes, only Dejerine's type can cause confusion. The age and heredity are the chief features. Newer studies on complement fixation and spinal fluid are wanting.

In amyotrophic lateral sclerosis the increased reflexes, the extension to the bulbar nuclei, the spasticity, and rapid course are characteristic. Multiple sclerosis, syringomyelia, chronic poliomyelitis, myotonia, and hereditary ataxia occasionally call for differentiation.

Prognosis.—Usually poor, but the disease is very chronic, and patients die after twenty to thirty years of intercurrent disorders. Sometimes stationary periods are met with.

Therapy.—Electricity, baths, massage, internal secretions, general tonics, and orthopedics are useful. A few cures are known.

Group 3. The Muscular Dystrophies or Myopathies.

This very large and extremely motley group has also been built up of a variety of forms since Duchenne, in 1849,¹ first described the fatty pseudohypertrophies, and later, in 1868, spoke of them as myoscleroses. Leyden² (1876), Möbius³ (1879) described certain hereditary forms, while Erb⁴ in 1883 first brought some order into the confusion of the atrophies and dystrophies by showing that in certain forms this lesion was predominantly muscular and not nervous. He made the first practical synthesis. Landouzy and Dejerine⁵ in 1884 described their well known form, and separated it from Erb's juvenile type. Since this time the group has been better unified,

¹ Union Med., 1853.

² Volkmann's Klinik, No. 171.

³ Comptes rendus, 1884, p. 53.

⁴ Klinik de R. II, 1876.

⁵ Neurol. Ctbl., 1883, p. 452.

its limits better recognized, and the various forms within it more thoroughly studied.¹

The myopathies make a fairly consistent group, although the forms may not resemble one another clinically at different periods of their development, yet they have a number of common factors.

Heredity is a common feature; they usually occur at an early age; the muscles become weak gradually and atrophy in a peculiar manner, in that true hypertrophied fibers are mingled with atrophied fibers. The muscular atrophy may involve all of the muscles equally, or may be irregularly distributed both as to the body in general or within the muscle itself. Reaction of degeneration and fibrillary contractions are usually wanting, although a gradual loss of electrical excitability goes on coincidentally with the atrophy.

Certain muscles, pectoralis major, rhomboid, serratus magnus, are oftenest the seat of early atrophy. These are also characterized as congenital aplasias (Bing). The muscle electrical reaction curve is striking.

The tendon reflexes gradually disappear, but the Achilles is apt to persist, or occasionally be increased, especially with much pseudo-hypertrophy. Sensory disturbances are usually absent, likewise bladder and visceral disturbances. Pseudo-contractures with limitation of movement is frequent, causing peculiar positions. The patients hop like frogs. Bony dystrophies are also frequent, mostly showing in thinning of the long bones, with cranial deformities, deformed hands, short hands and short feet.

Pathology.—Erb based his synthesis upon the changes he found in the muscles, but at the same time was inclined to attribute them to lesions in the trophic centers. These muscle changes consist in the main in hypertrophy and atrophy, and splitting of the muscle fibers, proliferation of the nuclei, new connective tissue proliferation with hyperplasia of the vascular tissues and fatty deposition. Macroscopically the muscles have lost their normal color, varying from pale pink to dark red. In places where the muscle substance has entirely disappeared white connective tissue is apparent.

The peripheral nervous system is usually negative. The muscle plates are frequently missing.

Loss of cells in the ventral horns has been described by Holmes² and others. Whether these are primary or secondary cannot yet be determined.

The change in the bones is in the nature of a rarefaction.

Our knowledge of the trophic cells of the cord is still too incomplete to overthrow Erb's original conclusion, although the evidence seems to point to this group as one of muscular origin.

¹ Batten, *The Myopathies or Muscular Dystrophies*, *Quart. Jour. Med.*, April, 1910. Lorenz, *Krankheiten d. Muskeln*, 1904; Jendrassik, *Handbuch d. Neurol.*, 1911.

² *Rev. Neur. and Psych.*, 1906, vi, p. 136.

Forms.—The forms are many and show considerable intermingling. They are:

1. Pseudohypertrophic (Duchenne).
2. Juvenile (Erb).



FIG. 163.—Pseudohypertrophic myopathy. Early stage. (Jendrassik.)



FIG. 164.—Pseudohypertrophic myopathy. Stage of atrophy. (See 163.) (Jendrassik.)

3. Facio-scapulo-humeral (Landouzy-Dejerine).
4. Atrophic myatonia congenita (Oppenheim).
5. Myatonic atrophica.
6. Distal (Gowers).
7. Mixed and transitional forms.

1. *Pseudohypertrophic Type* (Duchenne, 1849).—Semmola, in 1834, and Costa and Gioja, in 1836, antedated Duchenne in describing these cases, but pictures of earlier centuries give evidence of its presence. It is the type most frequently observed. It is more common in males (3 to 1), and usually begins during childhood. An hereditary form is very frequent. The parents first notice a certain clumsiness in the gait of the children, then the position of the body is peculiar,



FIG. 165.—Pseudohypertrophic myopathy. Later stage of atrophy. Compare 163, 164. (Jendrassik.)

the head being bent forward, and the cervical vertebræ are particularly prominent. There is an early beginning lumbar lordosis. The patient waddles, then commences to find it hard to go up stairs—often trips and falls. On rising from a recumbent position the arms are called in to aid, and the mode of rising is unique. The patient climbs up his legs as it were with his arms. In the final stages the patient is unable to raise himself at all.

The shoulder-blades are freely movable and rise with the rise of the arms. Atrophies are apparent.

The gait varies somewhat, according to the muscles chiefly involved. It is often wabby, like a pregnant woman; at times it has a high step



FIG. 166.—Pseudohypertrophic myopathy. Later stage. Compare 163, 164, 165. (Jendrassik.)

character; again the patient walks on his toes. The lower limbs often show marked hypertrophy in the early stages, the calves are plump and firm.



FIG. 167.—Pseudohypertrophic myopathy. Later stage. Compare 163, 164, 165, 166. (Jendrassik.)

The atrophy advances unequally. The muscles most affected in the lower extremities are the psoas, glutei, quadriceps, sartorius, adductors, gastrocnemius and soleus; in the trunk, the rectus abdominis, latissimus dorsi, erector spinæ, rhomboidei, infraspinatus,

serratus, trapezius, and pectoralis major, while in the upper extremities the muscles mostly implicated are the deltoid, biceps, brachialis, and brachioradialis.

In advancing cases all of the muscles go, save perhaps those of the face. In rare cases the face is involved (myopathic facies), and



FIG. 168.—Pseudohypertrophic myopathy.
(G. Hammond.)



FIG. 169.—Pseudohypertrophic myopathy. (G. Hammond.)

in a few cases bulbar muscles are diseased. In the less advanced cases many of the distal muscles can be utilized.

The patients usually die of intercurrent disorders after many years of illness.

2. *Juvenile Form* (Erb).—This usually develops about the age of puberty, with weakness and atrophy in the shoulder girdle. The

deltoid may show hypertrophies. The arm is usually thinner and more atrophic than the forearm, and typical winged scapulæ develop. In walking the patients not infrequently bend forward, from weakness of the trunk, and support the back by holding the thighs. Pseudo-hypertrophy of the calves is not uncommon.

3. *Facio-scapulo-humoral Type* (Landouzy-Dejerine).—Here the facial atrophies usually develop early, particularly the orbicularis oris. The sphinx-like face develops, the patients are unable to whistle, tapir mouth is often present, and their smile is distorted. The eye-lids hang and cannot be closed completely. The shoulder-girdle atrophy then advances, the waist is small, wasp-like, the chest flattened; finally the dystrophic process becomes universal.



FIG. 170.—Pseudohypertrophic myopathy. (G. Hammond.)

4. *Amyotonia Congenita*—Myotonia Congenita—Oppenheim¹ (Wilson).—It is not certain whether this disorder should be included among the dystrophies or not. Pathologically it seems identical, clinically it is quite at variance. Spiller made the first autopsy. The number of cases known (about 60—1911) prevents a definite answer at the present time.² Rothmann is inclined to ally it with the Werdnig-Hoffmann spinal nuclear atrophies as a congenital variety.

Symptoms.—The disorder is usually congenital, hypotonia is characteristic, with loss of tendon reflexes. Active motion is impaired by

¹ Monats. f. Neurologie u. Psychiatrie, 8, 1900, p. 232.

² Latest literature: Collier and Holmes, Brain, 1909; Batten, loc. cit.; Cassirer, Handbuch d. Neur., 1911; Griffith, Arch. Kindhk., 1910; Griffith and Spiller, Am. Jour. Med. Sci., August, 1911.

reason of weakness, but the limbs are not paralyzed. Usually the lower limbs are involved, in half of the cases the upper, and in a few, those of the trunk and neck. These little patients kaleidoscope as it were. The facial muscles are usually spared. The intercostals are but little affected.

Electrical reactions are normal, or show quantitative reduction. The knee and Achilles jerks are usually absent; those of the upper extremity less constantly gone. Atrophy is not definite, nor is pseudo-hypertrophy present. Mechanical irritability and fibrillary contractions are absent.



FIG. 171.—Landouzy-Dejerine myopathy.



FIG. 172.—Landouzy-Dejerine myopathy.

Contractures are not uncommon. The sphincters are intact. Sensibility is intact, also the special senses, and the children are usually bright mentally. The general condition is good, and vasomotor disturbances are absent.

The disorder has some tendency to improve, although Batten claims that not one has got well. Some of the patients learn to stand, but rarely unaided.

Intercurrent disorders, particularly respiratory, cause death in the majority.

5. *Distal Type*.—Gowers-Spiller,¹ and Spiller² first definitely separated this myopathy from its apparently related Charcot-Marie-Tooth

¹ Brit. Med. Jour., 1902.

² Jour. Nerv. and Ment. Dis., 1906.

atrophy. It varies little from this latter save in the absence of sensory disturbances.

6. *Myatonia Atrophica*.¹—A combination of muscular atrophy with slow relaxation of the muscles—possibly more closely related to Thomsen's disease than to the dystrophies (Pelz-atypical Thomsen's



FIG. 173.—Myatonia atrophica. Note dropped feet, atrophy of hand muscles, and facies. (Abrahamson.)

disease). The distribution of the atrophies is peculiar, corresponding to none of the well-known types of myopathies. There is usually weakness of the facial muscles, also masseters and temporals (50 per cent.), atrophy of the sternomastoids, the vasti, and dorsiflexors of

¹ Fűrnrrohr, Deutsche Zeitschrift f. Nervenheilkunde, 1907, 33.

the feet. Considerable variation in the distribution is known.¹ The intrinsic muscles of the hand are usually preserved. Batten and Gibb have collected all of the cases to 1909.

AMYOTROPHIC LATERAL SCLEROSIS.

History.—The position of the progressive muscular atrophies, with their many clinical variations, and the anomalous pathological findings is still in need of more detailed analysis. Charcot, in 1865, separated from this mass a special group which combined the features of a poliomyelitis and a lateral sclerosis. Atrophy with spasticity was the determining feature, and in 1872–1874 after previous studies with Joffroy and Gombault he gave it definite form. Dejerine later (1883) showed that the glosso-labio-laryngeal palsies of Duchenne were a constituent part of the disease, and the work of Kojewnikoff, Marie, Roth, Florand, Holmes, Spiller, Raymond, Cestan and others has served to establish this disorder upon a sound pathological and clinical basis.

It is a disease of the entire motor neuron, although Holmes has shown that this is not universal. Schultze has therefore suggested the term motor tabes in contrast with sensory tabes of the classical type.

Etiology.—This is a comparatively rare disease. Little is known concerning its causes. It has been thought of as a primary atrophy of the motor system—an abiatrophy, but this is only pushing the explanation back a step further. It is most prevalent in early adult life, thirty to forty, but it is also known to occur in children (Erb-Seeligmüller), and Soques, Roussy, Raymond, Probst and others have described cases occurring in the fifth decade. Women seem to have the disease somewhat oftener than men, but the differences are not striking. The statistics of the Vanderbilt Clinic for ten years show the reverse conditions—11 men and 6 women.

Arteriosclerosis, trauma, infection, intoxication, syphilis have each been shown to play some role in the causation in some patients.

Pathology.—While the disease is one in which the entire motor system is predominantly affected, this is not exclusively so, as Holmes has definitely shown. The spinal motor neurons are chiefly affected, both centrally and peripherally. The anterior horn cells are degenerated, and the motor nerve fibers as well.

The corticospinal tracts are also degenerated. This degeneration can be followed to the Betz cells of the Rolandic cortex in practically all of the cases of any duration, but in some patients the degeneration in the pyramidal tracts has not been traced beyond the pons.

Degeneration in the bulbar motor nuclei is the rule, and in the cortico-bulbar tracts, as was demonstrated by J. Dejerine in 1883. The third,

¹ Batten and Gibb, *Brain*, 1909.

fourth, and sixth nerve nuclei usually escape, though a few recorded degenerations here are known (Hoche, Pal, etc.).

Degenerations can also be traced in the corpus callosum. Following the degeneration there is some replacement sclerosis. This widespread degeneration in the motor system may be accompanied by other degenerations, however, although such degenerations seem to stand out less prominently in the clinical picture. Thus, degenerations in the lateral columns, Clarke's columns, posterior longitudinal bundles, spinocerebellar tracts, and ventrolateral ground bundles are recorded.

Symptoms.—The disorder may begin in almost any motor nerve region: bulbar, cervical, dorsal or lumbar; more than one area may be initially affected simultaneously, but the usual beginning lesions are predominantly in the muscle groups of the cervical cord. The order of involvement is predominantly radicular.



FIG. 174.—Amyotrophic lateral sclerosis showing atrophy of the arms.

The usual symptoms are muscular weakness, with wasting of the muscles, usually distally, with increased reflex excitability. The patients first note that the hands get tired, they are clumsy in dressing and undressing. Buttoning and unbuttoning, sewing, writing, and the carrying out of the more delicate finger manipulations become increasingly difficult, first from fatigue, then from stiffness and motor palsy. Atrophy of the thenar and hypothenar eminences occurs, then progresses up the muscles of the forearm, arm, and shoulder-girdle. Weakness and stiffness then make themselves evident in the lower extremities, and a similar progression takes place. Then after months or a year or so the muscles of the lips, of the tongue, and swallowing and phonation gradually become implicated. The lips become thinned,

the tongue atrophies, speech fatigue is followed by inability to speak; swallowing occurs with regurgitation, cardiac irregularities develop, and the patients die of aspiration pneumonia, weakness, or other intercurrent affections.

With the atrophies there develops a gradually increasing spasticity. The tendon reflexes are increased, there is increased jaw-jerk, or mandibular clonus; the biceps and triceps, and scapular reflexes are increased, patellar clonus, increased knee-jerks, increased Achilles, ankle clonus, Babinski and Oppenheim reflexes are manifest.

The muscles themselves show increased myotatic irritability, fibrillary contractions are common, or the atrophies may be so marked that flaccid conditions develop. The electrical reaction of degeneration occurs.

Sensory anomalies are extremely rare. Pain is probably present at some periods in many cases; it is that of fatigue or spasm, however. The bladder and rectal functions are not usually involved, and the majority of the patients show only the involvement of the motor neurones.

Mental symptoms are not prominent; increased emotionalism is usually present, and as the patients are often much depressed, the involuntary crying which is very frequent is augmented thereby.

As has been noted, the onset may be in any part of the motor system—thus the disorder may progress for some time, even to death, as a bulbar type of palsy, with increased reflexes in the motor cranial nerves; death occurring in some instances before there are any spinal symptoms. Again the distribution may be hemiplegic, and then triplegic, and finally quadriplegic and bulbar.

Again the disorder may bear the stamp of a lower extremity paraplegia, or an upper limb diplegia; again, whereas the distal muscles are usually initially involved, patients show proximal atrophies in either upper or lower extremities; and the more cases reported the greater are the possibilities encountered in the way of anomalous localizations.

But throughout, after a certain lapse of time, the combination of weakness, atrophy, increased reflex activity, without sensory changes, stamps the process as essentially a motor degeneration, and whereas one even finds the spastic phenomena antedate the atrophic changes, yet in the end the two level up, unless the course has been unusually active, and death results early from the disease or from intercurrent disorder. Here the pathological picture may seem to contradict the clinical findings, and the interpretation remains that the patient did not live long enough to show the classical syndrome.

On account of this polymorphism, certain authors have erected types which may be useful clinically, but which are rarely borne out, save for certain periods, during the development of the disease. Thus Raymond and Cestan (R. N., 1905) make (1) *an ordinary spinal type* with the classical *main en griffe* of Charcot,

increased reflexes, particularly in the upper extremities, less marked in the lower, with occasional failure of the Babinski reflex; (2) *labio-glosso-laryngeal type*—apparently more frequent in women—which begins in the muscles of the lips, tongue, pharynx, or larynx, occasionally with facial palsies, inability to close the eyes, etc., with fibrillary twitchings, lively masseter-jerk. Some of these, but not all, run a rapidly fatal course from four to nine months, and die before the lower neurons show their characteristic changes; (3) *an amyotrophic type* with pronounced atrophies which overshadow or mask the spasticities and the increased reflexes. These patients resemble chronic poliomyelitis, often for years, and constitute a most difficult group to differentiate. (4) *A spastic type*, in which the reverse condition exists, and increased reflexes and spasms, contractures, etc., antedate the atrophies. These resemble multiple sclerosis, lateral sclerosis, etc.

Finally it should be recognized that certain patients represent transition forms; they are not pure types, and the apparent clinical picture of amyotrophic lateral sclerosis is due to other than the recognized pathological foundations of this disorder.

Course and Duration.—The average time of duration of some hundred or more cases analyzed is about two years; but this by no means tells anything about any individual case. Death in four months after the onset has been the result in a score or more of cases, some have persisted ten years, and a few questionably diagnosed cases even much longer. The rule is toward steady progression; there are few leaps and regressions, as is seen in multiple sclerosis, and so far as is known the disease is a fatal one. A few stationary cases are reported. Bulbar palsies, when not among the initial symptoms, usually develop before the second year, and are often the cause of death by choking, or dyspnea. Cardiac irregularities, pneumonia, exhaustion, secondary infections, these are the usual modes of death. Suicide is unusual.

Diagnosis.—The chief disorders needing separation are multiple sclerosis and progressive spinal muscular atrophy or chronic poliomyelitis. The former rarely shows atrophies, and the eye-grounds, ocular muscle signs, and characteristic tremors should separate this; the latter disorder when it shows increased reflexes, as is occasionally the case, is separated with great difficulty. Lumbar puncture does not throw any light on the diagnosis.

Treatment.—No specific is known. Overfeeding, light massage, much rest, caffeine, strychnin are the only methods at present of service. Certain anomalous thyroid insufficiencies have been associated with progressive muscular wasting, and indicate a field for experimental therapeutics. Parathyroid myatonias, myasthenias, family periodic paralyses, etc., offer a suggestion in this line of a perverted parathyroid activity that may be seized upon for the study of the mineral metabolism, especially of calcium. Personal cases treated along lines suggested by the mineral metabolism of the body offer a glimmer of hope in a hitherto hopeless disease. Syphilitic cases need specific therapy.

FRACTURE AND DISLOCATION SYNDROMES.

Spinal cord injuries result from bullets, penetrating instruments, or from blows or falls.

Bullets and cutting instruments cause hemorrhage, with, as a rule, partial or complete severance of the cord, with septic infections of the cord and meninges. Blows and hard falls occasion fractures or dislocations, with crushing of the cord to a greater or lesser degree. Mild injuries may result merely in the bruising of the cord, or minute

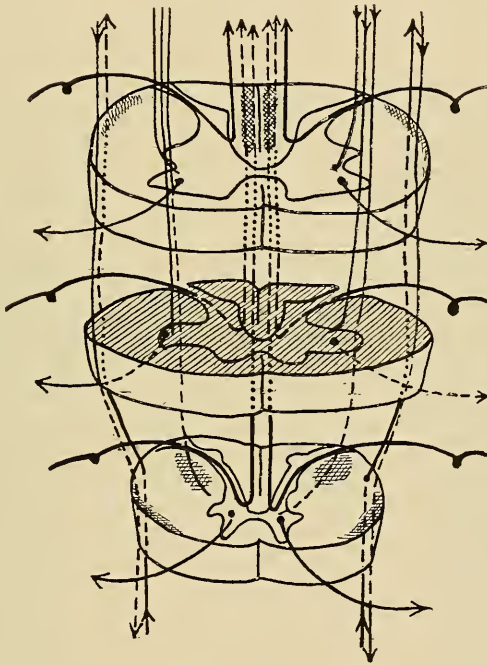
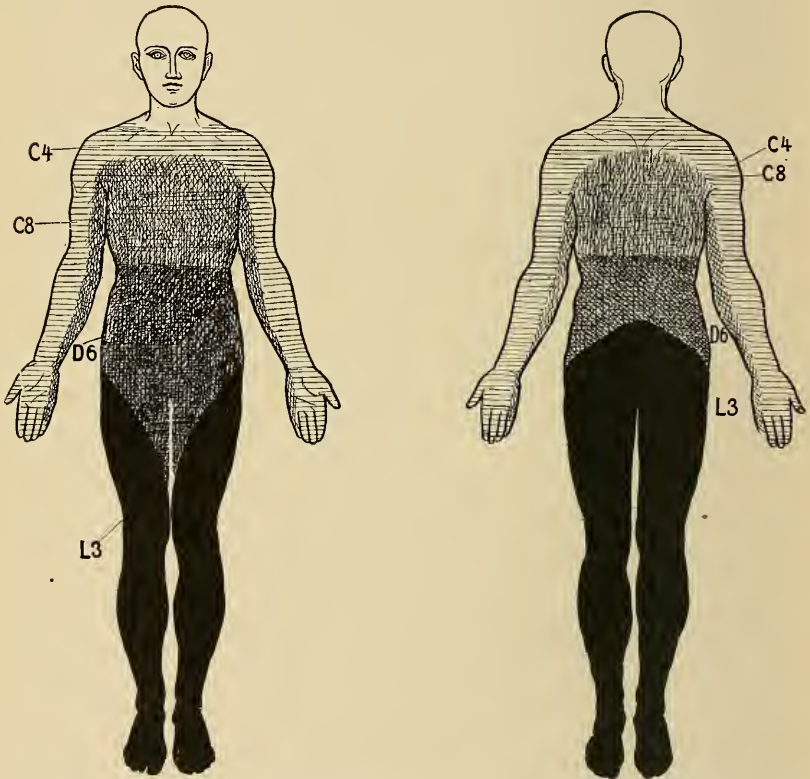


FIG. 175.—Total cross-section of spinal cord at the level of C_3 showing the results upon the long fiber tracts. Middle section indicates level of injury. Cross-hatching above and below indicate the degenerations or loss of function. (Veraguth.)

hemorrhages within the cord or of the pial or dural spaces only may be produced, sometimes even from excessive exertion, long marching, severe athletic exercises, sudden spinal torsions, etc. Dislocation of a vertebra practically always causes a crushing of the cord; the so-called dislocations without spinal cord injury are more apt to be wrenches of the vertebræ or very limited dislocations. Fractures may result with but few spinal symptoms; crushing of the cord causes more or less disintegration, usually accompanied by severe hemorrhages within or without the cord substances (hematomyelia, dural hemorrhage). Hematomyelia usually extend up and down the cord from

the point of destruction, sometimes involving several segments of the cord.

Symptoms.—Fractures or dislocations of the spinal vertebræ usually cause local deformity, much pain and muscular rigidity, particularly on motion. X-ray examination reveals the nature and extent of the bony lesion. The motor and sensory phenomena at the level and below the site of the lesion determine the location, extent and character of



FIGS. 176 and 177.—Showing superficial sensibility disturbances in complete transverse lesions of the cord at the levels of *C*₄, *C*₈, *D*₆, and *L*₃, respectively. (Veraguth.)

the injury to the spinal cord. The symptoms usually develop immediately following the injury, become slightly progressive, if hemorrhage only, and then slowly recede until after a variable length of time, usually from two or three months the residual symptoms indicate the permanent impairment of function.

The chief syndromes, which develop from injury to the spinal cord at different levels, are here described by means of charts. A careful sensory examination is indispensable and should follow the directions already laid down.

It is important to recall that the spinal cord segments and the vertebral segments, while nearly corresponding during infancy, do not in

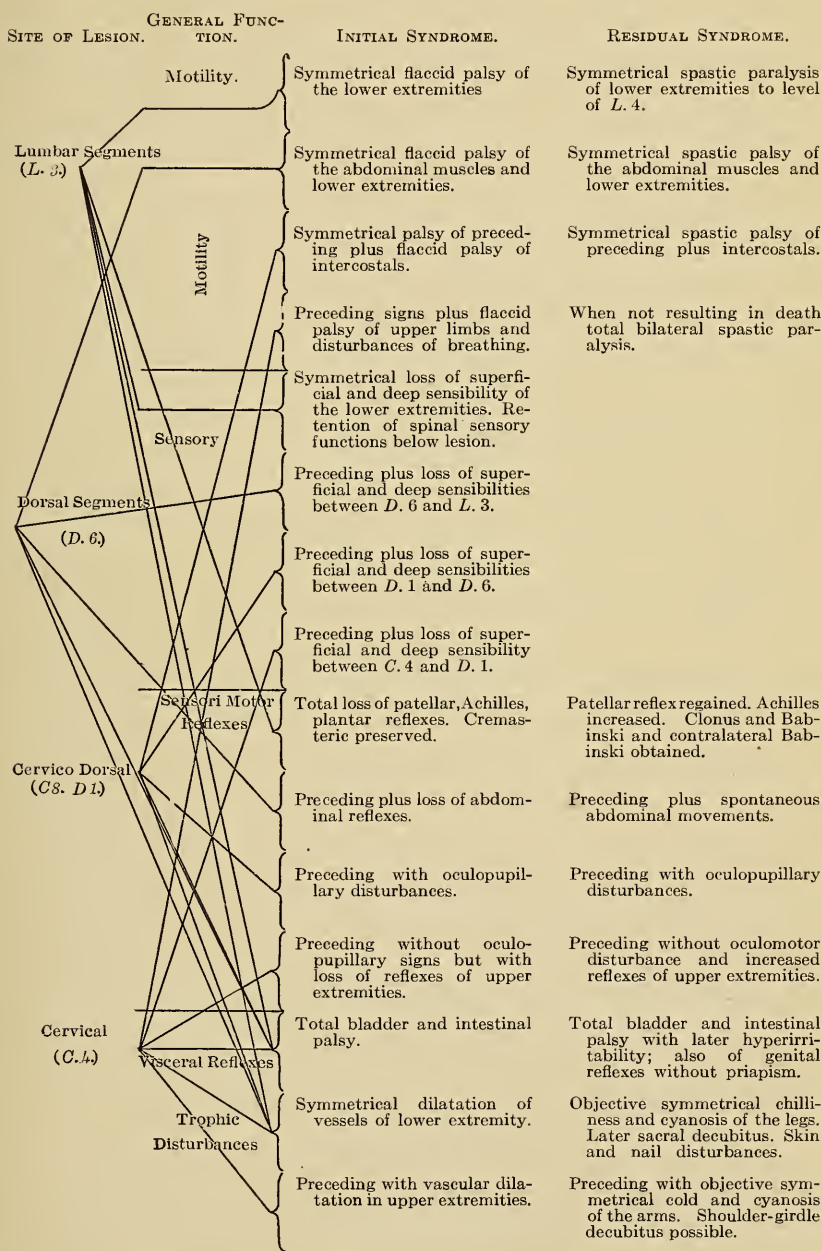


FIG. 178.—Localization of symptoms at different levels of the cord. (Veraguth.)

the adult. It is rare that absolutely symmetrical involvements result from spinal injury. Usually one side is involved more than the other.

A total destruction of the spinal cord will result in initial and in residual symptoms. This may result from accident, bullet wound, diving, caisson disease, myelitis, tumor—Pott's disease chiefly. The course, progression and mode of treatment will depend entirely upon the causative lesion. The chief symptoms of acute cross-lesion are expressed in the table on page 343 and diagrams according to the site of the lesion.

Lesions of the upper cervical segments usually causes instant death from respiratory paralysis. Injury lower down is very frequent, occurring in workmen from objects falling upon the bent neck or in reckless diving. The symptoms are charted. Many of these patients live for some time, even years, dying usually from bladder and kidney complications. Dorsal lesions are comparatively rare, and often show the Brown-Séguard syndrome, which latter may, however, develop from lesions at all levels above the conus.

Lower dorsal and upper lumbar lesions are the most frequent of all. Their symptoms are indicated in the chart.

Injury to the lower lumbar vertebræ causes cauda equina lesions, as the cord proper terminates at about the first lumbar.

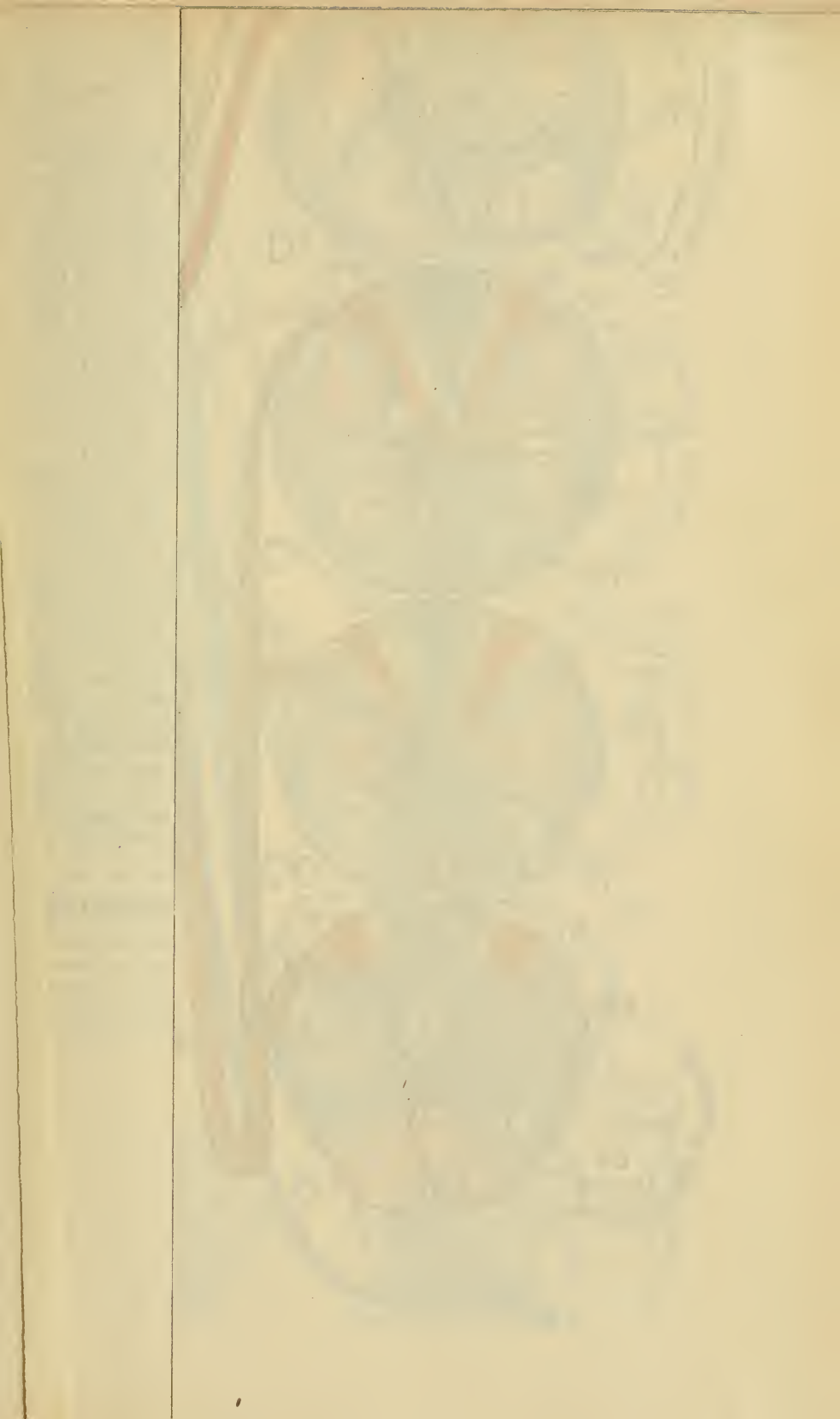
Intramedullary lesions—*hematomyelia*—have a symptomatology all their own.

Careful sensory testing shows that in an intramedullary lesion there is a complete separation of the impulses underlying the appreciation of posture, the discrimination of two points, and their correlated faculties from those of other sensory groups.

All painful and thermal impulses coming from the periphery, undergo regrouping after entering the spinal cord, and, whether they arise in the skin or in deeper structures, become arranged according to functional similarity. Then, after a longer or shorter course, they pass away to the opposite side of the spinal cord. (See Plate XI.)

This process of filtration leaves all the impulses associated with postural and spacial recognition to continue their course unaltered in the posterior columns; they are the survivors of peripheral groups broken up by the passing away of certain components into secondary afferent systems. At any point in the spinal cord, these columns transmit not only impulses from the periphery which are on their way, after a shorter or longer passage, to regrouping and transformation, but at the same time they form the path for impulses, arising both in the cutaneous and deep afferent systems, which undergo no regrouping until they reach the nuclei of the medulla oblongata.

Thus, a lesion confined to one-half of the spinal cord, even at its highest segment, may interfere with the passage of sensory impulses, some of which are travelling in secondary paths, whilst others are still within the primary level of the nervous system. All impulses concerned with painful and thermal sensations from distant parts, disturbed by



such a lesion, will be travelling in secondary paths and will have come from the opposite half of the body; for, after regrouping, they have passed across the spinal cord. But those impulses underlying the appreciation of posture, the compass test, size, shape, form, weight, consistence, vibration, will be affected on the same half of the body as the lesion. They still remain in paths of the primary level and have undergone no regrouping. (See Plates X and XI.)

In such a case the parts on the side opposed to the lesion may be insensitive to pain, heat and cold; but all the postural and spacial aspects of sensation will be perfectly maintained. Yet, all power of recognizing position, of estimating size, shape, form and weight, or of discriminating the two compass points, will be lost in the limbs which lie on the side of the lesion, although tactile sensibility and localization of the spot stimulated may be perfectly preserved.

This remarkable arrangement enables one to analyze, as Head has pointed out, the nature of the peripheral impulses upon which depend the power of postural and spacial recognition. Obviously, even at the periphery, they must be independent of touch and pressure. The power to distinguish two points applied simultaneously and to recognize such size and shape, requires as a preliminary the existence of sensations of touch; but the patient may be deprived of all such powers of spacial recognition without any discoverable loss of tactile sensibility. In the same way our power to appreciate the position of a limb, or to estimate the weight of an object, is based upon impulses which, even at the periphery, exist apart from those of touch and pressure, called into simultaneous being by the same external stimulus.

This long delay of the postural and spacial elements in reaching secondary paths enables them to give off afferent impulses into the spinal and cerebellar coördinating mechanisms, which lie in the same half of the spinal cord. The impulses which pass away in this direction are never destined to enter consciousness directly. They influence coördination, unconscious posture and muscular tone, and, although arising from the same afferent end-organs, they never become the basis of a sensation.

Finally, the last survivors of these impulses from the periphery become regrouped in the nuclei of the posterior columns and cross to the opposite half of the medulla oblongata in paths of the secondary level. So they pass to the optic thalamus and thence to the cortex, to underlie those sensations upon which are based the recognition of posture and spacial discrimination.

Diagnosis.—Accurate testing of the motor and sensory functions leaves no doubt as to the presence of a spinal cord injury of a major grade. The results of minute lesions may escape recognition. Total severance of the cord is unusual, most, even severe injuries, leave some pathways undivided, which, after the immediate effects of the injury have subsided, give some sensory response. Total, complete severance causes absolute anesthesia to all forms of stimuli, flaccid paraplegia,

with loss of all reflexes and all visceral reactions below the site of the lesion.

Prognosis.—This is bad in practically all spinal cord injuries save small hematomyelia or limited cauda equina lesions. High lesions are almost immediately fatal. Lower lying ones—fourth cervical and down—vary in their immediate and remote results, according to the site of the lesion and its extent.

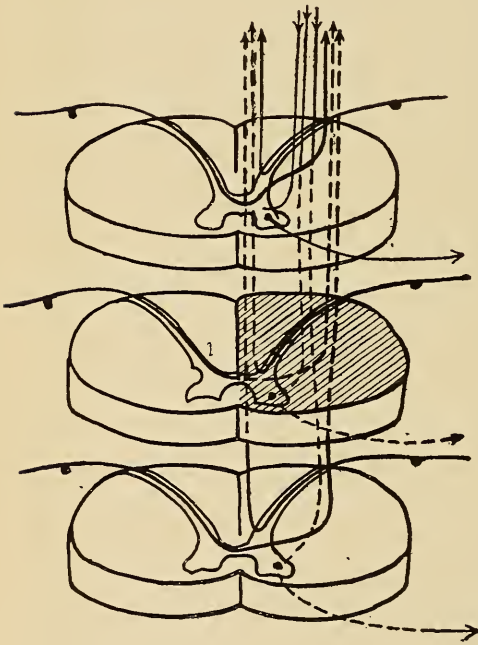


FIG. 179.—Lesion of the cord producing a Brown-Séquard syndrome. (Veraguth.)

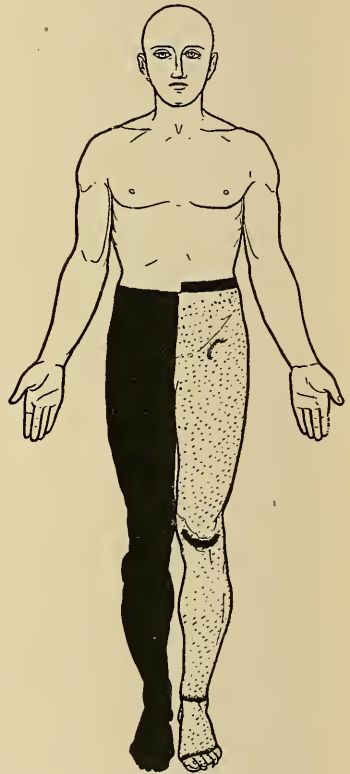


FIG. 180.—Brown-Séquard syndrome. Black represents superficial anesthesia in joint anesthesia; --- passing hyperesthesia and paralysis. (Veraguth.)

The question of regeneration of spinal neurons has not as yet been entirely settled experimentally, but the preponderance of practical evidence is against it. Pathways once destroyed remain so. Just at what period it may be said that the residual symptoms will be permanent is largely a matter of the extent of the lesion. As a rule the findings at three months are apt to be those of permanence, still occasionally marked improvement, particularly in bladder and rectal functions, may take place after a year or more of total loss. Many

spinal lesions, especially those dependent chiefly on hemorrhage, will show widespread symptoms early, and later these will be reduced to a minimum. The chief bugbear as to life is the bladder. Great care should be taken of it in the early stages, and every available measure taken to keep it sterile.

SITE OF LESION.	GENERAL FUNCTION.	INITIAL SYNDROME.	RESIDUAL SYNDROME.
In lumbo-sacral segments	Motility	On the side of the lesion. Flaccid palsy of muscles whose neurons lie caudad to lesion.	On side of lesion, spastic palsy of muscles whose neurons lie caudad to lesion. Normal on side opposite to lesion.
		Normal on opposite side.	
Dorsal segments	Sensibility	On the side of the lesion. Disturbance of deep sensibility, especially joint hypesthesia, caudad to affected metameres. Small zone of superficial anesthesia above the palsied motor zone. Hypesthesia for touch caudad to affected metameres.	Hyperesthesia disappears shortly.
		On the crossed side, and partly on the side of lesion, because of limitations in crossing space of fibers below lesion, superficial hypesthesia, especially thermohypesthesia and hypalgesia.	
Cervical segments	Reflexes	As above.	On the side of the lesion. Increase of tendon and skin reflexes. Babinski. Contralateral Babinski.
		On the side of the lesion. Loss of tendon and skin reflexes of the lower extremity. Babinski phenomenon.	
Cervical segments	Sympathetic signs	On opposite side. Loss of skin reflexes of the lower extremity. Preceding and loss of abdominal reflex.	Preceding and increased abdominal reflexes on side of lesion.
		Preceding and loss of tendon reflexes of the upper extremity of the side of the lesion.	
Cervical segments	Sympathetic signs	On the side of the lesion the skin of the caudad metameres red and hot.	On side of lesion the skin of the caudad metameres either normal or cyanotic or objectively cool.
		On the crossed side normal. Preceding plus oculopupillary signs on affected side.	

FIG. 181.—Brown-Séquad lesions.

Operations, after spinal injury, are often futile, yet with care rarely do any harm and, not infrequently, if the cord itself is only being pressed upon by hemorrhage, may be distinctly advantageous. Evident surgical indications (fracture, dislocation) should be met. The patient is rarely benefited but is entitled to the opportunity.

Therapy.—Surgical treatment is indicated in most spinal injuries even though the prognosis is not at all reassuring. The persistence of some form of sensibility (all types should be tested) is an indication that the cord is not completely severed. Operation during shock is not advisable, yet too long a delay is unwarranted, even if all sensibility seems abolished below the lesion. The functional loss practically always exceeds the anatomical defect. X-ray examination should be made immediately for diagnosis, and if there is evidence of compression, operation is advisable as early as is compatible with the patient's condition. In the absence of evidence of compression, earlier surgeons advised against operation as dangerous and futile. Since in the hands of competent surgeons most of the dangers of spinal cord injury have disappeared, there is a growing tendency to operate more freely and at times, fortunately, even though more often without any gain.

The general treatment of the patient is of great importance. It should be directed toward giving a maximum of relief from distress; avoidance of bladder infection, preventing bed-sores, and careful bowel attention. Antispasmodics and analgesics may have to be used for a long time, but morphin and its derivatives are to be avoided if possible, save in those agonizing cases where it gives the only relief from torture. Electricity is largely a placebo.

Half-sided lesions produce the typical Brown-Séquard syndromes, which vary according to the segments, as seen in the accompanying scheme (Fig. 181.)

COMPRESSION OF THE CORD.

Compression of the cord, as a slow chronic process results from (1) bony hypertrophies, (2) tuberculosis, (3) tumors, (4) syphilis, (5) aneurisms, (6) meningeal disease.

Bony Hypertrophies (Osteitis, Osteitis Deformans, Osteo-arthritis, Spondylosis Rhizomelique).—Under these various names one finds patients who present signs of more or less spinal cord compression, either with or without stiff backs or deformities.

They show gradually increasing weakness, going on to paresis, or complete paraplegia with spasticity and increased reflexes. Frequently there is severe pain, and when the bony disease impinges upon the intervertebral foramina neuritic pains and symptoms of peripheral nerve palsy develop. (See Plexus Palsies.) X-ray examinations reveal the nature of the bony changes and the location of the pressure.

Tuberculosis (Caries).—This is a most widespread cause of spinal cord compression. The tuberculous focus usually begins within the body of the vertebræ; breaking down and destruction of the vertebræ take place, with displacement and projection of the vertebræ either forward, backward or laterally, causing the various deformities of Pott's disease. The tuberculous process usually spreads to the spinal meninges (tuberculous pachymeningitis). Thus if the compression

arises it may be from both processes. Even more rarely, tuberculous myelitis occurs from direct extension, usually through the lymphatic channels.

Symptoms.—The chief symptoms are pain in the back, stiffness of muscles, rigidity and tenderness on motion in the early stages. With the development of a kyphosis, lordosis or scoliosis, deformities appear and symptoms of cord compression and plexus pressure begin. The plexus symptoms vary with the segments involved, as already discussed. (See Peripheral Plexus Neuritides.) The cord compression gives rise to increasing bilateral spasticity below the site of the lesion, slowly developing sensory impairment, which may develop into signs of a complete cutting off of the entire cord pathways below the site of the compression. (See table, p. 343.)

Lymphocytosis in the cerebrospinal fluid is a frequent sign and the cell-picture is striking. X-ray examination will reveal early the signs of tuberculous bony disease.

The symptoms usually show very gradually, as the tuberculous disease is usually a slow one. Occasionally they show a fulminating course, especially in childhood, or even in young adults.

The outcome depends upon the success obtained in combating the tuberculosis by orthopedic, surgical and general health measures. Early diagnosis is essential, and the spinal fluid examinations and x-ray findings will aid in such an early sizing up of the situation, which will lead to the proper procedures. Surgical therapy, early, Albee's bone splint or related procedure may help to do away with the cumbersome braces formerly so extensively used in treating Pott's disease.

Spinal Cord Tumors.—The consideration of spinal cord tumors makes a large chapter in contemporary neurology, which can only be sketched here. They are comparatively rare, yet frequent enough to put one on one's guard in any spinal cord condition showing compression phenomena, *i. e.*, weakness, spasticity, and increased reflexes, *i. e.*, pressure symptoms of the spinal motor neuron.

Spinal cord tumors are as variable as those found within the cranial cavity. They are found extradurally, durally, intradurally and intramedullary. They are small and large, and located at any, sometimes at all, levels (multiple sarcomata) of the cord. The tumors of the spinal cord are identical with those of the brain (*q. v.*). They induce different symptoms solely because their localization varies. For the most part they lie laterally and posteriorly, and are thus more accessible to surgical removal. They occur at all ages, and in both sexes, and show a frequency comparable to those located in the cranial cavity. The causes for intracranial tumors apply to those of the vertebral cavity.

Symptoms.—The chief symptoms of spinal cord tumor are pains, sensory signs on the skin of the body, and evidences of motor paresis.

Sharply localized pains at the site of the tumor are frequent, but may

be absent; all pains may be absent, but this is infrequent.¹ The more usual pains are those of the root area or areas involved by the tumor. The pains are usually unilateral, but widespreading tumors cause bilateral pain later. Any so-called neuralgia, sciatica, etc., may be the initial pressure pain of a spinal cord tumor (see discussion on neuralgias). Under the popular misnomer, "rheumatism," many spinal cord tumors and neuralgic pains are hidden until it is too late to obtain relief.

The x-ray picture is usually negative. The cell count of the cerebrospinal fluid often gives much information, and most tumors show a high globulin content in the cerebrospinal fluid. The Wassermann technique will rule out gummata as a rule. In some tumors there is an absence of fluid, or fluid under a very low pressure, below the site of the tumor, with normal fluid above.

The motor compression signs are variable, paresis advancing to paralysis (paraplegic), hypertonicity, spasticity, increased reflexes, Babinski, clonus, etc. At the level of the tumor there may be destructive lesions with signs of peripheral motor neuron disease, *i. e.*, atrophy, loss of reflexes in the diseased area, reaction of degeneration, trophic changes (bed-sores, etc.).

The sensory phenomena will vary also, one side often showing more markedly than the other. There is hyperesthesia at about the level of the lesion which is changed to various grades of anesthesia below the lesion. Slight loss of epicritic touch is apt to be an early sign.

Greater sensory loss advances with increasing compression. Bladder and rectal disturbances are frequent.



FIG. 182.—Osteophytes of spinal cord.
(Larkin.)

Small tumors (cysts, etc.), intramedullary, sometimes extramedullary, cause typical dissociation symptoms with retention of epicritic tactile sensibility and loss of protopathic pain and thermal sensibility.

The symptoms for localization of the tumor have already been discussed.

Diagnosis.—X-ray examination, spinal fluid examination, and a complete neurological status should enable one to arrive at a satisfactory diagnosis. The chief diagnostic problem is syphilitic meningo-myelitis. Bony disease is usually excluded by the *x*-rays. Multiple sclerosis is not infrequently ushered in with sensory signs, also syringomyelia. They present real difficulties in diagnosis. The application of proper neurological and psychoanalytic procedures will exclude the conversion symptoms of hysteria.

Intramedullary or extramedullary diagnosis is practically unimportant since spinal cord surgery has become so satisfactory. Theoretically employed the problem is of interest, practically less so. For localization symptoms consult Figs. 177, 178, 179, and Plates X and XI.

Treatment.—Apart from syphilis the treatment is surgical and it should be employed early and for practically any or all tumors; not necessarily with the promise of cure but as a routine exploratory procedure. In skilled hands the danger is slight, and apart from special contra-indications, the risk is very frequently worth while, as very unexpected things turn up within the spinal cavity.

The diagnosis for localization is the most difficult problem. Most tumors are found a couple of inches above the site usually sought for as determined by the level of the anesthesia. Surgical progress relative to spinal cord tumors is rapidly advancing and cannot be discussed here. The general prognosis has improved both with reference to the finding, as well as the successful removal of the tumors. Recurrences do occur, and many patients are not benefited. Little is to be expected in those long-standing tumor cases with marked signs of spinal cord-compression degeneration. Yet every patient is entitled to the benefit of the doubt. Surgical failure is better than a let-alone policy covered up by morphinism. This latter even is not necessary, since careful division of the affected sensory roots in inoperable cases may give relief from pain, even if life cannot be saved.

Most patients die of the myelitis bed-sore and bladder complications after one to two years in inoperable cases.

LATERAL SCLEROSIS GROUP.

Historical.—These disorders which clinically may resemble a number of spinal cord conditions, have been isolated from the mass of rachialgias of the eighteenth century, and from the paraplegias and myelitides of the nineteenth more particularly by Seguin, Türck and Erb. The

latter, in 1875,¹ posited a hypothetical degeneration of the pyramidal tracts in their spinal course, terming the disease primary spastic paralysis. Charcot accepted Erb's teachings and spoke of the disturbance as a spasmodic tabes dorsalis. The disorder, as understood by Erb, has been found to be much less frequent than was at first supposed, as the lateral sclerosis picture was found to be but one stage of a number of other cerebral and spinal affections, notably, as in multiple sclerosis, syringomyelia, hydrocephalus, tumor of the cord, anemia of the cord, diffuse myelitis, senile changes and amyotrophic lateral sclerosis, etc. Of recent summaries, those of Erb² and Spiller³ are available.

Pathology.—Ideally this consists of a simple degeneration of the pyramidal tracts which rarely ascends to the cortex. A replacement glia infiltration is present. But few autopsies are recorded with the ideal lesion.

Symptoms.—These are exclusively motor, and usually of the lower extremities alone, although the disorder may show itself in the arms. The ordinary picture is that of a spastic paraplegia, *i. e.*, motor weakness, increased reflexes, clonus, Babinski, spasticity, with no sensory or visceral signs.

Gradually increasing weakness of one or both legs is first observed after a long walk, or dancing, or any prolonged muscular exertion. A certain stiffness develops, but very slowly, often only after several years. The tendon reflexes are exaggerated at a very early date.

Slight motor weakness supervenes, and some stiffness to passive motion, and then the patients note that their gait is more constrained; they do not bend their knees, but shuffle somewhat and stumble easily. The limbs become stiffer and stiffer, the extensor muscles being more involved, this brings about extreme rigidity at the joints.

In this stage, which may be arrived at in a few years, or more often after many, the patient walks with a stiff, stubbing tread, perhaps the knees overlap (scissors walk) or knock each other; the toes are not lifted, but rather shoved along, thus wearing the shoes markedly at the toe.

The cutaneous and tendon reflexes are exaggerated. There is markedly increased knee-jerk (see Franz's distinctions), patellar clonus, ankle-clonus, Babinski, and paradoxical and Bechterew-Mendel signs, Strümpell's tibialis phenomenon is apt to be marked. Spasms frequency occur in the affected limbs. In the latest stages flexor contractures usually result.

Sensory symptoms, save the muscular pains of spasm, are absent. The bladder and rectum are not implicated. In rare instances the upper extremities are involved, and even the medullary and bulbar motor tracts.

¹ Virchows Arch., vol. lxx.

² Deut. Zeits. f. Nhk., 1903, No. 23.

³ Osler's Modern Medicine, vol. vii; Bono, Rev. d. Méd., March, 1908.

Forms and Varieties.—1. *Hereditary Familial Types* (Strümpell,¹ Newmark²).—Here the disorder shows itself either in childhood, or late in life, and many generations may be affected.

The symptoms are those of muscular hypertonia, spasticity, exaggerated tendon reflexes, and later palsies and contractures. The skin and anal reflexes are less apt to be involved in the familial types. In some cases (Newmark, Lorrain) there are more widespread disorders, optic atrophy, feeble-mindedness, muscular atrophies, etc. Deep sensibility is occasionally involved with slight Romberg.

The changes in these patients tend to become more or less stationary after a certain length of time,³ but the disease may extend to the upper part of the cerebrospinal axis.

2. *Infantile Types*.—Infantile types beginning from three to six years, due to developmental defect in the pyramidal system. Here the lower extremities are most involved. The advent of the Wassermann technique is relegating the greater number of these to some intra-uterine or early syphilitic process.

3. *Unilateral Ascending and Descending Types*.⁴—The validity of this type is not yet established. It shows itself as a gradually progressive hemiplegia, supposedly due to primary degeneration of the pyramidal tracts.

4. *Mixed Types*.—These previously described as due to syphilis, to lead, to lathyrus poisoning, anemia, etc., are more properly more or less irregular forms of myelitis, and are treated under that title.

5. *Congenital Type* (Little's Disease).—This will be considered under diseases of the brain. The pyramidal tract disease is secondary to other lesions.

Diagnosis.—Pure types of lateral sclerosis are rare. The underlying condition often develops after careful observation, sometimes extended over several years. The chief disorders to bear in mind are multiple sclerosis, compression from tumor, bone, amyotrophic lateral sclerosis, myelitis, old encephalitis, brain tumor, with or without hydrocephalus.

Hysteria can be readily excluded by the careful scrutiny of the reflexes (Babinski, Grasset, Hoover signs, etc.).

Treatment.—Foerster's operation, or posterior root section, may help the spasticities in some patients. At the present time, nothing is known that will stop the advance of the disorder. A Wassermann examination should be made in all cases, as in some an unknown syphilitic element has been revealed.

COMBINED SCLEROSES. COMBINED DEGENERATIONS.

One speaks didactically of a combined sclerosis as a system disease, in which the lateral and posterior columns are involved; clinically

¹ Arch. f. Psych., x, xvii, 1880.

² Deut. Zeits. f. N., 27, 1904.

³ Bono, Rev. d. Méd., March, 1908, for summary of all cases.

⁴ Mills-Spiller, Bull. Univ. Penn., 1906.

a combination of tabes and lateral sclerosis. It is a doubtful question whether any one disease exists which may be designated as a combined sclerosis, but one does find a great number of conditions in which both lateral and posterior columns are degenerated to a greater or less extent, and as a result one gets varying symptom-pictures as the one or the other is more or less implicated.

Thus in true tabes, degeneration of the lateral columns not infrequently occurs; in general paresis there are typical combined scleroses; in many cases of syphilitic meningomyelitis, degenerations of posterior and lateral columns occur, likewise in severe anemias, in poisoning from lead, ergot, pellagra, lathyrus; in the senile cord similar changes are found. In fact a great variety of degenerations are found in the cord involving both sets of columns to a greater or less extent. Sand proposes to divide this group into the pseudosystem diseases and the polysystem diseases.¹

Whether, as Westphal first maintained (1867), there is a true system disease of these columns is not yet a settled question. Among this large group, however, certain clinical types stand out, which permit of more or less clear-cut description. In practice they are separated one from another only with great difficulty. By Leyden and his school, most of these disorders were classed with the chronic myelitides. Henneberg² uses the term funicular myelitis for one group of non-system combined degenerations. These latter are usually due to blood changes, and are discussed in this volume with the myelitides rather than with the combined scleroses.

The more fixed of the combined scleroses types which are here considered are: (1) combined sclerosis (ataxic paraplegia (Westphal)), and spastic paraplegia (Strümpell) forms; (2) general paresis forms (see under General Paresis); (3) toxic forms; (4) senile forms. There are many intermediary forms.

1. **Combined Sclerosis** (Ataxic Paraplegia (Westphal) Types).—Here the characteristic features are those of a spinal tabes, with some signs of spasticity, *i. e.*, Babinski's reflex, and a crawling rather than a typical tabetic gait. Ataxia, pains, bladder disturbances, sometimes pupillary stiffness, etc., indicate that the meningoneuritic element is predominant, the pyramidal tract involvement of less marked extent. Certain patients start with typical ataxic signs, then gradually develop spasticities and the spastic element finally becomes predominant.

Spastic Ataxic Type.—Here the spastic element enters predominantly into the picture. Weakness precedes, the gait then becomes stiff, and the toes drag; there are increased skin and tendon reflexes, just as in atypical lateral sclerosis; then pains develop; radicular sensory disturbances commence; the knee-jerks become diminished; hypotonia gradually takes the place of hypertonia; one leg may be hypotonic, the other hypertonic; visceral disorders are added, and ataxia and

¹ Bull. de l'Acad. Roy. de Méd. de Belg., 1903.

² Arch. f. P., 40, 1905.

Romberg are present. Possibly there is added optic atrophy, or pupillary stiffness. Nystagmus is not infrequent. The Babinski reflex is apt to persist.

The course in both forms, of which there are all possible gradations, is chronic. Complete disuse of the lower limbs follows. The patients are bed-ridden with contracted, drawn-up limbs. Twitchings, spasms, and bed-sores supervene.

2. **Combined Sclerosis in Paresis.**—(See Paresis.)

3. **Toxic Forms.**—Here one may group a motley array of combined scleroses. These are due to poisoning from the lepra bacillus, to diabetes, to lathyrus, to pellagra, to ergot, to alcohol, carcinosis, malaria, tuberculosis, Addison's disease, etc.¹

4. **Senile Forms.**—Slowly progressing weakness of the limbs, with numbness, palsies, and stiffness is frequent in many old people. There develops a shuffling gait, and gradually a more or less complete paraplegia with increased reflexes, Babinski and clonus. The upper extremities share somewhat in the feebleness, tremor and spasticity. These senile myelopathies are very diverse in their nature. L'Hermitte² finds (1) perivascular sclerosis, (2) marginal scleroses, and (3) combined scleroses of the pyramidal and posterior tracts. Crouzon has found in this last group, paretospasmodic, ataxospasmodic, and ataxo-cerebello-spasmodic types. Thus it may be seen that the senile cord offers a great variety of pathological changes with a large series of closely related clinical pictures.

SYRINGOMYELIA.

Historical.—As long ago as 1564, Etienne described cavity formation in the spinal cord; it was further recorded a hundred years later by Bonet (1688) in his celebrated *Sepulchretum*. Morgagni (1740) and Portal (1800) saw and described cases, and Ollivier of Angiers (1834) first gave the name syringomyelia to what had been taught by Etienne to be a persisting central canal, but which Ollivier claimed was a pathological formation.

The studies of Gull, of Landau, and Nonat, with those of Stilling and Waldeyer, first gave the impetus to the correlation of the anatomical lesion, and the clinical symptoms. Duchenne (in 1853) then called attention to certain muscular atrophies with sensory anomalies, which differed markedly from the chronic muscular atrophies he was describing; but it was not until as late as 1882 that both Kahler and Schultze brought out the factors that permitted a diagnosis during life.

This marks the period of active neurological interest in the disease with a sudden growth in its symptomatology. Wichmann (1887) and Anna Bäumlér (1888) published monographs, the latter collecting 112 cases.

¹ For literature to 1903, see Sand, l. c.

² Thèse de Paris, 1907.

In the six years following, up to the appearance of the first edition of Schlesinger's masterly monograph, contributions appeared from everywhere, and the related subjects of leprosy and Morvan's disease took on an active interest. Schlesinger's (1894) monograph fixed the lines of research, which are shown in his thorough and monumental second edition of 1902, a monograph of some 600 pages, since which time few striking additions have been made.

Etiology.—Men are more frequently affected than women, in about the proportion of 2 to 1. About 70 per cent. of the cases occur before the age of forty, the greatest age of incidence being between the ages of twenty and thirty. It is not known that occupation has any bearing on the etiology. Toxic factors are not proved; infections may play a role in causing cord hemorrhages, emboli or thrombi, with secondary cavity formation—Schlesinger believes that in such developments the cord was not previously normal. Syphilis may be such an infecting agent, also the typhoid bacillus. Syringomyelia in mother and son, and in different members of the same family has been observed, but Schlesinger did not note any hereditary history in any of his numerous cases.

Traumatism undoubtedly plays a role. Hematomyelia develops after spinal traumata, and then may give rise to secondary cavity formation. Kienboch, however, has followed many of these cases of trauma and rarely found any consequent syringomyelia. Local traumata with ascending neuritis have possibly given rise to later developing syringomyelia. Leprosy may also condition a syringomyelia.

The essential feature is a congenital predisposition. The various factors just enumerated are purely contributory, either singly or one or more together.

Symptoms.—Like multiple sclerosis, syringomyelia is characterized by its extremely rich and variable symptomatology; like this disorder also, almost anything is to be expected, since with the gradual increase in extension of a cavity formation in the cord, new areas become involved, and older boundaries enlarged. It is therefore a disorder which is apt to show a gradually developing symptomatology, and persisting as it does many years, a rich array of pictures may be presented. Again it is very irregular in its manifestations, although certain features form the background of the disorder. These chief features are: (1) a peculiar dissociation of the sensory impulses; (2) muscular atrophy and other trophic disturbances, affecting chiefly the skin and joints; (3) motor disturbances either irritative or paretic in character. Not all of the cases show these symptoms, but inasmuch as the cavity formation is apt to occupy certain portions of the cord more often than others, this grouping of symptoms occurs more often than other groupings.

1. *Sensory Dissociation* (Kahler, Schultze).—This consists in a loss of ability to recognize sensations of heat and cold and sensations of pain but without any loss of touch, especially of epicritic touch.

This dissociation varies considerably. It may be absent. In one patient or in one area the thermo-anesthesia is very profound, the loss of pain sense less so—again, in others, the reverse is true. In the

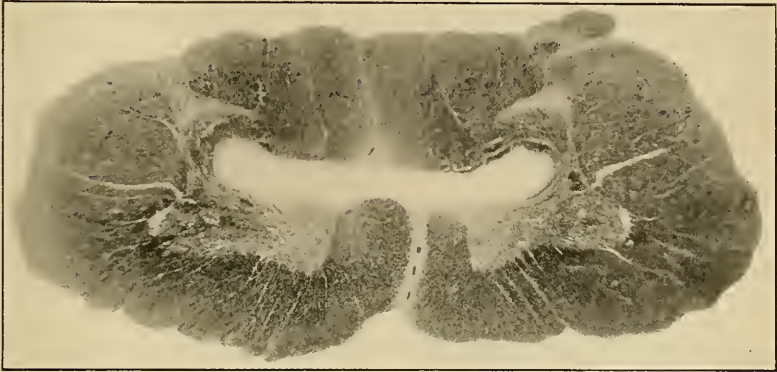


FIG. 183.—Syringomyelic cavity in the cord (cervical region).

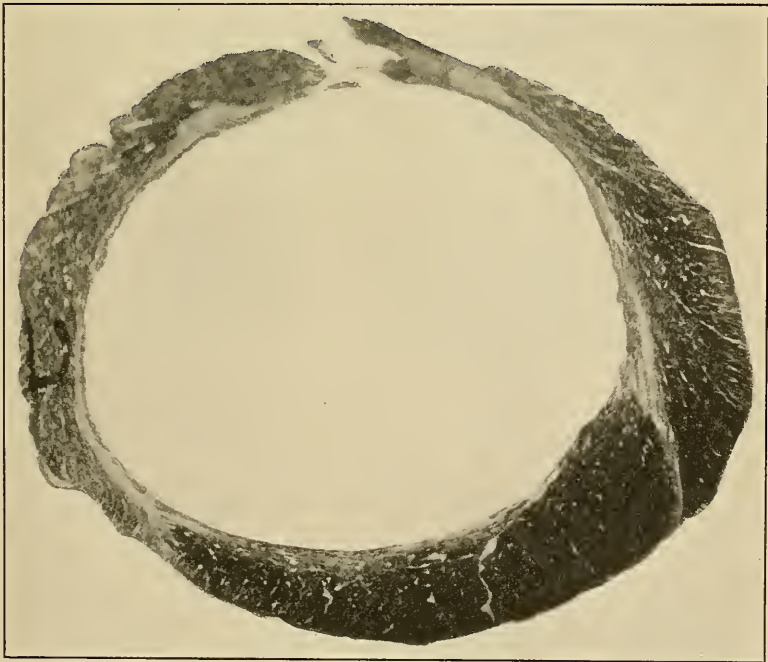


FIG. 184.—Another level of the cord, showing gliomatous tumor cavity.

majority of patients the loss is only a partial one, not an absolute one. Most cases if seen early enough will show only slight reductions—epicritic heat and cold are lost before protopathic heat and cold—

sometimes the reverse is true, or thermo-anesthesia and analgesia may be present on one side only. Again heat or cold alone may be affected. Epicritic touch may be involved, but it is not usual.

These sensory anomalies may be distributed over very small areas, rarely bulbous (mucous surfaces included), most often cervical; or they may extend almost throughout the entire spinal axis from the trigeminus to the cauda (loss of testicle pains, and analgesia of the bladder, etc.).

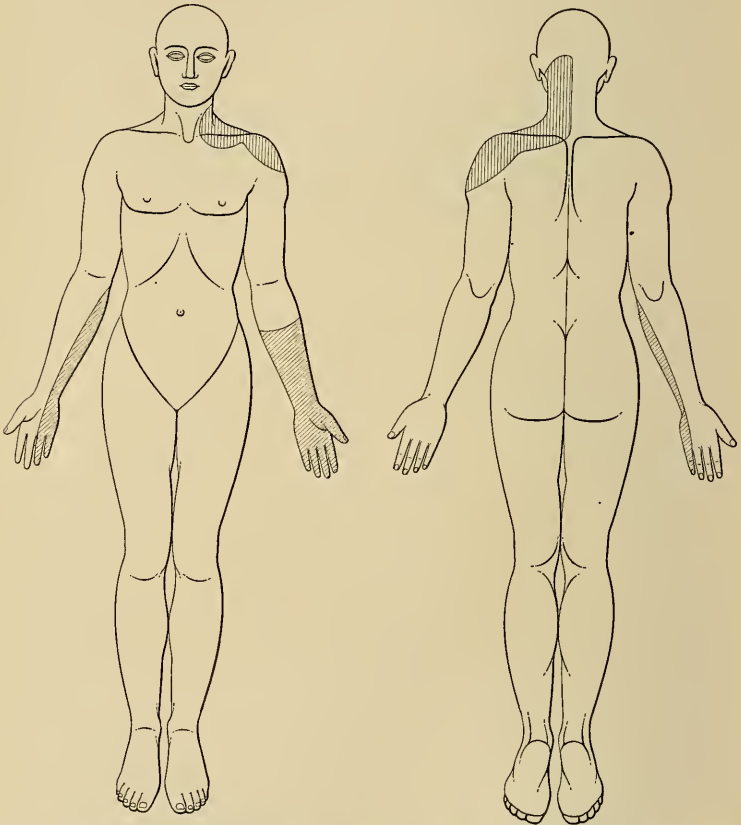


FIG. 185.—Syringomyelia, showing the dissociated loss of sensibility. Areas of loss of sensibility to pain.

The areas of diminished or lost sensibility to heat and pain are usually bilaterally asymmetrical, not infrequently are they unilateral for a time, then spread to the opposite side. They show the most unique distributions. The distribution may be exquisitely radicular; again, it is predominantly segmental or metameric. Schlesinger's most recent researches speak for the segmental type of distribution for the majority of the cases (Fig. 185).

In the beginning, one frequently encounters the glove and stocking types of sensory changes. Later a whole limb will be involved; one may get a girdle sensation. A hemianalgesia, or hemithermo-anesthesia may be present. Allochiria is usually absent.

The sensory changes usually take years to develop. This sensory dissociation is frequently preceded by paresthesiæ—burning pains, cold spots, neuralgic-like pains, etc. Position sense is rarely involved, even in the advanced cases. Tactile agnosia (astereognosis) is frequent. The bony sensibility is very frequently involved (Egger). It may also be, although rarely, an initial sign. Deep pressure sense is often involved. Romberg and ataxia are naturally not infrequent.

2. *Muscular Atrophies and Other Trophic Changes.*—These changes occur most often in the small muscles of the hand (ulnar distribution),



FIG. 186.—Syringomyelia ("prediger hände.")

and of the arms. Thus are produced typical claw-hands, which develop slowly and insidiously. Here again no absolute rule is followed. The interossei may atrophy first, or the muscles of the thenar or hypothenar eminences. A (median) typical monkey atrophic hand may be present, or the "preacher-hand." Or the atrophy in the hands may be skipped, or combined with that of the shoulder-girdle (Aran-Duchenne types), the wings of the scapula stand out, etc. Atrophy of the trunk muscles results in various distortions of the spinal column. Pes equinus, pes valgus, etc., occur in the lower extremity, though less frequently. The bulbar nuclei may be involved, causing speech disturbances; and these may be initial symptoms, though infrequent.

These atrophies are usually progressive; like the sensory changes, they are usually bilaterally asymmetrical, and show much variability.

Thus one may find a claw-hand on one side, and a monkey-hand on the other, etc.

Reaction of degeneration varies, being present in some and not in others. Increase of electrical excitability is present in others.

Fibrillary twitchings are common in the degenerating muscles, and even more active movements are observed, consisting in static tremors, choreic-like movements, intention tremors, or paralysis agitans-like tremors. Cramps are not infrequent, and peculiar myotonic contractions. These irritative motor phenomena belong more to the early stages.



FIG. 187.—Syringomyelia, showing the curvature of the back and the atrophy of the small muscles of the hands.

The motor power is uniformly diminished with the motor atrophy, and spasm or contractions and rigidity may be present. Spasticity is not infrequent in the lower extremities. This only argues for pressure upon or involvement of the pyramidal tracts by the lesion.

The gait is not infrequently involved. The patients tire easily; they commence to walk more slowly; hemiplegic types, paraplegic types, patients bent to one side, or bent strongly forward are seen in the later stages. Ataxic and tumbling gaits belong to the curiosities with rare bulbar localizations. Schlesinger reports a large variety of rare anomalies.

Trophic Disturbances.—These may be many. They may vary in number and extent in strict accordance with the involvement of the trophic cells in the cord.

In the skin one finds cutaneous hyperemias, either active—idiopathic congestive erythemas—or passive, with the formation of dark red patches, or various patches of a dark blue color—cyanotic. These changes are usually associated with others of the muscles or of the bones. Cutaneous anemias and cutaneous edemas (succulant hand), with or without distinct Raynaud symptoms, may occur. The glandular activities of the skin also suffer. The perspiration may be absent, or excessive, or one encounters curious anomalies, such as increased perspiration to cold stimuli, or islets of increased perspiration in normal skin, or perspiration-absent areas. These areas of altered perspiration show anomalous distributions quite comparable to those seen in the sensory sphere. Changes in the fatty secretions may be looked for.

Acute dermatoses are also encountered with exudative phenomena; urticarias, angioneurotic manifestations, phlyctenulæ, dermatitis bullosæ. Hemorrhagic infiltrations, or even ulcer and gangrene of the skin are known. True hypertrophies, sclerodermas, and changes in the nails are recorded.

The bony structures also suffer. Arthropathies are common (10 per cent.—Sokoloff; 25 per cent.—Schlesinger), but more often in the upper extremities, in sharp contrast to the lower limb distribution of tabes. They usually occur late in the disease and persist for many years. The onset is usually acute, with pain, swelling, and destruction. Mild cases cause little or no deformity. Both atrophic and hypertrophic changes occur, with fixation of the joints, dislocations or fractures. The sensory, secretory and trophic disturbances are usually in closely related areas, and bony sensory anomalies are to be expected. Occasionally suppurations occur.

Bony changes in the vertebræ, with thorax deformities, occur either as a consequence of arthropathies, or as muscular atrophies. These scolioses occur usually in the upper dorsal, and give rise to compensatory curvatures.

The bony structures of an entire limb may be involved—a hand or a foot—with atrophies of the muscles. Here acromegaly is to be ruled out usually by the deformed, contracted nature of the syringomyelic hand or foot, the marked muscular atrophy, and the isolated character of the limb involved. Syringobulbia may cause a facial hemiatrophy or hemihypertrophy.

Reflexes.—Great variability and changeability is present. The skin reflexes vary from complete loss to exaggeration. The Babinski reflex may or may not be present, as well as the abdominal, epigastric and cremasteric reflexes.

The tendon reflexes are often striking in that one reflex in the arm for instance will be lost, the others present, or even exaggerated. The

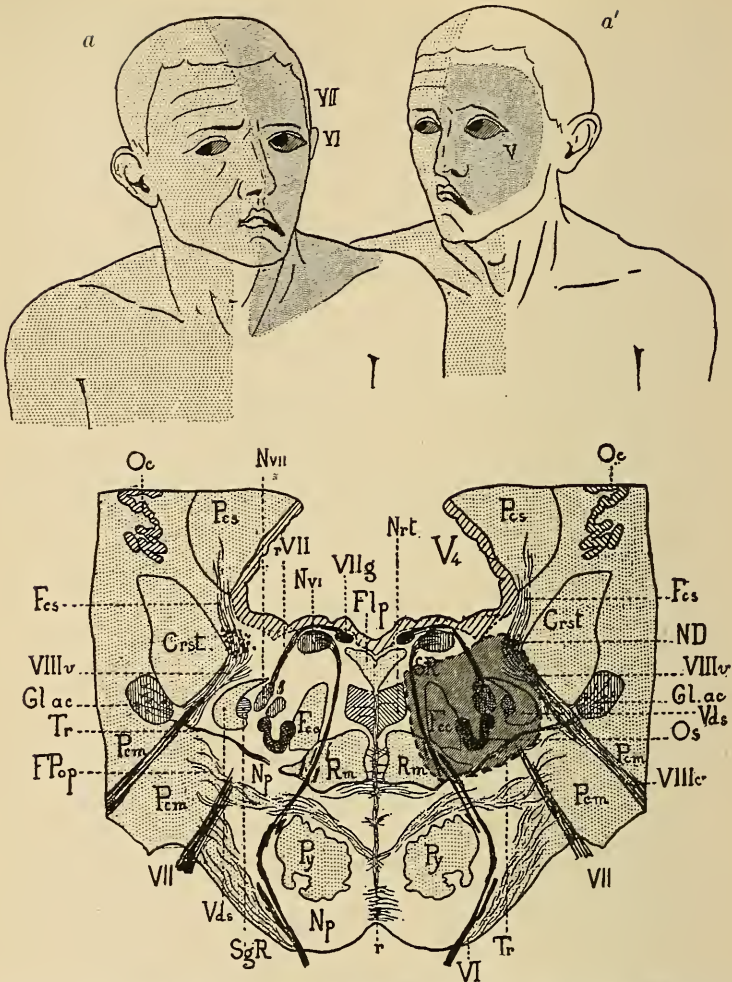


FIG. 188.—Pontine syndrome, with eye palsies of central origin and syringomyelic dissociation. There is here a crossed hemianesthesia with alternating paralysis of the VI and VII cranial nerves, anesthesia of the V nerve due to hemorrhage in the lateral and lower portion of the pontine tegmentum of the left side. The *right-hand figure* shows the hemianesthesia, dissociated as in *syringomyelia* (hemianalgesia and hemithermanesthesia due to lesion of the crossed sensory pathways of the lateral portion of the reticular formation. There is preservation of the tactile and postural sensibilities and of the stereognostic sense, because of the incomplete extension of the lesion to the median lemniscus (*Rm*). The *left-hand figure* shows (1) atrophic paralysis of the VII nerve with reaction of degeneration, lagophthalmia, drooping of the lips, loss of facial mimicry, paralysis of the entire left facial (VII) indicated (Fig. *a*); (2) anesthesia of the face, following involvement of the descending root of the trigeminal (see *V* on *a'*); (3) paralysis of the external rectus with convergent strabismus by reason of the overaction of the antagonists. Furthermore, there is a paralysis of the lateral movements of the eyeballs toward the left notwithstanding the integrity of the posterior longitudinal fasciculus (*Flp.*), of the nucleus of the VI and of the adjacent reticular formation. The lesion of Deiters' nucleus, and of the labyrinthine oculorotary fibers which unite Deiters' nucleus (*ND*) to the nuclei of the III and VI causes this. By reason of the overaction of the antagonists the patient looks to the right. (After Dejerine.) For abbreviations of the anatomical sketch see section on Midbrain.

same holds true in the lower extremity, but is less striking. Increased reflexes are here more apt to be found. Increased knee-jerks, Achilles-jerk, and even ankle-clonus are not infrequent, particularly in high-lying syringomyelias—bulbar, cervical and upper dorsal. Lost knee-jerks, unilateral or bilateral, may also occur in syringomyelia, either as an early or a late symptom, and may be associated with increased skin reflexes (Babinski).

Rare cases are encountered with increased jaw reflexes.

Visceral Symptoms.—Bladder disturbances are not the rule, although at times appearing, usually late in the disease. When appearing early they are apt to be transitory. They may be sensory or motor, irritative or paralytic. Cystitis is not infrequent in the later stages of the disease. In syringobulbias one finds anomalies of secretion, polyuria, glycosuria, diabetes insipidus, pollakiuria.

Obstipation is not uncommon; incontinencia alvi less so. Loss of sexual power and of desire also occur. Persistent priapism is one of the curiosities of this disorder; also analgesia of the testicles.

Bulbar Symptoms.—These are frequent, when isolated, giving rise to syringobulbia. They are usually more benign than when found in other affections (chronic bulbar palsy), have a very chronic course, are usually unilateral, and often implicate the vagus. They occur in about one-third of all cases. Certain affections of cranial nerves have been recorded for many years. Smell and hearing are involved rarely. Vestibular vertigo is also rare. A dulling of the sense of taste is not infrequent, and shows irregularities of disturbance—half-tongue (Dejerine); dissociation (Hitzig, Simon)—anteroposterior changes.

Optic atrophy or swelling occurs in advanced bulbar cases; a few cases show restriction of the color fields. The eye muscles may show nystagmus or nystagmoid movements, paralyse and sympathetic complications. H. Simon reports an interesting case of nystagmoid movements with both eyes, absent when only one was in use. Transitory diplopias, Schlesinger reports in 11 per cent. of the cases; they are not infrequently early signs, and do not necessarily recur. The abducens is frequently affected.

Ptosis is not infrequent, and is often an important early sign. Its relations to the sympathetic are intimate. Myosis is found in a small percentage of cases, and differences in the pupil are frequent, at least 25 per cent. (Schlesinger). They are mostly due to sympathetic palsies. Argyll-Robertson pupil has been observed, but chiefly in pre-Wassermann times, hence the absence of syphilis has not been proved; its presence in syringomyelia is anatomically conceivable.

The Klumpke syndrome is frequently met with and points to the upper dorsal localization of the lesion. Myosis, diminished lid aperture, retraction of the bulbi, and anomalous pupillary reactions (loss of cocaine dilatation, etc.) are signs of sympathetic involvement.

The trigeminus distribution shows sensory anomalies, either as

paresthesiæ and pains (tic douloureux) or as analgesiæ, with loss of the trigeminal reflexes and corneal trophic changes. Dissociation in the trigeminal distribution is also observed. Pain and temperature are mostly affected. Deep sensibility is less often involved. (See Fig. 188.)

The facial is involved in but few cases (3 per cent.) but impairment of ability to swallow is not infrequent. The tongue is involved, projects to the paretic side, shows fibrillary twitching, and hemiatrophy. The soft palate and laryngeal musculature is often involved at the same time, which with the loss of sensation often causes deglutition pneumonia.

Laryngeal palsies are infrequent. They are characterized by the palsy of one recurrent nerve, and the usually accompanying homolateral palsies of the pharynx and soft palate. Posticus palsy and homolateral bulbar palsies go hand in hand. From 15 to 20 per cent. of all syringomyelias have laryngeal symptoms; and they not infrequently antedate the other bulbar or cranial nerve symptoms.

Speech disturbances are common, as one or other of the related mechanisms are apt to be involved. Recurrent palsies give the high, rasping voice, tongue involvement, the thick, hot-potato speech of the bulbar paretic; palatal palsies cause a "nasal" voice—occasionally a scanning-like speech is observed.

Tachycardias, bradycardias, and dyspnea are among the rarer symptoms.

Apoplectiform or epileptiform attacks have been described, and are sufficiently frequent to attract attention. They, however, are usually accompanied by signs of vestibular involvement—rotatory vertigo, nystagmus, vomiting, etc.

Mental Symptoms.—Syringomyelia is often associated with chronic hydrocephalus which two conditions arise simultaneously. Here defect symptoms in the form either of a debility or imbecility are present. Other patients are reticent, surly and vindictive. Many patients come to develop a totally perverted feeling about their anesthesiæ, analgesiæ, etc., and cannot be convinced regarding its true character.

Course and Progress.—The great multiplicity of symptoms, and the almost fortuitous situation of the tumor and cavity formation make it impossible to generalize too sharply about the symptom groupings, but in general one can find four more or less classical types. These are the bulbar, the cervical, the dorsolumbar, and the sacrolumbar.

1. *Bulbar Types.*—*Syringobulbia.*—If restricted solely to the medullary lesions, this is the least common of the types (Schlesinger). Bulbar symptoms, however, are very often found in the other types. In this type the lesion is limited to the cranial nerves. The laryngeal palsies are prominent; difficulties in swallowing are present, either transitorily or persistently, and cause death. Atrophy of the tongue, and paresis of the ocular muscles are frequent. Sensory disturbances in the region of the trigeminus are frequently found.

Initial syringobulbias, in contrast to those developing with or following spinal signs are not as dangerous to life *per se*.

2. *Cervical Type*.—This is the commonest and that best known. The patients first complain of weakness in the small muscles of the hand with clumsiness for finer movements. Paresthesiæ and pains are frequent in the hands and arms. The patients frequently have severe sores on the hands from being wounded or burned, and then note the advancing analgesia, muscular wasting, and loss of ability to tell hot from cold, analgesiæ, total or partial, with intact touch sense. The difficulties are first unilateral, and later spread assymmetrically. The knee-jerks are apt to be exaggerated. Advancing disease shows itself in the greater atrophy, with claw-hand, preacher-hand, monkey-hand, the whole shoulder-girdle may show involvement. The knee-jerks are increased, clonus and Babinski may be present. There may be beginning scoliosis or kyphosis. Trophic disturbances in the upper extremities appear.

3. *Dorsolumbar Type*.—Strictly localized dorsolumbar types are rare. Here the typical syringomyelic sensory anomalies are met with. Paresthesia, pains followed by analgesiæ, thermo-anesthesia, preservation of touch, kyphoscoliosis are frequent. The muscles of the pelvic girdle become involved and the lower extremities show atrophies and deformities. The Klumpke type of sympathetic involvement is seen here as well as in the preceding type, *i. e.*, irregular pupils, irregularity in the palpebral fissure, recession of the eyeball. The gait becomes spastic paretic, the tendon reflexes usually increased—at times lost. Girdle sensations and involvement of the bladder and rectum are not infrequent. Secretory and trophic anomalies, already noted, point to the localization of the process. Many of these cases closely resemble tumors of the cord.

4. *Sacrolumbar Types*.—These are rare types, and are characterized by muscle atrophies, especially of the lower extremities, and the smaller muscles of the feet. The glutei may also be involved. Segmental sensory disturbances of the perineum and genital regions of the dissociated type referred to are present. Trophic disturbances are usually profound and extensive, such as fractures, ulcerations, running sores, etc. Vasomotor disturbances are frequent. The tendon reflexes are usually increased. Babinski may be present. Bladder and rectum are usually involved. Contractures are common, and kyphoses and scolioses occur.

Characteristic Groupings.—Not only do certain types stand out conditioned by the topography of the intramedullary lesion, but certain cases show predominant forms of lesion, such as motor, sensory, trophic, or secretory. Thus certain cases resemble amyotrophic lateral sclerosis very closely; others again have the general features of a spastic spinal paraplegia, others again show a characteristic Aran-Duchenne scapulohumeral atrophy. A few cases of general anesthesia are on record, and certain sensory types may be confused with hysteria.

Trophic cases with an isolated picture of Morvan's disease are striking, and tabetic-like forms are likewise puzzling. Schlesinger also calls attention to a pachymeningitic type.

The illustrative case of Schmitt and Baraban, which is not unique, shows the striking variability that may be present in the symptomatology of this disorder. At various times this patient was diagnosed by competent authorities as tabes, chronic diffuse myelitis, amyotrophic lateral sclerosis, and spastic paraplegia of unknown causation.

Differential Diagnosis.—The chief disorders that come in review are atypical multiple sclerosis, amyotrophic lateral sclerosis, tumor of cord with spastic paraplegia, diffuse sclerosis, progressive muscular atrophy, central myelitis, syphilitic meningomyelitis, polyneuritis, leprosy, pellagra and ergotism.

The *course* of the disease is very chronic. Large cavity formations involving the functions of the intestines and bladder soon lead to death—four to six years; whereas if the lesion does not compress the entire cord and lies outside of the more vital nuclei the patients may be followed thirty to forty years. Dejerine has reported a case of fifty years' development.

The patients die more often of intercurrent disease; tuberculosis in particular. Bladder sepsis, with kidney complications, is also frequent.

The patients frequently show an up-and-down course, quite analogous to the course seen in multiple sclerosis.

Pathology.—Syringomyelia, pathologically speaking, is due to a cavity in the cerebrospinal axis not in genetic relation to the central canal—dilatations of this latter structure are better termed hydro-myelias. Cavities due to hemorrhage are recognized as being different from those in syringomyelia proper and are classed with the hematomyelias. On opening the spinal canal, which is rendered difficult by reason of the vertebral deformities, the dura is usually found to be normal; occasionally a pachymeningitis complicates the picture. The pia is usually thickened, and evidences of swelling or of some internal irregularity are seen either as paths of grayish degeneration, or cystic-like irregularities, with flattening in places. The posterior fissure frequently appears deeply sunken in the cord, causing, as Thomas says, a double-barrelled shot-gun appearance. The cord collapses on cutting and clear fluid escapes. Repeated section, best made after hardening of the cord, shows the existence of one or several cavities, of variable length and diameter, and occupying various situations in the cord. Its most frequent site is behind the neighborhood of the central canal, usually involving the posterior commissure, and with a tendency to reach backward more than forward. The cavity seems to have a special fibrous wall, which is well limited, smooth or papillated; often a gliomatous mass fills the lower end of the canal. In the hydromyelic type the cavity is round and usually occupies the

center of the cord. The cavity is lined first by a layer of epithelium, and is surrounded by a gliomatous wall.

In the syringomyelic type there are also some ependymal cells, but they are less regularly arranged. These are interspersed with neuroglia cells and rest upon a solid wall of glia cells, many of which are in process of disintegration. Small vessels are frequent, among which may be found many undergoing hyaline degeneration. The picture is different at every level, and in many sections the cavity has no lining at all in places, bordering directly upon the nervous tissues. Sections through the glioma show perhaps no cavity at all. Fresh hemorrhagic remains of old hemorrhagic foci are frequent findings.

The glioma may be sharply delimited—central gliosis; or the neuroglial tissue may infiltrate the cord in all directions—diffuse gliosis.

Secondary degeneration in the parts impinged upon or invaded takes place by process of atrophy and then of tissue replacement with characteristic neuronophagia. Regenerated fibers are also encountered.

In cases complicated with pachymeningitis one finds the lesions of this process, and in the traumatic forms one usually finds the remains of an ancient fracture, with inflammatory thickening of the meninges and pia. The cavity is usually posterior.

Pathogeny.—No unanimity of opinion has yet been reached. The general hypotheses are: (1) it is due to a defect of development; (2) it is due to an intramedullary tumor formation which later breaks down; (3) it is the left-over remains of an inflammatory (chronic myelitis) or hemorrhagic process (hematomyelia); (4) it is due to a modification of pressure in the ependymal canal, brought about by a trauma, a compression. In general it seems that no one hypothesis can explain all of the cases.

Thus, according to Schlesinger, one divides the cavity formations that may occur in the spinal cord as follows:

1. Cystic formation, after
 - (a) traumatic destruction of tissue,
 - (b) traumatic hematomyelia,
 - (c) non-traumatic hematomyelia.
2. Softening of an inflammatory or non-inflammatory nature with short course.
3. Syringomyelias:
 - (a) true hydromyelia (as malformation),
 - (b) true tumor with cavity formation,
 - (c) syringomyelia gliosis,
 - (d) syringomyelia from vessel disease without gliosis,
 - (e) pachymeningitis and leptomeningitis with cavity formation.

In a similar manner the cavity formations of the medulla may be classified as follows:

1. Cysts following softening or hemorrhage.
2. Softening of inflammatory nature with acute course.
3. Cavity formation following degeneration of tumors.
4. True syringobulbias (in typical localities).
 - (a) Embryonal, lying in the center, and in combination with hydromyelia or syringomyelia.
 - (b) Without combination with hydromyelia or syringomyelia.
 - (c) Arising in later life and lying laterally as continuation of a syringomyelia due to circumscribed bulbar changes.

Therapy.—Since hemorrhage into the glial cavities is a frequent occurrence in syringomyelia, it is advisable to avoid hard, muscular work, since such seems to favor bleeding. Severe muscular effort also aids in spontaneous fractures. Occupations involving heat are to be avoided because of the danger from burning.

For the pains, the treatment is symptomatic. Here the various analgesic antipyretics may be employed. Aspirin, acetanilid, antipyrine in 5-grain doses are sufficient.

Specific therapy is as yet unknown. One is justified in recommending a spinal cord operation in the case of large hemorrhages with sudden increase in pressure symptoms. Here the principle of open expression of the clot can be practised to advantage (Elsberg).

Certain advantageous results have been reported from the use of high-frequency currents. These, however, have not been employed long enough to determine their absolute efficiency. Beaugard and l'Hermitte¹ recommend weekly applications of penetration rays, 79 (radio chronometer of Benoist), dose of 3 H, at a distance of 15 cent. between anticathode and the skin. Twenty-six seances are recommended.

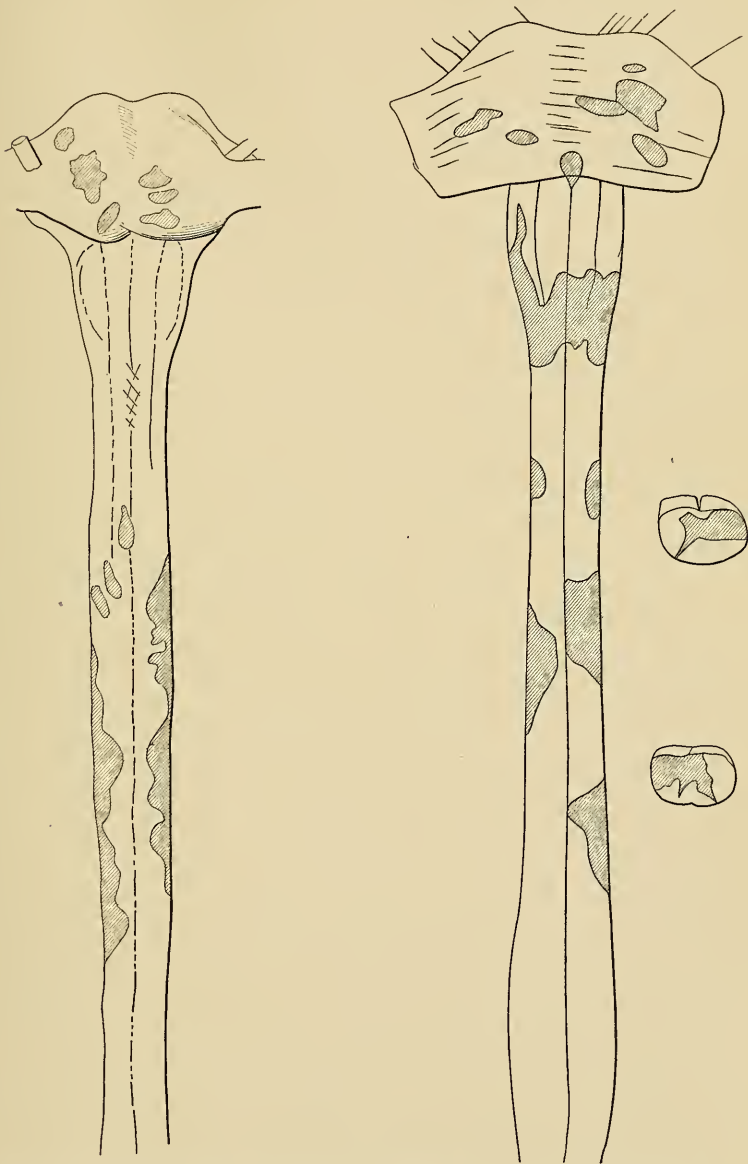
MULTIPLE SCLEROSIS.

Historical.—Because of the striking variability in the symptomatology of this disorder, it is not altogether surprising, historically considered, that the first clues to its final delimitation should have been gained from the pathological rather than from the clinical side.

Under the older nosological schemes one finds these patients grouped as epilepsy, paraplegia, tremor, chorea, dance of St. Guy chronica, and a host of other conditions. Although Sylvius de la Bœe recognized an intention tremor as different from other types, it cannot be said that the conception assumed anything like its modern form until the work of Vulpian (1862), Ordenstein (1868) and Charcot. The initial pathological notion was given, however, by Cruveilhier (1832-1845) in his famous case of Darges (in 1840), a cook in the Salpêtrière, a sketch of whose cord is here reproduced. Carswell's picture, appearing in 1838, is probably the very first illustration of this condition on record. From this time on, brain and spinal sclerosis became an

¹ Sem. Méd., 1907.

active subject of discussion, in which the work of Frerichs¹ and Valentin² stand out prominently. The former made a diagnosis of brain



FIGS. 189 and 190.—Early sketches by Carswell and Cruveilhier (1838–1840) of the sclerotic patches in multiple sclerosis.

¹ Ueber Hirnsclerose, Arch. f. d. g. Med. Haeser, 1849, p. 334.

² Ueber die Sclerose des Gehirns und Rückenmark, Deut. Klinik, 1856, p. 147.

sclerosis during life and found multiple sclerotic patches at an autopsy, and tried to erect a clinical entity with differential pathological diagnosis, which later was amplified by his student Valentiner, who collected a series of cases resembling those of Frerichs, and who also gave a remarkable summary of the symptoms. When these reports are read it may be seen that they would not be included within the modern concept, yet Valentiner picked out the facts of involvement in early years, the marked motor disturbances, hemipareses, tremors, speech disturbances, the remissions, the unilateral onset, the greater involvement of motor than sensory functions, the involvement of the cranial nerves, the long course and bad prognosis. The mental symptoms as outlined by Valentiner are not as characteristic. A diplegic idiot, for instance, is included in his series of fifteen cases.

The first real foundation of the symptomatology may be said to have been laid down by these two authors, and little practical progress was made until the work of Vulpian, Ordenstein and Charcot (1862-1869) and his pupils as summarized in the monograph of Bourneville et Guerard (1869) when the multiple sclerosis of the present day assumed definite form. As early as 1862, Vulpian and Charcot brought the classical triad, *scanning speech*, *nystagmus*, and *intention tremor* into prominence as diagnostic of the condition, and in the later studies of the Charcot school the clinical and pathological foundations were laid much as they were held up to within recent times.

It has been becoming more and more evident, however, that the picture laid down by Charcot was not a satisfactory one. Sensory pictures, neglected by him, had to be reckoned with in the later studies; also interference with the bladder. Decubitus as a symptom occurs. Then cerebellar syndromes were recognized; then Erb's spastic paralysis fell largely into the multiple sclerosis group, then this author's chronic dorsal myelitis with optic changes. Hemiparesis not due to embolism or thrombi became recognized. Alternating Millard-Gubler types were described. Then many bulbar palsies passed into its confines, pontine encephalic pictures, pure ophthalmoplegias, and occasional forms with painful attacks, resembling syphilitic meningo-myelitis. Amyotrophic lateral sclerosis (Dejerine) and pseudoparesis were later diagnostic possibilities, showing the extreme multiplicity of form under which this disorder may show itself. The best recent monograph of real value is that of Müller, 1904.

At the outset it is desirable to call attention to the fact that there exists a multiple sclerosis due to a fairly definite pathological process, and other clinical types resembling the former so clearly as to be clinically indistinguishable, but in which one finds multiple tumors, multiple endarteritic lesions (syphilitic, arteriosclerotic), multiple encephalomyelitic inflammatory processes, etc. These will be discussed under pathology. Whether the clinical picture to be described belongs only

to essential multiple sclerosis, and not to these accessory or accidental forms (secondary forms—Schmaus and Ziegler) is not yet definitely known.

Primary multiple sclerosis then is a disease essentially of adolescent or young adults, beginning very gradually, advancing slowly, initially, very varied, but ultimately assuming a very characteristic, almost monotonous character. It is characterized by beginning muscular weakness, with spasticity, by disturbances in speech, nystagmus, intention tremor, by forced laughing or crying, and by changes in the fundus—temporal pallor. Sensory symptoms may be present but are not usually prominent, bladder symptoms are not uncommon, while vasomotor and trophic disturbances are comparatively infrequent. The disease persists for many years, has striking remissions— noted by Valentiner in 1856—and the patients die of exhaustion or intercurrent disorder.

Multiple sclerosis is a relatively infrequent disease. Of 18,000 cases of nervous disease at Vanderbilt Clinic it was diagnosed as occurring 27 times, *i. e.*, a percentage of 0.001 per cent. In European clinics it would appear to be more frequent. Bramwell-Williamson show 2 per cent. Possibly better diagnoses account for these differences, since the American statistics are obtained from polyclinic material. These are not checked by autopsy findings, and are suggestive rather than conclusive of its frequency. Personal figures show 1 patient in 200 of nervous disorder, 0.5 per cent.

Etiology.—A definite position regarding this cannot yet be taken. Practically all of the infections have been held responsible, and it would appear that they stand in some sort of causal relationship. Certain metallic poisons, zinc, copper, manganese, give rise to a picture closely resembling multiple sclerosis. Trauma has been held responsible; so also have sexual excesses, cold and wet and childbearing.

Heredity plays an important factor, according to many (Pelizeus and others), and Strümpell contends that the essential feature is an abnormal congenital factor, which is made to develop by any one of the other causes here enumerated. Müller's critical summary would seem to exclude practically all the exogenous causes since they occur in but a very small proportion of his cases. He allows that a secondary multiple sclerosis, in the sense already outlined, may possibly follow infections, but that multiple sclerosis, in the narrow sense, develops only on the basis of a congenital predisposition.

Sex plays practically no part. Both the results of Charcot's and Whitoff's studies, which spoke for greater frequency in female and male material respectively are due to their particular clientele. Polyclinic statistics nearly always show a greater frequency of women, because they are freer to visit dispensaries, and usually go to all of them in a big city. The only reliable statistics are those of Müller, which were controlled by autopsy. They show practically no difference.

Age.—Three-fourths of the carefully observed cases occur between the years of twenty and forty. In our own statistics¹ two-thirds of the patients were under forty. Cases have been reported in children as young as five months, and in adults as old as seventy-five to eighty years, but for both extremes diagnostic mistakes are not ruled out. Marie's view that multiple sclerosis is common in children has not withstood the severe critique of autopsy material, and Müller takes the stand that the disease is found extremely seldom in very early youth. The initial stages, however, may be traced very often to the years of adolescence or early adult life. Occupation plays no role that is yet known.

Symptoms.—The accidental features of the distribution of the sclerotic patches in this disorder makes it possible for almost any combination of neurological signs, and the greater the number of cases studied the richer has become the symptomatology. Certain patients show comparatively few symptoms for years; others show additions almost from month to month, until they become veritable neurological museums, with signs of involvement from the frontal poles to the tip of the cauda equina. No two patients are alike, yet most seem to attain the same level in the end and almost come to present a stereotyped picture.

In this picture the most striking feature is the involvement of the motor system, giving rise to muscular weakness, hypertonus and general spastic phenomena, both in the cranial and spinal paths. The extreme range is the feature that characterizes multiple sclerosis in its fully developed form. One finds isolated or group phenomena all over the body just in the same proportion as the accidental distribution of the sclerotic patches is isolated or diffuse. For this reason it has been thought advisable to begin the description of the symptoms with those of the cranial nerves, and proceed systematically throughout the nervous system. The older and classical triad of Charcot is only partially true, and today a multiple sclerosis may be diagnosed from a temporal pallor of the optic disks combined with bladder incontinence in the absence of nystagmus, intention tremor, or scanning speech.

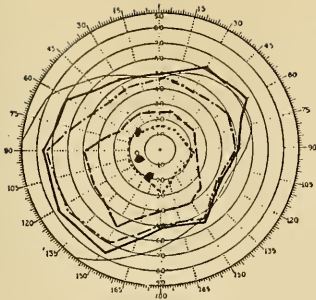
The classical picture of Charcot may be found in not more than 15 per cent. of all the cases, at a period when the presence of other symptoms permit one to make a diagnosis of multiple sclerosis. If one waits for the "classical" picture, one may have to wait for years.

Olfactory.—Hallucinations of smell are occasionally found but belong to the rarer and infrequently recorded psychical signs. Bilateral anosmia has been recorded.

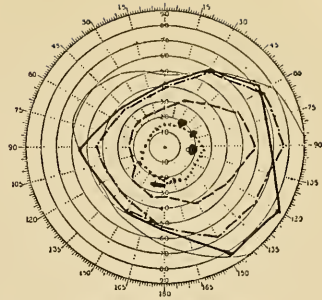
Optic.—The researches of Uhthoff have emphasized the frequent occurrence of changes in the optic disks. About 50 per cent. of the patients show changes in the papillæ, and it is striking that these

¹ Jelliffe, *Jour. of Nerv. and Ment. Dis.*, 1904, p. 446.

FIG. 191

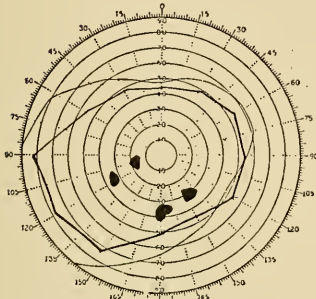


Left eye vision 20/30



Right eye vision 20/30

FIG. 192

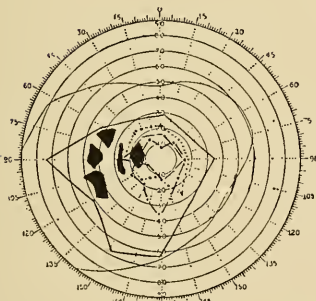


Left eye vision 15/40

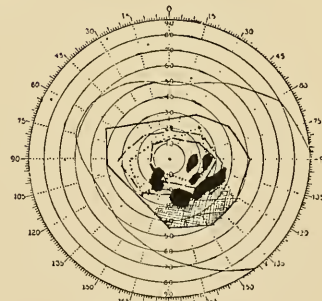


Right eye vision 15/40

FIG. 193

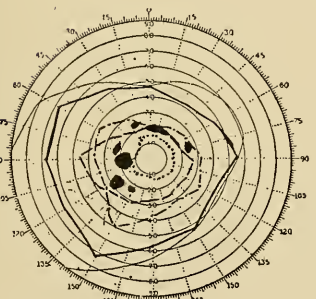


Left eye vision 15/30

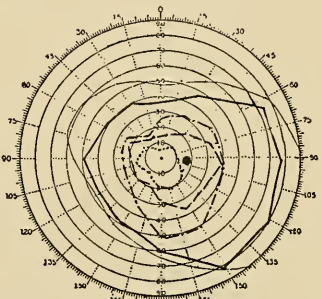


Right eye vision 15/50

FIG. 194



Left eye vision 20/40



Right eye vision 20/30

Field of vision——White - - - - - Blue

Green for 5 mm. □
Relative scotoma ▨

FIGS. 191-194.—Irregular scotomata seen in multiple sclerosis cases. (Klingmann.)

changes occur early. They are in the nature of partial atrophies, and show themselves for the most part as a simple atrophic pallor of the disk—usually most marked temporally. True optic atrophy is found in some of the cases, and again in still fewer a true papilloedema or choked disk. This usually recedes and either leaves no sign or an atrophy. A great number of variations are known.

The sight is frequently affected, sometimes blindness, partial or complete, unilateral or bilateral, is an initial symptom. This not infrequently clears up. Complete blindness with double-sided atrophy is extremely rare (Gnanck-Uthoff). Parinaud has made three classical types, but later studies have shown that there is no uniformity, and the variations in sector blindness and dimness are very many. The loss or diminution in sight in multiple sclerosis is characterized by its advancing and receding character, and by the fact that as a rule the ophthalmoscopic picture is usually more pronounced in severity than the symptoms, and offers no satisfactory register of the variability in visual acuity.

The fields of vision more often show central scotomata with peripheral clearness than peripheral scotomata. Unilateral scotomata, quadrant scotomata, and a great variety of other forms are known as well as a certain amount of variability from time to time in the individual case. The color fields show similar variations and variabilities. Optic hallucinations also occur. (See Figs. 191 to 194.)

Ocular Symptoms.—The characteristic phenomenon here is nystagmus. Its occurrence as a symptom of “brain sclerosis” was first pointed out by Valentiner (1856) and Charcot overvalued its diagnostic importance. True central nystagmus is rare, but nystagmoid movements on lateral motion of the eye occurs in from 70 to 80 per cent. of the cases, especially in the later stages of the disease. Vertical, oblique, rotatory nystagmus is included with the other forms. In cases with pronounced cerebellar involvement the nystagmus is of this type, and rotation of objects with subjective rotation and forced positions are to be expected. These eye movements may be summarized as: (1) continued rhythmical oscillations (true central nystagmus) analogous to the continuous movements of the head and body; (2) rhythmic oscillations set up on movement of the bulb in any direction, analogous to the intention tremor of the hands; (3) nystagmoid movements on extreme lateral or vertical movements, analogous to fatigue (paresis) movements; (4) ataxic movements. Of these (3) is the most frequent; practically all are conditioned by central disease, although peripheral involvements are described.

Eye Muscle Palsies.—These are important in diagnosis, and present themselves infrequently as isolated or complete palsies, transitory or persisting. Paralysis of convergence is more often found. In general, from 17 to 46 per cent. of the patients show palsies, the statistics varying according to the more or less strict interpretation of paralysis. Uthoff demands “double vision” as a criterion, which symptom is

not an infrequent early symptom of multiple sclerosis. Müller, on the other hand, admits milder palsies into his statistics (46 per cent.).

Ptosis is less frequently found (8 to 15 per cent.), usually one-sided, occasionally double, usually incomplete and ephemeral. One also meets with external ophthalmoplegia, abducens, and trochlearis palsies. Divergent palsy has been noted by Bielschowsky. Internal ophthalmoplegia has not yet been recorded.

Pupillary inequalities are not infrequent (24 per cent.—Müller), and these are changeable and frequently early. They may represent spasms or paresis of muscles. Miosis is frequent in the later stages (Parinaud), but the reflexes are usually normal. Pupillary unrest (hippus) is found (Frankl-Hochwart). Argyll-Robertson pupil was found only four times in 364 cases. One case of Uhthoff was controlled by autopsy; two cases by Rad,¹ Wassermann control.

V. Trigemini.—Facial neuralgia has been observed as the first symptom of a multiple sclerosis (Oppenheim), but both sensory and motor fifth nerve involvement is rare.

VII. Facial.—Facial palsies, usually associated with other central (hemiplegic) pontine (alternating palsy), or pressure symptoms, occasionally coming on very acutely, are not infrequent (15 per cent.); as an isolated palsy it is rare. Like other palsies their fluctuating character is striking, the severe permanent types being rare (Bouchaud), as are also bilateral palsies.

VIII.—Hearing is rarely affected. Complete or partial deafness has been recorded, as well as the occurrence of noises. Auditory hallucinations are possible, but not anatomically proved. Hyperesthesiæ are frequent.

The vestibular branch of the eighth nerve is not infrequently involved in its cerebellar connections. An explanation of some of the more severe nystagmus movements may be found here. Giddiness and dizziness are very prominent symptoms, and occur either from involvement of this nerve or other sensory cerebral or spinal tracts. Rotatory giddiness with tendency to fall, revolving of objects, etc., is present in a few cases of multiple sclerosis, and certain Ménière cases belong here.

Speech Disturbances.—Charcot's typical speech disturbance had a slow, monotonous, and scanning character. This special type is present, however, in only a small proportion of the cases, and then usually only in the later stages. Müller's autopsy controlled material (81 cases) gave only 25 per cent. of this character, whereas patients with other types of speech disturbance run higher—25 to 30 per cent.—making about half of all multiple sclerosis cases with some disorder of speech. Easy fatigability, with increasing unsteadiness; stuttering, with accentuated mouth movements, are other types.

Singers soon notice these alterations, especially the fatigability,

¹ Neur. Ctblatt, 1911, xxx, 584.

and an increasing inability to modulate the tones. Dysarthria and explosive speech are obtained in later stages. Articulatory disturbances with repetition of syllables, or words, is frequent in later stages—*r*, *l*, *p*, and *g* are particularly difficult. Aphonias or other laryngeal complications are occasionally met with (Rethi). The tongue occasionally shows mild transitory palsies, with mild atrophies, usually unilateral and fibrillary twitchings. Ataxic movements of the tongue are frequent in the later stages. Occasionally subjective sensations of fulness, thickness, and difficulty in movement are observed.

Chewing and swallowing are involved, the former rarely, the latter not infrequently, less often transitorily, as an early symptom more often as a permanent terminal sign. Excessive salivation seems very infrequent.

Pneumogastric involvement is rare. Müller reports a case of paroxysmal tachycardia in multiple sclerosis. Dyspnea is also known, but is infrequent.

The *taste* has been modified in a few cases.

Motor Disturbances.—The most characteristic are the intention tremor, ataxias, and palsies with later developing atrophies and contractures.

Ataxia.—By the finger-nose test or the finger-finger test, and by the static position one can demonstrate in the upper extremities an ataxia, separate from an intention tremor, in a large number of the cases (Müller, 70 per cent.). A similar ataxia may be shown in the lower extremities in even a more striking manner in testing by the knee-heel test. The ataxic movements usually precede the development of the intention tremor, and are later often covered up by the same. Occasionally the ataxias are increased on closure of the eyes, occasionally not, showing that at least two types are to be observed. Recent findings in the sphere of deep sensibility, position sense, etc., afford an interpretation of these ataxias. They vary clinically from the tabetic ataxia, particularly in the increased tempo of the individual movements and the irregular contraction of the agonist muscles in the former. The usual hypertonus of the multiple sclerosis patient is in marked contrast to the hypotonus of the tabetic. Crossed hemiataxias are recorded.

Intention Tremor.—Present in from 50 to 75 per cent. of the cases, this symptom is one of the most striking in well-advanced multiple sclerosis. It may be absent for a number of years, and yet a diagnosis may be made on other grounds. When present it speaks almost always for this disorder. Considerable care must be exercised in not confusing an intention tremor with an ataxia, and *vice versa*. Müller has laid considerable stress upon this point, and because of his more rigid criteria regarding the differences, states that real intention tremor was present in only 25 per cent. of his cases. This intention tremor is increased by prolonged effort and by emotional disturbances. It is not markedly increased by shutting the eyes.

It develops gradually, occasionally suddenly after an apoplectic attack, is more often bilateral, corresponding closely with the pareses. The arms are more frequently involved, then the trunk, and then the head; rarely the chin and muscles of the face.

Only exceptionally are the legs markedly affected by an intention tremor. In the trunk this disturbance gives rise to a type of continual balancing or rocking motion; it also involves the muscles supporting the head, causing a nodding and swaying series of movements. Passive tremors of the hands when at rest are also observable.

The causes for the intention tremor are as yet unsolved, purely spinal causes do not show it, and the greatest probability exists in the hypothesis of midbrain localizations, involving both incoming sensory tracts and outgoing motor paths. Similar tremors are seen in lesions of the cerebello-rubro-thalamic tracts. A complete analysis of the fiber tract involvements in multiple sclerosis has yet to be made before the question of localization is settled.

Writing shows characteristic changes due to both the intention tremor and the ataxia. The strokes are irregular—too thick or too thin—and the excursions in forming the letters irregular.

Motor Power.—Motor weakness is usually the earliest symptom noted in multiple sclerosis. It may be the arms, oftener the legs, or occasionally in some special group of muscles; larynx in singers, hand and arm in painters or players, occasionally bladder or rectum. Marked fatigability is an important symptom, and may precede the other symptoms for years.

Spastic paresis, with hypertonus, then develops, and though varying considerably in intensity, often dominates the picture, leading later to the most severe forms of contractures. The type may be hemiplegic or paraplegic, and usually indicates involvement of the pyramidal tracts. The lower extremities are involved much oftener than the upper. Irregular Brown-Séguard paralysis is occasionally present.

Gait disturbances are common and variable. They may be purely spastic, or in the cerebellar cases, spastic ataxic. Pure ataxic gaits are very rare. A spastic paretic gait marks the pre-bed stage. Occasionally one finds the tumbling or reeling gait of cerebellar tract implication.

Certain motor anomalies are encountered as the patients rise or sit down. A Romberg is not uncommon on standing, which is not much modified on closing the eyes.

Muscular Atrophies.—These occur, but are rare.

Sensory Phenomena.—Marie's earlier contention that sensory disturbances do not belong to multiple sclerosis has been definitely disproved.¹ On the contrary they are very frequently found, but largely by reason of their fluctuating character, both as to intensity

¹ Jeremias has collected the rich literature bearing on sensory disturbances in multiple sclerosis. Dissert., 1898; see also Müller.

and extent are overlooked or falsely interpreted. The frequently made false diagnosis, "hysteria," is usually founded on these fluctuating, sensory signs. Hoffman and Freund found sensory disturbances in from 70 to 90 per cent. of their cases. Müller found them in 76 per cent. Pains are not frequent, paresthesiæ, on the other hand, are very common. Anesthetic or hyperesthetic spots are frequent; the patients frequently complain of dead or numb fingers. Tactile insensibility is not infrequent, occasional tactile agnosia is present. The sense of localization is frequently faulty, and bony sensibility is also often involved, with diminution of position sense and disturbance of thermal sensibilities.

Skin Reflexes.—The abdominal and epigastric reflexes.—are almost invariably modified (80 per cent.). There may be unilateral diminution or loss, or more often bilateral diminution or loss (62 per cent.). Thus bilateral loss of these reflexes constitute a very important differential for the diagnosis of not only an organic nervous disease, but especially for multiple sclerosis. Careful studies by Müller have shown that when correctly tested they are modified in only about 5 per cent. of normal individuals, and these occurred most often in women who had had children, or in very obese subjects. The cause is to be sought in an interruption of the sensory tracts, either in the cord, in the median lemniscus, in the thalamic end stations, or in the post-Rolandic sensory distributions. The multiplicity of the sclerotic patches makes it liable that this reflex path will be implicated somewhere in its march to the cortex.

The cremasteric reflex is also often involved unilaterally, or bilaterally, but less often. Also the vomiting reflex, though not so often.

Babinski's phenomenon is very frequent; Müller says, almost constant. Crossed Babinski may even be observed. Oppenheim reflex is very often, though less frequently, found.

Like all of the symptoms of multiple sclerosis, the skin reflexes are subject to considerable variations. They frequently return after showing a loss.

Tendon Reflexes.—In concordance with the spasticity and hyper-tonus increased tendon reflexes are to be expected. The upper extremities show increased triceps-jerk and increased radius-periosteal reflexes. The Achilles-jerk is exaggerated, unilaterally or bilaterally, as also the knee-jerk, and usually there is unilateral or bilateral ankle-clonus. Patellar clonus is less often observed.

Vasomotor and Trophic Signs; Other Signs.—One finds a number of anomalies of this group in a large collection of multiple sclerosis cases, but, as a rule, they are sparse and isolated. Among these may be mentioned hyperidrosis, angiospasm (cyanosis), vessel palsies with edema, erythromelalgia-like affections, syringomyelic-like trophic disturbances, abnormal blushing. Hyperpyrexias are encountered with apoplectiform attacks. In the ordinary cases the patients show marked lability of the body temperature.

Pilomotor reflexes are frequently disturbed; thus dermatographia is common, and may show unique isolated localizations.

Bladder.—The bladder is frequently involved (80 per cent.—Oppenheim; 75 per cent.—Müller), often transitorily, and also it may be an early symptom. The patients feel a sense of insufficiency, and have to strain to pass their water. Complete paresis with incontinence is rare. There is a great variability, with many ups and downs in the bladder symptoms. Polyuria and glycosuria have been observed.

Obstipation is frequent; loss of control less so, but is present.

Menstruation, childbirth, parturition, etc., are not markedly affected. Genital hyperesthesia is at times pathologically increased and occasionally there is anesthesia with impotence.

Lumbar Puncture.—The experience thus far gained shows no appreciable changes in pressure. Cytologically, occasionally marked slight lymphocytosis has been observed, but the majority of the cases have been negative. Serologically, some positive Wassermann reactions have been recorded.¹

Mental Symptoms.—These are not constant nor prominent, and are for the most part absent, yet careful analysis shows them to be much more frequent than is usually taught. Mild intellectual reduction in the form of a dementia, hallucinations of hearing, of sight,² hallucinatory states with mild confusion, passing ideas of reference and of persecution, difficulty in thinking, spasmodic intermittent alternations in the capacity for attention and concentration, lapses of memory, etc.; these have all been recorded. The latter anomalies are not infrequent, and often are colored by depression (melancholic) or excited (manic) states, or more frequently the emotional tone is one of indifference. Pseudoparetic states may be expected at times.

Involuntary Laughing and Crying.—These were described by Cruveilhier in his classical case, and correctly appreciated by Bourneville. They are purely neurological signs, for neither the laughing nor the crying are necessarily accompanied by their usual emotional states, nor do they necessarily betoken any intellectual reduction (evidence against the James-Lange emotional hypothesis). They are very often found, forced laughing being the more frequent (40 per cent.—Müller). They are both to be referred to implication (in part) of the corticobulbar-cerebellar reflex paths. An analysis of all of the variations is not possible here.

Apoplectic and Epileptiform Attacks.—These occur but not as frequently as was held before one was in a position to rule out anomalous parietic attacks. But the frequent occurrence (20 to 25 per cent.) of mild attacks with transitory disturbances of consciousness, or attacks of giddiness or faintness, with unilateral or bilateral pareses, or sensory anomalies in the distal extremities, should be emphasized. Such mild attacks resembling arteriosclerotic attacks, often mark the

¹ Nonne, Deut. Zeit. f. N., 1910, 1912.

² Nonne, Mitt. Hamb. Staats k., 1910.

initial stages, or accompany the sudden progressions which are characteristic of this disorder. Epileptiform attacks are much less often observed and usually speak against a multiple sclerosis, but they do occur, and very frequently show as Jacksonian attacks (Gussenbauer).

Characteristic Forms.—As already noted, the classical signs of Charcot, with nystagmus, intention tremor and scanning speech, in reality are found in only a comparatively small proportion of the cases in the earlier stages of the disease. In the interest of early diagnosis, this must be born in mind, especially, as true nystagmus is always rare, nystagmoid movements are to be judged with care, and the tremor and scanning speech are usually later symptoms. The “non-typical” cases in Charcot’s sense (*formes frustes*), are really more frequently met with. Any attempt to state which special group of symptoms occurs with more or less frequency is apt to be misleading, especially in view of the great variability met with and the inconstant changes, the advance of certain symptoms and the retrogression of others.

For purely didactic purposes, one can divide the more common symptom pictures as follows:

1. Cases that begin with isolated or prominent cerebral symptoms. These occur less frequently than the spinal cases, perhaps than the bulbar, but hence are also more likely to be overlooked. Here optic atrophies, with defects of vision, ocular palsies, transitory diplopias, crossed eyes, etc., muscular weaknesses are prominent, and occasionally associated with giddiness, nausea and headache.

2. Cases with isolated or prominent bulbar symptoms. These begin as mild or severe bulbar palsies, and are rare.

3. Cases with irregular and prominent spinal symptoms, mostly involving the lower extremities. Here the patients complain of the legs becoming tired easily, and after a time they stumble, or trip easily. They then become somewhat stiff and walking upstairs becomes increasingly difficult. Mild bladder disturbances may have preceded or accompany the weakness and stiffness in the legs. Paresthesia are also frequent. These patients soon show spasticity, increased knee-jerks, clonus at times, Babinski’s sign. The abdominal and epigastric reflexes are diminished or absent unilaterally or bilaterally.

This is apparently the most frequent mode of onset, since very often the patients have paid no attention to the rapid passing of transitory diplopias, slight speech difficulties or weakness of the bladder, or mild attacks of giddiness, yet sharp questioning usually elicits some of these other signs as having preceded or accompanied these spastic paraplegic types. Whereas these signs usually come on very gradually, they may appear to have had a sudden onset, as after a long walk, or slight accident, or following childbirth, or other striking intercurrent event.

4. In the vast majority of the cases there is a gradual onset of both spinal and cerebral symptoms. Headaches, diplopia, difficulty in walking, slight changes in speech, giddiness, abnormal muscular tire, paresthesiæ, transitory bladder weakness, etc. These symptoms progress and then recede—usually attributed to the skill of the physician or thought of as hysterical—and then reappear in the same or in an entirely different order.

5. A small number of cases begin with a mild initial apoplectiform attack, and then either progress, or stand stationary for some years.

Non-characteristic Forms.—These unusual forms may be summarized as (*a*) forms which run a more distinctly psychic course, with the picture of slowly advancing dementia, and pseudoparetic forms; (*b*) forms that resemble brain tumor—hydrocephalus; (*c*) Jacksonian attacks due to patches in the motor area; (*d*) hemiplegic forms due to patches in the cerebral course of the pyramidal tracts; (*e*) bulbar paralysis and pseudobulbar paralysis types; (*f*) sacral forms as described by Oppenheim, resembling tumor of the cauda equina; (*g*) forms that give an acute onset resembling pontine encephalitis; (*h*) cerebellar types, resembling paralysis agitans (not infrequently called early paralysis agitans); (*i*) forms that give a picture like cerebrospinal syphilis; (*j*) progressive muscular atrophy and amyotrophic lateral sclerosis-like forms; (*k*) tabetic-like forms; (*l*) transverse myelitis and combined sclerosis types, and finally (*m*) latent and recessive forms which are differentiated from hysteria neurologically only after many years of the most exact scrutiny.

Diagnosis.—Enough has been said to indicate how a multiple sclerosis may appear under the guise of a number of organic diseases of the brain and spinal cord. The most important features in the diagnosis concern the age of the patients, young individuals, the usual lack of heredity, the failure of usual external causes to account for the symptoms, absence of Wassermann reactions and of spinal fluid findings. Of the more important objective findings for the early diagnosis one counts on the early feeling of motor weakness, feelings of giddiness, the onset of spasticity with increased reflexes, clonus and Babinski, the presence of the fundus changes, the loss of the abdominal reflexes, fine ataxic movements of the legs in the knee-heel test, and of the arms in the finger-finger and finger-nose tests.

In the later stages the full pictures as already outlined appear and make a diagnosis certain. One feature of perhaps the most striking importance is the peculiar advancing and receding course, the remissions during which the entire picture seems to fade away and which permit a patient condemned to a chronic organic nervous lesion to turn up in the physician's consulting-room apparently well.

It is for this latter reason perhaps more than any other that the false diagnosis, hysteria, is so frequently made in these cases. Then there are in addition the almost daily fluctuations in the sensory sphere which always suggest hysteria, and which only a rigid analysis

will exclude. Furthermore it must not be forgotten that not only does one experience the changeability in symptomatology suggestive of hysteria, but a helpful optimistic psychotherapy can most markedly alter the symptoms as well, causing some of them to entirely disappear. The rise in the level of nervous energy by such psychic means seems to enable the patient to force a better control of the disturbances. In the failure of a typical hysterical character—the hysterical constitution—this disorder should not be diagnosed. Finally one has always to reckon with a symptomatic hysterical reduction in the patient's resistance due to the presence of an organic lesion.

Further, diffuse sclerosis, general paresis, brain tumor, chronic hydrocephalus, arteriosclerosis, cerebral syphilis, infantile palsies, encephalitis of a diffuse disseminated type, bulbar palsies, chronic leptomeningitis, infantile ataxias, Friedreich's ataxia in particular, disseminated myelitis, meningomyelitis, Erb's spastic palsy, hereditary spastic palsies, syringomyelia, tabes, spinal cord tumor, paralysis agitans, chronic zinc poisoning, chronic manganese poisoning, chronic mercurial poisoning and spinal cord edemas all come in review in the differential diagnosis of multiple sclerosis.

Pathology and Pathogenesis.—On autopsy in the cases of true multiple sclerosis one finds, as a rule, a normal dura, the brain itself usually shows irregular atrophies with thinning of the cortex and some internal hydrocephalus. At times one can observe on the surface the irregular patch-like areas of atrophy. These usually show much more frequently on the surface of the pons, medulla and cord, as Carswell and Cruveilhier showed early. On section of the brain one finds few (five or six) or many (several hundred) irregular sclerotic patches, which are usually isolated one from another.

Microscopic examination invariably reveals many not seen by the naked eye. They vary in size from that of a pin-head to 5 to 6 cm., which larger patches are usually made up of several smaller ones. The general color tone is gray. The reddish patches usually belong to the secondary encephalomyelitides. The plaques either rise slightly from the surface or are level or show a slight depression; the tissues about are slightly edematous. At times, particularly in the optic nerves, there is distinct shrinkage in the tissues. The consistency is usually tough, or almost leathery. The soft plaques are apt to resemble disseminated myelitis, encephalomyelitis, etc. The patches are very sharply differentiated from the surrounding tissues. As to localization, they may be anywhere; in the brain, cord, medulla, pons, cerebellum, the roots of the cranial or spinal nerves, within the optic nerve itself. As a rule they are found in both brain and spinal cord, and seem to have a special fondness for places particularly rich in neuroglia, and there is a certain grade of asymmetrical symmetry in their localization.

In the cord, the median line, and the pyramidal tract region are favorite localizations, and the white matter more than the gray above

all. Central gliosis seems rare. In the medulla the olivary region, the floor of the fourth ventricle, and the posterior aspects are favorite sites, while in the cerebellum the dentate nucleus is usually most often implicated.

Histologically the patches are made up of masses of glia fibers. There is no areolar composition and the glia nuclei do not appear prominent. Small holes may be found within the plaques, around which the glia fibers cluster as about a bloodvessel. Bielschowsky preparations show the partially intact nerve axones passing through the glia masses in much reduced numbers; the medullary sheaths are partly modified in their qualities. Charcot first called attention to the persisting axis cylinders. The immense mass of glia fibrils is an entirely new growth, apparently not to take the place of destroyed nervous tissue, but a pure addition product in which one finds the almost normal elements still present, for a time at least. Secondary degeneration outside of the plaques is not present.

What relation the disease has to the vessels is still far from clear, but it seems certain that primary disease of the vessels is not a part of true multiple sclerosis.

The differentiation from disseminated myelitis, encephalomyelitis, and other secondary affections which may give rise to partial multiple sclerosis pictures must be sought in special works on pathology.

Prognosis.—In general the disorder is progressive, yet there are many stationary cases, and some few that apparently recover; these are possibly mistakes in diagnosis, but such mistakes, in view of the great number of similar affections, are almost impossible to avoid. Many patients extremely ill and bed-ridden, following one of the acute advances of the disease, recover almost completely; but usually there is another acute advance, and then others. The time between advances or the length of remissions cannot be stated; they have varied from six months to ten years; some very rare observations show a period of twenty years.

A few patients die rapidly of the disorder (six weeks to six months)—acute multiple sclerosis of Marburg, Frankl-Hochwart and others—but the majority live for many years, and die usually of intercurrent disorders, pyelitis, tuberculosis, pneumonia, etc. Müller's average was four years; Charcot's, six to ten years.

Therapy.—Specific therapy is not yet known. Prophylaxis also seems difficult to grasp since no definite exciting cause is known. Relative prophylaxis in the shape of advising against marriage for young affected individuals, or against childbearing in the married is necessary. Childbirth would seem to be an exciting cause for an active exacerbation.

Rest in bed is more than desirable in acute stages, it is imperative. The rest must be absolute, and should include eye rest. Active remedial treatment—massage, hydrotherapy, electrotherapy—are all disadvantageous. Warm baths for short periods are not contra-

indicated. Definite motor quiet and sensory quiet as well is desirable. Counter-irritation in those cases which show sharp myelitic symptoms is to be tried.

Pharmacotherapy has not yet devised any useful remedies. Arsenic is used on empirical grounds and in combination with quinin, ergot, strychnin and iron does some service.

Mild work in the open air, gardening, etc., is advisable. Psychotherapy is never to be forgotten, and a healthy optimism helps these patients enormously.

CHAPTER VIII.

DISEASES OF THE PONS, BRAIN STEM, AND MIDBRAIN.

DISEASE of the brain stem offers special problems of diagnosis of extreme difficulty, both with reference to the motor and sensory disturbances. Attention will first be directed to the latter. (See Plates X and XI.)

So long as the sensory paths were in their spinal route they were capable of a certain amount of isolation either as they entered the cord and made their first synapses, or as they continued up the cord in primary or secondary paths. But as these paths converge to enter the brain stem they become closer anatomically, disease processes are apt to overrun many paths, and thus the analysis becomes increasingly difficult up to the entering of these paths into the optic thalamus.

Head and Holmes have made the most searching analyses and their researches tend to show that the impulses underlying sensations of pain, heat, and cold seem alone to run unaltered, either directly or by intercalated fibers associated with the ganglion cells of the formatio reticularis, between the upper end of the spinal cord and the optic thalamus. Here are received the regrouped secondary impulses from the face which cross then join the specific paths for pain, for heat or for cold. These paths are so situated that they can be interrupted without disturbance of any other form of sensation of the body, and the analgesia and thermo-anesthesia so produced resemble in

GENERAL LEGEND ABBREVIATIONS OF PONTINE, PEDUNCULAR, AND MIDBRAIN SYNDROMES.

In all of these hemiplegia is indicated by oblique lines; hemianesthesia by dots, and alternate paralyzes by gray network. The lesions involve the cerebral peduncles and the pons at different levels.

ABBREVIATIONS. *Aq.*, aqueduct of Sylvius; *BrQp*, peduncle of posterior corpus quadrigeminum; *Crst*, corpus restiforme and inferior cerebellar peduncle; *Fcc*, central tegmental tract; *Fcs*, internal semicircular fibers of the cerebellum; *Flp*, posterior longitudinal fasciculus; *FPOa*, *FPOp*, anterior and posterior pontine fibers; *HC*, cerebellar hemisphere; *Lc*, locus ceruleus; *LN*, locus niger; *Lig*, lingula of superior vermis of cerebellum; *ND*, Deiters' nucleus; *Np*, nuclei of pons; *NR*, red nucleus; *NRL*, nucleus of lateral lemniscus; *Nrt*, reticular nucleus of the tegmentum; *NIII*, nuclei of third nerve; *NmV*, motor fifth nucleus; *NsV*, sensory fifth nucleus; *NVI*, nucleus of sixth nerve; *NVII*, nucleus of the facial nerve; *NVIII*, anterior nucleus of the acoustic (cochlear); *Oc*, cerebellar olive; *Os*, superior or pontine olive; *P*, lower stage of cerebral peduncle; *Pcm*, middle cerebellar peduncle; *Pcs*, superior cerebellar peduncle; *Py*, pyramidal tract in its pedunculo-pontine region; *Qa*, corpus quadrigeminum anterior; *r*, raphe; *Rm*, median lemniscus; *RL*, lateral lemniscus; *SAq*, gray substance of the aqueduct of Sylvius; *SgR*, substance of Rolando; *Tpo*, tenia pontis; *Tr*, trapezoid body; *V₄*, fourth ventricle; *Vs*, superior vermis of the cerebellum; *VV*, valve of Vieussens; *III*, root fibers of the third nerve; *V*, trigeminus; *Vc*, descending motor root of the fifth; *Vds*, descending sensory root of the fifth; *VI*, root fibers of the sixth; *VII*, *rVII*, root fibers of the facial; *VIIg*, knee of the facial; *VIIIv*, vestibular.

quality the loss of sensation to pain, heat, and cold caused by a lesion in the spinal cord. (See Plates X and XI.)

Thus when a lesion of the bulb interferes with sensation of pain, not only may the skin be insensitive to prick, but the readings of the

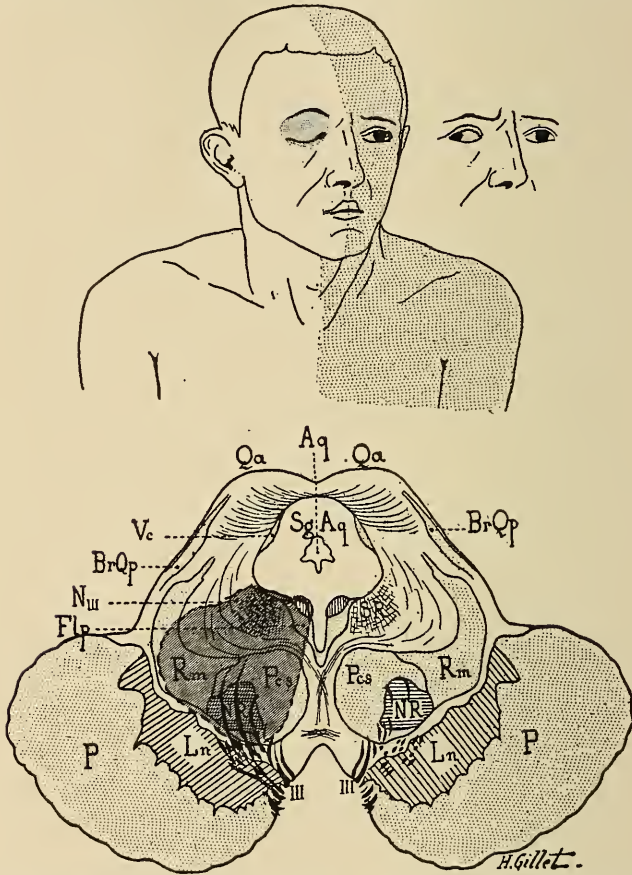
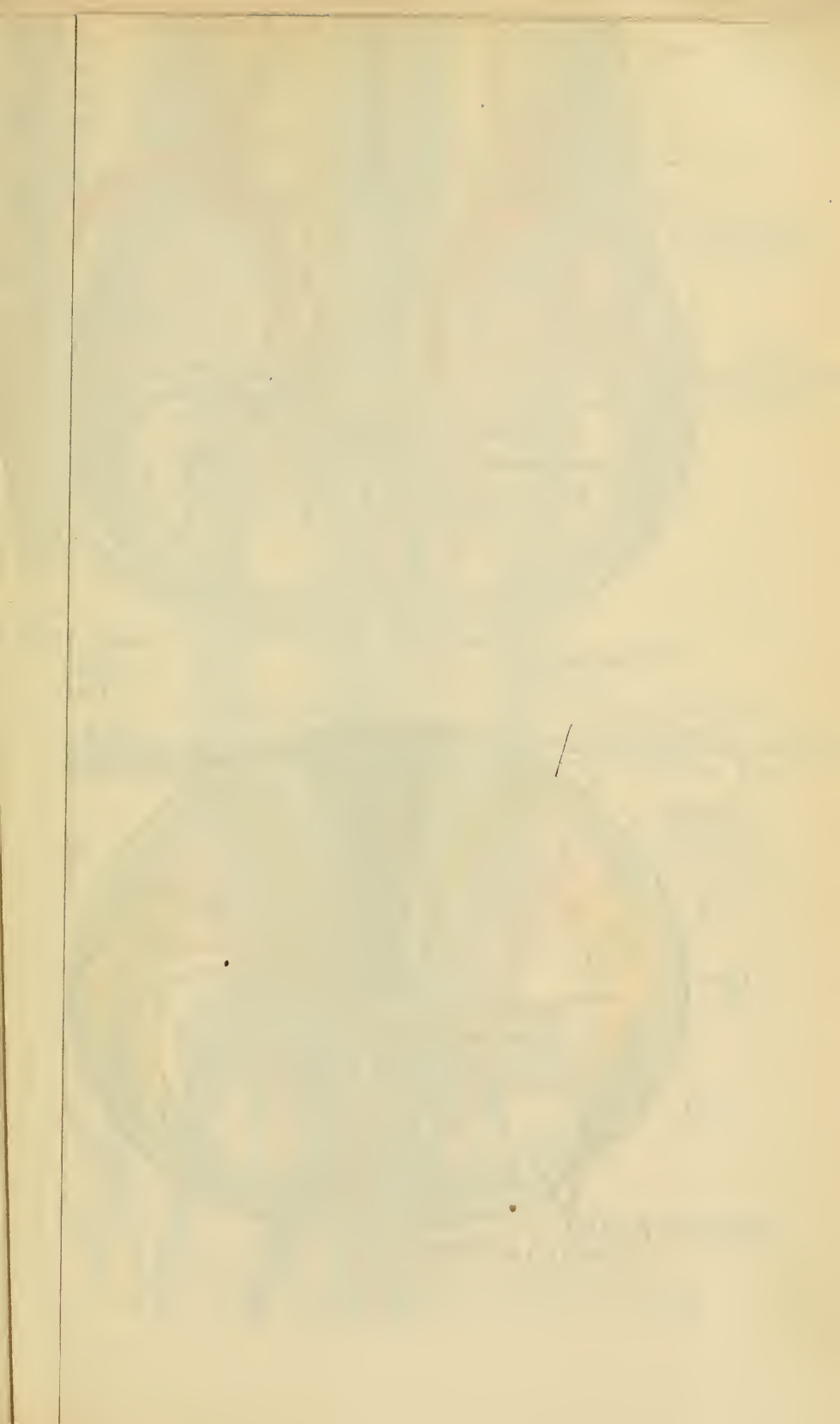


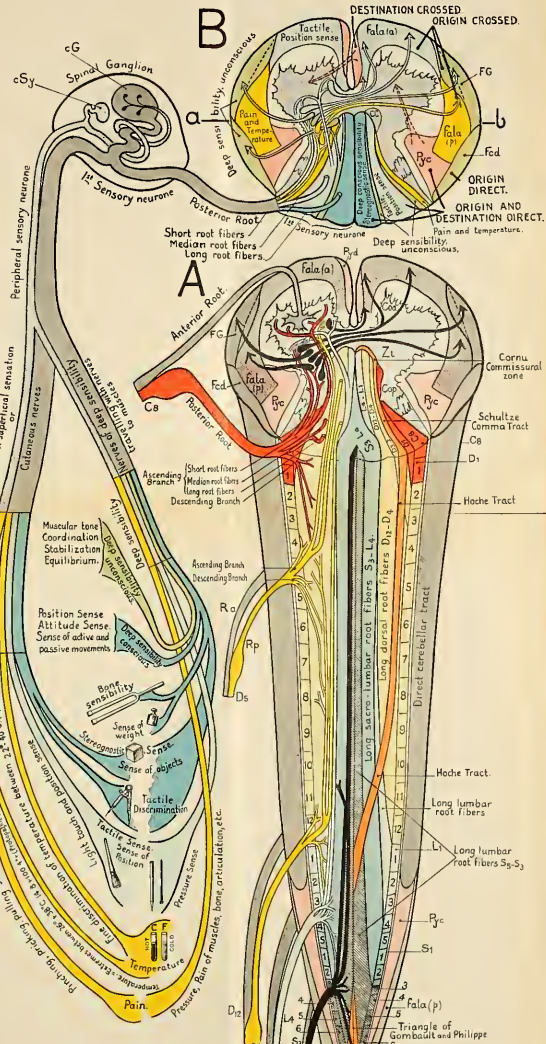
FIG. 195.—Posterior peduncular syndrome. Contralateral Benedict's syndrome. Hemianesthesia with choreo-athetoid movements and alternate paralysis of the third (III) nerve by reason of lesion of the right pontine tegmentum.

On the *left* side, contralateral hemianesthesia of the extremities and of the face from lesion of the secondary crossed sensory pathways (*Rm* and *SR*). Choreo-athetoid movements and at times tremors from lesion of the red nucleus and of the superior cerebellar peduncle (*Pcs*) below the decussation.

On the *right* side paralysis of the third nerve (*III*), with ptosis, external strabismus (non-resistance to external rectus), with or without mydriasis and pupils immobile to light and convergence, depending on the extent of the lesion of the root fibers of the oculomotorius (*III*). (Dejerine.) (See Chart, No. 6, p. 390.)

pressure algometer may be raised on the analgesic side. In the same way the affected area of the body may be insensitive to all degrees of heat, and to all stimuli capable of evoking normally a sensation of





Explanation of Plate XI

The First Sensory Neurons and the Origin of the Secondary Sensory Pathways of the Afferent Column of the Spinal Cord.

Made of extension of the posterior roots *Ca, Da, De, Dn*. Clashes the spinal nerve. Ascending and descending branches, short, medium and long root fibers. Composition of the posterior columns by the posterior root fibers of the lower sacral, lumbosacral, lumbal and lower cervical. The mixed fiber tracts of the root and endogenous fibers of the posterior columns, the nucleus commissural axis, nucleus tract of Scholtes, Heubner's tract, and nucleus tractus of Combarius and Philippe. In the figure A separate in a way the pathways of the sensitive tracts in Plate X. The colors correspond to those in Plate X. They indicate the location occupied in the posterior columns by the dorsal root fibers (Sho) (red and black), the lumbosacral fibers (SLa) (blue, blue), the lumbal fibers (La-La) (pink blue), the dorsal fibers (Da-Dn) (yellow) and the cervical fibers (Ca-Cn) (red).

Figure 21 shows the course of the pathways devoted to the different types of superficial and deep sensation in the peripheral nerve (sensory and sympathetic fibers), in the anterior root fibers and in the posterior root fibers and anterolateral columns of the spinal cord. The colors indicate the functions of the different sensory fibers. Pain and temperature sense travel in these colored fibers; tactile sense in those colored pale blue; in the dark blue fibers stereognosis sense, tactile discrimination of two points, the idea of weight, of body resistance to touch, vibration, deep conscious sensibility of position in space, of the attitude of the members, and of active and passive movements; in green the fibers conveying deep unconscious sensibility underlying muscular tension, coordination, formation of movement, and of the maintenance of static and dynamic equilibrium. The specific sensation of pain, temperature, touch, sense of localization, of tactile discrimination, and of stereognosis perception are discussed in the periphery and following the course of the superficial and deep nerves, while the perception of weight of body vibration, deep conscious and unconscious sensibility are functions of the deep sensory nerves (arterial, muscular, tendon, osseous, profuse, etc.). Pain and temperature sensations follow the course of the sympathetic fibers: the two categories of these, sympathetic and somatic, enter into the composition of the nerves of superficial and deep sensibility and participate in the terminal sign, hypodermal and muscular contracture—the Pralax, Miesner, Galva-Miesner, Engel, Thelax, Tomelox, etc., bodies. In the spinal cord a qualitative grouping of sensations takes place. Fine and temperature sensibility (yellow) enter the spinal cord by short root fibers and are transmitted to the spinal nucleus by the fibers of the posterior segment of the ascending anterolateral column (Sho, p) on the opposite side of the cord. Tactile impressions (blue) follow the course of the median root fibers and are transmitted to the optic thalamus by the fibers of the anterior segment of the ascending anterolateral column (Sho, a) which are concentrated after the site of emergence of the anterior root. Deep unconscious sensibility (green) is transmitted to the cord by the short and median root fibers, receive collateral stimuli from long root fibers, and give to the cerebellum by the regular component of the secondary sensory pathway of the cord—direct cerebellar tract and Gowers' tract, this latter is made up of fibers of crossed origin. As to deep conscious sensibility (dark blue) it follows the course of the long root fibers of the posterior columns of the cord and enters a secondary pathway only at the level of the neck of the medulla. A horizontal line (H), which indicates the base of the posterior column of the cord, is a posterior portion which passes above all the direct pathways ending or terminating in the homolateral side of the spinal cord and tracts of which determine homolateral symptoms, and an anterior portion which remains for the most part the crossed pathways which take their origin or which terminate on the gray substance of the opposite half of the root and trunks of which give rise to contralateral lesions. (Dejerine.)

Ascending and descending branches, short, medium and long root fibers. Composition of the posterior columns by the posterior root fibers of the lower sacral, lumbosacral, lumbal and lower cervical. The mixed fiber tracts of the root and endogenous fibers of the posterior columns, the nucleus commissural axis, nucleus tract of Scholtes, Heubner's tract, and nucleus tractus of Combarius and Philippe. In the figure A separate in a way the pathways of the sensitive tracts in Plate X. The colors correspond to those in Plate X. They indicate the location occupied in the posterior columns by the dorsal root fibers (Sho) (red and black), the lumbosacral fibers (SLa) (blue, blue), the lumbal fibers (La-La) (pink blue), the dorsal fibers (Da-Dn) (yellow) and the cervical fibers (Ca-Cn) (red).

cold. Here, however, in the bulb, in distinction to lesions of the cord, the grosser form of pain and discomfort may traverse other paths if the usual ones are closed, whereas in the cord all painful impulses are blocked by a unifocal lesion.

In the bulb, moreover, all three forms of sensibility may be affected together or any one may escape or be alone involved.

These impulses of pain, heat, and cold all run up in the neighborhood of the fifth nerve nucleus, and in cases of occlusion of the posterior inferior cerebellar artery the paths are usually implicated. This same accident may occasion a dissociation of the impulses underlying the appreciation of posture and passive movement from those concerned with spatial discrimination.

A summary of the findings which may occur in the lesions which cut off the sensory pathways between the nuclei of the posterior columns and the optic thalamus has been stated by Head and Holmes as follows:

1. The impulses for pain, heat, and cold continue to run up in separate secondary paths on the opposite side of the nervous system to that by which they entered. They receive accessions from the regrouped afferent impulses from the nerves of the head and upper part of the neck.

Although these paths are frequently affected together they are independent of one another, and any of the three qualities of sensation may be dissociated from the others by disease.

2. Lesions of the spinal cord tend to diminish simultaneously all forms of painful sensibility, but with disease of the brain stem the gross forms of pain and discomfort may pass to consciousness, although the skin is analgesic. This applies not only to painful pressure, but to the discomfort produced by excessive heat.

3. The impulses concerned with postural recognition part company with those for spacial discrimination at the posterior column nuclei. Up to this point they have travelled together in the same column of the spinal cord, but as soon as they reach their first synaptic junction they separate. Above the point where they enter secondary paths, the power of recognizing posture and passive movements can be affected independently of the discrimination of two points and the appreciation of size, shape and form in three dimensions.

4. It would seem as if those elements which underlie the power of localizing the spot touched or pricked become separated off from their associated tactile impulses before they have actually come to an end in the optic thalamus. The long connection of localization with the integrity of tactile sensibility is here broken for the first time.

All these changes are preparatory to the great regrouping which takes place in the optic thalamus.

The analysis of the motor pathways is equally involved. For economy in space and for convenience of reference the various syndromes are here tabulated.¹ (See Plate I.)

¹ Kindness of Dr. F. Tilney in collaboration.

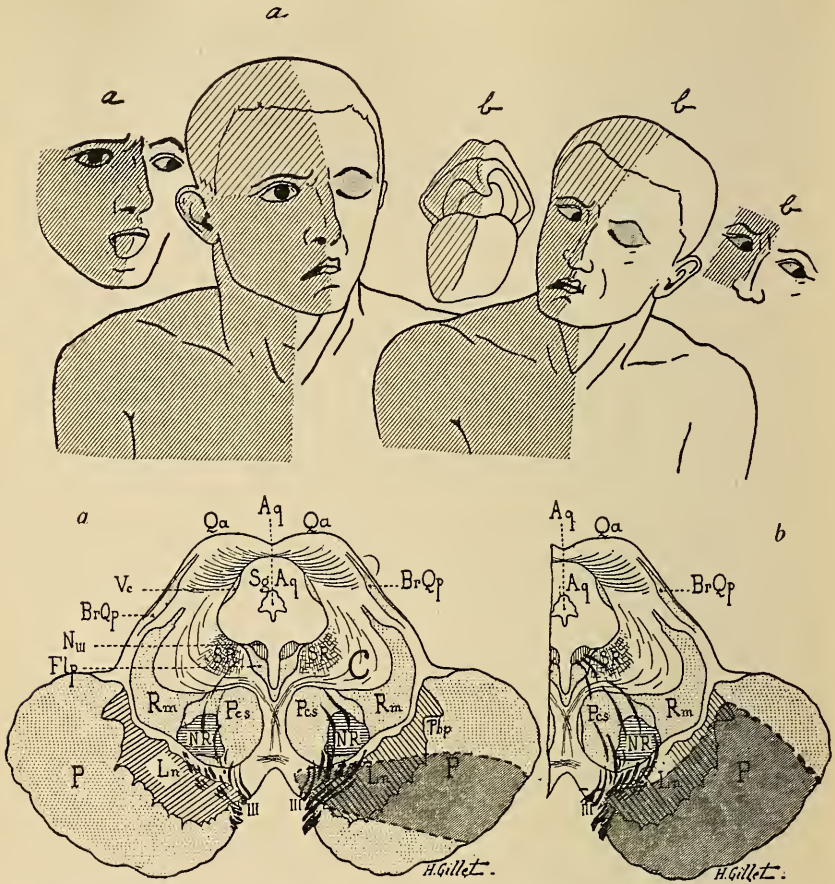


FIG. 196.—Anterior peduncular syndrome. Weber's syndrome. Superior alternate hemiplegia. Produced by cerebral peduncle and III root fiber lesion.

On the *right* side, contralateral hemiplegia of the trunk and extremities from lesion of the corticospinal pyramidal fibers. Facial hemiplegia inferior branches, hemiparesis of the tongue from lesion of the corticonuclear fibers of the hypoglossus. In (a) the lesion involves only a portion of the corticospinal and corticonuclear fibers. In (b) the lesion includes all of the pyramidal corticospinal and corticonuclear fibers, even those which in this region form the superficial and deep pes lemniscus fibers. (See Plate I.)

In addition to the symptoms common to (a) and (b) indicated in the illustrations (b) also shows a conjugate deviation of the head and the eyes from a lesion of the cortical oculomotor fibers destined to go to the nuclei of the oculomotorius and abducens (III-VI); difficulties in mastication from lesions of the motor corticotrigeminal fibers; difficulties in swallowing, of phonation, and of articulation from lesions of the corticonuclear fibers of the spinal vagus.

As a result of the predominance of the antagonists of the sound side (left) in (a) and (b), there can be observed a deviation of the mouth (facial), and of the jaw (masticators) toward the left, a deviation of the tongue to the paralyzed side (action of sound genioglossus) and in (b) further a conjugate deviation of the head and of the eyes toward the left (action of the external branch of the spinal accessory and of the levator ocular fibers) and a deviation of uvula toward the left (spinal vagus).

On the *left* side there is a direct paralysis of the third (III) nerve with ptosis, divergent strabismus, with or without mydriasis and pupils which do not react either to light or convergence, according to the grade of destruction of the root fibers of the oculomotorius (III). (Dejerine.) (See Chart, No. 5, p. 390.)

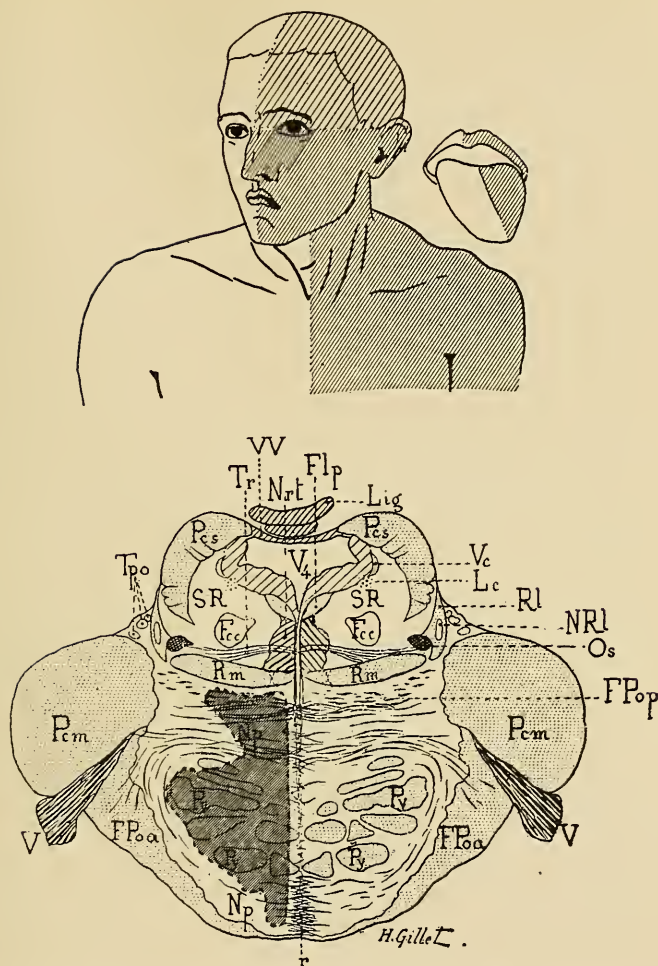


FIG. 197.—Anterior cephalic pontine syndrome. Hemiplegia of cerebral type due to thrombosis of the upper part of the basilar trunk. The lesion is unilateral, occupying the cephalad part of the pons, or the anterior portion of the right pons destroying there the corticospinal pyramidal fibers, the corticonuclear fibers of the facial, the masticators, and the hypoglossal. It does not involve the tegmentum nor the root fibers of the cranial nerves.

On the left side there is a crossed hemiplegia of the trunk and extremities with contractures and exaggeration of the reflexes. Left inferior facial hemiplegia, mild superior hemifacial paresis, slight widening of the palpebral fissure, slight drooping of the external border of the left eyebrow, slight impairment of independent closure of the eyes; hemiparesis of the masticators, especially of the internal pterygoid. Hemiparesis of the tongue. Integrity of electrical reactions.

By reason of the predominance of the muscles on the sound side the lips and jaw are drawn to the right and on protrusion of the tongue it points to the paralyzed side due to action of the right genioglossus. (Dejerine.)

ANALYSIS OF SYMPTOMS RESULTING FROM LESIONS AFFECTING THE PEDUNCULAR PATHWAYS.

Level of lesion.	Topography of lesion.	Structures involved.	Etiology.	Symptoms.	Special names and remarks.
1. Cortical	Motor area of cerebral cortex.	Pyramidal cells of Betz, irritative and destructive.	Thrombism, embolism, hemorrhage, tumor, tuberculous meningitis, encephalitis, meningo-encephalitis, traumatic meningeal hemorrhage. Same as above.	Contralateral hemiplegia; convulsions (Jacksonian) common; aphasia frequent, intelligence often disturbed; may be monoplegia.	Cortical hemiplegia.
2. Subcortical	Centrum ovale or corona radiata.	Pyramidal cell axons while passing through medullary substance of brain.	Same as above.	Contralateral hemiplegia; Jacksonian convulsions rare; monoplegia rare; intelligence seldom disturbed.	Subcortical hemiplegia.
3. Capsular	Genu and posterior limb of internal capsule.	(1) Corticonuclear; (2) corticospinal contingents of pyramidal system.	Hemorrhage most common. Thrombism embolism, tumors, cysts, and abscesses. Same as above.	Contralateral hemiplegia; convulsions, aphasia, and mental disturbance absent.	Capsular hemiplegia.
4. Capsulothalamic	Genu and posterior limb of capsule, with postero-inferior part of thalamus.	(1) Corticonuclear; (2) corticospinal contingents of pyramidal system with (3) thalamus.	Same as above.	Contralateral hemiplegia; hemianesthesia; hemiatetosis; sometimes hemianopsia; pain in affected parts.	Capsulothalamic hemiplegia; thalamic syndrome of Dejerine.
5. Peduncular (a)	Basal.	(1) Pyramidal fibers; (2) pes lemnisci profundus and superficialis; (3) emergent III nerve fibers.	Same as above.	Contralateral hemiplegia involving face, tongue, arm, and leg; ipsilateral ophthalmoplegia.	Basopeduncular hemiplegia; Weber's syndrome; hemiplegia alternans superior (Fig. 196).
6. Peduncular (b)	Tegmental.	(1) Superior cerebellar peduncle; (2) fillet; (3) red nucleus; (4) pes lemnisci; (5) emergent III nerve fibers.	Same as above.	Contralateral hemichorea-athetosis; hemitremor and hemianesthesia; ipsilateral external ophthalmoplegia.	Tegmentopeduncular hemianesthesia; Benedict's syndrome; hemiplegia alternans inferior (Fig. 195).
7. Pontile (a) cephalic	Basomesial.	(1) Corticospinal fibers; (2) corticonuclear to V, VII, and XII nerves.	Hemorrhage, embolism, thrombosis, tumor, or solitary tubercle, syphilis.	Contralateral hemiplegia; hemipropoplegia; hemiparesis; masticatory hemiparesis; lingualis.	Cephalic pontile basal hemiplegia (Fig. 197).
8. Pontile (b) cephalic	Mesial, basal, bilateral.	(1) Pyramids of one side complete; (2) pyramid of other side partial; (3) corticonuclear to VII and XII nerves on both sides.	Thrombosis of basilar artery.	Complete hemiplegia of one side; partial hemiplegia of other side; paralysis of both sides of face; paralysis of both sides of tongue with dysarthria.	Cephalic pontile mesial basal bilateral hemiplegia.
9. Pontile (c) cephalic	Basoarterial, disseminated.	(1) Fasciculus longitudinal posterior (internuclear oculoogyric fibers), (2) pyramidal; (3) corticonuclear to VII and XII nerves.	Multiple foci, multiple sclerosis, syphilis.	Contralateral hemiplegia; contralateral paralysis of VII and XII nerves; contralateral fixed conjugate deviation in extreme lateral position; cannot move eyes out of this position; looks toward paralyzed limbs.	Cephalic pontile basoarterial (disseminated) hemiplegia, with the syndrome of Foville (internuclear type).

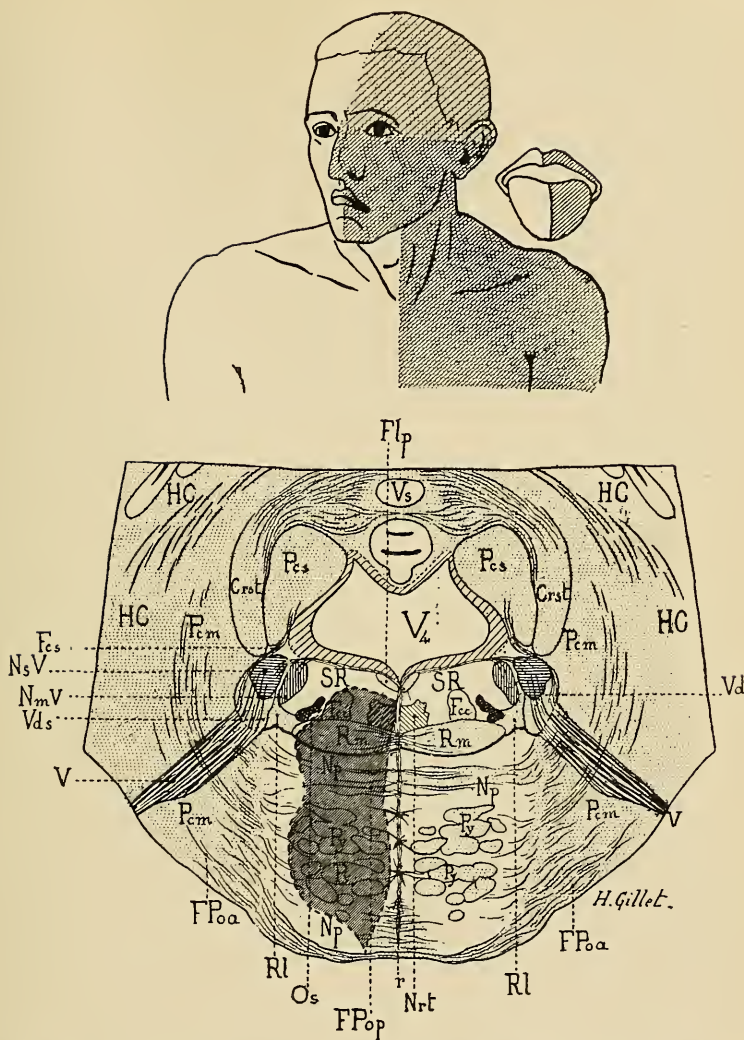


FIG. 198.—Anterior and posterior internal pontine syndrome. Thrombosis of the middle portion of the basilar artery involving the right anterior, mesial basotegmental portion. The lesion not going as far as the motor and sensory nuclei and avoiding the posterior longitudinal fasciculus. There is contralateral hemiplegia on the left side, of the cerebral type, of the trunk and extremities and of the inferior facial by reason of the lesion of the corticospinal pyramidal and facial fibers (*Py*). Mild hemiparesis of the upper facial, hemilingual paresis, and hemiparesis of the masticators (deviation of the chin to the sound side by implication of the aberrant pyramidal corticolingual, and corticotrigeminal (motor) fibers which travel in the median lemniscus (*Rm*) and the tegmentum (*Sr*)). (See Plate I.)

There is contralateral hemianesthesia for all kinds of sensibility but above all of the sense of position by lesion of the median lemniscus (*Rm*) and of the reticular formation of the tegmentum (*Sr*). There is no alternate-paralysis, the lesion not involving any of the sensory or motor cranial nerve nuclei. (Dejerine.) (See No. 11 in Chart, p. 392.)

Level of lesion.	Topography of lesion.	Structures involved.	Etiology.	Symptoms.	Special names and remarks.
10. Pontile (d) cephalic	Basotegmental.	(1) Pyramids; (2) cortical oculoogyric fibers; (3) cortical cephalogyric fibers; (4) corticonuclear fibers to VII and XII nerves.	Thrombosis.	Contralateral hemiplegia; ipsilateral conjugate deviation of eyes (patient looks to side of lesion); head inclined toward side of lesion; contralateral paralysis of tongue and face.	Cephalic pontile basotegmental hemiplegia, with the syndrome of Foville (oculoogyric and cephalogyric type).
11. Pontile (a) middle	Mesial basotegmental.	(1) Pyramid; (2) corticonuclear to V, VII, and XII nerves; (3) fillet.	Thrombosis of middle portion of basilar artery.	Contralateral hemiplegia and contralateral paralysis of face, tongue, and masseters; contralateral hemianesthesia involving all sense qualities, but especially posture sense.	Middle pontile mesial basotegmental hemiplegia (Fig. 198).
12. Pontile (b) middle	Lateral basotegmental.	(1) Motor and sensory V nucleus; (2) lateral portion of fillet; (3) superior cerebellar peduncle; (4) pyramidal in part; (5) corticonuclear fibers to V nerve.	Thrombosis, hemorrhage or tubercle (?), multiple sclerosis, syringomyelia, syphills.	Ipsilateral paralysis of jaw muscles, ipsilateral hemianesthesia of face, contralateral anesthesia of head, neck, arm, and leg (less here); ipsilateral hemichorea-athetosis; contralateral hemiparesis of jaw muscles.	Middle pontile lateral basotegmental hemiplegia; alternating trigeminal hemianesthesia.
13. Pontile (c) middle	Lateral tegmental.	(1) Motor V nucleus, also sensory; (2) superior cerebellar peduncle; (3) spinobulbar tract; (4) fillet in part.	Same as above.	Ipsilateral paralysis of jaw muscles; ipsilateral hemianesthesia of face; ipsilateral hemichorea-athetosis; contralateral hemianesthesia (most for pain and temperature).	Middle pontile lateral tegmental; dissociated hemianesthesia (Fig. 199).
14. Pontile (a) caudal	Transverse tegmental.	(1) Fillet; (2) F. L. P.; (3) N. VI; (4) N. VII; (5) vestibular and cochlear VIII; (6) corpus restiform; (7) descending V.	Tubercle, multiple sclerosis, syringomyelia, syphills.	Contralateral hemianesthesia; hemiataxia and lateropulsion; contralateral conjugate deviation; ipsilateral deafness and nystagmus; ipsilateral paralysis of palate and vocal cord (?); ipsilateral hemianesthesia of face, and paralysis ipsilateral of face.	Caudal pontile transverse tegmental hemianesthesia, with nystagmus and dysphonia (with syndrome of Foville) (Fig. 200).
15. Pontile (b) caudal	Basomesial.	(1) Pyramids; (2) emergent VI nerve fibers, or (3) emergent VI and VII nerve fibers.	Hemorrhage, thrombosis, embolism, tubercle, or multiple sclerosis.	Contralateral internal strabismus; or ipsilateral internal strabismus and facial palsy.	Caudal pontile basomesial; inferior alternating hemiplegia of Millard-Gubler type (Fig. 201).
16. Pontile (c) caudal	Mesial basotegmental.	(1) Pyramids; (2) fillet; (3) F. L. P.; (4) emergent fibers of VI and VII nerves.	Thrombosis of basilar artery or left medial pontine branches.	Contralateral hemianesthesia; contralateral ocular deviation (conjugate); ipsilateral internal strabismus and facial palsy.	Caudal pontine mesial basotegmental; inferior alternating hemiplegia of Millard-Gubler Foville type (Fig. 202).

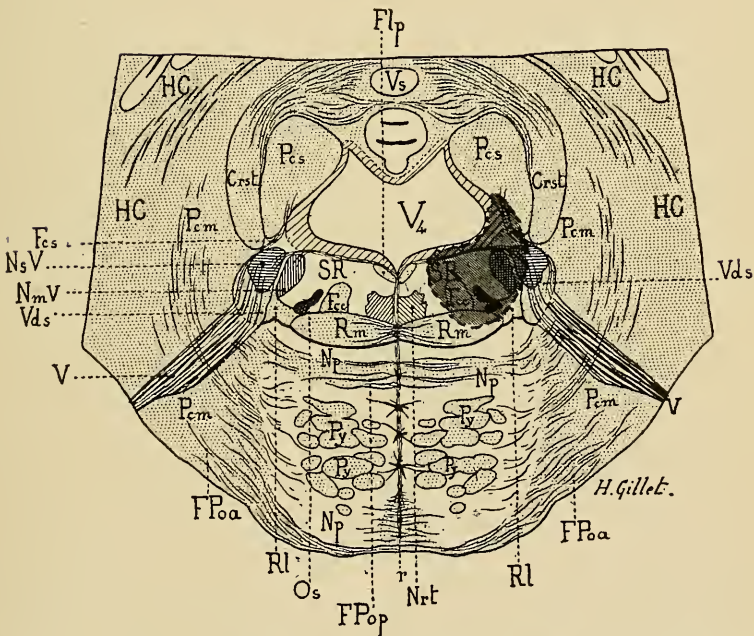
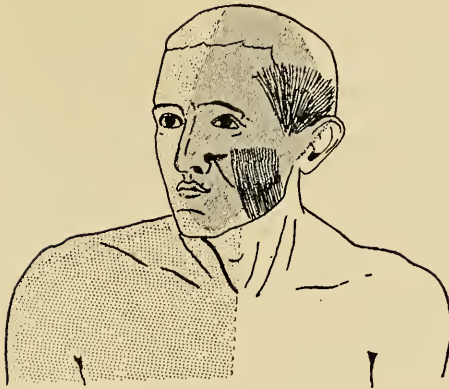


FIG. 199.—Pontine middle syndrome. Lesion of the lateral part of the left pontine tegmentum, involving the nuclei of the trigeminus, the crossed secondary sensory paths of the tegmentum, partially involving the superior cerebellar peduncle (*Pcs*) and the median lemniscus (*Rm*), and not involving the anterior portion of the pons.

On the right there is hemianesthesia of the extremities of the syringomyelic type, above all for pain and temperature sense.

On the left there is paralysis of the muscles of mastication (pterygoid, masseter, temporal) by lesion of the motor nucleus of the trigeminus. There is slight anesthesia in the trigeminus region (sensory nucleus *V*) and choreo-athetoid movements of the extremities from involvement of the superior cerebellar peduncle. (Dejerine.) (See Chart, No. 13, p. 392.)

ANALYSIS OF SYMPTOMS RESULTING FROM LESIONS AFFECTING THE PEDUNCULAR PATHWAYS—Continued.

Level of lesion.	Topography of lesion.	Structures involved.	Etiology.	Symptoms.	Special names and remarks.
17. Pontile caudal	Transverse tegmental incomplete.	(1) Fillet incomplete; (2) descending V; (3) emergent VII nerve fibers and VI nerve; (4) spinotthalmic tract; (5) Deiters' nucleus.	Hemorrhage, etc.	Contralateral hemianesthesia for heat and cold; contralateral hemianesthesia incomplete; ipsilateral internal strabismus and facial palsy; contralateral conjugate deviation.	Caudal pontile transverse tegmental; incomplete hemianesthesia with the syndrome of Foville.
18. Bulbar (a) olivary	Mesial basotegmental.	(1) Pyramids; (2) fillet; (3) nucleus XII.	Thrombosis of anterior spinal artery.	Contralateral hemianesthesia (posture sense mainly); ipsilateral paralysis of tongue.	Olivary bulbar mesial basotegmental hemiplegic hemianesthesia; alternating hemiplegia; hemianesthesia.
19. Bulbar (b) olivary	Transverse basotegmental (incomplete).	(1) Pyramids; (2) fillet; (3) XII fibers; (4) cerebellar afferents; (5) oculopupillary fibers; (6) nucleus ambiguus; (7) descending V.	Thrombosis of vertebral artery.	Contralateral hemianesthesia; ipsilateral hemiataxia and hemiasynergia; ipsilateral myosis, glossoplexia, laryngoplexia, cephalogyric paralysis, and hemianesthesia of face.	Olivary bulbar transverse basotegmental (incomplete) hemiplegia; alternating hemiplegia; hemianesthesia, including syndrome of Avellis.
20. Bulbar (c) olivary	Mesial basotegmental bilateral.	(1) Pyramids; (2) fillets; (3) emergent XII fibers.	Thrombosis of anterior spinal artery.	Bilateral hemiplegia; bilateral hemianesthesia; ipsilateral glossoplegia.	Olivary bulbar mesial basotegmental bilateral hemiplegia; hemianesthesia; alternating hemiplegia; hemianesthesia; alternating hypoglossal bilateral hemiplegia, hemianesthesia.
21. Bulbar (a) postolivary	Lateral tegmental.	(1) Cerebellar afferents; (2) spinotthalmic tract; (3) descending V; (4) N. ambiguus; (5) sometimes oculopupillary nucleus; (6) hypoglossal nerve fibers.	Thrombosis of inferior and posterior cerebellar arteries.	Ipsilateral lateropulsion; hemiasynergia; hemiataxia; hemifacial anesthesia; palatoplegia, glossoplegia; laryngoplegia and myosis; contralateral anesthesia to pain and temperature.	Olivary bulbar lateral tegmental hemiataxia, including the syndrome of Avellis.
22. Bulbar (b) postolivary	Nucleus ambiguus and nucleus accessorius spinalis.	Nucleus of X and XI nerves.	Hemorrhage, tubercle or may be traumatic injury of nerves.	Ipsilateral palatoplegia; laryngoplegia and cephalogyric paralysis of same side.	Syndrome of Schmidt.
23. Bulbar (c) postolivary	Nucleus ambiguus, nucleus spinal accessorius, nucleus hypoglossi.	Nuclei of X, XI, and XII nerves.	Same as above.	Ipsilateral palatoplegia; laryngoplegia and cephalogyric paralysis of same side, with ipsilateral paralysis and atrophy of tongue.	Syndrome of Jackson.

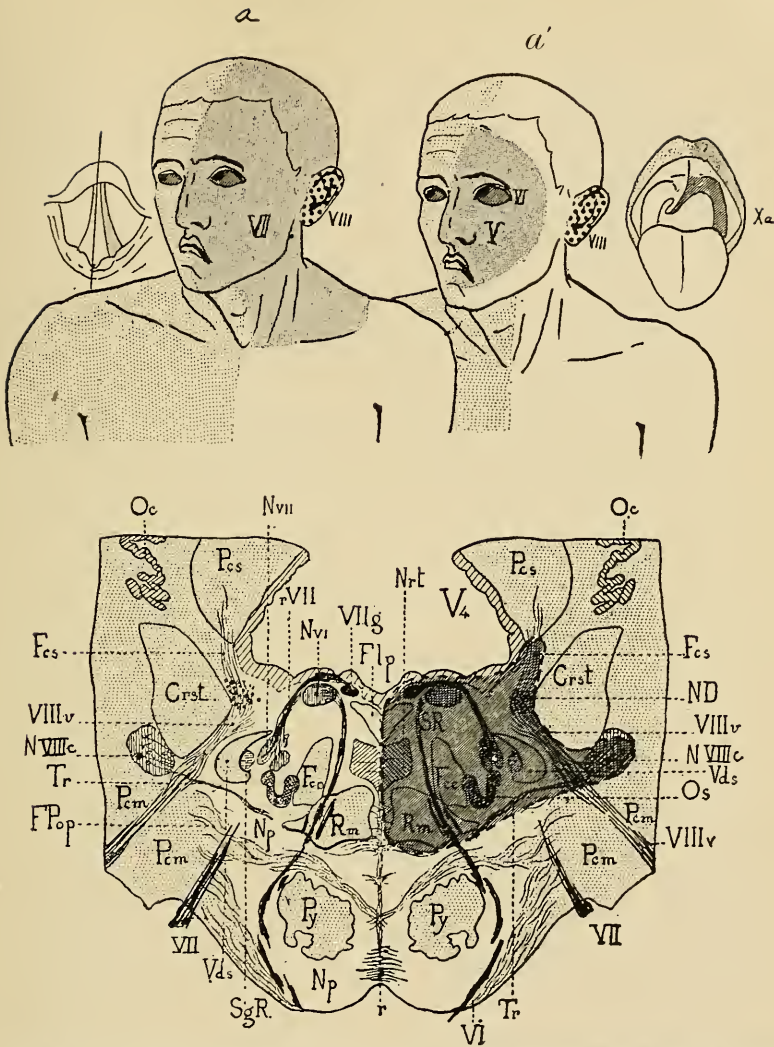


FIG. 200.—Posterior (caudal) pontine lesion. Contralateral anesthesia with alternate paralysis of the VI, VII, VIII pair and of the spinal vagus (*Xa*) from a tubercle in the inferior (caudal) portion of the left pontine tegmentum. The tubercle has pressed upon or destroyed the secondary crossed paths of the tegmentum (*SR*), the median lemniscus (*Rm*), the posterior longitudinal fasciculus (*Flp*), the nuclei and root fibers of the external rectus, of the facial, the vestibular, and the cochlear branches of the eighth.

On the *right* there is hemianesthesia of the body and of the face for all modes of deep and superficial sensibility. Cerebellar hemiataxia and lateropulsion by lesion of the cerebellar pathways.

On the *left* there is atrophic facial paralysis. (*a*) superior facial, inferior of the platysma, lagophthalmia, drooping mouth angle, cochlear deafness, vestibular nystagmus, convergent strabismus, paralysis of the lateral associated movements of the eyes toward the left by lesion of the internuclear and vestibular left oculorotary fibers. By reason of the predominance of the antagonists the patient looks to the right. There is a velopalatine paralysis and a paralysis of the vocal cords (nuclei and root fibers of the spinal vagus). Hemianesthesia of the face by lesion of the descending root of the trigeminus (*a'*). (Dejerine.) (See Chart, No. 14, p. 392.)

ANALYSIS OF SYMPTOMS RESULTING FROM LESIONS AFFECTING THE PEDUNCULAR PATHWAYS—Continued.

Level of lesion.	Topography of lesion.	Structures involved.	Etiology.	Symptoms.	Special names and remarks.
24. Bulbar (<i>d</i>) postolivary . . .	Nucleus ambiguus.	Nucleus of X nerve; fillet; spinthalamic tract.	Syringobulbia, tabes dorsalis, always central.	Ipsilateral paralysis of palate, vocal cord, and pharynx; contralateral hemianesthesia and loss of pain temperature sense (sensory troubles not essential).	Syndrome of Avellis.
25. Bulbar (<i>e</i>) postolivary . . .	Mesolateral tegmental.	Nucleus X, nucleus XII; or X and XII nerves.	Usually a peripheral lesion.	Ipsilateral paralysis of tongue and vocal cord.	Syndrome of Topia.
26. Bulbar (<i>f</i>) postolivary . . .	Mesial basotegmental (irregular).	(1) Pyramids; (2) fillet; (3) inferior cerebellar peduncle; (4) reticular formation.	Multiple lesions in distribution of vertebral artery.	Contralateral hemiplegia, and hemianesthesia, ipsilateral hemianysthergia, and lateropulsion; myosis and enophthalmia.	Syndrome of Babinski-Nageotte.
27. Bulbar (<i>g</i>) postolivary . . .	Mesial basotegmental.	(1) Pyramids; (2) fillet; (3) inferior cerebellar peduncle; (4) reticular formation.	Thrombosis of vertebral artery.	Contralateral hemiplegia and hemianesthesia; ipsilateral hemianysthergia and lateropulsion; myosis and enophthalmia.	Syndrome of Cestan-Chenais
28. Peduncular . . .	Mesial basal.	(1) Pyramids; (2) oculogyric fibers; (3) ophthalmic fibers.	Inflammation, etc.	Contralateral hemiconvulsion; in convulsive type, eyes gaze at convulsed limbs.	Peduncular convulsive syndrome of Foville.
29. Peduncular . . .	Same as above.	Same as above.	As above.	Contralateral hemiplegia; in paralytic type, eyes gaze at sound side.	Peduncular paralytic syndrome of Foville.
30. Subthalamic . . .	Mesial basal.	(1) Pyramids; (2) subthalamic corticonuclear fibers.	As above.	Contralateral hemiplegia; vertical paralysis of gaze (bilateral).	Subthalamic syndrome of Foville.
31. Bulbar . . .	Basal and tegmental.	(1) Pyramids; (2) labyrinthine oculogyric fibers.	As above.	Contralateral hemiplegia, ipsilateral oculogyric paralysis.	Bulbar labyrinthine syndrome of Foville.
32. Bulbar . . .	Basal and tegmental.	(1) Pyramids; (2) post. long. fasc.	As above.	Contralateral hemiplegia; ipsilateral oculogyric paralysis.	Bulbar internuclear syndrome of Foville.
33. Pontile . . .	Basal and tegmental.	(1) Pyramids; (2) post. long. fasc.; (3) facial nucleus; (4) fibers of VI nerve.	As above.	Contralateral hemiplegia; ipsilateral internal strabismus; facial palsy; oculogyric paralysis.	Pontile syndrome of Foville.
34. Pontile caudal . . .	Basal and tegmental.	(1) Pyramids; (2) fillet; (3) inferior cerebellar peduncle; (4) post. long. fasc.	As above.	Contralateral hemiplegia and hemianesthesia; ipsilateral tremor; hemianthetosis; hemianysthergia; hemiataxia; oculogyric paralysis.	Pontile syndrome of Raymond and Cestan.

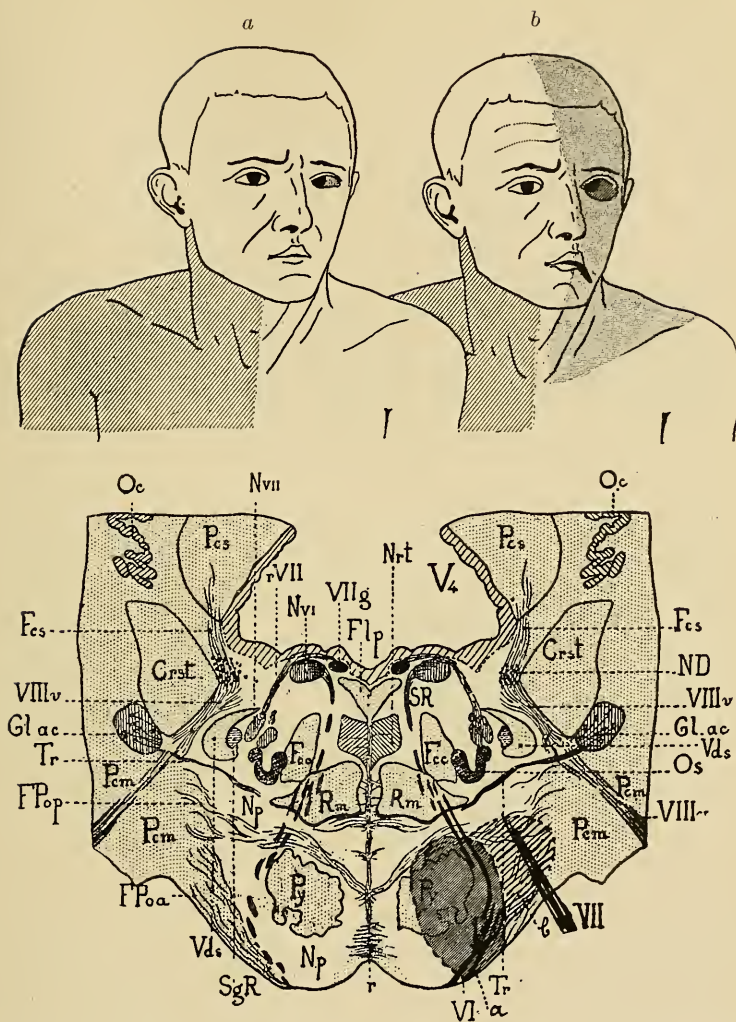


FIG. 201.—Anterior pontine syndrome. Inferior alternate paralysis—Millard-Gubler type. Contralateral hemiplegia of the trunk and of the extremities of the ponto-medullary type (without inferior facial palsy) without hemianesthesia, and without the syndromes of Foville, with alternate paralysis may be of the sixth nerve alone (*a*) or of the sixth and seventh (*a* and *b*). At this level the cortico-nucleo-facial fibers have left the pyramidal tract with the medullopontine aberrant fibers (see Plate II) and are in the tegmentum, hence the absence of the inferior contralateral facial palsy.

At *right* there is hemiplegia of the extremities without paralysis of the inferior facial.

To the *left* there is convergent internal strabismus by paralysis of the external rectus; the predominance of the antagonists carries the eye to the left and up. There is no forced position of the head, the associated lateral movements of the eyes being possible, only the left eye is involved.

In (*b*) there is added to the preceding an atrophic facial paralysis with *RD* (VII). The facial folds are gone, there is lagophthalmus, drooping of the angle of the mouth and paralysis of the elevators. (Dejerine.) (See Chart, No. 15, p. 392.)

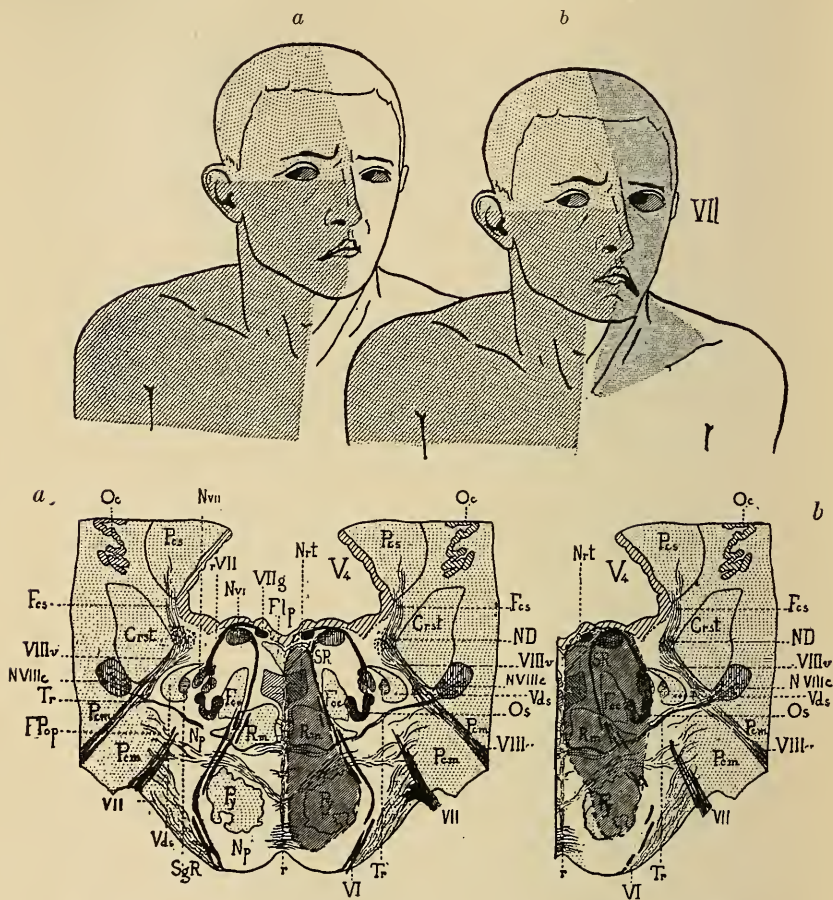


FIG. 202.—Pontine syndrome. Millard-Grubler-Foville syndrome. Hemiplegia of cerebral type with hemianesthesia of the extremities and of the head of the side opposite and alternate paralysis may be of the (VI) only (a) or of the VI and VII, (b) by thrombosis of the inferior portion of the basilar artery, in particular of the median pontine arteries of the left side. The lesion occupies the anterior part of the pons, destroys the pyramidal tract (*Py*) extends to the tegmentum, sections the median lemniscus fibers (*Rm*), the fibers of the reticular formation, the posterior longitudinal fasciculus (*Flp*), the root fibers of the VI pair. In (b) the more extensive lesion destroys in addition the root fibers of the facial and the nuclei of the VI and VII and the fibers of the lateral portion of the reticular formation.

On the *right* there is contralateral hemiplegia of the cerebral type—extremities and inferior facial, by lesion of the corticospinal pyramidal fibers and of the cortico-nucleofacial (aberrant-medullo-pontine fibers). Hemianesthesia of the extremities and of the face in (a) involving particularly the tactile sensibility and postural sense, and in (b) all modes of sensibility, these being more marked in the head than in the extremities.

On the *left* side there is paralysis of the external rectus (VI) internal strabismus, to which there is added in (b) a paralysis of the facial (VII) inferior, superior, and of the platysma myoides, lagophthalmia, drooping of the angle of the mouth, flattening of the facial lines.

Furthermore, there is paralysis of lateral movements of the eyes toward the left by lesion of the left posterior longitudinal fasciculus (*Flp*). By reason of the predominance of the antagonists the patient looks toward the right. (Dejerine.) (See Chart, No. 16, p. 392.)

CHAPTER IX.

CEREBELLAR SYNDROMES.

DEFECT or disorder of the cerebellum itself, or of its chief afferent and efferent paths gives rise to a number of fairly definite syndromes. These may be referred, with a certain degree of accuracy, to the structures involved. There are, furthermore, other disturbances, the precise nature of whose mechanisms is still unsatisfactorily analyzed, although it is recognized that cerebellar mechanisms are involved.

These syndromes may be the result of defect or disorder of the organ itself, or of its connections, or they may be due to or complicated by the position that the cerebellum itself occupies with reference to contiguous structures in the posterior cerebral fossa.

The cerebellum occupies the posterior cerebral fossa, is separated from the occipital lobes of the cerebrum by the tentorium, and rests upon the pons and medulla, forming part of the upper boundary of the fourth ventricle. It is connected with the rest of the nervous system by the anterior medullary velum, the superior, middle, and inferior cerebellar peduncles and posteriorly by the posterior medullary velum. The tela choroidea forms the posterior continuation of this latter structure, and serves as a roof to the posterior part of the fourth ventricle.

Being so intimately connected with structures in the midbrain, the red nucleus and the optic thalamus, with bulbar and pontine centers and with the cord; lying above important structures, and containing important nuclei, the dentate nucleus, Deiters' nucleus, nucleus globosus, nucleus emboliformis, tectal nuclei, etc.; with a multiplicity of afferent and efferent tracts, the possibilities of symptomatology are very numerous.

The cerebellum is the central organ, composed of groups of centers, for the coördination of the reflex system of the proprioceptors; that is, those sensory impressions coming from receptors throughout the entire body. It thus represents the entire body. These receptors receive impressions of thermal, tactile, gravity, weight, pain, sound, light, chemical and other stimuli, and by means of afferent paths, transmit them chiefly to the cortex of the cerebellum. Many of these paths are definitely known; others, particularly those connected with the viscera, are still under investigation.¹

From the cerebellar cortex, which may thus be looked upon as chiefly, if not exclusively, sensory, these impulses pass to the various nuclei of the cerebellum, and are there redistributed. It would appear

¹ Bechterew, *Die Funktionen der Nervencentra*, 1909, ii.

that these intrinsic cerebellar nuclei are mainly motor.¹ The movements of the head being chiefly referable to the intrinsic nuclei, those of the trunk and limbs to the paracerebellar nuclei.

The chief afferent or receptor paths arrive by way of the three cerebellar peduncles.

Bechterew (*loc. cit.*) describes seven paths as passing through the *inferior cerebellar peduncle*, or restiform body. These are in part:

1. The dorsospinocerebellar tract of Flechsig, which passes up the lateral ventral side of the lateral column, originating from cells in Clark's column, from the upper lumbar to the upper dorsal segments. This tract passes up through the inferior cerebellar peduncle (corpus



FIG. 203.—Hemorrhage of pons.

restiforme), and is thought to be distributed to the middle lobe of the vermis and the ventrolateral lobe of the lobus centralis² (Mott).

2. The posterior columns of Goll and Burdach send fibers from their nuclei in the medulla by way of the restiform body, dorsally and uncrossed to the inferior vermis, ventrally and crossed to the superior vermis. (Many authors claim that these bundles have no connection with the cerebellum.)

3. The olivocerebellar tract, which originates in the cord, ends in the inferior olive, from which it passes direct (?) to Deiters' nucleus, and crosses to the superior vermis.

¹ Horsley, *Functions of the Cerebellum*, Brain, 1906, xxix, 446.

² *Monatsschrift*, 1891, i, 104.

4. Vestibulocerebellar path from the vestibular ganglion, which sends its central fibers to the nucleus vestibularis, and to Deiters' nucleus in the tegmentum, and from thence to the inferior vermis. (The details of these pathways are given in the chapter on the vestibular functions, also in Plates X and XI.)

These pass up through the lateral part of the restiform body. In the median portion there are two groups of fibers: One contains sensory fibers from the cranial nerves, the trigeminus and vestibular. They form the direct cerebellar sensory path of Edinger. Other fibers connect the nuclei of the cranial sensory nerves to the cerebellum. Both of these bundles end for the most part in the tegmental nuclei. This tractus nucleocerebellaris is an indirect path.¹



FIG. 204.—Hemorrhage of pons.

In the *middle cerebellar peduncle* incoming fibers come from the nuclei of the pontine reticular formation and the ventrolateral pontine nuclei. Certain of these fibers are in relation to fibers coming from the frontal area to the pontine nuclei and form part of a frontocerebellar reflex path. (See Fig. 205.)

The fibers passing to the cerebellum by means of the *superior cerebellar peduncle* are four in number, according to Bechterew. The best known of these is Gowers' tract, which passes into the cerebellum by means of the superior cerebellar peduncle. According to Edinger and Horsley this tract is distributed to the entire cerebellar cortex. Fibers from the thalamus, from the red nucleus, and collaterals from the nuclei of the eye muscles have also been traced through the superior cerebellar peduncles.

¹ Bechterew, ii, 961.

Afferent Tracts.—It is also through the superior cerebellar peduncles that the chief connections between the cerebellum and the sensorimotor areas of the cerebrum are carried. These cerebellorubral and cerebellothalamic fibers originate in the dentate and other cerebellar

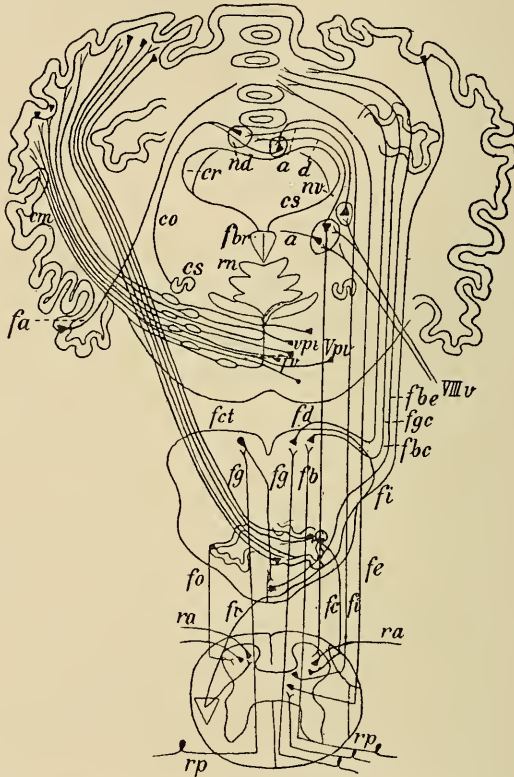


FIG. 205.—The fiber tracts of the posterior cerebellar peduncle. *ra*, anterior roots; *rp*, posterior roots; *fc*, posterior; *fc'*, ventral cerebellar tracts; *fi*, aberrant pyramidal fibers; *fG*, column of Goll; *fB*, column of Burdach; *fd*, descending tract from Deiters' nucleus; *VIIIv*, vestibularis; *fi*, fibers from lateral nuclei of medulla; *fgc*, *fbc*, cerebellar fibers from posterior column nuclei; *pv*, *vpi*, lateroventral and dorsomedian pontine nuclei; *nd*, Deiters' nucleus; *nv*, vestibularis nucleus; *d*, descending bundle of median portion of posterior cerebellar peduncle; *a*, ascending fibers of vestibularis; *fl*, fibers from Deiters' nucleus to posterior longitudinal bundle; *os*, superior olive; *fa*, fibers from flocculus to vermis; *cm*, median cerebellar peduncle; *fv*, fasciculus verticalis pontis; *fct*, central tegmental tract; *oi*, inferior olive; *fo*, olivary fasciculus. (Bechterew.)

nuclei, and passing contralaterally to the red nucleus and thalamus, end there to be continued further to the cerebrum.

The cerebellum therefore forms an important sensorimotor station in a complicated series of reflexes which work for the most part automatically. The cerebellar cortex acts as the primary reception center,

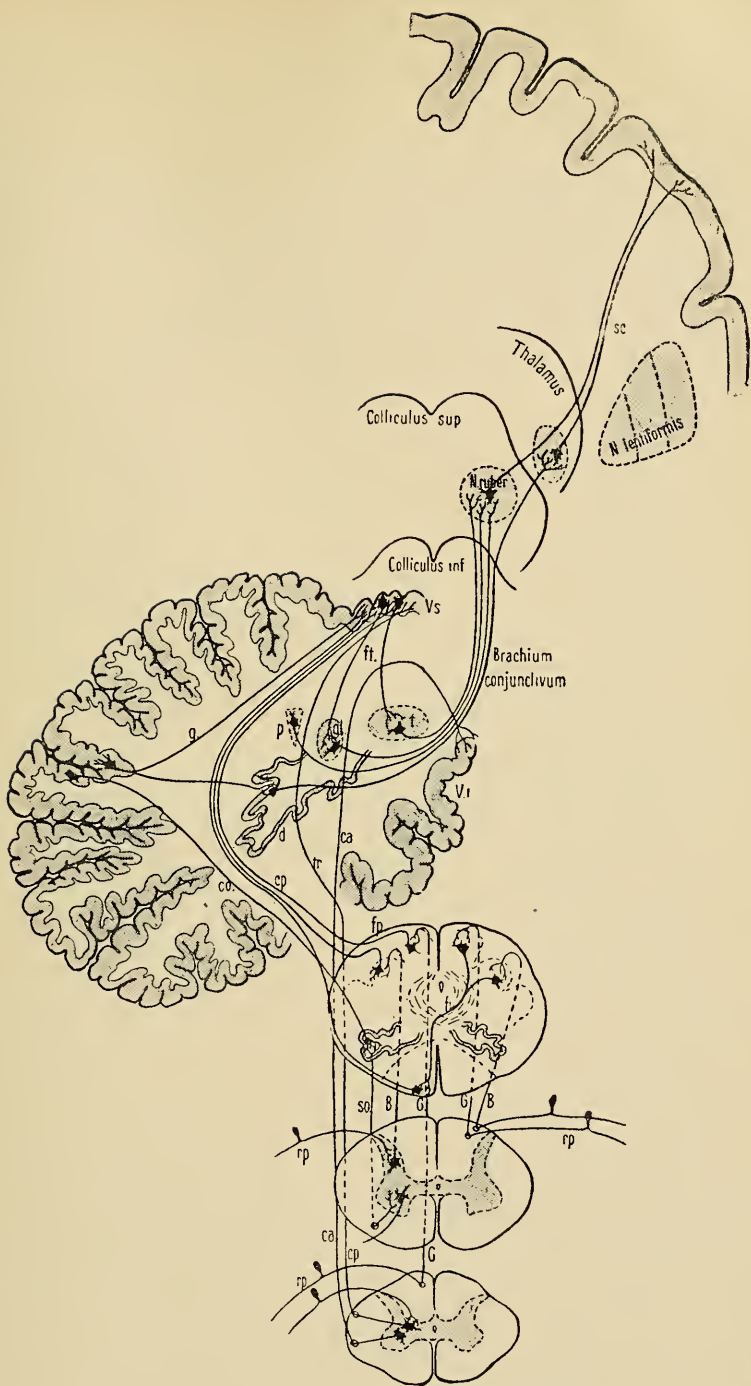


FIG. 206.—The ascending cerebellar tracts of the spinal cord and their central pathways; *B*, Burdach's column; *ca*, anterior cerebellar fasciculus to the vermis through the anterior medullary velum *co*, cerebello-olivary fibers; *cp*, common path from posterior column nuclei to vermis; *d*, dentate nucleus; *ft*, internal arcuate fibers; *fp*, posterior arcuate fibers; *ft*, fibers from vermis cortex to tegmental nuclei; *G*, column of Goll; *g*, corticovermis association fibers; *gl*, nucleus globosus; *p*, nucleus emboliformis; *rp*, posterior sensory root fibers; *sc*, subcortical paths of the brachium conjunctivum; *so*, olivospinal tract; *t*, tegmental nuclei; *tr*, fasciculus spinocerebellaris; *Vi*, inferior vermis; *Vs*, superior vermis. (Bechterew.)

the impulses coming from the cord and brain stem traversing the paths just enumerated, giving information chiefly concerning the states of tension of the muscular apparatus of the trunk, the extremities, and the head, and the states of pressure in the joints and tendons. The reflexes combine to regulate the constantly altering positions of

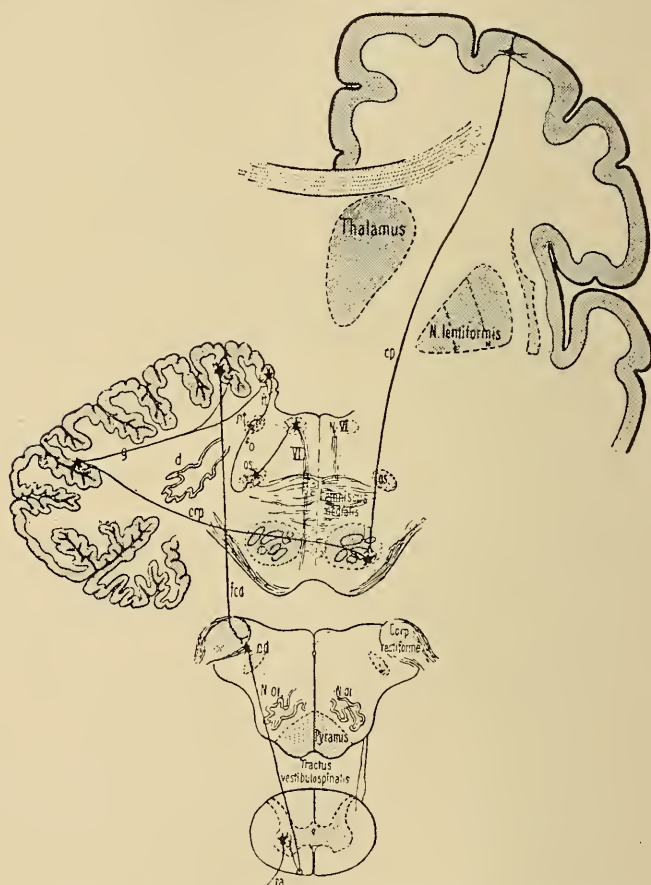


FIG. 207.—Descending pontine and cerebellar tracts: VI, root fibers abducens; cp, corticopontine fibers; crp, fibers from pons to cerebellum; d, dentate nucleus; fcd, cerebello-Deiters' fibers; fo, tegmento-olivary fibers; ft, fibers from vermis to tegmental nuclei; g, cortical association fibers; NVI, abducens nucleus; nd, Deiters' nucleus; Nol, inferior olive; nt, tegmental nuclei; os, superior olive; ra, anterior root fibers. (Bechterew.)

the entire body in space, and possibly some of the viscera. The cerebellum therefore, in this sense, acts as a regulating, coördinating organ for the estimation of the body in space. To the spinal, midbrain, cerebral reflex arcs there are also added fronto-pyramido-ponto-bulbo-cerebellar arcs which contain involuntary as well as voluntary regula-

tory coördinating impulses, acting to orient the body in practically all of its spatial relations. Cyon adds that therefore the cerebellum is intimately concerned with the mathematical sense in its objective space relations.¹

Symptoms.—The general symptoms of cerebellar disease therefore offer a multitudinous complex, the details of which, and their anatomopathological correlations, are still being rapidly added to. Among these, however, there are certain cardinal types which are capable of partial analysis at the present time, and to which attention will be directed.



Figs. 208 and 209.—The peculiar stand and gait of cerebellar ataxia. (Thomas.)

Cerebellar Ataxia.—This complex of disturbances is one of the classical signs of cerebellar disorder. It consists in a disturbance of the coördination of the larger muscular group actions, principally of the trunk and lower extremities, although the head, eyes, and upper extremities are not uninvolved. Thus, standing, walking, and the making of fine coördinated movements are interfered with in the absence of signs of distinct paralysis.

In walking, the wobbling, side-stepping gait, so well described by early French authors (Duchenne, of Boulogne) as the drunken gait, is characteristic. Incidentally it may be added they are identical,

¹ Cyon, *Das Ohrlabyrinth*, 1908.

since alcohol dissociates the sensory neurons by raising the synaptic junction threshold. In severe grades of cerebellar ataxia standing, and even sitting, becomes impossible. In milder grades one sees the same type of disturbance in many choreas, in paralysis agitans, in general paresis, multiple sclerosis, etc.

A partial study of the cerebellar gait has shown two characteristic trends of disturbance:¹ first, staggering (lateropulsions) toward the affected side, at times forward or backward, according to location of lesion in vermis (or dentate nucleus). The patient feels as though shoved to one side and in the attempt at restitution overcorrects (asynergia of Babinski) and thus sways; second, the entire orientation in space is influenced and the patient's movements, as a whole, swerve in the direction of the affected side. (Menagery movements, as seen in whirling white mice; tumbling movements, as seen in the tumbler pigeon.)

Conscious attempts at correction (frontocerebellar paths) produce the larger zig-zags in the general course of the progression. Forward and backward movements have their special localizing signs to be spoken of. From the anatomical considerations these ataxias may result from involvement of the spinocerebellar paths (Flechsig, Gowers—vestibular systems), as in the Friedreich and Marie ataxia group; from involvements in the cerebellum itself (tumors, cysts, agenesis, sclerosis); in affections involving the superior cerebellar peduncles—from bulbar and pontine involvements of these paths, and also from implication of the cerebello-rubral, cerebellothalamic, and frontocerebellar paths.

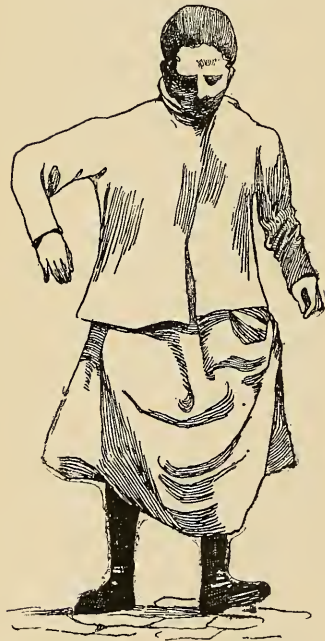


FIG. 210.—Cerebellar gait attitude.
(Thomas.)

Typical cerebellar ataxias are thus seen in some frontal tumors, with classical intoxication gait.

Chiefly associated with ataxias of the trunk and the lower extremities in standing, walking, etc., are the bilateral or unilateral (usually homolateral) ataxias (dysmetrias) of the upper extremities. The individual muscular activities are illy coördinated, both as to space and time, the desired act only being arrived at after several trials and errors, as in walking. Visual aid, or its lack, has little or no influence on the ataxia, as tested by the finger-nose test and finger-finger test, and the

¹ Stewart and Holmes, *Brain*, 1904.

ataxia is a constant one, non-increasing, as in an intention tremor (with varying graduations, for certainly the intention tremors of multiple sclerosis are often due to interference with cerebellar mechanisms).



FIG. 211.—Test for dysmetria in cerebellar disturbances.



FIG. 212.—Showing cerebellar dysmetria.

Adiadokokinesis.—Originally described by Babinski as characteristic of cerebellar disease, this symptom, consisting of a disability in

the performance of rapid movements involving the alternating actions of agonist and antagonist muscles (see Examination), is not invariably found nor always clearly indicative of cerebellar disorder; yet it is so frequently found as to merit special attention. It is in part a variant of ataxia brought out by a special test. Here the timing sense that is regulated by the cerebellum is at fault. In the absence of paralysis it usually is indicative of cerebellar path involvement. It is frequently absent in extracerebellar tumors in which there are other well-marked cerebellar signs, as in frontal tumors.

Vertigo.—As the chief organ of orientation in space, severe disturbance of certain of the cerebellar reflex paths causes vertigo, which is apt to be a prominent and a fairly constant sign. The vertigo is of a rotatory character. The patient may not only feel himself revolving in space, but objects may go around from right to left or from left to right; more rarely the vertigo has an up or down character. Each of these two characters is to be closely inquired into. Here the chief lesions are connected with the vestibular paths, as the labyrinth is the chief cephalic ganglion in the whole proprioceptive system, of which the cerebellum constitutes the coördinating center. Thus labyrinthine disease itself, as well as disease of its extracerebellar or intracerebellar paths may give rise to the symptom. By means of the specific tests devised by Barany (see chapter on Examination) a separation of labyrinthine diseases of extracerebellar origin is usually possible.

Further, enough experience has accumulated (Stewart and Holmes, *loc. cit.*) to show that, in general, objects rotate from the diseased to the well side for intracerebellar as well as extracerebellar affections, whereas the subjective sense of rotation is usually from the diseased to the well side in intracerebellar involvement, and the reverse in extracerebellar involvement of the paths. This generalization, a little too broad, is in need of further study, and of more accurate localization.

Nystagmus.—A fourth sign, rarely absent in cerebellar path disturbance, is nystagmus. It is also closely related to the vestibular reflex system, and may result from extracerebellar involvement as well as intracerebellar implication of the paths.

True vestibular nystagmus is almost invariably accompanied by vestibular vertigo and ataxia. Vestibular nystagmus itself is usually modified by the position of the head, hence every person with nystagmus must be examined with the head in the three planes; a patient with vestibular nystagmus tends to rotate within the plane of the nystagmus, and in the direction opposite to that of the quick nystagmus movement.

A patient with vestibular nystagmus then, who bends his head forward at 90 degrees will rotate in a direction directly opposite if he bend his head backward 90 degrees. The laws of intracerebellar nystagmus, apart from actual vestibular disease, remains to be investigated (con-

jugate deviations, skew deviations, and other eye displacements are to be interpreted in the light of forced movements having their analogies to nystagmus, and are considered in the chapter on Midbrain Disease. See illustrations of conjugate palsies; also in chapter on the Eye Paths. See plate of oculorotary and cephalorotary mechanisms (Plate VIII).

Closely associated with disorder of the vestibular system are pain in the muscles of the neck, nausea, vomiting, amblyopias, and loss of consciousness.

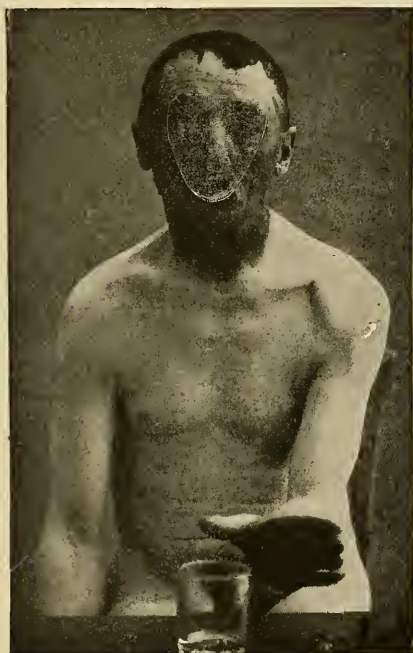


FIG. 213.—Asynergia of Babinski developed on attempting to take hold of a glass. The fingers are held very far open. (Thomas.)



FIG. 214.—Asynergia of Babinski. (Schaller.)

Cerebellar Hypotonus.—Palpation of the muscles, testing of resistance movements, and looseness on the performance of passive movements, reveal a type of muscular hypotonus in cerebellar affections which is usually homolateral. This hypotonia, or atonia, is usually accompanied by normal or even exaggerated tendon reflexes in contrast to that of peripheral neuritis or tabes. One feature of this hypotonus noted by Stewart and Holmes is striking. If a resistance to a definite movement be suddenly relaxed, in the normal flexing of the arm, for instance, there is a sudden flexor-jerk, followed by an extensor

recoil. In a cerebellar hypotonic reaction the flexor-jerk is excessive, and is rarely followed by a recoil.

Asthenia.—A paresis or asthenia, usually homolateral, is closely related to cerebellar hypotonus. Its presence in cerebellar disorder has usually been interpreted as due to a lesion of the pyramidal tracts by contiguity; unquestionably, however, it is a true cerebellar symptom. It is chiefly present when the cerebello-vestibulo-spinal and rubrospinal tracts are affected. Since the rubrospinal tract is to be interpreted as an auxiliary to the pyramidal tract, the different opinions of various authors may find a common adjustment.

Cerebellar Asynergia.—Described by Babinski¹ as a special symptom of cerebellar disease, this symptom is in reality one of the components of cerebellar ataxia, but in the anteroposterior plane, rather than in the lateral planes. It consists in the patient's inability to balance himself, whereby his legs either walk away from under him, or he pitches forward without their following. It is a severe grade of retro-pulsion and propulsion, as seen in paralysis agitans, and due in both instances to similar pathological foundations.

Cerebellar Fits.—Originally described by Jackson² as tetanus-like convulsive seizures, with characteristic holding of the body in extended rigid position.

Forced Movements.—These are present in the neck muscles, muscles of the eyes, and appear as irritative or as defect symptoms, due to disease of the hemispheres or of the middle cerebellar peduncle. (See Diseases of Midbrain.)

Speech Disturbances.—Dysarthrias usually indicate the same type of ataxia as found in other muscles of the body, adiadokokinesis. They are usually present with defects of the cerebellum, and may indicate general defect of the entire apparatus, disease of the bulbocerebellar tracts or pressure upon the bulbar nuclei from contiguous new growths.

Chief Syndromes.—In discussing disorders of the cerebellum it is convenient to take up first affections of the peduncles, although very rare, then of the cerebellum itself, and finally diseases of intracerebellar location, which latter occupying the posterior fossa implicate the cerebellar mechanisms, and those of its contiguous structures, the pons, medulla, and fourth ventricle.

Inferior Cerebellar Peduncle—Corpus Restiforme.—Isolated disease of this structure is rare. Pressure due to bulbar and pontine disease is not uncommon. The most characteristic symptoms are modifications of the eye movements, convergent and divergent strabismus, even skew deviation, forced positions, turning of body toward the site of lesion, and vertigo, with tendency to fall in the direction of the side of the lesion. The chief mechanism involved is the cerebello-vestibulo-spinal tract.³ (See Midbrain Lesions, 14, 19, 21, 26, etc.)

¹ Rev. Mens. Int., May, 1909.

² British Med. Jour., November 4, 1871. Reprint, Brain, 1906, p. 425.

³ Adler, Die Symptomatologie der Kleinhirnerkrankungen, Wiesbaden, 1899, has collected the cases.

Lesions of the Middle Cerebellar Peduncle.—These cause rolling movements of the body on its vertical axis, skew deviation of the eyes, Magendie-Hertwig syndrome, consisting in one eye being higher than its mate. The patients behave as though they had bilateral vestibular disease, causing the rolling motions; the eye symptoms depend on lesions of the fasciculus anteromarginalis, fibers to the posterior longitudinal bundle, and interference with the fibers to the abducens nucleus.

There are few uncomplicated cases on record. Pontine lesions often give rise to symptoms from implication of the middle peduncle. (See Midbrain Syndromes.)

Lesions of the Superior Cerebellar Peduncles.—Isolated lesions of these peduncles are rare. The symptoms are usually choreic, or paralysis agitans-like tremors on the same side of the lesion—possibly due to implication of the cerebello-rubro-spinal bundle in the tractus cerebello-tegmenti. Forced positions of the head to the side of the lesion have been described. Ocular implications rarely occur, although nystagmus has been observed. (See Midbrain Lesions.)

Lesions of Cerebellum Itself.—The most important of these are agenesis or aplasias, scleroses or atrophies, hemorrhage, softening, inflammation, abscesses, and tumors.

Aplasias of Cerebellum.—These are congenital, and represent a vast array of different conditions; total lack of cerebellum, absence of the lateral (in old sense) lobes, absence of vermis, unilateral loss, irregular defects, and general congenital smallness of the cerebellum and cerebrum. A consistent symptomatological grouping is not yet possible. Mingazzini¹ has attempted it. With the newer studies in localization by Horsley, Bolk, and others the entire study of cerebellar representation will see marked advance in the near future.

Mingazzini's grouping of the conditions is as follows:

1. Pure unilateral agenesis and atrophies.
2. Pure bilateral agenesis and atrophies.
3. Cerebellar atrophies associated with
 - (a) Disease of the cerebrum.
 - (b) Disease of the spinal cord.

1. *Unilateral loss* of a lateral (in old sense) lobe may be present without any symptoms according to present developed modes of testing. Few of these cases have been tested by more recent methods. In certain instances unilateral atrophy has been associated with epileptiform convulsions, or retropulsion, when the superficies of the affected hemisphere is atrophic; if the atrophy involves a part of the vermis, slight motor signs, such as slowing of the gait, have been observed.

2. *Pure Bilateral Agenesis.*—In some of the cases reported no symptoms have been observed (old cases). The commonest symptoms are

¹ Monatsch. f. Neur. u. Psych., 1906, xviii, 76.

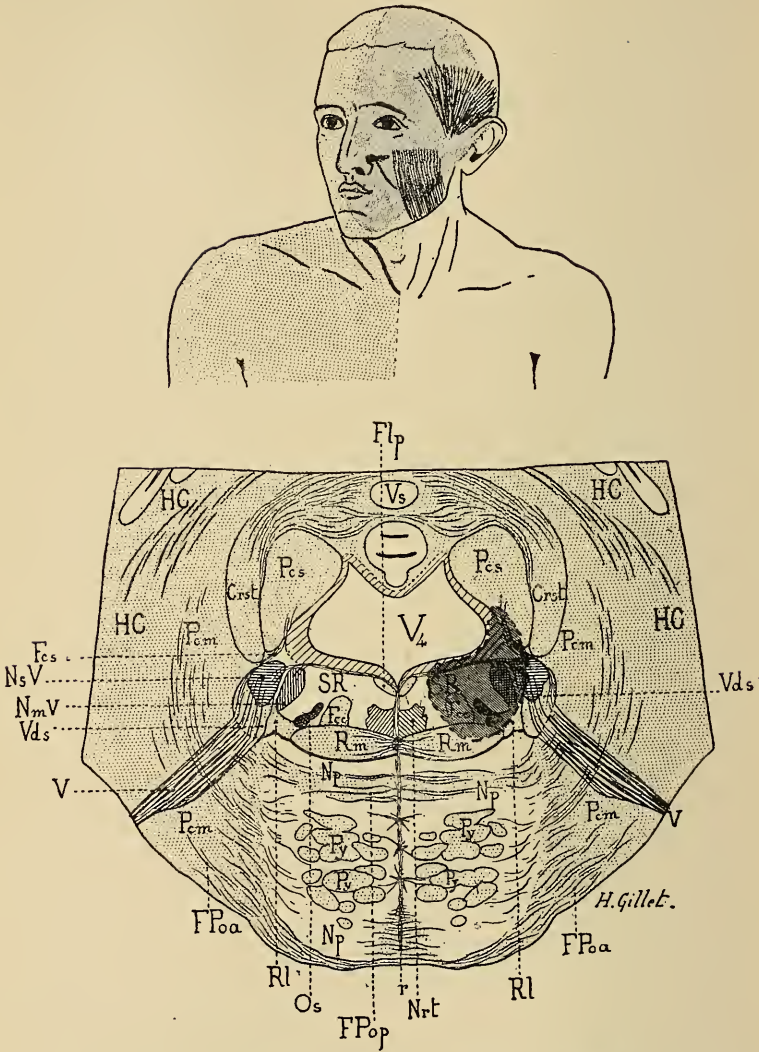


FIG. 215.—Pontine middle syndrome. Lesion of the lateral part of the left pontine tegmentum, involving the nuclei of the trigemius, the crossed secondary sensory paths of the tegmentum, partially involving the superior cerebellar peduncle (*Pcs*) and the median longitudinal (*Rm*), and not involving the anterior portion of the pons.

On the right there is hemianesthesia of the extremities of the syringomyelic type, above all for pain and temperature sense.

On the left there is paralysis of the muscles of mastication (pterygoid, masseter, temporal) by lesion of the motor nucleus of the trigemius. There is slight anesthesia in the trigemius region (sensory nucleus *V*) and choreo-athetoid movements of the extremities from involvement of the superior cerebellar peduncle. (Dejerine.) (See Chart, No. 13, p. 392.)

difficulty in standing and walking. The patient, in high grades of atrophy or aplasia, is unable to stand, or sometimes even to sit; in the milder grades the station is wobbly, the feet placed far apart, and walking is possible only with assistance. The gait is then the classical drunken stagger. There is marked asynergia of the trunk and lower extremities. Tremors, ataxias, incoördination (asynergias) of the upper extremities are also present. Hypotonus, muscular weakness, slow, irregular, hesitating or explosive speech are also present. Nystagmus may or may not be present, there is usually adiadokokinesia, the knee-jerks are usually normal, or even slightly exaggerated at times, even in the presence of hypotonus. Bilateral atrophies show similar symptoms.

It is evident that until the newer knowledge regarding cerebellar localization is coördinated with the older and newer findings the studies which have appeared up to the present time will lack precision.

Combined Aplasias of the Cerebellum and Brain.—Combette's (old period) patient, with absolute absence of the cerebellum had from birth epileptiform attacks, was imbecile, had weakness of the muscles of neck and extremities, was able to walk but fell often. Many of these patients are idiotic and imbecile, and show similar symptoms to those enumerated in the previous paragraph.

Mingazzini includes the olivo-ponto-cerebellar atrophies here, but these are discussed later.

Holmes¹ calls these cases "congenital smallness of the central nervous system, with cerebellar symptoms."

A number of conditions may be grouped here. Some of Marie's so-called hereditary cerebellar ataxias are best referred here. Irregular staggering gait, Romberg, disorder of speech, nystagmus, and ataxias of limbs are the chief symptoms. These patients have shown small cerebellums with apparently intact tracts in cerebellum and cord.

Olivo-ponto-cerebellar Atrophy.—This type was described by Thomas and shows a fairly definite syndrome. Anatomically there is atrophy of the cerebellar cortex, of the bulbar olive, and of the gray matter of the pons. There is total degeneration of the middle cerebellar peduncles, partial degeneration of the inferior cerebellar peduncles, and a relative integrity of the cerebellar nuclei. It is not necessarily hereditary, familial nor congenital. It comes on at an advanced age and progresses slowly. Clinically there is great defect in equilibration in standing and walking, drunken gait. Romberg is absent. Some irregular intention tremor, usually nystagmus, and also scanning speech is present.

*Cerebellum and Cord Atrophies or Aplasias.*²—Here also a motley group is on record. These cases will vary greatly in proportion to the varying degree of the lesion in the cerebellum and in the cord. Marie's hereditary cerebellar atrophies belong here. Some authors

¹ Brain, 1907, p. 546.

² Holmes, Brain, 1907, loc. cit., for literature.



FIG. 216.—Scoliosis of Friedreich's ataxia.



FIG. 217.—Friedreich's ataxia, showing the permanent extension of the great toe.

are disposed to refer Friedreich's disease to this group also, and to claim that between these two disorders one finds every gradation from the Friedreich type, with mostly cord and little cerebellar change, to Marie's type, with more cerebellar and little cord change.

The chief signs here¹ are ataxias of the leg, arm, facial, ocular, head, laryngeal, and pharyngeal muscles. Cerebellar reeling, no Romberg. Later palsies and contractions. Exaggerated tendon phenomena. In most of these cases the disorder is predominantly in the cord.

In other cases with degeneration of the spinocerebellar tracts with normal or only small cerebellum one finds patients with staggering gait, scanning, explosive, slow speech, nystagmoid jerkings of the eyes, muscular cramps, fatigability of muscles, normal or exaggerated knee-jerks. As mentioned, Friedreich's disease properly belongs to this rubric.

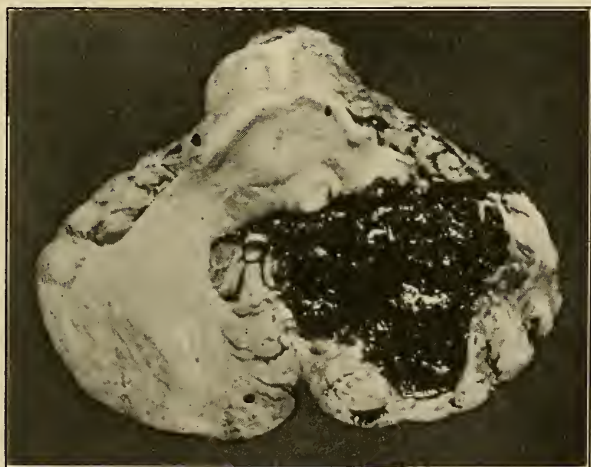


FIG. 218.—Hemorrhage of cerebellum. (Larkin.)

Primary Parenchymatous Degeneration.—Holmes² has described this condition. It usually sets in about middle age and progresses slowly. Staggering or reeling gait is an early symptom, then asynergia of the upper extremities, and later hesitating, scanning, or explosive articulation, nystagmus, tremor of the head and limbs. Tendon reflexes are normal or slightly exaggerated. No clonus, no Babinski. Sphincters intact and normal psyche. Most of the cases have shown a familial character.

*Hemorrhage of Cerebellum.*³—Cerebellar hemorrhage is probably extremely rare. Its symptomatology will depend largely on its size and the location of the effusion. The superior cerebellar artery is

¹ Brown, *Brain*, 1892, xv, 250.

² *Brain*, 1907, p. 466.

³ Starr, *Medical Record*, May 2, 1906.

oftenest involved, thus implicating certain portions of the dentate nuclei. Extension into the fourth ventricle is to be borne in mind.

The onset of the symptoms is usually sudden, or preceded by pain in the back of the head, with slight giddiness, or forced position of the head. There is usually marked vertigo, recurring on attempts to move, and usually persisting in non-fatal cases. Cerebellar gait, forced position of head, depending on location, nystagmus, especially on lateral movements. Operative interference is generally useless.

*Cysts of Cerebellum.*¹—Cysts and cystic tumors are not always distinguishable. Together they form a small part of tumor formations in the cerebellum (5 to 10 per cent.). The symptoms of cerebellar cysts are practically identical with those of cerebellar tumor, but the operative outlook is much better.

*Cerebellar Tumors.*²—Before discussing the subject of tumors of the cerebellum proper and their symptomatology a word may be said regarding the question of cerebellar localization. The researches of Bolk, and others³ in comparative anatomy, and of Horsley⁴ have served to make fairly certain that both in the cortex, and in the intrinsic nuclei there are definite localizations, the former with reference to sensory representations from different portions of the body, the latter with reference to motor representations.

With reference to sensory representation, studying practically only the terminations of Gowers' tract, Horsley⁵ concluded that there was no evidence of differentiation of the cerebellar cortex into localized receiving stations for the impressions (muscular, arthritic) which ascend from the arm, trunk, or leg muscles, joints, etc., respectively. This author holds that the results of the work of Bolk and others did not guard sufficiently against lesions of the adjacent nuclei.

While this may be true for the distribution of Gowers' tract, it is not true for the distribution of the olivocerebellar tract. Stewart and Holmes⁶ have shown that fibers from certain portions of the inferior olive pass to definite regions in the contralateral cerebellar cortex. The function of these olivocerebellar paths is still in question. As to definite localization for other receptor paths (chemical, etc.) exact knowledge is absolutely wanting at the present time.

So far as localization of motor functions in the nuclei is concerned, this seems to have received definite confirmation by the work of Horsley (*loc. cit.*).

Cerebellar tumors are relatively frequent. Almost one-third of the brain tumors occurring in childhood and adolescence are of cerebellar origin. In order of frequency one finds glioma, tubercle, sarcoma, and fibroma.

The chief general symptoms of cerebellar tumors are headache,

¹ Williamson, Review of Neurology and Psychiatry, March, 1910, for literature.

² Stewart and Holmes, Brain, 1904, xxvii, 522.

³ See Van Rynberk, Ergebnisse d. Physiol., 1907.

⁴ Brain, 1908.

⁵ *Ibid.*, 1909.

⁶ *Ibid.*, 1908.

usually severe, occipital or frontal, and apt to be confined to a sagittal plane; papilledema and later optic atrophy, which is rapidly

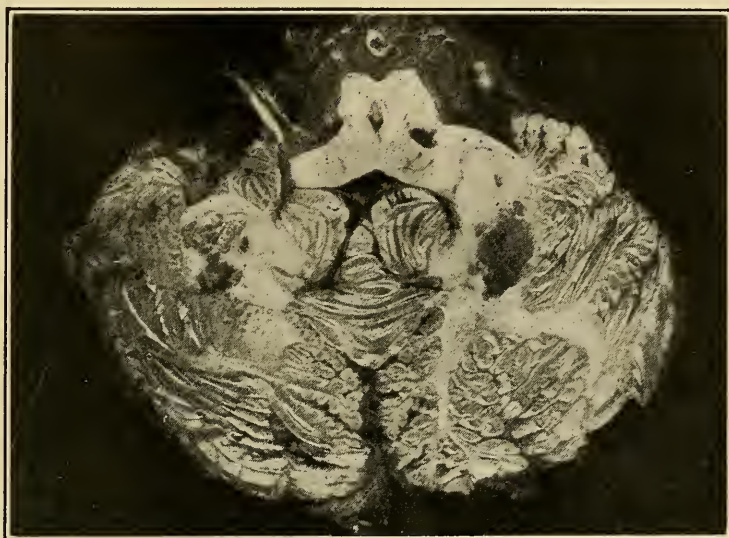


FIG. 219

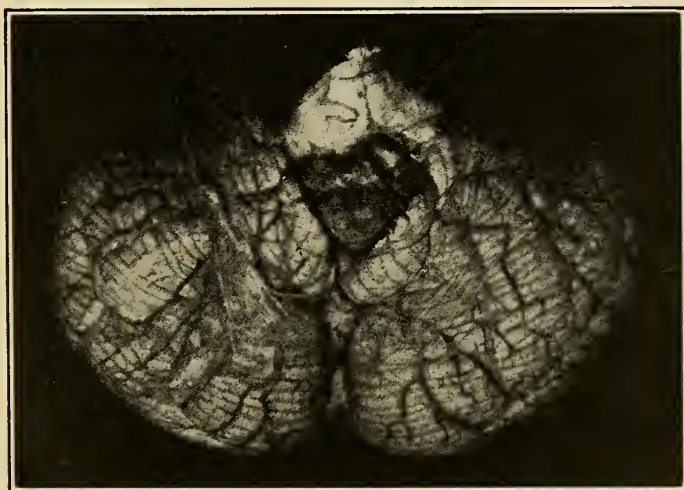


FIG. 220

progressive; vomiting, vertigo, and tenderness to percussion over the occiput. Tumors in the hemispheres, not involving the central tracts or the intrinsic nuclei, may give rise to no localizing symptoms. But

there is usually an implication of these contiguous structures with added symptoms.

These are the classical cerebellar syndromes of gait, and attitude, asynergia, ataxia, and adiadokokinesia of the same side, with hypo-



FIG. 221.—Tumor of cerebellum. (Goodhart.)

tonia of special character already described, and motor paresis of the same side. To these are usually added nystagmus and eye deviations



FIG. 222.—"Skew deviation" after removal of a tumor from the left lateral lobe of the cerebellum: the left eye is directed downward and inward, the right eye upward and outward. (Holmes and Stewart.)

already noted. The nystagmus is apt to be pronounced only on looking to the affected side, and is usually slower and coarser than the nystagmus of labyrinthine origin, or of involvement of the vestibular tracts. As these latter are frequently impinged upon, it is useless to

insist upon too fine distinctions in the character of nystagmus. Unless the pyramidal tracts are influenced by pressure, the arm and leg tendon reflexes are not markedly exaggerated, nor are they lost, and the Babinski, Oppenheim, Schaefer and Remak signs of pyramidal tract involvement are not present. The abdominal reflexes are usually unmodified.

As the tumors increase in size there are added symptoms due to encroachments or pressure upon contiguous structures. These are usually the signs of involvement of the pyramidal tracts, eye palsies, and of the cranial nerves—from the fifth to the twelfth. These all show on the opposite side of the body. The two lower branches of the facial are involved, the tongue protrudes to the paralyzed side, and is without atrophy or R. D. Occasionally the medulla is pressed upon and one finds all branches of the facial involved with atrophy of the tongue. Homolateral anesthesia of the cornea may be present, due to trigeminus involvement. Homolateral affections of the ears, deafness, buzzing and homolateral pain to pressure on the mastoid may aid in diagnosis. Percussion should never be neglected. Oppenheim has called attention to the cracked-pot sound often present in cerebellar tumors.

Unilateral signs are apt to pass over into bilateral signs as the pressure increases, with dysarthria, dysphagia, continued vomiting, and finally cardiac and respiratory signs.

Lumbar puncture may give important information in clearing up a diagnosis of meningitis-serosa or hydrocephalus.

Cerebellar Abscess.—These are relatively frequent, and originate mainly from middle-ear infections, either by way of the temporal lobes or the mastoid, and wounds, from trauma, which latter may have occurred a long time previously. Occasionally abscess may result from thrombi due to abscess of the lungs, ulcerative endocarditis, etc.

These abscesses vary considerably in size from that of a pea to an apple, and their development is either acute or chronic.

The characteristic symptoms are headache, usually occipital, and radiating into the neck region, producing marked stiffness of the neck, at times resembling the pain of a cerebrospinal meningitis. General unrest, nausea, vomiting and stupor are present. Temperature may be added, but a cerebellar abscess may run a course of several months without temperature. Of special cerebellar symptoms ataxia, nystagmus and rotatory vertigo are characteristic. This vertigo is usually rendered worse and vomiting is induced by movement of the body; hemiparesis and hemiasynergia are usually present on the side of the lesion, but this is not a constant sign.

By an increase in the size of the abscess, symptoms of pressure, precisely similar to those mentioned under Tumor, may develop. Papilledema, with optic atrophy, is not infrequent in large abscesses.

Brain puncture by special aspiration needles is advisable to clear

up the diagnosis in complicated cases. Lumbar puncture is useful in excluding meningitis.

Associated Posterior Fossa Complications.—*Occipital Lobes.*—These may be pressed upon anterosuperiorly by a foreign body growing upon or within the superior lobe of the cerebellum. Hemianopsia (mind blindness) may then occur.

Corpora Quadrigemina.—Ocular palsies of a nuclear character occur, first on one side, then upon the other. The oculomotor and abducens are oftener involved than the trochlear. There is less apt to be a paralysis of accommodation or changes in the pupils, and the loss of conjugate action of the eye is rarely found. Implication of the posterior corpus, and of the middle geniculate causes deafness, one- or both-sided, and if the lateral geniculate be pressed upon, amblyopia, without papilledema. Pineal tumors may cause the same picture.

Cerebral Peduncles.—If these structures are markedly impinged upon the Weber-Gubler syndrome of alternate hemiplegia and oculomotor, palsy may be found. From milder irritative pressures one obtains the Benedict syndrome, oculomotor palsy, with tremor of the opposite side. If the lemniscus is impaired there is crossed anesthesia and ataxia. (See Midbrain Syndromes.)

Pons.—Here one finds a number of syndromes added to the initial cerebellar symptoms. Crossed hemiplegia with facial palsy (Millard-Grubler) and R. D. Occasionally from a more anterior pressure there may be homolateral facial palsy without R. D.

Crossed Hemiplegia and Abducens Palsy.—Both are usually associated with hypoglossal involvements. Conjugate deviations to the opposite side of the lesion is observed in these cases when the abducens nucleus is involved. Such conjugate palsies speak more for tumors within the pons (*q. v.*). (See Midbrain Syndromes.)

Crossed hemiplegia and trigeminus involvement is occasionally found, and also alternate hemiplegias with cochlearis symptoms. Here the hearing defect is due to destruction of the intrapontine fibers of the cochlearis; or to pressure on the tuberculum acusticum.

Tumors of the Fourth Ventricle.—These may be considered here because of the cerebellar symptoms induced. These tumors are for the most part glioma, sarcoma, psammoma, carcinoma. They give rise to symptoms due to pressure on the medulla and pons and almost always cause a marked hydrocephalus with advancing stupor and confusion. Cysticercus may also be found here. Bruns has called attention to the following features. Alternating periodicity of headache, nausea, vomiting, vertigo, changes in pulse and breathing, with sudden let up of all these symptoms. The vertigo and vomiting are set up by changes in position of the head; sudden movement of the head may cause immediate unconsciousness. Cerebellar ataxia, mild nystagmus and occasionally diplopia are other signs.

One more word may be said about cerebellar tumors and their diagnosis. They may, in the presence of few signs only, be mistaken

for affections of the frontal lobes (frontocerebellar paths), parietal lobes (implications of central sensory components and of the optic thalamus).

In frontal lobe tumors special intelligence defects are usually found, if carefully examined for by the methods of Ziehen, Sommer, and Kraepelin. The tremor is apt to be very fine and rapid, hemiparesis, if present, is crossed, and shows spastic phenomena; the speech disturbance is aphemic; the conjugate deviations are irritative and not paralytic. Then anosmia, apraxia, and aphasia are often added. Skew deviations, and hypotonus are not known for frontal tumors.



FIG. 223.—Tumor (neurofibroma) of cerebellopontine angle. (Larkin.)

Parietal lobe disease only occasionally offers difficulties, while the characteristic sensory disturbances and central pains of optic thalamus involvement should exclude this structure.

*Cerebellopontine Angle Tumors.*¹—These should be discussed here because of the symptoms of cerebellar pressure and of vestibular

¹ Henschen, F., Ueber Geschwülste der hinteren Schädelgrube insbesondere des Kleinhirnbrückenwinkels, 1911, for full literature to date; also English literature, Fraenkel and Hunt, Medical Record, 1903, and Medical News, 1904; Stewart and Holmes, Brain, 1904; Weisenberg, Jour. Amer. Med. Assoc., 1908; Starr, Jour. Nerv. and Ment. Dis., 1910; Lewandowsky, Handbuch der Neurologie.

involvement. Two main types of tumor come under review. Those from the pia of the cerebellum, and those growing on or about the eighth nerve. Fibromata, myomata, and sarcomata are the most frequent.

These tumors press upon the pons and middle cerebellar peduncle and the symptoms vary slightly, according to the variations in pressure on these two structures. The eighth nerve is usually involved early; buzzing and deafness are observed. Facial palsy is usual with corneal anesthesia from pressure on the fifth; trigeminal pains are frequent. Ptosis may come on. Pressure on the cerebellum causes the typical gait and the ataxia, homolateral paresis, and hypotonus. A contralateral paresis from pressure on the pyramidal tracts usually develops. This gives the usual signs of a pyramidal tract involvement. Homolateral static tremor is frequent, and a sense of subjective rotation toward the side of the lesion.

Treatment of Cerebellar Conditions.—Gummata must be attacked by the usual antisyphilitic treatment, otherwise surgery offers the only opportunity, and this is limited to the attack upon cysts which is apt to give fortunate results. The results of operations for abscesses are improving, as are also those upon tumors. But as each case is a law unto itself, it is futile to generalize.

CHAPTER X.

PARALYSIS AGITANS, CHOREA, AND RELATED DISORDERS.

PARALYSIS AGITANS.

History.—The clinicians of the times immediately preceding Parkinson grouped the paralysis agitans cases of the present day in several different categories. Galen had noted the characteristic tremor, and the class of tremors of this kind; the “tremblement palpitant” of Preysinger was part of the earlier *palmos* of Galen. Francis de la Bœe was shrewd enough to notice the difference, afterward forgotten, between the tremor produced by attempts at motion and the tremors present while the limbs were at rest, and his term *tremor coactus*, for the tremors of paralysis agitans, was utilized up to Parkinson’s time. Juncker had also described a tremor, a paralytic-like tremor, *tremores paralytoidei*, which included some of these patients.

Not only was the tremor appreciated, but the clinicians of the eighteenth century (Gaubius, 1751), had called attention to the propulsion of these patients, and Sauvages groups them in his choreas, as *Scelotrybe precipitée* (*Danse de St. Guy precipitée*, L.).

Parkinson, in his famous thesis on the “Shaking Palsy,” London, 1817, made a synthesis of several of these conditions, and erected a new clinical form to which he gave the name shaking palsy (paralysis agitans), and gave the following short and striking description: “Involuntary tremulous motion, with lessened muscular power, with a propensity to bend the trunk forward, and to pass from a walking to a running pace, the senses and the intellects being unimpaired.”

All of the case histories cited by Parkinson were probably true cases of our present-day paralysis agitans, but the group as then understood still contained certain of the chronic choreas, and certain cases of multiple sclerosis, possibly certain thalamic cases, etc., which later clinicians have come to recognize. The chronic choreas were definitely excluded by the work of the Germain See, 1851, and the researches of B. Cohn,¹ Ordenstein,² and Charcot finally separated the multiple sclerosis syndrome.

Since the appearance of Charcot’s studies the monographs of Wollenberg, Heimann, Zingerle, 1910, and of Mendel, 1911, contain the chief steps made, showing the steadily advancing trend to regard

¹ Wien. med. Woch., No. 18.

² Thèse de Paris, 1868.

the disorder not in the light of a functional disturbance, as Charcot taught, but as an organic syndrome, and affecting either circumscribed or more diffusely, certain cerebellar and thalamic mechanisms.

Etiology.—The changes of age seem to be the most striking etiological factors. The majority of the patients are between 50 and 70—although cases of patients of 19, 15, 12, 10, and 3 years of age are recorded, some of which have been possibly faultily diagnosed, the vast majority of them being multiple sclerosis or encephalitis. Hereditary factors may play a role, probably through vascular disease. Berger, Gowers, Borgherini, Clerici and Medea and others have reported cases occurring in two generations or in more than one member of the same generation, and Erb reports that in 15 per cent. of his cases the parents or grandparents suffered from the same disease. Concerning indirect heredity, the least said the better, as the studies available are entirely too conflicting, and for the most part inapplicable.

Emotional disturbances are held accountable by many writers; it is difficult to determine here whether one is concerned with cause or effect. Sorrow, worry and emotional distress are such universal all-pervading, environmental factors, that too much stress must not be placed upon them. Sudden shock may perhaps stand in an accentuating, accidental relationship.

Trauma stands in a possibly closer relationship. It is highly improbable as a direct cause, but it may be a sufficiently exciting cause to bring the symptoms of a slumbering paralysis agitans to the surface, or those of a mild case rapidly to a severe stage.

Physical stress is a factor which, bearing upon arteriosclerosis, may be an accompanying factor in certain cases. Toxic factors of themselves are not known to play any necessary role. Their coincidental occurrence is frequently reported; the same may be said of infections. Cold, exposure to wet, and other factors are probably more accidental than vital; they may augment the action of an underlying factor, as yet unknown; they may represent purely coincidental features.

Arteriosclerosis is the chief factor in bringing about the syndrome. The central features of the syndrome are matters of localization in the implication of certain pathways by the sclerosing process.

The parathyroid hypothesis is unproved.

Symptoms.—These develop for the most part very slowly, although occasionally patients are seen who show fulminating types, and although atypical developments are known, the regularity and uniformity in the development is very striking.

The vast majority of the patients show, on close analysis, prodromal symptoms which are chiefly sensory, in contrast to the sensorimotor symptoms of the more advanced stages of the picture. The more characteristic of these prodromata are fugitive, irregular pains of a sharp, lancinating character, frequently found in the extremities first to be affected by the motor disturbances, and usually ceasing as these latter advance. Paresthesiæ are also frequent, causing sensations

of tickling, cold spots, hot spots, gastric distress—almost crises-like attacks, with diarrhea and colicky disturbances in the large intestine.

General malaise with headache, sweating, mild vertigo, palpitation, sialorrhea, anxiety, pressure of blood in the head, easy excitability, these are general symptoms accompanying many senile and presenile conditions, but are so frequently found as forerunners of the motor symptoms, and persist with such marked increase of severity throughout the disorder that their appearance is to be regarded as more than coincidental.

The symptoms of the more classical syndrome may be grouped as follows: (1) The main group of sensorimotor disturbances, varying in intensity and location in different individuals. (2) A number of sensory, vasomotor, trophic and secretory disturbances already indicated as often in part occurring as prodromes. (3) Psychological symptoms which are somewhat variable and possibly not essentially related to the disorder *per se*.

The sensorimotor disturbances are predominantly increase of muscular tonus, with rigidity and resulting contractures, and motor disturbances with tremor, compulsory gait, forced attitudes, forced movements, and loss of mimetic expression.

An increase in the muscular tonus is a most fundamental feature in the concept paralysis agitans. As a result of it there follows the rigidity, the mask-like countenance, and the contractures. The increase in the muscle tonus usually is a very early sign, although positive traces of rigidity may not appear until later. It is practically always found, whereas there are some patients who have little or no tremor¹ and yet the name paralysis agitans is properly used. Associated with the hypertonus and the rigidity there is a slowness of movement, and a steadily increasing stiffness, and also retardation of the motor and ideational impulses.

The muscular rigidity varies widely in its situation at the beginning. Practically the symptoms first become manifest on one side of the body, and the severity of the symptoms usually predominates on one side, it may be for years. In the well-developed syndrome the rigidity affects particularly the muscles of the neck and trunk; the patient assumes the bent-over attitude, such as one naturally assumes when shivering from the cold and the face is mask-like (corrugators) and staring, the eye muscles also sharing in the rigidity with Stelwag's sign.

Whereas the muscles of the neck and back are most affected, almost any group of muscles may be involved. The arms and legs are almost always implicated, and so are also the muscles of the face. Occasionally there is ptosis, or the patients open their eyes after closing them with difficulty. One of us has seen this as an initial symptom.

¹ Fürstner et al.; see Zingerle.

The patients read with difficulty because of the stiffness of the ocular movements. Ocular palsies may result—pseudo-ophthalmoplegias. One occasionally finds slow pupillary reactions. The pharyngeal and laryngeal muscles being involved, as others in the body, results in slow, difficult speech, becoming fainter and fainter as the years go by, until finally the patient, in addition to being unable to move, to dress himself, eat without help, finally is unable to talk or to swallow.

This hypertonus and rigidity, however, is not associated with the usual increased reflex signs of pyramidal tract involvement, the reflexes are either normal, or only slightly exaggerated, no clonus, Babinski, Oppenheim, etc., and the contractures may be easily overcome by passive movements, in marked contrast to the contractures

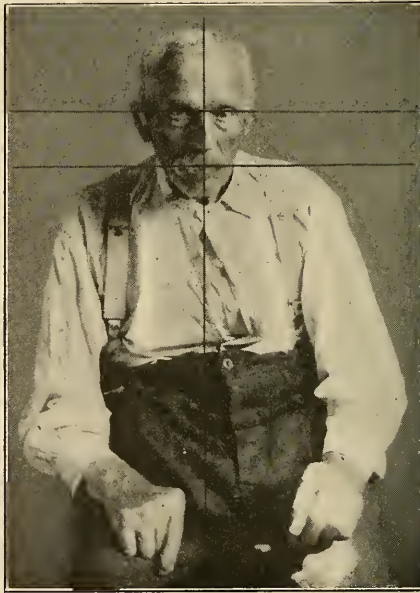


FIG. 224.—Attitude of paralysis agitans patient. (Tilney.)

of psychomotor cortical origin. The muscular power is also not so involved, the patients show muscular weakness, but not paralysis. There is a striking contrast between the strength of active movements and that of resistance movements. The former are weak, the latter rarely less than normal.

A few cases of extensor rigidity are recorded, but it is predominantly of the flexor muscles.

With the rigidity there is a feeling of tension that the patients dislike usually much more than the almost universal tremor. This causes them to feel as though they were bound. Their motor impulse seems interfered with. This may even affect their urination and defecation, and their deglutition.

Attitude.—This is that of a decerebellate rigidity in contrast to a decerebrate rigidity.

Tremor.—In the majority of the cases this objective sign seems to be the first, although, in reality, vasomotor signs precede as a rule. It was the symptom first noted by Galen. It is characterized by its uniformity and steadily increasing severity, both in point of advance of the disease, and also during the movements themselves. It is a tremor that Franciscus Sylvius first noted was present while the limb was at rest. It ceases during movement, especially if the movement is rapid, and in the beginning of the disease. In the later stages it becomes continuous, and some patients with paralysis agitans show some intention tremor. Again, a certain number of cases show little or no tremor.

The tremor is characterized by the uniformity of its excursions, which are at first small, slow, and rhythmical. They average about three to five in a second, according to the muscles involved. Tremor in the muscle of the thumb, which is often an early sign, gives rise to the well-known "pill-rolling" movements. Similarly, one has the movement of "beating the drum," and other conditions when the larger muscles of the arms are implicated. The muscles involved in the early tremors vary considerably; usually the upper extremity is involved before the lower, the hand particularly; but with the progress of the disease the tremor tends to become widespread, almost universal; most diverse localizations are on record, anything is to be expected. There is no great preponderance of one hand over the other, although there is a marked tendency to disproportion in the severity, and a hemiplegic type of onset and persistence is frequent, if not characteristic. Monoplegic types are encountered.

At first the tremor is absent during sleep, but in the advanced stages it frequently persists and constitutes one of the factors in sleeplessness which finally exhausts the patient.

Motion tends to diminish the tremor, as also does attention; emotional disturbances and cold increase it markedly. Grasping the tremulous member, touching it, or changing its position results in a temporary cessation of the tremor. The restlessness of these patients is largely dependent upon the constant and continual shifting of the body, *i. e.*, before they become too rigid, in order to obtain comfort.

Statistical studies show that tremor may be absent in as high as 20 per cent. of the cases—a much greater proportion than the rigidity which also may be absent or scarcely noticeable. These are variations in precise pathway blocking, the lesions showing slight variability in localization.

Disturbances of Equilibrium.—Propulsion, which apparently was first noted by Gaubius (1751), is one of the cardinal symptoms in Parkinson's original definition. The patient on walking tends to fall forward, and in his effort to keep his equilibrium goes faster and faster, until he either falls or stops himself by a stick or a foreign body.

Lateropulsion and retropulsion are also present. These are due either to the stiffness with the slowness of muscular movement, or to a central disturbance of equilibrium, which latter is perhaps the preferable explanation, since exquisite examples of gait disturbances are known without any marked stiffness or rigidity. In a few cases the loss of equilibrium in one direction alters that of another during the course of the disease.

Secretory, Vasomotor, Trophic Disturbances.—These make up the second category of symptoms almost universally found to a greater or less degree in paralysis agitans. As noted, many are prodromal symptoms. What relation these symptoms, all of which have some definite relation to the sympathetic nervous structures, possibly at lenticular levels, bear to the almost universally present arteriosclerosis is not yet apparent.

The most important of the secretory changes are increased perspiration—sometimes unilateral, increased salivation—one of the most distressing of the symptoms—and polyuria, with occasionally diarrhea.

Among the vasomotor changes are rushes of blood to the head, reddening of the face, cyanosis, tachycardia, acroparesthesia, with hot and cold spots, alteration of temperature—sometimes unilaterally disposed, and dermatographia, which is almost constant.

Trophic changes in the skin, such as atrophy, thickening, edema, are among the rarer findings.

Psychic Disturbances.—These probably do not constitute an essential part of the disorder, but represent almost normal psychological reactions to a most distressing and hopeless situation. Depression, anxiety, ideas of self-destruction, scornfulness, savage raillery, sarcastic pessimism, euphoric compensation and sublimation, resignation to the will of God, etc., these are but a few of the innumerable attitudes which these patients show at one or another time during their long period of almost unbearable suffering.

Parkinson said the “senses and the intellects are not impaired,” and intelligence tests bear this out. In some patients one naturally finds a senile dementia, and for most, in the later stages, the mental signs of an arteriosclerotic dementing process are present. Acute exhaustion, delirious states, often close the sad chapter; but these are not a part of the paralysis agitans.

Sensory Symptoms.—Tactile, thermal or pain disturbances are not definite. They do not constitute striking features of the disorder. The early pains are usually fugitive, and apart from the dull and most oppressive sensation due to the tension and stiffness, pain is not prominent. Irregular anesthesiæ, hyperesthesiæ, paresthesiæ are frequently found, but are so inconstant that one can say that definite sensory disturbances do not belong to the paralysis agitans picture. When present in striking fashion they are due to some complicating factors.

The *reflexes* are not markedly disturbed. Considerable variation

exists, but there is no constant picture as yet known which is pathognomonic of the condition. Reflex activities due to pyramidal tract involvement are occasionally found. Increased knee-jerks, clonus, and Babinski phenomena are at times found, but they are not constant, and represent occasional rather than essential features.

Loss of the Achilles reflex is a not infrequent symptom, the significance of which is as yet not definitely placed.

The abdominal, cremasteric, epigastric, and anal reflexes are not involved.

Other clinical findings are inconstant. The blood practically shows nothing, some anemia at times, but nothing striking; the cerebrospinal fluid is practically negative. The urine, apart from a frequent polyuria, shows no quantitative or qualitative anomalies, beyond the excessive phosphate elimination, which is indicative of the exhaustion.

Course and Progress.—Rudimentary forms are not unknown. Many senile patients show conditions closely approaching the milder grades of paralysis agitans, and intermediary stages, with muscular stiffness, slowness of movement, retardation of motor impulses, etc., are not infrequent. Certain stationary cases, non-progressing for twenty-five years, are also known, and infrequently patients make partial recoveries. These are possibly syphilitic cases showing the syndrome. But the usual course of the more frequent arteriosclerotic cases is a long, slow, and gradual progression, lasting over many years with annoyance, inconvenience, discomfort, distress, and agony until life becomes a burden. Remissions and exacerbations belong to almost every case.

Patients with little hypertonus and muscular rigidity seem to progress less rapidly, and many cases beginning in younger individuals do fairly well. Emotional disturbances act badly, almost invariably causing marked progress of the disease.

The majority of the patients get worse gradually. The increasing weakness and stiffness limits them more and more in their work, until that becomes impossible. Then the walking, to which they are unconsciously attracted, becomes more and more difficult. They are then confined to their chairs for a few years, or to bed, and finally come to be helpless invalids, who must be fed, turned over, attended to like living rigid statues, which in the presence of relatively intact intelligence, though robbed of the power of expression, reading, writing, even pantomime, constitutes one of the most ghastly afflictions in the entire realm of nervous disorders.

Decubitus, pneumonia, exhaustion delirium, and starvation, are the usual precursors to the end of a disorder whose prognosis is bad. Recoveries there are none, although stationary cases are occasionally seen.

Differential Diagnosis.—The diagnosis is rarely difficult. The attitude, gait, facial expression, and tremor are so characteristic as to stamp the patient at once. In the initial period, before the stiffness,

tremor, pulsions, etc., have developed, the diagnosis may be difficult, but after its classical development it cannot be mistaken for anything else.

Hysteria sometimes comes into review, but here the character of the hypertonus is quite different, the tremor is rarely classical, and can be more readily influenced by diversion and distraction. The exaggeration of the paralysis agitans symptom-picture is characteristic of the hysterical type.

Multiple sclerosis of the cerebellar type frequently shows the classical paralysis agitans picture, plus the evidence of pyramidal tract involvement, nystagmus, bulbar speech, etc., of this disease. It is usually present in younger individuals. Most of the so-called juvenile Parkinson cases are cerebellar types of multiple sclerosis.

Senile and presenile tremors have been mentioned.

Complicating diseases, such as tabes, hysteria, multiple sclerosis, hemiplegia, exophthalmic goitre, etc., are known.

Pathology.—A dogmatic presentation of the causes for paralysis agitans is not yet available. The trend of opinion is that it represents a senile or presenile degeneration of certain brain regions, and these are mostly contained within the cerebellar, thalamic, and lenticular mechanisms. Whether they are confined to the midbrain regions is not proved—neither are these mechanisms—but the evidence points in that direction. The increased tonus resembles cerebellar and not cerebral tonus. The rigidity, attitude, slowness of motor impulses, has its analogies in disorders of the frontocerebellar, cerebello-rubrospinal and thalamic systems. The disturbances of equilibrium are distinctly of the cerebellar type. The vasomotor, secretory, and trophic symptoms represent central sympathetic disorders which are referred with greatest probability to those thalamic nuclei other than the nuclei which are known to be the synapses of the chief sensory pathways.

The recent review of Zingerle (*loc. cit.*) brings these features to the fore. Few complete series of microscopic sections through the cerebellum and midbrain region have as yet been studied, but distinct atrophies in the ansa lenticularis, in thalamic, and lenticular regions are present in those studied by the serial section method.

The muscular hypotheses,¹ which are many, are totally inadequate, as is also the parathyroid hypersecretion theory upheld by Lundborg.

As to the nature of the process that brings about the degenerations in the regions involved, science is still in the dark. Arteriosclerosis is probably the chief factor.

Therapy.—Notwithstanding the gloomy outlook, much can be done to relieve the patients. They must be guarded against cold, and as far as possible, from emotional disturbances and mental and physical strain.

¹ Camp, Jour. Amer. Med. Assoc., 1910.

They should live in warm, dry, sunny rooms if possible, be much in the open air, eat a full, mixed diet, and possibly a minimum of purin-containing substances is needed. Alcohol and coffee may be used in moderation. Tobacco is not necessarily taboo; two to three cigarettes or cigars a day. The regime should under no circumstances be so strictly adhered to as to cause the patients, already suffering from irritating conditions, to become further annoyed thereby. Diet has relatively little power to modify the trouble and fussy dietaries are superstitious nonsense for the most part.

The greatest relief from rigidity comes from the regular use of passive movements. The Zander apparatus can be utilized. Working with carpentry or garden tools is often very helpful. Heat is grateful and helpful and passive motion combined with warm (not hot) baths is particularly gratifying. A few patients react badly to baths.

Such attempts at occupation therapy must be carefully dosed. Fatigue must be avoided. Five to ten minutes is sufficient in the early stages. Such therapy is solely of value from a psychological stand-point.

Drug therapy is at times of doubtful service in controlling the tremor. The remedies are those with marked action on incoming nerve impulses—notably the alkaloids of the belladonna group, of which hyoscin, duboisin, scopolamin, and atropin are the most available. In view of the chemical uncertainties concerning the alkaloids of this group one should obtain good products and try the different derivatives. The dosage must be tested with each case.

The analgesics, particularly in combination with salicylates, are useful in relieving the muscular soreness and pain of tension—phenacetin, aspirin, acetanilid combinations, etc.

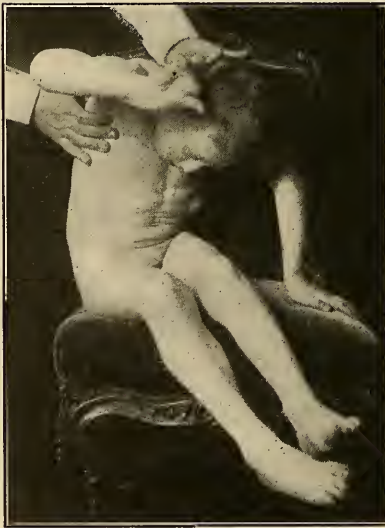
For sleep, the best form of hypnotic is not yet known. Bromides are at times available, at other times the alcohol hypnotics—trional, sulphonal, again urea substitutes, as veronal—are useful. One should avoid morphin as much as possible, particularly bearing in mind that the emotional hyperactivity may have little real feeling behind it. It is often mostly mimicry which is uncontrollable because of the motor defect.

DYSTONIA MUSCULORUM DEFORMANS.

Under this term, Oppenheim includes a peculiar syndrome, first called attention to by Ziehen as a tonic torsion neurosis. Flatau and Sterling term it a progressive torsion spasm. It is preëminently a disorder of children, most of the observed patients having been between eight and fourteen years, and almost all of the Jewish race. Apparently there are no sex differences. Oppenheim's cases did not lead him to any light on the disorder as regards heredity. Three of Ziehen's cases were brothers and sisters. Exciting or other causative factors are not known.

The illness comes on apparently gradually and subtly in one arm

or in both arms, occasionally first in the legs or in the spine, but in progression. The patient twists the spine in a peculiar fashion, tilting the pelvis, and bringing about a marked torsion of the entire vertebral axis, with lordosis, scoliosis, and tilting of the pelvis, the arms and legs moving in a peculiar manner. The mode of progression at times resembles that of *astasia abasia*. The whole musculature, when in action, is extremely stiff and hypertonic; when at rest, hypotonia is apparent. In the general attitude of the patient in walking one is inclined to regard the whole matter as one of extreme suggestibility; a psychogenic-hysteriform affair. The movements of the hip are very typical. It is tilted or thrust forward or backward in an awkward



FIGS. 225 and 226.—Attitudes in *dystonia musculorum* cases. (Flatau.)

manner. Oppenheim has likened it to a dromedary in some of the positions assumed. Walking seems to fatigue the patients greatly. They perspire, get red, and show signs of fatigue, getting out of breath, and one of our (J.) patients grunted involuntarily. One of Oppenheim's patients could walk backward better than he could walk forward. On sitting down or lying down the movements cease (Beling's case), or are much reduced in frequency and in clownishness. Ziehen's cases were at times continually in motion, and had to be kept in a special bed, against which they frequently bruised themselves by their impulsive movements. The peculiar activities come into play as soon as there is an attempt to make any voluntary movement. Writing

becomes difficult or impossible. There is no paralysis. Oppenheim speaks of a dystonia, Ziehen of a hypertonia. The movements are not athetoid nor choreic. They are wide, irregular, and yet partake of the nature of both, and at times resemble those of Huntington's chorea.

Tonic and clonic extension of the muscles, particularly of the biceps and rotators of the thigh, were marked in Oppenheim's cases. Thus there is an alternation in tonicity of the muscle.

The knee-jerks are apt to be much diminished, coming out in Oppenheim's cases only by Jendrassik reinforcement.

The relationships are difficult to state. Hysteria should be excluded. Double infantile athetosis shows a similar picture. These cases have been studied especially by Lewandowsky.¹ Cecile Vogt and Oppenheim have reported on infantile pseudobulbar palsy, which also is to be considered in this connection.² The patients apparently hold their own for some time, and but little is known of the development of the disorder. As yet no pathological reports are available. No known method of treatment seems useful. Suggestion is of no value, nor are bromides. The movements cease during sleep.

THE CHOREAS.

The choreas are due to definite but usually recoverable brain disorders, chiefly located in or involving the cerebellar static mechanisms. They are a vast conglomeration of conditions, certain trends of which have been separated out under a variety of types.

The detailed history of this sorting process would lead too far. The chief trends center about the study of the movements which are present. These are of two main forms: (a) spontaneous movements, and (b) coördination disturbances. In most choreas the two forms are present but in varying proportions.

The chief diagnostic entities which have been erected are:

1. *Chorea Minor or Sydenham's Chorea*.—The most widespread and frequent of the trends, usually found in children or young adults, and transitory.

2. *Chorea Chronica*.—A stationary form of the former, or when occurring in old age, chorea senilis.

3. *Chorea Huntington*.—A chronic progressive type with certain definite hereditary factors and one showing a vast variety of other choreic anomalies in the "non-Huntington chorea" members.

4. *Chorea degenerans of Brissaud*, occurring as a result of presenile breakdown in unstable neuropathic individuals.

5. *Chorea electrica of Dubini*, an acute, usually fatal disturbance, often occurring with epileptiform attacks, with paralysis and death.

6. *Chorea electrica of Bergeron and Henoch*, occurring in young persons, seven to fourteen years, with rhythmic lightning-like movements

¹ Deutsche Zeitsch. f. Nervenheilk., xxxix, 1908.

² Journal f. Neurologie, xviii, 1911.

of the neck, shoulders, and upper arms. It has allies in certain epileptic-like choreas.

7. *Chorea epileptica*, continuous with the preceding or related to cortical epilepsies. (See Epilepsy.)

8. *Choreas of general paresis*, in which spontaneous choreiform movements occur. (See Paresis.)

9. *Choreas of many Psychoses*.—Motility psychoses of Wernicke, chiefly schizophrenic individuals, in whom Kleist has endeavored to show an involvement of the cerebello-rubro-cortical tracts.

10. *Choreas of Congenital or Infantile Cerebral Palsies*.—(See Hemiplegia, Thalamic Syndrome.)

11. *Chorea Postaplectica. Posthemiplegic Choreas*.—(See Hemiplegia.)

12. *Chorea Thalamica*.—(See Thalamic Syndrome.)

13. *Choreas due to Disorder Involving the Superior Cerebellar Peduncles*.—(See Cerebellar Syndromes.)

14. *Choreas of Cerebellar Origins*.

15. *Chorea tabica* in tabes with choreic crises with or without pain.

Chorea Minor (*Sydenham's Chorea, St. Vitus' Dance*) is the most frequent of these disturbances of spontaneous and coördinating movements, which, occurring in children, usually recover.

Infection and maldevelopment are the most frequent etiological factors. The most widespread infecting agents are various streptococci, hence the frequent complications of infectious arthritides, endocarditis, with a mild meningitis which is usually present.

Girls are more susceptible to these infections, and hence show a higher percentage of choreic attacks.

Symptoms.—These show considerable variation, ranging from slight motor unrest and irritability to marked motor disturbances, with corresponding modifications of conduct and emotional response. The latter at times are so severe as to constitute a psychosis (choreic mental disease, *q. v.*). The motor symptoms are best grouped as pyramidal or extrapyramidal tract and cerebellar disturbances, either or both occurring in most patients. They are the results of definite irritations, occasionally of defect (diaschisis) lesions.

The *spontaneous* movements are quick and show comparatively wide excursions. In the distal extremities cramps of single muscles or groups of muscles occur, with complete rest following the movements. There is great irregularity in the affected groups; there will be one or two movements, which are followed by opposite muscle action.

In mild cases these movements are limited to the face and to single muscles. In the more severe ones the entire body musculature is involved.

The arms are flung about, the legs are wobbled and pulled, walking is impossible, the larynx, lips, and eyes are in activity. The patients gasp, snort, and groan. In the mild cases the movements may cease during sleep; in the more severe ones the movements are continuous.

Any sensory stimulus may increase these spontaneous movements. They do not behave like willed movements. Hence, extrapyramidal systems are involved.

Pyramidal Tract Disturbances.—Certain choreic, jerky movements are observed apart from the more usual ataxias and incoördinate movements. The latter diminish with rest, quiet, and relaxation; the former do not seem to diminish as muscle activity is withdrawn.

A group of minor signs appear on close examination. One of these is the Babinski hand sign. When the choreic patient extends the hand, palm down, one side, that most affected, has a tendency to sag. Or if the hands are hanging by the side, the more affected hand shows a position half-way between pronation and supination, whereas, since the normal muscle tonus is greater in the pronators, the more healthy side is held more pronated. This is in accord with the general tendency for one-half of the body to be more affected than the other. The affected side is hypotonic; the shoulder droops more. There is apt to be exaggerated flexion or extension of the arms. In the lower extremities, hyperflexions of the leg on the thigh occurs. Since hypotonicity is characteristic of underdeveloped, psychomotor integration, younger children do not bring these contrasts into relief.

The patient lying flat upon the back attempts to sit up, the arms being folded. The leg most affected is flexed on the thigh and raises from the bed, the healthy side remains flat. (Babinski thigh sign—Hoover). Similarly, the patient lying on the back raises the legs, simultaneously, the sound side rises higher than the other, or if one leg is raised and then the other, the sound side is raised higher and the choreic side also falls more readily.

The Hoover procedure by measuring the pressure of the leg on the heel during the movements just mentioned shows diminished pressure on the weak side. (See Examinations.)

Thomas showed that in choreics, particularly in the more unilateral cases, the closing of the fist of the sound side called forth an associated closure of the affected side, but not *vice versa*. Similar associated abductor or adductor movements occur in the lower limbs.

The Babinski, Oppenheim, Gordon, and Chaddock great-toe extension sign is very frequent in chorea, and should be looked for.

Strümpell's contraction of the tibialis anticus occurs when the patient lying on the back attempts to flex the leg on the thigh against passive resistance applied to the thighs. The foot assumes the equinovarus position.

The tendon reflexes, patellar and Achilles, are apt to be variable and often delayed.

These signs are all suggestive of mild types of hemiplegia, and have been collected under the title of the "Little Signs of Hemiplegia."¹ Someone or all may be present in even mild cases of chorea, especially

¹ Jelliffe, Little Signs of Hemiplegia, Post Graduate Medical Journal, 1912.

when the disturbance is at its height and particularly in the severe infectious types. Their disappearance often takes place with the stage of recovery. Mild cases may show only the most passing signs, or very mildly developed ones.

Cerebellar Signs.—These are chiefly adiadokokinesia and asynergia. The former is frequently found, especially on the more hypotonic side. It is sometimes complicated by the choreic movements.¹

Asynergia (dysmetria) is the usual choreic type of movement. It is the jerky, irregular movement of the muscles which fail to perform well-adapted movement. Thus the patients drop things, lurch, stumble or fall, spill their food, or speak in a jerky, at times mumbling, manner. They are incapacitated from writing, playing on the piano, or for making any finely adaptive movements. The finger-nose and finger-finger tests show this dysmetria by the overshooting of the mark, pseudo-ataxia. Attempting to grasp an object, the choreic opens the affected hand over widely; the pencil test also shows similar overshooting. (See Figs. 211 and 212.)

Leukocytes are usually increased in the cerebrospinal fluid. (See Meningeal Inflammation.)

Etiology and Pathogenesis.—A too narrow view of the causation of chorea has prevailed. While many patients undoubtedly develop a chorea following streptococcus or other infection, this whole group should be looked at from the developmental point of view as well. This renders fatigue factors comprehensible.

The so-called hereditary factor is probably dependent upon an inferior or slowly developing psychomotor-cerebellar integration. In certain patients the rapidly developing body calls for a higher grade of motor adaptation than the developing motor integration can subserve. This is seen normally in the cerebellar static apparatus in what is so widely called the awkward age, or in the "puppy" stage, where the analogues to mild choreic affection are obvious. Chorea may thus develop in adolescents from the mildest of infections or even from excess of motor activity. Thus chorea may be a fatigue symptom.

Ancestral syphilis is responsible for certain choreas, especially as affecting the full development of the nervous system, thus allying such choreas with related spinal, cerebellar, and cortical agenesis (Friedreich, etc.). Hence, the Wassermann tests should be applied in chorea diagnosis (provocative test desirable). Milian has found from 60 to 70 per cent. of fifteen choreics examined to show positive Wassermann reaction.² This may account for the good results from salvarsan in the treatment of some choreas.

Acute rheumatic infection loses much of its so-called specificity. Tonsillitis is also assuredly not the universal malefactor. These and other infections are important as reducing physiological efficiency, by interfering with the fiber-carrying capacity (through inflammatory

¹ Grenet et Loubet, *Rev. Neur.*, December 15, 1912, p. 632.

² Milian, *Soc. Méd. d. Hôp.*, November 29, 1912.

exudate at times), especially in some cases where meningitis is obvious, of an insufficient or tardily maturing and integrating motor apparatus, especially in its cerebellar static portion.

Diagnosis.—The chief disorders to be excluded are hysteria, frequently from imitation, and the more persistent and milder of the compulsive tics. The organic signs enumerated will be found in the majority of choreics, if carefully looked for. Lumbar puncture may be resorted to in difficult cases, although those with definite leukocytosis are apt to show the organic signs.

The diagnosis of hysteria will depend upon the finding of definite conversion mechanisms, while that of the tics will develop substitutions. The movements in both may be similar to these called choreic, but in both hysteria and in tics hypotonus is less liable to show. There is a greater likelihood that so-called hysterical movements will turn out to be choreas than the reverse, especially in younger children. In older children or in young adults the opposite may be more seriously entertained.

Treatment.—The best treatment is rest in bed, with partial isolation, no playing, local treatment of infectious areas, full diet, with increased fatty ingredients (milk and eggs), and intravenous use of neosalvarsan in some cases.

The rest in bed should be practically absolute for a week or two before the use of arsenic.

One may start with milder arsenical preparations in less severe attacks, and in those where sudden disproportionate growth seems to play a larger role, Fowler's solution Mv-xv , or the acidi arsenosi, may be used, either alone or in pleasing vehicles.

In severe and protracted cases the intravenous use of doses of 0.05 to 0.2 gm. of salvarsan, according to age, once a week for four weeks, is advantageous.

Most of the mild cases recover on prolonged rest in bed, without medication, if on a full diet, with milk and eggs in abundance.

Huntington's Chorea.—This is a disorder of the nervous system, named after George Huntington (born 1850), an American physician, who gave the first essentially comprehensive and distinctive description of the disease. Huntington's grandfather and father had observed the disorder in one of its American foci, Easthampton, Long Island, and chiefly through their studies of several generations of afflicted families the essentially hereditary nature of the malady became apparent.

Huntington's chorea has no relationships to Sydenham's chorea, that essentially infantile disturbance of cerebrocerebellar tract coördinations following so frequently upon infectious disease or exhaustion. Huntington's chorea is essentially hereditary and chronic, occurring, as a rule, in adults from thirty to fifty years of age.

The condition did not escape earlier observers. Thilenius gave a report of a case apparently as early as 1816. Rufs, another in 1834.

Waters made his striking comment in 1841.¹ Dr. Charles G. Gorman, of Luzerne, Pa., wrote an inaugural thesis, in 1848, on this affection, which has been lost. Dr. Irving W. Lyon, while house physician at Bellevue Hospital, wrote a paper on "Chronic Hereditary Chorea," which was published in the *American Medical Times* in 1863. Huntington's paper appeared in the *Medical and Surgical Reporter*, Philadelphia, in 1872. Since this time a rich bibliography has accumulated, which in the Huntington number of *Neurographs*, edited by Dr. William Browning, in 1908, mounted to 200 titles.

Etiology.—So far as is known the disorder is hereditary. From eugenic studies of Davenport, Muncie, and Jelliffe,² the chief factors or determinants behave as Mendelian dominants. Heilbronner has said that the disorder appears at later intervals in succeeding generations, but evidence of the extensive eugenic studies, which include the study of 1000 cases of Huntington's chorea, limited to a few families, shows that it appears at earlier years in succeeding generations. The data here indicated that the disease behaves as a complex in which age, motor disturbances, and mental defect behave more or less independently one of another. When all three factors combine, the result is Huntington's chorea. No other etiology is known.

Symptoms.—As it is not possible in a short résumé to discuss the separate factors, the older lines of description which regard the disorder as a unit will be followed. Thus one speaks of an insidious onset, usually coming on between the years of thirty and forty. The earlier signs are either slight changes in character, irritability, moroseness, eccentricities, or the choreic movements become prominent in the picture. The facial, neck, and upper extremity muscles are usually involved first. There are involuntary, jerky movements, usually of muscle groups, not of muscle fibers. The excursions brought about are massive, *i. e.*, excessive, loose, and hypotonic. The hand is thrown to one side, the whole arm sweeping outward; the neck is jerked backward, the head bowed forward in a quick, loose-jointed, jerky sort of way. The motor unrest spreads over the entire body.

The patient, after several years—for the motor disturbance advances slowly—becomes jerky, and although for a long time voluntary movement is able to check the excessive motion, finally control is lost and the patient is confined to a chair or his bed, making his peculiar broad, jerky movements. During sleep the motions cease.

Nearly all of the voluntary muscles may be involved. The eye movements seem to resist to the last. The speech becomes explosive, or grunt-like, very incoherent at times by reason of the involuntary movements of tongue, lips, diaphragm, and chest. Even swallowing is involved in the later stages. Writing soon becomes impossible by reason of the jerkiness of the hands and arms. Walking becomes

¹ Dunglison's Practice of Medicine, ii, 312.

² American Neurological Association, 1913.

successively more and more unsteady until the patient becomes bed-ridden.

There are few disturbances of sensibility and these are observed only late in the deteriorated phases. The knee-jerks are active or uninvolved; there are no atrophies, nor paralyses, nor hypertrophies.

The mental changes may develop apart from the motor ones, and in choreic families mental choreics are to be recognized who perhaps may never show motor signs or those who show choreic movements very late. This is the basis of Diefendorf's constitutionally defective group. These patients even in childhood may be excessively nervous, irritable, and excitable. They are often difficult children to manage. Their eccentricities become more and more marked with advancing years. Some show marked grades of feeble-mindedness and occasionally are born choreic and defective.

In the great majority of the cases the insidious and slow development of great instability and irascibility shows itself coincident with or following the choreiform movements. Angry outbursts and destructive impulses occur, often followed or preceded by periods of marked moroseness and despondency. This depression or gloom may be a forerunner of suicide. Diefendorf remarks that this despondency is not due entirely to the realization of having the disease. With some patients, however, the suicide is to be traced to the knowledge of the taint. Suspiciousness, paranoid ideas and jealousies are not infrequent mental signs. Emotional deterioration follows. The patient loses all interest in his work, his appearance, his home, etc. Some become tramps. Intemperance and free sexual activities may show themselves with this gradual deterioration. Indifference shades off into absolute incapacity and deterioration becomes profound, always, however, showing itself in the affective sphere more prominently than in the intellectual capacities, although these latter are not free from gross defect; the patients being forgetful, poor in ideas, disorderly in thought and weak in judgment.

Diefendorf speaks of a group in which the mental symptoms develop somewhat similarly to those seen in the hebephrenic types of dementia precox. These patients complain of insomnia and general malaise. They often then develop ideas of reference, anxiety, suspiciousness and ideas of infidelity. In some of these impulsive activities show themselves. Homicide has taken place. Kraepelin cites an illustration of a choreic father who killed his three small children by hanging, as he feared he could not support them, then quietly took a walk and was quite unconcerned about the affair at a judicial hearing. The eating is often impulsive and ravenous. In some of these cases, as with the inferior group, the mental symptoms may develop long before the choreic symptoms, and the diagnosis of a katatonic schizophrenic may be made as the motor symptoms become manifest. Here the diagnostic difficulties are very definite.

It would appear from Diefendorf and the studies cited that the

mental and motor traits are more or less independent one of the other. In inheritance they seem to show as such. Some patients have shown choreic movements for twenty to thirty years without mental signs.

Course.—This is subject to great variation. Often the patients suddenly develop great motor unrest; the mental signs augment rapidly, and the patient dies in exhaustion five, ten or fifteen years after the onset of the symptoms. Usually the disorders, *i. e.*, motor and mental, are progressive; rarely they may diminish after reaching a severe grade. Many die of intercurrent disease.

Pathology.—No unitary interpretation seems yet possible. The motor and the mental symptoms are best considered separately.

A number of autopsies have shown a variety of findings. At times there are chronic meningeal thickenings, again generalized brain atrophy. This reduction of the cortex may show to a marked degree in the loss of cells, particularly of the third layer. There is a compensatory increase in neuroglia. In some patients arteriosclerosis has been present, in others not. The older patients showed the arteriosclerotic changes. These cortical cellular changes are apparently more frequent in the frontal areas. They are correlated with the mental defects.

The pathology behind the motor manifestations is more obscure. Theoretical considerations as well as pathological findings point to an implication of the rubro-thalamo-cortical extensions of the cerebellar pathways as chiefly responsible for the perverted movements. Kleist, Zingerle, Jelgersma, and Winkler adduce observations from various sides which tend to show that these mechanisms are involved. Numerous autopsies show changes in the lenticular region which may be taken to support these contentions. Thus the motor signs have a pathology similar to that seen in paralysis agitans and other midbrain tremors.

Therapy.—There is no efficient therapy. Many patients need hospital care. Prophylaxis is important, Mendelian dominance arguing for certain factors at least, that these patients should not procreate. The percentage of chance of escape for Huntington choreics is about one in four at the best. Some branches escape, and a branch once free, is always free. Only a few exceptions to this are found in the Davenport, Muncie, and Jelliffe extensive series.

CHAPTER XI.

DISEASES OF THE MENINGES.

HERE diseases of the dura, the arachnoid, and the pia are to be recognized. Under the first various types of meningeal hemorrhage and inflammation—pachymeningitis—are found. Under the latter various forms of leptomeningitis and cerebrospinal meningitis.

DURAL DISEASE.

1. **Meningeal Apoplexy** (*Traumatic Meningeal Hemorrhage*).—**Etiology.**—Trauma from blows, instruments, violence of various kinds, causes either a rupture of the branches of the median meningeal artery, the veins, or of the cerebral sinuses. Such hemorrhages may be found at the site of the injury, or at the point opposite.

The hemorrhage may be extradural, between the pia and dura, epidural, or between the pia and cortex, arachnoidal hemorrhage. The bleeding may be diffuse or circumscribed. At childbirth such hemorrhages with partial destruction of the brain substance itself are extremely frequent. Here the bleeding is almost always exclusively venous.

Pathology.—Macroscopically such hemorrhages resemble hemorrhagic pachymeningitis, but microscopically they differ, especially in the absence of new elements—vessels, plasma cells, etc.

Not infrequently, in severe fractures, the brain substance is also involved.

Symptoms.—These will vary according to the severity of the causing lesion, the amount of blood thrown out, and the site and extent of the bleeding. In severe injuries there are signs of shock and concussion in addition to the symptoms of pressure. Unconsciousness becomes increasingly deeper, the pulse is slow in the beginning, then hastens, vomiting takes place, urination and defecation are involuntary, irregular respiration, with increasing temperature, and death often results unless operation relieves the pressure.

In less severe lesions the initial symptoms of concussion with varying grades of stupor partially clear up for an hour or more, even twenty-four, or a few days.¹ Then compression symptoms develop, with signs of excitement. Irritation and paralytic signs appear. There may be spasms, epileptiform convulsions, often of Jacksonian

¹ Connell, Free Interval in Meningeal Hemorrhages, Surg., Gyn. and Obstet., March, 1906.

type, monoplegias, hemiplegias, the arm often suffering the worst. The hemiplegia is usually on the side opposite to the site of injury, but in about 5 per cent. of the cases is found on the side of the lesion (uncrossed pyramids or *contra coup*). Aphasias are not infrequent in left-side injuries. The tendon reflexes are usually increased on the parietic side, while the skin reflexes are usually diminished. The Babinski phenomenon is frequent on the paralyzed side, and occasionally present on the side of the hematoma. Occasionally hemianesthesia and hemianopsia can be made out.

Bleeding at the base may show involvement of the cranial nerves; occasionally choked disk is present.



FIG. 227.—Pia-arachnoid hemorrhage from *contra coup*. (Larkin.)

The pupils vary greatly. Wiesmann has shown in 70 cases, that in 39 both pupils were dilated and immobile, in 20 there was dilatation on the side of the hemorrhage, in 7 both pupils were small, and in 4 there was dilatation on the side opposite the lesion.

In birth hemorrhages, Seitz has shown that sub-tentorial hemorrhages behave differently from convexity hemorrhages. In the former the children frequently show no signs of asphyxia, then after a few hours respiratory disturbances set in, the breathing becomes irregularly quickened, with spasms and cyanosis. Then spasms of the eyeballs occur, less often facial spasms. If the pressure is directed downward toward the medulla, opisthotonus and muscular rigidity develop, and not infrequently priapism.

In convexity hemorrhages the child shows considerable restlessness, refuses to suckle, and then develops signs of brain-pressure, pressure in the fontanelles, respiratory disturbances, drowsiness to unconsciousness, with some slowing of the pulse. Localizing symptoms may then show themselves, spasms of the opposite arm and leg, increased tendon reflexes, and slight hypertonus.

Diagnosis.—It is extremely difficult to determine whether one has to deal solely with a pure meningeal apoplexy, or whether there is also intracerebral disturbance. If there is a definite free interval after the initial signs of concussion, with the gradual or sudden onset of compression signs, the probabilities are for hematoma (80 per cent.).

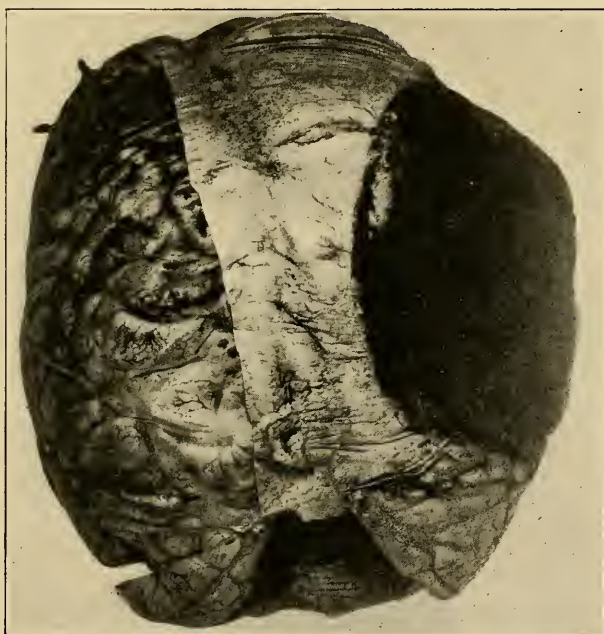


FIG. 228.—Traumatic supradural hemorrhage. (Larkin.)

The length of free interval offers no certain criterion as to site of hemorrhage. Choked disk, often transitory, also speaks for hematoma. Bloody spinal fluid, which does not clot, speaks for intradural hemorrhage, occasionally extradural. Neisser's brain puncture and lumbar procedures often help in clearing up a diagnosis. Long intervals speak for abscess.

Therapy.—This is surgical and should be immediate. The exact procedure must be determined largely by the symptoms. Even the intracranial hemorrhages of childbirth may be controlled by skilled surgical measures. The results of surgical interference are three times as good as leaving the patients alone.

Traumatic late apoplexy is a special variety in which degenerative processes complicate the picture. The patients may develop the signs of hemorrhage, usually intracranially, even months after the injury. Such cases are to be diagnosed with much caution. Senility, pronounced arteriosclerosis, and syphilis should be excluded.¹

2. Inflammation of Dura (*Pachymeningitis*).—(A) **Pachymeningitis Externa.**—Following severe trauma, purulent processes of the frontal sinuses, the middle ear, mastoid, erysipelas, caries, or osteomyelitis, one occasionally observes an inflammation of the external surface of the dura of the cerebrum. It is usually localized. A similar process may be present in the spinal dura.

Symptoms.—These are usually hidden in those of the causative lesion and vary with the acuteness and extent of the process. Localized cerebral or spinal pains, muscular twitching, spasms, marked scalp tenderness to percussion, and slight rise in temperature are the chief signs. At times symptoms of localized pressure of the cerebrum or of the cord, resembling tumor, are observed.

Treatment.—The treatment is that of the causative factors.

(B) **Pachymeningitis Interna Simplex.**—This may consist of a localized or circumscribed purulent exudate, giving in the main the symptoms of a brain abscess or a brain tumor. In rare instances (pneumonia, etc.), a secondary pseudomembranous productive inflammation occurs.

(C) **Pachymeningitis Interna Hemorrhagica.**—This consists of a chronic inflammation in the internal layers of the dura, associated with extravasation of blood. It is by no means infrequent² and gives rise to chronic psychoses of an ill-defined type.

History.—Morgagni noted the affection in the eighteenth century. Baillarger³ followed the older authors in assuming it to be a primary hemorrhage, with new membrane tissue formation. Heschl and Virchow⁴ first pointed out the inflammatory nature of the productive inflammation, and the consequent hemorrhage due to the rich formation of new bloodvessels. Jores and modern authors support these views.

Occurrence, Pathology.—Usually a disorder of advanced years, hemorrhagic pachymeningitis may be found in children.⁵

It is extremely frequent in general paresis, and Blackburn has found it to cause chronic excited and chronic depressed states, in patients running a course resembling senile dementia. It is frequently an alcoholic complication. It is seen in some chronic choreic affections. Tuberculosis, nephritis, leukemia, scorbutus, Barlow's disease, and hemophilia have seemed to stand in causal relations in some instances.

¹ Stadelmann, Deut. med. Woch., 1903; Allen, Jour. Nerv. and Ment. Dis., 1909.

² Blackburn, Government Hospital Autopsy Reports.

³ Maladies du Cerveau, 1854.

⁴ Würzburger Verhandlungen, 1856, p. 134.

⁵ Goppert, Jahr. f. Kinderheilk., 1905, lxi, 51; Freund, Monat. f. Kinderheilk., 1909, vii.

In the initial stages a productive inflammation causes the formation of a thin, delicate, yellowish-brown membrane on the inner surface of the dura. The base is rarely affected, the temporal regions more often. New bloodvessels form, whose walls give way, giving rise to extravasation of blood. The process of new membrane formation and bleeding goes on hand in hand until the whole membrane may be one or more centimeters thick, compressing the brain, with which it is usually closely united by new connective-tissue formation and new bloodvessels, and causing atrophies, degenerations, softening, or sclerosis of the near-lying portions of the cortex.¹ The process may extend to the spinal cord.

Symptoms.—The disorder may be present for years without symptoms. In paresis it may not add any definite symptoms to the underlying disorder, or it may cause a number of complicating pressure pictures. When the process has advanced to a definite extent both general and local symptoms show but are so indefinite as to defy diagnosis. Gradually increasing head discomfort, headache, often severe, some nausea or vomiting, irregular periods of confusion or distress in the head, with at times mild delirious excitement, are among the more characteristic earlier signs.

With rapid extravasations, acute pressure symptoms may develop, with epileptiform convulsions, hemiparesis, comatose states with slow pulse, aphasias, astasia-abasia. It is very characteristic that these may be transitory. Persistent monoplegias, with Jacksonian attacks involving irregularly distributed muscle groups may be early signs. Often persisting stereotyped muscle movements, chewing, automatic arm, hand, or leg movements may be the signs of a local irritative lesion. Constantly putting the hands to the head was considered suggestive of pachymeningitis by Fürstner. It is a frequent sign in paresis. The eye muscles are rarely involved (conjugate deviations, nystagmus), and still more rarely one finds involvement of the cranial nerves at the base.

Papillitis, or choked disk, is often present. The pupils are not infrequently irregular, occasionally myotic in the early stage, dilated in coma, and at times immobile to light and accommodation. Argyll-Robertson may be present with or without positive Wassermann. Increased tendon reflexes show the presence of irritation of the motor cortex, clonus and Babinski being occasionally present, and at times coming and going. Irregular but inconstant temperature changes are often present.

Course.—This varies considerably, is usually chronic, shows remission and exacerbations. At times the patient recovers completely, again the disorder is progressive, causes death after a long psychotic period of irregular excitement or depression.

¹ Jores u. Laurent, Ziegler's Beiträge, xxix.

Diagnosis.—It must be separated as a primary and as a secondary process in alcoholism, paresis, cerebrospinal syphilis, etc. Brain tumor, abscess, hydrocephalus, sinus thrombosis, leptomeningitis, apoplexy are to be borne in mind. Traumatic meningitis must be excluded if an antecedent trauma, even of mild grade, such as falling from the bed, in bath tub, hard crack on the head from running into door, etc., should have occurred.

Neisser's procedure of brain puncture is always desirable in those patients that give signs of brain tumor, brain abscess, etc.

Therapy.—In acute progressions, local bleeding is advisable. Hydrotherapy with stimulation of elimination is useful. Brain puncture has been of service, also lumbar puncture in children. Abstinence from alcohol is imperative. Mercury in the positive Wassermann cases is indicated. The pains are often relieved by analgesics, and by warm baths. Brain puncture with injections of gelatin have been successful.

DISEASES OF THE ARACHNOID AND PIA. LEPTOMENINGITIS.

1. **Acute Leptomeningitides** (*Cerebrospinal Meningitides*).—The studies of recent years have shown a host of causes for acute inflammation of the cerebral and spinal arachnoid and pia. These vary very widely as to severity—simple meningism to the gravest forms of general meningeal involvement—epidemic cerebrospinal meningitis, epidemic polio-myelo-encephalitis, general syphilitic meningomyelitis, etc. It becomes impossible to present a logical classification of these disorders, either from the etiological, pathological, or clinical standpoint. In general only the more circumscribed types of meningitis will be considered here.¹

Causes.—These are many. Trauma is one of the most important. Such trauma may act either through direct infection, as by compound fracture, or secondarily bring about a septic meningitis, through hemorrhage, thrombosis, and subsequent infection.

Traumatic early and late meningitis appear. Many tuberculous leptomeningitides arise from trauma. Occasionally one meets with late purulent meningitides, due to old encapsulated abscesses, old projectiles (bullets, splinters, etc.), old fractures of lamina cribrosa.

Infection from suppurative processes in the neighborhood is one of the most frequent causes of the type of meningitides under consideration, and chief of these is otitic meningitis. Suppurative otitis may lead to intra- or extracranial abscess, sinus phlebitis, thrombosis, direct contagion, contagion through labyrinth, through mastoid, etc. Serous meningitis may also have an otitic origin.

Nasal and frontal sinus infection gives rise to a small number of these meningitides; they may be purulent or serous. Operations upon the nose are frequently complicated by meningitis.

¹ Recent monographs are by Schultze (Nothnagel) and Finkelnburg, *Handbuch d. Neurologie*, ii, 1090, in both of which complete bibliographies are to be found.

Facial erysipelas occasionally is a cause; rarely facial furunculosis.

Specific organisms give rise to specific types of meningitis. Among these are measles, scarlet fever, varicella, typhoid, diphtheria. The influenza bacillus is an extremely important organism in this respect, often giving rise to severe epidemics of meningitis. Malaria, yellow fever, anthrax, leprosy, actinomycosis, pneumonia, whooping-cough, each may be the exciting factor. Septic extension from acute articular rheumatism is a factor. Tuberculosis is a large item. The epidemic type due to the *Diplococcus intracellularis* is one of the most characteristic of the types. Old abscess formations in the lung, liver, bladder, gonorrhœa, etc., all may give rise to a meningitis.

Occasionally one ascribes certain meningitides to chemical poisons: diabetes, lead, gout, are among these.

Symptoms.—These show certain variations according to the mode of infection and the type of infecting agent. Since simple purulent meningitis, tuberculous meningitis (usually a mixed infection) and epidemic cerebrospinal meningitis are the chief infections, the following description will apply to these and no attempt will be made to cover the symptomatology of the entire group.

Headache.—This is one of the earliest signs, and is frequent, usually increasing in intensity as the disease progresses. It is mostly diffuse, but may first appear in the occipital region, or in the forehead. The slightest movement, percussion, etc., increases it.

Stiffness of Neck.—This is a striking symptom and develops early, sometimes preceding the headache. Such early stiffness may or may not be apparent to the patient, but comes out on careful examination, and is not infrequently accompanied by painful pressure points over the cervical vertebræ. When well developed the least movement of the neck is painful and the attitude of the patient on movement is striking. In young children it is not infrequently absent, and in adults in chronic cases. It is a further characteristic that this stiffness and painfulness shows considerable variation even during the twenty-four hours.

Hyperæsthesia.—The skin, the muscles, the joints, can hardly bear the slightest touch or pressure, and sensitiveness to light and to sound are early signs of meningeal irritation. Occasionally the sense of smell is abnormally acute.

Nausea and Vomiting.—These are common initial symptoms (80 per cent.). Occasionally the vomiting persists. Early vertigo is frequent.

Mental Symptoms.—These occur early, particularly in children, and more especially in tuberculous meningitis. The patients become peevish, throw their toys away, are capricious, their attention varies. They not infrequently have mild delirium early, especially at night, or are confused. Very young children, one to three years, often show less mental involvement. Older patients are excited, sleep badly, are restless, mutter in their sleep, or have well-marked delirium. Later a

semicomma may gradually develop, with variations in intensity from slight confusion to a confused delirium. Marked ups and downs characterize the epidemic forms, but some degree of confusion or coma is more or less constant.

Temperature.—The patients practically always show a rise in temperature. High temperatures (104° to 106° F.) usually characterize the purulent meningitides; often preceded by chill. Such temperatures may remain high, or not infrequently show remissions. The tuberculous meningitides usually show a lower curve.

Motor Irritation, or Paralytic Phenomena.—Cramps and epileptiform convulsions occur more often in the early stages with children than with adults. They sometimes are very persistent. Circumscribed spasms, Jacksonian in type, are not infrequent. Occasionally there are choreic, athetoid, or tremor movements, which come and go, or are continuous.

Muscular rigidity is an early and persistent sign, showing early in the neck, later in the back, with opisthotonus, or stiff lordosis postures. These muscular rigidities are often subject to considerable fluctuation with the degree of internal or external hydrocephalus. Lumbar puncture often influences them greatly.

Kernig's sign is frequent, and is based upon this generalized muscular rigidity (reflex, spasmodic contraction of rectus femoris).

Paretic signs, monoplegia, or hemiplegia, are less frequently met with, and then more in the late stages. Such indicate localized purulent processes, with abscess formation in the motor area, and are usually accompanied by convulsions. Spinal pareses (paraplegia) are rare.

Speech disturbances, usually cortical, and aphasia are occasionally observed, more particularly in tuberculous meningitides.

Cranial Nerve Signs.—Disturbances of the cranial nerves are among the most characteristic signs in the meningitides. The pupils are often unequal; frequently markedly miotic in the beginning, they show irregular widening later. With increasing coma they usually widen, and react sluggishly or not at all. Convergence reactions are less easily tested, but also show slowness.

Eye palsies are frequent. Mild ptosis, unilateral or bilateral, divergent and convergent strabismus—the abducens is particularly prone to disturbance—and double vision is often present. These eye palsies vary from hour to hour in extent, and in intensity, becoming permanent in the long protracted cases particularly. Protrusion of the eyeball is a rare sign, nystagmus frequent.

Papillitis is very frequent, and early; optic atrophy is common (18 to 25 per cent.). Permanent blindness, however, is rare. The trigeminus is rarely involved.

Facial palsies are very frequent, but are usually incomplete, vary from day to day, and are rarely permanent.

Hearing is frequently affected. Complicating otitis, and laby-

rinthitis often leave these patients deaf. It is a frequent complication in the second and third week of the disease.

The vagus involvement causes pulse and respiratory anomalies which are also influenced by central processes. The pulse is initially hastened in practically all forms, and usually remains rapid in the later stages, save in those forms of less acute progress, notably in tuberculous meningitis, where it is often slow, especially after the second week of the disease (75 per cent.—Heubner). Marked irregularity is conspicuous.

The respiration is also irregular and Cheyne-Stokes type is frequent in the severe purulent and tuberculous types toward the end.

Reflexes.—The tendon reflexes are usually somewhat increased, particularly in the early stages, but may be missing from the beginning (medullary and ventricle pressure). With the advance of the disease they may be missing. Marked variations and irregularities are to be expected, thus lost knee-jerks may be associated with ankle-clonus and Babinski. This latter is a very frequent early sign and later disappears. It should be remembered that it is normally present in young children up to six to eight years of age, and hence is to be neglected in diagnosis in young children.¹

Lumbar Puncture.—This is of primary importance. The pressure is initially increased. Later this is not marked because it is purulent. In the ordinary purulent meningitides the fluid is usually cloudy, sometimes only microscopically so; again markedly purulent, in which the specific organisms may be found by proper methods. In tuberculous meningitis the fluid is less apt to be cloudy, especially early, but by proper technique the bacillus is found (33 per cent. early stages, 50 per cent. with pressure signs, 100 per cent. in paralytic stages—Pfandler).

Cytologically² one finds that in purulent meningitis there is a preponderance of polynuclear leukocytes, while in tuberculous meningitis the lymphocytes are increased. This is not an absolute rule and there are variations during the course of the disease. The cytological findings should be checked up with the clinical ones.

Irregular Symptoms.—Herpes is not infrequent (75 per cent., save in young children) in the epidemic form, and usually appears within the first week (two to five days). It is most frequent about the nose, lips, and forehead. It does not persist long as a rule. Other skin eruptions are not infrequent, especially the rose-colored spots of the diplococcus types. Eruptions resembling measles, scarlet fever, urticaria are occasional, while erythemas and hemorrhagic spots are rare.

Gastric disturbances are frequent, especially constipation. Diarrhea may be an initial symptom in young children.

Tonsillar and pharyngeal redness and soreness are not infrequent

¹ See Göppert for careful study of reflexes; *Klin. Jahr.*, xv, 523; *Berl. klin. Woch.*, 1905, 21, 22.

² Schönbron, *Klin. Vort.*, 384; *Med. Klin.*, 1906, p. 593.

in the epidemic types, and bronchitis is often present in the later stages.

Course and Prognosis.—Whereas most of the types of leptomeningitis show much the same general symptomatology, it is more particularly in their developmental course that the differences appear. These variations may be viewed as fundamentally due to specific differences in the microorganisms in question, although it may be said that in a number of instances the only differences observed by the best clinical observers have been those of the microscopical slide, or the test-tube. Hence it may be stated that under certain circumstances the pathological fact that the patient has a meningitis governs the entire picture and all forms are precisely alike, but in the main it is true that specific variations exist, and should be sought for when facing any particular case of leptomeningitis.

In general fairly sharp distinctions can be made between (1) the infectious, (2) the epidemic, (3) the tuberculous, and (4) the serous forms.

(1) *Infectious Meningitis.*—Here the initial disturbance in the ear, nose, mastoid, frontal sinus, fracture, etc., precede, and its symptoms often hide those of the suppurative meningitis that follows. Here the onset is usually acute. Headache, local or diffuse, is early. The temperature usually mounts rapidly to 104° F. or 105° F., with initial chill; the pulse and respiration are rapid. There is photophobia and hyperacusis within a few days. The mental signs come on soon. Confusion, somnolence, or coma are frequent, often punctuated by active delirious intervals. Lumbar puncture usually shows a purulent fluid. The tendon reflexes are usually increased and the cranial nerve signs are marked within the first week. No special skin eruptions are noted. Neither tache cerebrale nor dermographism are marked.

With irregular septic temperature, increasing coma or delirium, increasing signs of local pressure or destruction, convulsions, and paralyses the more serious cases end fatally within two to five days in children, or one to two weeks in adults.

Other patients show less grave symptoms, run a subacute course and recover in three to four weeks, but in general the prognosis is unfavorable. Those that get well probably have a circumscribed process.

(2) *Epidemic Cerebrospinal Meningitis.*—Known for centuries, first recognized as epidemic in 1805 in Switzerland, in 1806 in Massachusetts, this form has been extensively observed the world over.¹ Its epidemic onset is usually very insidious, and spring and winter are the months of predilection in the north temperate zones. It is now endemic in large cities. Children under ten are most prone to the infection. The exciting agent is the *Meningococcus intracellularis*

¹ See Report of State Board of Health, Massachusetts, 1898, for extensive review with literature to that date. Articles of Finklenburg, loc. cit., for later literature.

of Weichselbaum.¹ The disease is contagious, the contagion being possibly carried through the nasal passages. Abortive, subacute, acute, chronic, and fulminating cases are recognized with every possible intermediary type.

The malignant or fulminating cases may end fatally in twenty-four hours, with headache, nausea, vomiting, delirium, coma, convulsions, stiff neck, high temperature, high pulse, Cheyne-Stokes respiration and death from acute toxemia.

Abortive cases, which are more often found in adults than in children, show rudimentary meningeal signs. Headache, backache, nausea and some vomiting with slight stiffness of the neck occur, or the patients may only have slight vertigo, malaise, and nausea, and keep on with their work. Fever is usual and occasionally deafness develops in these abortive attacks.

The usual subacute or acute types show a latent period averaging three to five days consisting of irregular backache, headache, slight vertigo, and sweating, with signs of a nasopharyngitis. Then a chill with slight rise in temperature accompanied by vomiting indicates the onset. Children become restless, cry, and are very irritable. Usually within twenty-four hours meningeal signs appear. Stiff neck, headache, mental confusion are present; not infrequently convulsions appear in children. Photophobia, hyperacusis, excessive sensitiveness to pressure, and hypertonicity then show themselves with Kernig's sign and increased tendon reflexes. The cranial nerve signs then develop. Herpes labialis is frequent from the second to the fifth day, and a measly, macular eruption (spotted fever) may appear. Other skin eruptions develop in the first week. There is usually a leukocytosis, and the cerebrospinal fluid shows a characteristic picture.

The coma continues, shows considerable variation in intensity, and the patient gradually improves, or shows increasing signs of pressure, convulsions, palsies, and dies within seven to fourteen days, after ineffectual attempts at maintaining nourishment.

Other patients show a much more chronic course, lasting weeks or months, with considerable variation in the intensity of the headache, coma, or delirium, with eye palsies, mono- or hemiplegia, contractures, constant emaciation, and variable temperature until death takes place, often with increasing size of the head—hydrocephalus and signs of increased cerebral pressure.

In other more favorable cases, periods of clearness or of betterment become longer and more pronounced, intermingled with the severer symptoms already outlined. The variability in clinical course is enormous, but the prognosis, up to the time of the introduction of the Flexner serum, was distinctly unfavorable, the mortality ranging from 50 to 80 per cent. Apparently mild cases often die and some extremely ill children recover. Recovery with defect, either deafness,

¹ Fortschritte d. Med., 1877, p. 622.

blindness, cranial nerve palsies, monoplegias, hemiplegias, or mental defects, are not infrequent.

Treatment.—Since a specific antimeningitic serum has been elaborated by Flexner¹ the prognosis has been much improved. The mortality has fallen to 25 per cent. in some of the recent epidemics.² The best results are obtained in children of from five to ten years.

The effects of serum treatment are often seen very soon—twenty-four hours after injection. The Kernig sign and stiffness of the neck persist, however. The attenuation of the symptoms is very marked in many of the cases, as well as shortening of the disease. Lysis is the usual mode of recovery of non-serum-treated cases. Crises occur much more often in serum-treated cases (25 per cent.). Furthermore, the permanent sequelæ of the disease are markedly reduced by the serum treatment.

The general treatment will be considered with that of the other forms of meningitis.

(3) *Tuberculous Meningitis.*—This form was first isolated about 1830 (Rilliez et Barthez and Robert Whytte), although one can see its chief features in the *Nosologie of Sauvages*, written in 1763. In this form the onset is characteristically subacute or chronic, one to two weeks, exceptionally months, and is almost invariably secondary to tuberculosis in other organs. General symptoms, such as loss of appetite, irritability, loss of flesh, general malaise, with loss of desire to play, fretfulness and ready fatigability are the precursors. Headache and dizziness, with irregular fever movement is then observed, and occasional dreamy states, during which the patient's manner is peculiar.

Then gradually, or suddenly, the patient's manner becomes much changed; mild delirium or coma develops, and convulsions appear. The patients lie in bed, are restless, rolling from side to side with sharp cries or whimpers, and frequent placing of the hand to the head. Passive motions of the head forward invariably bring out resistance and pain. The sensitiveness of the skin to pressure or touch is marked, and hypertonus with muscular twitchings and Kernig's sign are present.

The temperature ranges from 102° F. to 104° F., and is usually less marked than in the suppurative or epidemic types. Lumbar puncture in the first week usually gives a clear fluid under pressure, with exceptionally the tubercle bacillus or pus elements.

The cranial nerve signs may then develop in the second to third week, and show more ups and downs than is usually present in the other types. Localized pressure signs with hemiplegias or monoplegias then develop and, not infrequently, the previously increased tendon reflexes diminish or are lost.

The patients gradually get worse, convulsions are frequent, and

¹ Jour. Exp. Med., 1907.

² Flexner, International Clinics, 1909; Jour. Amer. Med. Assoc., October 30, 1909.

rigidity is followed by flaccidity, and the patient dies with signs of intense exhaustion, sometimes with agonal rise of temperature immediately preceding. The whole attack may terminate within a comparatively short time, two to three weeks, or may run for months.

Atypical forms are especially frequent in adults such as apoplectic-form onsets, with aphasia, monoplegia, or hemiplegia. The picture may be that of a toxic delirium. The prognosis is bad.

(4) *Serous Meningitis*.—Dietl, in 1846, first isolated this type, which is of uncertain origin, and characterized by varying grades of edematous exudate with infiltration of round cells. The symptoms are usually those of a mild meningitis. Headache, stiff neck, marked sensitiveness are constant, whereas convulsions and signs of pressure are less frequent. Again the disorder may be acute and very severe, with high temperature and signs of cranial nerve involvement. The usual course is less stormy. Papillitis is a frequent symptom, and should be borne in mind in those patients in whom the serous exudate is more or less circumscribed, giving the general impression of a brain tumor. Lumbar puncture shows increase in pressure with some lymphocytes.

In the serous meningitis of alcoholic origin (wet brain, meningo-encephalitis), there is usually a busy delirium, excessive hyperesthesia, with marked twitching of the muscles.

Syphilitic Meningitis.—See Syphilis of the Brain.

Differential Diagnosis.—The chief disorders that may be confounded with the meningitides, especially in the beginning of the disorder, or in mild cases, are as follows: Internal pachymeningitis, which rarely gives temperature or lumbar puncture findings. Encephalitis, and encephalomyelitis: in the former localizing symptoms develop early, and the spinal fluid is clear; in the latter flaccid palsies rapidly develop, and the fluid is not purulent. Brain abscess may be complicated by meningitis, or localized, in which latter case the localizing symptoms, septic course, and clear spinal fluid are of aid.

Infectious sinus thrombosis may resemble meningitis very closely. There is a greater tendency for the lower cranial nerves to be involved, particularly the vagus, hypoglossal and spinal accessory. The spinal fluid is usually clear.

Delirium tremens shows an active occupation delirium, optic hallucinations, marked fine tremors and little temperature. Hysteria rarely shows temperature, and many of the organic signs described by earlier authors as found in hysteria are better referred to as organic brain disease with hysterical epiphenomena.

Treatment.—This must first be prophylactic, and involves general hygienic precautions in tuberculosis; prompt aural treatment in otitis, mastoiditis; surgical asepsis of wounds in all head operations. The isolation of the epidemic types is advisable, and nasal antisepsis imperative. The active treatment is surgical for most of these forms, especially if the symptoms show early signs of being circumscribed,

or when it seems possible to get at an initial focus of infection, as in the various septic infectious forms—ear disease, etc.

In diffuse general meningitis the responsibilities are great, and it is difficult to decide in the individual cases.

Epidemic types are best treated by serum. Surgical treatment of tuberculous meningitis has not yet met with sufficient success to warrant its advocacy.

Lumbar puncture has given very brilliant results in some patients, in others it has been of little service. With the development of acute hydrocephalus it is indicated, and it is in general a harmless procedure. The punctures may be made frequently, and 25 to 40 c.c. of fluid removed. The amount removed should be controlled by the pressure.

Of the serums, the Kolle-Wassermann in septic cases has given good results. Flexner's serum has been mentioned.

General treatment consists in keeping the patient quiet. Bromides, chloral, veronal and other mild hypnotics are of service. Hypodermic use of morphin or hyoscin may be necessary, but other things being equal, is undesirable. The continuous hot bath is especially valuable in delirious patients.

In the beginning a prompt use of calomel is called for: The room should be darkened and as quiet as possible. Pain may be controlled as far as possible by analgesics, and local counter-irritants.

Particular attention should be given to the nourishment and rest of the patient. Continuous fussing and overnursing is to be avoided in these cases. There are no specific remedies. Urotropin in large doses may be tried, as it is partly broken down into formaldehyde in the cerebrospinal fluid. The silver colloidal salts are disappointing.

In the long, tedious convalescence of many of these little patients, one's ingenuity is taxed to stimulate the appetite, provide the proper amount of outdoor play, and to strengthen the paretic or paralyzed muscles.

2. **Chronic Leptomeningitis.**—This usually is a secondary affection in paresis, senile dementia, chronic lead poisoning, and chronic pachymeningitis; it is rare as a primary affection save as syphilitic or tuberculous.

Chronic tuberculous leptomeningitis is usually of the convexity, usually anterior, involving the frontal and motor areas. It behaves as a low, mild-grade meningitis, often with intermittent circumscribed symptoms, simulating those of a brain tumor.

Chronic traumatic cases often show vertigo, pains, epileptiform convulsions, aphasias, nausea and vomiting, and gradual mental involvement. These signs should stand in direct relationship to the injury, and should not be complicated by signs originating at a distance from the site of the injury or from other causes (Argyll-Robertson from syphilis, for instance).

Chronic meningeal inflammations are occasionally found in children, giving the signs of a posterior basilar meningitis. They are often complicated with hydrocephalus. Opisthotonus is frequent. Many are due to syphilis, as the Wassermann tests show.

Hydrocephalus.—An accumulation of fluid within the cranial cavities takes place in a variety of affections. It is invariable in the acute meningitides, in greater or lesser degree; in tuberculous meningitis it is often extreme and comes on with great rapidity—acute hydrocephalus being practically synonymous with a tuberculous basilar meningitis.

External hydrocephalus is often synonymous with serous meningitis; as an entity it is infrequent.



FIG. 229.—Showing marked hydrocephalus. Clinically the patient showed epileptic attacks and was markedly feeble-minded. (Munson.)

Hydrocephalus is a very frequent complication in tumors of the brain, particularly in tumors of the third ventricle, the pineal, the corpora quadrigemina, and those causing pressure upon the aqueduct. These conditions are discussed under Brain Tumor. Ventricular hydrocephalus often results from such structural anomalies as cephalocele, and spina bifida.

In children one may find a primary chronic hydrocephalus of as yet unsettled pathology; but there is usually a chronic ependymitis present.

One to five pints, even gallons, of fluid may accumulate. The fluid is clear, slightly alkaline with sp. gr. 0.1005, containing the earthy chlorides, albumin, phosphates, and occasionally a sugar-reducing substance. The presence of high percentages of albumin and many cellular elements indicates a more active inflammation. As a result of the pressure the ventricles widen, the septum lucidum is displaced, the cortex thins, the infundibulum dilates, the optic chiasm is pressed upon. Extreme distention may convert the pallium into an enormous cyst, with the smallest vestige of a cortex. The thalamus may be flattened, even the pons.

The cranial bones may become separated. The head enlarges, usually symmetrically, at the vault and at the base. In older children, however, hydrocephalus may exist without these changes in the position of the cranial bones taking place. The average cranial circumference at birth is fourteen inches; at one year eighteen to nineteen inches. In hydrocephalic heads this is increased. As a rule the congenital types show the largest heads. These patients are rarely born alive, or they live a short time only.

Symptoms.—Two to three or more months after birth it is noted that the child's head is increasing in size with more than the usual rapidity. Somnolence and lethargy are frequent; the child may not be able to hold the head up. A whiny, peevish irritability, with frequent sharp cries is the rule. With fairly rapidly increasing internal pressure the coma is marked, showing great variability, however; vomiting is frequent, the hearing is affected, also the sight, disks, spastic extremities, with usually symmetrically increased reflexes. The pupils are usually contracted and sluggish to light early. With severe grades of pressure, there may be extreme dilatation. Convulsions appear and death results in from three to six months, with signs of emaciation.

In the cases of more gradual increase of intracranial pressure a marked degree of accommodation takes place. The symptoms are those of dulness or stupidity, the children are usually less bright, the choked disk may be very little marked, or may be excessive if the bones have not permitted distention, and may be followed by atrophies; but many cases recover with only a slight degree of mental reduction—weak-mindedness or only stupidity. There is a proverbial cheeriness in these patients. Some few make total recoveries, and may show brilliant mental capacities.

In the hydrocephalus of brain tumor in adults, with its up-and-down course, somnolence and headache, choked disk, dilated pupils, somewhat inactive to light, are characteristic.

A cracked-pot percussion note is often very characteristic in the young and also in many adults.

Therapy.—Many cases are hopeless from the onset. A Wassermann test should always be made, both of the blood and cerebrospinal fluid, for the double purpose of determining syphilis or other inflam-

matory disorder. Many cases of ependymitis are syphilitic in origin. For these prompt mercurial treatment is indicated; salvarsan or enesol are useful. Inunctions are also valuable in the less rapidly advancing cases. Iodin therapy is advantageously combined.

Cerebral irritative phenomena need hot baths and bromides.

Tapping is frequently of service, but not always. Lumbar puncture is not practicable to relieve pressure, as frequently the aqueduct of Sylvius is blocked; a dry lumbar tap is of diagnostic service.

CHAPTER XII.

DISEASES OF THE BRAIN.

ENCEPHALITIS—ABSCESS OF THE BRAIN.

ENCEPHALITIS refers to an inflammation of the brain substance. There are a number of forms, but the term is here restricted to the more acute processes, due to bacterial or toxic action. Primary idiopathic encephalitis does not exist. General paresis is a diffuse encephalitis, with exudations and proliferation of new glia and bloodvessels; multiple sclerosis is a type of disseminated encephalitis; cerebral gummata may be spoken of as localized encephalitis, etc., but the discussion is here limited more particularly to acute exudative inflammations of the brain substance proper. Abscess is a frequent secondary result.

Historical.—The encephalitides were for years included with the meningitides, possibly under the term phrenitis, which to the ancients meant any excitement, accompanied by fever. In Hippocrates the typical description of phrenitis, however, is a typhoid delirium. Traumatic encephalitides were known to this author.

Just how long this conglomeration of cerebral affections remained an entity in nosology cannot be determined. Separation of types is going on at the present time, and it must be recognized that a heterogeneous group at best must, for the present, be included under the head of encephalitis, a term used as early as 1554 by Actuarius.

Leaving aside the older works, the history of encephalitis practically begins with the works of Rostan (1820), Bouillard (1826), Lallemand (1830), and Fuchs (1835), in which processes of softening, of an inflammatory or non-inflammatory character are commencing to be recognized. Cruveilhier, in 1829, distinguished an apoplectic softening, suppurative softening, and softening with disorganization without pus or inflammation. Virchow's (1846) work on thrombosis and embolism threw much light on the subject, while the studies of Durand Fardel (1849), Haye (1868), and Huguenin (1876) commenced to give a modern touch. Then Wernicke (1881) described a toxic form, chiefly alcoholic, and Strümpell (1884) opened up the large study of the infective types. Finally, the modern study of epidemic poliomyelitis, by Medin and Wickmann, the recognition of syphilitic types by Freud and Plaut, and the work of Councilmann and his students have served to widen out the conception of the infectious types of Strümpell. Recent monographs of value are by Oppenheim and Cassirer, Nothnagel (1907), and Southard (Osler)¹.

¹ Modern Medicine, 1910.

Etiology.—Infections and intoxications are many and various. Among the former are found the organisms of influenza, streptococcus, typhoid, *Diplococcus meningitidis*, pneumonia, scarlet fever, measles, tuberculosis, syphilis, trypanosomiasis, malaria, rabies, poliomyelitis, etc. The commonest acute forms are due to the influenza bacillus, pyogenic organisms (external and internal infections), including mastoid, etc., poliomyelitis; among the more common chronic types are tuberculosis and syphilis, which latter, as Plaut¹ has well shown, is responsible for many of the infantile cerebral palsies. As for the intoxications, alcohol plays the chief role.

Acute Encephalitis.—*Symptoms.*—As so much depends upon the seat of the inflammatory process, it is natural that great variations in the clinical picture should be expected. The type of infecting agent also introduces a variant. The infecting agent of a polio-encephalo-myelitis, is different from that of a syphilitic encephalitis. Although similar structures may be involved and in a manner very closely related pathologically, yet nevertheless the mode of development will be different. For this reason some of the more pronounced types will receive separate consideration. Any attempt to generalize on the whole group results in a descriptive monstrosity untrue to any clinical type.

1. *Influenza Encephalitis.*—The work of Wickmann and others has shown that great care must be exercised in not confusing this with poliomyelitis. In the true influenzal type, the disorder is apt to develop in young adults some days, or even weeks, after an acute influenza. Oppenheim includes other infectious types here. The "cold in the head" seems to be clearing up, when most intense headache—often in the occiput—nausea, vomiting, apathy, or drowsiness commences to indicate something more than the usual depression of an influenzal attack. Sometimes there is a rigor. Confusion and mild stupor are frequent, the patient being aroused with some difficulty after a few days. Symptoms of meningeal irritation are not prominent, and a clear cerebrospinal fluid will separate this condition from the closely related picture of cerebrospinal meningitis. There is usually much febrile irregularity. The pulse is very variable, being not infrequently slow.

Focal symptoms develop irregularly. If the pyramidal region is involved there is premonitory weakness of the limbs, then paresis, then paralysis. Convulsive seizures may be present. The extent may be that of a monoplegia only, or a hemiplegia, which may not show in the coma. Various aphasias may occur, pseudobulbar palsies, or oculomotor palsies. Sensory anomalies are frequent, but are difficult to detect because of the mental state.

Many patients recover absolutely, others are left with slight motor defects, monoplegias, or severe hemiplegias. In a few abscess may

¹ Nervous and Mental Disease Monograph Series, 1911.

develop. In others there is a complicating ependymitis with hydrocephalus, and frequently there are psychotic complications.

2. *Polioencephalomyelitis* (Strümpell's acute encephalitis of children in part).—This has been mentioned under Anterior Poliomyelitis. The disorder shows the usual acute onset of this disease. The cerebrospinal fluid shows the characteristic bodies described by Hough and Lafora; the Wassermann reaction of the cerebrospinal fluid should be negative. The residuals here may be purely in the cerebrum, the midbrain, pons, medulla, and spinal cord escaping entirely. Various forms of infantile palsy (so-called Little's disease), idiocy, imbecility, monoplegia, etc., are to be encountered. Epileptiform convulsions are an infrequent residual of a motor zone focus. The prognosis is usually good. Many patients recover entirely, or with mild residuals. As a rule there are pontine, bulbar, or spinal complications.

3. *Polioencephalitis Hemorrhagica Superior*.—Wernicke called attention to a special form of diffuse encephalitis, with pronounced involvement of the midbrain (ophthalmoplegias). In one sense this is not a true inflammatory reaction. In the alcoholic cases the course of the affection resembles that of the Korsakow syndrome, with pronounced eye palsies. There is usually an initial delirium, sometimes appearing later. Headache, nausea, and vomiting precede the development of an irregular type of ophthalmoplegia, apparently nuclear, possibly neuritic. Ptosis, nystagmus, irregular palsies, optic neuritis are frequent. The gait is cerebellar; the speech is slurring; the mental disorientation for time and place and confabulation are marked. Somnolence and loss of sensory functions are also frequent, and point to the involvement of the thalamus. There is no fever, no leukocytosis, and a doubtful cerebrospinal fluid. The pulse is usually rapid—80 to 120—tachycardia. The nerve trunks and muscles are usually tender. Many of these patients clear up to a marked degree, but there is usually a residual mental defect, showing in a lack of initiative and mild demented states.

The lesions are those of a diffuse neuritis, with hemorrhagic foci.

A non-alcoholic variety has been described, in which somnolence is more marked than delirium. There is ptosis, complete ophthalmoplegia, with vertigo and ataxia, disorientation and frequently confabulation.

Occasionally poisoning by mussels, by fish, bromides or from rabies shows similar clinical pictures.

4. *Pyogenic Types*.—Here a multiplicity of affections is to be reckoned with. Age is no bar. Traumatism bulks high in the percentage; also ear disease. The onset is usually acute, particularly in the younger patients. Trauma, or middle ear, labyrinthine, sinus or other pyogenic infection is followed by malaise, headache, nausea, and vomiting with advancing stupor, or convulsions and increasing coma. Great restlessness, with developing delirium, usually develops within from three days to two weeks. Leukocytosis is usually present.

Lumbar puncture is usually negative, except when meningeal symptoms are also present.

Local topical signs are frequent. These are monoplegia, hemiplegia, epileptiform convulsions, cranial nerve palsies. If the delirium clears up, one may find aphasias, pseudobulbar palsies, various midbrain lesions, hemianopsias, and bulbar palsies.

Many patients die in coma. Others recover with marked mental defect (cerebral atrophies of childhood, idiocy, imbecility, debility). In others the general signs of brain abscess (*q. v.*) become apparent. Again others clear up with hemiplegias, diplegias (so-called Little's disease). Finally others recover entirely, or show small focal residual lesions.

Diagnosis.—It is impossible with present methods to clearly distinguish the various encephalitides. Many are not diagnosed during life, the course being so rapid, and facilities for laboratory research limited. Oppenheim has well said that at the present time (1911) any attempt at presenting the problem is only patchwork.

Wernicke's type is characteristic. It is, however, frequently reserved for the psychiatrist to make the diagnosis as the mental symptoms so mask the physical signs. Ophthalmoplegia, neuritis, disorientation for time and place, with confabulation, progressing to delirium or coma, the almost typical facies, closely resembling the Korsakow facies, tachycardia and no temperature are the leading features. Here the entire series of the alcoholic syndromes must be reviewed. (See Alcoholism.)

Polioencephalomyelitis is to be suspected during an epidemic. The acute onset, frequently with gastro-intestinal or nasal respiratory predecessors; the frequency of spinal and bulbar involvements; usual absence of signs of neuritis; lost knee-jerks, spinal fluid findings are suggestive.

Meningitis often calls for diagnosis. Here lumbar puncture is of great service, since many of the initial clinical signs are identical. Increased pressure, presence of cellular elements and pus are present in purulent meningitis. In epidemic cerebrospinal meningitis there is the characteristic organism.

Herpes is usually absent in encephalitis; the neck and muscle hyperesthesia usually less. Eye muscle palsies are often absent in the pyogenic types of encephalitis; they are not infrequent in meningitis. In syphilitic types the Wassermann is of great service.

Therapy.—Practically only in the malarial and syphilitic encephalitides is there any specific therapy. In the pyogenic forms, with abscess, surgery is demanded. The poisonings require withdrawal. Bromide poisoning is not to be overlooked.

All these patients require bed treatment in a darkened room. The toxic cases should have hot sheets and elimination be encouraged. Ice-bags to the head give comfort and counter-irritation; they do not

affect internal temperatures. Active catharsis is desirable, calomel and salines being advisable.

In the influenza types, salicylates are indicated. Otherwise the treatment is largely symptomatic. The residual symptoms call for their special therapy.

ABSCESS OF THE BRAIN.

History.—Brain abscesses have been recorded for many years: Cruveilhier's and Carswell's illustrations are classic. The steps that led up to the present-day conception of brain abscess are partly outlined in the section on Encephalitis. The history of these two groups is almost identical up to the appearance of Wernicke's *Lehrbuch d. Gerh rnkrankheiten*, 1883, when a division into infectious and non-infectious abscesses was foreshadowed. MacEwen's (1893) studies gave a great impetus to the understanding of the ports of entrance, while K rner's (1902–1908) studies have set in clear relief the importance of aural disease. Recent monographic treatments are those of Oppenheim and Cassirer (1909), Southard, in Osler's *Modern Medicine* (1912), and Brissaud and Soques, in Bouchard and Brissaud's *Traité de Médecine* (1904), ix.

Etiology.—Cerebral abscess is almost exclusively a secondary phenomenon due to infection. Many organisms have been found in cerebral abscess. They are derived from many sources; external trauma, the venous sinuses, and otitis media being among the commonest. Otitic infection supplies about a third, the usual communication being through the tegmen of the antrum. Metastatic abscesses, from pyemia, tuberculosis, osteomyelitis, abscess of lung, empyema, etc., are not infrequent. Actinomycosis and oïdium infections are among the curiosities. *Streptococcus pyogenes* is the most frequently found bacterium.

Symptoms.—There is always some antecedent disorder, although such may be overlooked or silent, the acute miliary tuberculosis of cardiac disease being an example. The symptoms will vary greatly with the exciting cause, the site, and the size of the abscess or abscesses. Certain traumatic abscesses run their fatal course in three to five days, whereas some recorded cases have persisted for years.

There are enough cases described to show that, speaking in general terms, one may recognize (1) a stage of irritation, (2) a stage of remission or latency, and (3) a stage of paralysis or of acute progression.

1. *Primary Stage.*—In the initial stages, general rather than localizing signs predominate. The patient usually has an acute rise in *temperature*; it is frequently slight, insidious, and variable, and often absent after a few days. The *pulse* is often slow. *Headache* is rarely absent but is very variable in intensity. It is most frequently dull, but gives a sense of tension in the head. There may be some vomiting.

Such a tense headache, with rise in temperature and slow pulse, following an otitis, after the discharge may have ceased, is of grave import. The pain is situated usually over the side affected; but is not infrequently slight or absent in cerebellar cases (*q. v.*) or here may be frontal in character. Movements are apt to increase the headache. Anxiety, insomnia, restlessness are the rule, and in younger patients, the sleep is often broken by loud cries. There may be some delirium or stupor in these initial stages, which occupy the first three or four days, occasionally a week. There is frequently some leukocytosis in this stage.

2. *Stage of Remission or Latency.*—A period of depression often sets in about this time. The headache diminishes, the fever diminishes, the patients are fatigued, somnolent, indifferent. The pulse is apt to remain slow. Percussion at this time may reveal tenderness. The blood count is variable, with a tendency to polynuclear increase. This stage shows extraordinary variation in different cases, sometimes persisting weeks or months.

In this stage beginning choked disk is not infrequent, especially if the disorder has lasted over a week or ten days. It is apt to first appear homolateral with the abscess. There are indefinite signs of a cerebral affection; variable headache, occasional nausea, vomiting, fainting attacks, or convulsive seizures.

The pupils are frequently unequal, if there is pus, and the more dilated pupil is apt to be on the side of the abscess. With maximum equal dilatation, the localization is difficult from the pupils alone, and such dilatation argues for a large abscess. With increasing size, immobility appears. Contracted pupils are found, in a few instances, toward the end in severe cases.

3. *Stage of Advance.*—After a period averaging from one to three months the symptoms of an advancing cerebral pressure manifest themselves. There are delirium, often epileptiform convulsions, all of the symptoms of a generalized meningitis, with death.

Occasionally encapsulation occurs with no symptoms for from ten to twenty-eight years¹ (Nauwerck).

Course.—The course just described is, statistically speaking, the most frequent, but it is by no means universal. In many cases there is an acute progressive course, without any intermission or remission. This may also follow a latent period, or may develop acutely after a trauma, or following a suppurative process in the ear or nose. In others there occurs an acute progressive course, with a remission which is incomplete. The chronic course is as described.

Forms.—The most marked are the traumatic, the otitic, the rhinogenic, and metastatic. In the *traumatic* cases the abscess is usually near the site of the trauma, and the symptoms of the abscess formation are apt to be complicated by the traumatic incidents. The symptoms

¹ See Oppenheim, *loc. cit.*, for literature of these rare cases.

of a leptomeningitis develop early, whereas those of a purulent destructive nature usually require from eight to fourteen days (Bergmann). The whole development is usually gradual, and the stages far from being sharply set off one from another. Headache, fever, vomiting, vertigo, confused delirium, these are the prodromal signs. The focal lesions occur several days later, either as an epilepsy, a cortical monoplegia, a speech or other motor or sensory defect.

Traumatic *late* abscesses are also known, weeks, months, even years go by, before the full truth is known.

Otitic brain abscesses are frequent, yet in proportion to otitis are rare (Jansen, 7 in 5000 cases). Here the original disorder, the localization, and the complications are of moment. The development has already been sketched. It is very insidious in many cases. The localization is usually either temporosphenoidal or cerebellar. The symptomatology of right- and left-sided temporosphenoidal abscesses or tumors offer considerable variations. A right-sided abscess may pursue its course with little or no localizing signs. Left-sided ones often give rise to aphasic complexes. Pressure upon the pyramidal tracts will give characteristic hemiplegic phenomena, there are apt to be disturbances of smell, direct or subjective, olfactory, or uncinat fits (Knapp, *loc. cit.*).

Rhinogenic abscesses are usually located frontally. In addition to the headache, there is frequently a certain degree of torpor, often associated with a tendency to joke (Witzelzucht), isolated choked disk, a cerebellar type of ataxia, when the rubrocortical fibers are implicated, isolated tremor of the hand, and olfactory disturbances. When the abscess reaches farther back, motor symptoms occur.

Diagnosis.—Many differential points arise. The etiology is the most important factor to bear in mind. Traumatic purulent meningitis, apoplexy, late apoplexy (Bollinger), pachymeningitis, brain tumor, tuberculous meningitis, syphilitic meningitis, hemorrhagic encephalitis, meningitis serosa, functional neurotic headache and sinus thrombosis are the chief disorders to be reviewed. The last often runs a very similar course, and is often associated with a purulent otitis, as a fundamental disorder. Here the fever is apt to be high and remittant, even on the same day, rigors, chills, and profuse perspiration, pulse usually rapid and irregular, convulsions rare, save perhaps in children, headache constant, eye-grounds, as in abscess, usually bilateral neuritis, more frequent than choked disk. Focal symptoms rare. Physical signs of swollen spleen, and metastatic invasions confirm the diagnosis of thrombosis.

A purulent meningitis may be more difficult to differentiate. It may be caused by similar factors; it is usually more acute and stormy in onset; it usually runs a shorter course, shows higher and more continuous fever, herpes, the pulse usually rapid, irregular, often slow in beginning as in abscess, initial unrest, irritability, confusion, delirium, in contrast to a heaviness or coma in abscess, headache constant, vomiting frequent,

convulsions common, general clonic and often muscular twitches, the eye-grounds are less often positive, meningeal irritation phenomena (Kernig, spasticity, clonus, etc.) common, local symptoms more confined to cranial nerves and basal signs rather than intracerebral focal signs, spinal symptoms frequent, whereas in abscess they are rare. Furthermore, the cerebrospinal fluid is more apt to contain globulin and show a lymphocytosis or even pus.

Prognosis.—When left alone, cerebral abscess almost invariably causes death. Calcification or external discharge occasionally occurs.

Treatment.—This is exclusively prophylactic in nose, throat, and otitic work, and surgical after the development of the abscess. The results of operative interference are highly satisfactory. The statistics vary with each new series of cases. In the hands of competent surgeons the risk from operation is *nil*. The temporosphenoidal and frontal abscesses can be readily reached and drained.¹

¹ Starr, Med. Record, 1906.

CHAPTER XIII.

DISEASES OF THE BRAIN (CONTINUED).

VASCULAR DISTURBANCES—CEREBRAL APOPLEXY.

THE quantity and quality (viscosity) of the blood, the heart action, and the size and activity of the bloodvessels are the chief factors governing the general blood flow, and hence that of the brain. Of all the bodily structures, the brain is among the most highly vascularized, arguing for its great functional activity. Furthermore, there is marked plasticity evident in the compensatory regulations. Not only is there a continuous balance maintained between different vascular systems, especially between those larger groups, such as the portal system, the vessels of the skin, the muscles, and the brain, but within the brain itself different balancing reactions are constantly going on between speech areas, visual areas, auditory areas, motor systems, etc., which are thrown into use for instance in specialized occupations or activities.

The anatomical structures used in regulating these compensating mechanisms are partly localized in the medulla and cord, as discussed in the chapters on the Vegetative Neurology of the Vascular Apparatus, but parts are within the vascular apparatus itself; at times within the walls, again within the circulating blood (viscosity disturbance).

The smaller cerebral vessels, in uniformity with the softness and plasticity of a developing organ, have less rigid walls, hence more easily overfill (congestion) and contract (fainting), are more liable to spontaneous rupture, and, furthermore, many differ in that they have few or no anastomosing branches. Thus special problems are connected with the cerebral circulation. The avenues of blood intake are through the two internal carotids and the two vertebrals. The course of the left internal carotid is more direct, and it has been taught that thrombi are, therefore, more common on the left side, which has no decisive facts to bear out the *a priori* hypothesis.

The vertebrals unite to form the basilar, which bifurcates into the two large posterior cerebral vessels which supply the temporo-occipital lobes, the corpora quadrigemina, crura, and parts of the optic thalami. The posterior communicating branches, usually small and symmetrical and subject to great variation, pass forward to join the internal carotids, and are given off to the base of the brain. The internal carotids form the middle cerebral and the anterior cerebral arteries, an anterior communicating branch completing the circle of Willis.

The middle cerebral arteries lie in the Sylvian fissure, giving off many

branches to the lenticular nucleus, the internal capsule, portions of the thalamus, and superficially irradiate the Island of Reil, Heschl's convolution, and much of the lateral aspect of the brain.

The anterior cerebral arteries supply the frontal areas, the olfactory apparatus, the upper margin of the frontal, parietal, and occipital lobes on their mesial aspects, and much of the corpus callosum.

The cerebellum derives its supply from the vertebral and basilar arteries.

Partial compensatory balance of the circulation is brought about chiefly through the circle of Willis. This circle, as well as the branches

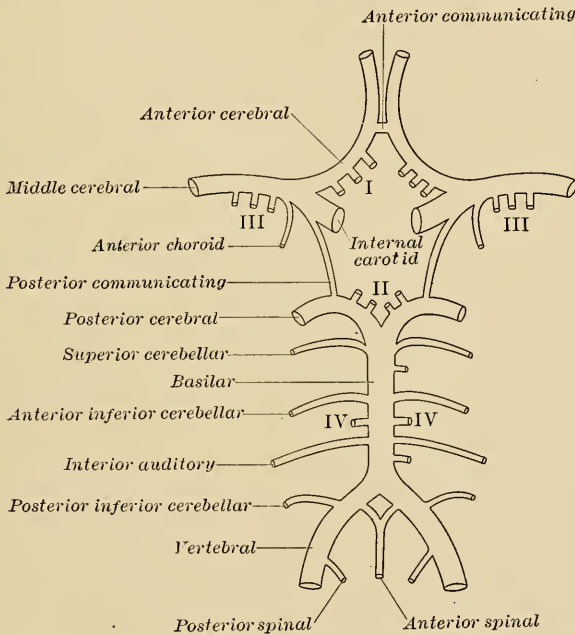


FIG. 230.—Diagram of the arteries at the base of the brain, including the circle of Willis. *I*, anteromedian group of ganglionic branches; *II*, posteromedian group; *III*, right and left anterolateral group; *IV*, right and left posterolateral group. (Gray.)

from it, shows a vast number of anomalies, and these in part determine many anomalous cerebral disturbances, possibly related, as Blackburn, Windle, Bullen, and others have shown, to faulty cerebral development, thereby laying the anatomical foundation for a psychosis or some aberrant vascular disease. Furthermore, such anomalies are of great surgical importance.¹

The chief cerebral arterial disorders arise from (*a*) temporary vascular instability (shock reactions, cardiac irregularities, internal secretory disturbances), (*b*) protracted regressive changes (arterio-

¹ I. W. Blackburn, *Jour. Comp. Neur.*, 1907, vol. xvii and xx, 1910, 185.

sclerosis, with or without miliary aneurisms), (c) hemorrhage, (d) thrombosis, (e) embolism.

Venous changes are less frequent than arterial ones. Sinus thromboses from infectious processes are the most important.

(a) **Vascular Instability.**—This is a very variable factor and has been discussed under the sections on Vegetative Neurology. Vagotonia and vagotropia occur in the cerebral vessels as well as in the somatic vessels. The results were previously grouped under the concepts cerebral anemia and cerebral hyperemia. Chronic vagotonic conditions are known. They should not be called cerebral congestions.

Anemia (Shock; noci-association—Crile) results from loss of blood, paracentesis, surgical handling of intestines, cardiac weakness, or from marked vascular instability, often of emotional origin. The symptoms are faintness, dizziness, black spots before the eyes, buzzing in the ears, and it may be loss of consciousness with or without nausea and vomiting. There may be partial consciousness, apathy, or semi-coma, with contracted pupils, and cold and clammy skin, occasionally loss of bladder or bowel function.

The therapy is heat and bandaging of the extremities, the horizontal position, camphor, caffeine, and cardiac stimulants, alcohol and ether.

Hyperemia.—Hyperemia may be active or passive. The former may result from vascular instability, or is not infrequent after excessive eating or drinking. Sudden emptying of the peripheral and somatic vessels may occasion it. It is frequent in certain thyroidisms, notably in exophthalmic goiter and in natural and artificial menopause states, often due to changes in the internal secretions. (See Vegetative Neurology.)

The symptoms of hyperemia are redness and congestion of the face and eyelids, pounding in the ears, or neck or head, headache, confusion, and usually contracted pupils. More severe attacks may lead to acute confusion, rise in temperature, and the general picture of an acute meningitis.

Signs of congestion of the face—flushing, a sense of fulness, etc., must not be taken to necessarily mean cerebral congestion. It should be borne in mind that the vessels of the face and those of the brain are controlled by separate mechanisms.

Passive hyperemias result from mechanical obstruction to the jugulars, chiefly from new growths, emphysema, and loss of compensation of heart disorders. Pressure of the head, abnormal wakefulness or sleepiness, dizziness, apathy or mild confusion, with anxiety, are the chief signs, combined with definite signs of passive congestion of the skin, face, and mucous membranes.

Efficient therapy consists in ameliorating the causative factors whether active or passive. The hyperemias are purely secondary. Acute states may call for bleeding, mustard foot-baths, active catharsis.

(b) **Cerebral Arteriosclerosis.**—This is a widespread disorder occurring chiefly after forty years of age when with increasing years it tends

to become more and more prominent. The arteriosclerotic process differs little in cerebral vessels from those located in other organs of the body, save in a tendency for a marked production of miliary aneurisms. These are largely conservative formations and are not to be considered as productive of hemorrhage, as taught by Charcot and Bouchard.¹ Cerebral arteriosclerosis is due to the same causes that produce arteriosclerosis elsewhere (heredity, syphilis, alcohol, etc.), and may be the expression of a general disease, or may be sharply delimited to the cerebral vessels.

Symptoms.—The symptoms of early arteriosclerosis may be general or localized. The chiefly early symptoms are sleeplessness, restlessness, headaches, especially if there are dizzy attacks, and renal changes, neural fatigue, increased emotionalism and irritability. To this, at times is added hebetude, ready forgetting, especially of new impressions, increased blood-pressure. These signs are often loosely spoken of as neurasthenic. Local signs may be added, such as temporary lapses, marked somnolence, tingling or numbness or other signs of focal disease.

The chief neurological interests are focussed upon the distribution of the focal lesions and are here discussed according to the syndromes presented. The psychiatric features are also dwelt upon here as well as in the special groupings under the senile and presenile psychoses.

These chief syndromes are:

I. Disorders due to disease of the twig and terminal branches.

(a) Cortical branches: irritative complexes, Alzheimer's disease.

(b) Medullary branches: lacunar complex.

(c) Diffuse types (arteriosclerotic dementias).

II. Disorders of chief branches.

(a) Anterior cerebral syndromes, crural palsies.

(b) Middle cerebral syndromes.

1. Palsies: hemorrhage, embolism, thrombosis, palsies apoplexy.

(1) Monoplegias: facial, brachial.

(2) Combined palsies.

(3) Complete palsies: capsular and supracapsular.

(4) Thalamic syndromes.

2. Aphasias.

3. Hemianopsias.

(c) Posterior cerebral syndromes.

(d) Inferior, superior, posterior, cerebellar syndromes.

III. Disorders of large trunks from obliterating lesions.

1. Basilar syndromes.

IV. Senile dementias, not necessarily arteriosclerotic.²

¹ See Pick, *Berl. kl. Woch.*, February 21, 1910, p. 325, for bibliography.

² Lambert, *States Hospital Bull.*, 1908, i, 475.

Clean-cut examples of these syndromes are rare. The patients present mixed conditions save in the few instances of embolism due to the closure of a main trunk. For this reason the general rather than the special type of apoplectic attack will be described. Special indications as to the localization of the region involved will be noted later. Pontine and midbrain localizations have already been discussed, as have also disorders of the cerebellum.

Cerebral Apoplexy.—**Hemorrhage, Thrombosis, Embolism.**—Men are more often affected (seven to five) than women, and four-fifths of the cases occur after forty years of age. Arteriosclerosis, as indicated, plays the more important role in hemorrhage and in thrombosis, and the smaller, rather than the basal, vessels are responsible. Cardiac hypertrophy with increased blood-pressure (180 to 225 mm.) is the chief determining factor, and is closely associated with the arteriosclerotic process. Chronic atrophic kidney disease is also a frequent concomitant factor (30 per cent.). Exciting causes, such as great physical exertion, lifting heavy weights, coitus, vomiting, coughing, sneezing, etc., were formerly given a prominent place. It is doubtful if they have much importance. In a large series of cases studied by Jones, in a considerable number the stroke occurred within a few minutes after getting out of bed, so that the sudden change in blood-pressure on awakening and getting about was chiefly responsible. Many patients develop apoplexy during sleep, although, other things being equal, sleep is a protection.

The localization of the hemorrhage (thrombus) varies greatly. Right and left sides are about equally involved. Morgagni's early dictum concerning the greater prevalence of right-sided hemorrhages has little support from extensive statistics.¹

The chief syndromes are of the middle cerebral type. The branches of the lenticulostriate arteries supplying the internal capsule, caudate nucleus, lenticular nucleus, and optic thalamus are most frequently involved. Thus the most frequent syndromes are the combined palsies, arm and leg; arm, leg, and face; arm; leg, face, with sensory symptoms and aphasic complexes.

Cortical hemorrhages are probably much more frequent than is usually supposed since many occur without the death of the patient. These result in more limited syndromes, such as apraxias, arm monoplegias (anterior cerebral syndrome).

Monoplegias of the leg, isolated aphasias, lower quadrant hemianopsias, and the thalamic syndrome belong more especially to the middle cerebral syndrome, while mind-blindness, and homonymous hemianopsias are the chief features of the isolated posterior cerebral syndromes.

Midbrain, pontine, medullary, and cerebellar hemorrhages are comparatively infrequent, and have a special syndrome described elsewhere.

¹ See 995 cases collected by Jones, *Brain*, 1905.

The Apoplectic Attack.—Nausea and vomiting are the most frequent precursors of the apoplectic attack. In thrombosis or embolic occlud-



FIG. 231.—Traumatic supradural hemorrhage. (Larkin.)

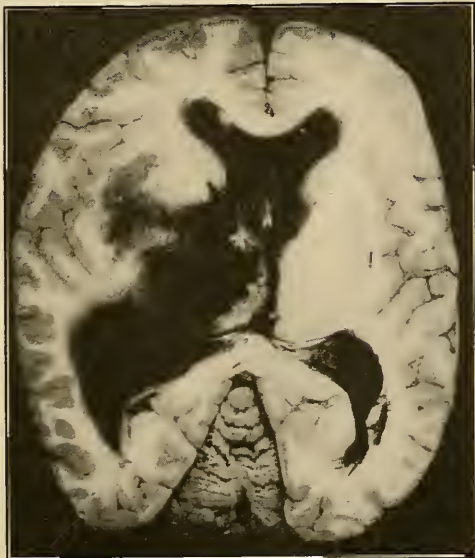


FIG. 232.—Cerebral hemorrhage within the ventricle.

ing lesions, twitchings or even convulsions are more frequent than in hemorrhagic cases. In hemorrhagic cases with convulsions the bleeding is more liable to have extended to the ventricles, and can frequently be demonstrated by lumbar puncture. This is not invariable, however.

Loss of consciousness is usual, especially in hemorrhagic cases, less often present in thrombosis or embolism, especially in the beginning. In Jones' series, 47.7 per cent. of cases of embolism showed loss of consciousness, impairment in 60 per cent. Thromboses show a similar percentage, while hemorrhage is accompanied by loss of consciousness in 75 per cent. of the cases. These figures are of minor value in determining a differential diagnosis.

The attack is usually abrupt. Dizziness, heaviness, anxiety, headache, paresthesiæ may be described by those who do not at first become suddenly unconscious, and yet in whom a gradually developing state

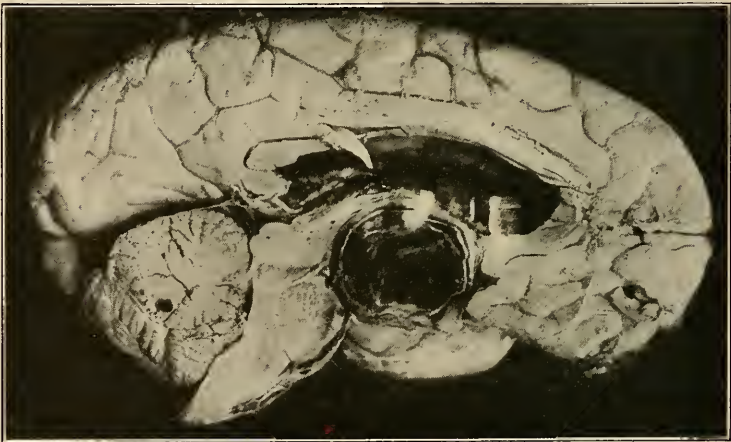


FIG. 233.—Cystic hemorrhage of hypophysis. (Larkin.)

of unconsciousness occurs, with paraphasia and gradual weakness of one side of the body. Many patients are able to describe the beginning symptoms with accuracy after recovery from even a profound coma lasting a week or more.

Coma developing is accompanied by stertorous breathing, by slow, full, regular pulse. The patient is pale, or the face may be congested, the extremities are cold. The limbs are completely relaxed. Minute variations in the excursion of the two sides of the chest, irregularity of the usually dilated and light-inactive pupils, minor signs of irritability (Marie-Foix) may enable one to locate the side of the hemorrhage even in this comatose state. Other signs are loss of corneal reflexes, no reaction to painful stimuli, loss of reflexes in general, occasionally involuntary urination and defecation. Retention is more apt to occur.

In severe states of coma with marked rise in temperature, 103° F. to 105° F., with twitching or convulsive jerks, very slow heart action, later

developing cardiac and pulmonary symptoms, the hemorrhage is probably very extensive, involves the ventricles, and death ensues in a few hours or a few days. Thalamic cases are frequently attended with marked coma, which may be very protracted, three to four weeks, without definite hemiplegic signs.

Jacksonian convulsive attacks usually indicate meningeal bleeding. Some patients show a tendency to clouding of consciousness which comes and goes.

Recovery from the immediate attack may take place in a few hours or after weeks or more. The residuals found will vary according to the location and extent of the lesion. It is in this stage that a diagnosis of the localization and extent of the lesion can be made.

In emboli, thrombi, and hemorrhage of the anterior cerebral arteries the lesion may be limited or very extensive. Apraxia may be an isolated symptom, or there may be combined apraxia and crural monoplegia.

Mild or severe and acute confusions with variously colored mental defect states are frequently the result of minute or more extensive changes in the vascular supply of the frontal lobes, also supplied by the anterior cerebrals. These patients not infrequently present lacunar symptoms when the medullary vessels are involved.¹ The clinical picture is protean, depending on the severity of the hemorrhage, or softening (from embolus or thrombus) and the distribution of these focal softenings. The course is more halting; epileptiform or apoplectiform attacks, with acute confusions occur, from which the patient is apt to make a clinical, not anatomical, recovery. The patient is usually aged forty, fifty, or sixty years. There is gradually increasing sense of incompetency. Vertiginous attacks occur, with headaches, often migrainous in character. Speech and thought associations are interfered with and minor monoplegias develop. In lacunar syndromes, apraxias, and hallucinoses result from arteriocerebral lesions chiefly; aphasias, agraphias, hemiplegias, hemianesthesias from middle cerebral lesions. General progression may result with marked mental enfeeblement—arteriosclerotic dementias—or a sudden new and more extensive lesion may cause death.

Irritative complexes from cortical vascular disease develop not infrequently, even in younger individuals following various infections (thrombi, emboli of cortical vessels), alcoholism, syphilis, lead. Here transitory mild shock may usher in the disorder with twitching, jerky, choreiform accompaniments, or these motor disturbances may later develop as residuals, with or without epileptiform attacks. Paresis, astereognosis, weakness of an arm or leg, or mild paresis of the facial musculature may develop. True epileptic residuals may be permanent symptoms of certain of these cortical arteriosclerotic focal lesions.

¹ Lambert, States Hosp. Bull., 1909, ii, 459.

The *middle cerebral syndromes* are the most frequent. Here *hemiplegia* is the most striking residual symptom. In total hemiplegia, usually capsular, supracapsular, the face, arm, and leg of the same side are involved. The upper branches of the facial, wrinkling of forehead, closure of eye are not usually involved. Nor are the eye movements involved unless the lesion is located lower down, crura or in the mid-brain, when another syndrome is present.¹ The face is drawn to the healthy side, the tongue projects to the paralyzed side on protrusion. Dysphagia, from hemiparesis of the palate develops. The soft palate hangs lower on the paralyzed side. The neck may be involved, but is less apt to be, whereas the paralysis of the arm and leg are characteristic. (See Plate VIII.)

The arm is flaccid, flabby, apt to be edematous, blue, cold, and boggy. The reflexes which during coma are lost, gradually return and shortly become exaggerated. Chaddock's wrist phenomenon develops. There is little or no atrophy, and the electrical reactions are normal. Later contractions may develop, or may be early if the lesion involves only a portion of the arm fibers or is apt to encroach on the thalamus. The abdominal reflexes are diminished or lost on the paralyzed side.

The leg is flaccid, is thrown about the hip like a flail, may be edematous and flabby, and also shows later the signs of pyramidal tract, upper neuron disorder, *i. e.*, increased knee-jerks, ankle-clonus, Babinski and Chaddock reflexes, with increased muscle tone and normal electrical responses.

The grade of the paralysis varies greatly. With some it is a transitory weakness which passes in a few weeks or a few months, in others it is a complete permanent and marked paralysis, with later developing contractures and total unilateral disability. Any intermediary grade may be expected. In the milder forms of hemiplegia the little signs—see Examination Methods—are of great value in clearing up the situation, since some mild hemiplegias may be confused with psychogenic palsies, especially in thalamic cases.

Hemianesthesia may accompany the hemiplegia, or it may be an isolated symptom. This argues for the implication of a portion of the thalamic fibers. The patients complain of numbness, coldness, and, depending on the location of the lesion, may lose their stereognostic sense, may not be able to distinguish heat, nor cold, or may have subjective sensations, haptic hallucinations of the limb areas.

Sensory disturbances from cerebral lesions are extremely diverse and important from the diagnostic point of view. They are best discussed with a consideration of the thalamic syndrome.

The Thalamic Syndrome.²—If the thalamus alone is involved or with partial implication of related extrapyramidal tracts, a characteristic neurological complex develops, termed by Dejerine and his pupil

¹ Millard-Gubler; Benedict, etc., *q. v.*

² Jelliffe, The Thalamic Syndrome, Medical Record, February 1, 1910, for references.

Roussy the thalamic syndrome. It is one of the middle cerebral artery syndromes.

The chief features show usually after an apoplectic attack with the ordinary signs of a severe hemorrhage, thrombosis, or embolism. In certain syphilitic cases the attack may be comparatively slight, or the syndrome may develop with no signs of an attack.

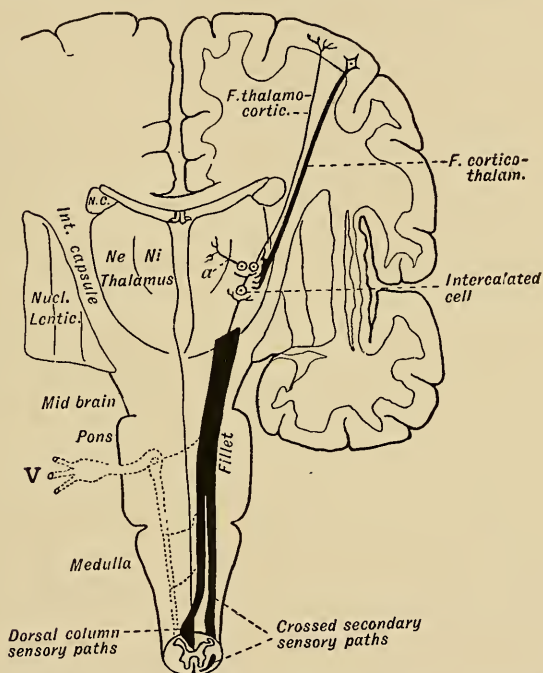


FIG. 234.—To show the position and relations of the optic thalamus in the central sensory path. Two distinct paths exist in the spinal cord; a crossed secondary path in the ventrolateral column which conveys impressions of pain, temperature, and touch, and a second uncrossed path in the dorsal column which also carries touch, and in which run impulses that underlie the sense of position, the appreciation of movement, the discrimination of two points, and the recognition of vibration, size, shape, form, weight, and consistence. This second path decussates in the lower part of the medulla oblongata, but runs separate from the first path, at least as high as the pons. All these secondary sensory fibers, now crossed, terminate in the ventrolateral region of the optic thalamus. The impressions they carry are regrouped here and, through intercalated neurons, are distributed along two distinct paths; the one carries impressions to the cerebral cortex, the other toward the more mesial parts of the optic thalamus. The corticothalamic fibers, which terminate in the lateral nucleus of the optic thalamus, are also shown. (Head and Holmes.) (Compare Plate XI.)

Practically the entire mass of sensory fibers carrying impulses of all kinds—the tests for most of which have already been outlined—have synaptic junctions within the optic thalamus. No discussion is here given of the numerous fibers coming from the chemical receptors of the respiratory, gastro-intestinal, or genito-urinary tract, nor those from the organs of internal secretion, nor even of the sympathetic—

all of these make up an enormous *terra incognita* in the thalamus or related structures.

The chief features of the thalamic syndrome are:

1. A persistent loss of superficial sensation of one-half of the body and face. This loss to touch, pain, and to temperature is more or less definite, subject to considerable variation and to partial recovery, but the loss of deep sensibility, deep pressure, postural sense, etc., is much more pronounced, and is more apt to persist. This latter is usually more marked distally and in many instances diminishes proximally.

2. There is slight hemiataxia and more or less complete astereognosis.

3. There are, in the complete syndrome, acute pains on the affected side, which are very persistent, coming on in paroxysms. They are frequently extremely severe and rarely respond to the ordinary analgesics. These pains may involve a single member, may be limited to the side of the face, simulating a trigeminal neuralgia, or they may involve one whole side of the body.

4. There is usually a more or less distinct, though slight, hemiplegia which, in the unmixed syndromes, rapidly clears up. Contractures rarely develop in the pure syndrome. In the mixed syndrome—with extension of the lesion to the internal capsule—contractures may be present.

5. Choreic, athetoid, or paralysis agitans-like movements may be present on the affected side.

These are the chief symptoms of optic thalamus disease, but in addition to these, Head and Holmes have pointed out an extremely suggestive series of affective reactions which are due to lesions which cut the optic thalamus from its cortical connections. They have opened up an attack upon the analysis of the sensory content of emotional reactions. They show that in this type of lesion there is a tendency to react excessively to unpleasant stimuli. The prick of a pin, painful pressure, excessive heat or cold, all produce more distress than on the normal half of the body. Thus, in one of Head and Holmes' patients, if a pin was dragged lightly across the face, or trunk, from the sound to the affected side, there was felt an excessive discomfort as it passed the middle line. She not only complained that it hurt her, but the face was contorted with pain, and all this notwithstanding the fact that she was less able to distinguish head from point, yet the prick hurt her more. This very anomalous state of affairs is a purely thalamic reaction.

This excessive reactivity is subject not only to pin-prick, but also to deep pressure, to extremes of heat and cold, to visceral stimulation, to scraping, roughness, vibration, tickling, to pleasurable stimuli, and to ideational emotional states. Not all patients show all of these reactions, but in practically 90 per cent. of the thalamic cases examined by Head and Holmes, excessive affective response to one or more measured stimuli were found. For heat and cold, and other forms of

sensibility as well as for pain, the excessive response may be present, and yet the patients are unable to detect—*i. e.*, are anesthetic to—the stimulus itself. So far as the ideational affective reaction is concerned, these patients express themselves as follows: On hearing affecting music, “a horrid feeling came on in the affected side, and the leg screwed up and started to shake.” The singing of a comic song left one patient absolutely cold, but a tragic song produced a very distinct unpleasant effect. One patient said, “my right hand seems to crave sympathy, my right side seems more artistic.” In practically all of the cases the increased affective reaction was accompanied by actual sensory loss.

A more detailed study of the loss of sensibility in thalamic disorders made by Head and Holmes revealed the following: No sensory functions are so frequently affected as the appreciation of posture and the recognition of passive movement. The amount of this loss varies greatly from a scarcely measurable defect to complete want of recognition of the posture of the limbs of the abnormal half of the body.

Tactile sensibility is frequently diminished, but, excepting in a few cases, where all appreciation of contact was destroyed, a threshold could be obtained. It was always possible to show that increasing the strength of the stimulus improved the proportion of right answers unless the observations were confused by the disagreeable tingling or other accessory sensations.

Localization of the spot touched was defective in half the cases where sensation was sufficiently preserved to carry out accurate tests. This inability to recognize the site of simulation was equally great, whether the patient was pricked or touched. In cases where localization was gravely affected, the disagreeable sensation, so easily evoked, tended to spread widely on the abnormal half of the body. A prick on the hand may cause an extremely painful sensation in the cheek or side, and sometimes the patient simply recognized the stimulus as a change within himself, and did not refer the discomfort from which he suffered to the action of any external agent.

Sensibility to heat and cold may show all degrees of change from total loss to a slight increase of the neutral zone. Heat and cold are not dissociated; and if one form of sensation is lost, the other will be gravely disturbed. The apparent exceptions arise from a misinterpretation of the sensation evoked by high or low temperatures on the affected half of the body.

Not infrequently the compass test cannot be carried out because of the gross loss of sensation and inability to recognize contact; but whenever this method can be applied a threshold can be worked out, and widening the distance between the points increases the accuracy of the answers.

The power of estimating the relation between two weights is frequently disturbed on the abnormal half of the body. If the appreciation of posture and movement is affected, the patient can no longer

recognize the identity or the difference of two weights placed in the unsupported hands. But so long as tactile sensibility is not diminished, he can still estimate the relation between weights applied one after the other to the same spot, and can recognize the increase or diminution in weight of an object already resting on the hand.

The appreciation of relative size is often disturbed in these cases. With care it is usually easy to demonstrate a difference in the threshold. Shape and form in three dimensions are frequently not recognizable on the affected hand. But, if tactile sensibility is not grossly affected, the patient usually retains an idea that the object possesses a form, and may give a considerable percentage of right answers.

Vibration of the tuning-fork in almost all thalamic cases is felt, but the length of time during which it is appreciated is usually shorter, and sometimes the rate of vibration is thought to be slower on the affected half of the body.

Roughness, as tested with Graham Brown's esthesiometer is usually recognized, except in those cases in which the loss of all forms of sensation is unusually severe.

Partial syndromes are more frequent than the classical syndrome described. To summarize: the main symptoms of the complete syndrome are:

1. Persistent hemianesthesia, more or less marked for superficial sensibility, tactual pain, temperature, most marked for deep sensibility, and hence loss of postural sense and astereognosis.
2. Mild hemiplegia, usually regressive.
3. Mild hemiataxia, with choreo-athetoid movements either spontaneous or on an attempted movement. *Adiadokokinesia* at times.
4. Paroxysmal, shooting, neuralgic pains, often very persistent and severe and not helped by the usual analgesics.
5. Over-response to affective reactions, particularly on affected side, with emotional hyperactivity to varied sensory impressions.

The analysis of sensory stimuli in cerebral and thalamic disorders is of the greatest importance in cerebral localization, hence an extended presentation of contemporary work is desirable, particularly that of Head and Holmes.

Sensory Changes in Cortical Lesions, Supra-thalamic Pathways.—In the chapters on Peripheral Nerves the chief sensory syndromes of the first sensory neurons were discussed. Those of the cord are presented elsewhere. Redistributions take place in the medulla and mid-brain. Those disturbances due to lesions within the thalamus have just been presented. Finally the thalamocortical pathways make a fifth regrouping of sensory phenomena in the entire course of the sensory neuron. (See Plates X and XI.)

The analysis of these phenomena introduces complex factors, and it is necessary to abandon all generalizations, even, for instance, light touch, cutaneous sensation. The results of the tests must be stated in terms of the tests employed. (See Sensory Examination Methods.)

Using graduated tactile stimuli, such as von Frey's hairs and the pressure esthesiometer, the chief results obtained by Head and Holmes and others have been that a cortical lesion may reduce the accuracy of response from the affected part to graduated tactile stimuli. The form assumed by this defective sensibility differs from that produced by lesions at other levels of the nervous system. The affected part may respond to the same graduated hair as the normal hand; but this response is irregular and uncertain. Increasing the stimulus may lead to no corresponding improvement, and even the strongest tactile hair may occasionally evoke less certain answers than a hair of much smaller bending strain. Moreover, a touch with the unweighted esthesiometer may be as effective at one moment as the same instrument weighted with 30 grams at another. In such cases no tactile threshold can be definitely obtained. This irregularity of response is associated with persistence of the tactile sensation and a tendency to hallucinations of touch. Where the sensory defect is not sufficiently gross to abolish the threshold, persistence, irregularity of response and a tendency to hallucinate may still disturb the records.

In all cases where tactile sensibility is affected, whether a threshold can be obtained or not, fatigue is induced with unusual facility. Although the patient may cease to respond to tactile stimuli over the affected part in consequence of fatigue, his answers may remain as good as before from the normal parts. The fatigue is local and not general.

With stationery cortical lesions, uncomplicated by states of shock or by "diaschisis," sensibility to touch with cotton-wool is never lost over hair-clad parts. Over hairless parts, stimulation with cotton-wool may produce a sensation which seems "less plain" to the patient, and his answers may show the same inconstancy so evident when he is tested with graduated tactile stimuli.

A pure cortical lesion leads to no change in the threshold to measurable painful or uncomfortable stimuli. Nor does the patient express greater dislike to these stimuli on one side than on the other. A prick may be said to be "plainer" or "sharper" on the normal than on the affected side; but this is due to a defective appreciation of the pointed nature of the stimulus and bears no direct relation to the painfulness of the sensation evoked.

Temperature tests show that (a) the neutral zone, within the stimulus, was said to be neither hot nor cold, was considerably enlarged in comparison with that observed on similar normal parts of the same patient. (b) The patient complained that, although he recognized correctly the nature of the stimulus, it seemed "less plain" than over normal parts. His answers were less constant, and less certain; a temperature recognized without difficulty at one time seemed doubtful at another. (c) The power of discriminating the relative coolness of two stimuli or the relative warmth of two hot tubes may be diminished. Thus 20° C. may be said to be the same as ice, although both

are uniformly called cold, and 40° C. may seem as warm as or even warmer than 48° C. The faculty of appreciating the relation to one another of two temperatures on the same side of the scale is disturbed.

Tests for *posture* and for *passive movements* show that (a) cortical lesions most frequently disturb the recognition of posture and of passive movements. Whenever sensation is in any way affected in consequence of a cortical lesion these two functions suffer. (b) In all their cases the disturbance in the faculty of recognizing posture and passive movements was greater toward the peripheral parts of the affected limb. (c) When a patient with unilateral disturbance of these faculties attempts to point to some part of his body, defective knowledge of its position causes greater error than want of recognition of posture and movement in the hand with which he points. (d) When testing the patient's power of appreciating passive movement, the answers are frequently uncertain and hallucinations of movement may occur. And yet the patient may be remarkably consistent and accurate when normal parts are tested.

Localization tests show that (a) the power of localizing the stimulated spot is not infrequently preserved, although sensation may be otherwise disturbed as a consequence of cortical lesions. (b) This faculty is independent of the power of recognizing the position of the affected limb; appreciation of posture may be lost, although localization is not in any way diminished. (c) If the power of localization is lost, the patient will be unable to recognize not only the position of a spot touched but also the position of a prick. (d) When localization is defective in consequence of cerebral lesions, the patient does not habitually localize in any particular direction, but ceases to be certain where he has been touched or pricked.

The *compass test* shows that (a) a cortical lesion may destroy the power of discriminating two compass points, both when applied simultaneously and collectively. If this is the case, no threshold can be obtained for either form of the test; increasing the distance between the points does not constantly improve the accuracy of the answers. (b) This disturbance is not caused by changes in tactile appreciation; for it can be demonstrated equally well with two painful as with two tactile stimuli. (c) The condition of tactile sensibility and the accuracy of the simultaneous compass test are closely associated; a disturbance of the tactile threshold is usually accompanied by a raised threshold for the appreciation of two points applied simultaneously. (d) Should the power be preserved of recognizing two points when the compasses are applied consecutively, localization will be found to be intact. The patient's appreciation of the two points when they are separated by an interval of time is due to the recognition of the separate locality of the two spots touched.

Appreciation of weights shows that (a) the power of estimating the relation of two objects of the same size and shape is readily disturbed by cortical lesions. (b) Though the patient may retain sensations of

contact when the weight is placed in his hand, all power of recognizing the relative heaviness of the object has disappeared. (c) This faculty is equally disturbed in most cases whether the weights are placed on the supported or the unsupported hand.

From these and related studies, Head and Holmes maintain that sensory impulses pass from the thalamus to the cortex in five groups:

1. Those concerned with the recognition of posture and passive movement. If these impulses are affected the power of discriminating weights on the unsupported hand may be also diminished.

2. Certain tactile elements; integrity of this group is necessary for the discrimination of weights placed on the fully supported hand.

3. Those impulses which underlie the appreciation of two points applied simultaneously (the compass test); on this group also depends the recognition of size and shape.

4. Those which underlie the power of localizing the situation of a stimulated spot. Recognition of the double nature of two points applied consecutively also depends on this group of impulses.

5. All thermal impulses are grouped together to underlie a scale of sensations with heat at the one end and cold at the other. At the level with which we are now dealing these impulses have already excited the affective center and are passing away to the cortex.

The functional integrity of the cortex enables attention to be concentrated upon those changes which are produced by the arrival of afferent impulses (Head and Holmes). When this is disturbed, some impulses evoke a sensation, but others, from lack of attention, do not affect consciousness. Attention no longer moves freely over the sensory field to be focussed successively on fresh groups of sensory impressions. Sensations, once evoked, are not cut short by the moving away of the focus of attention as when cortical activity is perfect. Hence arise persistent sensations and hallucinations which are so prominent a feature of lesions of the cortex.

The cerebral cortex is the organ by which we are able to focus attention upon the changes evoked by sensory impulses. A pure cortical lesion, which is not advancing or causing periodic discharges, will change the sensibility of the affected parts in such a way that the patient's answers appear to be untrustworthy. Such diminished power makes the estimation of a threshold in many cases impossible. Uncertainty of response destroys all power of comparing one set of impressions with another and so prevents discrimination.

But in addition to its function as an organ of local attention the sensory cortex is also the storehouse of past impressions. These may rise into consciousness as images, but more often, as in the case of spacial impressions, remain outside central consciousness. Here they form organized models of ourselves which may be termed "schemata." Such schemata modify the impressions produced by incoming sensory impulses in such a way that the final sensations of position, or of locality, rise into consciousness charged with a relation to some-

thing that has happened before. Destruction of such "schemata" by a lesion of the cortex renders impossible all recognition of posture or of the locality of a stimulated spot on the affected part of the body.

In daily life all stimuli excite more or less both thalamic and cortical centers, for most unselected sensations contain both affective and discriminative elements. But among the tests employed in sensory analysis, some appeal almost entirely to the one or the other center. The test for recognition of posture, as carried out by Head and Holmes, is purely discriminative; while the pain produced by squeezing the testicle, or to a less degree by the pressure algometer, appeals almost exclusively to the more affective center.

Sensory impulses arriving at the optic thalamus are regrouped in such a way that they can act upon both its essential center and the sensory cortex. The essential organ of the thalamus is excited to affective activity by certain impulses, and refuses to react to those which underlie the purely discriminative aspects of sensation. These pass on to influence the cortical centers where they are readily accepted. In a similar way, the primary centers of the cortex cannot receive those components which underlie feeling tone; in this direction they are completely blocked.

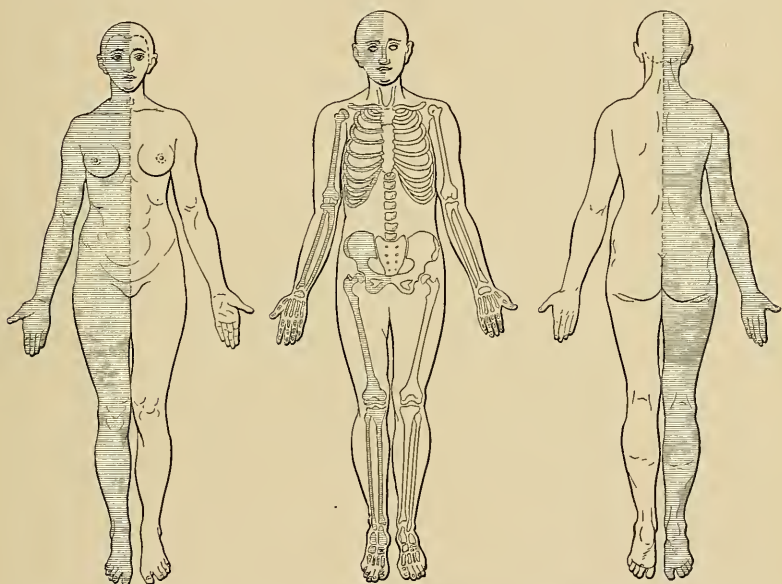
It has long been recognized that sensations are endowed with feeling tone to different degrees. In those which underlie postural appreciation this quality is entirely absent, while visceral sensations are, in some instances, little more than a change in a general feeling tone; the former set of impulses appeals almost exclusively to the cortical center, the latter to that of the optic thalamus. All thermal stimuli, however, make a double appeal. Every sensation of heat or cold is either comfortable or uncomfortable; the only entirely indifferent temperature is one that is neither hot nor cold.

In the same way, some unselected tactile stimuli appeal both to the sensory cortex and to the optic thalamus. For not only is a touch always related to, and distinguished from, something that has gone before it, but we have shown that contact, especially of an object moving over hair-clad parts, is capable of exciting thalamic activity. Vibrations of the tuning-fork also make a double appeal, for when the cortical paths are cut the amplitude of the vibration must be greater in order that it may be appreciated; on the other hand, the vibratory effect may be stronger on the abnormal side in those thalamic cases where the affective response is excessive.

But these two centers of consciousness are not coequal and independent. Under normal conditions the activity of the thalamic center, though of a different nature, is dominated by that of the cortex. When the sensation normally produced by a prick is examined it is recognized that the pain develops slowly and lasts a considerable time after the stimulus has ceased. Moreover, the same intensity of stimulation will produce a different effect on the same spot on different occasions. A long latent period, persistence and want of uniformity

are characteristic of all painful sensations. This is seen in an exaggerated form in cases where the thalamic center has been freed from control. The response to prick is slow, but persists long after the stimulus has ceased. Moreover, the reaction, when it occurs, tends to be explosive; it is as if a spark had fired a magazine and the consequences are not commensurate with the cause.

On the contrary, the sensations normally produced by moderate tactile stimuli are characterized by a short latent period, and disappear



FIGS. 235, 236, and 237.—Topography of the sensory disturbance, in a case of the thalamic syndrome, right side, of six years' duration in a woman, aged fifty-five years. There is mild hemiplegia of the right side with intense choreo-athetoid movements of the hand and foot. There is a marked ataxia in all voluntary movements of the same side. Exaggeration of the tendon reflexes of the right side. No Babinski reflex.

There are very active and severe spontaneous pains over the entire right side, tactile, pain, and thermal sensibility is diminished, but not abolished, on the entire right side. Compass discrimination markedly affected. Complete astereognosis. Taste, smell, and hearing are diminished on the right side. Vision is unimpaired and there is no hemianopsia. Deep sensibility is more altered than superficial. The sensory position is altered, bony sensibility is much diminished. (Thomas and Chiray.)

almost immediately on the cessation of the stimulus. A lesion of the sensory cortex disturbs both these characteristics. Tactile sensations become uncertain and incalculable, and no threshold can be obtained; persistence and hallucinations mar the uniformity of the records.

The work of Head and Holmes tends to show that the sensory cortex is the organ by which attention can be concentrated on any part of the body that is stimulated. The focus of attention is arrested at any one spot by the changes produced by cortical activity. These are

sorted out and brought into relation with other sensory processes, past or present. Then the focus of attention sweeps on, attracted by some other object.

All stimuli which appeal to the thalamic center have a high threshold. They must reach a high intensity before they can enter consciousness, but once they have risen above the threshold they tend to produce a change of excessive amount and duration, and this it is the business of the cortical mechanism to control. The low intensity of the stimuli that can arouse the sensory cortex, and its quick reaction period, enable it to control the activity of the cumbersome mechanism of the thalamic center.

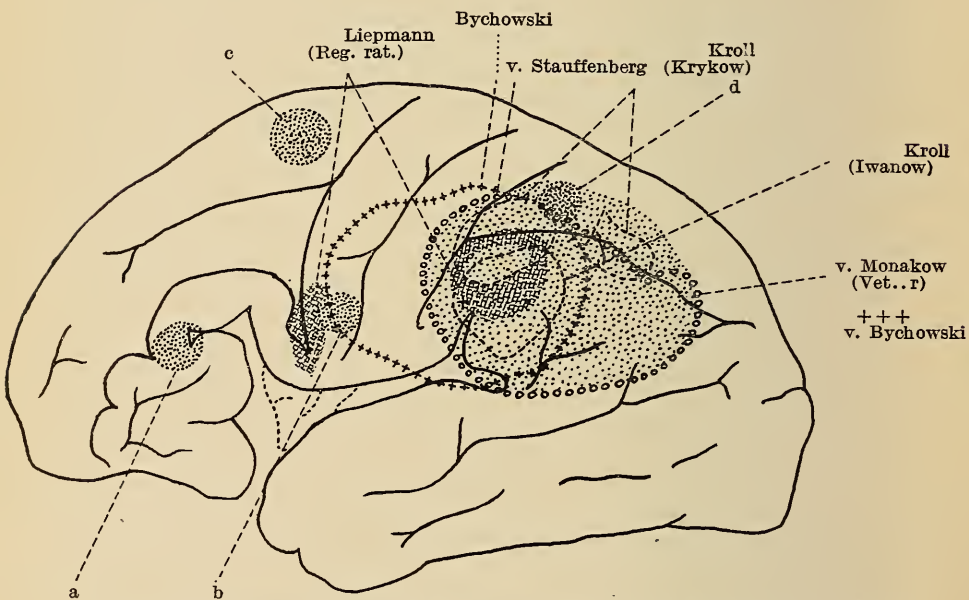


FIG. 238.—Localization of apraxia. Positive cases from lesion in the left supra-marginal gyrus and (a, b, c, d) small metastatic tumors from one case of total bilateral apraxia. In this case foci were also found in both optic thalami. (von Monakow.)

This view of the sensory mechanism explains many of the facts recognized by neurologists and psychiatrists. It enables one to understand how integrations can occur at all afferent levels of the nervous system, and makes development possible even in the individual. The aim of human evolution is the domination of feeling and instinct by discriminative mental activities. This struggle on the highest plane of mental life is begun at the lowest afferent level, and the issue becomes more clearly defined the nearer sensory impulses approach the field of consciousness.

Apraxia.—This term was first used by Gogol in 1873, in a Breslau thesis on Aphasia. His patient ate his soap, urinated in his water

pitcher, and was described as having lost his understanding for objects. Such defects had been noted before, and it is worthy of note that Hughlings Jackson, in 1866, called attention to a similar type of phenomenon, and attached much importance to it. Quaglino, in 1867, described a case, Finkelnburg, in 1870, another, in which recognition of things and people was lost, and created the term *asymbolia*. Wernicke, in 1874, expanded the term *asymbolia*, while Freud finally utilized the term *agnosia*, to cover all types of loss of sensory or motor object images, apraxia then being arranged as a form of loss of knowledge of objects, really a form of visual agnosia.

From this early use of the term apraxia, there has been a distinct variation, brought prominently into the foreground by Liepmann in 1900. He defined the disturbance as a lack of knowledge of the use of objects, although there was no true agnosia, or loss of recognition of what they were. Out of the later studies of Liepmann, Pick, von Monakow, d'Hollander and others has come the following general definition of apraxia:

It consists in an inability to perform certain subjectively purposeful movements, or movement complexes, the motor power, sensation, and coördination being intact. Such an inability will naturally depend upon at least three factors; one may be unable to recognize the object which is to be used, in which case we can speak of a sensory apraxia, in the same sense as one speaks of a sensory aphasia, or a visual agnosia. Should the patient recognize the object, call it perhaps by name, state its use, and yet in attempting to use it totally fail in proper motor act, one speaks of a motor apraxia. It being understood here that there is no necessary change in the motor arc, either on the incoming sensory or outgoing motor side. Thus one can speak, as Wilson has done, of a motor aphasic, as having an apraxia of his speech musculature. In grave intracerebral changes the knowledge of the proper kinetic images to carry out purposeful actions in the arms and legs may be complexly involved. Here one speaks of an intrapsychic apraxia. Clinically it is usually overlaid in the general psychic loss, and is often included loosely in the term dementia.

Apraxia may be then either sensory or motor; it may be unilateral or bilateral, it may be extensive, involving many muscular groups or may be limited to a few, such as an inability to protrude the tongue on demand or close the eyelids, etc., with perfect power in other movements.

A certain patient with motor apraxia, on being given a cigarette holder and cigarette, recognized the objects, said they were for smoking, but on being told to put them together was unable to make the correct movements, and finally gave up. Another patient was given a candle and a match-box. She took out a match, made rubbing movements with it in the air above the candle, and then reinserted it in the box.

Liepmann's celebrated case was able to do things with his left hand, but failed entirely with his right. When told to brush the examiner's

coat, he picked up a corner of it carefully in his left hand, then picked up the brush in his right hand, with which he made movements as if to brush his hair. Asked to pour water into a glass from a carafe,

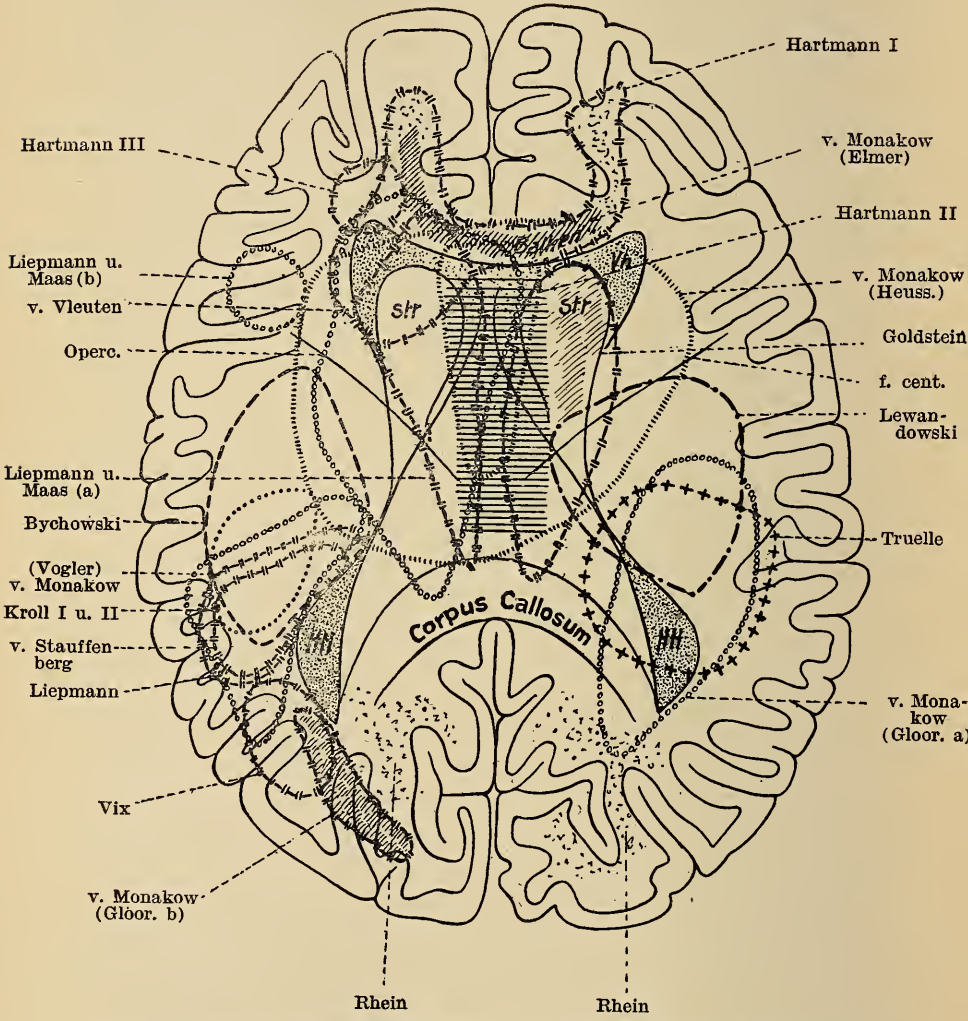


FIG. 239.—Site of the lesion in 20 cases of apraxia marked according to the author's localizations. The shaded places in the illustration indicate the spreading of the lesion in certain cases. *Str*, corpora striata; *VH*, anterior horn of ventricle; *HH*, posterior horn. (von Monakow.)

he grasped the carafe with his left hand, to pour water into the glass held in the right hand, after which the glass was brought to the mouth without any water in it. These patients fail to carry out the simple commands to blow a kiss, make a threatening fist, soldier's salute, etc.

In ideomotor apraxia the situation is more complicated. One patient given a tooth-brush recognized it, then began to brush his beard with it clumsily; another being given a pistol, which he named correctly, on being told to shoot it, grasped the barrel, blinked and put the muzzle into his left eye. Another patient, being given a cigar and a match-box opened the latter, stuck the cigar in it, and tried to shut the box as though it were a cigar cutter. Then taking the cigar out rubbed it on the side of the box as though it were a match. The entire order of procedure was badly devised.

Apraxia is usually an accompaniment of certain apoplectic attacks. The lesions center about the fibers of the corpus collosum and the medullary fiber areas from the second parietal region.

In left hemisphere disease, the apraxia may be homo- or heterolateral, and in homolateral apraxias the corpus callosum is usually involved. In left frontal disease apraxia has been found especially with lesions of F.1 and F.2. Anything that brings about an isolation, diaschisis, of the left frontal area from the right frontal area will seem to bring about an apraxia.

Hemianopsia is an infrequent sign occurring as a result of the apoplexy. As a symptom of middle cerebral disease it may result from thalamic lesions (pulvinar, geniculates) and is usually a permanent residual. It is rarely a temporary condition but may show marked improvement. The type is a bilateral homonymous hemianopsia. Quadrant hemianopsias more often belong to the middle cerebral syndromes, although limited lesions of the cuneus may cause quadrant hemianopsias.¹

The visual fields will vary greatly according to the tract involvement. In posterior cerebral syndromes occipital cortical types of hemianopsia are present. These show very irregular visual fields. Psychical blindness (loss of visual memories) may also result from posterior cerebral involvements. Here the portions of the occipital cortex about the calcarine fissure and cuneus are implicated. It is usually an accompanying symptom of the apoplectic attack but may be isolated and often combined with a hemianopsia.

Aphasia is a frequent result of the hemiplegic attack (right hemiplegia in right-handed persons; left-sided in left-handed persons).

The type of aphasia depends entirely upon the location of the vessels and the areas served. Transitory aphasias are extremely common, occurring in at least one-half of the attacks. Permanent aphasias are rarer, about one-half of the patients recovering. The different forms of aphasia and the localization significance are discussed under Disorders of Speech. (See p. 237.)²

Papillary edema, even choked disk, may be a symptom of apoplexy,

¹ Compare Willbrand and Saenger, *Die Neurologie des Auges* for complete discussion and bibliography; also, Henschen, in Lewandowsky's *Handbuch der Neurologie*.

² Comp. v. Monakow's *Die Lokalisation im Grosshirn*, 1914, for most recent discussion of the complicated aphasia question.

and is found on the side affected. It is most frequent with large lesions.¹

Diagnosis.—A separation of hemorrhage, thrombosis, and embolism is highly desirable, although not always possible. The end-results are identical, but the antecedent conditions vary. Hemorrhage is comparatively rare in the young, but it may be seen at all decades, and even in intra-uterine life. When occurring in early youth it is usually a result of parental syphilis or some acute illness. In cases of hemorrhage the patient is usually over forty, shows signs of arterial disease, frequently with hypertrophied heart, and often nephritis. The attack is usually sudden, often preceded by emotional disturbance or sudden change in position that modifies the blood-pressure. The attack is more likely to be accompanied by unconsciousness (75 per cent.). Severe ventricular hemorrhages show blood on lumbar puncture, but this procedure is rarely called for save in supposed traumatic cases.

Thrombosis occurs also in older individuals, and in syphilitics particularly. Unconsciousness is less apt to occur, or occurs in stages as it were. The prodromata already mentioned are more apt to have been present. Markedly atheromatous arteries speak for thrombosis, and mild signs of deterioration—lacunar syndromes—speak for thrombotic types of disease.

Embolism is almost invariably associated with some acute disease—typhoid, acute septic infections (articular rheumatism, gonorrhea, scarlet fever, malaria, etc.). Acute endocarditis is often present and the individuals are apt to be young. Unconsciousness is less apt to occur; when occurring it is likely to be acute, and is more often present in basilar and carotid occlusions than when other arteries are blocked.

Hemorrhages, especially when small, frequently show the maximum symptoms early, with gradual betterment, while thromboses usually show the reverse, the symptoms having a tendency to spread or to deepen.

Ophthalmoscopic examination is always imperative. Dilatation of the pupil, usually present, can be obtained by cocain in a few minutes. Diffuse retinitis, so-called retrobulbar neuritis, is highly indicative of basal syphilis or syphilitic endarteritis. In hemorrhage the vessels of the disk are apt to be engorged. This is not so in embolism nor in thrombosis. Choked disk points to a neoplasm.

The blood-pressure affords diagnostic criteria. It is apt to be high in hemorrhage and in embolism, but low in thrombosis, also in neoplasms, syphilitic endarteritis, and cerebral abscess.

Other disease processes to be distinguished are hysterical hemiplegias, syncope, epileptic attacks, general paresis, uremic, alcoholic, or encephalitic coma.

¹ Uthhoff, Neurol. Centralblatt, 1909.

Hysterical hemiplegias are rarely accompanied by unconsciousness. Hysterical delirium may complicate the picture. Later the signs of pyramidal tract involvement, such as increased reflexes, Babinski, Chaddock, loss of abdominal reflexes, Grasset and Hoover phenomena are not present in hysterical hemiplegia. In certain mild thalamic cases the sensory changes may be regarded as hysterical, but careful summing up of the results outlined on pages 55-63 will show the involvement of the thalamus. Personal experience has shown that many thalamic cases are diagnosed as hysterical.

Certain lacunar syndromes, especially in the anterior cerebral distribution, which are mild and which present momentary confusion (worse at night), emotional instability, irritability, tendency to weeping, etc., are frequently termed hysteria.

Lacunar syndromes with softenings in the frontal areas are also mistaken for *manic* attacks. There is confusion, some disorientation, excitement, and after a short time the whole thing may clear up, leaving only slight defect, easily overlooked, unless careful intelligence tests are applied (presbyophrenic excitements, presenile excitements—see Senile and Presenile Psychoses.)

Uremic coma is usually of more gradual onset. There are preceding signs of heaviness and toxemia; convulsive movements are not infrequent, with little signs of irregular respiration of the two sides of the chest, possible pupillary inequalities, possible variations in response to sensory stimuli on the two sides, involuntary reflex motor responses to joint squeezing and finger squeezing (Marie-Foix) are usually absent in uremic coma. The general odor of the patient is often characteristic in uremia. Some uremic patients develop apoplectic attacks as well.

Diabetic coma shows similar difficulties. Acetone odor, large amounts of sugar-containing urine, slow onset, and the previous history must be relied upon.

Alcoholic coma is frequently diagnosed by the police as apoplexy, since many alcoholics do have apoplectic attacks. Some severe intoxications resemble apoplectic coma very closely, but, as a rule, the coma is less profound in alcoholism; the reflexes are often preserved, in a measure, and the uniformity in the bilaterality of the relaxation is evident (absence of Marie-Foix signs). One should be on one's guard, however, and look for all of the little signs of hemiplegia.¹

Epileptic and syncopal attacks rarely present great difficulties. The history of a previous attack, the scarred body or head and tongue of the epileptic is often evident, while in syncope the coma is usually shallow and the feeble respirations and superficial heart action point to the difficulty.

An apoplectiform attack may be the first sign of *paresis*. It is a cerebral edema, and may be at first indistinguishable from an apoplexy (non-paretic). The later history and examination will usually

¹ Dejerine, *Semiologie*, 1914, 2d ed.; Jelliffe, *Little Signs of Hemiplegia*, Postgraduate, 1912.

establish a diagnosis, although the pseudoparesis of arteriosclerotic softening is often only distinguishable from paresis by the cytobiological four reactions. (See chapter on Syphilitic Diseases of the Nervous System, p. 511.)

Prognosis.—Recovery from the attack and amelioration of the residual symptoms are separate problems.

Cerebral hemorrhage is usually more immediately fatal than either thrombosis or embolism. Deepening coma, Cheyne-Stokes' respiration, irritative phenomena, jerking, convulsions (very high, over 230 mm. or very low, under 90 mm), blood-pressure, marked rise in temperature are the usual lethal signs. General convulsions, retinal hemorrhages, blood on puncture, bilateral paralysis are unfavorable signs. After recovery from coma, continued temperature, advancing symptoms, restlessness, delirium, loss of sphincter control, trophic disturbances, indicate a grave prognosis and probable death in from two to three weeks.

Early attacks of lacunar softening are rarely fatal, but indicate that a fatal termination from a more severe type of attack will be probable in from one to three years. Inasmuch as this is a form which is usually called hysterical in the early mild attacks, sometimes diagnosed as a mild manic attack, from the excitement and confusion, one should be on one's guard in this not infrequent syndrome.

The recovery from the residual symptoms will vary greatly upon the nature, localization, and extent of the lesion. A careful plotting of the entire symptomatology will determine the area involved in the destructive process, and one must first separate out the effects of diaschisis from those of actual tissue destruction, since the former are more apt to disappear.

Aphasias are usually recovered from almost invariably in left-sided hemiplegias in the right-handed. In right-handed hemiplegias aphasias are usually recovered from in about one-half of the patients, especially in the intelligent, who will make an effort to reëducate themselves. If the lesion lies directly within the aphasic area the chances for recovery are less.

Hemianopsias are not infrequently diaschitic. When so they pass within a few weeks. In thalamus lesions they are apt to be permanent, and cortical hemianopsias persisting over a few weeks are apt to be permanent.

Hemiplegia, if total, is apt to persist in some degree at least, although most patients are able to get about in three months or more. Helplessness persisting over six months or a year has been partially recovered from. Facial palsies usually recover. Leg palsy is rarely as persistent as arm palsy, but both react favorably to appropriate treatment. Early contracture is a bad prognostic feature.

Thalamic involvement is a bad prognostic feature—the pain and irregular movements (choreo-athetoid), hemianesthesia, etc., usually persist.

Mental defects may clear up almost entirely, especially when slight, but careful intelligence tests (see chapter on Mental Examination) should be utilized in all cases, especially to decide medicolegal problems which may arise—testamentary or contract capacity, responsibility, etc. A sensory or motor aphasia alone is not necessarily a sign of an intelligence defect; many aphasics are very intelligent. They simply cannot utilize, in speech, the knowledge they have. (See chapter on Senile and Presenile Mental States.) A motor aphasic who cannot get his symbols over, as it were, may not be any more of a dement than an Englishman trying to make a Chinaman understand what he is saying.

Treatment.—Prophylaxis applies particularly to those over fifty years with arteriosclerosis, and sustained high blood-pressure. The tendency to hemorrhage here is great. Such patients should slow down somewhat in their work, if strenuous and calling for intense and sustained effort, and especially if emotional calls are frequent. A moderate amount of work and freedom is desirable. Invalidating a prospective hemorrhagic case is inadvisable.

Dietary faddism is to be avoided. Excessive eating is to be avoided and all alcohol should be restricted. Protein-free diet—vegetable proteins are the same as animal—keeps down the blood-pressure in many cases. Some search should be made to see if specific protein sensitization exists.

Careful regulation of the gastro-intestinal tract is called for, the kidney functions should be scrutinized, and the liver metabolism regulated.

It is doubtful if drug therapy is of any service in prophylaxis. The iodides have been used widely, but their utility is still undecided.

Treatment of the attack, even if there is no unconsciousness, requires immediate rest in bed if possible. If stricken away from home the patient should be moved as little as possible. With high tension, hot foot-packs will help to reduce it (not hot enough to burn the unconscious patient). In cases in which the blood-pressure rises steadily, keeps above 250 mm. and with very deep coma, blood-letting (10 to 12 oz.) is advisable.

Hypodermic medication by blood-pressure reducing drugs in hemorrhage is alone advisable in coma, and only very small quantities of water should be used, as water raises blood-pressure. Hydrochlorate of gelsemine in doses of $\frac{1}{15}$ grain is fairly active and reliable. The nitrites are not available.

If the patient can swallow, tincture of aconite in \mathfrak{M} v doses may be given, watching the blood-pressure. The dose may be repeated in an hour. Pressure should be kept below 200 mm. if possible. Tincture of veratrum viride in \mathfrak{M} v–xv, every two hours, or the fl. ext. of gelsemium in same doses at same intervals.

Pressure-reducing drugs should be used with caution. The high pressure following hemorrhage is usually compensatory and for the

purpose of keeping up an effective vascular irrigation of the medullary nuclei following a rupture in the arterial pipe line. These drugs should be used only when it is known that a high pressure preceded the attack.

Early purgation is desirable; 2 gtt. of croton oil is useful in states of deep coma. This may be placed with butter on the back of the tongue.

If there is marked excitement, chloral, gr. v-xv, or paraldehyde, ʒj-ij, may be given by mouth or by rectum. Tepid sponge baths help restlessness. Veronal and trional are useful in the restlessness of lacunar softenings.

Care of the mouth, drooling, moving the patient, removal of all obstructions to breathing, should be attended to at once.

Nourishment may be omitted in the early stages. A purin-free diet, mostly milk and gruels, to which sugar and eggs may be added later, should constitute the standard diet. In the presence of dysphagia, milk enemas are to be given.

In cardiac atonic cases, with low blood-pressure—chiefly thromboses—vasodilators, camphor, and alcoholic stimulants may be necessary.

Surgical treatment may be advisable for meningeal or subcortical hemorrhages. Certain nephritic cases are helped by lumbar puncture, and the withdrawal of 20 to 50 c.c. of fluid. The patients should be kept quiet several weeks, attention being paid to giving ease and comfort by cushions, props, supports, and frequent changes of position.

The late treatment should be gradually adapted to the residual symptoms. The gastro-intestinal tract and the skin need special attention. The care of the paralyzed muscles by systematic exercises, the avoidance of contractures and treatment of shoulder, of hip, or knee arthritides constitute the major portion of the late therapy.

Electricity is of doubtful service, save as a psychic aid to the general management of the case. Galvanism aids some of the pains, the anode should be over the painful area.

The general care of the invalid will depend largely upon his economic status. Travel is helpful in supplying a mental stimulus and the warmer climates and more interesting foreign resorts are enjoyable, and thus of direct therapeutic value. Varied occupations suited to the individual's temperament and habits should be sought for, wherein resourcefulness is a great asset.

Speech training for aphasia may accomplish much. Its details, as well as many others, cannot be entered into here.¹

¹ Consult White and Jelliffe, *Modern Treatment of Nervous and Mental Disease*, vol. ii, for detailed discussion of all of the features of treatment in articles by F. Tilney and S. A. K. Wilson.

CHAPTER XIV.

TUMORS OF THE BRAIN.

TUMORS of the brain are relatively infrequent. They occur at all ages, are found in every conceivable location, and are of a greatly varied pathology. Extensive monographs have been published, and no feature of nervous disease has attracted more attention, not only from its practical importance, but also from the stand-point of cerebral localization and function. The chief literature to 1915 may be found in Starr,¹ von Monakow,² Oppenheim,³ Tooth,⁴ and Redlich.⁵

In 18,000 cases of nervous disease occurring in ten years at the Vanderbilt Clinic there were 48 brain tumors. This is a minimum computation.⁶ Bruns states it as high as 2 per cent., Cushing as 0.75 per cent., Redlich, from 4000 cases of nervous disease, the same.

Etiology.—The causes for certain tumors, such as tuberculoma, syphiloma, actinomycoses, are well known. For both syphilitic and tuberculous tumors, and possibly other tumors, traumatism may be an additional element for a brain localization. Metastatic tumors follow from their primary sources. Certain teratomata, dermoids, angiomas are congenital conditions, while cholesteomata, chordoma, chondroma, lipoma, and myxoma are also developmental anomalies.

The traumatic genesis of tumors in general, or of any one type in particular, apart from aneurisms, is problematical, yet if the trauma and symptoms are related in special ways the causative role may be assumed. Thus if the injury is sufficiently intense to definitely injure the skull, the time interval between the accident and the development of the symptoms not too great, and the localization of the probable tumor near to the site of the injury, the relationship may be justifiably maintained.

Varieties.—The chief forms met with are (1) true tumors, (2) infectious tumors, (3) parasitic cysts, (4) aneurisms, (5) vascular cysts.

1. **True Tumors.**—Of these *gliomata* are the most frequent. They preponderate over any other class in adults. In Tooth's summary of 500 cases, 49 (2 per cent.) were gliomata. Children rarely come to the National Hospital in London, hence his low percentage of tuberculous tumors. Gliomata occur throughout the brain as circumscribed or as diffuse tumors, and are extra- as well as intracerebral. The

¹ Textbook of Nervous Diseases, 4th ed., Brain Surgery.

² Gehirnpathologie, 2nd ed., Die Lokalisation im Grosshirn, 1914.

³ Die Geschwülste des Gehirns, 2nd ed.

⁴ Brain, 1912.

⁵ Handbuch der Neurologie, Lewandowsky, vol. iii, 1912.

⁶ See Report of Clinic of Prof. M. Allen Starr, 1900-1909.

general tendency is toward diffuseness and principally in intracerebral growths degeneration with hemorrhage and fatty and cystic formations takes place. Thus the symptoms are apt to develop slowly and intermittently in cortex or basal ganglia, occasionally from the ependyma. They may invade an entire hemisphere.

Sarcomata are less frequent, restricting the term sarcoma more strictly than is usual, and rejecting the compromise gliosarcoma. Nearly all gliomata show elements indistinguishable from sarcoma. They usually develop slowly in the brain.

Chloromata are leukemic in origin and are rare. They involve the periosteum or the base and thus cause compression phenomena which often persist for some time.

Fibromata (neurofibromata) are comparatively common brain tumors (10 per cent. in Tooth's collection, including fibroglioma).

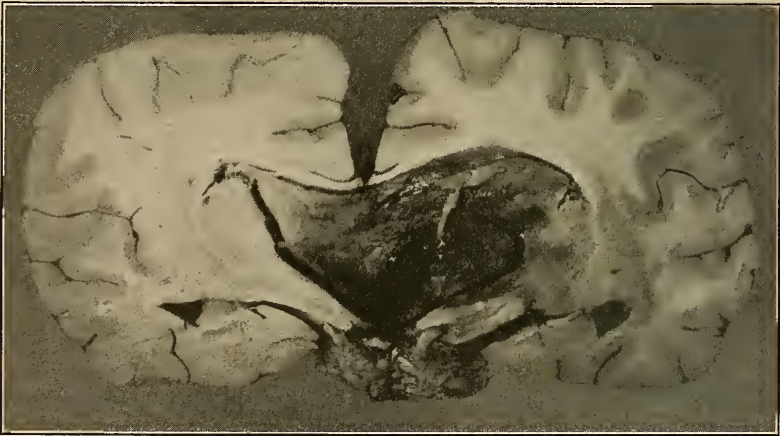


FIG. 240.—Infiltrating glioma of basal ganglia.

They develop chiefly about the cerebellopontine angle (acoustic) but may develop along other cranial nerves. Occasionally they are multiple. They develop slowly.

Endotheliomata are comparatively frequent (14 per cent. in Tooth's collection). They seem to confine themselves chiefly to the anterior fossæ. They are usually small and multiple, develop slowly and chiefly in the falx region.

Chordomata are infrequent, and only rarely reach a considerable size.¹

Carcinomata (5.8 per cent.—Tooth), closely related to the endotheliomata are usually secondary (metastatic), rarely primary.

Psammomata, chordomata, lipomata, enchondromata, angiomata, osteomata, adenomata, cholesteomata, teratomata (pineal),² and dermoids are among the rarities.

¹ Jelliffe and Larkin, *Journal of Nervous and Mental Disease*, January, 1912.

² Bailey and Jelliffe, *Tumors of the Pineal Body*, *Arch f. Int. Med.*, December, 1912.

2. **Infectious Tumors.**—These are tuberculous, syphilitic, actinomycotic.

Tuberculomata are possibly the commonest of all tumors, certainly in children (Starr, 193 in 600 tumors recorded). They are extremely rare after forty years. They are frequently conglomerate or multiple in type, hence giving rise to mixed syndromes. There may be a few very small miliary tubercles or a large broken-down tubercle mass, with every conceivable intermediary stage. They develop chiefly in the cerebellum, peduncle, basal ganglia, pons, and cortex. In Zappert's group of 89, 37 were in the cerebellum, 29 in the cerebral cortex, 13 in the basal ganglia, 5 in the pons. They have a bad prognosis.



FIG. 241.—Chordoma of base. (Jelliffe and Larkin.)

Syphilomata.—Gummata are not infrequent. They are practically limited to adults, and are not recorded from congenital syphilis. They may appear from one year to thirty years after infection. They occur chiefly as flat, infiltrating, irregular masses—less often as definite nodular masses, chiefly at the base of the brain. They are discussed more fully in the chapter on Syphilis of the Nervous System (*q. v.*).

Actinomycosis of the brain is a rarity.

3. **Parasitic Cystic Tumors.**—*Cysticercus of the brain* is a rarity and is secondary.

4. **Aneurismal Tumors.**—Aneurisms are very frequent in cerebral vessels. They are mostly small, but large aneurisms occur at times and give symptoms of pressure. They occur in patients usually from

forty to seventy years old, and are mostly of the basilar. They cause pressure symptoms at times, with obstructive symptoms—basilar syndrome—or they rupture and produce symptoms of cerebral hemorrhage.¹

Symptoms.—Brain tumors, even of a large size, may be found at autopsy, and yet not have given rise to any recognized symptoms. Tuberculomata are thus frequently found in children. With more precise investigation such latent tumors are becoming rarer, especially since the importance of mental symptoms—psychoses, so-called hysterias, etc.—unaccompanied by sensorimotor syndromes, is becoming recognized. Many small tumors, especially osteomata, psammoma, slowly developing and circumscribed gliomata, cholesteomata cause very few symptoms. Occasionally a tumor will show monosymptomatically, as by epileptic convulsions, mild speech disturbances, mild sensory defects, optic, olfactory, auditory hallucinations or hemianopsia, without being recognized.



FIG. 242.—Gumma of brain.

The symptoms are best considered as (1) general and (2) local or focal.

The *general symptoms* are indicative of the effects of the tumor as a whole, irrespective of its special nature or localization. They are due in general to the effects of increased intracranial pressure, which in certain tumors, notably of the posterior fossa and cerebellum, appears early and is usually marked even with small tumors, whereas tumors elsewhere often may show little of such pressure symptoms. Sometimes the focal symptoms appear before the general ones. General symptoms rarely have any localizing diagnostic value as many of them may lie remote from the site of the tumors. Some of the general symp-

¹ Beadles, *Brain*, 1907, p. 285; Reinhardt, *Ueber Hirnarterienaneurysmen und ihre Folgen*, Mitt. a.d. Grenzg. d. Med. u. Ch., 1913, xxvi.

toms of pressure, cranial nerve palsies for example, may even tend to mislead one as to a localizing diagnosis. Furthermore, in cerebral tumors, acute swellings, not identical with but related to edemas, often give rise to very anomalous symptoms and tend to obscure the diagnosis and render it uncertain.

The chief symptoms of general value are headache, nausea, vomiting, dizziness, respiratory and cardiac disturbances, metabolic changes, mental signs with sleeplessness—sometimes drowsiness and optic nerve changes, and convulsive phenomena. These general symptoms have a tendency to be progressive, but may vary considerably in their intensity from time to time, especially in syphilomata, tuberculomata,



FIG. 243.—Aneurism of basilar artery. (Larkin.)

and gliomata. At times they remain stationary, again they may regress and disappear.

Headache.—This is frequent. Most patients will have headache, especially if the tumor is of protracted growth. Headache is an early sign. Practically every patient with a headache should be questioned and examined for the possibility of its being caused by a cerebral tumor. The headache usually starts more or less irregularly, is frequently intermittent in the early stages and then becomes persistent, being dull or severe; if dull, with periods of excruciating exacerbation. Diurnal variations may be noted, and emotional excitement or general causes for increasing intracranial pressure increase it, often with addi-

tional symptoms, such as vertigo, vomiting or even agitated or comatose confusions. Not infrequently aneurismal tumors will reveal pulsation by auscultation. Migraine-like headaches are very frequent in early stages, particularly in basal cases, hypophysis, basal gummata, neurofibromata, or there may be migraine-like exacerbations on a dull, heavy, gray background of pain. Children usually respond to such variations by attacks of screaming, pulling the hair, or beating the head.

Later, somewhat mentally dulled patients may even deny any headache, or even forget having had a period of great distress.

The headache of cerebral tumor is mostly diffuse, but it may be localized, in which case it may serve to indicate the general site of the tumor. Such is rendered more probable if further substantiated by percussion tenderness, a highly important procedure, percussion dullness, and *x*-ray shadow. The site of a headache is a very uncertain guide for localization purposes, however. Frontal tumors often give rise to occipital headaches and *vice versa*; right-sided tumors to left-sided pains and *vice versa*. The *general* drift for localized pain is, however, in favor of a similarly located tumor. Among head symptoms may be mentioned the occasionally found auscultation notes of aneurismal tumors; the presence of enlarged head (hydrocephalus), and the overfilling and increased tortuosity of the veins on the forehead, face, conjunctiva, etc., of the affected side.

Trigeminal neuralgic attacks may be general or at times a focal (cerebellopontine angle) sign.

Nausea, vomiting, and dizziness are frequent in late stages of a cerebral tumor, and more often found in children and in those patients with rapidly increasing signs of intracranial pressure—posterior fossa tumors particularly. Such vomiting may occur spontaneously—projectile in type—or as an accompaniment of the headache crises, especially when migrainous in type. Vomiting often is absent entirely even with large tumors. When present it more often occurs in the morning, and at times it is so persistent as to lead to inanition, exhaustion, and death.

In cerebellar, peduncle, pontine, and medulla tumors, and those causing pressure on the superior cerebellar peduncle, or its incoming pathways, the nausea and vomiting may be accompanied by unilateral vertigoes or with tendencies to turn or to fall in one direction. Here general and focal symptoms coincide. Frontal tumors may occasionally cause such one-sided vertigoes from implication of the frontal extensions of the cerebellar pathways. Vertigoes from ocular palsies are localizing symptoms (quadrigeminal syndromes—occasionally cortical).

Cardiac and Respiratory Signs.—Slowness of the pulse, at times marked—30 to 40—is a general sign of intracranial pressure, and more especially in marked grades. Hence it is apt to be a late rather than an early symptom, unless one of direct irritation of the vagus (medulla pressure). The bradycardia, at times arrhythmia, may appear period-

ically during headache exacerbations, or at times independent of the same (acute swelling reaction).

Medulla tumors cause respiratory changes, at times slowness, again irregularity, and Cheyne-Stokes—with acute pressure symptoms (hydrocephalus internus). Hiccough, yawning, and related respiratory signs are occasionally present.

Metabolic Disturbance.—These are irregular in their development and evolution. Fever is infrequent save as a complication of the late stages.

Cachexia and marasmus are present with certain carcinomata, and marked adiposity. Ovarian, and in particular testicular aplasia, are frequent in certain hypophyseal (pituitary) tumors or those causing internal hydrocephalus by possible implication of the infundibular region through general pressure (pineal and corpora quadrigemina tumors). Acromegaly is a special case, as are also the dystrophia adiposogenitalis syndromes which are discussed in the chapter on the disorders of the endocrinous glands.

Mental Signs.—These are of great value, both general and localizing, in from 60 to 85 per cent. of the cases.¹ They vary considerably, and are particularly prominent late in the disease, although here masked under the general symptoms of apathy, confusion or coma. Tumors of any region, large and small, and independently of their pathological nature, may cause psychical changes. Certain localities cause special psychical alterations to be discussed under focal and localizing symptoms.

In the early stages, slight impairment of attention, with slowness and difficulty in grasp, retardation in motor response, and a confusion or bewilderment may be present. Ready forgetting, slight esthetic lapses, and moral breaks—with the telling of shady stories, showing of bad taste, exhibitionistic fancies, even gross lapses—such as open masturbation, etc.—occur. These are the precursors of a more marked grade of retardation of mental function, leading to apathy, listlessness, lack of initiative, at times with confusional episodes—getting lost—fugues, fussiness, emotionalism, etc. In marked states of confusion and disorientation a typical Korsakow's syndrome (*q. v.*) without polyneuritis, will show itself.

Later stages show typical pictures, not distinguishable from those of arteriosclerotic dementia, or paresis—*i. e.*, so far as the purely mental picture is concerned.

Certain less general symptoms often present themselves and are of a certain localizing value. Hallucinations of smell speak for olfactory lobe or olfactory tract involvement. Those of sight for occipital cortex localization, those of sound for temporal lobe trouble.

The tendency to joke, be facetious, show manic-idea associations, even flight of ideas with euphoria, is at times present. This is more

¹ Gianelli, Schuster, and others. See Bibliography in Redlich, Hirntumor, Lewandowsky's Handbuch der Neurologie.

often found in prefrontal tumors, especially left-sided, but may occur in tumors of other regions, usually, however, all reaching to and involving the cortex. These symptoms are possibly thalamic over-responses from thalamocortical interference at cortical levels.

Certain patients show definite depressed states—with hypochondriasis or even melancholic suicidal ideas. Others show manic pictures with wild flight or marked maniacal delirium. Certain patients develop delirium during certain of their headache paroxysms. Paranoid trends also manifest themselves in a few instances. So-called hysterical symptoms are frequently encountered. Careful analysis shows, however, no psychical conversions. Emotionalism and delirium are incorrectly termed hysterical because of a loose application of the term hysteria.

Optic Nerve Changes.—These are of the greatest importance in diagnosis of brain tumor. The general features are discussed in the chapter on Cranial Nerves. From 60 to 80 per cent. of all patients show optic nerve changes, which vary largely, depending upon the grade of intracranial pressure and the size and location of the tumor. Those tumors causing great intracranial pressure (posterior fossa particularly) naturally cause choked disk and optic neuritis earlier and in more marked degree. Optic nerve changes may be absent even with large tumors, and small tumors of the pons, medulla, motor area, basal ganglia, corpus callosum, and hypophysis may give rise to no optic nerve changes. The optic nerve changes develop gradually. They are unilateral, later bilateral or develop bilaterally synchronously. As a rule tumors of one side show beginning nerve changes on the same side earlier and more markedly than on the opposite side. The reverse can also be true and the localizing value must not be overestimated. The general trend, however, is as stated (Gunn).

The visual power may not be lost even with marked grade of swelling or of atrophy, but there is later a gradual loss of vision—often seen in early signs by the irregularity of the color field loss (interlacing phenomena, scotomata, etc.). The hemianopic changes which are not infrequent in chiasm, posterior tract, pulvinar, and parts of the pathways are discussed in Chapter V, p. 183.

Motor Phenomena.—These are local or general. Epileptiform attacks are frequent—particularly in children. When limited Jacksonian attacks are present, the localizing value in the motor cortex is evident, save for the few exceptions of pontine, cerebellar, and peduncular Jacksonian attacks. Petit mal attacks are also not infrequent. Some patients die in the convulsive seizures.

Focal or Local Symptoms.—These may be among the first of the symptoms encountered, especially in circumscribed cortical tumors, but, as a rule, the focal symptoms develop after the general ones; again they advance together. They are best considered with reference to the areas involved, such as the symptoms of the frontal localizations, central convolutions, parietal lobes, temporal, etc.

Frontal lobe tumors are those located ahead of the precentral sulci. Those of the frontal poles, foot of third frontals, are frequently termed prefrontal tumors. The functions of the frontal areas are chiefly those of coördination of psychical, chiefly intellectual processes.¹ They contain motor areas for the innervation of the muscles of the neck, throat and abdominal muscles and the third frontal convolution—Brocas' convolution—is the motor aphasia area, Marie and his pupils notwithstanding. On the base the olfactory and optic pathways may be involved by direct or indirect injury, and pressure posteriorly causes pyramidal tract symptoms. Cerebellar pathway projections also lie in the frontal poles.

Thus the symptoms of tumors lying within the frontal lobes may show considerable variation according to their size and site. Many



FIG. 244.—Frontal lobe tumor. (Larkin.)

small tumors located deep in the cortex are apparently symptomless from the neurological point of view. Paranoid states, occasionally seen from such tumors, are readily overlooked, also mild depressed states which are called neurasthenia. One symptom of special importance is a tendency to make jokes (Witzelzucht), or a tendency to talk or answer beside the point—at times an apparently intentional effort to mislead. One does not necessarily locate a tumor in the frontal lobes by reason of this tendency to joking alone. At times the behavior is infantile and childish, and diagnosed hysteria. Again patients are irritable, excitable, churlish, even have furious outbreaks of wrath and are violent, capricious, or the picture of gradually advancing stupidity, with inability to grasp, loss of initiative, slowness of power of applica-

¹ Franz, Functions of the Frontal Lobes, Archives of Psychology, 1907.

tion, is seen. In right-handed tumors, the psychical disturbances are more frequent—Schüster¹ (80 per cent.), Pfeiffer,² Müller.³

Orientation for the external world, time and space, is apt to be involved more than personal orientation. Complete disorientation, as in Korsakow's syndrome, is met with.

At times hallucinations of smell appear from pressure on the olfactory pathways, or hallucinations of sight, photomata, from similar pressure on optical pathways at the base.

Vertigo, with a drunken gait, may be ascribed probably to the higher association of space perceptions and indicates frontal involvement of cerebellar components, the gait being closely related to that of cerebellar syndromes—the patient staggers to the tumor side: adiadokokinesia and asynergia are usually absent here, however. (See Cerebellar Syndromes.)

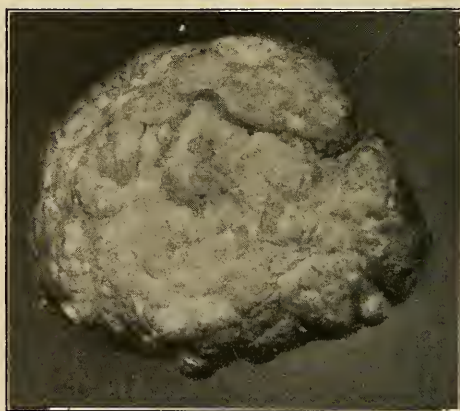


FIG. 245.—Frontal lobe tumor removed. This patient had loss of smell on the tumor side, Witzelsucht, staggering gait, and loss of control of feces at times, with otherwise unimpaired intelligence.

Involuntary defecation or urination occurs at times; most frequently with somnolent patients. The patient whose tumor is here figured had a marked cerebellar gait, was keen and active, jocular and happy, with occasional loss of bladder and rectal function and hallucinations of smell. The tumor occupied the left frontal pole (Fig. 245).

Apractic disturbances are occasionally met with in frontal tumors, and those involving or pressing upon Brocas' convolution, left side, cause mild (paraphasic), or severe motor aphasia in right-handed individuals, usually of gradual onset and often remittent in character.

Other motor signs are stiffness in the neck with forward and backward fixations of the hand and tendencies to tremor of the hand on

¹ Thesis, Stuttgart, 1902.

² Arch. f. Psych., xlvii.

³ Deut. Zeitschrift f. Nervenheilkunde, vols. xxi, xxii, xxiii.

the tumor side. In a third of the cases, epileptiform attacks, often Jacksonian, occur, from pressure on the motor area.

Central Convulsions.—The functions chiefly involved are those of the voluntary muscular activity, hence paresis, paralysis, spasms. Tumors of this region are never latent. Irritative phenomena, spasms, convulsions, speak for cortical locations; paralysis for deeper-seated lesions involving the pyramidal paths from the motor areas. Small tumors, cortically located, cause isolated Jacksonian attacks; the more extended the tumor the more widespread the muscular involvement; even small tumors, however, may cause widespread Jacksonian or grand mal symptoms. Often the first observed motion accompanied by tingling affords a clue as to the more definite localization of the



FIG. 246.—Depression in brain after removal of a frontal tumor. (Goodhart.)

tumor; again an orderly and uniform progression in the development of a Jacksonian attack is valuable in localization.

Monoplegias¹ and monopareses are not infrequent from small lesions, and in the beginning of the tumor growth. The slow extension of the paresis or paralysis is of diagnostic moment. Advancing hemiplegia is often accompanied by the vasomotor phenomena seen in hemorrhage—especially in deep-seated lesions. The usual signs of an organic paralysis (*q. v.*) are present.

Psychical symptoms of general nature are not infrequent. Occasionally large tumors will cause a Korsakow syndrome. Katatonic

¹ Bergmark, Monoplegia, Brain, 1910.

symptoms may also appear. Sensory phenomena are frequent in postcentral convolution tumors. The phenomena have been extensively described on p. 479, when speaking of sensory changes due to cortical and subcortical lesions.

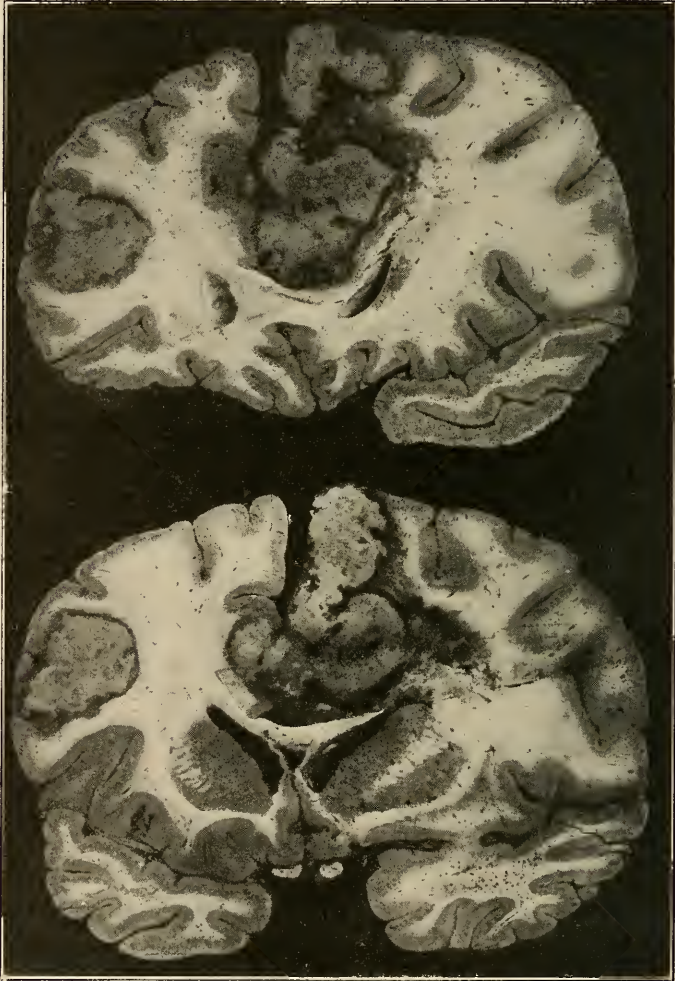


FIG. 247.—Multiple sarcoma of brain. (Larkin.)

Central convolution tumors are among those more readily localizable, and usually more accessible to operative relief.

Parietal Lobes.—When the tumors press forward toward the posterior central lobes sensory signs are produced like those mentioned on p. 479. Left-sided tumors, in right-handed persons, especially of the inferior parietal lobes, cause cortical sensory aphasia of Wernicke

of various grades. Alexia and agraphia may also be found in left-sided lesions.

Epileptic attacks with hallucinatory auras of taste occur; also a general Korsakow syndrome may develop. Other pathways from the sensory areas may be cut off by tumors in this region; hence either hemianopsias, optic agnosias or optical aphasias (gyrus angularis). Apractic disturbances are of value in localizing left-sided tumors. Katatonic syndromes, confusion, apathy, or general loss of orientation may be encountered but are equally present in right-sided and left-sided cases.

Occasionally parietal tumors give rise to ptosis, paresis of the lateral movements of the head and the conjugate motion of the eyes to the opposite side. (Cephalorotary and oculorotary paralysis, see Plate VIII.)

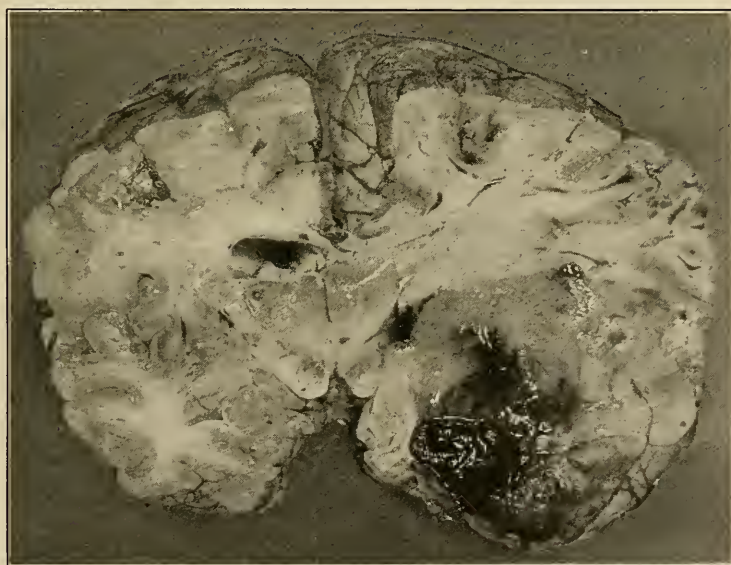


FIG. 248.—Glioma of temporal lobes.

Deep-seated lesions may impinge upon or involve the motor pathways.

Temporal Lobes.—The cortical end stations of the auditory pathways which are both crossed and uncrossed are contained in the first and second temporal lobes, particularly in Heschl's convolutions. Cortical deafness is practically impossible in unilateral lesions but has resulted from bilateral involvement.

Word-deafness is the most striking result in left-sided lesions. This is a progressive affair, often beginning with difficulty in finding words, paraphasia, and resulting in more severe forms in alexia, agraphia,

logorrhea, and total word-deafness. Large tumors also cause indirect symptoms and may lead, by pressure on motor areas—Broca—to total aphasia, and epileptiform convulsions.

Auditory hallucinations are not infrequent, showing as auræ in generalized grand mal attacks. Gustatory and olfactory phenomena of similar nature result from hippocampal or closely related lesions—uncinate fits.

Tumors of the under surface may cause hemianopsia, through pressure on the optic tracts, and by pressure on the pyramidal or fillet tracts cause hemiparesis or hemianesthesia.

In certain cases, as Knapp¹ has shown, there may be an orderly progression of symptoms due to direct or indirect action in temporal lobe tumors. The most striking of these is a homo- or contralateral

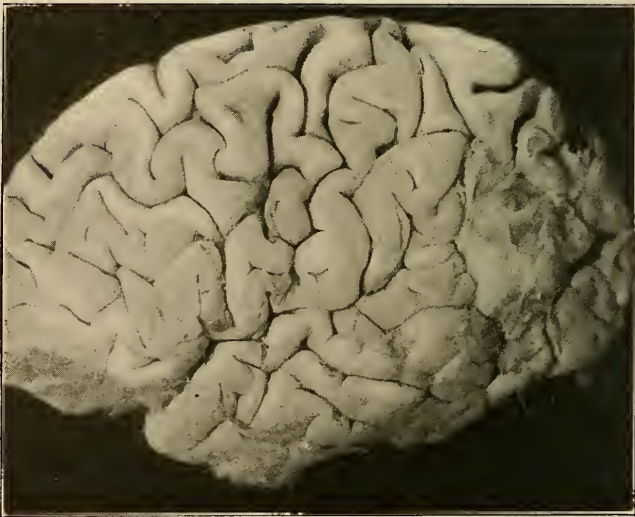


FIG. 249.—Tumor of temporo-occipital lobes. (Larkin.)

temporary or persistent oculomotor palsy, chiefly ptosis and mydriasis, crossed hemiplegia, and cerebellar ataxia.

Occipital Lobe.—The end-projections of the optic tracts are located here particularly in and about the calcarine fissure. Complete homonymous hemianopsia is the chief symptom of tumors of this area. This hemianopsia usually spares the papillomacular bundles and is often unperceived by the patient. Quadrant hemianopsia is also found. Tumors may exist and hemianopsia be absent. It is most often present with tumors of the median aspect of the occipital, also with those lying on the convex surface, and hence the more readily removable. Various stages of blindness may also result, and there

¹ Die Geschwülste d. rechten u. linken Schläfenlappens, 1905; Münch. med. Woch., 1908.

may exist a mind-blindness from left-sided tumors, also alexia, agraphia and sensory aphasia.

Other optic signs, such as phonemes, various scintillating scotomata, optical hallucinations and illusions occasionally result from occipital tumors, and these may exist as auras preceding general epileptic convulsions.

Tumors on the inferior surface, by compression of the cerebellum, will cause cerebellar signs, and occasionally palsies result from tumors lying on the external surface of the occipital lobe.

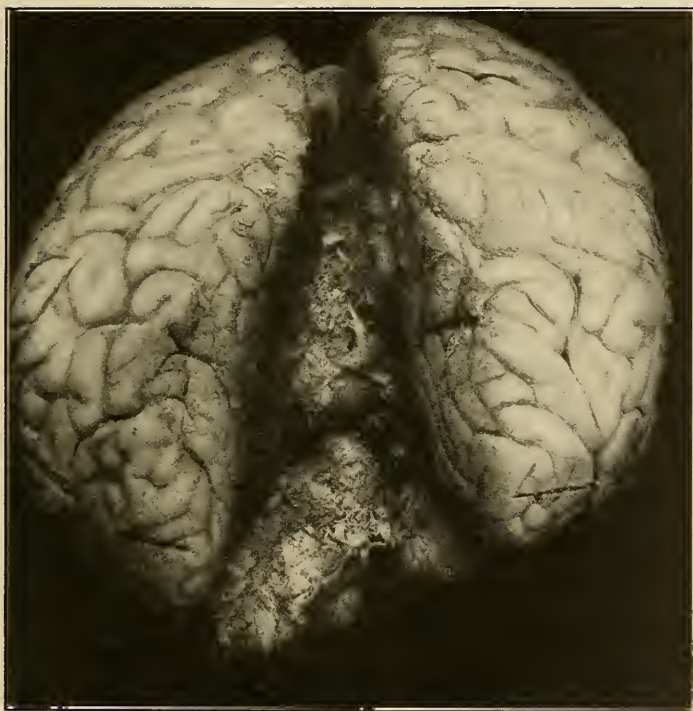


Fig. 250.—Tumor arising from the meninges and pressing down to the corpus callosum. (Baldwin.)

Corpus Callosum.—Isolated callosal lesions are extremely rare. Schüster gathers 37; Williams 38.¹ Congenital absence is known with no definite symptoms. Apraxia (dyspraxia) of the left hand is an occasional symptom due to interruption of forward-lying callosal fibers, especially those between the sensorimotor areas, from left to right, and is of special significance in diagnosis. Possibly mind-blindness results from lesions of those callosal fibers uniting the two optic

¹ Journal of Nervous and Mental Disease, 1901.

fields (Brodmann 18, 19), also termed the visual psychic area. This is not yet established definitely.¹

Paretic syndromes of the extremities occur with callosal tumors. Dupré locates them in the anterior callosal region if the paretic symptoms are in the muscles of the face and tongue; pareses and ataxias of the upper extremities from midcallosal fibers; paresis, ataxia and hemianopsia from posterior callosal fibers. Van Valkenberg's extensive studies of the callosum do not at all corroborate Dupré's deductions.

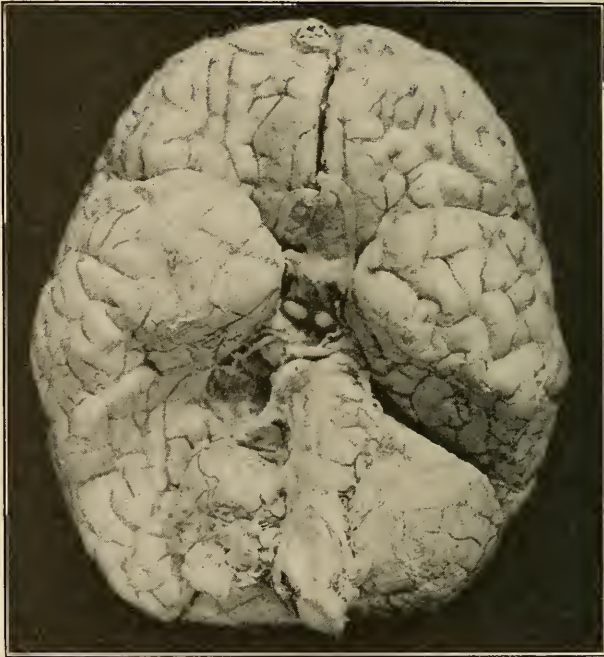


FIG. 251.—Tumor with atrophy of the cerebellum.

Tumors of the callosum are more apt to show compound symptoms due to involvement of the neighboring parts; if lying forward, frontal signs are added; midcallosal region, pyramidal tract, epilepsies, etc. General psychical symptoms are usually present, and appear early. They approach the type due to general pressure. Korsakow and paretic syndromes develop. There is nothing diagnostic about the mental signs, save as might appear from the neighboring pressed upon areas.

Tumors of the Optic Thalamus.—The symptoms are discussed under the section on the thalamic syndrome (*q. v.*). Involvement of the

¹ Van Valkenberg, *Brain*, November, 1913.

lenticular nucleus (see Wilson's disease), tumors of the midbrain, pons and medulla have been discussed. Those of the cerebellum and cerebellopontine angle will be found under Cerebellar Syndromes.

Diagnosis.—Multiple sclerosis, paresis, arteriosclerotic disease, all forms of headache, tuberculous meningitis, chronic hydrocephalus, and hysteria are the chief conditions causing difficulty.

Multiple sclerosis, if the patches are solely cerebral, may cause confusion, especially in the acute cases, as described by Marburg and others. The bitemporal pallor of the disks in this disorder differs from the usual pressure changes in the disk. Nystagmus is not a frequent brain-tumor sign. Other signs of pyramidal tract implication may be identical. Headaches are usually absent, also nausea and vomiting.

Pseudoparetic and arteriosclerotic psychical syndromes (Korsakow's psychosis) are frequently confused with cerebral tumor, *i. e.*, the cause of the mental picture is overlooked. Thus a tumor, which might have been removed, has been missed under the psychotic disguise. Eye-ground changes are usually positive in these cases, yet may be absent.

Headache should always be scrutinized carefully. A postinfluenzal occipital headache which is very frequent and extremely severe and persistent, is frequently highly suggestive of brain tumor. The headaches from lead poisoning, anemia, and nephritis are also to be excluded.

Brain abscess and tuberculous meningitis must be excluded on the ground of their difference in development of symptoms. Symptomatically speaking they may be considered as tumors. Hence, see Symptoms, p. 462. This is also true of chronic hydrocephalus.

Cerebral puncture, withdrawing a small plug of brain tissue through a trephine opening and canula is often of great aid in diagnosing very puzzling cases.

Prognosis.—No definite prognosis can be laid down. Everything depends on the site of the tumor. In general, apart from surgical relief and from medicinal treatment of syphilomata, the outlook is pessimistic. Sudden death is not infrequent and lumbar puncture is an extremely dangerous procedure with brain tumor, often leading to sudden collapse and death, especially with tumors of the posterior fossa.

Syphilomata and gummata of the brain have a fair prognosis. Better results are obtained with mercury by inunction and by iodides than by salvarsan in the beginning treatment. Salvarsan may be used later to attempt to kill off all the spirochetes, but with well-advanced syphilomata, salvarsan is apt to set up a dangerous reaction. Round, hard gummata do not absorb, as a rule, and are best considered surgically.

Treatment.—Medicinal treatment, excepting for syphilomata, is useless, and involves a waste of valuable time. The chief objects to be attained are *early diagnosis*, immediate exclusion of syphilis by sero-

logical tests, eye-ground examination with particular study of the color fields, *exact localization* as soon as possible, and *surgical removal* or *palliation* (decompression) to save the eyesight or to gain time for a more exact localization.

The details for applying these principles have already been noted. The results to be expected in any particular case are problematical, yet from 10 to 20 per cent. of all brain tumors (seen in the large) have been removable, with at least in 10 per cent. practical recovery. Even with such chances against him the patient should have the benefit of the doubt if a competent surgeon is available. Surgical skill is a very large factor in the results; a good abdominal surgeon is not necessarily a good brain surgeon. The brain is semifluid and an intricate switch-board of highly important structures; there are *no unimportant areas* in the brain; many surgeons have treated it in the past as though it were an abdominal viscus. The results have been disappointing.

Most brain operations are best done in two stages. Decompression, usually subtemporal, alone is often the only possible procedure. It often relieves a recently acquired blindness.

The situation referable to brain surgery for cerebral tumors is rapidly advancing, and better and better results are being obtained and regions hitherto impossible to reach (hypophysis, etc.) have been approached with results which a decade ago would have been impossible.¹ Notwithstanding all this, the general attitude should be one of extreme caution.

¹ See Starr, Brain Surgery; Halsted, Treatment of Brain Tumors; White and Jelliffe, Modern Treatment of Nervous and Mental Disease.

CHAPTER XV.

SYPHILIS OF THE NERVOUS SYSTEM.

SYPHILIS of the nervous system is so protean in its clinical forms, that the significance of syphilis as an etiological factor in nervous diseases frequently is entirely overlooked. The physician with his eye riveted upon a clinical picture, be it an amyotrophic lateral sclerosis, a failing memory, a persistent nervous weakness, a progressive muscular atrophy, or a protracted sciatica, and mindful of the somber prognosis given in the majority of his available guides, may readily overlook the fact that syphilis is the cause for these syndromes.

There is no field in medicine wherein similar diseased pictures may arise from as many differing causes as in the domain of the nervous system. Nor, on the other hand, where a single etiological factor may give rise to so many dissimilar clinical pictures. Hence, the complexity of the whole subject, and the need for iterating and reiterating the advice that in nearly all of the clinical pictures which have or have not been given descriptive terms in nervous or mental disease, the possibility of syphilis as a direct etiological factor or as a complication, or as causing confusion, should be borne in mind. Hence, the necessity for detailed and minute inquiries into all of the possible symptomatology, of hereditary or acquired syphilis, which in all questionable cases should be supplemented by complete serological and cytological examinations.

The formerly very distinct dividing lines between the lesions of hereditary syphilis, acquired syphilis, and metasyphilis are slowly being obliterated, and in time it may probably seem strange that it could have been thought that spinal and cerebral syphilis on the one hand should ever have been considered different, let us say, from tabes and general paresis on the other.

Since it has come to be believed that both tabes and general paresis rest upon a syphilitic basis, the variety of syphilitic disorders showing fairly clear clinical entities has been enlarged. Further, with the recognition of many acute and subacute psychoses due to syphilis the psychiatric borders have been further extended.

In the present chapter, then, the discussion will concern itself chiefly with clinical forms, without any extended attempt being made to differentiate types, which in reality are so kaleidoscopic that they defy description.

At the same time it needs to be emphasized that such intermediary forms are ever-present realities. Classical pictures of a disease are

largely literary efforts. They are the product mainly of the descriptive art. The actual processes going on in nature in the conflict of man against the spirochete, does not show classical types but a multiplicity of variations, with here and there statistical prominence of this or that trend in the reactive compromise.

History.—Leoncino as early as 1497 described paralysis as a consequence of the disease. He here referred to what is known as hemiplegia, which may result, as is well known, within a few years, even a few months, after infection.

Joseph Grünbeck (1503), Emser (1511), Ulrich von Hutten (1519), all lay writers, mention paralyzes of the limbs as due to the disease. Emser speaks of his patient, a syphilitic paralytic, and with a psychosis, as having made a remarkable recovery, under treatment by Bruno, by making a vow. Paracelsus (1530), although still confusing gonorrhoea with syphilis, as had been and is still being done, left indications of a description of syphilitic meningitis, and in speaking of the syphilitic virus said that it affected all of the organs of the body. Nowhere in Fracastorius (1521), who gave the name syphilis, are direct references to the nervous system to be found. Nicolaus Massa (1556) gave an early description of syphilitic neuralgias. Borgarutius (1567) also described neuralgic pains due to syphilitic disease of the meninges. Amatus Lusitanus (1561) described headaches due to intracranial osteitis of syphilitic origin. Botalli (1563) made an observation that blindness might be due to syphilitic disease of the brain.

During the following century many references have been found showing the recognition of the relationship of syphilis to nervous disease. Only a few can be mentioned here. Thus, Guarinoni (1610) described epileptic attacks from syphilis of the brain. Vidus Vidius (1611) described epilepsies as due to syphilitic cranial caries. Thiery, de Hery (1634) and Zechius (1650) also called attention to syphilitic spasms as well as epilepsies. Zacutus Lusitanus (1644) described cases of blindness due to gumma of the brain, quoting Botalli a century ahead of him. In 1696 a special treatise on syphilitic pains was written by Blagny.

Attention might be called to the works of Scholtzius (1610) and Willis (1672) *a propos* of the subject of general paresis. To Willis has always been ascribed the honor of the first description in which one could definitely recognize general paresis.

By the end of this (seventeenth) century a fairly broad view of syphilitic nervous disease had been obtained. Syphilitic headaches were described by Felix Plater (1641). Rhodius (1657) described gummata of the dura and syphilitic hemiplegias; Ballen (1663), spinal syphilitic disease and spasms in the facial region; Cummius (1684), diplopias and eye palsies. Astruc has reviewed these writings completely.

During the eighteenth century the picture expanded rapidly. Intercostal neuralgias (1762), deafness, loss of smell, caries of base of

skull (1762), sciatica (1745), psychoses. Syphilitic mania was described by Sanché in 1777, amaurosis (1748), facial palsy (1758), leptomeningitis (1766), syphilitic arterial disease (1766), paraplegias (myelitis) (1771), and a number of other conditions were described, and may be consulted in Lagneau's interesting monograph in which 234 case histories are collected. Astruc, Bonet, and Morgagni offer the richest literary sources.

It may be recalled in this place that John Hunter in 1787 stated that he never observed syphilis in the internal organs, including the brain. The weight of his authority retarded progress for many years, especially in England. Indeed, it was not until Ricord's sound observations were published that Hunter's enormous blunder was fully remedied.

Virchow's studies (1847) on phlebitis and arteritis had laid the foundation for our modern knowledge of bloodvessel syphilis, although it may be recalled that Morgagni (1766) and Horne (1782) both made extremely important studies on vascular syphilis. These have been fully developed by Huebner (1874) and Alzheimer (1904).

The studies of Virchow on the formation of gummatous granulomata and related syphilitic phenomena, practically established the modern era of study of the pathology of this disease.

The succeeding years have filled in the picture with a number of details, the chief additions having been those of Nissl and Alzheimer, who have established the highest criteria for the pathology of this disease so far as the nervous system is concerned.

The latest chapter in this interesting history is that dealing with the discovery of the exciting agent, and the final clearing up of the entire subject of etiology and modes of infection. Schaudinn (1905) demonstrated the parasite which he called *Spirochaeta pallida*. Its varied synonymy to accord with principles of botanical and zoological nomenclature need not detain us. *Spironema pallida*, *Treponema pallidum* are those most frequently employed. Doele (1892) is thought to have first seen the parasite, but Schaudinn, then Epaschen, Fischer, Metchnikoff, and Roux established its identity and its affiliations.

It is found in congenital syphilis of the nervous system, in syphilitic gumma of the brain and spinal cord, in syphilitic meningitis, even in the cerebrospinal fluid, both of congenital and acquired syphilitics. Moore (1913), Noguchi, Nichols, and Hough found it in the paretic brain, and it has been reported to have been found in the spinal meninges of tabetics.

The final studies of Neisser, Metchnikoff, Roux, and others have laid bare the entire story of the inoculability of the disease and its transmission from animal to animal, while utilizing the knowledge gained by Bordet and Gengou, Wassermann and his pupils have elaborated a serobiological technique which has made one independent of clinical or anamnestic data relative to a knowledge on the part of the patient of the infection.

Thus, in hardly more than seven years, a flood of light has been thrown upon the disease and its relation to other protozoal-caused diseases, notably trypanosomiasis, which has illuminated and made clear the entire path which has been so busily travelled since Columbus brought back this most portentous exotic to the old world.

Diagnosis.—The diagnosis of syphilis of the nervous system presents certain difficulties which rapidly increasing perfections in laboratory technique are resolving with considerable success. These laboratory findings, combined with those of the neurological examination, permit an almost certain diagnosis of this disease in the nervous system, either as congenital, acquired, or as para- or metasypylis.

The chief features in such diagnosis are: (1) search for the organisms; (2) serological investigation of the blood and cerebrospinal fluid; (3) cytological examination of the cerebrospinal fluid; (4) chemical examination of the cerebrospinal fluid; (5) clinical examination of the pupillary reflexes.

1. *Search for Organisms.*—The parasite has been found in the cerebrospinal fluid, but as yet in but few instances. It has been cultivated from the cerebrospinal fluid (Nichols and Hough). Increasing experience along these paths of study will probably develop important diagnostic aids. These are, as yet, of less practical use than others to be enumerated.

2. *Biological or Serological Tests of Blood and Cerebrospinal Fluid.*—*(a) Blood.*—Practically all forms of early syphilis of the nervous system should show a positive Wassermann reaction in the blood. There are exceptions, but syphilis of the nervous system, like syphilis elsewhere, gives a positive reaction in early cases.

Whereas, the number of positive findings should be as high as 100 per cent. in cases of early syphilis of the nervous system, in its secondary and tertiary stages the number of positive results may fall to even 70 per cent., and in the latent period may sink to 50 per cent. Whether these reduced percentages speak for the good results of treatment or are dependent upon other as yet uncertain factors is yet to be decided. The alcohols interfere with the reaction. Hence the results of a Wassermann test are apt to be unreliable if the patient has taken alcohol within forty-eight hours of the time of making the test.

(b) Cerebrospinal Fluid.—The behavior of the cerebrospinal fluid to the Wassermann reaction is of special significance in the diagnosis and treatment of syphilis of the nervous system. It is almost uniformly positive in general paresis, even when small quantities (0.02 c.c.) of the serum are employed. By the use of such small quantities it would appear, from the work of Hauptmann and Hössli, that paresis alone will cause a positive result, but with larger quantities of cerebrospinal fluid (0.4 to 0.8 c.c.), practically all forms of cerebrospinal syphilis will give a positive reaction; tabes, cerebral syphilis, meningomyelitis, etc. (Hauptmann, Holtzmann, Swift and Ellis.)

Syphilis without nervous involvement usually gives a negative reaction, even when large quantities of the fluid are used.

It must constantly be borne in mind in the diagnosis of syphilis of the nervous system that the reaction of the cerebrospinal fluid in the Wassermann test is purely monosymptomatic. The positive or negative results must always be interpreted by association with other laboratory and clinical tests. As Nonne has well said, the Wassermann reaction is only a symptom. Like other symptoms in a syndrome it may or may not be present without affecting the validity of the syndrome from its diagnostic aspects.

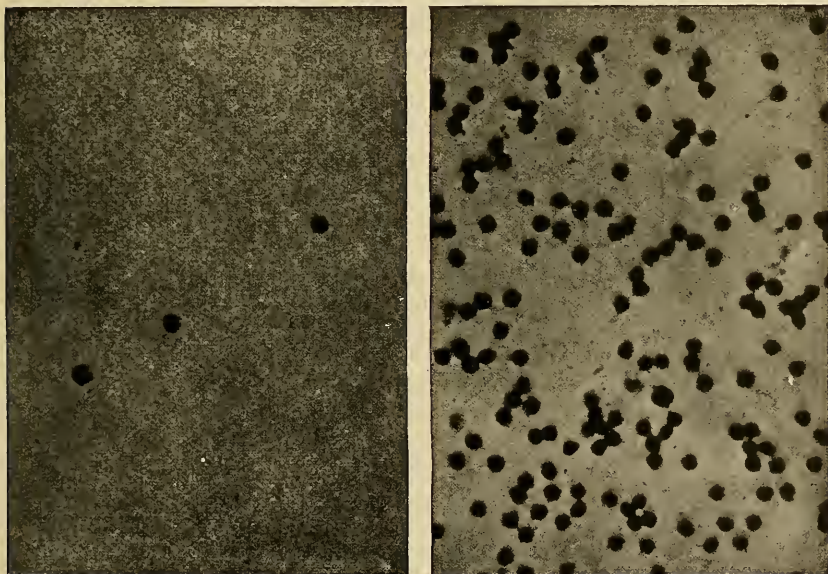


FIG. 252.—Lymphocytes in normal (left) and paretic (right) cerebrospinal fluid. (Kraepelin.)

3. *Cytological Examination of the Cerebrospinal Fluid.*—The technique of puncture cannot be entered into here, but one point should be borne in mind: the procedure is not always without danger. It should be done with care, the fluid being withdrawn very slowly, dropwise in some cases, and the patient should rest in bed several hours, preferably both before and after the operation. One of the functions of the cerebrospinal fluid is to maintain an equality in the intracerebral pressure, and any sudden alteration, such as is produced by the withdrawal of 5, 10, or 15 c.c. of the fluid, is apt to disturb such equilibrium. Headache, nausea, vomiting, dizziness, are among the unpleasant effects in those who react badly. Such are few, but they exist, and care is imperative. Some are helped by lying quiet, with foot of bed

elevated and with aspirin, 10 grains, repeated if necessary. In patients in whom brain tumor is suspected special care should be taken, as here a few cases of sudden death have been reported following lumbar puncture with withdrawal of fluid.

In pathological cerebrospinal fluid an increase in lymphocytes is the most striking feature. Over 7 to 10 lymphocytes to the c.mm. indicates pathological fluid. Other cells may also be found, such as polymorphonuclear leukocytes, plasma cells, and occasionally eosinophile and endothelial cells. Red blood cells usually come from the wound of the puncture.

The fluid is best fixed and imbedded, and then stained by the Alzheimer method. This is the most complete and satisfactory method thus far devised, as it permits of a counting and study of all the cells. The Fuchs-Rosenthal counting chamber, however, is that used in the greater number of instances. Its results are more quickly obtained, although they lack the finality of the Alzheimer method.

A normal cerebrospinal fluid is a clear liquid. It has a specific gravity of 1006, a slightly alkaline reaction, and is almost free from cellular constituents—1 to 5 lymphocytes per c.mm., as estimated by the Fuchs-Rosenthal method, may be considered normal.¹

Changes in Cerebrospinal Fluid.—In cerebrospinal syphilis, in tabes, and in general paresis there is usually an increase in the number of lymphocytes. They may run up into the hundreds, especially in some cases of acute meningomyelitis; also in some fulminating cases of paresis. In fact, most meningitic processes are accompanied by increase in cellular content. Polynuclear cells are frequent in the acute types, sometimes tuberculous meningitis excepted, and as the process tends to chronicity there is a tendency to lymphocytosis and loss of other cell types. The pleocytosis of syphilis has usually been attributed to a meningitic process. It has been suggested as due to a periarteritis as well (Szecsi).

Certain general variations may be recorded. The earlier students were more didactic in their statements concerning specific differences in the number of cells as distinguishing cerebrospinal syphilis, tabes, and paresis. Further extension of the studies shows them to have been in part unwarranted. Thus it has been said that the lower numbers point to cerebrospinal syphilis, the higher number to tabes, and the highest to paresis. This is perhaps so, but it is not an absolute rule. The number of cells seems to bear a more definite relation to the *activity* of the underlying irritation or inflammatory process than to its *kind*. Thus a stationary tabes may show few cells, also a paresis in remission, whereas an acute cerebrospinal syphilis or an acute meningomyelitis may show many cells.

A fluid rich in polymorphous cells is indicative of a very active process, syphilitic or otherwise.

¹ See Thomsen, Hill, Halliburton, Proc. Roy. Soc., vol. lxiv, for normal fluid.

In cerebrospinal syphilis, tabes, and particularly in paresis, it is of great importance to note that a pleocytosis, oftentimes of very marked grade, may antedate all neurological symptoms of the after-coming disorder. This has been shown repeatedly by Sicard, by Ravaut and others. This preparetic period has been diagnosticated as long as two years before the onset of the disease. This point is well to bear in mind when one's advice is asked as to the advisability of marriage of syphilitics in the forties. It is not at all an infrequent experience to find the outbreak of paresis occurring in men of from forty to forty-five, who have been from one to three years married, and many have waited this long, feeling that because of an early syphilis it were wise to defer marriage until a safe period. A return will be made to the prophylactic features later in the more detailed discussion of the therapy.

How long after infection by syphilis may lymphocytosis appear in the cerebrospinal fluid? Varying answers are available, but Ravaut has reported its presence at least two months after infection. The presence of lymphocytes almost invariably speaks for nervous syphilis. In patients who have thus far been followed, the development of nervous accidents, hemiplegia, paraplegia, meningomyelitis, etc., has taken place.

With active therapy the pleocytosis is apt to disappear. Are those patients who retain their lymphocytosis on the way to tabes or paresis? This is an important problem which has been answered yes and no. Its definite answer is yet to come, as the necessary time during which the full significance of the results of cytological study of the fluid has not yet elapsed.

From the therapeutic point of view then the whole subject of pleocytosis of the cerebrospinal fluid is full of significance, and in need of constant attention. In this connection it should again be emphasized that lymphocytosis alone does not mean syphilis alone. It can be said that absence of lymphocytes at least negatives tabes and paresis—to this generalization there are but few recorded negative observations.¹

Lymphocytosis is not limited to syphilis of the nervous system, however. It is marked in sleeping sickness, an allied disease (Spielmeier, Mott). It is often also high in tuberculous meningitis, but is here usually complicated by the presence of polymorphonuclear leukocytes. In a few cases of multiple sclerosis lymphocytes have been found. In the acute stages of poliomyelitis lymphocytes may be found, also in epidemic cerebrospinal meningitis and in herpes zoster.

4. *Chemical Examination.*—Here the presence of a reducing agent (Fehling's) and of globulin is to be estimated. Most fluids contain

¹ Klieneberger, *Archiv f. Psychiatrie*, 1911; Foster, Lewandowsky, *Handbuch der Neurologie*.

the former. When present in large quantities it may point to a tuberculous meningitic process.

Increased globulin content is a characteristic feature of paresis. In tabes increased globulin is the rule, as is also the case in cerebrospinal syphilis, but to a less extent. Markedly increased globulin content is not infrequent in spinal cord tumors, gummatous or non-syphilitic. The globulin reaction is apt to run along with the lymphocytosis. It has no apparent alliances with the findings of the Wassermann test.

Summary of Laboratory Findings.—Four Reactions.—Before passing to the diagnostic significance of the clinical examination of the eye reflexes a word should be said relative to the value of these “four reactions,” as Nonne has called them. It has been said that taken alone they may mean nothing positive, so far as a differential of the different types of syphilis of the nervous system is concerned, but when read together they afford important guides to diagnosis and to treatment.

Nonne's conclusions are perhaps the most extensive that we possess on this point. They are founded upon his own experience with 167 patients with tabes, 179 with paresis, 97 with cerebrospinal syphilis (arterial types), 68 patients with multiple sclerosis, 38 with brain tumor, and 14 with spinal cord tumor.

Expressed schematically, which schemes, as he well remarks, are not products of nature but of man, the following results of the four reactions are to be expected: (I) blood and (II) fluid.

I. Blood examination.

Wassermann reaction.

- (a) Positive. Is characteristic of syphilis with few exceptions (already noted). A positive Wassermann of the blood serum says nothing further than that the individual has come in some manner in contact with syphilis, either through heredity or by infection. It does not say that the disease from which he suffers is due to syphilis.
- (b) Negative. Is differentially diagnostic against paresis, since it is only very rarely that the blood in paresis gives a negative reaction.

II. Study of cerebrospinal fluid.

- (a) Normal fluid. Pressure 90 to 130 mm. water. Globulin reaction negative—not over 5 or 6 cells to c.mm. (Fuchs-Rosenthal).
- (b) Pathological fluids.
 1. Increased pressure—over 15 c.mm. water.
 2. Positive phase I. Globulin reaction.
 3. Increased cell count. (These three symptoms, in coördination or alone indicate the presence of an organic nervous disorder, syphilitic or non-syphilitic.)

- (c) If the disease of the nervous system is syphilitic, then the Wassermann test of the fluid will show. If the Wassermann reaction (original method—0.2 c.c. of the fluid) is positive, there is great probability that the patient is a paretic, or a taboparetic, much less often a cerebrospinal syphilitic, or a pure tabetic. In nearly all cases of cerebrospinal syphilis and of tabes the Wassermann reaction becomes positive by using 0.4 to 1 c.c. of fluid.

Nonne's typical findings are as follows:

I. Paresis or taboparesis.

1. Wassermann reaction in blood positive (100 per cent.).
Pressure increased.
2. Phase I, globulin reaction positive (95 to 100 per cent.).
3. Lymphocytosis (95 per cent.).
4. Wassermann in fluid.
 - (a) Positive—about 85 to 90 per cent. with original method and 0.2 c.c. fluid.
 - (b) Positive in 100 per cent. with larger quantities of fluid.

II. Tabes without paresis.

1. Wassermann reaction in blood positive (60 to 70 per cent.).
Pressure usually increased.
2. Phase I, reaction globulin and positive (90 per cent.).
3. Lymphocytosis positive (90 per cent.).
4. Wassermann in fluid.
 - (a) Original method, 0.2 c.c. positive (5 to 10 per cent.).
 - (b) Larger quantities (100 per cent.).

III. Cerebrospinal syphilis.

1. Wassermann reaction in blood positive (80 to 90 per cent.).
Pressure frequently increased.
2. Phase I, reaction usually positive, exceptionally negative.
3. Lymphocytosis nearly always positive.
4. Wassermann in fluid.
 - (a) Original methods (0.2 c.c.) positive in about 10 per cent.
 - (b) Larger quantities nearly always positive (of value in diagnosis of multiple sclerosis, cerebral and spinal tumor).

These results of Nonne's summarize fairly accurately the present-day attitude on the value of the four reactions. The full significance of the findings can be gained only by reference to the originals. This field of work is rapidly expanding, and that which now appears on the frontiers of our knowledge will undoubtedly be much modified by the rapidly advancing army of investigators.

ADDITIONAL TESTS.—These additional tests are valuable because syphilis may be present with a negative Wassermann. These cases of so-called latent syphilis with negative Wassermann occur in about

35 per cent. of cases with no active signs of the disease but with a clear history of infection.

The Luetin Test.—This is a cutaneous test with a suspension of killed *pallida* cultures as prepared by Noguchi, known as luetin. A positive reaction appears in the form of a red papule with indurated areola in five or six days. This test is especially valuable in tertiary and latent syphilis, conditions in which the Wassermann reaction is sometimes negative. It is valuable also in differentiating from other conditions which might give a positive Wassermann, for this test appears to be a specific for syphilis.

Provocative Wassermann.—This test, like the luetin test, is valuable where syphilis is suspected, but the Wassermann is negative. It is dependent upon the fact that a negative Wassermann may be changed to positive after an injection of salvarsan. This change may occur quite promptly or only after several days. Nichols recommends making the Wassermann twenty-four and forty-eight hours after an injection (0.4 gram salvarsan or 0.6 gram neosalvarsan) and again after seven and fourteen days.

Lange's Colloidal Gold Test.—This test is dependant upon a color reaction which makes it very delicate. It has an added value because a single test, depending upon the degree of discoloration, tends to show whether we are dealing with a frank luetic or a metaluetic process. For this reason it is to some extent replacing the Wassermann test. For a description of the technique the student is referred to special works. A number of other tests have recently appeared, but those mentioned are the most important.

5. *The Eye Reflexes.*—In the diagnosis of syphilis of the nervous system the neurological examination of the eye reflexes is of paramount value. Here irregularity in the size of the pupils, irregularity in the pupillary margins, the impairment of the consensual light reflex, the slowing in reaction to light, fatigability of the light reflex, alterations in response to accommodation efforts, the full development of the Argyll-Robertson syndrome are all to be considered. These, one or all, constitute extremely delicate and valuable criteria for the clinical appraisal of syphilis of the central nervous system.

A fully developed Argyll-Robertson syndrome—loss of direct pupillary light reflex, with free and ample response to accommodation reflexes in one or both eyes—represents for the most part a fairly positive criterion of syphilis of the nervous system.

There are many who believe that this syndrome affords positive proof of nervous syphilis. This we do not believe to be true, not only upon clinical, but also upon anatomical grounds. Clinically the Argyll-Robertson syndrome has been observed following direct injury of the midbrain structures (pistol shot—Guillain), it has been observed in poisonings other than those of syphilis, alcohol (in Korsakow, Wernicke's polioencephalitis superior). It may result from press-

ures (tumors of third ventricle, pineal), from poliomyelitis, from trypanosomiasis, and from other rare anomalous disorders. Anatomically the syndrome represents implication of certain reflex paths in certain peculiar combinations, and such implications and combinations are purely fortuitous and accidental, *i. e.*, so far as nosology is concerned.

As a matter of fact, however, these combinations rarely take place except as a result of the widespread changes induced by one particular type of poisoning—the syphilitic virus—so that for clinical purposes the presence of a permanent, bilateral, Argyll-Robertson syndrome is nearly enough positive for syphilis to permit one to assume its presence, and to therapeutically guide one's self accordingly (Rose).

Here again, however, one meets with the pertinent suggestion that the testing for the Argyll-Robertson syndrome is not as simple as it is usually supposed. The ordinary devices of having a patient face the window, and then cover and uncover the opened eyes with the hand; focusing the eye upon a distant object, and then upon the finger in close proximity to the nose; these tests for the most part are entirely too crude to permit one to judge with certainty that the Argyll-Robertson syndrome is present. Such methods may suffice for the majority of instances, but in no field of neurology is it more desirable to utilize the most accurate methods than in dealing with the vexed question of syphilis of the nervous system.

Few clinicians can follow out the intricacies of Weiler's complicated methods, but they may be necessary in certain doubtful cases.

Repeated examinations, under carefully regulated supervision, in the daylight, and in the dark-room, are therefore desirable when testing for anomalies in the pupillary reflexes. Careful checking of the results obtained by the small pocket electric lamps is imperative, as occasionally they give anomalous results and may lead to serious errors (Oppenheim). The presence of a fully developed Argyll-Robertson syndrome may be said to be a highly probable positive proof of syphilis of the central nervous system, particularly of the cerebral and mid-brain neurons. Its absence, however, by no means negatives syphilis of the nervous system, since cerebral gummas, cerebral syphilis, paresis, tabes, syphilitic meningitis, meningomyelitis, cord gummas, syphilitic radiculitis, syphilitic neuritis, all may be present without any anomalies in the pupillary reflexes. It has already been pointed out that the laboratory findings of paresis and of tabes have antedated the development of neurological signs by at least a couple of years, and, moreover, it has been emphasized that pathological alterations in these pupillary reflexes are after all only chance happenings, that certain reflex arcs in certain combinations are caught in the mesh of the infiltrative, syphilitic alterations. The chance is a large one, it is true, but still it is purely a statistical matter of what has happened. One word may be added: in many cases of cerebrospinal syphilis one can gauge the progressive amelioration of the patient's condition

by the gradual return of the anomalous pupillary reflexes to a more normal condition. Thus, an absolute Argyll-Robertson syndrome may become a relative one. A unilateral Argyll-Robertson may be lost; slowly reacting pupils may show prompt reactions, irregularities in size may disappear; rapid fatigability may let up; a consensual light loss, often the first anomaly to appear in cerebrospinal syphilis, will clear up; irregularities in the pupillary outlines will make way to regular outlines, etc.

An inability to modify pathological pupillary reactions by ample syphilitic therapy argues in part for the chronicity of the process, or the inefficacy of treatment. This is not an absolute rule, however. It may be possible for a syphilitic process to permanently destroy portions of the pupillary reflex paths, and then be completely and permanently arrested. The pupillary reflexes, however, remain impaired.

In relation to this question of the pupillary reflexes and anti-syphilitic treatment the problem arises, What is the probable outcome of a syphilitic process which comparatively early in its course has destroyed the pupillary reflex paths? Can it be decided, say after two, five, or ten years, during which time there has existed an Argyll-Robertson syndrome and little else, that the disease has been completely and permanently arrested? Since the rapid extension of knowledge concerning the cerebrospinal fluid this question can be answered better at the present time than ever before.

If the signs of a meningitis—acute, subacute, or chronic, *i. e.*, increase in cellular elements, increase in globulin, and positive fluid Wassermann, remain absent, then it can be taken as highly probable that the disease process has been arrested. Judging by clinical methods alone a long-standing and unchangeable Argyll-Robertson pupil may be the only lesion in a practically cured syphilis of the nervous system. It is, however, to be realized that such a pupillary anomaly may exist as long as from twelve to sixteen years (alone) and then the patient may develop a paresis or tabes. Of eleven personal observations, now existing twenty years, only two syphilitic patients with long-standing Argyll-Robertson pupil have not developed further signs of brain syphilis. Certain deep-seated, chronic, syphilitic arterial processes, which may lead to focalized lesions, hemiplegia, aphasia, etc., may, however, go on for some time without distinct signs of meningeal irritation with the characteristic cellular reactions.

Clinical Forms.—It has already been stated that the clinical forms of syphilis of the nervous system are largely abstractions. The pathological processes are predominantly either meningeal, arterial, infiltrative, *i. e.*, gummatous in character, or parenchymatous alone or in combinations, and the clinical manifestations are extremely variable, complex, and confusing, depending upon the interactions of the pathological trends and the variations in anatomical paths interfered with.

Fortunately for the therapy, the clinical type is of secondary con-

sideration, yet there are certain therapeutic variables that render it desirable that a fuller analysis of clinical forms should be made than would at first sight seem advantageous. For instance, it may be recalled that certain patients with meningeal infiltrations of the base, with or without gummatous nodules, either of the base or of the convexity, may be at times clinically indistinguishable from a parenchymatous type. A nihilistic therapeutic attitude relative to the latter process would therefore work greatly to the disadvantage of a patient with the former. A like situation, though perhaps less frequently, is present in primary syphilitic endarteritis of the vessels. That type of syphilitic endarteritis of the small vessels of the cortex to which Alzheimer has called particular attention is another case in point.

The following clinical forms are to be distinguished:

1. Syphilis of cranial bones.
2. Syphilis of the basal meninges.
3. Syphilis of the convexity; epilepsies.
4. Cerebral syphilis—arterial types.
5. Cerebral syphilis—parenchymatous types (general paresis, tabo-paresis).
6. Syphilitic psychoses, acute and subacute.
7. Tabes.
8. Syphilitic spinal meningitis; meningomyelitis; myelitis; syphilitic radiculoneuritis, and related syphilitic syndromes.
9. Hereditary syphilis of nervous system.

For a fuller consideration of the protean variations the monographs of Rumpf, Nonne, Mott, Plaut, Oppenheim, Forster, Schaffer, and Fournier should be consulted.

1. Syphilis of the Cranial Bones Causing Nervous Symptoms.—

These were recognized as early as the end of the sixteenth century. Syphilis of the cranial bones shows itself practically in the form of gummata. Caries of the cranial bones alone, while known, rarely gives rise to nervous symptoms, headache excepted. These gummata may be circumscribed, in which case, if large enough, they give rise to the symptoms of a tumor of the brain, which signs are largely determined by the precise location of the gummata. These circumscribed gummata may attain enormous proportions. A personal observation (J.) recalls a gumma of the left frontal region (the size of a tennis ball) which originated in the bone dura and protruded into the right frontal lobe. Similar gummata are not infrequent.

Cranial bone gummata—usually involving the dura as well—are more frequently flattened and spreading. Here the symptoms of brain tumor are usually present. Headache, nausea, vomiting, sleeplessness, are among the general symptoms, while localizing signs in great variety, depending upon the situation of the gummatous masses are present. Epileptiform convulsions, lasting for years, may be the sole signs of such gummatous formations, occupying or due to

pressure upon the motor areas. Such patients are often mistakenly treated as epileptics, and the monographs of Mott, Oppenheim, Rumpf, Nonne, in recent years, are replete with autopsy records of such cases. Monoplegias of various sorts result from such, as also aphasias, word-blindness, and various cranial nerve palsies.

In addition various forms of periostitis and of osteitis occur. This latter may lead to exostoses or to osteoporosis. In connection with syphilis of the vertebræ it should be remembered that the processes are more often involved in distinction from tuberculosis that more frequently involves the body. Syphilis, too, more often affects the cervical, while tuberculosis more often the dorsal vertebræ.

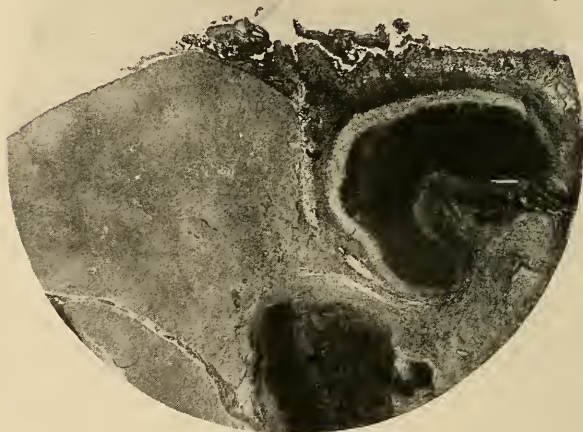


FIG. 253.—Gumma of brain.

Rarer cases of cranial bone caries of the base (sphenoid) complicated often by caries of the upper vertebræ, are also known. Petren has studied these in detail, and has shown the value of the *x*-rays in their diagnosis.

2. Syphilitic Meningitis of the Base.—This is the most common form of cerebral syphilis. Its most frequent site, in the beginning, is in and about the interpeduncular space, thus almost invariably involving the optic chiasm. From here it tends to spread in all directions, pressing into the sulci, thickening the meninges, by infiltration, by arterial disease, or by gummatous growth. Usually all types of pathological alteration are found. The gummatous masses not infrequently invade the brain structures as well, grow about the emerging or entering cranial nerves, and even involve the bones of the skull,

and the upper cervical vertebræ. Thus, spinal meningeal infiltrations almost invariably accompany this basal syphilitic meningitis. Whereas, the diffuse, conglomerate types are more frequent, isolated vascular disease, circumscribed gummata, or other simpler manifestations of the disease may occur, in which latter case the syndromes are apt to be simple.

The clinical course of the more frequent types of basal syphilitic meningitis often resembles general paresis, especially in the beginning, but the gradual extension of the infiltrating or gummatus developments introduce variants which often permit a differential diagnosis.

Headache is a frequent and early sign, often preceding other symptoms by weeks, months, or even years. It has the frequent nocturnal exacerbations so frequently pictured as characteristic of syphilitic headache. It is described variously as boring, stabbing, and percussion at the base, may show tenderness, though less frequently than in convexity meningitis. The cervical complications spoken of often result in stiffness of the neck.

An early implication of the optic nerve is to be expected. It shows itself (20 per cent. to 40 per cent. of the cases) either as a pressure neuritis (neuritic atrophy) of the nerve in one disk, then in the other, later, if headache and vomiting or other signs of intracranial pressure are prominent, choked disk in both eyes is apt to be present. Atrophic degeneration and optic neuritis are less often found. Marked diminution in visual acuity may be present without any disk evidences of disease.

The third nerve is frequently and usually irregularly involved. It is characteristic of basal syphilitic meningitis that successive branches are implicated. First one eye may show a ptosis, then perhaps an internal rectus palsy, then the other eye may show a dilated pupil, slow in its reactions to light, then ptosis develops here. Occasionally the accommodation reflex is lost. A series of cases will show a great variety of oculomotor palsies. A true Argyll-Robertson syndrome is not infrequently obtained.

Other cranial nerves are often included. Variations in the corneal reflex, in the sensibility, pain, anesthesiæ of the face, point to a trigeminal complication. A peripheral facial palsy may be present. In some individuals the deeper-lying cranial nerves (IXth, Xth, XIth, XIIth) are caught in the syphilitic extension, with their characteristic symptoms. The eighth nerve is probably frequently involved, but often too slightly to elicit complaint.

The mental picture is usually very striking. It is frequently that of a slowly developing apathy, or heaviness advancing to coma, or unconsciousness, with periods of acute confusion, possibly violent delirium. There is a marked variability in these patients from day to day and also considerable differences in different patients. Some patients develop a sort of drunken delirium; others are heavy and stupid and apathetic; others are furiously violent.

One special feature is frequently met with. This is a rapid altera-

tion in the mental picture, when, after a period of acute confusion or deep coma, the patient becomes almost practically clear within a few hours. This occurrence may even follow a period of convulsive seizures. Careful tests reveal an underlying series of defects it is true, but from a lay view-point the patient may appear to have made a complete recovery so far as his psychosis is concerned. Without treatment, however, the patient again develops his apathy, confusion, delirium, or coma and not infrequently dies in this state. Sometimes death results by suicide as the patient develops, slowly or rapidly, a distinct depression with possibly persecutory ideas.

Thus, mentally, the patients may show the old-time rubrics of acute confusion, dementia, mania, melancholia, paranoia, etc. This alone

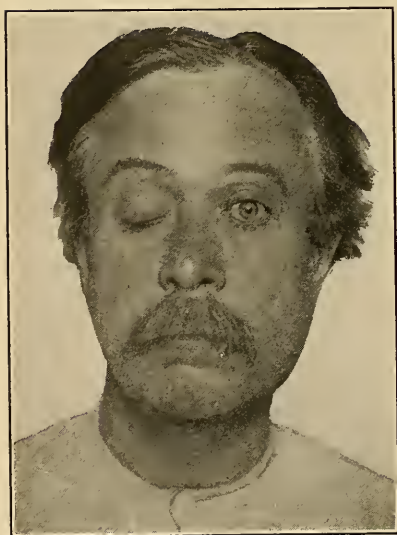


FIG. 254.—Cerebral syphilis ptosis.
Third nerve palsy.

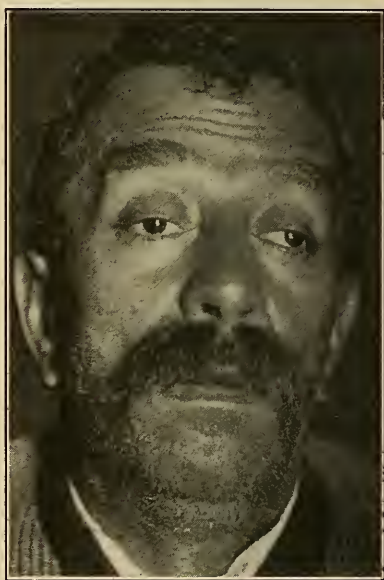


FIG. 255.—Cerebral syphilis with third
and fourth nerve palsies. (Sharp.)

indicates the futility of regarding the symptom pictures which have gone by these names as diseases, some for hundreds of years. Happily present-day psychiatry, largely under the influence of Kraepelin's teaching, recognizes them as only the protean and kaleidoscopic picture-formation of not only syphilis, but other disease processes as well.

Biological and cytological methods have permitted this definite change in attitude, and have shown the essential and close relationship of many diverse neurological and psychotic syndromes.

3. **Syphilitic Meningitis of the Convexity.**—This differs from the former only in the trend of its symptoms. The pathological processes are practically identical. Many individuals show that the process is



[Fig. 256.—Cerebral syphilis of the convexity. Chronic epileptiform convulsions.

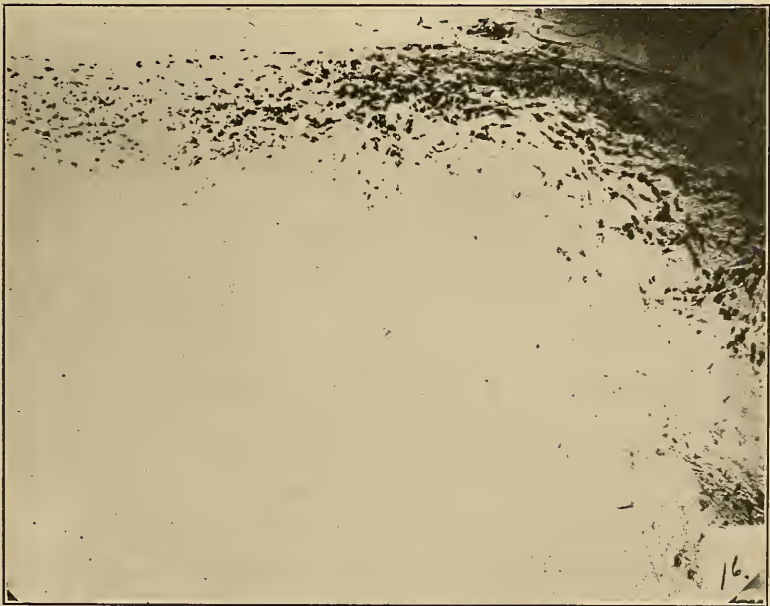


FIG. 257.—Syphilitic meningitis with giant-cell formation.

general, involving both the base and the convexity; in some instances the pathological changes being more marked on the convexity than on the base.

Convexity syphilis, like the basal variety, may be a fairly localized affair, or it may be diffuse. It may be limited to the meninges, or involve the bones, or the brain, or, as is most usual, all three.

Here, headache is a prominent sign. It is paroxysmal, and often shows a nocturnal increase in severity. Percussion affords valuable evidence, as localized tenderness is very common.

Here the general syndrome of brain pressure is usually less emphasized. Nausea, vomiting, giddiness, may be present, but are usually late in development, or more transitory. Optic nerve changes are less frequent. Isolated symptoms are more prominent. Epileptiform

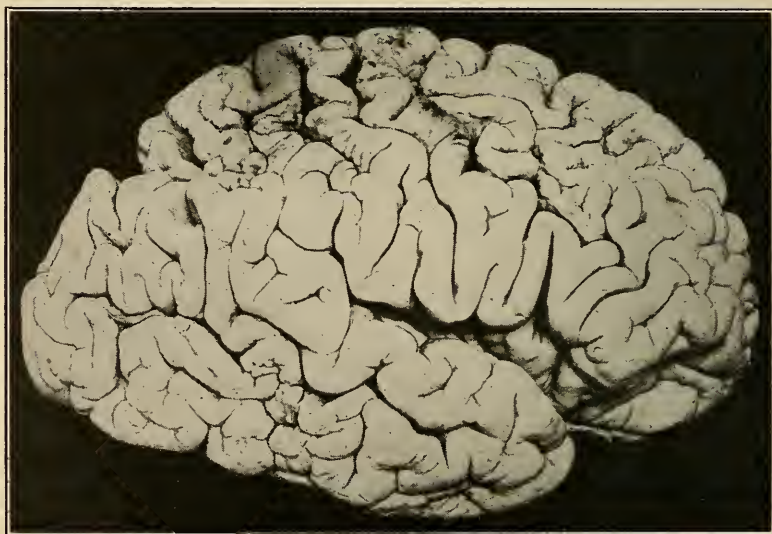


Fig. 258.—Chronic epileptiform convulsions due to syphilitic leptomeningitis and syphilitic vascular disease. (Lafora.)

convulsions indicate that the process is in or about the motor areas. Not infrequently the attacks are of the Jacksonian type. Involvement of Broca's convolutions produces temporary or more enduring motor aphasic attacks. Sometimes these aphasic attacks clear up in a few minutes, an hour or so, or a few days. Minor speech difficulties may only indicate the possibility that an aphasia might develop. Complete motor aphasia develops only, as a rule, with hemiplegic or monoplegic accompaniments. Pseudobulbar palsy attacks indicate a bilateral involvement, probably both cortical, less frequently cortical on one side, and subcortical on the opposite side.

Monoplegias of varying types are not infrequent. Bergmark¹

¹ Brain, 1911.

has devoted a large monograph to this study. Sensory disturbances, hemianesthesiæ, astereognosis, haptic hallucinations are met with.

With diffuse meningo-encephalitic changes the picture of general paresis is assumed, and it is particularly difficult to differentiate this disorder. The clinical pictures may be as various as those of paresis. Possibly the only means that we possess to distinguish is that claimed by Plaut, and apparently substantiated by Nonne and several others, that in paresis the four reactions are all positive; the cerebrospinal fluid showing a positive Wassermann with 0.05 c.c. of fluid. With meningo-encephalitis Hauptmann and Nonne have shown that the fluid is negative when small quantities are used, but positive when 0.4 cm. are employed.

This generalization seems to hold true not only for convexity meningitis, but also for those forms in which the base is more especially involved, although as will be later pointed out in the discussion of paresis, variability in the Wassermann reaction is not unusual.

4. Cerebral Syphilis.—Vascular Types.—In considering the symptomatology of patients who are thought to have cerebral syphilis, attention may again be called to the fact that the dividing lines between cerebral syphilis, basal meningeal syphilis, convexity syphilis, etc., are indefinite. Rarely does one find a pure basal or convex meningitis without some involvement of the cerebral substance on the one hand, while conversely it is as rare to find syphilitic processes strictly limited to the cerebral substance, and not involving the meninges. One can postulate pure types for purposes of description, but disease is rarely a respecter of one cerebral tissue more than another.

SYMPTOMS.—It is for this reason that one is constantly reminded of the multiplicity of symptoms found in cerebral syphilis. In this connection it would not be without profit to glance for a moment at the diagnoses of certain cases reported by Nonne in his monograph several times alluded to. The patients were illustrations of basal, or convexity, or encephalic brain syphilis, usually combined forms. The short descriptive diagnoses run as follows: Specific headache a year after infection, with secondaries in skin and mucous membranes; headache and pupillary anomalies; headache and obstinate vomiting, with tertiary testicle signs; progressive simple dementia cured by treatment; progressive dementia with defect; combination of convexity meningitis and paresis; gummatous meningo-encephalitis of convexity with general symptoms, choked disk and antisymphilitic treatment unavailing; surgical treatment of gummas with cure; Jacksonian epilepsy; cortical epilepsy, choked disk, paresis of left leg; cortical epilepsy, optic neuritis, arterial hemiplegia; cortical hemiepilepsy and general cortical symptoms; arterial hemiapoplexy with hemiepileptic convulsions; generalized epileptic seizures; hemianesthetic attacks with cortical general signs; uremia, etc. Such illustrations might be almost indefinitely continued. They are not

the exceptions, they are the rule. One is tempted to indulge in the generalization that one hundred consecutive patients with the types of cerebral syphilis under consideration would show one hundred different clinical syndromes. Practically all of those just enumerated belong to the convexity types of cerebral syphilis. Their enumeration may prove of service in localizing the process.

A similar series for the basal types may be equally of service: Gumma of right frontal lobe; pressure neuritis of optic nerve; choked disk with general cerebral symptoms; bilateral neuritic optic atrophy; recurrent optic atrophy; hemianopsia; bitemporal hemianopsia; homonymous left-sided hemianopsia with right-sided abducens palsy; temporal hemianopsia; cranial nerve and epilepsy; hemianopsia;



FIG. 259.—Syphilitic meningitis. Endarteritis of basilar artery.

hemianopic pupillary reaction, oculomotor palsy; epilepsy, oculomotor palsy; ptosis; internal rectus palsy; optic atrophy; partial oculomotor palsy, fifth, seventh palsy; second, third, fourth, fifth, sixth nerve involvement; fifth, sixth, seventh, eighth nerve, right arm and leg palsy; seventh, eighth, psychosis (paranoia acuta) with manic moods; third, fourth, sixth, tenth, eleventh nerve palsies; second, third, fourth, sixth, seventh with epilepsy; third, fourth, seventh, polyuria; isolated internal ophthalmoplegia—to mention any more

would be to needlessly extend this chapter. The lesson such findings indicate is obvious.

But eliminating, as far as possible, the varied syndromes of cortical or basal syphilitic meningitis, and limiting the discussion of the present section to these forms of cerebral syphilis due more particularly to arterial disease, what is its more frequent symptomatology?

In the first place it may be mentioned that arterial types of brain syphilis may be found very shortly after infection—as short a time as two or three months. Naunyn in a thorough study found that 48 per cent. of 335 cases reported on by him developed signs of cerebral syphilis within three years. On the other hand, forty years have been known to elapse between infection and the development of a cerebral syphilis.

Here the prodromal symptoms are usually headache, dizziness, sleeplessness, irritability, inability to apply one's self continuously to one's work, lack of interest in work, etc.; in general, the so-called neurasthenic syndrome. These are naturally not absolute.

Headache.—The headache is usually very disagreeable; it usually has a migratory character—here, there and elsewhere, usually dull, it is at times boring. It is inconstant, intermittent, often, not by any means always, worse at night than in the daytime. It may disappear for weeks or months, and then suddenly reappear. It may also be the only sign of cerebral syphilis for months or even years.

Dizziness.—Dizziness, in shorter or longer attacks, is very insignificant of the arterial disease. It is usually associated with the headache but may appear as the single symptom of brain syphilis. Like the headache it is apt to be increased by mental or physical work.

Insomnia.—Insomnia is frequent, often obstinate, quite variable and not infrequently sleep is made irregular and non-restful by the sense of heaviness in the head or actual headache.

Psychical Disturbances.—Psychical disturbances are the rule in these patients with arterial disease of the cerebrum. They become more or less apathetic, lose interest in their work; are unable to work because of forgetfulness or inefficiency. With this there is increasing irritability, an inability to size up the situation. Such severe disturbance is arrived at only after some time as a rule.

Abnormal Sleepiness.—Abnormal sleepiness, coming on in attacks, is not infrequent—such periods of torpor or apathy often intermitting with periods of anxiety or of acute restlessness or excitement. Periods of stupor or semidrunken states may occur. They often presage more distinct neurological signs, being based as they frequently are, upon sudden extravasation, infiltration or thrombotic plugging off of the blood from small areas of brain tissue.

Many patients with cerebral syphilis of this general character remain in this condition, it may be for some time; they show a picture precisely similar to certain patients with general paresis. It is in this general group that the greatest difficulties in diagnosis occur. As

has been previously stated, a positive "four reactions" is the sole criterion for differentiating the two in the present state of our knowledge.

Brain tumor is also to be thought of in diagnosis. Paresis, brain tumor, possibly gumma, cerebral syphilis, at times cannot be distinguished one from another clinically. With brain tumor, non-complicated by syphilis, the absence of the four reactions affords a positive criterion.

Local Symptoms.—As a rule, however, the greater number of individuals with cerebral syphilis develop local symptoms, and neurologic rather than psychiatric syndromes come into relief, or the latter are intermingled with the former. Palsies develop. These are transitory, partial, not widespread, or may be severe, complete, and permanent, showing various hemiplegic syndromes, according to the anatomical site of major disturbance—usually thrombotic—cortical, capsular, mid-brain, peduncles, pons, or medulla—the symptomatology of the different forms of which are dealt with elsewhere.

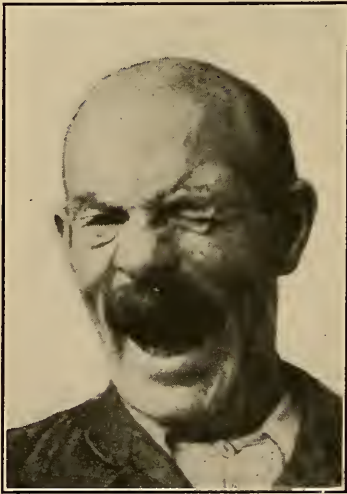


FIG. 260.—Pseudobulbar palsy from syphilitic disease. (Tilney.)

Successive attacks of mild and transitory palsies are very significant of cerebral syphilis. Monoplegias are not infrequent, one arm, one leg, one side of the face, possibly the cortical speech areas with, in case of double lesion, pseudobulbar palsy. Minor speech disturbances are extremely frequent, tremors of the facial muscles usually accompanying the stumbling, stuttering or drawling speech.

A list of the usual clinical diagnoses similar to those already outlined for basal or convexity meningitis would show a multiplicity of phenomena, no less complex, in the neurological field and certainly infinitely more varied in the mental symptom pictures. As these latter will be discussed more in detail in the section on the psychoses no further mention will be made of them here.

Parenchymatous Form.—General Paresis.—It is usual to separate paresis as well as tabes from other syphilitic disorders of the nervous system, under the general caption of para- or metasyphilitic disorders. Fournier is largely responsible for this, and to paresis and tabes he has added a number of other disorders, in other parts of the body, to which he applies the term para- or metasyphilitic.

Just why para- or metasyphilis is not known, especially so far

as the nervous system is concerned. Many ingenious hypotheses have been formulated with the purpose of explaining the differences between paresis and tabes, on the one hand, and other forms of nervous syphilis, secondary or tertiary, on the other. It would serve little purpose to enumerate them in detail, since none has as yet compelled conviction.



FIG. 261.—Cerebral syphilis (arterial type) with softening. Aphasia, hemiplegia, advancing dementia. (Lafora.)

To return to the syphilitic etiology of paresis. It is practically conceded "no syphilis, no paresis." One is not speaking now of those few individuals who, either because of the presence of brain tumor, or the existence of arteriosclerosis, or of other cerebral disorder, show a close clinical resemblance to paresis.

Finally the findings by Moore and Noguchi of *Treponema pallidum* in twelve of seventy parietic brains serve to render more certain the relationship of the organism to the disease.

But syphilis does not by any means necessarily lead to paresis, for-

tunately. The most recent studies of Mettler show that about 2 per cent. of those infected by syphilis develop paresis. A considerably larger percentage develop cerebrospinal syphilis—how large, can only be surmised.



FIG. 262.—*Treponema pallidum* in the brain of a paretic. (Moore.)

The question then arises, How is it that in certain cases a disorder arises, usually more than five, more frequently about ten years after infection which, while closely resembling many forms of cerebral syphilis, yet differs from it in certain very noteworthy particulars, and what underlies these differences? In other words, Why para- or metasyphilis?

Naturally there are those who say there is no difference, either anatomically, biologically, or therapeutically. They are in the minority with certain well-developed arguments, some of which are as yet unanswerable. The present-day attitude is to maintain a distinction between the strictly cerebral syphilitic disorders and general paresis, chiefly because the histological pathology is unique, the biological tests are different, and the results of therapy diverse.

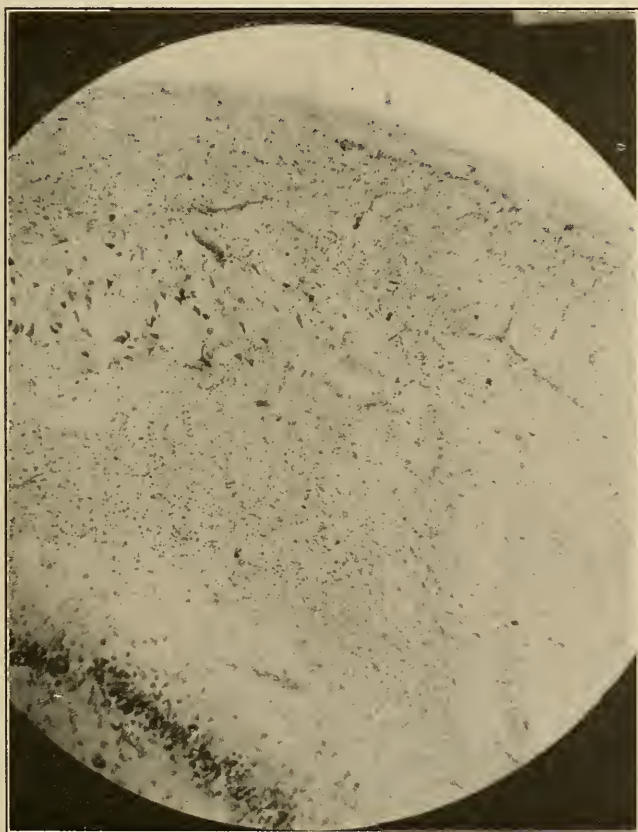


FIG. 263.—Ammon's horn in paresis. Pyramidal-cell layer has disappeared, vessels infiltrated.

Alzheimer and Nissl have laid down fundamental distinctions in the pathological picture. The chief points are quantitative, and to a less extent qualitative. In paresis the parenchymatous changes are predominant; in syphilis the vascular. Their researches have remained uncontroverted, although modified.

SYMPTOMS.—The syndrome is characterized by a bewildering multiplicity of forms, which, shifting in the individual patient from

month to month, at times even from day to day, prevent any clean-cut description that will embrace its many vagaries. Attempts have been made to create empirical types. Thus, Binswanger, in a notable study, created three types based on certain anatomical peculiarities—meningitic-hydrocephalic, hemorrhagic, and taboparetic forms. Such an anatomical subdivision has no reliable clinical counterparts.

Attempts at the erection of clinical types are perhaps slightly more encouraging, for there are certain patients who will run a course true to type.

The earlier monographs of Simon and Kraftt-Ebing, and the later ones of Joffroy, Obersteiner, Klippel, and Kraepelin¹ suggest the following groups:

1. Simple dementing types.
2. Simple depressed types.
3. The expansive or so-called classical type.
4. The agitated types.
5. The irregular types with localized symptoms, Lissauer, taboparetic form.
6. Juvenile paresis.

Before even attempting a description of these purely artificial creations, pictures which are constantly shifting and showing combinations of details, a brief glance at the chief symptom components is advisable.

These have frequently been divided into the mental and physical, but as this is a purely arbitrary distinction it will not be emphasized here. As has been noted, a diagnosis of an impending paresis may be made, at times some years before its onset, by the findings in the cerebrospinal fluid, but attention is here first focussed upon the mental picture. A peculiar psychological weakness is one of the early phenomena. A difficulty in perceiving external impressions shows this intellectual loss. In the early stages it may require special study of reaction times, which are usually lengthened, but soon absent-mindedness, inattention, loss of details, forgetfulness of important facts, become apparent. There is a gradually developing loss of ability for prolonged mental effort; in conversation finer shades of meaning are lost, the patient is no longer alert and keen, as perhaps has been his normal habit. The mental deterioration going on leads to many changes in his usual conduct, until the patient may be no longer quite sure of himself in his customary surroundings.

Certain patients develop a state of dreamy consciousness, as though in a mildly intoxicated state.

Increased fatigability is another early symptom. Much has been written of the preneurasthenic stages of paresis. This excessive fatigue may prevent him from starting anything new—sometimes he even falls asleep while at work or in conversation.

¹ General Paresis, Nervous and Mental Disease Monograph Series, No. 15.

Defects of Retention and Memory.—Retention and memory soon commence to show defects. Careful studies in the early stages have shown difficulties in association, lessened capacity for learning, dis-



FIG. 264.—Paresis. Cortical changes. Epileptiform convulsions. (Lafora.)

turbance in attention, often with good retention. The patients forget recent happenings more readily, not knowing what has transpired



FIG. 265.—Paresis with Korsakow syndrome; Wernicke's polioencephalitis superior.

a week ago, yesterday, sometimes a half-hour ago. These grosser defects belong to the later stages as a rule. The memory of time relations gradually slips away, the patient being unable to arrange suc-

cessive phases in an orderly series. Thus, many of these patients show the greatest defects in their appreciation of time differences, when married, age of oldest child, and related striking facts of life. In later stages all sense of time may become effaced.

Impoverishment of Ideas.—A gradual impoverishment of ideas takes place, varying in degree from month to month and showing also great variability with different patients. Those associations most in use usually persist the longest, and all tests should take into consideration special aptitudes. In the later stages the patients have lost practically all of their mental possessions—the gap at times being filled in by retrospective, confabulatory reminiscences. Certain of the patients resemble patients with a Korsakow syndrome.

Loss of Judgment.—Loss of judgment naturally goes on *pari passu* with the general psychical disintegration. In the early stages even refined methods of testing, such as those of Gregor, show that uncertainties, contradictions, logical lapses are not infrequent. The patients are easily distracted by sound associations, $9 \times 9 = 99$, etc. As this loss of judgment goes on the patients may make the most absurd plans. They do the most unusual things, often involving their entire fortunes and playing havoc with all of their carefully raised social fabric. Dream world and real world become hopelessly confused in this fundamental psychic crumbling.

Hallucinations, illusions, and changes in simple sensory perception are found, but they are not, as a rule, prominent features in paresis. Delusion formation is naturally present in many instances, although certain patients may go through the disorder with but few delusional developments.

Delusions.—The delusional ideas vary immensely—they are usually senseless and fantastic and when combined with active creative phantasies, as they frequently are, especially in agitated or excited periods, pass all bounds. These patients think in millions, billions, quadrillions, etc. They are princes, kings, emperors, potentates, priests, Christ, god, supergods. They have rubies, pearls, diamonds, emeralds; two wives, a dozen, a harem, thousands of beautiful women, etc.

These delusional ideas, simple or phantastic, are also liable to great lability. They are always changing; contradictory as well as unconscious. New ones come, old ones go, revivals take place. Progressions may go backward: Now they have millions, next moment have thousands; now a king, in ten minutes a fine soldier. One can at times, by talking with these patients, expand or contradict their delusional exuberance almost at will.

The same characteristics may be noted in regard to delusions of a depressive or hypochondriacal nature. Nihilistic delusions such as believing they have no stomach, no heart, or are dead are not infrequent in these types.

Alteration of Emotional Activity.—The disposition or emotional reactivity is involved, as is the intelligence. As a rule the patients

in the early stages are hyperexcitable—others, however, are markedly depressed. They are apt to be touchy, surly, cross, even having violent outbursts for the most trivial events. There is often a distinct damper in their higher ethical feeling, so that the stimulus of conversation, the joy of music or art, of various social relations gives way to a careless indifference, often at great variance with the psychomotor activity of the patient.

As the disorder progresses the mood is apt to be colored by the delusional interpretations. Anger and laughter may follow one another in quick succession, and a great variety of fleeting, changeable, often contradictory, emotional states are passed through.

Character Alterations.—The character alterations are predominant. Decision is progressively lost; instability and foolhardiness alternating with obstinacy and perverseness. Initiative is reduced, and the patient may become as clay in the potter's hand; such periods often alternate irregularly with impulsive heedlessness. Kraepelin relates the case of a patient who stepped out of a second-story window to pick up a cigar that he happened to notice on the walk beneath him. Criminal actions may be committed in just the same manner as the case of the paretic who shot at Mayor Gaynor, of New York. Suicide may occasionally take place in the same manner. Stealing is by no means infrequent, and sexual misdemeanors and crimes are extremely prevalent. This blunting of the repressions inculcated by the force of civilization is particularly noticeable, and predominantly in the sexual sphere. Hence results the frequent telling of lewd stories, consorting with people of quite inferior social status, exhibitionism, shameful and open masturbation, and even sexual assaults.

Neurological Signs.—Here one finds not infrequently in the beginning phases a dull, heavy headache. Hyperacusis often precedes the blunting of the special sense, and various localized disturbances, such as word blindness, word deafness, auditory hallucinations, apraxia, asymbolia, astereognosis, indicate a special localization for the time being in more or less definite cortical areas. Optic nerve atrophy occurs, at times early, in from 5 to 10 per cent. of the cases. Special changes in the optic disk are recognizable in from 12 to 50 per cent. of the cases.

Changes in Cutaneous Sensibility.—Very frequently cutaneous sensibility is modified—sharp pains, numbness, itching, etc., occur, and in those forms recognized as taboparetic these often show the special localizations of the tabetic. Out of these changed sensations delusional interpretations frequently arise. A general insensibility to peripheral stimuli develops later, and the patient may then pay little attention to any kind of irritant, heat or cold, full bladder, distended rectum, etc. Occasionally such patients mutilate themselves, cutting off a finger, or the tongue, or the testes in order to get rid of what seems to them a foreign body.

Motor Incoördinations.—Motor incoördinations, from initial trembling to more high-grade ataxias, apraxias, adiachokinesias, Romberg, asynergias, are common. Intention tremor is not infrequent, and perseveration is almost never missed in the later stages.

Disorders of Speech.—In speech these motor difficulties have been specially studied since Esquirol first laid stress upon such changes in mental cases. Frequently beginning with slight stumbling, a slurring over certain letters or syllables, *r*, *l*, etc., the paretic develops very characteristic speech anomalies. These come out with marked prominence in the use of test phrases—electricity, Methodist Episcopal, organization, truly rural, third cavalry brigade, etc.—when certain letters are repeated, stumbled over, or elided. Paraphasia, aphasia, ataxia of speech, perseveration are among the frequent later developments until only a mumbling may be possible in some of the last stages. Similar changes take place in writing.



FIG. 266.—Set of pictures of parietic convulsion. (Kraepelin.)

Eye Symptoms.—In the ocular movements, analogous difficulties are observed and in the pupils one observes a variety of changes. Statistical studies show these pupillary anomalies to be extremely frequent. Differences in size from 50 to 80 per cent. (Rache); distortion of the pupillary outlines, 74 per cent. (Joffroy); Argyll-Robertson pupil, 50 to 70 per cent. (Westphal, Junius, Arndt, etc.). Many of these pupillary anomalies undergo considerable variation, changing from time to time even without treatment. Loss of consensual light reflex, as already noted, is often one of the earliest, and at the same time one of the most persistent of the pupillary anomalies.

Convulsive Phenomena.—Convulsive phenomena, epileptiform or apoplectiform in character, are rarely missed in paresis. They are usually of the cortical epileptic type. They often occur early in the disease or may punctuate any period in its development. At times limited, they more often are generalized, and frequently have prodromata, such as dreamy states, motor incoördination, thickness of speech, twitchings, etc., as a rule occurring early. As isolated phenomena, typical status attacks may be observed, with as many as 100 or more epileptiform crises in twenty-four hours. An attack in the

very early stages may last only a few seconds; the patient suddenly sinks back on his chair, and is all right in a few moments—while, on the other hand, status attacks may persist a week or even more. Unconsciousness is usual, though it may be very slight or fleeting. A vast variety of focal residuals have been described.

Similar changes may be observed on the sensory side of the nervous system, and so-called psychic equivalents, as in the more classical epilepsies, are frequent.

Statistical studies show the very great frequency of these attacks, Obersteiner recording them as often as in 90 per cent. of his patients; while Junius and Arndt in their recent extensive study give them as occurring in 53 per cent. A personal study (*J.*) of two hundred cases showed them in 78 per cent. of the patients. Kraepelin believes that treatment in bed limits the number and frequency of the attacks; his Munich statistics show an incidence of about 65 per cent.

Alterations in Reflexes.—The tendon reflexes—triceps, radius, knee-jerks and Achilles—are usually positively involved, either excessive, in the greater number of cases, or diminished, especially in those patients with posterior cord involvement, which is frequent. When the deep reflexes are found to be increased other symptoms of involvement of the pyramidal tracts are not infrequent. Babinski reflex, very frequently Chaddock's external malleolar sign, at times the paradoxical reflex of Gordon occurs. Possibly there is an ankle clonus, and spasticity in gait is present. If, on the other hand, the deep reflexes are diminished, other signs of involvement of the position sense and deep sensibility fibers, travelling the posterior column pathways, are usually found. Ataxia, Romberg, girdle sensations, anesthesiæ, etc., pains of the radicular type, are also often encountered in these taboparetics.

In most of the patients there is great variability in the two sides. Occasionally one finds spasticity of one and hypotonia and ataxia of the other, and combined symptoms are to be expected in the later stages, especially in those patients with prominent cord localizations.

In the final stages contractures occur in the bed-ridden patient. They are unable to do anything and muscular twitches, spasms, localized atrophies, and a veritable museum of anomalies is to be looked for.

Findings in Cerebrospinal Fluid.—The findings in the cerebrospinal fluid have already been discussed. Suffice it to say here that they are of paramount importance and a diagnosis of paresis without the signs obtainable in the cerebrospinal fluid must always be regarded as lacking in a most important element.

Enough has been said to show that the clinical picture of paresis may be closely counterfeited by a number of other pathological states—notably brain tumor, cerebrospinal syphilis, arteriosclerosis, chronic alcoholism, sleeping sickness, etc.

The findings in the fluid are very definite. A positive four reactions,

the fluid used in small quantities—0.05 to 0.2 c.c.—is almost certainly diagnostic of paresis, yet at times it would appear that positive four reactions are found in other syphilitic processes which do not behave like paresis. The earlier didactic attitude of Plaut seems to be in need of some revision, but at the present time there is not sufficient autopsy-controlled material to permit absolute dicta.

Vasomotor and Trophic Disturbances.—Vasomotor and trophic disturbances may appear early and come and go; among them skin eruptions, such as herpes, and pemphigus are the commoner types met with. The ready appearance of bed-sores and abscesses indicate the lowered resistance of the skin and subcutaneous structures.

The bodily temperature may show considerable variation, even on opposite sides of the body; it is usually subnormal in the later stages, save following convulsive seizure. Sleep is irregular, especially in the excited stages, when the parietic may not sleep for days—whereas, in torpid stages or in those quiet, dementing forms the patient sleeps or is in a doze much of the time.

The appetite is capricious, and the bodily weight is apt to fall off in the early stages and during excitement, to become much increased in the torpid, quiet states.

Disorders of the bladder, and incontinence of urine and feces, all sooner or later come within the outlines of the picture.

Remissions.—One clinical feature which is very striking is the tendency of this disorder to show marked remissions. In certain respects this is a general law in disease processes, but in paresis it appears most striking because of the almost miraculous change that takes place in the patient. Such patients one would say were about to die; they become absolutely helpless, convulsion follows convulsion; in the interim they know nothing, are bed-ridden, have to be fed, soil themselves, and are reduced simply to breathing, heart-beating automata. They may remain in this condition for weeks and months, and then pick up a little, and then more and more, and within a space of six weeks to three months many such patients appear to be almost well and like themselves. They have risen from the dead, and strange to say, although the relatives, friends, and business associates have been told over and over again perhaps, for they should be, that this is not a cure, that it is only a remission of symptoms, the patient is frequently restored to all his civil rights and given full control of his affairs. In the majority of cases this is disastrous; he may launch out into new lines, involve his fortune, marry unwisely, and then after a few months, perhaps a year—the longer remissions on record have been five or six years—the average in about six months—the symptoms return, often in rapid progression, and usually lead to death after variable intervals of from six months to a few years.

Forms.—To return now to the subject of the forms—those more or less artificial groups which for the purposes of description psychiatrists are agreed upon.

The symptomatology of paresis varies within such wide limits because of the extent and distribution of the pathological changes; because of the individual make-up of the patients, and because the disease process not only affects the highest psychological levels but strikes deep into the physico-chemical and sympathetic foundations upon which these higher levels are built. The disease presents, therefore, a combination of psychological symptoms more or less explainable at that level coupled with disintegrations of a much more material character and stable organization.

1. *Dementing Form.*—That which characterizes this general group is the progressive mental deterioration with motor paresis. Excite-

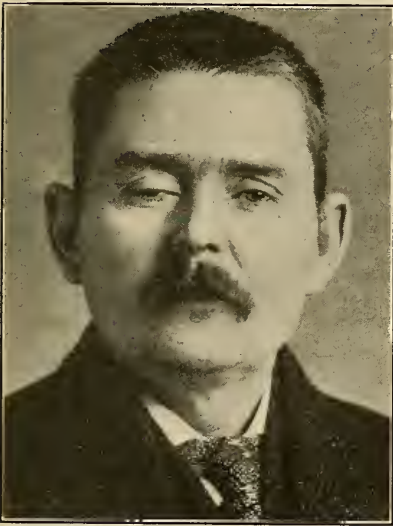


FIG. 267.—Simple dementing form of paresis.



FIG. 268.—Paresis. Demented type of facies.

ments, convulsions, extravagant delusion formations are not prominent and when present are transitory. In these individuals there is the early period of nervous irritable weakness, with loss of mental alertness, moodiness, inability to work, forgetfulness, and steadily increasing poverty of thought. Naturally, the picture beginning in this way may suddenly change. This is sufficient to throw the patient into another group—but if the development is of the slow, progressive nature, gradually advancing mental weakness, fleeting delusional ideas, often with childish, weak-minded features, these are the general symptoms of the dementing type of paresis.

2. *Depressed Forms.*—Here anxious depression is in the foreground of the mental picture. Hypochondriacal, delusional states are promi-

ment. The patients continually complain about bodily discomfort; have lost their intestines, or have destroyed their manhood by masturbation or sexual excesses. These delusional ideas become more and more nonsensical. In many instances the hypochondriacal ideas are dependent upon fancied sinful actions or wrong-doing. They are great sinners, they must be protected from the police, they fear they will be sent away. Such patients often have persecutory ideas, and when such are prominent early in the disease, before there is marked deterioration, they are frequently regarded as "paranoiacs" especially by those schools which regard names as disease entities and seek for diagnoses from a "pathognomonic" symptom. These paretics with persecutory ideas often have pronounced hallucinations of hearing.

Notwithstanding these hypochondriacal or persecutory, delusional interpretations, these patients are markedly indifferent; they are apathetic, talk and move in a monotonous, dull manner, and take little interest in their surroundings.

Thus the loss of energy, the libido in Jung's sense, not being able to go forward in the affairs of life, a marked pathological introversion takes place, and the regression takes hold of all sorts of nonsensical, childish, infantile, and archaic phantasies. Pathological projection also is common, and one has a regular chaos of pathological mental mechanisms. The sense of reality is so markedly impaired, and the affective relativity so cut off, that the nonsensical beliefs have no corresponding or adequate emotional relationships. The personality is fragmenting and disintegrating.

Childish regressions of hero formation appear. The patient is a god, a king, an emperor; like Jack and the beanstalk, he is miles high; as in the Lilliputian, he is a great giant; as in Midas' touch, or Aladdin's lamp, he breaks the bank at Monte Carlo, or is the owner of immense gold mines, fabulously valuable jewels, etc.

Contrasting states of great inferiority, weakness, poverty, cause them to be very fearful, easily confused, easily lost; they beg for protection, hide from anger, or ask piteously for food, preserve scraps, etc.

They become unmanageable in bed, and finally in many the agitation and fear develop great resistance and violence. Self-destruction may be attempted, mutilation occasionally occurs. Most of these attempts, however, are fragmentary, non-sustained and bungling.

Stuporous states show a contrast to this marked violence. They may persist for weeks, months, or even years. The patients lie stupidly, "depressed," or anxious in bed, unclean and unmanageable. Special rigidities, catatonic-like in their nature, may develop.

The special statistics show that from 15 per cent. to 20 per cent. of the material in some of the larger European hospitals and clinics may be in general thrown into this depressed category.

3. *Expansive Types.*—This general type has been for years considered "classical," yet they are not as frequent as the demented types. Thus, Kraepelin gives 30 per cent. in his Heidelberg series, Junius

and Arndt 27 per cent. in their Berlin material. It is to be regretted that the specialist has failed to emphasize this feature, which is of so much value to the general practitioner, and has confused the issues by speaking of a "change in type." It is of more value to insist upon the comparative rarity of the megalomaniac features of paresis, since, as the average medical man has been taught to recognize paresis by this sign, it is not to be wondered at that so much delay has occurred before the recognition of paresis. The emphasis should not be laid upon the comparatively rare expansive cases.

In this megalomaniac type the boastful ego rises to superior heights. Everything is seen from the stand-point of a feeling of abundant energy. At first the ideas are those of great exaltation, within the bounds of normal human experience, but soon the patient loses his earthly bonds and soars to superhuman unrealities. His strength is appalling; his education superior to any others in the world; he speaks ten, nay, all languages; has all wealth; all power; figures mount from thousands to millions, to pages of ciphers. And in kaleidoscopic changes, and great individual variation one learns of many marvels of superior excellence only dreamed of in childish phantasy, or seen in the boasts of inferior peoples.

One feature of this frightful megalomania, which has its very great ups and downs, should never be overlooked; namely, the tendency for such patients to commit sexual indiscretions, even atrocities; or to engage in the most foolhardy enterprises, thus jeopardizing life and property.

This feature in paresis is of so much importance that special attention should be devoted to the legal measures which should be invoked to prevent the worst consequences of this mental weakness.

That megalomania has a distinct deterioration background is seen in the frequent combination of a poor clerk, in a state institution, who speaks of the million-dollar novel he is writing. It consists of a few miserable scrawls on toilet paper, or on the edges of a daily newspaper. This is only a type. Such inconsistencies may be read of in the classics of psychiatry, from the work of Arnold, in 1700, to the present time.

These phantastic, exalted, euphoric states very frequently elaborate on sexual themes. Thus the patients have hundreds, millions of wives or concubines—"Solomon was a piker in this matter," boasted



FIG. 269.—Paresis, showing grandiose type.

a Bellevue patient. The children are more numerous and more beautiful than any promised to the ancient Hebrew heroes.

One patient, mentioned by Kraepelin, could lift ten elephants, was two hundred years old, 9 feet tall, was a beautiful Adonis, weighed four hundred pounds, had an iron chest, an arm of silver, a head of gold, 100 wives, 1000 million boys and girls, his urine was Rhine wine, and his feces were gold.



FIG. 270.—Excited parietic. (Kraepelin.)

The illustrations might be repeated *ad infinitum*. They are to be found in richer or poorer elaboration, in shorter or longer intervals of excitement, in this exalted euphoric type, but one may see a hundred parietics, as a general practitioner may see them, in the early stages, and never get a ghost of an idea of such experiences. Of this 100, sometime, sooner or later, 25 to 30 of them will be liable to exhibit the exalted, euphoric, megalomaniac picture here indicated rather than described.

Consciousness is usually much clouded in this type, especially while the delusional projections are in their full growth. Time, place, the great world, is a dreamy, far-off world of little moment to the mind engaged in its ambitious program. Continuity of thought is practically impossible, and chaos and anarchy exist. In such minds hallucinations are frequent.

The mood is happy, overflowing with schemes for good deeds and generosity, and all-embracing in its brotherly love. But coherence is not to be expected. Hypochondriacal ideas, such as delusions that there are worms in the head may rest in bizarre connection with the delusion of being a great philosopher, a Shakespeare, etc., and changes in mood are of frequent occurrence. Weeping follows ecstasy, and is replaced by beatific, sublime happiness. Sudden, passionate excitement leaps up under restraint, to subside, or to be diverted by such a trifle as a falling leaf, or a ring at the door bell.

The great psychomotor excitement is a striking feature, and one difficult to manage. These patients walk miles, are on the go, meeting people, busily engaged in everybody's business, making plans for self and others, and, when confined, the limits of a parietic's violence knows no bounds. He is transformed into a raving animal.

Throughout all of the excitement, divertibility, constant changing of plans, mixture of silly pleasure and superficial sadness, there is the note of great deterioration of critique and emotional degradation and deterioration which shows particularly in the conventions relative to one's person. Carelessness in dress, uncleanliness, grossness in eating, loss of finer susceptibilities, coarse expressions, frank immoralities—these are but a few of the possibilities in such lax conduct.

In watching such patients from day to day, one is struck by the immense variability in the picture. The ideas of grandeur may all vanish, the patient denies he ever said any such thing, he may get angry in a dispute over the matter, and then launch into a magnificent grandiloquent invective of colossal outlines.

As the dementia increases, these large ideas may entirely disappear, or be preserved, and appear on the surface only as a few words, or murmurs, "good to eat," "fine women," "millions," etc.

Finally in the later stages, the patients all sink to a more or less common level—"sans everything."

Among the expansive forms may be found the quick, galloping cases who die within a short time. Increasing experience seems to show, however, that these excited types indicate a very severe reactive process, and hence, if they do not die in the height of the reaction (galloping cases), they provide the greater number of the more stationary and protracted forms—those who make a partial recovery with defect, and who later disintegrate. Remissions seem to be common in this type as well.

4. *Agitated Forms.*—Those patients who show a predominant motor activity in the beginning may be said to be grouped here. Great

restlessness runs through the entire picture. The mental content is very variable—euphoric, depressed, hypochondriacal, mood colorations flit in and out. Galloping cases are usually grouped here, in which an extremely rapid and fatal course is present.

This is really only a subgroup of the preceding type, only artificially separated off by reason of the more consistently persistent psychomotor restlessness. Remissions are frequent, as are also the apoplectiform and epileptiform attacks. The pathological process simply has a wider extension in the motor areas.

The acute delirious cases, somewhat resembling delirium tremens of alcoholism, and independent of it, are arranged by Kraepelin in the agitated group.

5. *Irregular Types.*—*Lissauer*, etc. These patients, showing irregular forms of development; neurosymptomatic groupings, hemiplegias,

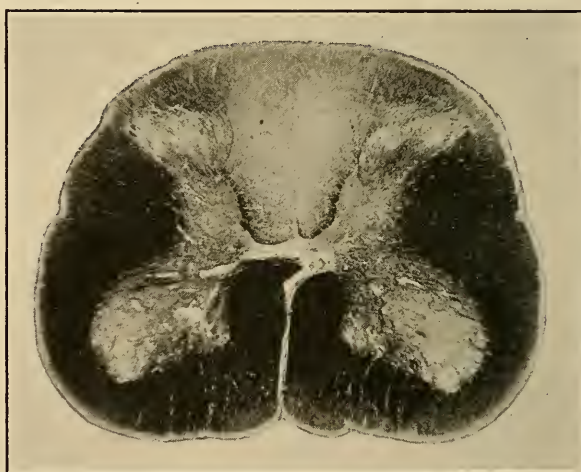


FIG. 271.—Paresis with tabetic changes in spinal cord.

etc., are here brought together. The hemiplegic and taboparetic groups are the more frequent.

Taboparesis.—Taboparesis is the more striking of these irregular forms and deserves a further outlining.

It has been assumed by many, especially by neurologists (*Schaffer*, for summary, 1912) that tabes may be regarded as a spinal paresis, and paresis a cerebral tabes; that is, the disease varies only by reason of the greater severity of the process in the one or the other localization.

Kraepelin, on the other hand, accentuates the opposing psychiatric view, that whereas, the two disorders are undoubtedly fundamentally syphilitic, yet they are two different kinds of processes, and that when the symptoms of tabes are added to paresis the changes in the cord are not exactly similar to those found in tabes limited to the cord. The different findings in the cerebrospinal fluid in the two disorders would point to some sort of a difference as well.

The matter still rests in the lap of the gods.

Clinically, taboparesis shows in a combination of the symptoms observed in the two forms. Raymond and Nageotte would have it that every paretic would show tabetic signs if he lived long enough. In those patients with pronounced tabetic onset one finds the frequent pupillary anomalies, the diminution or loss of the patellar reflexes, Romberg sign, ataxia of lower or upper extremities, or both, hypotonia, lancinating pains, crises, and arthropathies. These are found in the more definite taboparetics closely associated with the psychical



FIG. 272.—Juvenile paresis in the emaciated stage.

disturbances already outlined. In the more classical tabetic patients the mental disturbances, to which Cassirer and O. Meyer have devoted their attention, are very distinct from those of paresis.

6. *Juvenile Paresis*.—This form is quite distinct. It was apparently first recognized as late as 1877 by Clouston. It appears at the present time not infrequently, since the Wassermann-Plaut findings offer such certain criteria for its determination. Such tests seem necessary, since the clinical picture may be so extremely variable—hence it was overlooked—many patients dying diagnosed as “imbeciles.”

Here the patient may make a comparatively normal development to five or ten years of age—certain non-developmental forms probably belong here, but are now disregarded. Then the child's mentality seems to drop. In older children, ten to sixteen, this drop is more apparent. Poor memory, bad motor adaptation and gradual dementia appear. Childishness, fabrication, excitements and depressions, fears, and anxieties, are frequent. Epileptiform convulsions appear—many juvenile paretics are gathered into the almshouses and epileptic colonies as "epileptics with feeble-mindedness"—and after a course of three or four years, with gradually deepening mental disin-

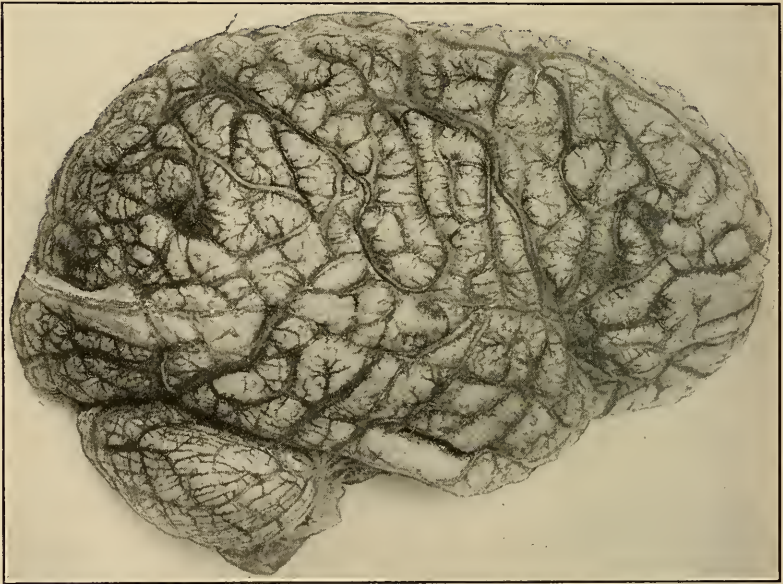


FIG. 273.—Brain of a patient with a syphilitic psychosis of acute maniacal type. Enlarged and engorged vessels. Syphilitic meningitis.

tegration, the patient dies. The histopathological changes are identical with those of the adult form.

It is noteworthy that the age of onset, from seven to twelve years, is the same length of time that in the adult form elapses between infection and the outcrop of the metasyphilitic disease.

SYPHILITIC PSYCHOSES.

In this section are included the psychoses which are associated with cerebral syphilis and with tabes. In the present state of our knowledge we are not prepared, as already stated, to make a clear distinction either on pathological, clinical, or psychological grounds between the so-called metasyphilitic and the more clearly syphilitic

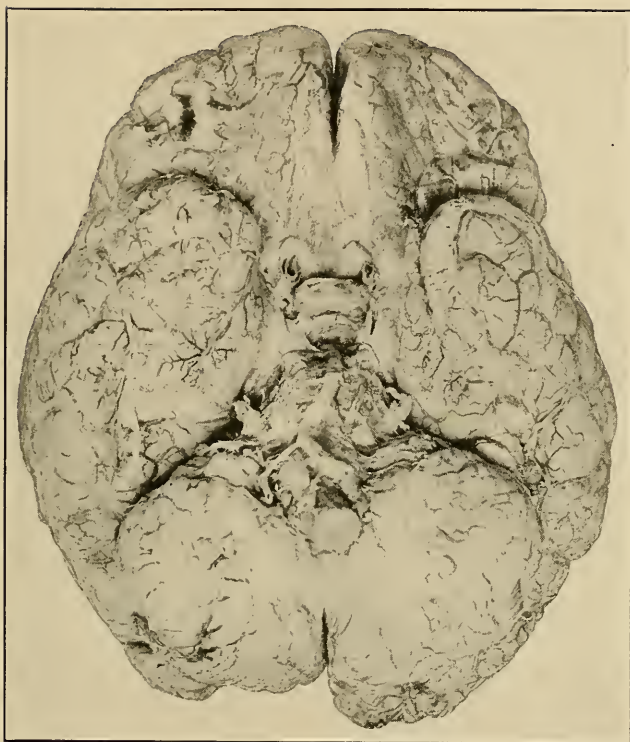


FIG. 274.—Brain of a patient with a chronic syphilitic psychosis of maniacal type. Syphilitic leptomenigitis, pachymeningitis.

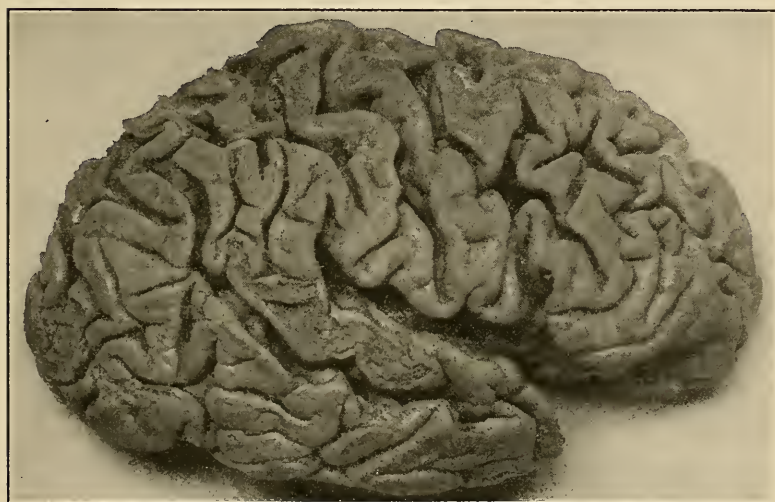


FIG. 275.—Brain of juvenile paretic showing marked atrophy.

conditions, and there are undoubtedly all sorts of gradations between these two practical divisions.

Forms.—*Neurasthenia.*—Kraepelin speaks of a syphilitic neurasthenia—a form of mental disturbance much written upon by earlier authors. The preneurasthenic phase of a cerebral syphilis or of a paresis is not now under review. It is apt to appear shortly after infection and manifest itself in a nervous discomfort, difficulty in thinking, irritability, disturbance of sleep, pressure in the head, variable and changeable discomfort and pain. To these may be added slight depression, dizziness, confusion, anxiety, slight difficulty in finding words, temperature variations, paresthesiæ, and nausea.

Many students prefer to interpret these symptoms as a direct result of the infection, and not as a circumscribed syndrome; but slight vascular changes, minute pupillary alterations, and particularly evidence of meningeal irritation as shown by the spinal-fluid lymphocytosis, point in the direction of its being something more than a simple, infectious reaction.

Plaut has described the *psychoses* which are associated with cerebral syphilis and with tabes in ten groups. The groups are as follows:

1. *Simple Luetic Weakness of Mind.*—This is the weakness of mind which usually goes with gross lesion of the brain, marked by hemiplegia or monoplegia. It is generally the result of the blocking of a considerable vessel by a thrombotic process and usually occurs in relatively young people. There is no well-defined type of mental defect resulting, as this is dependent, of course, upon the location of the lesion and upon the make-up of the individual. There may be depression or euphoria or a simple forgetfulness and indifference. Occasional cases of arteriosclerosis occurring early in life simulate this condition very closely.

2. *Syphilitic Pseudoparesis.*—Here we have a group of cases which seem to occupy all portions of the territory between the true syphilitic psychoses and paresis. On the mental side the distinction between pseudoparesis and paresis is practically impossible to make. Persistent auditory hallucinations, however, seem in experience to have pointed quite strongly to pseudoparesis. The most reliable diagnostic criterion is the behavior of the cerebrospinal fluid toward the Wassermann reaction, it being often negative in vascular syphilis and positive in paresis. While this is not an absolute differentiation and larger doses of the serum may produce the positive reaction, it is still, however, one of the most important differentials. It has to be remembered, too, that some cases of paresis are found with negatively reacting fluid, and rarely cases of lues with positively reacting fluid.

3. *Paranoid Forms Combined with Tabes.*—In this group are found patients who do not show any considerable deterioration, but present ideas of persecution with numerous auditory hallucinations over a considerable period of time. There is no self-reproach, they remain lively and affable, and what seems to be peculiar, present marked hallucinations of common sensibility accompanied by phantastic ideas.

4. *Paranoid Forms Without Tabetic Symptoms.*—In this group are found paranoid ideas combined with auditory hallucinations, rather

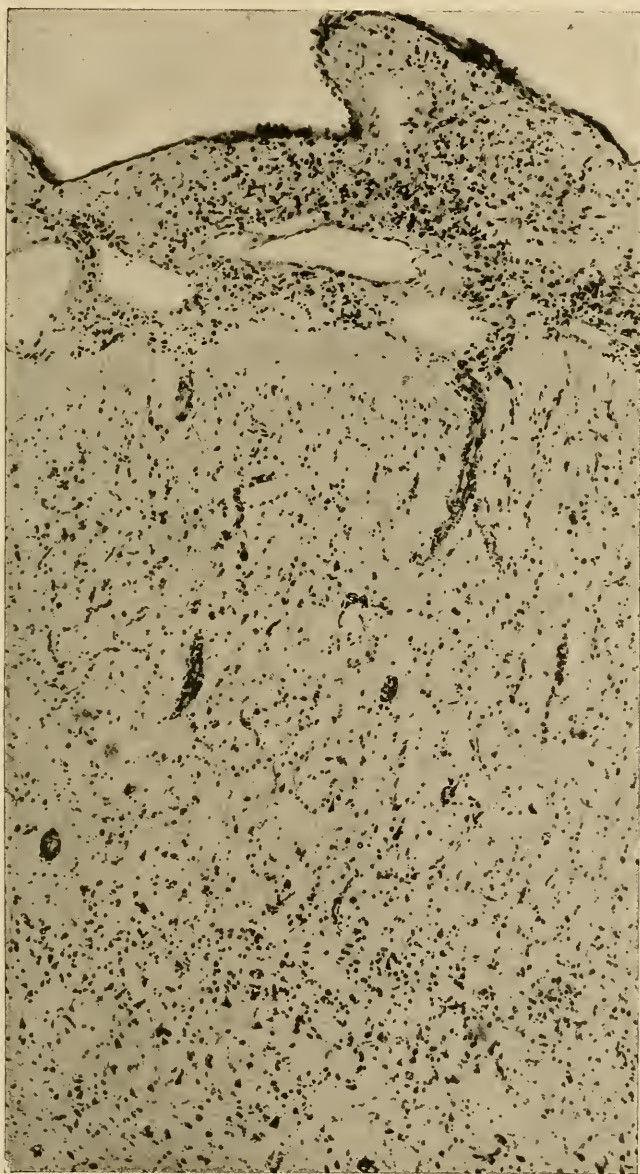


FIG. 276.—Devastation of cortex in paresis. (Kraepelin.)

resembling the alcoholic hallucinoses. A pronounced delusion of jealousy was present in one case described by Plaut. They have to be

separated from manic-depressive psychosis and particularly from dementia precox. The separation from the latter is made from the absence of catatonic signs and failure to develop marked evidences of defect.

5. *Certain Epileptic Forms.*—These appear to be due to the endarteritic changes in the small cortical vessels and may be combined with paralytic phenomena which develop as a result of the participation of the larger vessels in the disease process. The cases resemble very closely genuine epilepsy and must be differentiated by the neurological and serological findings. A case described by Plaut showed transitory dream states.

6. *Short Hallucinatory Confused States.*—These resemble the confusions associated with the epileptic forms, and the French have considered them in the same class with the crises.

7. *Psychotic Disturbances Associated with Syphilitic Cardiac Disease.*—This condition probably develops most frequently in connection with syphilitic aortitis.

8. *Psychoses Resembling Manic-depressive Psychosis.*—Here conditions are grouped which superficially very closely resemble the manic-depressive psychosis. As a rule, however, there is something to attract attention as indicating at least an aberrant form. On the mental side the delusions are more grotesque, more out of harmony with the personality of the patient, or show an unreasonableness which is not commensurate with the degree of excitement. On the physical side, of course, inactive pupils should lead to a serological examination. Occasionally such episodes occur a long time before the outcrop of frank symptoms of metasyphilitic disease.

9. *Mental Disorder Due to Syphilis as a Psychic Trauma.*—Here a psychogenic psychosis which is more apt to take a depressive form is included.

10. *Hereditary Luetic Mental Disturbances.*—This envisages psychopathically defective subjects and weak-minded children withluetice tiology. The exact relation between lues and various forms of weak-mindedness is not accurately known, but it is known that a large number of the feeble-minded group areluetice. Syphilitic brain disease may occur in early infancy and proceed for some time, producing only transient symptoms, perhaps an occasional convulsion, and ultimately lead to serious defect.

TABES.

History.—In any historical presentation a sharp distinction must be made between the name tabes dorsalis, and the disease as now understood. So far as is known, the former had its origin with Hippocrates, the latter, if one accepts the Post-Columbian origin of syphilis, could have come into existence among Europeans and their descendants after the Sixteenth century only.

The various interpretations given to the Hippocratic term through-

out the ages is a chapter of surprises. Spermatorrhea, gonorrhœa, leucorrhœa, gleet were its initial meanings, with or without signs of organic disease of the cord; when combined with cord signs—mye-

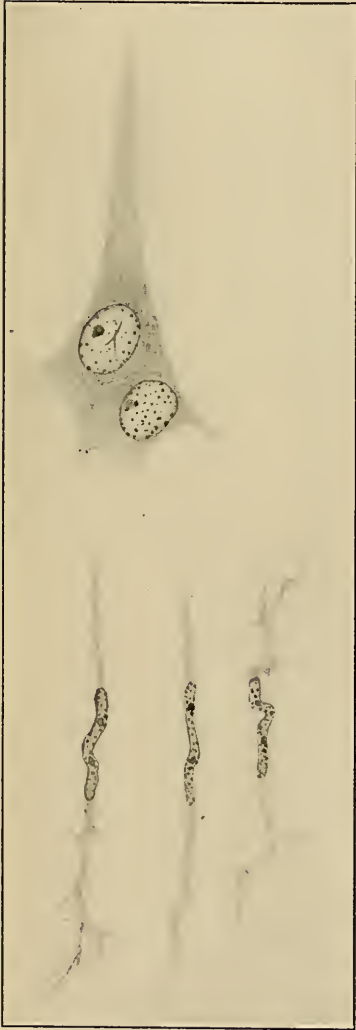


FIG. 277.—Ganglion-cell degeneration and rod cells in paresis. (Lafora.)



FIG. 278.—Axis-cylinder degeneration in paresis. (Kraepelin.)

litic processes, usually tuberculosis—Pott's, etc.—it was called tabes nervosa or myelophthisis. Out of this mass the disease of the present day was separated. It was natural that excessive venery should have

been attributed as the cause. As late as the early part of the last century (1804) the term persisted in the sense of a "nervous weak-

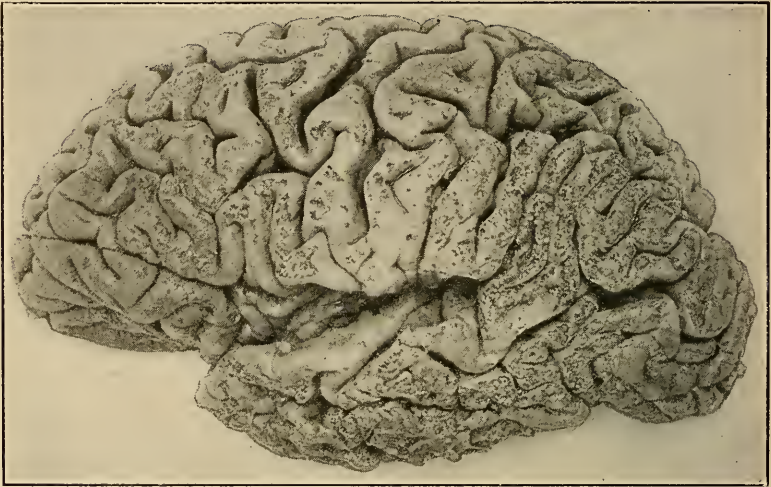


FIG. 279.—Brain of a parietic showing marked atrophy. Flaking of the cortex due to adhesions of the pia.

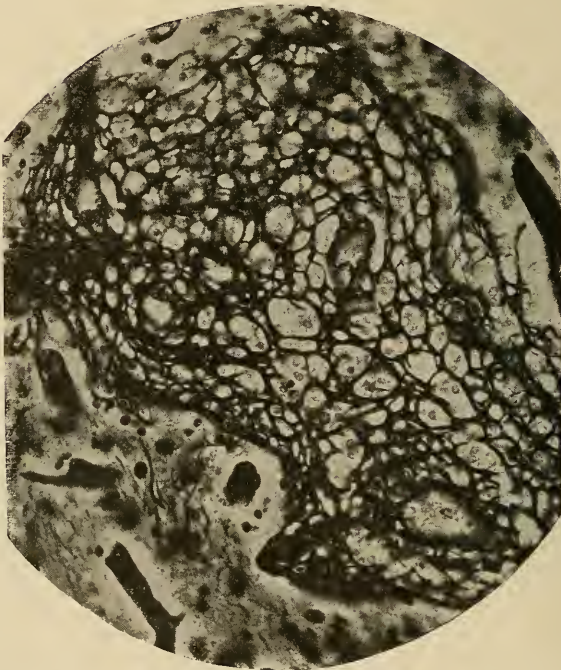


FIG. 280.—Organization of Virchow-Robin lymph spaces in general paresis. Tannin silver. (Achucarro.)

ness," *i. e.*, what today one would designate a "sexual neurasthenia," or a "sexual phobia." So far as can be made out from Littre's Hippocrates this is exactly what Hippocrates wrote about under the term *tabes dorsalis*. Even in the theses of Harles and Brera, in the early part of the nineteenth century, the relation between the cord, the vessels of the lower abdomen, and secretions from the genito-urinary organs was held to be a close one.

As to the earliest observations of modern *tabes*, the traces are very indistinct. To the clinicians of the sixteenth and seventeenth centuries, the confused mass of paraplegias was practically insoluble, and it would appear that it was only in the beginning of the nineteenth century that the process of differentiation took place. E. Horn (1816) called attention to a number of the important features including blindness; Weidenbach (1817) attempted to make a separate disease of it—he contested the inflammatory origin—said it had nothing to do with consumption, although still unable to break away from the belief in the excessive venery etiology then rampant. Schesmer (1819) described the peculiar gait in an unmistakable manner, while W. Horn (1827) emphasized the real absence of a true paralysis, and spoke of an ataxia whereby this affection was different from other forms of myelitis. Decker (1838) called attention to the swaying and unsteadiness with closed eyes, which was taken up by Romberg three years later and rechristened Romberg's sign.

Pathologically the characteristic cord signs were not unobserved. Hutin (1828) describes them, Ollivier of Angiers (1837) gave the picture, reproduced here in part, while Cruveilhier (1832-1845), in his Atlas, gives masterly clinical and pathological descriptions.

Romberg in the first edition of his *Lehrbuch* gave greater precision to the description, and Steinthal (1847) threw together the incomplete paralysis (or ataxia), the Romberg sign, and the characteristic gait, but without any real grasp of the situation clinically or patho-

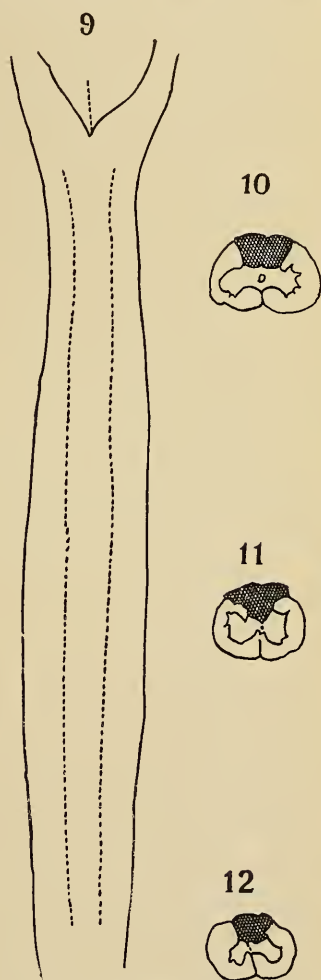


FIG. 281.—Illustration of the cord of a tabetic given by Ollivier of Angiers in 1837.

logically. Later Romberg, in the second edition of his text-book (1851) gave greater precision to the concept, and gave a classical description. Finally, Duchenne, in the years 1852 to 1858, elaborated the general idea, and gave the first complete and adequate description of the disorder. It may be said that Horn and Romberg had practically made out of the general tabes dorsalis collection a special tabes dorsalis collection in the sense of our present-day conceptions in the physiological and pathological fashioning of which Tod (1847), who seemed to grasp the fact that incoördination and posterior columns were related, Rokitsansky (1854), Virchow (1855), Türck (1856), Landry (1858), and Gull (1858) made lasting contributions. The recent history gives us the names of Charcot (1868), Vulpian, and Topinard, and in 1863 three monographs appear by Eisenmann, Leyden, and Friedreich, while the later contributions of Westphal (1875), Argyll-Robertson (1869), Marie, Redlich and Obersteiner, Nageotte, Oppenheim, Dejerine, Goldscheider, Erb, Nissl, Schaffer, and Alzheimer record the detailed studies in etiology, symptomatology, pathogenesis and spinal cord conditions of the last two decades.¹ Finally the even more recent work of the serologists, particularly Wassermann, Citron and Plaut,² and Noguchi have said the final word regarding the etiological factor, syphilis.

Etiology and Occurrence.—Syphilis is the only cause of tabes. The statistical method had almost proved the syphilitic etiology of tabes, but with the advent of the objective methods of Wassermann and his students, all doubts have vanished. In the more recent work, of the most competent serologists, syphilitic antibodies are found in the blood serum of practically every case (Plaut, *loc. cit.*). Furthermore, the cytological examination of the cerebrospinal fluid shows the presence of cellular exudates characteristic chiefly of the syphilitic processes, and as will be seen in the consideration of the pathological features; the syphilitic nature of many of the findings is beyond controversy. Finally *Treponema pallidum* has been found in the spinal cord areas in tabetics.

Pseudotabetic syndromes are known to occur in multiple sclerosis, in tumor, in caries, in poisoning by alcohol, pellagra, diabetes, ergot, etc.

What secondary factors are necessary to determine why this or that patient infected with syphilis should develop tabes cannot yet be answered. Less than one-half of 1 per cent. of the infected develop the disease, and other factors are demanded on *a priori* grounds to explain why one syphilitic individual develops it and 99 syphilitics do not; and this is true only for certain races. The many cases of congenital tabes, following a syphilis from the same source, suggest

¹ Jelliffe, On Some of the More Recent Literature of Tabes Dorsalis, Pathology and Etiology, *International Clinics*, 1907, ii, 257. Later literature, see Schaffer, Lewandowsky Handbuch and work by Massary, 1909.

² The Serodiagnosis of Syphilis in Psychiatry (*Nervous and Mental Disease Monograph Series*, 1911).

variations in the virus. Similarly the large number of tabetics who are known to have followed the wake of certain syphilitic prostitutes (Morell, Lavalee, Erb, Brosius—glass-blower cases). The other factor must be due to variations in resistance.

Both of these factors are impossible to measure, but it is highly doubtful if any of the many causes usually mentioned, such as exposure to cold and wet, trauma, excessive venery, etc., have anything to do with the after-development of a tabes. The multiplicity of assigned causes makes it more than probable that none are concerned. Ford Robertson's specific bacillus is certainly not a proven secondary factor. Trauma is highly doubtful. It may cause a pseudotabes, or may hasten the fuller development of the symptoms. Of heredity little is known. Charcot, Borgherini, Erb, and Gowers have laid much stress upon it.

Occurrence.—The majority of cases occur in the fourth decade, but this is largely due to the fact that the disorder comes on ten to twenty years after infection, and syphilitic infection usually occurs between the twentieth and thirtieth years. The disease may appear at almost any period after infection (fifteen to seventy years of age). The average runs from thirty-five to forty, the average interval after infection about fifteen years, with extremes at four to thirty-five. Dejerine and Raymond report cases forty-five and fifty years after infection. In certain races with a high syphilis percentage (Algiers) tabes is practically unknown, and there is every reason to believe that the syphilis has been there present since the sixteenth century. In other races, from three to five of every one hundred syphilitics develop the disease. In certain countries the ratio of men to women is 4 to 1, in others 10 to 1.

Occupation seems to play only that role that speaks for increased opportunities for syphilitic infection.

Symptoms.—The symptomatology of tabes is markedly diverse, and whereas it seemed at one time that it presented a more regular picture than other nervous disorders, accumulating experience shows that typical pictures, so-called, are the exception rather than the rule. In other words, there is no one symptom that may not be absent, and very few symptoms that may not be present in disorders other than tabes.

Nevertheless one can rely fairly well upon the following grouping of symptoms: Lancinating neuralgic pains, mostly in the lower extremities, usually preceding all of the other symptoms; paresthesia and related sensory disturbances, analgesias, hypo-esthesias, loss of the tendon reflexes (patellar, Achilles), incomplete or complete Argyll-Robertson pupil, unilateral or bilateral ataxia in both extremities, Romberg's sign, bladder disturbances, hypotonia, ocular palsies, positive Wassermann in the blood and lymphocytosis in the spinal fluid.

A host of other symptoms may be present in individual cases, either

early or late, but those just mentioned belong more particularly to the majority of the cases, and are usually sufficient to make an early diagnosis. A description of the individual symptoms will show somewhat of the proportionate occurrence, both in point of time and frequency.

Pains.—Severe lancinating pains occur in about 90 per cent. of the cases, and usually as an initial sign (60 per cent.). The patients complain in an almost stereotyped manner of having had sharp, severe, fugacious pains usually in the sciatic and crural distributions, which they speak of, and unfortunately are regarded even by physicians, as rheumatic. These pains come on in attacks, last a few minutes or more, several hours, a few days and then disappear, to again recur. They may precede the development of other symptoms by a few months or even many years (22 years—Erb). The average varies widely. They are an indication of the leptomeningitis or radiculitis which is one of the fundamental results of the syphilitic virus or products induced by it. They are usually of extreme severity and are much dreaded by the patient.

Whereas the distribution is predominantly sciatic or crural and radicular at first, the pains may be widely distributed, and may in fact start in any sensory root, cranial or spinal. Thus trigeminal neuralgia may be an initial sign, or the pains may affect the larynx, or the stomach, or the heart, the bladder, the testicles, the intestines, and give rise to various forms of crises, so characteristic and so much feared. The pains may be felt in the skin, or deep within.

Deep, boring pains are also present, usually later. Not infrequently the larger nerve trunks are somewhat painful to pressure. This fact may lead to confusion in separating an alcoholic or other neuritic pseudotabes.

Crises.—These have some relation to the pains of tabes, and are probably due to similar pathological alterations, but located in other sensory and sympathetic root areas. The best known are the gastric crises, noted as early as 1856 by Gull and recognized by Charcot (1868) as belonging to the general picture of tabes. The patients have sudden, violent gastric pain, radiating in all directions, and in the severe attacks accompanied by nausea, vomiting and great prostration. Like the pain attacks, these crises may last for hours, or a few days and then disappear for weeks or months to recur at irregular intervals. They disappear as rapidly as they come, and quite analogous to the lancinating pains, may be early or late symptoms. Certain cases of tabes begin with such crises.

Similar crises affecting other internal organs have the same etiology and course. Thus there are intestinal colics with diarrhea, rectal pains with tenesmus and diarrhea, vesical crises with strangury, urethral crises, renal colic-like attacks, testicular crises, vulvovaginal crises, laryngeal and diaphragmatic crises with cyanosis and dyspnea, pharyngeal crises with obstinate hiccough, bronchial crises with cough,

cardiac with angina-like attacks. Sneezing attacks have been described as an initial tabetic sign.

Sensory Involvement.—As a result of the implication of the meninges of the sensory roots, alterations in the sensory functions take place. The pathological process in some is so gradual or mild as not to give rise to pain, and in many, initial paresthesias may precede the pains, but, more often the same pathological process gives rise to both. Tingling, numbness, crawling sensations, flashes of hot

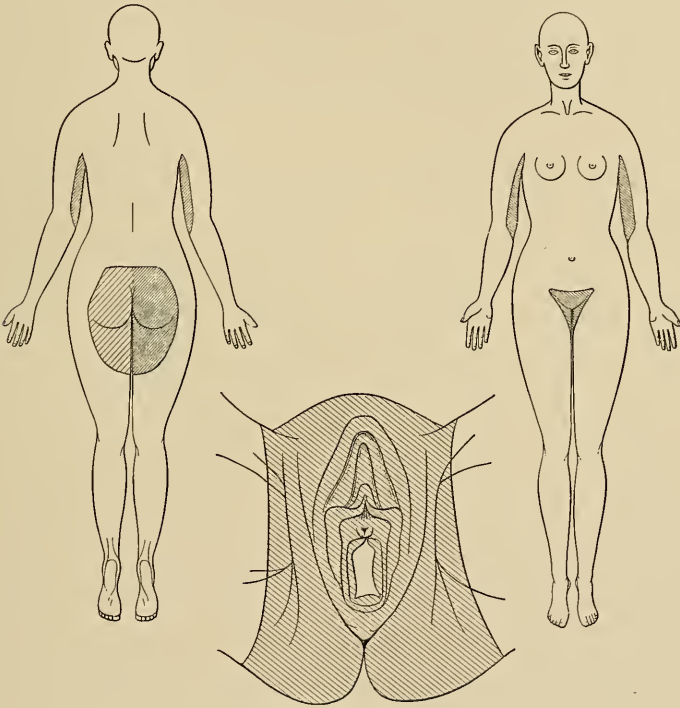
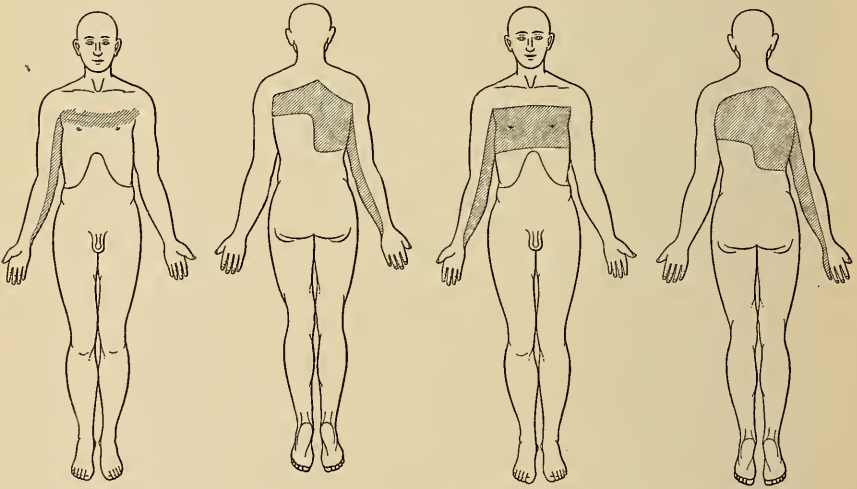


FIG. 282.—Tabes involving the cauda equina, showing the radicular distribution of the sensory disturbances. Tactile pain and thermal sensibility were involved. The patient first had pains and sphincter disturbances. The second dorsal root was also slightly involved, hence the sensory changes in the arms (Dejerine).

and cold, slight benumbing of the tactile sensibility, causing the sensation of wearing a glove, or walking upon a textile are the usual forms. They may be expected almost anywhere from the region of the trigeminus through any sensory cervical nerve branch to the tips of the toes, although the ulnar region seems a site of special predilection. Occasionally they cause the peculiar girdle-band sensation, at one time considered so characteristic of tabes. A whole limb may be involved, but under any condition the tendency for the sensory disturbances is to show a radicular distribution (Dejerine) (Figs. 282–286).

Increasing sensitiveness is also frequent, so that the patient dreads the cold, or draughts, or sudden shocks, or the clothing, not only on account of the abnormal sensitiveness, but because these may bring on the severer pains. Tactile anesthesia follows this and is a direct resultant of the degenerative process in the root area. These are also irregularly distributed.

Other sensory functions also become involved. The *pain sense* may become lost in irregular areas. Retardation in the carrying of pain impulses is present at times. There may also be numbing or loss of *heat* and *cold* sensibility. Bony sensibility as tested by the tuning-fork also may be absent and shows irregular distribution. In general, deep sensibility is more profoundly affected than epicritic sensibility.



FIGS. 283, 284, 285, and 286.—Radicular distribution of sensory disturbances in tabes; Figs. 283 and 284 represent the distribution of tactile anesthesia; Figs. 285 and 286 that of loss of pain and temperature sense (Dejerine).

Ataxia.—The most prominent sign of the sensory involvement just noted is seen in the gradually (sometimes suddenly) developing ataxia. The fibers conducting the impulses from the joints and the muscles to the chief organ for their coördination, the cerebellum, are degenerating, and there results an imperfect knowledge of the position of the joints and of the states of muscular tension necessary to the proper performance of motor functions. There results a hypotonus and swaying of the arms in the finger-nose test and finger-finger test, and of the legs in the knee-heel test, and in walking. The patients are partially or completely unaware of where their limbs may be, and are unable to control the same, save to a certain degree through other avenues, namely, the eyes. Hence with closed eyes all of these signs of ataxia are markedly increased. With this great uncertainty of

movement there is no muscular paralysis, and little loss of muscular strength, save as the patient generally becomes weaker.

Gait disturbances are a direct result of the ataxia, and are extremely characteristic. They were described by Schermer as early as 1819. In the early stages the patient notes a difficulty in going up and down stairs, or finds himself insecure on uneven surfaces. He stumbles and at times falls. At night he finds it more difficult to get about, and he soon notices that he must keep his eyes glued to his legs or the surface on which he is walking if he is to be able to control them. Later he must walk with a cane, and his legs are thrust somewhat wider apart, are thrown somewhat irregularly forward, and are then brought to the ground with a sharp stamp, the knee being stiffened or even bent slightly backward—overextended—at times so much so as to cause him to fall.

His step becomes quicker and more stamping, until he reaches a point where, unless he has trained himself to walk anew, he becomes bed-ridden. His manner of rising from a chair or sitting down soon becomes very characteristic. The ataxia in the upper extremities, which is usually less than that in the lower, affects the patient's writing, the buttoning of the clothes, his dressing, etc. His hands are constantly moving—one finger and then another is raised or lowered, or the wrist turned. Oppenheim has called particular attention to these spontaneous movements, which closely resemble a static ataxia, and are found throughout the body.

Other muscles naturally may be involved in the ataxia, particularly those of the face, mouth, tongue, larynx, pharynx in which case speech, singing, swallowing, etc., are affected. Many patients die of aspiration pneumonia through ataxia in the swallowing apparatus.

Romberg's sign is another result of the loss of position sense. It may be an early sign, but is more apt to develop later in the disease. Many patients without well-developed Romberg are unable to balance themselves on one foot, and further, a mild Romberg may be more readily demonstrated by having the patient bend slightly forward.

Tendon Reflexes.—Westphal first emphasized the importance of the diminution or loss of the tendon reflexes—notably of the knee-jerk and the Achilles reflex. These belong among the initial symptoms in the larger number of cases. The knee-jerk may be first diminished on one side, but demonstrable by the Jendrassik method, or lost, and this for years, perhaps, before the development of a complete Westphal phenomenon. The Achilles jerk is lost in a similar manner and not infrequently even before the loss of the knee-jerk (Babinski method). In the upper extremities the diminution or loss of the biceps and radial periosteal reflexes are likewise early and fairly constant phenomena, though less marked than the changes in the lower extremities.

These tendon-reflex changes are all referable to the degenerations in the root zones and sensory columns.

Cranial Nerve Involvement.—Any one or all of the cranial nerves may be implicated. Loss of smell is rare.

Pupillary Reflexes.—Here a striking phenomenon is observed. Patients with tabes—as with many other syphilitic affections of the medullary or midbrain region—show a diminution or loss of the pupillary light reflexes, without any loss of the reflex of convergence or of accommodation. This is the *Argyll-Robertson* phenomenon. It is present in over 60 per cent. of the cases, and may be present for many years without other symptoms. The pupils are apt to be at first irregular in size, and also not infrequently in shape. The light reaction is at first less prompt—usually in one eye before the other—later



FIG. 287.—Tabes. Third and sixth nerve palsy.

both eyes are involved. Myosis in marked degree is then apt to develop. Loss of the consensual light reflex is one of the earliest signs of this pupillary change (Weiler). The sympathetic dilatation of the pupils is also soon diminished or lost.

Optic nerve changes are frequent and may occur early. There is an irregular gray atrophy, with narrowing of the macular vessels and gradual narrowing of the visual field, first for colors and then blindness, partial or complete, with advanced optic atrophy.

Ocular Palsies.—These very frequently belong to the early symptomatology and are often fleeting. Persisting ocular palsies are more often encountered in the later stages of the disease. These palsies in tabes occur quite irregularly and show themselves as atonic ptoses,

irregular diplopias—from fourth or sixth or third nerve affections—and rarely show signs of a complete ophthalmoplegia, internal or external.

The *trigeminus* involvement may show not only neuritic pains as already noted, but also trophic disturbances of the cornea, herpes, irregular development of the bony parts, etc. The motor root may also be partly paralyzed.

Facial palsy occurs, but not often.

The *acoustic* is occasionally involved in both cochlear and vestibular branches, producing unilateral or bilateral deafness, or vestibular nystagmus, Ménière-like crises, etc. (Bonnier's syndrome).

The *vagus* and *accessory* nerves are frequently implicated, causing crises already noted, and also slowing of the pulse, dyspnea, tachypnea, etc. Taste is not infrequently lost. Bulbar palsies are frequent.

Visceral Symptoms.—The bladder is usually involved at some stage of the disorder. The crises have been referred to. Weakness of the bladder is the most usual occurrence. Cystitis usually develops and is a cause of general weakness. Incontinence is not infrequent. Constipation is also frequent, but fecal incontinence not common. Loss or increase of sexual desire is an early sign; impotence is common; continuous priapism unusual.



FIG. 289.—Tabetic arthropathy of the rib.



FIG. 288.—Tabes. Third nerve palsy.

is the joint of special predilection. The arthropathies usually develop with surprising rapidity—with edema and swelling but none of the

Trophic Symptoms.—These may involve any of the tissues of the body, but particularly skin and bones. *Arthropathies* are very frequent, usually occurring after the disorder is well advanced, not infrequently as an early symptom. The knee-joint

usual signs of a rheumatic joint; then new bone formations take place, with or without subluxation. Almost any joint may be affected, even the jaw.



FIG. 290.—Tabetic arthropathy of ribs.



FIG. 291.—Tabetic arthropathy in the wrist.

Fragility of the bones is a further complication. Perforating ulcers of the feet constitute another trophic disorder.



FIG. 292.—Tabetic arthropathy with destruction of the joint and hypotonus.



FIG. 293.—Charcot joint.

Muscular Atrophies.—An initial neuritis may give rise to an early muscular atrophy, but atrophies are not common.¹

¹ See Lapinsky. Arch. f. Psy., 40-42.

Blood Serum and Cerebrospinal Fluid.—The Wassermann reaction of the blood serum is positive in most of the cases of tabes. The cerebrospinal fluid is positive in from 60 to 80 per cent., and Plaut is of the opinion that it will be found to be more often so with improved technique. Nonne and Hauptmann, by using larger quantities of serum, have established this. It seems to be less often positive than in paresis—which, in view of the opinion held concerning the identity of the two disorders is food for reflection regarding the local reaction of the brain and cord proper.

There is a marked lymphocytosis of the fluid, which is usually found in the early stages of the disorder, and is an index of the meningitic syphilosis usually found.

Course and Variation.—Tabes is essentially a chronic disorder, usually mildly progressive, developing fully after many years. In a small number of cases it runs an acute course, causing death within a few years. In others it is stationary and in this sense gets well.

Duchenne (1858–1859), in his memorable description, made a division into three stages, which have been somewhat modified. The periods usually considered at the present time are as follows: (1) Prodromal or preataxic stage; (2) ataxic period; (3) paralytic period.

Such a division is of arbitrary value only; no two cases are exactly alike. The preataxic stage may last many years, or there may be none, paralysis and ataxia developing with extreme rapidity. The symptoms are so many, and the times of their appearance so variable, that a so-called typical course is the exception rather than the rule, yet a not unusual course is one extending over about ten years, with two to three years of pains, with or without crises, then the gradual development of the loss of knee-jerks, Argyll-Robertson pupils, gradually increasing difficulty in walking, worse in the dark, Romberg—then the patients are confined to bed, and then the stage of paralysis and atrophies.

Death results from the disease itself, or from complicating disorder—bulbar accidents causing pneumonia, laryngeal choking, cardiac syncope, kidney complications, very often; intercurrent disease such as tuberculosis, in large part, pneumonia, typhoid, and erysipelas in smaller percentages.

Prognosis is always most sinister. Stationary cases are known, but a question concerning diagnosis may be raised respecting the cases in the older literature. With the newer objective Wassermann and cytological symptoms it is to be seen whether such stationary cases exist. Since such are found as paretics, it is not improbable that the same will hold true for tabes.

The cases with severe bladder complications usually do badly. The duration of life has varied from six months to thirty and more years. The general average runs between ten to fifteen years.

Forms.—Certain types are worthy of special mention as forms, these are:

(1) *Juvenile Tabes*, like juvenile paresis, occurs in children from five to ten years of age, or in young adults apparently up to about twenty-five years. The higher age incidence is rare. The pupillary signs are early, and the bladder is soon involved. Ataxia and paralysis then develop. The objective serological and cytological changes are usually positive—the number of cases examined is as yet too small to permit wide generalizations, but syphilis of the parents is an essential.

(2) *Late Tabes*.—The appearance of the disorder after the age of fifty is rare, yet cases are recorded in which the initial signs have come on as late as seventy years. One case is on record of infection at twenty, tabes at fifty-nine. After all it is mostly a question of when infection takes place—since cases of infection at eighty or over are known, it is practically impossible to determine the tabes age limit. No noteworthy symptomatic variations are known in late tabes.

(3) *Rapid Tabes*.—Here the disease advances very rapidly, rendering the patient incapacitated in a few months, with death as a result of the paralysis. The paralysis and atrophies occupy the foreground in the picture, the ataxias being less prominent. The pupillary signs are present. Death may take place within six months.

(4) *Slow Cases*.—These are the more usual cases already described.

(5) *Stationary or Benign Cases*.—A particular type in which blindness comes on early and which follows a benign course was first described in 1881 by Benedikt. Dejerine and Martin (*Thèse de Berne*, 1890) called attention to the fact that it is rare to find a case of tabes beginning with blindness that advances to the second stage.

Furthermore, amaurotic tabetic cases seem to have fewer pains. The pathological features are the same as in other cases, save as to extension, and the explanation of this variation is difficult to find. A certain diminution in the general symptoms in amaurotic tabes has been observed.

Diagnosis.—Little difficulty exists after the development of the Argyll-Robertson pupil, lost knee-jerks, ataxias, and Romberg. Practically the only differential at this stage is a polyneuritic process, principally of alcoholic origin. Here the pupillary disturbances are less in evidence, but can occur, and if present there are usually more grave cerebral symptoms and a diagnosis of taboparesis is more in question. The serological and cytological results determines a diagnosis almost at once. Still the most difficult cases to differentiate are those of alcoholism complicated with syphilis. Where the alcoholism causes a pseudotabetic picture, and the syphilis gives its serological and cytological findings with slight meningitic affection as its only spinal or cerebral concomitant. These cases are diagnosed only with the greatest of difficulty.

Other toxic pseudotabes offer no difficulties—such as those due to ergot, diabetes, lead, pernicious anemia, and the infectious toxemias. Here the pupillary signs are absent for the most part, and the serum and spinal fluid normal.

Certain cerebellar atrophies and new growths cause symptoms closely resembling those of tabes, but the gait is more widely swaying, the individual movements have less ataxia, the absent knee-jerks, and Argyll-Robertson are wanting, and there are, moreover, to be found the definite signs of cerebellar involvement in the nystagmus and its alterations by the Barany vestibular tests. Normal serum or cord findings are to be expected.

Multiple sclerosis can occasionally cause a tabetic syndrome if a patch should involve the sensory neurons, but here the other symptoms, the nystagmus, the signs of spasticity, masked by the hypotonus, Babinski phenomena, etc., should afford the clue. Rare sclerotic patches in the pons and midbrain regions have caused unilateral Argyll-Robertson.

Syringomyelia occasionally develops with an initial tabetic picture, but soon the classical dissociation shows itself, and permits a diagnosis. The blood and spinal fluid findings are also to be reckoned with.

Hysteria as an astasia abasia occasionally causes difficulty, but here careful examination can exclude the entire tabetic symptomatology.

Pseudotabetic neuritides and eye signs, *i. e.*, irregular pupils, gastric crises, may occur in masked myxedema (hypothyroidism) of later years.

Pathology and Pathogenesis.—Both Ollivier d'Angiers and Cruveilhier gave descriptions of the general gross anatomy and both recognized the sclerosis of the posterior columns. Todd associated the sclerosis with the ataxia. Burdon and Luys (1861) called attention to the relation of the sclerosis of the posterior roots, and the posterior column sclerosis, since which time the development of knowledge concerning the pathological processes in tabes have been most actively studied, although be it said, without yet arriving at general uniformity.

Seen with the naked eye, the cord is usually markedly atrophied, hence the origin of the old term consumption (tabes); the posterior roots are atrophied, sometimes more marked in one region than another. The diminution in volume of the cord also varies in places, being more pronounced, as a rule, in the dorsal and sacrolumbar region than the cervical, and, as Cruveilhier noted, the atrophy preponderates markedly in the posterior columns. The pia and arachnoid are somewhat swollen but translucent with slight opalescences or cloudiness. Meningeal involvement is very frequent; a fact brought out more in recent years, and in strict conformity with the findings of the cellular contents in the cerebrospinal fluid.

Throughout the entire length of the cord one finds a graying discoloration, the margins are slightly sunken below the normal level, and the discolored areas are harder to the touch. The atrophy seems to cease with the sensory medullary nuclei—save in those cases where the cranial nerves are markedly affected when irregular atrophies are encountered in the upper sensory neurones.

Histologically the picture is fairly uniform. There is a mild inflammatory thickening of the pia and arachnoid with lymphocyte and plasma-cell infiltrates. This leptomeningitis varies considerably in its localization, and in its intensity, and the vessels are not infrequently involved. The whole process closely approaches that of a syphilitic meningitis, but is less intense, and is not accompanied by the presence of spirocheta.

The posterior roots are irregularly atrophied; the sacral and lumbar roots may show not a single unchanged fiber, whereas the cervical roots are less involved, save in the case of cervical tabes, where the reverse holds true.

The posterior columns show the most uniform lesions. These are greatly diminished in volume. There is a gradual degeneration, often, however, more intense on one side than the other. The atrophy is

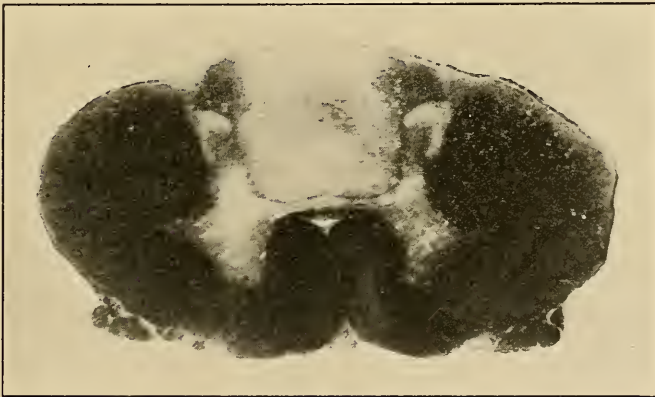


FIG. 294.—Tabetic changes in cord in paresis.

not universal, for many fibers are intact. These are usually collaterals, originating within the cord itself. The direct fibers in the columns of Goll and Burdach, from the degenerated posterior roots, are those chiefly involved. A great deal of variability exists as to the respective distribution of the atrophic degenerated fibers; but such bear a direct proportion to the distribution of the degenerated posterior roots. Certain portions of the cord escape, due to anatomical reasons; these are more particularly the commissural zones, Flechsig's centrum ovale, Gombault and Phillippe's triangle in the lumbosacral region; the cornucommissural zones, Schultze's comma tract and the fascicles of Hoche. (See Plate X.)

As a result of the disappearance of the fibers a secondary neuroglia infiltration takes place. This consists of glia with fine prolongations, and also spider cells. In the cases of taboparesis one finds lymphocytes and plasma cells within the cord substance. With a limited

tabetic process they are not usually encountered.¹ Many ganglion cells of the posterior horns show degenerative changes. They are diminished in size, there is definite chromatophilia, or vacuolization, and other signs of degeneration. The cells of the column of Clark are diseased only to a slight extent.

In tabes with cranial nerve signs the degeneration can be traced in the medulla and pons with secondary atrophy of the sensory nuclei. Atrophy of the optic nerve is not infrequent.

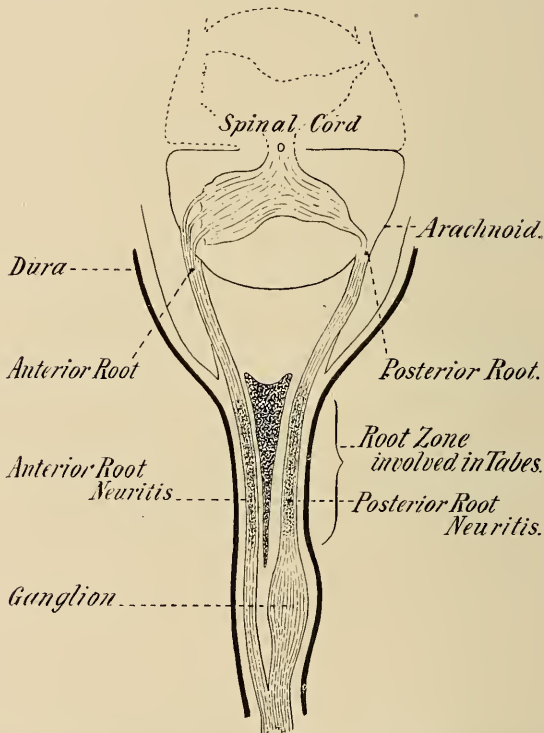


FIG. 295.—Nageotte's scheme representing the location of the chief lesion of tabes in the posterior root zone.

Jendrassik, Schaffer, Epstein and Krauss have also shown that the brain cortex is not uninvolved even in typical tabes. There is a diminution in the nerve fibers, and alterations in the vessels and pia—there may be lymphocyte and plasma-cell infiltration even in the absence of pronounced mental symptoms. Cerebellar degenerations are also known. The anterior horn cells do not always escape. In the patients with pronounced atrophy many motor cells of the anterior horns are found degenerated. Similar changes are known where there are bulbar, laryngeal or other cranial nerve palsies. The cerebrospinal

¹ Alzheimer, N. Arb., i, 14.

fluid also shows the effects of the mild inflammatory process by its increased lymphocytosis; a count of over 10 cells to the c.cm. (Fuchs and Rosenthal chamber) is to be regarded as positive, 50 to 100 are not unusual. Plasma cells are also to be found.

Serologically a positive Wassermann is to be expected, although here the number of positive results would seem to fall below that obtained in paresis. Possibly paresis indicates a more acute process.

Nonne phase I reaction is also frequently positive.

The posterior ganglia are also affected in many cases, although not uniformly. There is atrophy and destruction of the cells, and proliferative inflammatory exudates of the capsule. The changes are not sufficiently constant to permit one to assume that the primary part of the disorder is located in the posterior ganglia.

The peripheral nerves are also frequently found degenerated; Nonne says constantly; thus showing the complete degeneration of the sensory neuron, central as well as peripheral.

Sympathetic System.—This shares in the general destruction, and shows particularly in the regions involved by gastric or other visceral crises.

Pathogenesis.—Unanimity of opinion has not yet been reached. The various hypotheses evoked have upheld vascular (meningeal), medullary, radicular and the neuritic theories, not to mention the idea of a primary system intoxication. The general tendency is to regard as *fundamental* a primary syphilis involving particularly the posterior roots—a modified syphilitic radiculitis as the most constant feature in the pathogenesis. The process is one of chronic specific poisoning in which there is (1) an involvement of the posterior radicular fibers, and of the peripheral nerves, (2) an extension to the motor nerve system, and (3) to the sympathetic fibers.

Concerning the nature of this poison little is known. It shows certain analogies to an anaphylactic substance, but not sufficient enquiries have been pushed far enough along these lines to permit a working hypothesis.¹

SYPHILITIC MENINGOMYELITIS.

Nonne has devoted a large portion of his noted monograph to a consideration of the lesions of syphilis of the spinal cord and its membranes. This is a general indication of its extreme frequency, yet most patients showing syphilitic lesions of the cord also show signs in the brain or its meninges. They are nearly all examples of cerebrospinal syphilis. For practical purposes, however, it has been found of value to arbitrarily divide this large conglomeration and discuss it under two captions: cerebral syphilis, and spinal syphilis or meningomyelitis. This means simply that we are dealing with cerebrospinal

¹ See Head, Brain, 1913.

syphilis with predominant cerebral and minor spinal symptoms on the one hand, and with predominant spinal and nerve root, with less prominent cerebral signs on the other. It again seems advisable to accentuate the purely arbitrary nature of all such classifications.

Symptoms.—In considering meningomyelitis as a unit, we find cause for further emphasis upon separable symptom groups. Within this conglomeration again clinical neurology shows four fairly clear tendencies:

1. Syndromes due to pronounced meningeal implication.
2. Syndromes due to root and cauda equina disease. Radiculitis and neuritis.
3. Myelitic syndromes due to indiscriminate transverse disease.



FIG. 296.—Meningomyelitis. Radiculitis. Degeneration of Burdach's columns.

4. Syndromes of less extensive transverse lesions and fiber tract isolation. System syndromes.

A combination of all would make a complete meningomyelitic syndrome. This is a not unusual picture in a rapidly developing case; in its more chronic course the emphasis seems to be laid upon one or another of the just-mentioned groupings.

These are characteristic syndromes of the early and secondary stages of syphilis. The syndromes may develop within a few months after infection, or only come on after many years. In the former case the acute myelitic changes are frequent, also root lesions (many neuralgias, sciatica, etc.). The later developing cases show more the systemic lesions and gradually advancing meningopathies (later secondary meningitis) with compression (spastic) phenomena.

In all one expects to obtain a positive Wassermann; cerebrospinal fluid Wassermann is negative, save with large quantities of fluid; lymphocytosis is frequent—often the cell count being very high, always indicating the grade of meningeal involvement. The lymphocytes are not found before the stage of roseola; are abundant in the active secondary stages, and less frequent in the tertiary stages of a meningomyelitis. The protein content varies considerably.

1. *Meningeal Syndromes*.—Severe pains are signs of meningeal involvement. They shoot across the shoulder-blade, in the neck, across the hips, dart down the arms or legs and cause a stiffness of the neck, the shoulders and the thighs. The spinal column is usually sensitive to pressure, and to percussion, and local intensities may show both these signs, and also the peripheral signs of a definite zone localization.

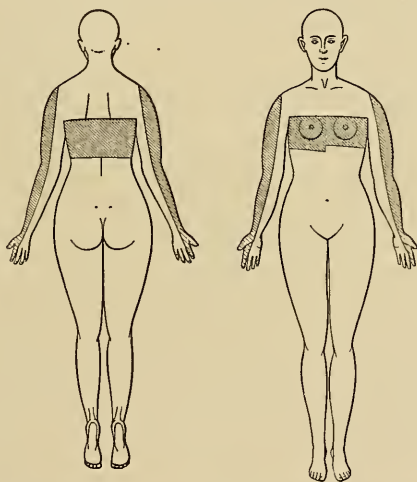


FIG. 297.—Radicular distribution of sensory loss in tabes, particularly to tactile pain and thermal sensibility. (Dejerine.)

Pain is frequently preceded by paresthesiæ, like the crawling of ants, numbness, and coldness. There is a gradual increase in the reflex excitability of the cord, due to pressure and evidenced by increased knee-jerks, possible Babinski sign, Oppenheim or Chaddock signs. When pressure is exerted in the sacral segments bladder and rectal disturbances are frequent.

With chronic meningeal thickening these pressure symptoms increase markedly, and spastic paretic phenomena augment, especially when gummata add their special pressures. Gummata may give rise to a "cord tumor" syndrome.

2. *Root Syndromes*.—Here pain is frequent and neuritic atrophies appear. Sensory losses of a root distribution are in evidence. Many show the characteristic reversal of epicritic touch loss being less exten-

sive than protopathic pain loss as pointed out by Head as pathognomonic of radicular lesions.

The atrophy of the muscles also follows the radicular distribution.

Many obstinate neuralgias are due to syphilitic radicular disease. Possibly one-half of the sciaticas are of this nature. Dejerine has put them as high as 80 per cent. in Paris. A very large proportion of the brachial neuralgias, so long looked upon as rheumatic or gouty, or what not, are due to a syphilitic, root meningitis.

Neuritic muscular atrophy, from pressure on the anterior roots, is further complicated by pressure on the anterior horns by the thickened meninges. Thus very anomalous atrophies result. When occurring in the eighth cervical and first dorsal region one obtains classical Klumpke paralysis with dilatation of the pupil, and narrowing of the palpebral fissure of the affected side. Lower localizations result in intercostal palsies, back muscle atrophies, hip girdle, thigh or cauda lesions. In this latter situation striking dissociations are obtained, as in the upper arm region, and radicular sensory disturbances and lost reflexes are the rule with atrophies.

3. *Myelitic Syndromes.*—These indicate the complete involvement of the cord, and also point to intraspinal vascular disease, rather than to a meningeal lesion. Complete flaccid palsy is the usual result. This is combined with sensory loss as well. The completeness of the sensory loss varies considerably, and indicates the severity of the lesion. Absence of a lymphocytosis points to a purely vascular, and usually focal lesion within the cord. The bladder and rectal functions are implicated as well.

In the regressive stage an increase in spasticity marks the subsidence of the inflammatory reaction, and many anomalous syndrome mixtures result. This phase of meningomyelitis offers abundant opportunity for very heterogenous syndromes. A Brown-Sequard complex, poliomyelitis, tabetic syndrome with atrophy, amyotrophic lateral sclerosis syndrome—these are but a few of the possible combinations.

4. *System Syndromes.*—These occur not so much as residuals of the previous myelitic changes, nor as due to meningeal compressions, but represent disease in or about the long motor tracts, tractus corticospinalis, especially. They give rise to the forms of primary lateral sclerosis (Erb), some combined scleroses, and particularly to clinical pictures closely resembling multiple sclerosis. Combined disease of the posterior and lateral columns is quite apt to be syphilitic.

Special localization of one or more of these forms of meningeal syphilis gives rise to the special forms of hypertrophic cervical pachymeningitis, which have been described by Joffroy and Charcot, and to Kahler's disease.

The anterior horns may be predominantly involved usually, however, as a result of a transverse myelitis (Nonne) giving the picture of an anterior poliomyelitis.

In *hypertrophic cervical pachymeningitis* one finds an enormous thickening of the meninges, with or without gummata, and located in the cervical region. Here root and compression symptoms are present. Pains in the neck and shoulder, stiffness of the cervical spine, shooting pains down the arms. Sensory loss may then show, particularly to pin prick, with relatively intact sensibility to cotton-wool. The ulnar and median are particularly implicated. Fibrillary contraction of the muscles, atrophy and loss of electrical excitability occur. The special type of deformity known as preacher-hand is one of the frequent expressions of the involvement of the brachial plexus in the cervical meningitis.



FIG. 298.—Pachymeningitis hypertrophica cervicalis.

CONGENITAL OR HEREDITARY SYPHILIS.

Effect of Hereditary Syphilis. Serological studies have thrown much light on the question of the mode of transmission. This cannot be entered upon here. These studies as particularly carried out by Plaut, Mott, and others have shown the enormous importance of transmitted syphilis in the pernicious effects upon the nervous system. Linser, moreover, has shown that two-thirds of the children of syphilitic parents show a positive Wassermann reaction, although much fewer show signs of congenital syphilis.

It may be recalled that Fournier stated the proportion as high as 98 per cent., and that 68.5 per cent. of the children died. This does

not include the aborted offspring. Should these be reckoned, one could obtain a true idea of the morbidity of syphilis in the young. Hochsinger reports an interesting group of cases in this connection. In 72 families there was paternal syphilis. The mothers were not syphilitic. Seventy mothers gave birth to 307 children—110 still-born, 166 syphilitic, and 31 healthy. The healthy were all the last born save in four instances.

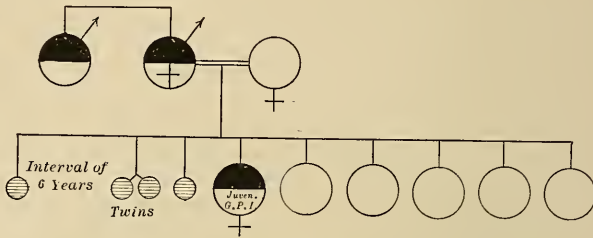


FIG. 299.—Congenital syphilis: Juvenile paresis, at first considered as "imbecile." (Mott.)

Of the children of tabetics, one obtains the same story from Mott, Mendel; and others. Either no children, many abortions, many dead children, few living, and no one knows as yet the fate of these. Certainly one-half are doomed to disease and disorder of the nervous system.

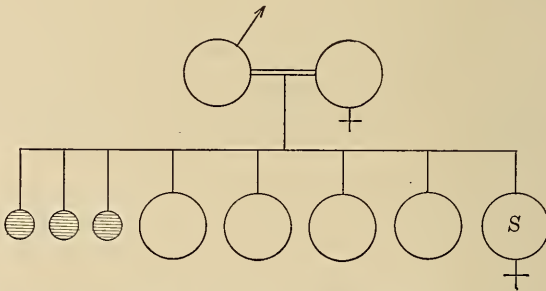


FIG. 300.—Congenital syphilis: Three miscarriages; then five children born alive and well. Last child snuffles, Hutchinson teeth. Did well in school, then deteriorated, noisy, maniacal; thought to be parietic. Autopsy showed generalized cerebrospinal gummatous meningitis, perivascularitis, and endarteritis. (Mott.)

It would appear that the common effect of such syphilitic infection is to reduce the resistances of the body and its powers for full development both in the general body and nervous tissues. Syphilis diminishes the vital energy of the germ plasm prior to conjugation, and can cause pathological variations in nervous structures, just as it can transmit the disease through the germ cells. The abundant studies on alcohol and its influence on the germ cell affords an analogy in understanding how this takes place with another type of toxemia.

The classical formula of Fournier seems to hold—abortion, dead child, early death, living, healthy child. This is in need of amendment; it is worse. The formula reads: Complete sterility, miscarriage, abortion, stillbirths, children dying in infancy or convulsions, marasmus, meningitis, hydrocephalus. Then follow children who are comparatively healthy, but who in later life develop late hereditary syphilis.

A study by Hochsinger (1911) says that of 208 children of syphilitic parents who had been under observation over four years, 89, or 43 per cent., had some disease of the nervous system. Of these there were 9 cases of hydrocephalus, 2 of Little's syndrome, 6 epileptics, 2

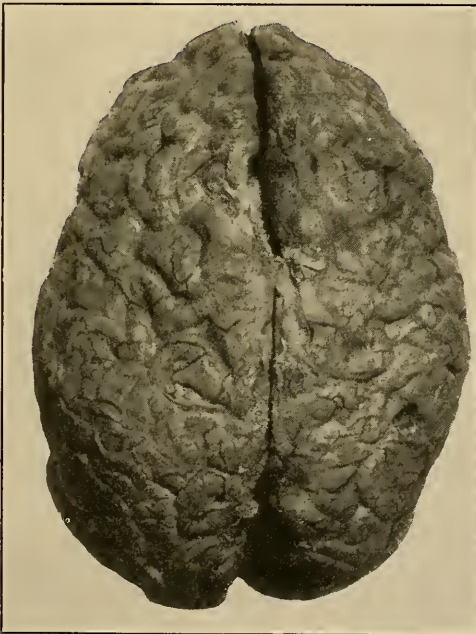


FIG. 301.—Brain of a congenital syphilitic idiotic child.

paralysis, 1 tabes, 6 Argyll-Robertson pupil, 36 extremely neurotic, 5 hysterical, 14 chronic headaches, 10 imbeciles. This is in strange contrast to the statements of Johnathan Hutchinson, who, but a few years ago, taught that nervous syphilis was negligible.

It has been abundantly shown that nearly every form of adult syphilis of the nervous system can be encountered in hereditary syphilis, and, as Mott well says, if congenital syphilis were not so fatal to infant life the number of people suffering from brain disease and syphilis would be appalling. It would therefore be of little service to repeat what has already been written regarding nervous syphilis of adults as it appears in children, and the present discussion

will be limited to a consideration of such forms of juvenile nervous syphilis as are present only in children. These are, particularly, certain forms of feeble-mindedness, of hydrocephalus, ependymitis, Freidreich's ataxia, primary optic atrophy, and encephalitides or encephalomalacias, leading to various hemiplegic syndromes, often loosely grouped together as Little's disease. Juvenile paresis and juvenile tabes are among the commonest congenital disorders of later infancy or adolescence.

Congenital syphilis of the nervous system may show itself before or at birth; it may develop in earliest infancy or in adolescence; it may develop as late as twenty, or even forty-two to forty-three years (Müller). How long may the virus remain latent, finally to develop a definite syphilitic reaction? As yet the answer will depend upon the general bias of the answerer, rather than upon empirical data. It would appear from the evidence at hand that no definite age limit can be put upon the time when such a latent factor becomes activated by causes as yet unknown. That an activator of some sort plays a role in such disorders as tabes and paresis seems at present a justifiable hypothesis. When a newborn child, known to have been syphilitic biologically, can be followed throughout life, his serum reactions being tested from year to year, as is now being done, then the question can be finally decided.

In this connection a line may be devoted to the subject of congenital syphilis "unto the third generation." While of late years doubts have been accumulating relative to this matter, certain positive cases are being reported. The rationale of this seems plain in view of the observations of recent years made by Levaditi, Bab, and others, that *Treponema pallidum* may be found in the ovum, and in an apparently resting stage similar to the resting stage known for other flagellate protozoa closely allied to the organism causing syphilis.

The fact of the whole matter is that opinions and statistics relative to congenital syphilis of the nervous system, supported by clinical observation alone, and uncontrolled by the available biological tests, are insufficient approximations, and very insecure. When relied upon for negative purposes they are harmful to the advance of thought, and detrimental to the relief of sick humanity.

Notwithstanding the importance, and often the strikingly gruesome character of these late-appearing congenital cases, the attention of the practitioner should be riveted upon the numerically preponderant miscarriages, stillbirths, and early syphilitic deaths if he would get in the right attitude toward the therapeutics of this disease, as it affects the nervous system.

In congenital syphilis, as has been stated, one finds a replica of what has been found in adult syphilis. Pathologically speaking, the lesions are nearly always combined. There is a variable composite of endarteritis, of leptomeningitis, of pachymeningitis, gummata, large and small, localized or infiltrating gummatous neuritis, diffuse degenerative

changes in the cells of the spinal cord, in the basal ganglia, or of the cortex. Thus the clinical pictures are apt to be conglomerate, and almost unanalyzable. Those more accentuated trends which permit a nosological term will be considered here.

Hydrocephalus.—As a result of congenital syphilis this condition has been suspected for two hundred years. Hasse, in 1828, Cruveilhier in his atlas, Van Rosen in 1862, and Virchow reported definite examples of it. It arises in these congenital forms largely from syphilitic disease of the cerebrospinal fluid-producing structures—choroid, ependyma, or from definite obstructive factors in the cerebral foramina, gummata, vascular swelling obstructing the iter, etc.

It is a not uncommon sequel of congenital syphilis and is undoubtedly more frequent than is realized. In Hochsinger's series of 362 cases of congenital syphilis 34, or nearly 10 per cent. showed hydrocephalus. In his series, which affords a fairly average review of the situation, the hydrocephalus began three to eleven months after birth; sometimes it was fetal. In 11 cases there were no nervous symptoms, *i. e.*, up to the time of reporting. In the others restlessness, sleeplessness, chronic vomiting, convulsions, contractures, nystagmus, and feeble-mindedness were the objective phenomena.

The more usual clinical picture is that of a child, boy or girl, from three to six months of age, who following, or not, an insignificant blow on the head, or some gastro-intestinal or bronchial disturbance, develops within a few days grave cerebral disturbances. There is great irritability and sleeplessness, screaming, and kicking. The head is usually drawn back, the eyes and the fontanelles are apt to bulge somewhat. Vomiting is frequent, and there are signs of oculomotor involvement. Internal strabismus from paresis of the external rectus is not unusual. It is frequently preceded by, or accompanied by, nystagmus and irregular pupils—often not responding to light.

Pain is present, as the child cries and struggles, and not infrequently the active movement of the arms—often highly spasmodic or convulsive—seem to try, in a blind reflex sort of way, to get at and brush away the source of it, *i. e.*, the head, pulling the hair, grasping and rolling the head. Spasticity, rigidity, and other signs of intracranial pressure may at times be demonstrated. As a rule the temperature is only slightly, or not at all raised, and the minor signs of an epidemic cerebrospinal meningitis, *i. e.*, herpes, temperature, flushed and spotted skin, are absent. The diagnosis of all of these infantile meningeal disturbances is fraught with much difficulty.

Feeble-mindedness.—Syphilis undoubtedly plays a much larger role in producing mental defectives than is suspected. The early statistics are comparatively worthless. They are quoted at great length even in modern works on idiocy, imbecility, and the like.

The more correct appreciation of this chapter on syphilis and feeble-mindedness began with the studies of Fournier on parasyphilis. Those truths, somewhat uncontrolled, were forced upon him by his

clinical observations. The early English, German, and American figures varied from 0.1 (Shuttleworth) to 17 per cent. (Ziehen). Whereas, the results following serological investigations start with the higher figures, and mount upward, in some cases as high as 40 per cent. The American figures available (Atwood and Clark) showed that 20 per cent. of the idiots, imbeciles, and morons at Randall's Island, New York, were syphilitic.

Intra-uterine feeble-mindedness is more or less a contradiction. These children who would date their mental defect to disease going on in the uterus rarely live. Plaut expresses the opinion that feeble-mindedness may be regarded, so far as syphilis is concerned, as the result of an extra-uterine syphilitic disease undergone in infancy. In some there are signs of an acute brain disease. Some instances of recovered hydrocephalus show the signs of havoc in their inability to develop normally. Many others show no acute stage, but fail to develop. Many recorded observations are available to show the very gradual development of mental defect, without convulsions or fever, altogether without signs of organic disease, which arrived at a definite termination, and which left behind entirely stationary, perhaps even improvable, idiots or imbeciles.

That type of hereditary syphilitic child without any tangible disease, formulated by Fournier—his "enfants arrieres"—who are described as unintelligent, simple, silly, limited children, always behind, not infrequently shows the Wassermann reaction.

Again, one is convinced by the researches of others that mental defect in less-marked grade, or more properly speaking, along more restricted or special lines, is allied with this broad group on the basis of congenital syphilis. Thus Nonne reports cases of general irritable weakness of the nervous system. The patients are highly excitable, are extremely nervous, they are very moody, suffer from headaches, irregularities of appetite, sudden fits of passion—not associated with other forms of epileptiform analogies—and for whom mercury and the iodides worked wonders.

Still another chapter has been opened in this hereditary syphilis problem in its relation to mental defect. It concerns many so-called psychopathic children. These children are bright, but they show marked ethical defects. Here one can conceive of the mental defect in terms of limited cortical control to the affective response of the sexuality, and to the nutritional instincts. These children want and take without going around by the circuitous routes devised by cultural standards. Anatomically one can posit a defect of certain corticocortical association areas on the basis of the syphilitic poisoning.

Taking the whole group of feeble-mindedness, it is evident that clinically one cannot pick out the hereditary syphilitic child in all instances. Indeed, it should be emphasized that too much weight is given to the anomalies in physical structure—Hutchinson teeth,

saddle nose, striae about the mouth, prominent veins, scaphoid scapula, etc.—if one rejects those who from the hereditosyphilitic class fail to show such anomalies. Nor can we recognize any certainly pathognomonic psychical anomalies. A careful neurological examination frequently aids in enlarging the group—particularly in the study of pupillary anomalies. The cytological tests are of the highest importance, and every child born of syphilitic father or mother should be systematically examined by these cytobiological methods.

Treatment.—The treatment of syphilis of the nervous system appearing in any of the forms previously enumerated, is often extremely satisfactory. In fact, at times one might say the results are often too good, as the speedy relief not infrequently leads the patient to forego further treatment, or causes him to pursue his course half-heartedly.

For more complete discussion consult *Modern Treatment of Nervous and Mental Diseases*, White and Jelliffe, Vol. II.

Cerebral syphilis for the most part is accompanied by active spirochetes; hence, the therapy is to be directed against this organism. For spirochetal poisons we possess mercury, arsenic, and iodine. Mercury and arsenic are active, iodine is very weakly toxic, but Neisser's most recent studies tend to show that it has actions other than that usually ascribed to it, namely, to promote the taking away of breaking-down syphilitic tissue or its product—its so-called resorption action. Neisser shows that iodides in large doses are toxic to spirochetes as well.

The treatment of nervous syphilis then should be an attempt to follow out a general antisiphilitic treatment, with special attention to certain structural peculiarities of the nervous system. These peculiarities are of much importance. In the first place, small lesions in the central nervous system, by impinging on important centers, bring about disastrous results, optic atrophy, pontine, medullary hemorrhages, etc. The enormous importance of correct nervous and mental functioning in the struggle for existence is self-evident. The difficulties in the way of repair in nervous tissues are enormous—at times insuperable.

Hence, one's attack upon nervous syphilis should be prompt and complete. A complete sterilization is desirable—and this is often extremely difficult to bring about in nervous tissues.

It must always be borne in mind that cerebral syphilis is often present with a negative Wassermann—this finding then should not deter one if there are clinical signs of diagnostic import. Not infrequently active antisiphilitic treatment (salvarsan) causes the appearance of a positive blood reaction—how often this occurs is not yet known.

Study of the cerebrospinal fluid is often a better guide to the correct appreciation of the situation as well as a reflection of the activity of the process. Lymphocytosis is often present months or years before

any definite nervous signs. Lumbar puncture is too often neglected in cerebral syphilis. Many authors state that if the blood is negative to Wassermann there is little occasion for studying the cerebrospinal fluid. This leads to bad results. Vascular and meningovascular processes may progress for years in nervous tissues without giving rise to a positive Wassermann. Here is an occasion in which this symptom fails to be present.

An energetic treatment should therefore be carried out if there are suggestive signs of nervous syphilis, even should there be a negative Wassermann.

In certain patients rapid action seems imperative; in others the need for this rapidity is not so much in evidence. Granted a knowledge of these requirements, the choice of remedies is not as simple as it might seem. Iodides given by the mouth in doses of 30 grains, 2 grams a day, show evidences of activity in about one week. Mercury by inunction shows results in about five days, while the newer arsenical preparations show reactive capacities in about forty-eight to seventy-two hours. None of these figures should be accepted as final, so far as curative action is concerned, nevertheless they are worth something. It is not apparent that iodides have a rapid toxic action; hence, in lesions which are characteristic of the more florid aspects of spirochetal growth—basal meningeal types of acute onset particularly—they should not be chosen in the initial attack.

Inunction Method.—For years neurologists have taught—chiefly under the influence of Erb—that nervous syphilis is best attacked by the inunction method—combined with iodides. In those situations in which the time element is of less moment this attitude seems justifiable, especially for gummatous types of the disease.

Oleate of Mercury.—Different possibilities are here presented. The oleate of mercury is of value in that it is comparatively cleanly and produces results as rapidly as other mercurial preparations applied to the skin. A dram of the 10 per cent. oleate is to be used night and morning for four days. The patient then takes a vapor bath and the same dose is used once a day for four days more. If sponginess and soreness of the gums do not appear—with cleaned teeth and gums—the double dose may be continued; otherwise a single dose should be utilized. In using the oleate one usually employs a small piece of flannel in the rubbing—the first dose should be larger as the flannel absorbs it, and the same piece of flannel should be used continuously.

The oleate may irritate the skin, but as it is absorbed fairly well from all parts of the body one can shift around more readily with it than with other mercurial ointments. Another object of using the oleate is on the ground of secrecy.

This line of treatment should continue at least six weeks, after the first week 10 grains (0.6 gram) of potassium iodide t. i. d., should be administered during the course of treatment. There is very little advantage in raising the amount of iodide above 60 grains (2 grams)

a day. After six or eight weeks the treatment should be discontinued absolutely—to be renewed not later than three months after the termination of the last treatment. A third and fourth course is advisable, even imperative if a positive Wassermann is present in the blood or lymphocytes above 10 to the c.mm. are obtained from the cerebrospinal fluid.

Unguentum Hydrargyri.—Unguentum Hydrargyri is much used and widely recommended. It has the disadvantage of being dirty and of attracting attention. The latter may be partly obviated by adding some non-stainable coloring matter, or some smelling compound such as balsam of Peru. Attention can thus be diverted from its characteristic color. The ointment is used in daily doses of from ʒj (4 grams) to ʒij (8 grams) best rubbed in in the evening in a fairly definite manner, and in places where the skin is more permeable. One uses the inner surface of the arm and forearm for the first rubbings, covering them with bandages, then the inside of the groin, then the popliteal space, then the abdomen and back. The fifth or sixth day the patient omits his rubbing—takes a Turkish bath and then starts over the same course. This course is kept up for thirty doses.

Other Details.—The care of the skin and of the mouth is naturally to be kept in mind. The blood Wassermann should be tested at the end of the period, and if strongly positive, or if spinal puncture shows active lymphocytosis, or if clinical signs seem slow in responding, the inunctions should be continued at least two or three weeks longer.

Checking up by Wassermann and lumbar puncture three to four months later, or any increase in clinical signs should determine a repetition of the treatment along identical or more strenuous lines.

Iodides, 30 grains daily, are to be given throughout the course of the inunctions. All medication should cease at the end of the cure, unless there are definite indications for its continuance.

Other mercurial inunction masses may be used. Those of value are the hydrargyri vasenol, vasogen, mitin, resorbin, which have special indications which may render them particularly valuable.

Injection Treatment.—Injection treatment attempts an even more rapid and energetic attack upon the spirochete. Many battles have been fought among syphilographers as to the comparative merits of the insoluble or soluble salts. When so much diversity of opinion can be found, it usually indicates that the real differences are usually minimal. Hence, ease of administration, safety, painlessness, etc., determine the choice of the remedy in each case.

Calomel, mercury salicylate, and thymol acetate are among the more favored insoluble salts. Calomel has occupied a high rank and can be utilized in the following forms:

℞—Hydrargyri chloridi mitis	5.0 gm.
Sodii chloridi	5.0 gm.
Aq. dest.	50.0 gm.
Mucilagio arabici	2.5 gm.
℞—Hydrargyri chloridi mitis.	
Ol. sesami, 10 per cent.	

Pravaz syringeful every second or third day, preferably into the muscles of the thigh or back, for 12 to 15 doses.

The hypodermic use of calomel is often accompanied by much pain. Abscess and necrosis is not uncommon, and lung emboli may occur—with care, however, calomel given by hypodermic is free from danger.

Creams of calomel, devised by Lambkin, have been extensively used, as they cause less pain and give rise to no complications if blood-vessels are avoided. These creams should be sterile. The formulæ of some in use are as follows:

R—Calomel	5 gm.
Creosote	20 gm.
Camphoric acid	20 gm.
Palmitin	100 gm.

Inject 10 m of this cream once a week for one month, to be replaced by the following:

R—Hydrargyri (metallic)	10 gm.
Creosote	20 gm.
Camphoric acid	20 gm.
Palmitin	100 gm.

This is injected in doses of 10 m twice a week for three weeks.

After six doses have been given, no more doses for two months. Four injections of the metallic cream are then given at fortnightly intervals. Then a rest for four months. Then four injections as before, and a rest for six months. Then a repetition of four fortnightly doses—an interval of one month, and a final series of four metallic cream doses.

English syphilographers have found these creams admirable in army and navy work. They are adapted for early stages better than for nervous syphilis, but are worthy of more extended trial in nerve syphilis.

The use of the insoluble salts has the advantage of a much more prolonged action of the mercury. They also have the disadvantage—all mercurial salts share in this, however—of irritation of the kidneys. If albumin is found before the use of mercury one should look for a syphilitic albuminuria. Tuberculosis, diabetes, alcoholism, marked cachexia are additional factors to be carefully dealt with. Gastro-intestinal disturbances are frequent, but it is extremely rare that mercury causes a neuritis.

Thorough cleansing of the mouth, and the use of chlorate of potash mouth wash is imperative.

The soluble salts in use are very numerous. They include the soziodolate, bichloride, lactate, succinamide, biniodin, benzonate, and cyanide. Fournier lists about 30. They may be injected within the muscles or into the skin, superficially. All are somewhat painful, and accidents are possible. In general the dosage is from $\frac{1}{4}$ to $\frac{1}{2}$ of a grain. The injections are given twice or three times a week.

General Scheme of Injection.—The following general scheme is suggested:

1. The site usually chosen is the posterior third of the buttock, to avoid the sciatic nerve and vessels.

2. The injection should be made deeply into the muscle, using each buttock alternately.

3. The syringe and piston should preferably be of glass, easily sterilized, and the needle of platinum iridium, about $1\frac{1}{2}$ inches in length, and sterilized.

4. The skin should be scrubbed with ether soap, washed with freshly boiled water, and swabbed over with an antiseptic solution.

5. After insertion of the needle, the piston should be slightly withdrawn, and if any blood appears the needle should be reinserted in order to avoid injection into a bloodvessel.

Solutions.—The injection of the solution free from air bubbles can then take place.

Various solutions are in use. Only a few can be mentioned here.

R—Hydrargyri sozoidolate	gm.	0.2	gr. iij
Sodii iodidi	gm.	0.3	gr. v
Aq. dest.	gm.	10.0	ʒiiss

Dose—10 to 20 minims constitute the dosage.

R—Hydrargyri lactatis	gm.	0.2	gr. iij
Aq. dest.	c.c.	18.0	ʒiv

Dose—10 to 25 minims.

R—Hydrargyri succinamidi	gm.	0.2	gr. iij
Aq. dest.	c.c.	10.0	ʒiiss

Dose—10 to 25 minims.

R—Hydrargyri chloridi corrosivum	gm.	0.5	gr. viij
Sodii chloridi	gm.	3.0	gr. xlv
Aq. dest.	gm.	100.0	ʒiij

Dose—1 to 2 c.c. daily or alternate days.

The use of corrosive sublimate—following Lewin—is usually very painful.

R—Hydrargyri cyanidi	gm.	1.0	gr. xv
Cocaine hydrochloridi	gm.	0.3	gr. v
Aq. dest.	ad	gm. 100.0	ʒiij

Dose—1 to 2 c.c.

A useful variant of this combines the cyanide with arsenic and strychnin, as follows:

R—Hydrargyri cyanidi,				
Strych. arsenatis	āā	gm.	0.6	āā gr. ix
Cocain muriat.		gm.	0.3	gr. v
Aq. dest.		gm.	60.0	ʒij

Dose—5 to 10 minims every other day for 20 to 25 doses.

Cocain may be added to any of the soluble salts. Its addition lessens the pain.

Fournier has always advocated the use of the biniodide dissolved either in sterilized oil or in water. It is, he claims, painless, sure, and free from dangers. In 2457 injections only 9 produced pain. Such results, however, obtain only when the physician is very careful. Careless use with the biniodides will produce all of the accidents, pain, abscess, etc.

℞—Hydrargyri biniodide	gm. 0.4	gr. vj
Olive oil (sterilized)	gm. 10.0	ʒiiss

Dose—One Pravaz syringeful every other day.

℞—Hydrargyri biniodide	gm. 0.2	gr. iij
Sodii iodide	gm. 0.2	gr. iij
Aq. dest.	gm. 10.0	ʒiiss

Dose—1 to 2 c.c. daily or on alternate days, with gradual elevation of the dose if stomatitis or gastro-intestinal signs are not in evidence. Twenty to twenty-five injections constitute a course of treatment.

℞—Hydrargyri benzoatis	gm. 1.0	gr. xv
Sodii chloridi	gm. 2.5	gr. xlv
Aq. dest.	gm. 120.0	ʒiv

Dose—1 to 2 c.c. daily or on alternate days.

Combined arsenical and mercurial injections were very much in vogue before the introduction of the salvarsan preparations. One of the most popular of these has been the arsenical salicylate or enesol. This remedy has been used widely in nervous syphilis and often with surprisingly good results. Schaffer speaks very highly of it.

The combined use of the cacodylates and of mercury has been observed to give good results. The early reports of optic nerve disease apparently following the use of the cacodylates served to force these salts into the background. Inasmuch as such optic nerve changes apparently occurred in other than syphilitic patients, it would not appear that they are to be interpreted as instances of those neurorecidives which have been so actively discussed since salvarsan has been introduced.

Salvarsan and Neosalvarsan.—Any attempt at an exhaustive summary of the various reports upon this remedy in the treatment of nervous syphilis would require a special volume. A simple enumeration of the bibliography alone—best obtained in brief in Lewandowsky's *Handbuch der Neurologie*, articles by Forster and Schaffer and others—in Nonne's discussion, referred to later, would require dozens of pages. Only the present (1915) drift of opinion will here be expressed.

In the exudative, hyperplastic, gummatous, and arterial forms salvarsan is by far the most efficient remedy that we possess at the present time. One form needs to be excepted, that of the large gummata, for which surgery alone is adequate. It would also appear that much larger doses of salvarsan are required for nervous syphilis

than were used in the earlier stages of its administration. Since the use of adequate dosage the so-called neurorecidives have almost entirely disappeared. It is the present trend of opinion that mercury and salvarsan combined gives the best results. Whether or not arsenic and mercury, both active spirochetal drugs, supplement each other in this combined use is not certain, but the results obtained have in many instances been very satisfactory.

At the same time it needs to be observed that a number of patients have relapsed, and the final results of therapy in nervous syphilis have been far from being as hopeful as had appeared. Because this has been so is no reason why it should remain so. The most obvious reason that stands out in many of the recent discussions that have taken place relative to this point is that the patients have been insufficiently treated. Finally, salvarsan has not been long enough in use to warrant anything but as yet tentative conclusions.

With this short summary of conclusion, a few words may be said as to its application and dosage. Whether neosalvarsan is to replace salvarsan or not cannot yet be determined. The intravenous application of salvarsan is the best method of giving it. It should not be given unless the patient is under some sort of supervision—in a hospital or remaining in bed—and minute attention to the technique is absolutely necessary to avoid certain dangers. It is highly important that fresh, distilled water be employed if salvarsan is to be used intravenously.

To kill spirochetes in the nervous system, however, is one thing, and to overcome the results of tissue changes is quite a different one, and this above all is the stumbling-block in the treatment of nervous tissue syphilis. Nevertheless, if nerve tissues have not been extensively destroyed, one can hope for excellent results by a proper combination of salvarsan therapy with mercury.

Salvarsan must be used in much larger quantities, however, than was at first thought. At the end of this section the outlines of an energetic combined therapy is given, and reference may be made to those pages for the general indications of such a course of treatment. Modifications to a less active mercurial salt than calomel may have to be made. Every patient needs individual treatment.

Notwithstanding the very evident fact that salvarsan and neosalvarsan are active spirocheticidal drugs, it is still an important problem how to reach them in the nervous system. Careful chemical investigation of the cerebrospinal fluid has heretofore failed to obtain any trace of arsenic when salvarsan has been given in the usual manner. One may infer that the arsenic has become fixed in some chemical combination which fails to react to the usual chemical tests. It is not yet comprehensible why nervous syphilis is so resistant to treatment and why the hopes aroused by the striking results of salvarsan therapy in general syphilis seem not to have been borne out in nervous syphilis.

Swift and Ellis have attempted to place a spirocheticidal solution directly into the cerebrospinal fluid. Salvarsan and neosalvarsan were employed by direct injection into the spinal canal through the Quincke lumbar puncture. This method they found was to be condemned. It failed to give any beneficial results, and, moreover, caused marked pains. It is probably a dangerous procedure, as animal experimentation has shown.

An attempt was then made to introduce into the patient's cerebrospinal fluid some of his own blood serum which had previously been mixed with the salvarsan by intravenous infusion. This procedure is carried out in the usual manner. After a certain length of time—one hour was found to give the most active serum—blood was withdrawn, separated from its corpuscles, after twenty-four hours, diluted with 40 per cent. of normal saline, and then heated to 56° C. for thirty minutes. A lumbar puncture is then made, and from 5 to 15 c.c. of cerebrospinal fluid is withdrawn, *i. e.*, until the pressure falls to 30 mm. of mercury. Thirty cubic centimeters of the warmed serum is then injected into the subarachnoid spaces. The patient must lie quiet, the foot of the bed usually being raised. After ten days to two weeks the injections, which are usually well borne are to be repeated.

They thus obtained very striking results in the action upon the serobiological factors known to accompany cerebral syphilis. Then a more crucial experiment was planned. This consists in the introduction into the subarachnoid spaces of serum taken from another individual, usually a secondary syphilitic under treatment. The technique being that just outlined. In the treatment of tabes by a heterologous serum most excellent results have been obtained in some cases. In others they have been *nil* or positively dangerous. Why the striking character of the results, for it is at once evident that the amount of spirocheticidal substance in a few cubic centimeters of serum taken from the body of another patient who had received the usual intravenous salvarsan therapy must be very small indeed, *i. e.*, reckoned as arsenic? If other factors than the salvarsan itself enter into the situation these are as yet unknown. One significant fact, however, would tend to indicate that other forces are operative. Extensive experiments carried on by Swift and Ellis with the heated and unheated serums show that the heated sera are three times as spirocheticidal to *Spirocheta duttonii* in mice.

This would raise the question whether or no the heating caused alterations in the salvarsanized serum which were advantageous in the therapy.

The most striking suggestive result is an almost immediate diminution in the number of pathological cells in the cerebrospinal fluid. The globulin reaction diminishes, positive Wassermanns of the cerebrospinal fluid with small quantities of fluid require larger quantities to show positive or become negative, and the amelioration of the symptoms has in a few cases been rapid.

Mercury by the Mouth.—Mercury by the mouth will always remain one of the simplest, and yet, at the same time, least efficient methods of treating syphilis of the nervous system. Here again one has a rich choice of remedies. Those most in use are: protoiodide grain $\frac{1}{8}$ to $\frac{1}{3}$, sublimate grain $\frac{1}{20}$, calomel 2 to 5 grains. Various vehicles are used.

The disadvantage of treatment by means of the intestinal canal are many—chief of which is the slow and weak action of the remedies employed. Moreover, the gastro-intestinal tract suffers.

The chief advantage is that mercury may be combined with the iodides. Furthermore, conveniences of medication must often constrain one to use this mode of giving antisyphilitic remedies, but only as a necessary choice.

In nervous syphilis it would appear that mercury medication by mouth is not radical enough. One may use it after an energetic treatment, by the methods outlined, has been employed, but oral administration is rarely a method of certain value, and hence is not advisable, save under particular circumstances.

Among the newer mercury preparations which future experience may prove to be of value are: Mercury dicarboxylate, two of which are on the market, with pronounced toxic action on spirochetes in rabbits. It is claimed to be twenty times as toxic to spirochetes as corrosive sublimate, and yet shows no action on the body. Its dosage has not yet been worked out.

Iodides.—Sodium and potassium iodide have been used in the treatment of syphilis of the nervous system for years, and often with good results. According to Neisser the iodides are weak spirochetal poisons. Our belief in its resorptive powers is justified on empirical, if not on pharmacological grounds. Personal experience does not confirm the belief in the efficiency of specially large doses, although that is the American preference.

The use of the iodides in doses of from 10 to 30 grains t. i. d. combined with mercury is particularly valuable in the gummatous type of cerebral syphilis. It is folly, however, to try to do away with large gummata by means of massive doses of iodides.

The dosage of the iodides will depend upon the individual. There are many idiosyncrasies to be borne in mind. At times small doses cause marked disturbances and cannot be borne. Here one may employ other combinations than those of sodium or potassium. Hence, strontium, rubidium, and organic iodine preparations have come into use. Iodopin, sajodin, iodoglidin, iodoval, iodocitin, iodostarin are among the newer of these combinations.

Iodopin may be injected as well as administered by the mouth. In the former case it is used in quantities of 10 c.c. on alternate days, or smaller doses 1 to 3 c.c. at more frequent intervals. In giving it by hypodermic both the syringe and the remedy should be slightly warmed, the needle should have an ample bore, and the drug be introduced slowly. It is also given by the mouth in 3j doses. In the form

of iodopin, large quantities of iodine may be introduced without toxic effect. Its action on nervous syphilis has not been extensively studied. Good results are reported by its use in syphilitic labyrinthitis.

Iodoval and iodocitin, the latter a lecithin albumin compound, have been found to be borne well in the course of salvarsan-mercurial treatment. The former is given in doses of about 5 grains t. i. d. throughout an energetic salvarsan-mercury treatment—the latter in about the same doses.

Plan of Intensive Treatment.—Nervous syphilis is treated too gingerly by most practitioners. It is difficult to kill the syphilis organism, hence an energetic course of treatment is here outlined:

First day	0.03 calomel (or other mercurial) hypodermically.
Third day	0.05 calomel (or other mercurial) hypodermically.
Fifth day	0.4 salvarsan intravenously.
Seventh day	0.5 salvarsan intravenously.
Ninth day	0.05 calomel hypodermically.
Eleventh day	0.05 calomel hypodermically.
Thirteenth day	0.4 salvarsan intravenously.
Fifteenth day	0.5 salvarsan intravenously.
Seventeenth day	0.05 calomel hypodermically.

This should be continued for a six weeks' cure, or until at least 5 gms. of salvarsan are administered. The whole cure can be compressed into three weeks if a soluble mercury salt is given, and at least 5 gms. of salvarsan can be administered in that time. The patient should be watched very carefully, especially with reference to the kidneys. Furthermore, there are patients who do not bear calomel well. Vagotonic individuals react excessively to mercury, especially to minute doses. (Compare article on Syphilis of the Nervous System in *Modern Treatment of Nervous and Mental Diseases*, White and Jelliffe, Vol. II.)

PART III.

PSYCHICAL OR SYMBOLIC SYSTEMS.

CHAPTER XVI.

THE PSYCHONEUROSES AND ACTUAL NEUROSES.

Introduction.—The field of the neuroses and the psychoneuroses is not only the broadest field in psychiatry, but perhaps the broadest field in all medicine. Not only is the field an extensive one in point of the actual number of persons who suffer from these afflictions, but it is a field of very great importance for the understanding of mental phenomena both in the realm of disease and in the realm of the healthy. It is in the manifestations of these disorders, which have been well termed borderland states, that one can find and can study the early departures from the normal, which, in much more aggravated form, appear in the psychoses.

Then again problems that lend themselves much more satisfactorily to therapeutic attack are to be found here. Thus we are dealing with conditions which, although they may represent practically any degree of departure from the average, in the main represent lesser or moderate degrees of departure from the normal which are, as a rule, capable of material alleviation, if not actual cure by therapeutic measures. When one considers the immense number of people who are affected by neuroses or psychoneuroses, the great amount of suffering that these diseases entail, the impaired efficiency in which they result, and then consider that they are, for the most part, susceptible of great improvement, if not actual cure by therapeutic endeavor, it will be seen that this department of medicine is not only the most attractive, but is one which perhaps offers most in the way of results.

The number of people actually afflicted with these conditions is difficult to estimate. The frank cases of the psychoneuroses and the actual neuroses are very numerous, as are also more or less larvated conditions, while on the other hand every specialist in medicine is dealing constantly with manifestations of these conditions as they appear upon the physical side. Perhaps these physical manifestations are best known to the gastro-enterologist, the gynecologist, and the genito-urinary surgeon, but the ophthalmologist, the laryngologist, the internist, and in fact every specialist has his share.

The wide distribution of the neuroses, but more particularly of the psychoneuroses, can best be understood if certain psychological considerations are first briefly gone into.

The baby in its mother's uterus has no desires; it has to do nothing for itself, not even to breathe; it rests quietly, far removed from sources of outside stimulation and irritation, every function being performed for it by the mother. After the baby is born this condition of affairs still continues, or at least an effort is made for it still to continue. The baby, to be sure, has to begin to breathe for itself, to eat for itself, to perform the functions of digestion and elimination for itself, but on the other hand, there stand about the army of the household, not satisfied to wait upon desire, but with every heartstring of emotion tense to forestall it. Everything possible in the way of the baby's needs is anticipated. He is waited upon hand and foot by all; he is, in the sense that every desire is satisfied, truly omnipotent.

As the days go by and development proceeds apace, as the sense organs become more acute, the muscular adjustments more refined, the baby's contact with the world becomes progressively and increasingly complex, and try as they will the loving attendants cannot forestall all of his desires, and there come times when food is not offered at the instant it is needed, when sleepiness overtakes the baby but he cannot woo it if he is in a bright and noisy street or on a clattering car far from his soft bed. And so there arises insidiously but necessarily the mental state of desire, things wished for because they are not had.

Still even in this stage of development the discrepancy between desire and attainment is not great. Attainment, in fact, is usually very near at hand, the hunger is not permitted to last long, the baby does not have to be kept awake by noises, except for a brief period, while in the matter of other desires, such for instance, as the desire to empty the bladder, that is indulged in forthwith without any further consideration of the matter. As development progresses, however, desires become more and more numerous, because the baby touches reality at more numerous points, and each one of these points offers a new possibility for a frustrated or delayed desire, while with such matters as emptying the bladder there soon steps into the situation the social repressions represented by the prohibitions of the mother.

Thus growing up in the life of the baby, beginning even in the earliest days, an ever-increasing discrepancy between desire and attainment takes place, and as the years go on it will be seen, without the necessity for further illustrations, that the amoral, egocentric baby must gradually take into consideration the world about him. He is forced to lay his conduct along certain lines which imply a putting off of the satisfaction of desire into an ever-receding future. Later in life, when he is hungry and wishes to eat, he can only satisfy this desire provided he has worked and earned the wherewithal to buy food, and if he en-

deavors to satisfy it otherwise by taking any food that may be at hand he offends the social usages and becomes a thief. If he wishes to empty his bladder he has to wait until he gets to an appropriate place; it cannot be done anywhere and at any time. He has to adjust himself to the requirements of society or run serious risks if he fails. As he becomes progressively more complex, as his desires become more and more difficult of fulfilment, as he demands more and more of the world, the individual finds that he has to put off fulfilment further and further into the future and be satisfied to struggle perhaps for years to attain some specific end.

Conflict is therefore at the very basis, the very root of mental life; the adjustment of the individual to the world of reality is by no means the passive molding by external forces, but the individual is constantly and actively, in his mind at least, reaching out and trying to mold the world to suit himself.

It is from this basal fact of conflict that there take origin two forms of thinking, an understanding of which is of great importance for the comprehension of the psychoneuroses, in fact for all behavior, sick or well. Thinking which is dominated by the reality motive, the thinking which is a conscious intentional effort at efficient relation with reality, is the thinking to which the word thinking is usually applied. But there is another kind of thinking, the thinking by phantasy formation, which is of great importance. In this form of thinking it is not the reality motive that dominates, but the pleasure-pain motive. The other horn of the conflict is here represented, and in moments of quiescence when the real world slips away from our vision and we settle back within ourselves, our thoughts flow without reference to this outside world, they come and go without critique on our part. We are dreaming, perhaps in sleep or perhaps in waking, and these fancies which come at these moments of rumination are all wish-fulfilling fancies controlled by the pleasure motive and represent the satisfaction of desires which are either put off or rendered incapable of fulfilment in the real world. These thoughts are not only the thoughts that dreams are made of, but the thoughts which the psychoneuroses are made of, and are therefore of immense importance for their understanding.

From the very first the immediate satisfaction of desire is frustrated, to be technical it is repressed and some other form of activity has to be substituted, for example in later life, to use our same illustration, instead of maintaining the immediate relationship between hunger and food, there is introduced another series of factors, represented by work and compensation for work in the shape of money, which money may be exchanged for food. And so, instead of the immediate relationship that maintains in infancy a more remote relationship is maintained, and the activities instead of going straight to their goal take a more or less circuitous and involved path. The original relationship therefore tends to be lost sight of, and the more involved

and complicated one takes its place. There are, therefore, gradually throughout the period of development, all sorts of desires being repressed which, thus put out of consciousness, are replaced by other forms of activity. The desires which belong to infancy and which thus are early repressed and substituted by other forms of activity, constitute the material out of which the unconscious is formed and the material from which come the activating moments for phantasy formation. The discrepancy between desire and fulfilment, then, is compensated in later life by the wish-fulfilling phantasies that have their origin in the repressed material of infancy and occupy the realm of the unconscious.

Between this realm of the unconscious, which contains relatively infantile material only, and the realm of the clearly conscious, there lies the realm of the so-called fore-conscious, which contains the material of recent experience, material which is quite easily made conscious. In other words, it is just out of mind and it is not difficult to bring back into the focus of attention when the individual so desires.

The region of the unconscious is of very great importance for an understanding of the psychoneuroses, because it represents the region of the deepest repressions, of the thoughts that are least like the present conscious thoughts, and which, therefore, when they break through into consciousness, produce symptoms that are so grotesque and strange appearing upon the surface and non-understandable, not only to the onlooker, but to the patient himself. It is therefore desirable to know somewhat of the nature of the unconscious and of its content. To do this certain features in the development of the child, particularly those, of course, that are at a later date repressed and substituted by other activities, will have to be described.

During the early infancy of the child the child's love is very naturally given out to the only people to all intents and purposes who constitute his milieu, namely the members of the immediate family, the father, the mother, the brother, sister, and perhaps nurse. This love, contrary to the usual way of thinking of it, is very definite in its direction, and from a very early date presents certain sexual characteristics. Of these sexual characteristics jealousy of a younger brother or sister who comes into the family and deflects a certain amount of affection which the child would otherwise enjoy is within the observation of most people, while the fact that the love of the child is given out to the members of the family, characteristically the parent of the opposite sex, is not a matter of such common observation, but a matter of great importance psychologically. As the child develops these loves are repressed and covered into that all inclusive amnesia for the infantile period, and when adulthood comes along and the child has grown to manhood or womanhood and finds its mate, the love which had before been spent upon the members of the family now finds its true object.

It is this infantile love for the members of the family that is the root

for so many of the incest phantasies of the psychoneuroses and the psychoses. This love for the parent of the opposite sex, for example, if it breaks through into the clear realm of consciousness becomes a horrid thing incompatible with the individual's peace of mind. Such things are quite common. For example, a patient marries a man who unfortunately presented a number of very close resemblances to her father. These resemblances served to stir into activity the unconscious love for the father, and she therefore, in her feelings toward her husband, is outraged beyond all endurance, for it is as if she were married to her father. Life with her husband is quite unendurable. She is constantly flying into passions, assaulting him, upbraiding him, etc.

If this psychology is the usual psychology, why is it that all people are not in danger from such sources? Perhaps they are to a limited degree, but it is necessary to bear certain things in mind to understand how the unconscious becomes mixed up, as it were, in the daily life of the individual, as in the case just cited. An individual with such an unconscious father complex will get along in life perhaps quite well until they meet some difficulty. The difficulty drives them back within themselves, it prevents the outward flow of interest into reality, makes them egocentric, introspective, they are unable to make an efficient reaction, and they therefore are driven back to phantasy formation where things come true and the difficulties are all removed. The reason why this driving back of the psychophysical energy within the individual under conditions of stress, the reason why this should stir up a particular complex, is because in the life of the individual there has been an undue fixation at that point in the course of development. This patient just cited had never been able to emancipate herself as she should have from the necessity for the loving care and tenderness and protection of the father and to go out into the world and, so to speak, stand upon her own feet, and when difficulties arose in her life and she was thrown back upon herself, she went back to that point at which there had been an infantile fixation.

From the few words of description of this patient's condition, who had symbolically married her father, it will be seen how important it becomes to know the content of the phantasies, and this is best determined by a study of the dreams, and without going into the principles of dream analysis, which are out of place here, it is well to remember that the neurosis or the psychoneurosis, like the dream, is not only a compromise between desire and fulfilment, but it is a wish-fulfilling mechanism that brings to pass the fulfilment both of the wish in the foreconscious, the wish with reference to the difficulty that caused the introversion in the first place, and also the wish in the unconscious, the wish at the fixation-point, which serves as a pull-back once the introversion has started. (See Psychoanalysis in Chapter II.)

Bearing these facts in mind it will be easy to understand that the child's first sexual feelings have reference to its own body, it is auto-

erotic; that next its sexual feelings are transferred upon those immediately about him, upon someone most like himself, therefore of the same sex (homosexual stage). It seeks, in other words, outside of itself, but still an object as much like itself as possible. And finally, the period of object love, when fulfilment is had in an entirely different individual and of a different sex (heterosexual stage).

In addition to the above facts the child not only passes through these various stages of psychosexual development mentioned, but in its earliest infantile state it is susceptible, theoretically at least, of deflection in any direction. So, for example, at the period when the love is given out to those in the immediate surroundings it not infrequently is given out to a member of the same sex; differences in sex are not appreciated in these early days and come only with later development. Other differences are equally indefinite. The erogenous zones of which the genital organs are only one, and the anus and the lips constitute the most important additional ones, are still more or less indefinite, and sexual erethism may be predominantly focalized in any one of them. And so the roots of the various so-called perversions are found in these early fixations. The determining factor in the early fixations, the mechanisms that have brought them about, in short, their uncovering, can only be accomplished by fathoming the unconscious. This is the work of psychoanalysis and the most prominent means at its disposal is by the analysis of dreams. (See Chapter II on Mental Examination.)

THE PSYCHONEUROSES.

Hysteria.—Historical.—To write the history of hysteria would mean practically to write the history of medicine, for hysteria stands throughout the ages as the type of functional disturbance of the nervous system which, protean in its manifestations, is found associated with all great therapeutic movements in medicine. Whether it be the therapeutics of religious conversion, of Perkin's tractors, or hypnotism, or more recently of persuasion, a considerable proportion of the patients who recover and thereby become largely responsible for the vogue of the particular therapeutic measure involved, belong to the great clinical group of hysteria.

Hysterical manifestations have been prominent in mental epidemics that have swept over whole continents, while the more specific and the more grotesque symptoms have always been observed and described.

The modern period in the history of hysteria might be said to have begun with Charcot. This period is still so recent as to be within the memory of many, and the influence which the Charcot school exerted is still all too dominant in certain quarters. The picture of hysteria as Charcot drew it, particularly of the *grande hystérie* with its regular march of histrionic attitudinizing, as set forth in the world-renowned pictures of Richer, is familiar.

For many years following Charcot the most brilliant work in elucidating the hysteria problem was done in France, and many illustrious names are crowded into a few years. All sorts of explanations were formulated, theories that were physiological, that were psychological, and that were biological, with numerous variants of each. The most illuminating worker in this field for many years, the one whose theories produced the greatest influence in the study of this disease was Dr. Pierre Janet of Paris. His was a theory of dissociation, and he believed hysteria to be purely a mental malady. It was due to a poor synthesis of the personality which enabled certain groups of ideas to drop away from effective association with the main portion of the personality and occupy a region which Janet termed the subconscious, and there existing more or less independently, produce their results irrespective of corrections from the rest of the personality. The hysterical manifestations, then, were the manifestations of these split-off parts of the personality. Janet's views were a great advance upon the current concepts of hysteria, but although they rendered possible a deeper insight into the nature of the disease and the disease processes, they were still largely descriptive, though, of course, the description was much refined from that of Charcot.

Various kinds of dissociation theories have been built up by investigators since Janet, and the dissociation theory was variously elaborated, particularly in this country by Sidis, White, Prince and others. It remained, however, for a Viennese physician, Sigmund Freud, to get beyond the point of description into a true interpretative attitude toward the disease.

Freud showed that the reason for the dissociation was that the dissociated ideas were out of harmony with the rest of the personality, that they represented ideas that were in conflict with the ideas forming the consciousness of the individual, and that they were therefore repressed. *Repression* became with Freud, then, the fundamental factor at the basis of hysterical manifestations, an active, not a passive, mental factor which tended to put out of mind certain unacceptable groups of ideas, and was therefore the cause of the dissociation.

The Mechanism of Hysteria.—Starting with dissociation as the most fundamental descriptive term applicable to the hysterical state—the doubling of the personality, in the sense of Janet—it has been seen that there is at the basis of this process of dissociation an active process called repression, which has as its function the splitting-off of unacceptable idea constellations—complexes—from the main body of the personality, and thus, so to speak, putting them out of mind. It has also been intimated that these split-off complexes, because of being split off, do not therefore cease to act. As a matter of fact they go on functioning, but the functioning is independent, more or less, of the balance of the personality.

This process of repression and dissociation, following upon conflict, is a very general one and is found in divers mental states and is in

fact a normal process. It is not these processes or mechanisms which are characteristic of any particular mental disorder, but it is the *way in which the split-off complexes manifest themselves* that produces the different types of mental disorders.

From what has been said it will be seen that if the individual, or more specially, the psyche, be considered as being a complex of adaptive mechanisms which is always making an effort to come into closer adaptation with the environment, then the meaning of a conflict is that there enters into this mechanism certain factors to which it cannot make efficient adaptation. This results in repression and splitting, but the whole tendency of the machine is to readjust effectively by bringing about in some way a new state of affairs. In the conflict there are two groups of tendencies in the psyche which are diametrically opposed one to the other. No solution of the conflict can possibly be brought about by a fulfilment of one of these groups, because manifestly the conflict would still remain. Therefore any readjustment that takes place must in some way bring to pass the tendencies of both groups. Inasmuch as these groups are opposed to each other, such a result cannot actually be brought to pass in the world of reality. Therefore an artificial world which is not governed by the strict laws of cause and effect has to be brought into existence wherein these opposing forces can both, as it were, allegorically find their ends attained. This is well shown in the following dream: The patient said "she saw herself dead, lying in a coffin, with a red rose in her hand." The red rose symbolized her sweetheart because of the frequent presents of red roses which he had made to her. Being dead in a coffin probably has several meanings, but among others has the meaning of a regression. The coffin is the matrix, it symbolizes a going back to the protection of the mother, and so the dream symbolizes the two opposing desires, one infantile, the other adult and recent.

In the different mental disorders this end is brought about in different ways. The hysterical mechanism is different from the other mechanisms inasmuch as while it is a general rule that the painful affect of the split-off complexes is drafted off by various channels and thus finds expression, and while it is a general rule that this expression is not consciously associated with the idea content of the complexes themselves so that the patient is saved from a realization of their true nature, is thus conserved from an appreciation of the pain that would result if they were understood at their true value, in hysteria the painful affect is drafted off into bodily innervation, thus producing the somatic phenomena of hysteria. This is the process of *conversion* and is characteristic of hysteria. The so-to-speak strangulated, unreacted-to emotion of the split-off complexes manifests itself as the physical symptoms of the psychoneurosis and in this way the strong affect of the split-off complex is weakened. The complex is robbed of its affect, which is the real object of conversion and hence its value to the individual.

Symptoms.—The symptomatology of hysteria is naturally a very complex one, but from what has been said it will be seen that it tends to group itself more especially about disturbances of motion and of sensation. In addition to this it also tends to manifest itself in certain crises.

All forms of paralyses and anesthetics may manifest themselves. Paralyses of the limbs, either singly or hemiplegia with or without contracture, are common, while anesthetics may be distributed in almost any way, involving the superficies or the special senses. As a rule, of course, the distribution of these various phenomena do not follow the anatomical areas of nerve supply. They show some symbolic grouping.

The *disturbance of sensibility* are of many forms. Very characteristic are the glove and stocking anesthetics, involving the extremities of the limbs, hands, a lower part of forearm and feet, and lower portion of legs. Patches of anesthesia may be found upon any portion of the cutaneous surface and they may be widely distributed and often not constant in location but varying with different examinations. Hemianesthesia, especially of the left side of the body, crossed and alternating forms are found. Light touch is more often involved, frequently deep pain also, while insensitiveness to heat and cold also occurs.

A characteristic form of anesthesia which is very frequently found is concentric limitation of the field of vision.

These anesthetics do not follow anatomical areas and experiments will readily determine that they are psychological. If for example an anesthetic area is stimulated, the patient will say he feels nothing, but if asked to guess the nature of the stimulus, will show a surprising number of correct replies.

Hypo-esthesias, hyperesthesias, various forms of neuralgia, especially visceral, and headache are also frequently hysterical.

The *disturbances of motility* are largely various forms of paralysis. Hemiplegia, monoplegia and paraplegia, with or without contractures, are the commoner forms. Astasia abasia is a characteristic hysterical condition.

Spasms of various parts are not infrequent. Spasms of the tongue, of the face and of the extremities when not demonstrably of organic origin are hysterical as a rule. Tremors, myasthenic types of reaction, and easy fatigability are frequent. Choreiform movements, tics, and certain occupation spasms are often hysterical.

The speech is involved frequently. Hysterical aphonia is well known and usually a diagnosis is warranted if the patient can only whisper replies to questions and an examination discloses healthy vocal cords. Stuttering is frequently hysterical and an analysis will show that the words with which there is difficulty have especial significance for the patient. Other respiratory disturbances of an asthmatic character may also be hysterical.

Visceral disturbances, especially of the gastro-intestinal tract, many of the false gastropathies with gastric crisis of vomiting and diarrhea, are quite frequent. There may also be hysterical attacks simulating renal or hepatic colic, gastric ulcer, etc.

Vasomotor disturbances, localized edemas, disturbed reflexes, fever, secretory and trophic disorders have all been described.

Symptoms which cannot be accounted for on anatomical and pathological grounds should always lead to an analytic examination of the psyche. Even marked disturbances may have originated in the psychic and continued so long as to produce organic changes, as for example, muscular atrophy from prolonged disuse of a limb, the paralysis of which was of psychogenic origin.

Among the episodic phenomena are found disturbances of emotion, either exaltation or depression, which can be understood because of the displacement of the affect. There are various types of delirium which may or may not be associated with convulsive seizures, producing, especially, when long drawn out, the so-called somnambulisms, during which all sorts of ideas may be manifested and the patient be quite disoriented. Dream states not infrequently occupy the field and lead by development to all sorts and degrees of double personality, which is simply a more elaborate expression of the split-off complexes, indicating that they form a relatively large part of the personality. In fact, these split-off systems, provided recovery is not possible, tend to gather to themselves more and more of the personality and thereby to lead a more and more independent and broader existence.

Amnesias of course are frequent in the symptomatology. Any portion of the personality which is active may be amnesic for any portion of the personality which is opposed to it in the conflict.

In the analysis of hysterical symptoms one finds it relatively easier to account for them logically. While the symptoms, on the surface, often resemble dementia precox, one is not so often brought face to face with the crude outcropping of the unconscious. It is more frequently found that the symptoms lead directly back to actual situations, as in the case of¹ Lucy R., published by Breuer and Freud. This patient was disturbed by a subjective sensation of smell, which was traced back to a smell of burning pastry in a perfectly well-recalled scene where the children had forgotten the pastry and it had become burnt. Why the smell of burning pastry should be chosen for hysterical conversion was again traced to the young woman's love for the children for whom she was governess and the repressed wish that she might take the mother's place as the result of her love for her master. And in the case of Freud's of² Elizabeth, who, while engaged in nursing her sick father spends one evening away from home at the solicitation

¹ Freud, S., Selected Papers on Hysteria and Other Psychoneuroses, Nervous and Mental Disease Monograph Series, No. 4.

² Loc. cit.

of her family. Upon this occasion she meets a young man and on her walk home with him gives herself up to the happiness of the situation. On the return, however, finding her father much worse, she bitterly reproaches herself for forgetting him in her own pleasure. This thought, however, is repressed. In the course of her caretaking she had each morning to change the dressings on her father's swollen leg. To do this she took his leg upon her right thigh. The suppressed complex seized upon the feeling of weight and pain of her father's leg upon her thigh as an efficient avenue of expression for her repressed wish which thus comes into consciousness under the disguise of a painful area on the right thigh corresponding in extent and location to the place upon which the father's leg rested.

From these examples it will be seen that the hysteric is the victim of the spontaneous and aberrant activity of repressed and split-off complexes that have to do with past events in the patient's life and that the expression of these complexes produces the symptoms of the psychoneurosis, and that so far as the hysterical manifestations are concerned the hysteric may be said to live in the past, for each access of symptoms is but a reanimation of past experiences.

Like all psychoneurotics the hysteric is infantile. In other words, there is a certain defect in psychosexual development, and the difficulties which they meet in life tend to drive them back upon themselves, to cause an introversion of the libido, that is, to remove their interest from the actual world of reality and to center it back again in themselves. As already explained, this introversion process tends to reanimate progressively lower psychosexual levels, and with a patient who is already infantile the tendency to reanimate, for example, the autoerotic level is easily manifest. This is well shown in certain symbolic masturbatory acts which recur during the hysterical seizures of which probably involuntary micturition is one.

The symptomatology of hysteria is then the symptomatology of the activity of the split-off dissociated idea constellations or complexes and their manifestation by the mechanism of conversion thereby producing symptoms of physical disorder.

These split-off complexes tend always to become dynamic and manifest themselves episodically in the hysterical seizures. The process of dissociation or splitting, once begun, tends to continue and new material tends constantly to be added to these split-off elements by further cleavage, and thus this new portion of the personality continues to grow at the expense of the total personality. Energy accumulates in these split-off systems, and when it becomes sufficient in amount it breaks through, so to speak, and produces the attacks. These attacks are made up characteristically of a living over again of those experiences which constituted the etiological moments of the psychoneurosis. In hysteria, as has been pointed out, the breaking through of the energy from the split-off complexes manifests itself in bodily innervation—the symptoms of the disease are physical.

Aside from these episodic manifestations, the crises or paroxysms of the disease, there are the so-called interparoxysmal symptoms, which, harking back to a middle age demonology are still termed stigmata. These are most characteristically various anesthetics, anesthetics which are rarely complained of by the patient, often entirely unknown to him, being only brought out upon examination. It is because of this latter fact that Babinski has been led into the error of supposing that they were entirely the result of the examination, a position the erroneousness of which one can demonstrate to one's own satisfaction. Even though it were absolutely true, the fundamental fact, the why of the symptoms, the reason for certain patients reacting in such a way to an examination, remains unexplained by this renowned French neurologist.

An analysis of the stigmata shows also, and usually without much difficulty, a logical connection with preceding experiences, as for example, the smell in the case of Lucy R., or the anesthesia of the thigh in the case of Elizabeth, already cited.

There is another group of symptoms which follow of necessity as a result of the splitting of the personality. It can be easily seen from this dynamic conception of the nature of the disease that a person who is not at one with himself has not at any one time the full quota of his energies available, and therefore it is found that the general efficiency of this class of patients, particularly in the psychic sphere, is very greatly reduced; it is reduced in proportion to the amount of the personality which is represented by these split-off complexes. These patients, therefore, are not equal to the tasks they once could do. They are nervous and irritable, they tire easily, they lack capacity for consecutive application, and forget readily. These are general symptoms of the condition, secondary symptoms, the result of any splitting, and which are added to the primary symptoms which are expressions of the actual conflict.

A connecting link between the two portions of the personality is seen in the hysterical phantasies and also, of course, in the dreams which themselves belong to the realm of phantasy formation, phantasies which are thoughts that come without being bidden at moments of mental abstraction, thoughts that flow along without volitional choice, that replace one another without the exercise of critique, in other words, day-dreaming or night-dreaming as the case may be. These phantasies represent the activity of the submerged complexes as they break through and manifest themselves in the upper consciousness. They are of great importance in discovering the nature of the conflict and are very common features of the hysteric, although usually the patient does not realize it until his attention is addressed to these vagrant mental manifestations, because they not only come unbidden, but when they go they leave no tell-tale traces in the conscious memory. A further connection between the unconscious and conscious are the conscious phantasies. These are phantasy forma-

tions which apparently lie in clear consciousness and are not repressed. Phantasies of this sort are permitted in clear consciousness only because they are not understood at their true value. They really represent chiefly repressed material.

To resume, hysteria is the result of a splitting of the personality in which certain split-off complexes are sexually determined, and leading an existence more or less independent of the total personality express themselves by the mechanism of conversion in bodily innervation. The hysterical symptoms, then, become the representation through conversion of the unconscious phantasies which originate in the repressed complexes, while the structure of the hysterical attack is in every way similar to that of a dream. The attack is the breaking through of the energy of the repressed systems and manifests itself by a wish-fulfilling delirium, the elements of which may be over-determined, displaced, and inverted for purposes of disguise, as are the elements of a dream.

Compulsion Neurosis.—Compulsion neurosis contains probably the majority of that complex group to which Janet gave the name of psychasthenia. Janet's group, however, contained not only the compulsion neuroses, but a number of other things, particularly the anxiety neuroses, probably many anxiety hysterias, perhaps some neurasthenias, schizophrenias, and hysterias.

The characteristics of the compulsion neurosis are the presence in the mind of certain compulsive tendencies to act or think in various ways. The patient is forced against his will and without apparent reason to think certain ideas or certain thoughts or to do certain things. The compulsion increases until it is yielded to, then a period of calm follows which may be of variable length, until, so to speak, the energy has again accumulated, when the compulsion again manifests itself and must again be relieved by yielding. The patient has perfect insight into the matter, knows the whole business is foolish, but he cannot help it.

Mechanism of Compulsion Neurosis.—In hysteria the repressed material manifests itself by conversion. The accumulated affect of the split-off complexes is drained off through bodily innervation. The high affect-laden complexes are thus deprived of their emotion. The hysterical attack is a wish-fulfilling delirium which brings to pass in a sort of allegorical dramatization the fulfilment of both elements in the conflict.

In contradistinction to these characteristics of hysteria, in the compulsion neurosis, there is no conversion. The affect of the repressed complexes is drained off, not through bodily innervation, but by attachment to otherwise indifferent ideas. The affect is displaced. This displacement, quite as in conversion of hysteria, is a distortion mechanism and serves equally with it to disguise from the patient the real source of the affect.

Then again, while in the hysterical attack both elements in the conflict come to contemporaneous fulfilment, such unification through the symptoms is less evident in the compulsion neurosis, although the attempt is made to bring it about. What occurs on the surface, at least, is a constant alternation between the ascendancy of the two factors in the conflict, which two factors in their ultimate analysis resolve themselves, perhaps always, into love and hate.

The compulsion neurosis is a true defense neurosis and its symptoms, at least the compulsive acts, which develop late in the course of the disorder are of the nature of ceremonials, which not only serve to disguise the true situation from the patient, but so to speak, atone for evil.

Freud has very well said that it is much more correct to speak of obsessive thinking than of obsessive ideas. It is the obsessive element, the compulsion, the so-called *Zwang* of the Germans that is the essential thing in this neurosis, and which may express itself in all possible ways.

The mechanism of the production of the symptoms of the compulsion neurosis is a quite complicated one. It cannot be expressed better than by quoting the language of Freud.¹

"Sexual experiences of early childhood have the same significance in the etiology of the compulsion neurosis as in hysteria, still we no longer deal here with sexual passivity but with pleasurable accomplished aggressions, and with pleasurable experienced participation in sexual acts, that is, we deal here with sexual activity. It is due to this difference in the etiological relations that the masculine sex seems to be preferred in the compulsion neurosis.

"In all my cases of compulsion neurosis I have found besides a subsoil of hysterical symptoms which could be traced to a pleasurable action of sexual passivity from a precedent scene. I presume that this coincidence is a lawful one and that premature sexual aggression always presupposes an experience of seduction. But I am unable to present as yet a complete description of the etiology of the compulsion neurosis. I only believe that the final determination as to whether a hysteria or compulsion neurosis should originate on the basis of infantile traumas depends on the temporal relation of the development of the libido.

"The essence of the compulsion neurosis may be expressed in the following simple formula: Obsessions are always transformed *reproaches* returning from the repression which always refer to a pleasurable accomplished sexual action of childhood. In order to elucidate this sentence it will be necessary to describe the typical course of compulsion neurosis.

"In a first period—period of childish immorality—the events containing the seeds of the later neurosis take place. In the earliest childhood there appear at first the experiences of sexual seduction which

¹ Hitschmann, Freud's Theories of the Neuroses, Nervous and Mental Disease Monograph Series, New York.

later makes the repression possible, and this is followed by the actions of sexual aggressions against the other sex which later manifest themselves as actions of reproach.

"This period is brought to an end by the appearance of the—often self-ripened—sexual 'maturity.' A reproach then attaches itself to the memory of that pleasurable action, and the connection with the initial experience of passivity makes it possible—often only after conscious and recollected effort—to repress it and replace it by a primary symptom of defense. The third period, that of apparent healthiness but really of successful defense, begins with the symptoms of scrupulousness, shame and diffidence.

"The next period, the disease is characterized by the return of the repressed reminiscences, hence, by the failure of the defense; but it remains undecided whether the awakening of the same is more frequently accidental and spontaneous, or whether it appears in consequence of actual sexual disturbances, that is, as additional influences of the same. But the revived reminiscences and the reproaches formed from them never enter into consciousness unchanged, but what becomes conscious as an obsession and obsessive affect and substitutes the pathogenic memory in the conscious life, are compromise formations between the repressed and the repressing ideas.

"In order to describe clearly and probably convincingly the processes of repression, the return of the repression, and the formation of the pathological ideas of compromise, we would have to decide upon very definite hypotheses concerning the substratum of the psychic occurrence and consciousness. As long as we wish to avoid it we will have to rest content with the following rather figuratively understood observations. Depending on whether the memory content of the reproachful action alone forces an entrance into consciousness or whether it takes with it the accompanying reproachful affect, we have two forms of compulsion neurosis. The first represents the typical obsessions, the content of which attracts the patient's attention; only an indefinite displeasure is perceived as an affect, whereas, for the content of the obsession the only suitable affect would be one of reproach. The content of the obsession is doubly distorted when compared to the content of the infantile compulsive act. First, something actual replaces the past experience, and second, the sexual is substituted by an analogous non-sexual experience. These two changes are the results of the constant tendency to the repression still in force which we will attribute to the 'ego.' The influence of the revived pathogenic memory is shown by the fact that the content of the obsession is still partially identical with the repressed, or can be traced to it by a correct stream of thought. If, with the help of the psychoanalytic method, we reconstruct the origin of one individual obsession we find that one actual impression instigated two diverse streams of thought, and that the one which passed over the repressed memory, though incapable of consciousness and cor-

rection, proves to be just as correctly formed logically as the other. If the results of the two psychic operations disagree, the contradiction between the two may never be brought to logical adjustment, but as a compromise between the resistance and the pathological result of thought an apparently absurd obsession enters into consciousness beside the normal result of the thought. If both streams of thought yield the same result, they reinforce each other so that the normally gained result of thought now behaves psychically like an obsession. Wherever neurotic compulsion manifests itself psychically it originates from repression. The obsessions have, as it were, a psychical course of compulsion which is due, not to their own validity, but to the source from which they originate, or to the source which furnishes a part of their validity.

"A second form of compulsion neurosis results if the repressed reproach and not the repressed content of memory forces a replacement in the conscious psychic life. Through a psychic admixture, the affect of the reproach can change itself into any other affect of displeasure, and if this occurs there is nothing to hinder the substituting affect from becoming conscious. Thus the reproach (of having performed in childhood some sexual actions) may be easily transformed into shame (if some one else becomes aware of it), into hypochondriacal anxiety (because of the physical harmful consequences of those reproachful acts), into social anxiety (fearing punishment from others), into religious anxiety, into delusions of observation (fear of betraying those actions to others), into fear of temptations (justified distrust in one's own moral ability of resistance), etc. Besides, the memory content of the reproachful action may also be represented in consciousness, or it may be altogether concealed, which makes the diagnosis very difficult. Many cases which on superficial examination are taken as ordinary (neurasthenic) hypochondria often belong to this group of compulsive affects; the very frequently so-called 'periodic neurasthenia' or 'periodic melancholia' especially seem to be explained by compulsive affects or obsessions, a recognition not unimportant therapeutically.

"Beside these compromise symptoms which signify the return of the repression and hence a failure of the originally achieved defense, the compulsion neurosis forms a series of other symptoms of a totally different origin. The ego really tries to defend itself against those descendants of the initial repressed reminiscence, and in this conflict of defense it produces symptoms which may be comprehended as 'secondary defense.' These are throughout 'protective measures' which have performed good service in the struggle carried on against the obsessions and the obsessing affects. If these helps in the conflict of the defense really succeed in repressing anew the symptoms of return obtruding themselves on the ego, the compulsion then transmits itself on the protective measures themselves and produces a third form of the 'compulsion neurosis,' the compulsive action.

These are never primary, they never contain anything else but a defense, never an aggression. Psychic analysis shows that despite their peculiarity they can always be fully explained by reduction to the compulsive reminiscence which they oppose.

“One example instead of many: An eleven-year-old boy has obsessively arranged for himself the following ceremonial before going to bed: He could not fall asleep unless he related to his mother most minutely all experiences of the day; not the smallest scrap of paper or any other rubbish was allowed in the evening on the carpet of his bedroom. The bed had to be moved close to the wall, three chairs had to stand in front of it, and the pillows had to lie in just such a position. In order to fall asleep he had to kick with both legs a number of times, and then had to lie on the side. This was explained as follows: Years before, while putting this pretty boy to sleep, the servant girl made use of this opportunity to lay over him and assault him sexually. When this reminiscence was later awakened by a recent experience it made itself known to consciousness by the compulsion in the above-mentioned ceremonial which sense could really be surmised and the details verified by psychoanalysis. The chairs before the bed which was close to the wall—so that no one could have access to it; the arrangement of the pillows in a definite manner—so that they should be differently arranged than they were on that evening; the motion with the legs—to kick away the person lying on him; sleeping on the side—because during that scene he lay on his back; the detailed confession to his mother—because in consequence of the prohibition of his seductress he concealed from his mother this and other sexual experiences; finally, keeping the floor of his bedroom clean—because this was the main reproach which he had to hear from his mother up to that time.

“The secondary defense of the obsessions can be brought about by a forcible deviation to other thoughts of possibly contrary content; hence, in case of success there is a compulsive reasoning regularly, concerning abstract and transcendental subjects, because the repressed ideas always occupied themselves with the sensuous. Or the patient tries to become master of every compulsive idea through logical labor and by appealing to his conscious memory; this leads to compulsive thinking and examination to doubting mania. The priority of the perception before the memory in these examinations at first induce and then force the patient to collect and preserve all objects with which he comes in contact. The secondary defense against the compulsive affects results in a greater number of defensive measures which are capable of being transformed into compulsive actions. These can be grouped according to their tendency. We may have measures of penitence (irksome ceremonial and observation of numbers), of prevention (diverse phobias, superstition, pedantry, aggravation of the primary symptom of scrupulousness), measures of fear of betrayal (collecting papers and shyness), and measures of becoming uncon-

scious (dipsomania). Among these compulsive acts and impulses the phobias play the greatest part as limitations of the patient's existence.

"There are cases in which we can observe how the compulsion becomes transferred from the idea or affect to the measure, and other cases in which the compulsion oscillates between the returning symptoms of secondary defense. But there are also cases in which no obsessions are really formed, but the repressed reminiscence immediately becomes replaced by the apparent primary defensive measure. Here that stage is attained at a bound which otherwise ends the course of the compulsion neurosis only after the conflict of the defense. Grave cases of this affection end either with a fixation of ceremonial actions, general doubting mania, or in an existence of eccentricity conditioned by phobias.

"That the obsessions and everything derived from them are not believed is probably due to the fact that the defense symptom of scrupulousness was formed during the first repression and gained compulsive validity. The certainty of having lived morally throughout the whole period of the successful defense makes it impossible to give credence to the reproach which the obsession really involves. Only transitorily during the appearance of a new obsession, and now and then in melancholic exhaustive states of the ego do the morbid symptoms of the return also enforce the belief. The 'compulsion' of the psychic formations here described has in general nothing to do with the recognition through belief, and is not to be mistaken for that moment which is designated as 'strength' or 'intensity' of an idea. Its main characteristic lies in its inexplicableness through psychic activities of conscious ability, and this character undergoes no change whether the idea to which the compulsion is attached is stronger or weaker, more or less intensively 'elucidated,' 'supplied with energy,' etc.

"The reason for the unassailableness of the obsession or its derivative is due only to its connection with the repressed memory of early childhood, for as soon as we succeed in making it conscious, for which the psychotherapeutic methods already seem quite sufficient, the compulsion, too, becomes detached."

The mechanism of the compulsion neurosis is therefore seen to be an extremely complicated one and one which produces a great variety of symptoms, with all possible ramifications of meaning.

This mechanism as set forth in this rather intricate statement by Freud may be more simply stated by saying that, in distinction from hysteria in which the disguise is brought about by a transfer of the repressed material into symbols of bodily ailment—conversion—in the compulsion neurosis the disfigurement is kept wholly within recognized psychological territory. The distortion is produced by displacement of the affect upon indifferent ideas and the development of a purificatory ceremonial. Fear of animals (snakes, mice, etc.) may be the accepted conscious equivalent of fear of sexuality with

a type of ceremonial, and is well illustrated in the case of the eleven-year-old boy cited.

It is important to bear in mind that the theory of infantile sexual trauma has long since been discarded by Freud himself. More attention is being paid to the present difficulties of the patient, particularly under the influence of Jung, who would always ask what task does the patient wish to avoid? The neurotic way of avoiding a task or duty is to revert to infantile methods. The infantile experiences do not explain the difficulty but only the symptoms. They only show why certain symptoms are used, because they represent material in the life of the patient and are merely being brought in this stage to serve a purpose.

Symptoms.—The symptoms of the compulsive neurosis are very varied. They have to do with all types of obsessional thinking and acting, that is, thinking and acting which takes place aside from the volition of the patient, which he cannot prevent but which he must yield to, as already described. This is the compulsion element which gives the name to the neurosis.

The symptoms have been variously divided and may be described under the form of motor symptoms, obsessive acts of various sorts; tics, spasmodic torticollis, even epileptic attacks; sensory symptoms, obsessive sensations, amounting at times to well-marked hallucinations; affective symptoms, obsessive emotions, more particularly those of doubt and fear; and ideational symptoms, obsessive ideas, such as continual questioning.

The commonest and best known of the obsessions are the *phobias* or fears which usually refer to some very specific object or set of conditions which acquire their quality of fear as the result of taking over an affect by displacement which is of deep though unconscious significance to the patient. Thus there are *misophobia* (fear of dirt or contamination), *metallophobia* (fear of metal, door-knobs, money, etc.), *agoraphobia* (fear of wide or open spaces), *claustrophobia* (fear of narrow or closed spaces), *pyrophobia* (fear of fire) and so on indefinitely.

The obsessions of *doubt—folie de doute*—are common and result in a state of mind in which the patient is torn between two courses of conduct and cannot choose, or having done something, such as turn out the gas before going to bed, is seized with a doubt as to whether he really did do it or not and must get up and satisfy himself. Then doubts when they refer to religious or philosophical matters lead to continuing questionings and elaborate processes of reasoning from which the patient cannot free his mind.

Quite allied to the phobias and doubts are certain moral obsessions such as overconscientiousness and exaggerated scrupulosity.

Of the various obsessional activities the so-called *manias* are best known. Thus there are *kleptomania* (a compulsion to steal), *pyromania* (a compulsion to set something on fire), *dipsomania* (a compulsion to drink), etc.

In addition there are all sorts of less easily classified and more complex forms of obsessional ways of thinking, feeling and acting. There are complicated ceremonials, such as that of the eleven-year-old boy already cited, ways of arranging things that must be carried out. Strange, and to the patient unexplicable, attractions and repulsions, dislikes for certain kinds of food, or persons with a particular color of hair, all manner of tics, habits, mannerisms, ceremonials, the necessity for touching things—*delire de toucher*—fixed ideas, hypochondrias, etc.

Such psychological phenomena must, of course, have a reason for their existence, and if the reason is not apparent it cannot be explained by the patient; it must be sought by psychoanalysis in the unconscious.

These obsessions produce a tremendous amount of mental unrest and suffering if they are not yielded to, and a sense of relief is experienced when they are yielded to, oftentimes, however, with a following sense of remorse for having yielded.

The compulsion ideas and acts represent compromise formations which permit the patient to obtain satisfactions in infantile ways, *i. e.*, to revert to old ways of gaining pleasure which were active and important in infancy when the erogenous zones were as yet not clearly differentiated. Here we find the explanation for urinary and fecal phantasies, for certain cutaneous, anal, and gastro-intestinal satisfactions which are used as ways of getting pleasure when driven away from reality. They become infantile ways of reacting to reality situations and so are inefficient, sick ways.

Anxiety Hysteria.—Anxiety hysteria, as the name indicates, occupies a midposition between conversion hysteria on the one hand and anxiety neurosis on the other. There is, so to speak, a combination of the two conditions, although this is not quite the situation. In conversion hysteria the affect of the repressed complexes is drafted into bodily innervation and produces the physical symptoms of the disease. In anxiety hysteria the affect remains in the mental sphere, producing there various phobias. In anxiety neurosis, as will be seen later, the anxiety has its origin not at the psychical, but at the physiological level and is a representation in the psychic sphere of a disturbance in the somatic. In anxiety hysteria, the anxiety is also produced at the physiological level, but it is a secondary symptom and is the result of the physiological accompaniments of the emotions which go with the phobias, such as difficulty of breathing, cardiac palpitation, etc.

Anxiety hysteria is one of the most widely distributed diseases. It is particularly the disease which manifests itself in childhood and from which most of the so-called nervous children suffer. It is much more easy of approach therapeutically than the compulsion neurosis, its accessibility being comparable to that of hysteria, and so offers greater opportunities for treatment. Probably many cases of this disorder are included under Janet's psychasthenia.

THE ACTUAL NEUROSES.

Anxiety Neurosis.—The anxiety neurosis was separated from the general group of actual neuroses and psychoneuroses by Freud. The name anxiety neurosis indicates that the symptoms all group themselves about the cardinal symptom of anxiety, and it is significant of this anxiety, that, while it is a psychic fact, it is still not of psychic but of somatic origin. The anxiety, therefore, is not susceptible of being analyzed into psychic components, but its source can only be found at the physiological level. This anxiety arising at the physiological level and manifesting itself in the psychic sphere then becomes a "free-floating anxiety" which may attach itself to any idea and therefore appear to be of psychic origin. On the other hand it may express itself simply as anxiety without ideational content.

Anxiety may thus be seen to be the correlative of fear. While fear is the emotion which corresponds to a danger threatening the organism from outside, anxiety corresponds to a danger which threatens the organism from within.

It will help to make understandable what has to be said about the anxiety neurosis if it is understood at the start that the whole sex relationship which is consummated by the sexual act consists of two parts, a somatic and a psychic. In contrast to neurasthenia, which results when the discharge of energy is inadequate upon the somatic side, anxiety neurosis occurs whenever the discharge is inadequate in the psychic sphere.

Symptoms.—The following is the description of the symptomatology of anxiety neurosis as given by Freud:¹

"1. *General Irritability.*—This is a frequent nervous symptom, common as such to many nervous states. It is mentioned here because it constantly occurs in the anxiety neurosis and is of theoretical significance. Increased irritability always points to an accumulation of excitement or to an inability to bear accumulation, hence to an absolute or relative accumulation of excitement. The expression of this increased irritability through an auditory hyperesthesia is especially worth mentioning; it is an oversensitiveness for noises, which symptom is certainly to be explained by the congenital intimate relationship between auditory impressions and fright. Auditory hyperesthesia is frequently found as a cause of insomnia, of which more than one form belongs to anxiety neurosis.

"2. *Anxious Expectation.*—I cannot better explain the condition that I have in mind than by this name and by some appended examples. A woman, for example, who suffers from anxious expectation thinks of influenza-pneumonia whenever her husband, who is afflicted with a catarrhal condition, has a coughing spell; and in her mind she sees a passing funeral procession. If on her way home she

¹ Loc. cit.

sees two persons standing together in front of her house she cannot refrain from the thought that one of her children fell out of the window; if she hears the bell ring she thinks that someone is bringing her mournful tidings, etc.; yet in none of these cases is there any special reason for exaggerating a mere possibility.

"The anxious expectation naturally reflects itself constantly in the normal, and embraces all that is designated as 'uneasiness and a tendency to a pessimistic conception of things,' but as often as possible it goes beyond such a plausible uneasiness, and it is frequently recognized as a part of constraint even by the patient himself. For one form of anxious expectation, namely, that which refers to one's own health, we can reserve the old name of hypochondria. Hypochondria does not always run parallel with the height of the general anxious expectation; as a preliminary stipulation it requires the existence of paresthesias and annoying somatic sensations. Hypochondria is thus the form preferred by the genuine neurasthenics whenever they merge into the anxiety neurosis, a thing which frequently happens.

"As a further manifestation of anxious expectation we may mention the frequent tendency observed in morally sensitive persons to pangs of conscience, scrupulosity, and pedantry, which varies, as it were, from the normal to its aggravation as doubting mania.

"Anxious expectation is the most essential symptom of the neurosis; it also clearly shows a part of its theory. It can perhaps be said that we have here a quantum of freely floating anxiety which controls the choice of ideas by expectation and is forever ready to unite itself with any suitable ideation.

"3. This is not the only way in which the anxiousness, usually latent but constantly lurking in consciousness, can manifest itself. On the contrary it can also suddenly break into consciousness without being aroused by the issue of an idea, and thus provoke an attack of anxiety. Such an attack of anxiety consists of either the anxious feeling alone without any associated idea, or of the nearest interpretation of the termination of life, such as the idea of 'sudden death' or threatening insanity; or the feeling of anxiety becomes mixed with some paresthesia (similar to the hysterical aura); or finally the anxious feeling may be combined with a disturbance of one or many somatic functions, such as respiration, cardiac activity, the vasomotor innervation, and the glandular activity. From this combination the patient renders especially prominent now this and now the other moment. He complains of 'heart spasms,' 'heavy breathing,' 'profuse perspiration,' 'inordinate appetite,' etc., and in his description the feeling of anxiety is put to the background or it is rather vaguely described as 'feeling badly,' 'uncomfortably,' etc.

"4. What is interesting and of diagnostic significance is the fact that the amount of admixture of these elements in the attack of anxiety varies extraordinarily, and that almost any accompanying

symptom can alone constitute the attack as well as the anxiety itself. Accordingly there are rudimentary attacks of anxiety, and equivalents for the attack of anxiety, probably all of equal significance in showing a profuse and hitherto little-appreciated richness in forms. A more thorough study of these larvated states of anxiety (Hecker) and their diagnostic division from other attacks ought soon to become the necessary work for the neuropathologist.

"I now add a list of those forms of attacks of anxiety with which I am acquainted. There are attacks:

"(a) With disturbances of heart action, such as palpitation with transitory arrhythmia, with longer-continued tachycardia up to grave states of heart weakness, the differentiation of which from organic heart affection is not always easy; among such we have the pseudo-angina pectoris, a delicate diagnostic sphere.

"(b) With disturbances of respiration, many forms of nervous dyspnea, asthma-like attacks, etc. I assert that even these attacks are not always accompanied by conscious anxiety;

"(c) Of profuse perspiration, often nocturnal;

"(d) Of trembling and shaking which may readily be mistaken for hysterical attacks;

"(e) Of inordinate appetite, often combined with dizziness;

"(f) Of attack-like appearing diarrhea;

"(g) Of locomotor dizziness;

"(h) Of so-called congestions, embracing all that was called vasomotor neurasthenia; and

"(i) Of paresthesias (these are seldom without anxiety or a similar discomfort).

"5. Very frequently the nocturnal frights (*pavor nocturnus* of adults) usually combined with anxiety, dyspnea, perspiration, etc., is nothing other than a variety of the attack of anxiety. This disturbance determines a second form of insomnia in the sphere of the anxiety neurosis. Moreover, I became convinced that even the *pavor nocturnus* of children evinces a form belonging to the anxiety neurosis. The hysterical tinge and the connection of the fear with the reproduction of appropriate experience or dream, makes the *pavor nocturnus* of children appear as something peculiar, but it also occurs alone without a dream or a recurring hallucination.

"6. '*Vertigo*.'—This in its lightest forms is better designated as 'dizziness,' assumes a prominent place in the group of symptoms of anxiety neurosis. In its severer forms the 'attack of vertigo,' with or without fear, belongs to the gravest symptoms of the neurosis. The vertigo of the anxiety neurosis is neither a rotatory dizziness nor is it confined to certain planes or lines like Ménière's vertigo. It belongs to the locomotor or coördinating vertigo, like the vertigo in paralysis of the ocular muscles; it consists in a specific feeling of discomfort which is accompanied by sensations of a heaving ground, sinking legs, of the impossibility to continue in an upright position, and at the

same time there is a feeling that the legs are as heavy as lead, they shake, or give way. This vertigo never leads to falling. On the other hand, I would like to state that such an attack of vertigo may also be substituted by a profound attack of syncope. Other fainting-like states in the anxiety neurosis seem to depend on a cardiac collapse.

"The vertigo attack is frequently accompanied by the worst kind of anxiety and is often combined with cardiac and respiratory disturbances. Vertigo of elevations, mountains and precipices, can also be frequently observed in anxiety neurosis; moreover, I do not know whether we are still justified in recognizing a vertigo of stomachic origin.

"7. On the basis of the chronic anxiousness (anxious expectation) on the one hand, and the tendency to vertiginous attacks of anxiety on the other, there develop two groups of typical phobias; the first refers to the general physiological menaces, while the second refers to locomotion. To the first group belong the fear for snakes, thunderstorms, darkness, vermin, etc., as well as the typical moral over-scrupulousness, and the forms of doubting mania. Here the available fear is merely used to strengthen those aversions which are instinctively implanted in every man. But usually a compulsively acting phobia is formed only after a reminiscence is added to an experience in which this fear could manifest itself; as, for example, after the patient has experienced a storm in the open air. To attempt to explain such cases as mere continuations of strong impressions is incorrect. What makes these experiences significant and their reminiscences durable is after all only the fear which could at that time appear and can also appear today. In other words such impressions remain forceful only in persons with 'anxious expectations.'

"The other group contains agoraphobia with all its accessory forms, all of which are characterized by their relation to locomotion. As a determination of the phobia we frequently find a precedent attack of vertigo; I do not think that it can always be postulated. Occasionally, after a first attack of vertigo without fear, we see that though locomotion is always accompanied by the sensation of vertigo, it remains possible without any restrictions, but as soon as fear attaches itself to the attack of vertigo, locomotion fails, under the conditions of being alone, narrow streets, etc.

"The relation of these phobias to the phobias of obsessions, which mechanism I discussed above,¹ is as follows: The agreement lies in the fact that here as there, an idea becomes obsessive through its connection with an available affect. The mechanism of transposition of the affect therefore holds true for both kinds of phobias. But in phobias of the anxiety neurosis this affect is (1) a monotonous one, it is always one of anxiety; (2) it does not originate from a repressed idea, and on psychological analysis it proves itself not further reducible, nor can it be attacked through psychotherapy. The mechanism of

¹ Die Abwehr-Neuropsychosen, Neurol. Centralbl., 1894, No. 10, u. ii.

substitution does not therefore hold true for the phobias of anxiety neurosis.

“Both kinds of phobias (or obsessions) often occur side by side, though the atypical phobias which depend on obsessions need not necessarily develop on the basis of anxiety neurosis. A very frequent, ostensibly complicated mechanism appears if the content of an original simple phobia of anxiety neurosis is substituted by another idea, the substitution is then subsequently added to the phobia. The ‘protective measures’ originally employed in combating the phobia are most frequently used as substitutions. Thus, for example, from the effort to provide one’s self with counter-evidence that one is not crazy, contrary to the assertion of the hypochondriacal phobia, there results a reasoning mania. The hesitations, doubts, and the many repetitions of the *folie du doute* originate from the justified doubt concerning the certainty of one’s own stream of thoughts, for, through the compulsive-like idea one is surely conscious of so obstinate a disturbance, etc. It may therefore be claimed that many syndromes of compulsion neurosis, like *folie de doute* and similar ones, can clinically, if not notionally, be attributed to anxiety neurosis.¹

“8. The digestive functions in anxiety neurosis are subject to very few but characteristic disturbances. Sensations like nausea and sickly feeling are not rare, and the symptom of inordinate appetite alone or with other congestions, may serve as a rudimentary attack of anxiety. As a chronic alteration analogous to the anxious expectations one finds a tendency to diarrhea which has occasioned the queerest diagnostic mistakes. If I am not mistaken it is this diarrhea to which Moebius² has recently called attention in a small article. I believe, moreover, that Peyer’s³ reflex diarrhea which he attributes to a disease of the prostate is nothing other than the diarrhea of anxiety neurosis. The deceptive reflex relation is due to the fact that the same factors which are active in the origin of such prostatic affections also come into play in the etiology of anxiety neurosis.

“The behavior of the gastro-intestinal function in anxiety neurosis shows a sharp contrast to the influence of this same function in neurasthenia. Mixed cases often show the familiar ‘fluctuations between diarrhea and constipation.’ The desire to urinate in anxiety neurosis is analogous to the diarrhea.

“9. The paresthesias which accompany the attack of vertigo or anxiety are interesting because they associate themselves into a firm sequence, similar to the sensations of the hysterical aura. But in contrast to the hysterical aura I find these associated sensations atypical and changeable. Another similarity to hysteria is shown by the fact that in anxiety neurosis a kind of conversion⁴ into bodily

¹ Obsessions et phobies, *Révue neurologique*, 1895.

² *Neuropathologische Beiträge*, 1894, ii, Heft.

³ *Die nervösen Affektionen des Darmes*, *Wiener Klinik*, 1893.

⁴ Freud, *Abwehr-Neuropsychose*.

sensations, as, for example, into rheumatic muscles, takes place which otherwise can be overlooked at one's pleasure. A large number of so-called rheumatics, who are moreover demonstrable as such, really suffer from an anxiety neurosis. Besides this aggravation of the sensation of pain I have observed in a number of cases of anxiety neurosis a tendency toward hallucinations which could not be explained as hysterical.

"10. Many of the so-called symptoms which accompany or substitute the attack of anxiety also appear in a chronic manner. They are then still less discernible, for the anxious feeling accompanying them appears more indistinct than in the attack of anxiety. This especially holds true for the diarrhea, vertigo, and paresthesias. Just as the attack of vertigo can be substituted by an attack of syncope, so can the chronic vertigo be substituted by the continuous feeling of feebleness, lassitude, etc."

The Etiology and Occurrence of Anxiety Neurosis.—The following remarks on the etiology and occurrences of anxiety neurosis are quoted from Freud's original paper:¹

"In some cases of anxiety neurosis no etiology can readily be ascertained. It is noteworthy that in such cases it is seldom difficult to demonstrate a marked hereditary taint.

"Where we have reason to assume that the neurosis is acquired we can find by careful and laborious examination that the etiologically effective moments are based on a series of injuries and influences from the sexual life. These at first appear to be of a varied nature but easily displayed the common character which explains their homogeneous effect on the nervous system. They are found either alone or with other banal injuries to which a reinforcing effect can be attributed. This sexual etiology of anxiety neurosis can be demonstrated so preponderately often that I venture for the purpose of this brief communication to set aside all cases of a doubtful or different etiology.

"For the more precise description of the etiological determinations under which anxiety neurosis occurs, it will be advisable to treat separately those occurring in men and those occurring in women. Anxiety neurosis appears in women—disregarding their predisposition—in the following cases:

"(a) As virginal fear or anxiety in adults. A number of unequivocal observations showed me that an anxiety neurosis, which is almost typically combined with hysteria, can be evoked in maturing girls, at the first encounter with the sexual problem, that is, at the sudden revelation of the things hitherto veiled, by either seeing the sexual act, or by hearing or reading something of that nature;

"(b) As fear in the newly married. Young women who remain anesthetic during the first cohabitation not seldom merge into an anxiety neurosis which disappears after the anesthesia is displaced by

¹ Loc. cit.

the normal sensation. As most young women remain undisturbed through such a beginning anesthesia, the production of this fear requires determinants which I will mention;

“(c) As fear in women whose husbands suffer from ejaculatio precox or from diminished potency; and,

“(d) In those whose husbands practice coitus interruptus or reservatus. These cases go together, for on analyzing a large number of examples one can easily be convinced that they only depend on whether the woman attained gratification during coitus or not. In the latter case one finds the determinant for the origin of anxiety neurosis. On the other hand, the woman is spared from the neurosis if the husband afflicted by ejaculatio precox can repeat the congress with better results immediately thereafter. The congressu reservatus by means of the condom is not injurious to the woman if she is quickly excited and the husband is very potent; in other cases the noxiousness of this kind of preventive measure is not inferior to the others. Coitus interruptus is almost regularly injurious; but for the woman it is injurious only if the husband practises it regardlessly, that is, if he interrupts coitus as soon as he comes near ejaculating without concerning himself about the determination of the excitement of his wife. On the other hand if the husband waits until his wife is gratified, the coitus has the same significance for the latter as a normal one; but then the husband becomes afflicted with an anxiety neurosis. I have collected and analyzed a number of cases which furnished the material for the above statements.

“(e) As fear in widows and intentional abstainers, not seldom in typical combination with obsessions; and

“(f) As fear in the climacterium during the last marked enhancement of the sexual desire.

“The cases (c), (d), and (e) contain the determinants under which the anxiety neurosis originates in the female sex most frequently and most independently, of hereditary predisposition. I will endeavor to demonstrate in these—curable, acquired—cases of anxiety neurosis that the discovered sexual injuries really represent the etiological moments of the neurosis. But before proceeding I will mention the sexual determinants of anxiety neurosis in men. I would like to formulate the following groups, everyone of which finds its analogy in women:

“(a) Fear of the intentional abstainers; this is frequently combined with symptoms of defense (obsessions, hysteria). The motives which are decisive for intentional abstinence carry along with them the fact that a number of hereditarily burdened eccentrics, etc., belong to this category.

“(b) Fear in men with frustrated excitement (during the engagement period), persons who out of fear for the consequences of sexual relations satisfy themselves with handling or looking at the woman. This group of determinants which can moreover be transferred to

the other sex—engagement periods, relations with sexual forbearance—furnish the purest cases of the neurosis.

“(c) Fear in men who practice coitus interruptus. As observed above, coitus interruptus injures the woman if it is practised regardless of the woman’s gratification; it becomes injurious to the man if in order to bring about the gratification in the woman he voluntarily controls the coitus by delaying the ejaculation. In this manner we can understand why it is that in couples who practise coitus interruptus it is usually only one of them who becomes afflicted. Moreover, the coitus interruptus only rarely produces in man a pure anxiety neurosis, usually it is a mixture of the same with neurasthenia.

“(d) Fear in men in the senium. There are men who show a climacterium like women, and merge into an anxiety neurosis at the time when their potency diminishes and their libido increases.

“Finally I must add two more cases holding true for both sexes:

“(e) Neurasthenics merge into anxiety neurosis in consequence of masturbation as soon as they refrain from this manner of sexual gratification. These persons have especially made themselves unfit to bear abstinence.

“What is important for the understanding of the anxiety neurosis is the fact that any noteworthy development of the same occurs only in men who remain potent, and in non-anesthetic women. In neurasthenics, who on account of masturbation have markedly injured their potency, anxiety neurosis as a result of abstinence occurs but rarely and limits itself usually to hypochondria and light chronic dizziness. The majority of women are really to be considered as ‘potent;’ a real impotent, that is, a real anesthetic woman, is also inaccessible to anxiety neurosis, and bears strikingly well the injuries cited.

“How far we are perhaps justified in assuming constant relations between individual etiological moments and individual symptoms from the complex of anxiety neurosis, I do not care to discuss here.

“(f) The last of the etiological determinants to be mentioned seems, in the first place, really not to be of a sexual nature. Anxiety neurosis originates in both sexes through overwork, exhaustive exertion, as, for instance, after sleepless nights, nursing the sick, and even after serious illnesses.”

Neurasthenia.—The term neurasthenia, since it first came into use only a little over a generation ago, has been applied to almost every conceivable condition. Almost all illnesses are accompanied by a certain amount of easy fatigability, emotional instability, and a general out-of-sorts feeling. All combinations of this kind which cannot be specifically diagnosed and placed under some well-known caption are easily dropped into the miscellaneous group of neurasthenia. Not only have all sorts of conditions, therefore, been included under this term, but the most varied symptoms have been thereby designated as neurasthenic. Not only have many physical conditions been included, such as general arteriosclerosis, but it is not uncommon for some of the more

serious psychoses, especially in their milder manifestations, as the cyclothymias, to be diagnosed as neurasthenia. It is highly desirable, therefore, to limit the application of the term to a definite condition.

It is better to consider neurasthenia as the expression of a very marked auto-erotic fixation, as a return to that infantile period of development in which the child takes a preponderating interest in its own body. Masturbation is quite liable to be indulged in as a means of auto-erotic satisfaction but the physical act of masturbation is perhaps relatively unimportant as compared with the crippling effects of the auto-erotic introversion.

This condition is known as a primary fatigue neurosis and has certain quite characteristic and constant symptoms which are in the main a feeling of pressure on the top of the head, more or less insomnia, spinal irritation, with perhaps pain in the back, certain paresthesias, easy fatigability, emotional irritability, and some depression.

This condition, despite outward evidence to the contrary, has been traced in most instances where careful analysis of the symptoms has been made, to a specific sexual etiology, namely to excessive masturbation or frequent pollutions. In contrast to the etiology of the anxiety neurosis, which, as has been said, is dependent upon an inadequate utilization and incomplete discharge of the energy of the sexual act in the psychic sphere, in neurasthenia the specific etiology is dependent upon an inadequate discharge in the physical sphere.

One has to think in addition to the specific etiology of the fact that in most instances where masturbation is practised into adulthood there is a serious moral conflict. The individual feels ashamed, chagrined, humiliated by having yielded to the physical demand. This, of course, adds to the difficulty by increasing the amount of energy dissipated. In addition to this the moral conflict is usually very greatly enhanced either by being told or reading of the awful results of this habit. This is especially so when these results are told to the child in order to frighten him into desisting and are accompanied by threats of cutting off the organ and the like.

It must not be lost sight of in dealing with this class of patients that a moderate amount of masturbation during infancy, about the third or fourth year, at the end of what Freud calls the first latency period, is normal and probably has as its function the focalization of the sexual erethism upon the sex organs. It will be remembered that before this time the various erogenous zones of the body such as the sex organs, the lips, the anus, are of practically equal significance. For the function of reproduction the sex organs must emerge with a preponderant erethism, otherwise some one of the perversions will take the place of normal sexuality. It seems, therefore, the function of masturbation to help produce this result.

When masturbation, however, is indulged in about the period of puberty and later on into adult life it has certain dangers in addition

to those which are more nearly at the physiological level and which are productive of neurasthenia. The individual in his psychosexual development passes through an auto-erotic period when he finds his sexual interests in himself, then through a period in which his sexual interests are transferred to the immediate members of the family, the period of narcissism in which at first he is most interested in those members of the family most like himself, namely of the same sex. Passing through this homosexual and narcissistic period he finally reaches, after having passed the period of puberty, to the possibility of giving his love out to someone else, not only besides himself, but someone removed from the family circle and someone of the opposite sex. He becomes normally heterosexual and attains the period of object love. Now one of the serious dangers of masturbation is the danger it has of fixing the individual at some intermediate point in his psychosexual development. The principal danger is, of course, fixation at the infantile auto-erotic period, which is naturally the particular quality of sexuality that masturbation ministers to. This fixation not only prevents the proper psychosexual development, but drags the whole personality back upon itself and prevents that open, free, and outward manifestation which is essential to success in life, to a finding of one's place in the world. These people are too thoroughly occupied with themselves to be able to deal with the outside world of reality with any degree of efficiency.

In the act of masturbation the individual is both the subject and the object. He has to supply the energies from both sources, not only the energies from within, but all of the energies and stimuli which normally would come from without from the person of another. During his indulgences he frequently develops all manner of phantasies and these phantasies often throw a considerable light upon the nature of his psychosexual development, quite similarly as do the dreams in those patients who suffer from frequent pollutions.

It will be seen, therefore, that in neurasthenia there is a condition that is by no means simple and that requires careful analytical study in order to be able to deal with it intelligently.

A final word as to the causes of neurasthenia: The alleged causes of this disorder have been as multiform as the conditions which have been ranged under it. There are a large group of cases which are supposed to be dependent upon injuries, traumatic neurasthenia, and another large group that is supposed to be dependent upon overwork. Although it is not quite possible to speak dogmatically with regard to the traumatic group at this time, still from analogy, as the result of cases studied in the group supposed to be dependent upon overwork, it will be seen that the same reasoning applies to both and it is again the same reasoning that may apply to anxiety neurosis, or in fact to any of the conditions described in this chapter, but more particularly perhaps to the actual neuroses. The traumatism or the overwork, as the case may be, or any other apparent assigned cause can be said not

to be the true cause of the neurosis, but only its occasion. The traumatism or the overwork could not produce the neurosis in the absence of the specific etiology. It is quite understandable that a given individual may stand a series of sexual traumatisms over a considerable period of time, but be strong enough to resist the development of a neurosis. On the occasion, however, of having his resistance reduced as the result of an injury, or as the result of long-continued overwork the neurosis crops out. This is the explanation for many of these conditions, and it is the reason why a banal cause may develop a result that is out of all proportion in both *quantity and quality*.

Mixed Neuroses.—Hysteria, the compulsion neurosis, anxiety neurosis, and neurasthenia have been described. If the etiology and mechanisms of these four conditions be considered it will be seen that they do not of necessity mutually exclude one another and as a matter of fact not infrequently certain admixtures are found in clinical experience. Anxiety hysteria, for example, has taken a rather definite place among these conditions, while as will be readily seen from the nature of the etiological moments neurasthenia and anxiety neurosis are not infrequently found associated in various proportions, while, of course, it follows that the etiological moments of the actual neuroses are not excluded from operating in the same patient who may have a psychoneurosis. Such combinations are, therefore, of not uncommon occurrence.

Aside from the possibility of the mixture of the neuroses it should also not be lost sight of that the etiological moments of the neuroses may also operate in persons who are suffering from the more severe psychoses, as for example, manic-depressive psychosis and dementia precox. We quite commonly find hysterical symptoms in the precox, while neurasthenic and anxiety states are not infrequent in the depressions of manic-depressive psychosis. Other combinations, of course, might be mentioned. The important thing to bear in mind, however, is the nature of the etiological moment and the mechanism of the condition, and then these will be recognized when the individual patient is under analytic observation.

Finally, the purely psychic element is found more and more in association with the actual neuroses. This was intimated in the description of the etiology and mechanisms of neurasthenia and it has also been spoken of in the description of the anxiety neurosis. More will be said on this point under the head of Treatment.

Treatment of the Neuroses.—In the treatment of the actual neuroses the main thing, as indicated by the description which has been given, is to correct the sexual life of the patient. In dealing with neurasthenia the habit of masturbation, if it be present, must, of course, be stopped before anything definite can be accomplished, while, of course, with both neuroses matters of coitus interruptus, ejaculatio precox, abstinence, etc., must be carefully inquired into and the sexual life modified as indicated so that the evil effects which result from them

may be remedied. These are the simple things to do, and in many cases will produce marked betterment, if not apparent recovery.

Rest cures, hydrotherapy, massage, electricity, exercise, and all such therapeutic agents have their place in the treatment of the neuroses, particularly the actual neuroses, but their place is a secondary one. It has already been indicated that the precipitating factor in the outbreak of a neurosis may, for example, be overwork, but that the neurosis would not eventuate in the absence of the specific etiology. The explanation of this occurrence was that the patient was strong enough to stand up under the results of sexual traumata until his general resistance was reduced by overwork and then the neurosis appeared. It will be seen, therefore, that the usual methods of treatment very frequently bring about a cure, but not in the way in which they are supposed to. By changing the patient's sexual habits, removing him from his surroundings, sending him away to a sanitarium, oftentimes the sexual situation is more or less well solved temporarily. Now, if during this period of residence in a sanitarium he is on careful diet, given regular exercise with baths and massage, it is perfectly understandable that his general resistance will be increased so that he may overcome the effects of the sexual traumata. In this way he may get well without any direct attack upon the factors of the specific etiology. This is the principle which is oftentimes seen in operation in the improvement and alleged recoveries of neurotics as a result of the rest cure.

Inasmuch, however, as even the actual neuroses usually present some admixture of purely mental symptoms, either primary or secondary in origin, it may be necessary ultimately and before satisfactory results can be obtained to deal with these mental symptoms. If so, they must be dealt with in precisely the same way as the mental symptoms of the psychoneuroses, namely by psychoanalysis.

The main principle involved in psychoanalysis may be said to be an analysis of the patient's mental condition sufficiently complete to thoroughly understand the symptomatic manifestations of his malady. Before psychoanalytic methods were employed no explanation was sought for mental symptoms and apparently it rarely entered anyone's mind that they had any. The patient who had a phobia or a tic was simply looked upon as being nervous, perhaps having had some fright or bad impression earlier in life, and was usually treated by tonics or rest or travel or some other such means that was not addressed to the solution of the problem in any way. From what has already been said about the psychic development of the individual it will be readily appreciated that no mental fact can fail to have a thoroughly logical and understandable reason for its existence, and it is the object of psychoanalysis to find this out.

Briefly, the technique of psychoanalysis is about as follows, being of course, modified in detail to some extent by the exigencies of the occasion and as the result of the special predilections of the physician

practising it. When the patient calls upon the physician the physician should let the patient, as far as possible without interrupting him by questions or otherwise, detail to him his difficulties. This may take only a few minutes, or may be quite a lengthy recital, but it is usually worth while to listen carefully to the whole thing, perhaps occasionally by a suggestion, keeping the patient to the point if he tends to be too circumstantial. This original statement contains a description of the things from which the patient is suffering, and if careful attention is paid to it one may get many hints as to how to pursue the further inquiry. As a result of this conversation and perhaps another, if it is necessary, the physician makes up his mind whether the patient's illness is a suitable one for psychoanalytic treatment, and if it is he so states and then makes arrangements for regular consultations, preferably not less than three times per week, of an hour's duration each, and all other preliminaries such as the fee, etc., are arranged too. Should it appear that the patient has any symptoms of physical illness he should be sent to a competent physician skilled in the particular trouble that appears to be present, for the psychoanalyst should under no circumstances undertake to treat the physical condition. The reasons for this will appear later. It is preferable, too, that the physical condition be attended to fully before the psychoanalytic treatment be taken up. In other words, it is undesirable to endeavor to do psychoanalysis while the patient is under the care of another physician.

Having arranged all the preliminaries, the patient calls at the appointed hour and the psychoanalytic conversation proceeds about as follows: Bearing in mind the ultimate goal, the psychological explanation of the patient's symptoms, the patient may be approached by beginning the discussion of one of the symptoms, either simply asking about it or else pursuing some line of inquiry that was suggested in the original conversation. On the other hand, the method may be pursued of endeavoring to first get a clear understanding of the whole life of the patient, beginning from the earliest recollections and tracing the development to the present. It really makes very little difference how one starts, because in a very short time there will be all manner of suggestions to develop inquiries along various lines, and these will have to be followed out for a complete untangling of the situation.

During the course of the psychoanalytic conversations one will get very shortly to a point from which progress seems to be impossible, for it does not take long to exhaust the consciousness of the patient. One then has to penetrate the foreconscious, which is relatively easy, and the unconscious, which is relatively difficult. The method of procedure here is the method by free association. Perhaps a point has been attained in the conversation, a situation has been unfolded, which has no apparent explanation. The patient cannot give any reasons which adequately account for it. Under these circumstances the patient is asked to, so to speak, take the situation as a starting-point, and then

relaxing into a condition of perfect passivity observe the thoughts that come to his mind and recount them as fast as they appear. In other words, he is asked to place himself in a mental state favorable to phantasy formation, he is asked to relinquish his grasp upon his mental life, to permit his ideas to flow untrammelled and unselected by his volition and critique. He is asked as it were to become the observer of his own ideas, to sit as if he were sitting in the window of a moving train, recounting aloud the objects as they passed by. So he is asked to observe his ideas and to tell them as they come. This sounds like rather a simple procedure, but it is a very difficult one for the patient to learn, and in fact when the patient can do it and do it easily he is approaching the end of his treatment.

Experience shows that when patients are instructed in this way they take the situation as directed as a starting-point, and with the intention of telling the ideas that come to their mind they will relax into a condition of passivity. Perhaps then for a considerable time they do not speak, and if they are asked why they do not tell what comes to their mind they will say that nothing comes. This is, of course, the interference of the repressed complexes; it is the resistance which they have to coming into consciousness which is manifesting itself in this way. The whole thing, then, has to be gone over with the patient. It has to be explained to them that their mind cannot be an absolute blank, and they have to be warned especially not to exercise choice as to what ideas they shall tell and what they shall not tell, that it makes no difference how absurd or inconsequential the idea is that comes to their mind they must tell it; even if the idea is extremely disagreeable they must tell it, for no matter how little connection it may appear to have with their trouble if the starting-point has been from some problem in the case these ideas that come must have some connection with that problem. It must be explained to them that, of course, they cannot see the connection, but that they must tell the ideas so that the physician may have them and that he will be able to see what bearing they have in the situation. Of course he may not be able to see at once, but it is so much material which, if it does not come in for utilization today, can be used perhaps tomorrow, or the day after.

This is the process of *free association*, one which is very difficult for the patient to learn and one which requires much skill and no little art on the part of the physician. The physician must be ever on the alert. It requires the most intense application to the question in hand, for everything must be watched with the utmost care. Every little detail must be observed as containing perhaps a hidden meaning behind it. The hesitations, the stammerings, the mistakes, the slips of the tongue, all have their significance. For example, in talking to a young man, who told about his previous illness, he said that the physician had prescribed four quarter-grain tablets of "quinin" for him. He had no sooner mentioned the name of the drug than he immediately

corrected himself and said "calomel." An inquiry into the meaning of quinin to him brought out a most important event in his life, an event undoubtedly of significance in his neurosis. The repressed complex was struggling, as it always is, for expression. A favorable opportunity presented itself. Calomel and quinin are easily mixed in the pronunciation, and the repression slipped its moorings for a moment and found expression. It remained for the observer to be sufficiently keen to see the possibility of meaning in such a mistake and find out that meaning.

The most important single aid in determining the content of the unconscious is the dream. The split-off complexes are, in accordance with the theory set forth, actively repressed by the individual, they are not permitted to come to expression if he can help it. They therefore can only express themselves in symbolic form, in which form they are disguised not only to others, but to the patient himself. In this disguised form they are not recognized for what they are, and therefore the painfulness of their recall to consciousness is avoided. A previous moral delinquency may thus come to the surface under a complete disguise without causing any particular distress, whereas it would be quite unbearable if it came forth in its true colors. It can be seen from this why it is that obstacles are so quickly reached in the psychoanalytic procedure. The dream is the best and most useful avenue for overcoming this type of obstacle. Here the repressed complexes come upon the stage in all their paraphernalia of symbolic disguise and find an opportunity for expression. The patient, not understanding what the dream means, will pretty generally recount it in all its details, a thing which he would absolutely refuse to do, in many instances at least, if he had the slightest suspicion of what it could mean. The physician is therefore, so to speak, in a position to come up on the blind side of the patient, to see the play of his unconscious phantasies, to be let behind the scenes, as it were. Now if he can penetrate these disguises then he not only is capable of determining the meaning of the dream, but also he learns in this way the nature of the repressed complexes, and is therefore in a position to begin to read meaning into the symptoms of the neurosis. The whole matter of dream interpretation constitutes a special chapter in psychology and is hardly discussable in a text-book of this sort with its necessary limitations.

In addition to the dream analyses one should also inquire into the phantasy formations of the patient, phantasies which are formed in the day time. They are interpreted on the same principles as the dream.

The events uncovered by the method of free-association must be dealt with as facts. It makes little difference whether they even did happen in reality or not they are nevertheless *psychological facts*; they represent the way the patient thinks and so have just as much value as if they represented real occurrences.

It will be seen, therefore, that psychoanalysis is a lengthy, painstaking, detailed dissection of the mind of the patient sufficient for the explanation of the symptoms. This dissection starts at the surface and may go to practically any depth. Bearing in mind the principles already elucidated it will be seen why it is possible to effect an apparent cure at various levels; why sometimes a single conversation may apparently produce the miracle of a cure, while in other patients a half-dozen will produce the same effect, and in still others months of careful work are required. The individual has been thrown out of adjustment by causes which, in their last analysis, have been operative the greater part of his life. Up to a certain point, however, up to a certain difficulty, he has been able to get along. Now, when this difficulty comes he breaks and the neurosis makes its appearance. If he can be patched up, so to speak, rehabilitated back to the point where the break occurred, an apparent cure results. For these apparent cures it will be seen that it is only necessary to carry the patients back to a point at which they are capable of making adjustment. This is what very often happens, especially in methods of treatment other than the psychoanalytic. For a real cure of the patient, however, something very much more radical than this is required, that the analyst has to proceed to ever and ever deeper levels until he has sought out and found the final stronghold of the neurosis. Anything short of this can only serve to effect a compromise.

This seems to be the most advantageous point to answer the question which is constantly being asked and which it seems impossible to make many people understand, namely, the question of how the analysis, the unraveling of the symptoms, produces a cure. In order to understand this it is necessary to recur to the statement that the illness is due to a conflict and that one element of the conflict is unconscious to the patient; he does not know what it is that he is fighting. He therefore cannot deal with it frankly, openly, intelligently. One is reminded of the story of a certain king who propounded the question to the wise men as to why a bowl of water was not increased in weight when a live fish was put into it, while it was increased in weight when a dead fish was put into it. This created a tremendous disturbance. All sorts of arguments and reasons were propounded, heated discussions arose, and the wise men were quite generally out of tune with one another. Finally, it occurred to some one to try the experiment and see what the facts were. As soon as the experiment was tried and the facts were determined there was no longer any cause for argument. The conflict subsided. This is quite comparable to the position in which the patient finds himself, fighting something which is unconscious and about which he knows nothing. The facts in the case in regard to the fish in the bowl of water were unknown, and as long as they were unknown nothing but chaos reigned among the wise men. As soon as the facts were brought to light, however, by proper experimentation, then there was no longer

anything to fight about. In addition to these reasons for the subsidence of the conflict there are others that are of more or less importance in different cases; among them is a large element of reëducation to which the patient is subjected throughout the period of psychoanalysis. The cause of his neurosis implies that he is somewhat infantile, somewhat undeveloped. The physician, who should be a man of wide learning, whose business it is to deal with the problem of right living, cannot help but infuse into the patient in the many hours of conversation a philosophy of life which is helpful, and this undoubtedly happens as one of the most important elements in the reëducation, development and rehabilitation of the patient.

While the simple uncovering of the meaning of a symptom is often enough to make it disappear, for the real rehabilitation of the patient the whole meaning of infantile ways of reacting must be finally understood and the patient must be willing to forego this means of obtaining pleasure in order to advance to a higher level of adjustment, to solve the difficulties by attaining a higher level which means at once renunciation and fulfilment.

And finally, about the matter of transference. The neurotics and the psychoneurotics and the vast majority of persons who require psychotherapeutic treatment, are introverted, that is, their interests are turned within, upon themselves, and they cannot be made over into efficient people capable of dealing with the outside world of reality until their interests can be made to flow outside of themselves, until they can become interested in persons and things and events. In the course of psychoanalysis, if it is to proceed successfully, one of the earliest things that happens is that the interest of the patient begins to flow upon the physician. It is transferred to him. This is a matter of utmost importance. It is a matter which should be watched with the greatest care, for it is the barometer of the relationship between physician and patient. It is the factor in the personal equation which plays such a great part and which was supposed in the old days to play practically the only part. As soon as transfer begins to take place then the patient will begin to bring dreams for analysis, and in other ways to show, so to speak, every desire to please the physician by doing as he wishes, and before long one will generally find that the dreams are occupied with the idea of the physician, they are transfer dreams. Now in these dreams one may find exactly how the physician is held in the mind of the patient. In symbolic form the transfer dream may indicate that the physician is held in high regard and that he is respected, and this is of course as it should be. On the other hand, he may have failed to deal with a certain situation adequately, and the dream will show that the patient is disappointed or that perhaps some idea that the physician suggested the patient thinks is ridiculous and silly, or the physician may make the mistake of talking over the head of the patient so that the patient cannot understand, and the dream will say what the patient could not say

himself, that it is all too deep for him, that he cannot follow, and that the physician is altogether beyond him in the whole matter, and it all seems quite hopeless. The transfer dream therefore becomes a very important matter and requires the physician constantly to look within and to exercise his self-critique, for he must always realize that if he fails at a certain point the trouble is not with the patient, but with himself, and sometimes the dream will indicate what the trouble is. The meaning of the transfer is of course that the patient cannot unburden his very soul, cannot stand unclothed in all the nakedness of his real self, cannot, in other words, completely confess himself to an indifferent person. There must be something in the physician which commands the patient's affection, respect and confidence, and therefore the psychoanalytic work, while it makes great demands upon the patient also makes great demands upon the physician.

Sometimes the transfer is of such a character and sufficiently intense of itself to interfere with the progress of the analysis. Under these circumstances it must be discussed with the patient, sufficiently discussed to do away with the resistances it has raised, and finally at the completion of the analysis the whole matter of the transfer must be freely gone into, and analyzed, so that there is no misunderstanding as to what its real meaning might be. The patient is to be placed in full and complete possession of all of the facts, which means a full and complete possession of himself. Nothing must be hidden from him, the mirror must be held up so that he can see himself in it in every detail.

It is because of the necessity of transfer to successful psychoanalytic handling of a case that it is undesirable to have another physician treating the patient at the same time. The other physician might acquire the transfer, and this would prevent the psychoanalyst from accomplishing anything. This is especially apt to be the case where the other physician has to do with the physical condition of the patient and has to come into personal contact with him in making examinations and the like.

As already said, the transfer is the result of the beginning flowing outward of the patient's interests into the world of reality. The physician naturally is the one toward whom this interest first flows. He therefore, so to speak, puts himself in a position to be utilized by the patient; he becomes a bridge by which the patient is able to get back into the world. At the completion of the treatment, when the transfer has been fully analyzed and the patient understands what it means, then the physician by so doing steps aside. Having served the purpose of a bridge, having gotten the patient back into reality, he steps aside and leaves the patient there to stand upon his own feet.

The great obstacle to the psychoanalytic method of treatment is the general obstacle that the patient in a certain real sense does

not want to get well. There are two contrary trends struggling for supremacy in his psyche. He has two sets of desires each trying to gain fulfilment and each diametrically opposed to the other. Now the symptoms of his neurosis constitute a compromise, and in his then state of mind the only compromise, the only solution of the problem possible to him. He therefore, although he wants to get away from the suffering of his neurosis, still is unwilling to give up the symptoms which compensate him, even though that compensation be inadequate. We see this illustrated in many ways. For example, the patient insists upon leading the physician back to infantile occurrences in order to escape a frank discussion and facing of present problems. Again, by the free associations. The patient will lead the physician up all sorts of blind alleys for the same purpose. The patient always wants to avoid his task. It is the function of the physician to hold him to it. To this end it is important that the physician should have a wide knowledge of the meanings of symptoms and symbols and be able fairly clearly to see at once their significance otherwise he will be led into interminable and futile discussions. It is only in the final stages of the analysis, when the patient is made whole, at one with himself, that he can understand why it is that these things have come about and how it is that he no longer needs his illness, but can dispense with it. So for a long time the physician has to contend against an innate desire on the patient's part to retain the symptoms of his illness.

Various accessory forms of treatment, such as baths, massage, sanitarium treatment, travel, etc., have the same place here as with the actual neuroses mentioned before. They should never be considered primary, but only as secondary. If they are utilized without a thorough analytic understanding of the patient they are quite as apt to do harm as good; because by no possibility can it be foreseen what the results will be unless the matter which has to be dealt with is known beforehand.

It must be borne in mind that the symptoms of a neurosis or a psychoneurosis may cover and conceal a true psychosis. This will appear in the course of the analysis, but cannot always be foreseen as the result of the first examination. It is a possibility that should be borne in mind so that the physician may be guarded in what he says in regard to the possibilities of treatment and the ultimate outcome of the case.

CHAPTER XVII.

MANIC-DEPRESSIVE PSYCHOSIS.

THE general concept of the manic-depressive psychosis has been an extremely difficult one for some reason or other for many people to adequately grasp. From the earliest times the marked cases of melancholia and of maniacal excitement have of necessity been observed and in many instances ably described, and at one period of time the manias and the melancholias constituted by far the larger portion of the types of mental disorder. At that time in the history of psychiatry, when the diagnosis hair-splitting was at its height, innumerable varieties of mania and melancholia were described and given specific names. They were differentiated on the basis of whether hallucinations were present or not, whether delusions were present or not, and upon like matters of what seems now superficial observation. It was observed also that there were a certain few cases in which states of excitement alternated with states of depression. These so-called circular types have been recognized for a long time. It goes without saying that during this period when psychiatry was in a purely descriptive stage, a stage from which it has not yet by any means fully emerged, when the excitements and the depressions were the symptoms in evidence, that excitements and depressions belonging to all sorts of conditions, dementia precox, general paresis, arteriosclerosis, toxic and infectious psychoses were included in the broad concepts of mania and melancholia that were prevalent and that the formulation of the manic-depressive group has been the result of a gradual weeding out from all of these various sources of the things that belong together and the elimination of those which further analysis showed were not similar.

The manic-depressive concept, however, was not definitely formulated until Kraepelin, by a study of life histories, described the condition as a disease of affect fluctuations which might at one time manifest itself by the profoundest depression and at another time by the highest grade of excitement. Even after this formulation many thought that the term manic-depressive psychosis applied only to the so-called cases of "circular insanity" and failed to appreciate that there were inherent and fundamental relations between the two extremes of affect disturbance. It was difficult to understand how there could be any relationship which bound together cases of such unlike outward appearances and they failed to see that a patient who manifested a single attack either of depression or excitement

could be diagnosed as a manic-depressive solely because the depression or the excitement, as the case might be, presented the symptomatology of the depression or the excitement as found in other patients who presented a series of attacks of both kinds.

The present-day concept of the manic-depressive group considers it as a mental disease essentially of affect fluctuations manifesting itself sometimes by depression, sometimes by excitement, sometimes by a mixture of the two, and finally the concept has grown to include on the one hand, a manic-depressive character which tends to react on the basis of a labile affectivity, and on the other various atypical manifestations which present secondary symptoms that tend to mask the fundamental affective ones. And so the concept emerges that finds the root of the psychosis in certain character traits that never amount to pathological manifestations, and certain very mild affect fluctuations, the cyclothymias, and including certain atypical varieties with secondary symptoms that are of greater practical significance than the disturbances of affect.

Here also, as elsewhere, it is seen that the manic-depressive type of reaction merges into other types so that we get reactions that closely resemble the manic-depressive in certain phases of precox, whereas various of the depressions and excitements from other causes also closely parallel the manic-depressive types in their symptomatology. With this concept of reaction types in mind, these mergings into adjacent territories are understandable. With the concept of a disease entity, one which looks upon disease as a definite something back of the symptoms and which produces them, it is impossible to understand the meanings of these attenuated and border states.

Etiology.—In the first place there are certain hereditary factors to deal with in this class of cases as there are in the precox group. There are certain families which show a preponderance of the manic-depressive psychosis, as there are families that show a preponderance of the precox type of reaction.

In harmony with the hereditary tendencies which appear to be present in this group of cases it is found that the group may be widely differentiated into two extremes, the one in which the constitutional factors appear to be predominantly in evidence and in which the various attacks appear to originate either without any cause at all or at least without a determinable or apparent cause that is sufficient. On the other hand there is a group which appears to be more or less largely determined by causes which are apparent such as the inability of the individual to adjust to certain conditions of life, and repeated breakdowns with the return of these conditions. It is important to recognize these two groups of cases between which of course every intermediate variety may be found, because of the significant bearing which the type of etiological factor has upon the probable outcome of the therapeutic attack.

In describing this type of mental disturbance the two principal

phases—the manic and the depressive—will first be described and then the various forms of periodic psychosis of the manic-depressive group will receive consideration, while finally certain less common combinations of symptoms known as the mixed states will receive attention.

Manic Phase.—The cardinal symptoms of the manic phase of manic-depressive psychosis are three in number, namely (1) *flight of ideas*, (2) *psychomotor hyperactivity*, (3) *emotional exaltation*. These three symptoms may manifest themselves with any degree of severity, and the severity of the symptoms may vary within wide limits at different times throughout the course of the attack. The three symptoms, too, may not be all of the same degree of severity, for example, the flight of ideas may be extreme and out of proportion to the degree of psychomotor activity which may show only a slight increase.

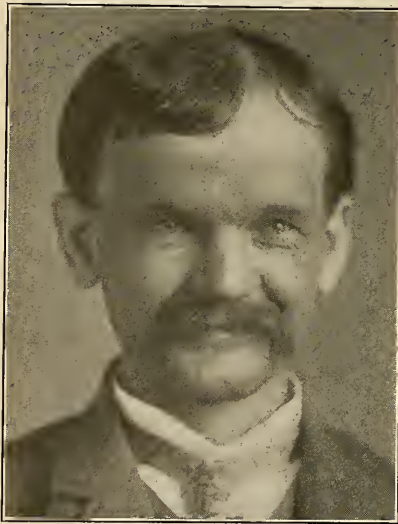


FIG. 302.—Facial expression of hypomanic with flight of ideas.

Taking the attack as a whole the ordinary acute varieties are generally designated as acute mania or acute maniacal excitement. Still milder grades are spoken of as hypomania and the more severe grades are generally termed acute delirious mania. These three degrees of excitement are the most convenient captions under which to describe the manic phase.

Hypomania, which is the mildest of these three degrees, merges of course, upon the one hand into higher grades of excitement, such as the acute maniacal, and on the other into conditions of cyclothymia, which may be termed hypomaniacal, but which constitute a special group of this class of cases which will be considered separately. In this condition, at least in the simpler cases, there is a disorder which involves the process of thinking more than it does the content of thought. The particular ideas and acts may not be unusual and yet when taken together in association with each other are seen to be abnormal. This state of affairs is excellently illustrated by the hypothetical case cited by Mercier:¹ “Its subject rises early, full of schemes of business or pleasure. He fusses noisily about the house, indifferent to his disturbance of other people’s slumbers. He is very impatient of delay, he cannot wait a minute for anything that he wants, and if

¹ A Text-book of Insanity, The Macmillan Co., 1914.

it is not forthcoming on the instant, he flies into a rage. The course of the post is not expeditious enough for him. He sends his letters by telegraph, and his letters are extraordinarily numerous. They would be numerous in any case, but their number is doubled, and more than doubled, by the frequent changes of his mind, and by the impulsiveness with which he acts upon every passing whim. He determines to make some purchase, probably a very unnecessary one, but one for which he can adduce twenty plausible reasons, and he writes to tell his solicitor that he will call the next morning. Scarcely is the letter posted when he sees that he will attain his object more quickly by asking his solicitor to lunch. He telegraphs accordingly. Before his messenger returns, it occurs to him that he had better ask the vendor to lunch also. Another telegram is dispatched, and since he cannot entertain more than one visitor at his club, another must be sent to the solicitor to announce the change to a hotel. Then he remembers that he has been drawing heavily of late on his banking account, and that he may not have the necessary funds available. Another telegram to the bank. But if there are insufficient funds in the bank, he will have to sell stock to raise the funds; another telegram to his broker. Then he determines that it will be better to pledge the stock to the bank rather than to sell it. More telegrams to the broker and to the bank. The broker won't like the contradictory orders—never mind; ask him to dinner—ask them all to dinner. Put off the lunch and have a dinner instead, and ask the solicitor, the vendor, the banker and the broker. Yes, and why not Smith and Jones and Robinson as well? More telegrams; and then, since two out of three of the invited guests decline, the whole thing is postponed, also by telegraph. Meantime, in the intervals of telegraphing, his hands have been full. He has been constantly ringing the bell and giving orders—giving them, modifying them, and countermanding them—constantly wanting something fresh, running up and down stairs, writing letters, haranguing this person and that, flying into a rage upon the slightest opposition, tearing the bell down on the slightest delay, and talking almost incessantly.”

In this example the subject's acts might, almost all of them, be considered normal, with of course the exception of those due to undue irritability or anger. Aside from this, however, each act is consistently directed to some definite aim. The disorder is not in the content of thought so much as in the process of thinking, and manifests itself by a rapid and too frequent change of direction. This is the phenomenon known as *flight of ideas*, and may be perhaps better illustrated by a stenogram:

“Do you know I was kidnapped to be sent here twice. I saw a mock funeral of me before I left home. This was done because I am a great inventor. The pope of Rome is the greatest human being in the universe. He is the head of the Catholic Church. My head (association of the word head in two different meanings) is good and

sound, and I am certainly not insane. Do you hear the ticking of the clock? (External association.) It says, 'call the little heifer, the heifer is sick.' Did you ever see the gloves veterinary surgeons use when they doctor sick cows? (Internal association.) Say! what are you keeping me here for anyhow? I want to go home. (Here he was asked how he slept at night.) I have slept excellently; that is because I am of such a strong constitution. The Constitution of the United States (association as above with the word head—probably the association is in large part at least a sound or, as it is called, a *clang association*) was signed by Thomas Jefferson. He was just a man, but he was not the inventor I am."

In this phenomenon of flight of ideas the patient either has no guiding idea or else at once loses it so that there is no consistent and sustained effort directed toward attaining a goal idea, and the thought therefore wanders here and there under the influence of chance associations. As a result the train of thought instead of progressing, changes direction, frequently returns upon itself, and never reaches a logical end. The various ideas are not on that account, however, incoherent, that is, they do not fail to cohere or to be connected with one another, although it may be quite impossible at times to see wherein this connection lies. If the associations are external, that is, originate in the surroundings, it is often quite possible to place them; when, however, they are internal, that is, originate within the patient's mind, it may be quite impossible to conceive what they are. In the example just cited, while there are many places where the connecting link is missing, probably because it was an association formed entirely within the patient's mind, still the connection can be made out in a sufficient number of instances to establish the characteristics of the train of thought. One of the principal characteristics of this type of the train of thought is, as we have seen, its great tendency to change of direction, and when, for example, this change of direction takes place under the influence of external associations, such, for example, as the ticking of the clock, as noted in the stenogram given, the phenomenon is known as *distractibility*. Any sensory impression is liable to be the starting-point of idea association, so that these patients' trains of thought may be turned at will, almost, by such devices as shaking a bunch of keys before them, saying some word loudly, showing them a newspaper, or in other words momentarily distracting their attention.

The *emotional exaltation* is also well shown in Mercier's example. The patient is constantly doing things which testify to his idea of his own importance. One is struck, however, by a symptom in the emotional field which is perhaps more fundamental than the simple exaltation and which corresponds to the symptom just cited and the motility disorders to be described, that is, the great lability of the emotions, the rapid play of different emotional reactions, exaltation giving place to irritability, to anger, annoyance, and the like. There

is no sustained emotional attitude, as there is no sustained direction in the train of thought. The psychomotor hyperactivity is also well illustrated by this case. The activity of the patient is seen to be constant and unremitting, but again it has the same qualities as the train of thought and the emotional attitudes inasmuch as it is not sustained for any length of time in any particular direction, it does not get anywhere, while from time to time, under the influence of rapid changes of emotion the acts tend to impulsiveness. There is marked *pressure of activity* just as there is *pressure of speech*, and the patient appears to be living under terrific and unremitting tension without power of direction.

In this condition, therefore, the patient is constantly active, busying himself about one thing and another, talking continuously meanwhile, often in a loud and rather boisterous manner, while emotionally, exaltation is manifested by good humor, a smiling countenance and increased self-esteem, punctuated mayhap by attacks of irritability or impulsive anger from little or no cause. His confidence in his own ability is unqualified and is shown in the outlining of all manner of schemes of work, investments, business enterprises and the like. Flight of ideas is marked, though not of high degree, the conversation changing at frequent intervals from subject to subject and the activities show a like characteristic, there being no consistent effort directed at any one aim for any length of time. Letters are often written in great numbers and their contents exhibit the same characteristics as do the speech and conduct. The patient is fully oriented, there is no clouding of consciousness nor delusions. In spite, however, of the lucidity and apparent abundance of energy the real efficiency of the individual is greatly reduced because of the lack of consecutiveness in application.

Ofttimes the picture is complicated by the addition of symptoms due to alcoholic indulgence which is very common with patients in this condition, many of whom show marked moral delinquencies, but because of their lucidity and facility of expression often elude the authorities, being at once discharged after examination when apprehended because of supposed mental disorder. This complication with alcohol will be spoken of again under the head of Diagnosis. Sexual excitement is also quite frequently and characteristically in evidence in these cases and leads to moral delinquencies which show a still further departure from the patient's normal manner of conducting himself. When complicated with alcohol the whole conduct may be quite non-understandable except to those trained in the recognition of this class of cases. The sexual excitement, of course, is an especially unfortunate and dangerous symptom in young women and may lead to particularly regrettable actions.

Acute Mania.—This next grade of maniacal excitement presents perhaps the most characteristic picture of this phase of the disease, exhibiting the symptoms to best advantage, though it must be under-

stood that the symptoms of the different grades differ only in degree, intermingle and are found alike in all the conditions.

In this degree of excitement the flight of ideas is well marked and may even become so extreme at times that the train of thought has the appearance of being quite incoherent. Distractibility is a prominent feature and the patients are constantly diverted by inconsequential happenings in their environment. The tendency to rhyme is quite frequent and the words heard by the patient spoken by those about him, although they may have no reference to him or be addressed to him in any way are often woven into or form the starting-point for these rhymes or for associations. The characteristic of these associations is their superficiality and when words that are heard are introduced into their conversation the basis of their choice is often nothing more than the sound similarity (clang association). It is quite remarkable how such a patient who is apparently paying no heed to what is going on about him will catch a chance word or phrase uttered by some one, perhaps a considerable distance away, and introduce it into the stream of his conversation. Consciousness may be somewhat clouded and there may be at least apparent disorientation, particularly for persons. This apparent personal disorientation, however, is dependent in the main upon two factors. In the first instance the patient does not adequately perceive the environment, he does not dwell long enough upon any one particular element of it to comprehensively grasp it in the rapid and transitory survey which it receives from him; its elements are not adequately perceived and therefore are often misunderstood, partly because of this superficial attitude toward the environment and partly, also probably, because of deeper reasons. Slight resemblances to friends or relatives are often seen in the patients and nurses, and these resemblances are magnified out of due proportion, and so these various persons are addressed by the names of members of the patient's family for instance. These resemblances do not necessarily result in a permanent and fixed mistake. The person who is at one moment addressed by one name is a little later addressed by another, and not infrequently the whole situation is further complicated by the wit reaction of the patient who gets a good deal of fun out of his facetious remarks and his apparently meaningless mistakes. These errors being not firmly fixed are frequently spontaneously corrected by the patient, at least at times.

The disorders of attention, flight of ideas, and distractibility are all elements which produce a transitory and a superficial survey of the environment by prohibiting any fixation or dwelling upon any particular element of the environment or even of the ego and tend to produce a condition of the content of consciousness in which all of the ideas are given the same value. No one thing is attended to long enough to enhance its importance over that of others. The patient voices ideas first about this subject and then about that, changing from one to the other, not because of any appreciation of differences of importance,

but in response to the pressure which makes it impossible for him to rest anywhere, so that all ideas tend to reach the same level of importance in his consciousness. There is what is called a *leveling of ideas*.

While hallucinations are not an essential part of the picture they may occur, but when they do, like all of the other elements, they tend to be only transitory and usually are rather simple and elementary in character.

The delusions also are inclined to be changeable. They partake characteristically, when present, of the grandiose character, but usually lack the element of extreme improbability found in conditions of dementia precox and general paresis. Occasionally we see a persecutory paranoid system of delusions develop in the manic phase of this disease, but this class of delusions is more apt to develop and present a fairly well-organized system in the milder grades of excitement.

The psychomotor activity is constant. The patients are unable to remain at rest (pressure of activity), they run and jump and turn somersaults, wave the arms about, tear up clothing, destroy plants, break furniture, howl and yell all night long, and go almost absolutely without sleep. Sexual excitement may be prominently in evidence. The excitement may be so great that the patient does not even take time to eat; food placed before him is perhaps tasted and then thrown about like everything else that comes in his way, so that with the lack of nutrition, lack of sleep, and with the unremitting activity, emaciation is a constant feature. In less-marked degrees of excitement, however, where the feeling of well-being is the controlling factor, it is common for the patient to gain somewhat in weight.

The emotional exaltation is marked and shown by boisterous laughter and remarks showing exaggerated ideas of self-esteem. Patients, however, are spasmodically apt to be irritable, bursting into attacks of anger without adequate reason and often are a constant source of trouble, annoyance and agitation upon the wards where they are confined. The emotional condition is as changeable as the trend of thought or of the direction of the activities, and emotions of radically opposed qualities may easily replace each other.

Delirious Mania.—This condition is merely an aggravated state of the acute mania already described. The flight of ideas here has proceeded to almost complete incoherence. The activity is unremitting and consciousness is more clouded and hallucinations more in evidence. The lack of sleep and proper nourishment, with rapid emaciation, soon leads to great physical exhaustion, while the constant activity not unusually results in slight wounds which, even though properly dressed, are soon exposed to infection when the patient tears off the dressing. Local areas of suppuration thus develop, there is a mild degree of infection with some temperature, which coupled with the exhaustion and the toxemia, produce a picture more distinctly delirious with marked clouding of consciousness and great incoherence.

The *acute delirious mania* which used to be described and was

regarded as always fatal, was undoubtedly in a certain proportion of cases the hypomania of the manic-depressive psychosis to which perhaps had been added, as just described, symptoms of infection, toxemia, and exhaustion, which made the picture one of delirium. Many other cases were undoubtedly also included in this general caption, more particularly deliria associated with acute diseases of the internal organs, such as acute nephritis or pneumonia. One who has had experience with these cases can understand how such conditions might go unrecognized, owing to the almost physical impossibility of subjecting such patients in their wildly excited condition to anything approaching an adequate physical examination.

Chronic Mania.—There are a very few cases that pass into a condition of chronic mania and usually, though not always, have mild excitements that may last for a number of years. These conditions, on the other hand, may be practically nothing else but character anomalies. Such prolonged phases of the disease, however, must be borne in mind as possibilities.

Depressive Phase.—Like the manic phase this phase also manifests itself by three cardinal symptoms each diametrically opposed to the corresponding symptom of the manic phase, namely (1) *difficulty of thinking*, (2) *psychomotor retardation*, (3) *emotional depression*.

This group of symptoms may, as with the manic group, manifest itself with any degree of severity, and the three symptoms may severally and individually vary, irrespective of each other. The retardation, for example, may be quite out of all proportion to the depression.

As with the manic phase, it is convenient to consider the depressive phase in three different grades.

Simple Retardation.—The word retardation is here used to refer not only to psychomotor retardation, but to the difficulty of thinking also, probably quite similar phenomena, the one more particularly in the sphere of thought and the other more particularly in the sphere of psychomotility. These patients move and speak slowly and perhaps in a low voice, by preference answering questions in monosyllables. These outward evidences of difficulty of thinking and moving are, however, more marked in the next stage of the depressive phase, that is, in acute melancholia, while here it is more usual to see the patients merely preferring to be by themselves, disinclined to associate with others, keeping to their room, and quite unable to make any mental effort. They are not equal at all to going on with their work. They may not, for example, feel equal to writing letters or even to reading the newspaper.

Emotionally these patients are usually somewhat depressed, but the depression may not be especially marked, it may only appear on questioning. Consciousness is clear and the patients are fully oriented and often have a realization of their mental invalidism.

Acute Melancholia.—In this grade of depression the three cardinal symptoms are manifested in a much more pronounced way. The

patients are characteristically inactive, sitting by themselves, showing little or no tendency to associate with others, their movements are slow and deliberate (executive retardation) and it often takes a considerable time to initiate them (initial retardation). The speech is similarly affected; it is slow, often monosyllabic, and sometimes almost inaudible. Initial retardation is noticeable here also. The emotional depression is profound and is indicated in the general attitude of the patient which is one of flexion of the body, the hands lying limp in the lap, the head inclined forward, the chin resting on the breast, and a marked facial expression of sadness. The subjective state of these patients is described by a feeling of difficulty of thinking and grasping the meanings of things and of their feeling of inadequacy, of incapacity for all effort, or even thought. There is a marked feeling of decrease in the mental activities, and the patient does not feel that he has control of his mind and can use it effectively. In the same way he feels an interference when he comes to exert his will in the performance of voluntary acts. There is a lack of energy, lack of ability to initiate or to sustain an act or a series of acts, and in the mental sphere alone the patient finds himself quite unable to carry out a series of consecutive mental acts which lead to a logical issue. He cannot come to conclusions, he has an overwhelming sense of weariness, of relaxation, of inadequacy.



FIG. 303.—Severe depression of several years' duration.



FIG. 304.—Depressive facies.

This general feeling of inadequacy and difficulty of thinking as above described fits into and forms a part of the emotional attitude and acts with it in determining the character of the delusions. The delusions are typically self-accusatory and hypochondriacal. The patients think themselves responsible for all the sin, wickedness, privation and suffering in the world; they are the cause of the unfortunate condition of their fellow patients; they themselves have committed some great sin and their souls are forever lost. As they occupy themselves with their own moral states so they occupy themselves with their bodily condition and believe themselves sufferers from incurable

disease, think that their organs are decayed, something has happened to their brains, their bowels are stopped up, their bones broken, and other such somatopsychic ideas. When the organic sensations are

altered patients have strange feelings which they interpret as indicating some mysterious thing going on within their body, and such sensations may be at the basis or associated with some of the hypochondriacal ideas. The emotional depression may at times reach a very high grade and express itself in anxiety attacks, moods of apprehension, fear of impending danger, a nameless dread of something going to happen, and the like. The whole world is looked at, so to speak, through blue glasses. The sad, depressed mood colors every perception, and so the perceptions are more or less incorrect and distorted to fit the mood.

Hallucinations may occur, but consciousness is usually clear and the patient well oriented. There may, however, be a lack of orientation toward their surroundings dependent upon the fact that they are wrapped up in their own thoughts and the environment is not attended to.

Physically there is almost always constipation, a coated tongue, indicanuria, poor appetite, loss of weight, disturbed sleep, and often circulatory disturbances with cold extremities.

Depressive Stupor.—This is the third and most severe grade of the depressive phase. In this condition the retardation, both in the field of psychomotility, and in that of thought, has proceeded to the extent that the patient lies wholly inactive and mute; he has to be tube fed, and his every want ministered to.

During this period of absolute inactivity it may be that the patient is suffering from delusions and hallucinations of a depressive and horrifying nature which perhaps are shadowed forth by an anxious expression of countenance, but the details of which can only be learned after the patient has aroused sufficiently from his stupor to be able to express himself. The hallucinations may appear to the patient much as in a dream and absorb his attention to a very great extent. This condition of stupor is not uncommon in the course of the depressive phase, but usually occurs as an episode rather than as a distinct form of the disease.

Chronic Depression.—There are certain patients who present for long periods of time a depressed mood. These cases may be mild depressive phases of manic-depressive psychosis or they may be character anomalies and so show the close interrelations between the normal fluctuations of emotion and those that are pathological.

The Periodical Types.—Under this head are included those forms of the manic-depressive psychosis which from time to time have been severally described as recurrent mania, periodic mania, intermittent mania, recurrent melancholia, insanity of double form, alternating insanity, circular insanity, etc.

All of these psychoses are merely different manifestations of manic-depressive psychosis, the manic and depressive stages being represented in various relations, often separated by a recovery interval. Thus recurrent mania would be recurrent attacks of a manic phase separated by

well intervals, similarly for recurrent melancholia, while alternating insanity would consist of manic and depressive attacks, each followed by a recovery interval; circular insanity, on the other hand, being cycles of manic and depressive phases without intervals of separation, while insanity of double form would consist of cycles of excitation and depression, each cycle followed by a lucid interval. Other varieties might be described, but it suffices to say that the three phases—manic, depressive, and lucid interval—may be combined in any possible way, and that further in a given case any degree of the manic or depressive phase may occur. It is common, too, to see during attacks of the manic phase transitory attacks of depression, while during the depressive phase it is equally common to see transitory periods of euphoria.

In a number of these cases the attacks reproduce themselves often at very definite intervals with practically photographic accuracy so that the patient leads a life the events of which can be predicted with almost absolute precision. Such patients not infrequently know some little time beforehand when an attack is coming on, and the physician may be able to see the approach of an attack the moment he steps into such a patient's room by a little difference in the arrangement of things that indicates the way matters are going.

It would seem that the patients who present such definite cycles occurring at stated intervals, each exactly like the others, belong to the group of cases with severe constitutional taint. In the other group of cases, that group in which external conditions seem to play a large part in the etiology of the several attacks, there is much less tendency to regularity in their occurrence, and as heretofore intimated, much greater hope for the results of therapeutics.

The following is the account of an intelligent woman of her feelings in both periods of excitement and depression:

"I have suffered all my life from excitements and depressions, although it was not until I was fifty-eight years of age that my family and I realized I was really mentally sick, and required institutional care. During youth and middle age my excitements were of a mild character, and during these periods I considered myself normal. I felt peculiarly happy and care-free. I managed my household affairs with the greatest ease. I entertained and mingled in society with pleasure and zest. I was lively, talkative and I have reason to believe I was witty and entertaining. I could work without an effort. I at times accomplished almost Herculean tasks. On one occasion I remember preparing and conducting a church entertainment by which the sum of \$800 was raised. Of late years my excitements have grown more severe. I begin by taking an overactive interest in everything going on around me. Everything seems rosy. I feel happy and nothing depresses me. I feel propelled by some unknown force to constant action. I am possessed with the idea of righting wrongs and straightening out things in general. All the faults in the administration of the ward, the hospital, and the government must be corrected.

"My excitements have never led me to commit any acts of violence. I occupy myself largely in talking and writing letters. My room is often in disorder because I cannot stay at one job long enough to complete it. As I feel these excitements approaching, I request the physician in charge of me to take up my parole, as I know I shall be moved to do and say many foolish things of which I will be ashamed later. No one who has not had experience can realize the mortification of having been insane.

"My depressions in early life were as mild as my excitements, the onset was gradual. I felt a disinclination to mingle in society. When forced to do so I sat like a 'dummy' and could think of nothing to say. My household duties became a burden. One after another of these was dropped until the care of the household was entirely given over to relatives or servants. I learned from experience a treatment of my own. As soon as I felt a depression approaching, I promptly dropped everything and left home for a time. I found by getting away from family cares and responsibilities, and from the demands of society, to some quiet spot, I could shorten the duration of these depressions. In recent years the depressions have appeared suddenly. One day I went to town to do some shopping for a friend. I went to a grocery store to make some purchases. It suddenly occurred to me that I could make these to much better advantage at the market only a block away. Suddenly I realized that I did not have sufficient energy to go to the market, and that another depression was upon me. It was with the greatest difficulty that I ordered the goods, paid for them and came home. At these times my brain feels paralyzed. I have not the strength or ambition to do anything. I am apprehensive lest some harm has befallen the members of my family, but to save my life, I could not write or telephone to find out if my fears are true. I have the impulse to act, but it seems as if something shuts down and prohibits action. I see my clothes becoming soiled—I know I should change them, but I cannot pull out the drawer of my bureau and get clean ones. This inertia is greater in the morning than at night. Before I came to the hospital for treatment I had servants who slept at home, and came to my house early in the morning. When my husband was away and my children were small, it devolved upon me to admit these servants early in the morning. I knew that when morning came to dress and go down stairs would be impossible. I solved the difficulty by dressing the night before and sleeping in my clothes. When the depression is most profound, I move in a fixed groove. I never vary a hair's breadth. At first I have a desire to remain in bed. Once this is overcome I have no choice but to remain up. I sit in the same seat and in the same attitude for weeks. As I come down stairs in the morning I am apprehensive lest my seat be taken, and I wonder what I shall do if it should be occupied, although the sitting room is well supplied with comfortable seats. I bring a shawl with me, and place it in the chair so that no one will appropriate it while I am at breakfast.

"After each depression, I suffer from intense pain in my back, side, shoulders and arms. This is dull and aching in character, and remains with me for weeks after the depression has disappeared. After the last depression I suffered from a severe attack of the shingles. The skin eruption has now disappeared, but the pain still remains."

The Cyclothymias.—This group of cases presents the mildest excitements and depressions. They deviate less from the normal than the other groups and are only considered separately because of their great practical importance. They are quite usually not recognized and the symptoms are attributed to all sorts of things other than the real trouble. It must not be lost sight of in considering these mild manic-depressive fluctuations that a slight depression may recur without the psychosis expressing itself by a fluctuation to the opposite condition of excitement, and *vice versa*. So that the picture is seen of patients presenting from time to time mild degrees of depression or mild degrees of excitement without anything approaching delusional formation or disorders of the sensorium and therefore attracting no particular attention from the mental side. The following example illustrates this exceedingly well: He is a man who devotes himself largely to literary work, and the fluctuations in his mental state are shown excellently well by his ability to write. The onset of a depressive phase is usually shown by a gradual, though more or less rapid, falling off in his literary ability. He is first unable to compose, then he gets progressively less able to write until he is only able to write the simplest things. It is the same way in his reading. He gravitates all the way from reading connected with his work down through the different grades of literature until he gets to fiction. He finally finds himself quite incapacitated, sitting for hours gazing out of the window or at a blank wall, and while rather enjoying company, it is almost impossible for him to initiate the procedure that is necessary to go anywhere. He finds it almost impossible to dress, to get out, to take the cars, and the like. This state is one almost entirely of retardation without marked emotional depression. During the opposite condition of affairs he has a feeling of well-being and efficiency in marked contrast to his feelings during the depressive period, and finds himself quite able to work for long intervals very effectively.

The remarkable transitions from phase to phase are shown well by one of his experiences. One day, having been writing all of the afternoon, he, as usual, went out to dinner, leaving his papers on the table, intending to resume work on his return. When he came back and took up the pen to write he found that the incubus of his depression was upon him. He had difficulty in finding words and finally after two hours' effort he gave it up. This was the beginning of a depression which lasted about a month. During this time he constantly tested his ability for composition, but without favorable result. Almost exactly a month after this incident he undertook to answer some personal letters, intending to write only short letters of perhaps three

or four pages, but when he started to write them he found himself writing easily and his letters spontaneously expanded to eight or twelve pages and he went on into his work again.

The *hyperthymic types* show exaggerated activities in the way of the usual business occupations, writing letters and the like. The judgment is apt to be rather poor at these times and many of the business ventures come to grief, though not necessarily so. Work is easily done, without having made effort, and the patient expends enormous amounts of energy over long periods of time. Certain types of cases are meddlesome and troublesome, tending to engage in disputes and altercations, and to bring law suits, while exaggerated eroticism and alcoholic predilections oftentimes very considerably color the picture.

In the *dysthymic types* are found the depressions which are attributed in large part to neurasthenia and to various visceral disturbances.

These cyclothymic cases not infrequently show fluctuations at periods of recurrent physiological activity such as the menstrual period, while it must never be lost sight of that not a few so-called dipsomanias are really recurrent manic-depressive attacks in which the alcohol is resorted to shortly after the attack commences and then quite usually all the symptoms from which the patient suffers are attributed to the alcoholic indulgence. It is important to bear this class of cases in mind, not only for diagnostic purposes, but in order that the patient should be dealt with fairly as a sick man.

Perhaps the most important of the disturbances in this group of cases are the visceral disturbances. There are a large number of conditions, particularly the false gastropathies, enteropathies, cardiopathies of Dejerine, etc., many of which belong here. Inasmuch as the psychosis is not recognized, these conditions are quite naturally credited with being the cause of the condition of the patient. Patients with mild depression are called neurasthenic, those with mild excitement are called nervous, and the accompanying physical condition is credited with making the trouble. The patient and the relatives consistently take this attitude and the physician naturally falls into it. No one wishes to acknowledge the possibility of a mental disorder, and therefore these other explanations are readily accepted. In fact, the condition is hardly recognizable at its true value, even by the practised observer, unless a full account of the patient's life is available.

After a while the symptoms of visceral disturbance clear up along with the disappearance of the mental symptoms, and the change is attributed to some form of treatment, a special dietary regime, or whatever has been resorted to for the relief of the symptoms. Here is a group of cases who during their attacks regularly seek the specialist and are subjected to all sorts of gastro-intestinal treatment, gastric lavage, special dietaries, gynecological manipulations of one sort and another, metabolism experiments, treatment for auto-intoxication, eye-strain, and almost everything in the category of medical specialism,

and yet characteristically in these cases nothing is found in the physical condition that adequately accounts for the symptoms. Another group of these cases are the paranoid types. These patients present typical paranoid symptoms with emotional accompaniments that seem to be hardly in excess of what is demanded as normal reaction to the delusional state. This is the group of cases that has given origin to a great deal of recent discussion with regard to the real basis of paranoia, its relations to manic-depressive psychosis, the affective origin of paranoia, and its basis in what Specht calls the "affect of suspiciousness."

The Mixed States.—The mixed states are forms of manic-depressive psychosis in which the three cardinal symptoms of the manic and depressive phases are mixed so that the resulting state is neither one. They are: (1) *maniacal stupor*, (2) *agitated depression*, (3) *unproductive mania*, (4) *depressive mania*, (5) *depression with flight of ideas*, (6) *akinetie mania*. It will suffice to merely mention the symptoms of these groups.

Maniacal Stupor.—Emotional exaltation, decreased psychomotor activity, difficulty of thinking.

Agitated Depression.—Emotional depression, increased psychomotor activity, flight of ideas.

Unproductive Mania.—Emotional exaltation, increased psychomotor activity, difficulty of thinking.

Depressive Mania.—Emotional depression, difficulty of thinking, increased psychomotor activity.

Depression with Flight of Ideas.—Emotional depression, flight of ideas, decreased psychomotor activity.

Akinetic Mania.—Emotional exaltation, flight of ideas, decreased psychomotor activity.

Still the possibilities are not exhausted. It is quite uncommon to see any one of the conditions already described continue pure from the commencement to the end of the attack. In the manic phase symptoms of depression not infrequently crop up and occupy the field temporarily, while during the depressive phase it is quite as common to note transitory periods of excitement. Then it is quite common for manic attacks to be preceded by a longer or shorter attack of depression, and sometimes such a period of depression follows, not infrequently but partial depression, of the type of unproductive mania. The depressive phase shows similar variations, more particularly it is followed by a short period of exaltation. Then, again, at any stage of the disease a mixed state may crop up for a time, so that we may see during the course of the manic phase psychomotor retardation occur or during the phase of depression emotional exaltation may develop, while in the various forms of the periodic psychoses it is quite the rule to find these mixed states at the transition places from one phase to the other, all of the symptoms of one phase not equally and contemporaneously graduating into their opposites. Thus during

the course of a circular psychosis the affect may change from depression to exaltation before the psychomotor retardation has given place to increased psychomotor activity, thus producing a temporary mixed state.

Involution Melancholia.—The group of cases comprised under the term involution melancholia, which was originally used by Kraepelin, has now been pretty generally conceded to belong to the manic-depressive group. The characteristics of the disease are those of an anxious depression occurring in later life. The group is such a considerable one and of such practical importance, however, that it will be specially considered along with other depressions of later life in another chapter. (See Chapter XXIV.)

Pathology.—There are no specific pathological findings in this psychosis, although certain degenerative products have been described in cases of death from depressive stupor. Patients, however, characteristically recover from this condition, or if they die during attacks, the death is due to some intercurrent disease which itself would produce changes in the nervous system that would gradually cloud and perhaps entirely obscure any pathology that the psychosis might have. A condition producing death itself must produce serious alterations of the central nervous system, that most sensitive of reacting portions of the human body.

Nature of Manic-depressive Psychosis.—This psychosis perhaps, as thoroughly as any other, has withstood throughout the years any attempt at understanding it, while as opposed to dementia precox the symptoms of which appear quite unpsychological, the symptoms of the manic-depressive psychosis in either one of its phases, more particularly perhaps in the depressive phase, are quite psychological, that is, quite understandable. The patients, to begin with, present largely normal types of personality before the advent of the psychosis, and during the symptoms of the psychosis they ordinarily are not so far removed in their conduct or in the character of their ideas as to place them, so to speak, in a class by themselves. They are still quite like the rest of us. The roots of the psychosis appear to spring more distinctly from the normal life, the fluctuations of the emotions being quite comparable to the fluctuations that occur in everyone.

However, it seemed quite impossible to understand how the patients could vary from one extreme to its diametrical opposite and what could possibly be the explanation of such shifts of position. For many years, under the domination of Meynert, the changes were supposed to depend upon changes of blood supply, upon anemias and hyperemias. When psychiatry, however, advanced beyond such cross types of explanation it was left practically without anything to fall back upon. Recently, however, the suggestive work of Bleuler¹ has seemed to indicate what may at bottom be the true explanation. He has demonstrated what

¹ The Theory of Schizophrenic Negativism, Nervous and Mental Disease Monograph Series, No. 11.

he calls the ambivalency of ideas. This ambivalency gives, as he understands it, to the same idea two contrary feeling tones and invests the same thought simultaneously with a positive and a negative character. Along with this ambivalency there is an ambitendency which sets free with every tendency a counter-tendency. With this basal supposition it can be understood why the fluctuation of the manic-depressive is a fluctuation between conditions which are diametrically opposed. If each idea has associated with it by preference the idea which is absolutely its opposite, if each feeling has associated with it by preference the feeling which represents its exact antithesis, then there is reason for understanding how the manic-depressive gravitates between these two conditions. It is the path of opposites which is met with at every turn in psychiatric experiences. Nothing suggests white more easily than does black, nothing suggests love more readily than hate. The opposed ideas and feelings stand with relation to each other in the path of least resistance, and when one would go from a certain idea or a certain feeling in any direction he finds the path to the antithetical idea or feeling more easily passible than the path to any other goal.

Assuming the hypothesis of ambivalency and ambitendency, still what is the explanation of the affect fluctuations in this psychosis? Here as elsewhere in the mental field some fundamental psychic conflict undoubtedly has to be sought to which the patient is making efforts of adjustment. This is precisely the starting-point from which, for example, dementia precox has to be viewed. But here are individuals who present a different possibility of reaction, a different reaction type to the conflict than do the precox patients. This statement, of course, must not be taken as meaning any more than a mere putting into words of what is found, because it is not understood what the differences are that make different people react in different ways. Psychiatrists are only upon the verge of being able to ask such a question intelligently. They are not yet able fully to answer it.

The manic-depressive psychosis, however, may be conceived of as an effort at compromise and at defense, resulting from an endopsychic conflict. In the depressive phase the affect has broken through and invades consciousness, while in the manic phase the patient by feverish and restless activity, by a constant alertness, fights off every approach that might touch him on a painful point, that might reach a vulnerable spot. It would seem as if he were wildly beating about to keep off all intruders, not only real intruders, but all possible, prospective, or thought-of intruders. And so the manic patient is already quite inaccessible and all of his reactions are especially superficial, as witness the word associations and the clang associations. He moves over the surface, which he endeavors to cover completely in order to prevent penetration at any point.

This is only a general statement of what may or may not be found by further study to be the case, but already certain indications

are known that give some hope that the near future may unravel the meanings of manic-depressive reaction. Abraham¹ particularly has worked with this group and lays special stress upon the resemblance between it and the obsessional neuroses. The relationship to hyperthyroid states has already been discussed in the chapter on the Internal Secretions. (See p. 147.)

Course and Prognosis.—The individual attacks vary in duration from a few days to several months. Recovery from the single attack is the rule, while the likelihood of subsequent attacks is considerable. In general, therefore, the prognosis is good for the separate attacks and is rather poor as to ultimate freedom from attacks. As pointed out previously, the severe constitutional types have a worse prognosis than those types in which the etiological factors are capable of removal. In the severe constitutional types also the recurrent attacks tend to repeat with photographic accuracy preceding attacks, while in the long run the general tendency is for an increase in the length of attacks and a decrease in the length of the free intervals.

The disease pursues its course without any special tendency to deterioration. Although mild grades of dementia have been described, terminating protracted attacks, the dementia which usually supervenes in the course of the disease is that which is superadded as the result of the changes incident to arteriosclerosis and the senium. Inasmuch as this psychosis tends to recur throughout life, not a few of the patients ultimately reach senescence.

Differential Diagnosis.—The manic phase in its mildest forms is often mistaken, especially where it leads to alcoholic and sexual excesses, for a form of moral obliquity. In the somewhat more pronounced attacks it may be difficult to differentiate it from other excitements, more particularly catatonic excitement. In general the manic excitement is more free and open, there is less tendency to constraint along any particular line, while the productivity and the psychomotility are not as meaningless or non-understandable as with dementia precox.

The depressive phase in its milder manifestations is not infrequently mistaken for neurasthenia, and in its more pronounced form it is extremely difficult to differentiate it from the depression of dementia precox. This is particularly so because the feeling of inadequacy of the manic-depressive is very close to the empty feelings with loss of affect of the precox, while the blocking of movement and expression in the latter condition outwardly closely resemble the retardation in manic-depressive psychosis. The stupor of catatonia outwardly also closely resembles the manic-depressive stupor, except that it is more apt to be associated with marked negativism, muscular tension, and perhaps grimacing.

The greatest difficulty, as between manic-depressive psychosis and

¹ Psychoanalytic Investigation and Treatment of Manic-depressive Insanity and Allied Conditions, *Zentralblatt für Psychoanalyse*, vol. ii, No. 6; abstracted in the *Psychoanalytic Review*, vol. i, No. 2.

dementia precox, lies with the differentiation of the mixed states. Here the resemblances are quite close and prolonged observation is often necessary to make the differentiation.

It must not be forgotten that the manic-depressive psychosis is by no means a clear-cut definite entity, that it merges in all directions into other conditions, and that its closest affiliation with the other psychosis appears on the surface to be with the dementia precox group. There are quite a considerable number of cases in which a study of the individual attack leaves one in doubt as to which group to place the patient in, manic excitement, for example, being associated with certain catatoniform symptoms, catatonic excitement presenting a fairly typical flight of ideas and the like. In general the principle of differentiation is first, the history of previous attacks, and next to this history of repeated attacks is a history of attacks of both manic and depressive character occurring in the individual and showing no marked tendency to deterioration. Practically, however, it is quite impossible to make a differentiation in many cases and patients that are at one time placed in one group, for example, in the manic-depressive group, are found later to probably belong in the precox group, because, for example, of the appearance of deterioration. And so the history of the diagnosis in these two groups shows a constant tendency to fluctuation, at one time the dementia precox group being enhanced by cases which at another time are placed in the manic-depressive group. And so the situation fluctuates back and forth, the best evidence of an inherent relationship between the two.

The most important group from the practical standpoint is the cyclothymic. These patients are practically always wrongly diagnosed at first and often over and over again for considerable periods of time. Most generally, as already mentioned, they fall into the hands of the specialist under the theory of some disorder of the internal organs. When an attempt is made to really understand these patients one is impressed with their close resemblances to the obsessional neurosis. It may be quite impossible, at least by any means other than a very careful and detailed study of the patient, to differentiate between the two conditions, and from the discussion on the nature of the manic-depressive psychosis it will be seen why this similarity exists. It should be borne in mind also that it is not difficult to confuse mild cyclothymic attacks with the anxiety neurosis.

It should, of course, be added that one must be careful and not confuse excitements and depressions that may have other origins as, for example, particularly paresis, the symptomatic and toxic psychoses.

Treatment.—There has been the general feeling in years past about this psychosis that the attacks were self-limited. This has probably been dependent to no small extent at least upon the extreme regularity of the attacks in certain patients. These patients, however, are the patients with the more profound constitutional taints, and it is perhaps generally true that in this class of patients attacks do tend to run a

regular course, each attack being approximately of the same duration as the former similar one. With the more frankly reactive types, however, this becomes progressively less true, so that the matter of treatment here easily becomes something more than mere intelligent custodial care.

In the very mild types of the disease the patients have to be carefully safeguarded, because their condition is not appreciated by others. In the excited phases alcoholic and sexual excesses are especially apt to occur, and it must never be lost sight of that during what outwardly appear to be the mildest depressions suicide is a possibility.

In the more pronounced attacks the handling of the patients calls for the very greatest amount of tact. During the excitement the patient's strength must be guarded, as insomnia is a constant symptom, and food may be taken in insufficient quantities. Mechanical restraint should, of course, be avoided if possible and it may be said that it is practically never necessary. Its application in the high degrees of excitement is often not understood by the patient, and produces an increase of excitement and resistance, and perhaps a state of anxious apprehension, and even terror. Chemical restraint is equally undesirable as it tends to shut out the real world and thereby increases the difficulty of adjustment to reality. Hypnotics may be necessary and such simple ones as veronal are the best—paraldehyd, trional, sulphonal, chloralimid are useful, but opium and its derivatives should be avoided if possible. Whereas the various kinds of restraint are highly undesirable, isolation may be resorted to and is not infrequently welcomed by the patient. All that may be necessary is simply to put the patient in a room by himself without locking the door. They may be very glad to stay there and so escape from sources of irritation.

It is in the condition of excitement that the continuous bath is so valuable. The patient is placed in a tub, preferably one constructed especially for the purpose, being long enough to permit the body to lie in it without the limbs being flexed. The water, which should cover the body completely, is kept at a temperature of from 96° to 98° F., that is, just above the normal surface temperature. It adds to the comfort of the patient if a canvass hammock can be slung in the tub on which he can lay, and a rubber air pillow be placed under his head. The warm water of the continuous bath is the best sedative treatment we have for this class of patients, as it produces sedation without any clouding of consciousness or other disagreeable features. The patient may be left in the tub for such a period of time as is deemed necessary, usually three or four hours at a time. On the Continent patients are not infrequently kept in the tub not only for days, but even months, sleeping in the tub and being fed in it. On the whole the patients enjoy this, the warm water is soothing, and they are grateful for its calming influence.

It is in the excited phases that the nurses ingenuity will be taxed to the utmost, and if she is not tactful all sorts of artificial symptoms

will be created in the way of antagonisms toward the nurse, increased irritability, etc., so that the adjustment of the nurse to the patient, particularly in excited conditions, becomes an important practical problem.

In the depressed phase of the disease the patient is often best treated in bed, particularly if the depression approaches the stuporous stage. Under these circumstances refusal of food is a common symptom and tube feeding must be resorted to at regular intervals. With the tube feeding it is easy to give such medicines as may be required, either hypnotics, cathartics, or anything else indicated. One must remember that in endeavoring to probe the consciousness of depressed patients the depression may be increased and if suicidal tendencies are present this should be borne in mind and guarded against.

If the patients are stuporous the usual precaution should be taken to see that the position of the body is changed from time to time and that the bladder and rectum are not permitted to become overloaded.

The danger of suicide in depressed conditions is an ever-present one. The only safe way to deal with these patients is to assume that they are all suicidal. Probably one of the reasons why more patients in this condition do not commit suicide is because of the marked retardation which makes it so difficult for them to initiate any form of activity. It is the depressed patients who are suicidal who most frequently require to be sent to a hospital, although their general condition may not seem to warrant such a move. The watching and the caring for a depressed patient with a view to preventing them from committing suicide is practically only understood in institutions for the treatment of mental disease. The general hospital nurse, the general practitioner and the family rarely have any idea of the degree of watchfulness that is necessary and for this reason alone oftentimes the patients must be sent to institutions.

During the period of convalescence, occupation, outdoor exercise, and the like are all in order. Care should be taken not to force the patient too fast.

Here as elsewhere in mental medicine an attempt should be made to analyze the mind sufficiently at least to understand the nature of the disturbing factors that are at work, and if possible the way in which they have brought about the psychosis. This, of course, is essential to an intelligent treatment of the patient. Such analysis, however, is almost impossible with many patients, particularly during the attack, and can only be resorted to when the patient is at least approaching the normal condition. All the information gained, however, is valuable as pointing the way toward regulating the patient's life and in many instances as indicating the nature of the etiological factors and thereby showing what must be avoided in the future if further attacks are to be prevented. Of course much more is to be hoped for in the frank reactive than in the profoundly constitutional types.

Prophylaxis.—Prophylaxis resolves itself into two parts: first, the prevention of the disease, and second, the prevention of subsequent attacks after the disease has manifested itself. The prevention of the disease is a problem of eugenics. We need very much more information as to the way in which the manic-depressive psychosis conducts itself with reference to the laws of heredity. It has not as yet been adequately worked out so that definite advice is possible.

As to the prevention of subsequent attacks the most important thing is to prevent if possible the recurrence of the etiological factors that have been found to play a part by the psychoanalytic study of the patient.

CHAPTER XVIII.

THE PARANOIA GROUP.

PERHAPS no term in psychiatry has undergone wider variations of meaning than the term paranoia. In its earliest days, in the Greek period, it meant little more than just craziness, although perhaps it may have been used somewhat more specifically in some instances, and later on, well into the middle ages, it was still a term that was not infrequently used to include the whole group of the so-called insanities.

The term did not come into general use as applied to a special grouping of mental symptoms until the early part of the nineteenth century, when a German psychiatrist, Heinroth, made an effort to classify various mental diseases and gave paranoia a distinct placement in his scheme. The classifications of this day, however, were extremely complex and there was a marked tendency to follow the dichotomous method with its binomial nomenclature, which had come into such popular vogue with the appearance of the work of the great Swedish botanist, Linnaeus, in the middle of the eighteenth century. A disease that was classified under the specific names of hallucinatory, confusional, depressed, or what not, might change its name and its nature over night, as it were. This led to great confusion and to the final throwing out of the whole scheme by the French, under the leadership of Pinel, who reduced the classification to manias, melancholias, and dementias. Esquirol followed with his monomania, under which the paranoias found a place, and this term has been in use ever since, largely by the English school, and it still finds application in the courts. It is based upon the simplistic conception that the brain is one organ and that it has one disease, and that disease is insanity, and not only simplistic to this extent, but that the disease may affect any part of the organ and therefore a person may be insane upon one subject, conceptions which are hardly worthy of a school boy, but yet are still held in some quarters today.

From this time on the general concept of paranoia became somewhat more definite and it tended more and more to concentrate and crystallize itself about a condition which presented essentially delusions, more or less clearly formed and of a persecutory type generally associated with hallucinations, especially auditory. Even this concept, however, included such a mass of material of such dissimilar types that it never became very well defined. The paranoia controversy during this period, namely from the middle to the end of the

nineteenth century, is largely taken up with a discussion upon the basis of the old faculty psychology, and the division of the mind into three parts, the intellect, the emotions, and the will. There had already been a tendency to consider paranoia as a primary intellectual disorder. The discussion took up the question as to whether the emotions were involved primarily, secondarily, or at all. This whole bootless procedure, based upon the faculty psychology, of course came to naught, simply because the mind is not split up into mutually exclusive compartments. Another one of the concepts upon which such discussions were based, and which was assumed in the discussion, was that there were such things as mental disease entities which had as much individuality and definiteness in the conceptions of the psychiatrist as tumors had in the minds of the pathologist.

Without going into a description of the different ideas of paranoia which have been extant, and which in their later development will be described in the body of the chapter, it need only be added that the general result of all this discussion is first that the brain is not a single organ. It is a great number of organs crowded into a very close space and that the functions of its different parts need be no more closely related to one another than the functions of the adrenal glands and the hypophysis. The cortex alone consists of at least fifty histologically differentiated organs, while the thalamus is composed of at least nine ganglia. The red nucleus is an organ by itself, as are the different portions of the lenticular nucleus, and the separate ganglia innervating the ocular muscles. In some way or other there issues from this complex of organs, or more properly is associated with it, the phenomena of mind. Mind is not a single thing any more than is the brain. It is not only as complex as the organ which subserves its function, but infinitely more complex than this organ as we know it today. The mind cannot be conceived as divided into compartments like the will, the intellect, and the feelings, each presided over by a mythological demon, so to speak, but must be conceived of as a complex of adaptive mechanisms interrelated with one another in the most intricate manner, so that the mind must be conceived of as capable of having not only one, but many kinds of disorders, which disorders are not entities in the sense of foreign bodies or diseases which enter from outside, but are inefficient ways of functioning, special combinations of mechanisms, and so we have not so much mental diseases after all as types of mental reaction. The disease is not, therefore, something which comes from without, but it is a function of the interrelation between the individual and his environment, and only in proportion as this interrelation is inefficient may it be conceived of as disease, and only in accordance with the type of mechanism which is utilized, the special trend of reaction, can we speak of a disease in any specific sense at all. This is quite parallel with the concepts on the physical side and is important to bear in mind if we are not to be enslaved by a limiting terminology.

The most recent advances, therefore, in the concept of paranoia are, a getting away from the consideration of it as a disease entity, or as involving a special faculty of the mind, or as a merely mono-symptomatic classification, and a coming to consider it as a type of reaction which manifests itself in certain individuals, probably as the result of certain specific types of noxa. The descriptive attitude toward the problem is being replaced by the interpretative. This is significant, as paranoia has long been the stronghold for descriptive psychiatry and it has been the last to yield to anything like an interpretative approach.

Description.—The general concept of paranoia which has been prevalent for many years is that of a psychosis presenting delusions of persecution of a pretty clearly defined type, well supported and defended by the patient, in other words systematized. These delusions generally involve a more or less circumscribed portion of the mentality, although they tend to spread out slowly and involve more and more. With this state of mind there is no marked tendency toward deterioration, the disease having essentially a chronic course and remaining unchanged for years. Associated with the delusions and harmonized with them in content are frequently auditory hallucinations—voices.

This is the general concept of the disease which has received various modifications and descriptive clothings by different authors. For many years the *délire chronique a évolution systématique* of Magnan in France and the paranoia of Krafft-Ebing in Germany have been the paradigmata under which the various forms have been arranged. The *délire chronique* of Magnan was a disease which progressed regularly through four stages: first, a hypochondriacal stage, or stage of subjective analysis; second, a stage of persecution; third, a stage of transformation of the personality; and sometimes, fourth, a stage of dementia.

In the first or the hypochondriacal stage, or stage of subjective analysis, the patient is self-centered and depressed and has ideas of reference. He also complains of many physical symptoms, such as dizziness, weakness, headaches, etc. Everything that happens about him tends to be referred to himself, so that he is in a constant state of morbid introspection about things which he does not understand. In the second stage the explanation of all these things finds itself in the delusions of persecution. The reasons why people have slighted him, why they have said disagreeable things about him, why they talk about him and spread rumors about him is all understandable because of the conspiracy which there is against him. These delusions are reënforced often by hallucinations of hearing, and he hears actual evidences of all of the things which are being done to annoy, to persecute, or to destroy him. Explanatory delusions follow which give the reasons to the patient why he is thus persecuted, and usually attribute the persecution to some special person or group of persons, or some society or institution. In this persecutory period the patient

may be very dangerous. He may react upon his persecutors if he knows who they are, that is, if he has defined them in his own mind. On the other hand, he may develop elaborate defenses against them or may endeavor to flee from them entirely. Later on, as a result of the elaboration of the whole system with the effort at finding further explanations for them all, there are developed the ideas of self-importance. The patient concludes that if so many people are leagued against him that he must indeed be an important personage. When this occurs we are in the face of the third phase of the disorder, namely, the transformation of the personality. By this time the delusional system has become extremely complex, with interminable ramifications in all directions involving all sorts of occurrences, all manner of people, and reaching backward into the past and finding delusional explanations of events even in the patient's childhood. These explanations often refer to facts which antedate the period of the psychosis, and are therefore known as retrospective falsifications. Following the transformation of the personality there may occur a certain amount of mental enfeeblement.

This, in a few words, is Magnan's *délire chronique*, a disease which is extremely rare if this gradual evolution of the several stages described by him is looked for; that it does occur from time to time cannot be disputed. In all probability a number of cases of paranoid dementia precox were included in this group, while today the group has been recognized in a much restricted sense by Kraepelin¹ in his *paraphrenias*.

Krafft-Ebing, in Germany, developed a picture of the disease which for many years had much vogue in this country, and found particular favor, largely, probably because of its easy applicability. In the first place he divided all paranoidias into two great groups, the *original* and the *acquired*. The original paranoidias, after Sander, were those which presented their psychosis as the natural unfolding of an abnormal character, while the acquired or *late paranoidias* were the paranoidias which developed in people who up to a certain point had appeared normal. A further subdivision of these groups was into *paranoia hallucinatoria*, in which hallucinations played a prominent part, and *paranoia combinatoria*, in which they were absent or of minor importance. Following these great groups there were secondary groups which stood upon a purely symptomatic basis, and so he described *persecutory paranoia*, *expansive paranoia*, *querulous* or *litigious paranoia*, and *inventive, reformatory, religious, and erotic types*.

More recently there has been a tendency to gradually restrict the paranoia group to narrower and narrower limits. One of the most recent attempts at more clear definition is that of the French authors, Sérieux and Capgras,² who have described two types of psychosis

¹ *Psychiatrie*, 8th Auflage.

² *Les folies raisonnantes*, Paris, 1909; and *Diagnostic du délire d'interprétation*, *Revue de Psychiatrie*, January, 1908.

to which alone they think the term paranoia applicable, namely, the *delirium of interpretation* and the *delirium of revindication*. In the *delirium of interpretation* the patient has ideas of reference, and because of his lack of critique and egocentricity comes to all sorts of false interpretations of what is going on about him. These delusional interpretations become systematized and reach more or less coherency without any special dependence upon disorders of the sensorium. There is no deterioration, and lucidity is maintained throughout the evolution of the psychosis. Unlike certain other paranoid conditions the false interpretations have their origin in actual facts.

In the *delirium of revindication* a chronic systematized psychosis which takes its origin in a fixed idea appears. It is a monoideism, and its various ramifications, like the other form of paranoia, do not tend toward dementia. They describe two varieties of this psychosis, the *egocentric type*, the subject of which are usually persecutors making claims for wrongs suffered that may or may not have some foundation in fact. Then there is the *altruistic type*, characterized by abstractions and impersonal theories. To this group belong the inventors, the reformers, and the prophets, becoming, however, in their endeavor to realize their ideals, oftentimes dangerous fanatics, mystics, anarchists, regicides.

In Germany Kraepelin limited the paranoia concept perhaps more than any one else. He confined the term to a very circumscribed and very small group. His conception of the disease is a chronic incurable psychosis of insidious origin developing slowly by the gradual systematizing of endogenous delusions. This system of delusions is enduring and unshakable and exists along with the retention of the logical and orderly process of thinking. There is no marked tendency to mental deterioration, and hallucinations play no essential part in the picture.

Kraepelin has recently, in the eighth edition of his work, still more clearly defined his paranoid group by describing a group, *paraphrenia*, which contains certain paranoid types that closely resemble his paranoia, but which provisionally he includes in this group for purposes of greater definition. This group of *paraphrenia* is divided into four sub-groups, as follows:

Paraphrenia systematica, which is for the most part Magnan's *déire chronique*, with the exception that the well-defined precox types with marked deterioration are excluded.

Paraphrenia Expansiva.—This form affects only women and is marked by the development of ideas of grandeur with mild excitement and exaltation. No dementia follows.

Paraphrenia Confabulans.—Here the delusions both of persecution and grandeur are specially marked by their foundation upon and reference to memory falsifications, as the name indicates.

Paraphrenia phantastica is the term applied to cases with a certain amount of exaltation, with the recounting of remarkable adventures

and incoherent changing delusions. This condition is characteristically accompanied by hallucinations of hearing. This group includes the cases previously described by him under the term dementia paranoides.

Interpretation.—The rather simplistic attitude which dominated shortly after the term paranoia came into general use and which saw in every combination of fairly well-defined and fixed persecutory ideas, especially those supported by hallucinations of hearing, the disease paranoia had to give way in a very few years to a broader, if somewhat less well-defined, attitude. It soon became evident that a fairly systematized and fixed delusional system of persecutory character might occur as the expression of a psychosis from which recovery took place. And so the element of the concept of paranoia which considered it as essentially chronic, progressive, and incurable had to be readjusted. These so-called acute paranoidias have been recently studied quite extensively by Friedmann,¹ and their origin traced to actual situations in the patient's life, so that the delusions appear as logical outgrowths of experience, and have as a consequence fallen into the group of the psychogenic psychoses.

Not only was the idea of chronicity associated with paranoia seriously shaken, but from other sources the idea of the specificity of the persecutory delusion also had to give way, for it was soon found that ideas of persecution of paranoid character were not at all infrequent in connection with other psychoses. This was particularly evident in the psychoses of chronic alcoholism. It soon developed that there was a special form of dementia precox presenting paranoid ideas, while later studies showed luetic forms with paranoid symptomatology, presenile forms, paranoid states of mind of the deaf, and others who are isolated from close contact with the world, to say nothing of the recent paraphrenia group of Kraepelin and many other less well-defined conditions, which have included more recently not only the manic-depressive psychosis, but certain of the milder cyclothymic manifestations of this disorder.

From these considerations it appears that here, as elsewhere in the field of psychiatry, that the important thing to consider is not so much the special content of the particular psychosis in a given individual as the mechanisms which are involved, for here we see a similar content in all sorts of mental disorders, some acute, some chronic, and are therefore forced to look beneath and see whether it is not possible to understand these manifestations by attributing them to a common mechanism.

The studies of Friedmann, already mentioned, went a long way toward showing the dependence of paranoid trends upon actual situations in the patient's life, and demonstrated how paranoid delusions in given cases might grow as a result from these situations. In other

¹ Contributions to the Study of Paranoia in Studies in Paranoia, Nervous and Mental Disease Monograph Series, No. 2.

words, they are of psychogenic origin and are perfectly understandable when all of the circumstances have been uncovered.

Among others, Gierlich¹ has shown that paranoid ideas often accompany fluctuations of affect which could only be considered as manifestations of a manic-depressive psychosis and that many of the paranoid conditions which were associated with only slight affect manifestations which belonged to the manic-depressive psychosis might easily be overlooked as coming under that group and be mistaken for true paranoia. In this way he accounted for a very large number at least of the so-called acute paranoidias, as these patients of course got well from the attacks as the manic-depressive cases usually do.

More recently Specht² has at great length endeavored to demonstrate that the underlying condition in paranoia was an affect of suspiciousness and therefore he brought the paranoid group into close alliance with the great affect group of the psychoses, namely, the manic-depressive group. This whole discussion has broadened out in all directions and has become very complex and extremely involved, and therefore it is not a proper subject for further elaboration in a text-book. It might be added, however, that Blueeler,³ who has made a most incisive study of the psychology of paranoia, denies absolutely that suspiciousness is an affect at all, and therefore departs radically from Specht's position. He believes suspiciousness is a state of mind based entirely upon perceptions and the resulting conclusions, and is therefore of purely intellectual origin, but that it is accompanied by affect, as are all mental states. Here, again, the fallaciousness of the old faculty psychology that would separate the mind into different parts, such as the intellect and the emotions, should be emphasized. The two invariably occur together, and suspiciousness of course, therefore is accompanied by its affect.

Blueeler is of the opinion that paranoia takes its origin in certain constellations of ideas or complexes and the dominant affect with which they are loaded, that these complexes are precisely of the same nature as are found in normal individuals, and that the abnormal element which leads to the elaboration of a psychosis is the fixation upon this complex, the inability to get away from it, or as might be said, the inability to reach an efficient adjustment to it.

One here sees what is everywhere apparent in dealing with mental disorders, that the delusion is not the disease, the delusion is only one expression of the disease. The mechanism involved has to deal with a certain content; this content is delusional, but is therefore only the outward expression of the disorder beneath. Therefore the delusion really expresses an effort upon the part of the individual to

¹ Periodic Paranoia and the Origin of Paranoid Delusions in Studies in Paranoia, Nervous and Mental Disease Monograph Series, No. 2.

² Ueber den pathologischen affect in der chronischen Paranoia. Cited by Bleuler, in Affectivity, Suggestibility, Paranoia, New York State Hospital Bulletin, vol. iv.

³ Affectivity, Suggestibility, Paranoia, New York State Hospital Bulletin, vol. iv; February 15, 1912.

reach an efficient adjustment. The constellation of ideas with its dominant, painful affect has been one to which the patient could not effectively relate himself, and therefore the next best thing had to be done, and this next best thing was the formation of certain delusions which rendered the existence of the painfully affected complex more endurable. The delusion, therefore, speaking in physical terms, is more comparable to scar tissue than to disease tissue. It represents the location of the wound and the result of the reparative process. To make the matter more clear, an individual who is ambitious, and yet who lacks ability, may develop the delusion that his lack of success is due, not to his lack of ability, which he persistently refuses to see, but to the interference of enemies who are jealous of him and who persecute him and try to belittle him in the eyes of his superiors. In this way an unacceptable fact—his inefficiency to which he cannot make adequate adjustment—is so distorted that it would appear that the results of this inefficiency emanate not from within, but from interferences from without. And so the patient creates a situation in which he, so to speak, finds himself able to get along, for as painful as a system of persecution of the sort which he creates for himself may be to him, it is less so than a realization of his own inherent defects. It will be seen, therefore, what is meant when it is said that the delusion, speaking in physical terms, represents scar tissue, and it will be seen also how a destruction of the delusion could in no way cure the disease. If the delusion could actually be destroyed the patient would be in much the same position as a patient who had a scar cut out; another delusion would have to take its place, because the patient would be thrown back upon the same unacceptable situation to which he would find himself again incapable of making efficient adaptation.

Another mechanism which is revealed in this illustration is of great importance as being characteristic of the paranoid reaction type, namely, the mechanism of projection, whereby the individual projects, as it were, upon the outside world his own mental difficulties which return to him in this instance in the form of persecution. This mechanism of projection is a very common one and appears to be fundamental in paranoid trends. It is at the basis also of the ideas of grandeur. Here the patient projects, not his difficulties, but his ambitions, and his hopes come back to him from the outer world realized.

The most elaborate attempt at interpretation of the paranoia syndrome was made recently by Freud¹ in his analysis of the Schreber case. Herein Freud voiced the view that paranoia was dependent upon a homosexual fixation in the psychosexual development of the

¹ *Psychoanalytische Bemerkungen über einen autobiographischen beschriebenen Fall von Paranoia (Dementia paranoides)*, *Jahrbuch für psychoanalytische und psychopathologische Forschungen*, Bd. iii, 1911. A very excellent and full account in English of Freud's analysis of this case can be found in the *Psychoanalytic Review*, vol. i, No. 1.

individual. To make this statement somewhat clearer it should be recalled that the individual in his psychosexual development is first auto-erotic, that is, interested only in his own body, that his next interest is in those immediately about him, the members of his own family, and particularly those of his own sex, in other words, those who have bodies most like his own, and that these stages have to be passed through before the normal end-result in a heterosexual object love is attained. The unconscious homosexual interest in the members of his own family is designated as narcissism, and the paranoiac mechanism is dependent upon a fixation and development at this period. In the normal development of the individual the unconscious homosexual tendencies are not entirely eliminated by any means, but the homosexual libido is sublimated, that is, its energies are utilized in other channels, more particularly it is utilized in all those forms of association with the same sex that one sees in friendships, social organizations, clubs, games, and in the higher social activities. But with a fixation-point at the narcissistic period of psychosexual development the patient is constantly in danger. Any serious conflict is liable to cause a regression of the sublimation to the point of fixation, and this is considered by Freud to be the mechanism at the basis of paranoia. "Persons who cannot rise completely out of the stage of narcissism and are thus prematurely fixed or arrested in the evolution of their dispositions, are exposed to the danger that a flood of libido which finds no outlet, sexualizes their social tendencies and reverts the sublimations achieved in the course of development." The libido of the paranoiac is then projected upon those about him.

The whole process is briefly and ingeniously set forth by Freud by means of ringing the changes—supposing the paranoiac to be a male—upon the basal sentence "I love him," thus:

Delusions of persecution contradict the verb. "I love him" is resented by the individual who reacts to the feeling by "I do not love—I rather hate him." Then this feeling of hate is projected with the result "he hates (persecutes) me, which justifies my hating him." As a result, this feeling, appearing to come from an outer perception becomes "I really do not love him—I hate him—because he persecutes me."

Erotomania contradicts the object. "I do not love him—I love her," then "I notice that she loves me," then finally, "I do not love him—I love her—because she loves me."

Delusions of jealousy contradict the subject. "Not, I love the man—she loves him."

Delusions of grandeur result from a total contradiction, a rejection of the whole sentence. "I do not love at all," and hence, "I love nobody." As the libido must be accounted for this is equivalent to "I love only myself."

Diagnosis.—Attention has already been called in the body of this chapter to the different conditions which have to be borne in mind

in making a diagnosis. There are many paranoid states, and wherever the paranoid mechanism is present then it is proper to speak of a paranoid state. These paranoid states are found in many of the psychoses. They may be more or less permanently associated with the special attack, as in the manic-depressive psychosis, or as in alcoholic hallucinosis, or there may be transient episodes, as in general paresis. It will be seen therefore that there are many and various types of symptomatic paranoid states and that perhaps the main consideration in the matter of diagnosis is that a condition which is symptomatic and transitory should not be mistaken for a chronic, progressive, and probably irrecoverable psychosis. This differentiation cannot always be made on the basis of a cross-section, but the patient must be studied carefully over a considerable period of time, and a reasonably full history antedating the period at which he came under observation must also be had in order to see what the general progress indicates.

Treatment.—For a considerable time past the general attitude toward the group of cases included under the designation of paranoia has been that they were incurable. The outlook has been an extremely dark and pessimistic one, and correspondingly therapeutic efforts have been paralyzed at their very inception. A somewhat changed attitude toward the whole group was the natural result of the development of a concept of paranoia which was more circumscribed and applicable to a more limited number of patients, and when correspondingly it was learned that there were many paranoid states associated with essentially recoverable psychoses; in other words, as the paranoia concept has become more and more contracted it has been realized that a great many of the paranoid conditions, which formerly were grouped under the head of paranoia, really belonged to recoverable transient conditions, and therefore the outlook for them was good. On the other hand, as the paranoia concept has contracted it cannot be said that there has been any increase, at least until very recently, in the hopefulness for this limited group of cases.

It could hardly be expected that at a time when paranoia was considered to be an absolutely hopeless and irrecoverable psychosis, chronic and progressive in its very nature, that therapeutic results would offer much, or that there would be found many who would even give any material effort in this direction, but here and there scattered through the literature are reports of cases of paranoid type which seemed to have been influenced by this or that form of therapeutic procedure, and more recently, since the doors have been opened and one has been able to enter more intimately into a knowledge of the mechanisms that are involved in the development of the psychoses and when it has been seen that these mechanisms at least are quite the same in mental disease as they are in health even, and that the mechanisms of chronic psychoses are quite the same as those found in recoverable conditions and also in health, and that the abnormal feature was not so much the mechanism as the fixation of the individ-

ual at certain periods of development or with reference to certain constellations of ideas, it became at once an open question whether these conditions might not be susceptible of the same sort of modification as they are in less serious conditions. And so within the past few years there has been an awakening of interest in these chronic psychoses and efforts are being made here and there to penetrate their mysteries and to modify their course, with the result that already a number of cases of paranoia have been reported as having had a favorable outcome.

The general principle of treatment, at the psychological level, resolves itself into as complete as possible an unraveling of the tangled skein of the patient's mental life, an uncovering of the activating circumstances in his career which have been the etiological factors in the development of the psychosis, and by so doing modifying his mental trends by a progressive process of readjustments and reëducation. This is the work of one skilled in the analysis of psychological situations and is of quite the same nature as the psychoanalytic treatment of the neuroses.

It seems certain, from the results of dealing with these paranoid conditions, that aside from any definite ability to modify the course of the psychosis or to produce a definite curative result, that the psychoanalytic method of attack may, not infrequently at least, lead to a certain amount of transfer, that the physician may come to be highly respected and affectionately regarded by the patient to such an extent at least that he may very largely control the patient's activities: This has been known to happen under rather extraordinary circumstances, showing a very high degree of personal influence by the physician over the patient, despite the fact of well-marked and fixed delusional beliefs.

In dealing with paranoiacs it must always be remembered that one is dealing with a class of patients who are potentially dangerous, that to this group belong perhaps the most dangerous of the so-called insane, and aside from matters of psychoanalysis, or in fact of any questions of therapeutic endeavor, it must be realized that where it is evident that the welfare of the individual and the welfare of society cross, the welfare of the individual must give way in favor of that of society. If the paranoiac is actually dangerous it is necessary to intern him in some institution where he will get proper care.

The question as to whether a given paranoiac is dangerous or not, in the absence of any overt acts, is often an extremely difficult one to decide. In any case it is a question to be decided by a study of the individual case and it always includes a consideration of many factors. Among these factors the following may be mentioned: It is important to find out how completely the mentality of the patient is permeated by the delusional system, in other words, how much or how little freedom he has from delusional control, whether all of his mental forces, so to speak, go to reënforce the delusion, or whether,

on the other hand, he is left reasonably free for a considerable portion of the time, in contact with reality, rather than plunged into the depths of his unreal world. It is important, too, to note how clearly defined may be his belief in the activity of any specific individual in his delusional system, whether he believes some person who is living, perhaps nearby, someone whom he can easily come in contact with, is responsible, at the bottom of his persecutions. It is important to see whether the patient, in the consideration of his delusional ideas, is at all subject to the reality motive, whether he has any critique left, or whether his belief is shakable in any degree by others, whether he can be influenced materially by his physician when it comes to the question of his delusional beliefs, or whether they dominate the situation absolutely. It is important to judge the general attitude and mood of the individual, whether he is entirely shut out from any consideration of others, of the world at large, whether he considers himself quite a law unto himself, whether he, for example, is exalted, egotistic, beyond criticism, self-sufficient, and believes that anything that he may decide to do is justifiable. It is important to know whether the patient in his past career has been impulsive, whether he has shown tendencies to do unusual, bizarre, or grotesque things, or to fly into passions, or to be uncontrollable from slight, inadequate, or unexpected reasons. Threats have to be evaluated and an opinion reached as to whether the patient really means to carry them out, or whether they are used as a means of emotional catharsis. The general education, bringing up, and ideals of the individual are important as indicating what he is liable to do. A person who was brought up originally with a proper regard for the proprieties, who is essentially a gentleman or a gentlewoman, is by that very token not so apt to commit some vulgar, rowdy, indecent act.

And finally, it may be said that while the general attitude toward paranoia has perhaps not materially changed, while there is still pretty good ground for believing that a certain class to whom the term paranoia is perhaps alone applicable, are inaccessible to therapeutic endeavor and are doomed to suffer from their psychosis throughout their lives, still even if this is so there is no absolute way of determining this fact except by a consistent and sufficiently prolonged effort to modify the course of the disease, and with the several cases already in the literature which indicate that conditions that might well have been considered chronic and irrecoverable if taken at their face value can still be materially benefited and perhaps cured, no one is in a position to pass final sentence upon any patient after an examination or two, but on the contrary, has the right to feel that there is some hope for all of them, and that at least hope should not be abandoned until constant therapeutic efforts have been applied for a reasonable time.

CHAPTER XIX.

EPILEPSY AND CONVULSIVE TYPES OF REACTION.

EPILEPSY, the "falling sickness," has been known from the earliest times, the very word itself carrying in its history (it is derived from a Greek verb meaning "to seize upon") evidences of the animistic hypotheses of earlier and relatively more primitive ways of thinking.

The word epilepsy is used as a symbol under which are grouped a great variety of conditions which in general are characterized by sudden and relatively transient *attacks* involving for the most part disturbances of consciousness ("faints," "absences," "blanks," amnesias) and convulsive seizures involving the voluntary and involuntary musculature. Such attacks are the outward manifestations of a wide variety of conditions ranging all the way from the so-called functional neuroses and psychoneuroses (hysteria, compulsion neurosis), the more frank psychoses (dementia precox), toxemic states (uremia, alcohol), many organic brain diseases (paresis, cerebral syphilis, abscess, softening and tumors) to the grosser defects of development (idiocy).

The natural evolution of the concept symbolized by epilepsy in the recognition that similar "seizures" may result in such a multiplicity of conditions has resulted in a tendency to speak of "the epilepsies" rather than of "an" epilepsy and makes it worth while to consider the attack as due to a faulty distribution of energy which may be brought about in many ways and through divers mechanisms. The wide variety of conditions, as a part of which convulsive reactions with associated disturbances of consciousness occur, cannot be too much emphasized. The toxic states (endogenous or exogenous) are usually transitory and depend upon the continuance of the toxemia, but in defective parathyroid functioning with disordered calcium metabolism the convulsive phenomena continue because the underlying metabolic disorder cannot be permanently relieved. Marked organic changes which are responsible for convulsive attacks are usually cerebral (tumor, softenings, hemorrhage, meningitis), but certain organic conditions resident elsewhere, notably the cardiopathy of Stokes-Adams disease and the condition resulting in animals from thymus extirpation, appear to be sufficient causes. While still more obscure factors determine vagal and vasovagal, gastric and intestinal attacks which appear to be dependent upon elements of constitutional make up at the level of the vegetative nervous system. It is worth while, therefore, to attempt to get a view-point of all these conditions dependent upon their common element—faulty energy distribution.

For the purpose of comprehending the epileptic phenomena then, the nervous system may be viewed in a very simple way. From the stand-point of structure it may be thought of as consisting of receiving organs, designed either to come in contact with the external world (exteroceptors) or with other parts of the body (proprioceptors) spoken of collectively as receptors. The combined material accumulated through these receptors forms the basis upon which certain extensions of the nervous system (effectors) are devised, whereby the reactions of the body are conditioned in a way to bring about that adaptation essential to life or to the maintenance of the social structure. In this way the nervous system is viewed as a mass of interrelated reflexes redistributing the energy received, for the purposes of the organism.

As the incoming stimuli are multitudinous, so the outgoing activities are correspondingly diverse, and a healthy organism is able, by reason of its nervous mechanisms, to so distribute the energy received as to bring about a series of harmoniously adjusted activities, be they physicochemical, sensorimotor, or psychic. This view-point, that energy distribution takes place at all of these levels, should not be lost sight of, as there is a tendency to think of the problem solely in terms of muscular work. Atwood's "man in the box" broke up more nitrogen compounds during mental than during mechanical work. This breaking up of nitrogen compounds is, however, only one form of registering the energy output.

That which characterizes a well-adjusted nervous mechanism is its ability to properly and in an orderly manner distribute its energy, but that which characterizes the pictures of the disorders included in this chapter is inefficiency in this regard which may be more especially emphasized at the physicochemical, the vital, or the psychic levels.

An explanation for epileptic attacks which finds its ultimate expression under such symbols as eye-strain, floating kidney, gliosis, or like specific indictments fails to realize that the nervous system contains representations of all of the organs and that the final activity of the human body is the result of the balance which has been struck among innumerable tendencies. The part that any particular organ plays can only be understood when taken in consideration with the organism in its totality and realizing the specific part that the organ in question plays in the whole problem.

Bearing in mind this view of the nervous system, as a great mass of complexly interrelated reflexes, and further, the law of avalanche (Cajal) which insures the continuous breaking up of the original sensory stimulus into an ever-increasing number of avenues of discharge, it will be seen how many ways are open to interfere with the orderly procession of energy through this complicated series of reflex arcs. The nature of the epileptic discharge, essentially a manifestation of energy at greatly reduced adaptive efficiency, and the destructive character of certain pathological lesions which initiate it (impaired

metabolism, gross destructions, psychic imbalances) indicates that the essential defect is not irritant but destructive, the blocking or closing of many paths of outlet structurally or by inhibition, and so accumulating the discharge within relatively narrow confines. Such a conception would apply equally well to the "idiopathic" or "genuine epilepsy" with Ammon's horn gliosis and to the epilepsy associated with marked developmental defects (idiocy) in which it may be conceived that the wider paths for avalanche discharge have not been laid down. This view-point is also consistent with the different levels at which the discharge may take place—psychic, physiological, and physicochemical—the character of the attack as limited to certain levels, the psychic (hysteria, dementia precox), the physiological (Jacksonian types), the physicochemical (tetany); the distribution of the discharge, general attacks ("genuine epilepsy"), localized attacks (Jack-



FIG. 305.—Epileptic, showing scars over eyebrows from falls.

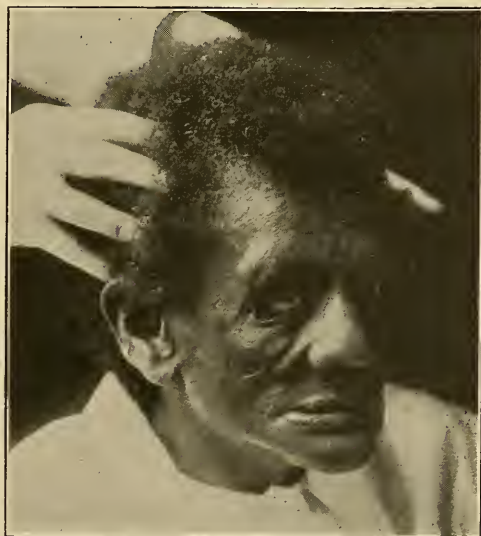


FIG. 306.—Epileptic, showing scar from burn due to injury during convulsion.

sonian types), and as being initiated by sensory, motor, or psychic prodromes.

Symptom Groups.—Convulsions may occur, as already indicated, in a great variety of conditions while distinctly explosive attacks, not convulsive in the sense of muscular spasms, occur under still wider conditions in states not definitely epileptic but, with reference to the more essential epilepsies, in what may be referred to as borderland conditions.

The essential epilepsies have been divided into the *late epilepsies* occurring relatively late in life and dependent upon toxemias and gross organic changes and the *early epilepsies* which occur relatively early in life, generally before or during adolescence.

Epilepsies of Gross Brain Disease.—These occur in paresis, cerebral syphilis, brain cysts (echinococcus, etc.), hydrocephalus, the cerebral meningitides (syphilitic, tuberculous, serous, and pachymeningitis), bony tumor of skull, traumatism (fractures, insolation, concussions, hemorrhages), multiple sclerosis, cerebral sclerosis and gliosis, cerebral arteriosclerosis, the encephalitides and myoclonias.

The Epilepsies of Toxic and Infectious Origin.—The toxemias include those of endogenous origin (uremia, diabetes), of exogenous origin (metallic—such as lead and arsenic, and strychnin, alcohol, and carbon monoxide).

The infections are more especially the exanthemata, influenza, rabies, malaria, rheumatism, syphilis, malaria, etc., operating either through the mechanism of an overwhelming toxemia or by meningitides or encephalitides.

Anomalous and Borderland Conditions.—Here are included certain internal secretory imbalances, particularly diseases of the thymus, thyroid, and parathyroids and degeneratio-adiposo-genitalis.

Certain high level attacks occur in hysteria, compulsion and anxiety neuroses, and in dementia precox, especially the catatonic form.

Here also should be included the vagal¹ and vasovagal attacks, slight passing disturbances of consciousness associated with vertigo and sometimes sensory disorders as loss of sight, possibly certain sensory disorders of fulminating character such as migraine, and some disturbances of sleep (narcolepsy) disturbances of consciousness of syncopal nature, and the affect epilepsies of the Bratz type.

Pathological Groups.—The preceding clinical grouping gives a fair idea of the pathological conditions which may be found. The following grouping is given by Alzheimer² as a result of the histological examinations of 63 cases. These simply indicate the reasons why and how the structure of the brain is modified thus changing its functional capacity as an energy distributor.

¹ Wm. R. Gowers, *The Borderland of Epilepsy*, Philadelphia, 1907.

² Alzheimer and Vogt, *Die Gruppierung der Epilepsie*, Jahresversammlung des deutschen Vereins für Psychiatrie, 1907; Ref. Allg. Zeitschr. f. Psych., Bd. lxiv, 1907.

- A. Cases with very obscure etiology (genuine epilepsy):
1. This group comprises 60 per cent. of the cases:
 - (a) With sclerotic changes in Ammon's horn.
 - (b) With superficial gliosis of the hemispheres.
 - (c) With signs of an acute process (status) besides *a* and *b*.
- B. Cases due to external poisons:
1. Alcohol: Different anatomical changes, as in chronic alcoholism. Besides these sometimes acute changes, as in delirium.
 2. Lead: Different changes. Experimentally lead produces a genuine encephalitis.
- C. General diseases:
1. Syphilis: Different forms of brain syphilis, especially the endarteritis of the finer vessels (Nissl, Alzheimer).
 2. Arteriosclerosis.
- D. Focal diseases: Most of the cases in this group are cases of epilepsy with idiocy after encephalitis.
- E. Arrests of development:
1. Stadium verrucosum (Ranke).
 2. Sclerosis tuberosa.

This survey will suffice to show what a wide variety of conditions have been included under the term epilepsy and also to point out the various groups that are being at present split off and separately identified. What has been said about the distribution of energy applies to the broad group of convulsive reactions. The disease "genuine epilepsy" will now be briefly described, although it is extremely difficult to do this at all accurately as it must be borne in mind that heretofore all matters of description and questions, such as those of heredity, are with practical uniformity considered with reference to "epilepsy," without effort at discrimination, much as is the case with the corresponding conglomerate "insanity."

CLASSICAL EPILEPSY

This roughly corresponds to the group "genuine epilepsy" of Alzheimer.

Heredity.—The genuine epileptic usually comes from a badly tainted stock. Epilepsy may not appear in the ancestors but they and often the collaterals show evidences of ill-defined nervous disorders (according to Davenport and Weeks,¹ migraine, chorea, paralysis, and extreme nervousness). Epilepsy and feeble-mindedness show a great similarity in their hereditary reactions and both appear to be due to a defect of the germ plasm, that is, they are both recessives. As should be expected, therefore, the two conditions are frequently found associated. This is also of significance in relation to what

¹ A First Study of Inheritance in Epilepsy, *Journal of Nervous and Mental Disease*, 1911, vol. xxxviii, No. 11.

has already been said about the developmental failure to lay down the paths for the higher avenues of sensory avalanche.

The Epileptic Constitution.—This type of epileptic is apt to be morose, irritable, suspicious, and hypochondriacal. He is quite characteristically unreliable and with it all frequently presents a very aggressive form of sentimental, shallow religiosity. This type of epileptic, in general then, is very sensitive, irritable and insincere. He is egocentric to a very considerable degree, paying great attention to himself, his own feelings, his state of health, his physical comforts, and his immediate surroundings. His interests are variable and he presents light variations of mood with perhaps headache and a tendency generally to hypochondriacal fixations. His interests all tend to be concentrated in this egocentric constellation. His reactions of irritability and unreasonableness present infantile characteristics. Many epileptics are feeble-minded or more profoundly defective, and rather in conformity with this frequent finding the word associations from epileptics have close analogies to the word associations of the imbecile. In addition to these traits of character these epileptics are usually lazy, frequently they lie openly, present an attitude based on high moral standards of great respect and consideration to one's face and quite the opposite when one's back is turned. Their general health is apt to be good and they often have enormous appetites, and are especially fond of proteids. While good-natured, even-tempered, well-disposed epileptics exist they are more apt to be most difficult problems to get along with, and as a class in the hospital they are extremely difficult to care for. Passing attacks of mental disturbance occur in the interparoxysmal period without apparent relation to seizures. Attacks of *transitory ill-humor*, according to Aschaffenberg, occur in 78 per cent. of cases. This is a condition of irritability, unreasonableness, sometimes associated with delusions and hallucinations. The patient is in a "touch-me-not" state and very apt to get into quarrels or make attacks. Rarely the disturbance is expansive in type and in these cases may be associated with religious fervor.

A study of the sexual characteristics of epileptics has recently been made by Maeder.¹ As a result, he finds the sexuality of the epileptic still largely undeveloped, still only little removed from the infantile stage. The sexual feelings are very prominent and are aroused in many ways: autogenically, constituting *auto-erotism*, and giving rise to such phenomena as masturbation, and by stimuli from without, constituting *allo-erotism*, which gives rise to a normal libido (heterosexuality), homosexuality, exhibitionism, etc. Maeder uses the term *polyvalent* to describe this characteristic of the epileptic sexuality which permits it to be aroused by many kinds of excitants.

The epileptic state leads in a certain proportion of cases, if it has begun in early life, to conditions of feeble-mindedness, imbecility, and

¹ Sexualität und Epilepsie, Jahrb. f. Psychoanalytische u. Psychopathologische Forschungen, 1909.

idiocy, or, depending upon the same causes, is associated with these conditions. Epilepsy tends, in many cases, to produce a general mental deterioration (*epileptic dementia*) which may become very profound.

A recent study of the personality of epileptics by Clark¹ has led him to the conclusion that this general type of character as described above is a result of the disease and not a precedent condition.

Scripture and Clark² have described the epileptic voice sign and found it in 75 per cent. of cases. The voice has been studied by the "air puff" method of recording on the kymograph. A measure of the wave gives the rates of vibration. A line connecting the tops of the ordinates produces the "melody plot." Normally each vowel has a rising and falling melody. In epilepsy the vowels run along on an even tone—"plateau speech." This is very characteristic and easily recognized.

Muskens, in his study of the muscular phenomena, has found fatigability and weakness of single muscles or muscle groups, startings, shocks and cramp-like contractions especially just before or after going to sleep. Gastro-intestinal and vasomotor disturbances have also been noted.

The Seizure.—The classical major epileptic attack (*grand mal*) is sudden in onset, often preceded by a warning—*aura*. The patient falls and the attack immediately develops into a tonic spasm with unconsciousness. The tonic is replaced in a few moments by clonic spasms which gradually subside. There is then often a short period of automatic activity followed by a gradual return to full consciousness, or the patient sinks at once into a deep sleep from which he awakes complaining of lameness and weakness in the muscles that were convulsed, and perhaps headache.

The attack has been described by authors in great detail, but is really different in practically each case, although the type tends to remain the same in each patient.

The *aura* may be sensory, motor, or psychic. The sensory warnings may occur in any of the sensory fields: the visual (flashes of light, hallucinations), the olfactory (odors, usually bad—*uncinate fits*), the auditory (simple sounds or hallucinatory voices), etc. The epigastric *aura* is most common and consists of a wide variety of disagreeable sensations in the epigastrium.

The muscles first involved vary greatly, as does also the order in which they are involved—*march of the convulsion*. The patient falls at the beginning of the spasm, the direction of the fall being dependent upon the muscles first affected. In a few moments all of the voluntary muscles are convulsed, including the muscles of respiration, producing cyanosis, and the jaw muscles, resulting in biting the tongue. With the beginning of the clonic stage frothy, bloody saliva issues from the mouth and the cyanosis gradually disappears with the resumption of

¹ A Personality Study of the Epileptic Constitution, Amer. Jour. Med. Sci., November, 1914.

² Researches on the Epileptic Voice, Proc. New York Neurol. Soc., November 12, 1907.

respiration. Urine and feces may be passed during the attack—usually only the former. In the partial or incomplete seizures—*petit mal*—the convulsive phenomena are much milder and may even escape observation altogether, while the disturbance of consciousness is shorter in duration and less profound. The patient may blanch, become confused for a few moments, perhaps falter in what he is doing, or fumble for a few moments about his clothing in a dazed fashion and then go on about his affairs as if nothing had happened. These attacks are also often preceded by an aura.

The psychic disturbances associated with the attack, before and after, or replacing it are many and varied.

In a great many epileptics there is a marked disturbance preceding the convulsion, sometimes of several days' duration, and those who are accustomed to the patient can tell that a fit is impending. This change manifests itself in increased irritability, complaining, sometimes by depression or dulness, and there may be associated disturbances of the sensorium, hypochondriacal complaints and hallucinations. All these conditions are commonly promptly relieved by the fit.

Immediately after the convulsion there is often a temporary condition of confusion. The patient rises clumsily, looks about him in a bewildered manner and often does some semiautomatic acts, such as taking off his clothes. Also following the attack a transitory exhaustion paralysis in the overacting muscles makes itself apparent.

Just before, or more commonly after, the convulsion a condition of active excitement may occur which may reach the stage of frenzy. In this state the patient is a veritable wild man—*epileptic furor*. He is liable to kill anyone who approaches or even himself. Fortunately his efforts are diffuse and not coherently directed. During this attack, which is usually brief, he has to be restrained and at the end is quite completely exhausted.

An attack of mental disturbance may take the place of the convulsion and thus become an *epileptic equivalent*. These attacks of *psychic epilepsy* frequently take the form of so-called *epileptic automatism* or *epileptic dream states*. In these conditions the patient may do almost anything and when he comes to himself he has absolutely no recollection of what has happened. Usually the attacks are of short duration and the acts rather simple—more simple than in the dream states of alcohol or hysteria. However, they may last for days, all sorts of things may be done, crimes may even be committed, so that the condition often becomes of great medicolegal importance. The crimes of violence are often noted for their ferocity and brutality.

It must not be forgotten that these states may be associated with a seizure that was so slight as not to have been noticed. Evidences of such a seizure, especially in medicolegal cases, should always be looked for.

Transitory conditions of *depression, excitement, confusion, delirium,* and *stupor* may develop and quite characteristically a condition of

ecstasy with hallucinations. The patient sees the gates of Heaven open and as the heavenly hosts appear he hears himself addressed by the voice of God.

The transitory states of ill-humor, as described by Aschaffenberg in the interparoxysmal state, might also be considered as psychic equivalents; these are frequently associated with drinking.

Besides these conditions, *paranoid psychic states* are quite common, while of the more transitory psychic manifestations *fugues* are frequent and certain types of *dipsomania* appear.

In addition to the symptoms thus far indicated various observers have found evidences of an altered blood picture such as leukocytosis and hypo-eosinophilia, while disorders of metabolism with hyper-toxicity of the secretions, has long been adduced as proof that the manifestations were dependent upon chemical poisons due to faulty metabolism.

Meaning of the Attack.—Bearing in mind what has already been said about the distribution of energy it will be of advantage to pursue this line of thought somewhat further.

Energy flow may be blocked, dammed up, and break through in diffuse discharge at any level—psychic, sensorimotor, or physico-chemical. This being so it would be expected, as is the case, as already indicated, to find disturbances at each of these levels. In the classical epileptic attack all these levels are involved, but what is of equal or greater significance to the general hypothesis is that there are attacks practically limited to one level, and a study of the several types of convulsive reaction will show a series of cases reaching through all of the intermediate stages from the highest to the lowest instinctive levels.

The hysterical convulsion offers an example of a high level convulsive type of reaction. This seizure is admittedly psychogenic in origin and presents the picture of a conversion of psychological into physiological symbols. In other words, the patient escapes from his painful ideas by converting them into physical symptoms. (See Hysteria.)

The disturbance of consciousness in these hysterical attacks is relatively slight, much less than it outwardly appears to be, while the whole situation is quite near the surface and with very little effort can be brought to conscious control.

Next lower in the scale of levels are the psychasthenic convulsions of Oppenheim (compulsion neurosis type) which are expressions of a more severe grade of neurosis but still within strictly psychological levels.

Then come the very interesting affect epilepsies of Bratz and Leubuscher. These are distinctly epileptoid types of reaction conditioned by purely psychological situations. Here the outward semblance to a deeper level epilepsy is much greater but the situation is still a psychological one. The reaction of the patient here is to conditions that are absolutely intolerable and to which no adjustment is possible,

such a situation, for example, as a young man has to confront when the key is turned upon him and he is called upon to realize that he is in prison with a life sentence to face. Under these circumstances the patient may become a veritable "wild man," beat his clenched fists against the bars, rush aimlessly about destroying clothes and bedding, and beat his head against the walls in ineffectual attempts at self-destruction. Hallucinatory disturbances may accompany these attacks and amnesia follow them, though consciousness during the attack is not entirely lost. That these patients are much more seriously burdened constitutionally and more nearly allied to "genuine epilepsy" than the psychasthenic types of Oppenheim is indicated by the fact that they give a history of "fits" in childhood while the psychasthenic types show tics, phobias, and compulsions.

In the classical epileptic seizure the greater severity and seriousness of the attack is indicated by the complete loss of consciousness and the still further reduction in the purposeful and coördinated adjustment of the muscular reactions. These have now become utterly disorganized. The attack has involved far more than the psychological levels and included the sensorimotor and, as indicated by the toxicity of the excretions, the biochemical.

The low instinctive level to which the epileptic is reduced by his seizure can be appreciated by observing his activities as he is "coming out" of the attack. His respiration is at first distinctly abdominal (infantile type), he makes characteristic sucking movements with his lips, and his movements, from the complete disorganization into which they have been thrown, assume at first an aimless fumbling with his clothes, a tentative feeling about as he instinctively tries to readjust himself to reality, to "find himself" again. In this tentative "feeling about" he repeats in a few minutes the process of relating himself to reality which is a normal period of development in the child.

Ferenczi¹ has endeavored to classify the neuroses with reference to the stage of development they represent and in accordance with this scheme suggests that epilepsy belongs to the period of wish-fulfilment by means of incoördinate movements. It is known how some children when thwarted will cry out, thrash about and sometimes straighten out rigidly, "lose their breath" and become blue. Attacks of "temper" the mother calls them. Later on the child will kick the chairs and tear up its books under similar circumstances while regression to approximately the same level is shown when an adult stamps his foot, clenches his fists, grinds his teeth, and otherwise shows reactions of anger which are quite ineffectual to effect any change whatever in conditions. The meaning of it all is an absolute inability to accept or to adjust and an equally determined attitude that it *is not* so because it just *cannot* be so. An effort to force circumstances to be different by a supreme effort of thinking them different which when it fails results in a flight from

¹ Entwicklungsstufen des Wirklichkeitssinnes, Internationale Zeitschr. f. Aertzliche Psychoanalyse, 1913, vol. i.

the whole thing into the rigidity and unconsciousness of the epileptic seizure.

This way of viewing the epileptic attack is warranted because it follows along a path that proceeds from the known to the unknown. The mechanism at the higher psychological levels can be worked out and although those at lower levels cannot it would appear that the two extremes are connected by a regular series of intermediate stages as represented in types of cases. It can only be assumed that the disorder in "genuine epilepsy," has to do with adjustments at deep instinctive levels, adjustments that are profoundly biological in character, and that the conflict may almost threaten life itself in order to understand the severity and seriousness of the attack as a reaction to failure. A study of the aura as a clue to the point at which blocking of energy commences, and a detailed analysis of all the elements of the attack plus an analysis of the make-up of the individual in the interparoxysmal period would seem to offer the mode of approach to a further understanding of the mechanisms in individual cases. The depth to which such an analysis would have to go and the severity of the constitutional burden in the classical types of the disease is indicated, for example, by the prevalence of the epigastric aura the characteristics of which indicate the possibility, at least, that the disorder reached as deep as the vegetative nervous system level.

It can be seen from this discussion why the epileptic, burdened by deeply instinctive defects of biological adjustment, should dement. It is also consistent with this view that, in general, this should not be true of symptomatic epilepsy. A localized lesion of the cortex, for example, may produce convulsive reactions in well-defined groups of muscles only without loss of consciousness (Jacksonian type). Here there is no defect of biological adjustment involving the individual as such. A group of muscles only has been cut off from effective associational relationship with higher levels and so becomes reduced in its possibilities of reaction to relatively incoördinate, automatic and purposeless types. A portion only of the machinery has been damaged, the individual remains otherwise intact. The disorder is confined to the sensorimotor level.¹

From this point of view it seems that the toxicity of the blood and urine is only an outward evidence of the depth of the disorder rather than an indication of its cause. It is true that certain toxic substances do produce convulsions, but they do it by damaging the machinery like the cortical lesions just referred to and the seizures cease with the removal of the poison. This is seen in severe forms of infection, febrile states in children, alcohol, uremia, etc.

It is interesting and significant to note, in this connection, that the form of dementia precox in which convulsive seizures are most prone

¹ See Clark, L. P., *Nature and Pathogenesis of Epilepsy*, N. Y. Med. Jour., February, 1915 *et seq.*, for a complete statement of this viewpoint with numerous clinical proofs of its value.

to occur is the catatonic. In the other forms types of compromise formation take place while the catatonic endeavors to cut out, to enucleate, so to speak, a certain portion of his psyche, a condition much more favorable to blocking.

Varieties of Convulsive Attack.—The Jacksonian type has already been referred to as has also the distinction between the grand and petit mal attacks and various equivalents (larvated or masked epilepsy).

In addition to *isolated seizures*, there are *serial attacks* in which several seizures follow each other at relatively short intervals and finally *status epilepticus* in which large numbers of attacks accumulate, following each other at short intervals until unconsciousness becomes continuous, the attacks then merging into one another—overlapping. The temperature rises in this condition, life is threatened and indeed it is the typical mode of death of the epileptic and his ever-present danger. Serial attacks are usually grand mal, but may be petit mal, while status, even if it begins as petit mal, soon takes on the seriousness of the major variety. Psychic seizures may also be serial and status attacks are possible without or with very minor convulsive manifestations. Status may, and frequently does, develop in the symptomatic epilepsies due to gross lesions of the brain as well as forming a frequent termination of the partial or incomplete seizures of the Jacksonian type.

Myoclonus epilepsy appears to be an association of myoclonia and epilepsy. It would seem that the myoclonic shocks, however, gradually eventuated by a process of summation into an epileptic seizure. Some epileptics have myoclonic shocks between their attacks which appear to be forerunners of the seizure rather than true forms of myoclonia.

Continuous epilepsy.—Similar to the myoclonic varieties are the polyclonia epileptoides continua of Choroschko and the epilepsia corticalis continua of Koshewnikow. This is a condition of continuous myocloniform shocks in single muscle groups, usually unilateral and without loss of consciousness.

Diagnosis.—From what has been said it can be seen that epilepsy is not an entity, that the term includes a great multitude of widely different conditions and that the problem of diagnosis is therefore the problem of differentiating the particular one of these several possibilities in an individual case. Diagnosis, however, does not mean giving a name to a thing, but understanding it. Every individual epileptic should be accepted as an individual problem and although it may often be quite impossible to fathom the mechanisms involved, it is only by such a method of approach that anything worth while can be hoped for when the question of treatment comes to be considered.

The possibility of exclusively nocturnal attacks—*nocturnal epilepsy*—should be borne in mind. It is suspicious if the patient awakes tired and lame, as if his muscles had been beaten, particularly if he shows conjunctival ecchymoses, a wounded tongue, and flecks of blood on the pillow. A localized muscular weakness that passes off promptly would add certainty to the diagnosis.

Treatment.—The only efficient prophylaxis is not to transmit the defective germ plasm. It would seem that in the purely symptomatic epilepsies, such as those due to cortical traumatism, that the germ plasm might escape indictment, but this does not necessarily follow. A certain proportion of these cases will be found to have had convulsions in infancy so that they might have been considered as potential epileptics predisposed to react by convulsion-producing mechanisms. This possibility is emphasized by the frequency with which the symptomatic epilepsies develop status attacks.

Treatment of the Attack.—Once the attack has started it is essential to so care for the patient during his period of helplessness that he may not be injured in any way. If he has fallen in a safe place he may be allowed to remain there, perhaps only removing him from proximity to furniture or the wall against which his limbs might be injured as they are in the throes of the convulsive seizure. The clothing should be loosened about the neck to permit free breathing, and if possible a towel end rolled up and pressed between the teeth to prevent injury to the tongue. As a rule, he should be permitted to remain on his back or side, according to the position the contracted muscles force upon him. A wound received in falling may need care and a broken limb needs protection from the severity of the convulsive contractions to prevent additional injury by the broken ends. Epileptics, in general, should not be permitted to sleep unobserved or alone for fear they may roll over and smother during an attack. If vomiting occurs the patient should be rolled on his side and care should be exercised to prevent aspiration of the vomitus. In the automatic period following, watchful care is needed, but direct efforts at control should be avoided if possible as they are not understood and may only excite antagonism. The patient should not be permitted to get up until it is seen that no bones are broken. In this automatic state an attempt to walk on a broken leg might easily compound the fracture for consciousness is so reduced that pain would not be felt or reacted to.

Treatment of Status.—Serial attacks indicate possible danger of status and sedatives should be used to control their frequency, while sudden withdrawal of bromides should be avoided as tending to precipitate a number of seizures. Clark¹ recommends the following as an emergency prescription when status is threatened:

R—Tr. opii deod.	℥v
Potas. bromid.	gr. xxv
Chlor. hyd.	gr. xx
Liq. morph. sulph. (U. S.)	ʒj—M.

S.—One dose: repeat in two hours if necessary.

This prescription may be given after the first four or five seizures, after that sedation must be pushed to control the attacks as they are themselves a source of serious danger to life. Chloroform may be

¹ Wm. P. Spratling, *Epilepsy and its Treatment*, Philadelphia, 1904.

given in emergency by inhalation, but the most valuable of the drugs are chloral and the bromides. While it is necessary to push them, it must be constantly kept in mind that these very drugs are contraindicated in the next, the stuporous stage, and therefore no more should be used than is actually necessary to control the situation as the following coma will be deepened thereby. Chloral and bromides are, as a rule, best given by rectum. The heart needs watching and may need stimulating, especially if large doses of chloral are administered.

In the stuporous stage the treatment is stimulating and supporting. Careful nursing and feeding and protection during the great exhaustion.

Treatment Between Attacks.—As there is no disease entity (epilepsy) there is no treatment that applies to all of the cases included under that term. Each of the various conditions requires treatment suited to it as does each individual require individual consideration.

The various surgical conditions, tumor, cyst, abscess, trauma, etc., require appropriate surgical intervention. In tumor, for example, as in other organic conditions, when the location of the trouble is not evident, a study of the attack together with the aura may give valuable evidence to guide the surgeon. It is desirable to have all such cases reside in a hospital long enough for their attacks to be accurately observed before operating.

Conditions of infection and toxemia require no special mention here. In arteriosclerosis with softening the general condition overshadows the special manifestation as is also generally the case following hemorrhage. Conditions of marked arrest of development, either congenital, as due to serious birth injuries, or early inflammations naturally offer little prospect for improvement.

Internal secretion imbalances should be corrected as far as possible, but for the most part little more than palliation can be expected, although the near future may well have something to offer in this realm.

Middle-ear disease should be adequately treated before meningeal symptoms and lateral sinus thrombosis take place.

The syphilitic meningitides, whether acquired or inherited, offer a promising field for relief by appropriate antisiphilitic treatment, which should be intensive and include intravenous injections of salvarsan.

As to the pharmacotherapy of "genuine epilepsy" it can be inferred from what has already been said that there is none except such as may be called upon to meet or prevent emergencies such as status. Bromides have been so constantly used, however, that they need to be briefly discussed.

Bromide acts as a motor depressant—it raises the threshold of motor discharge and does therefore inhibit the convulsive expression. That it does this is sufficient explanation for its extensive use. Many popular medicines have much less to recommend them. When we realize, however, that the convulsion is not the disease, that it is not the cause nor a first expression even of the disease, but only its outward expression and the end-result at that, it may be well questioned whether

bromide medication is rational. Experience seems to show that the fit postponed by bromides comes to pass ultimately any way and that the bromide may, in fact, operate unfavorably by tending to produce a summation of attacks and thus increase the danger of status. Added to this is the potentiality for disturbing digestion which the bromides possess so prominently, so it may be said that they had best not be given at all unless under most carefully regulated conditions. The true function of the bromides is to control the convulsive manifestations when they, as such, become a source of danger as in serial attacks and threatened status.

Bearing in mind the theory of the disease that has been elaborated, the rational treatment in all cases where the underlying mechanism cannot be unearthed (as in the symptomatic epilepsies), is to assist in the orderly discharge of energy, to help the process of sublimation. This is best effected by manual training, steady occupation graded to suit the intellectual level and other requirements of the patient, and preferably conducted under institutional (colony) supervision. In many individuals much is to be expected from psychoanalysis. No results will follow from short treatment, however. The most favorable cases need from twelve to eighteen months.

The social position of the epileptic is most pitiable, often so impaired mentally that his labor is at a discount, he loses his job on the occasion of the first fit, even though he may have succeeded in getting one that is free from the dangers to which he is particularly exposed on account of his infirmity. He is thus seriously handicapped in the struggle for existence and barely gets to earning his livelihood in one position before he finds himself jobless again, without recommendation, and forced to begin all over again. He is thus apt to be poorly nourished, poorly clothed, and the subject of intense social repression operating from without that drives him back upon himself and aggravates greatly his trouble. He becomes discouraged and depressed and only too often takes to alcohol, the very worst possible thing he could do.

In the colony all these social handicaps are removed. Here he may have a fit in peace and comfort without feeling that he is disgraced or in imminent danger of losing his means of livelihood. This relief alone goes a long way toward permitting him the use of his avenues of expression and in bringing about a relative peace and quiet, so essential as a therapeutic adjuvant. If in addition to this he is trained in some form of healthy occupation, preferably outdoor, that is interesting and affords an added means of expression the best possible has been done for him. In the colony, too, he is provided a home, congenial surroundings, a regulated diet, and is under that careful and continuous skilled supervision for a prolonged time which is so essential to the best results. Under colony care Spratling thinks 5 per cent. of cases as they go can be cured and that this percentage could be doubled or perhaps trebled if all the cases could be gotten under treatment early.

CHAPTER XX.

DEMENTIA PRECOX (SCHIZOPHRENIA) GROUP.

THE term dementia precox has been the occasion of a great deal of discussion. Coming into general use as it did as the result of the studies of the Kraepelinian school it was conceived to apply to a group of psychoses belonging to the period of adolescence and presenting dementia as a fundamental element in the symptom picture. When, however, it was seen that what appeared to be the same disease might occur later in life, even after thirty years of age, it seemed hardly proper to use the term precox as applied to psychoses of early life. It was therefore proposed that the term precox should refer not to the age of the patient, but to the relatively early appearance of dementia in the course of the disease. The term dementia was here used to mean a permanent mental impairment, and when it was realized that many cases made good recoveries without any apparent or at least material defect remaining, another reason was evident for the inapplicability of the term. The concept, then, might be formulated that it was a disease in which dementia was a relatively early symptom, and that the recoveries occurred only when the disease had not progressed to any extent. This also, unfortunately, does not meet the facts, because many cases get well after prolonged and apparently chronic courses. In the absence of any well-defined criteria of dementia it was impossible to predict when it was or was not present, and therefore the term presents very many undesirable features. Although it is somewhat of a bootless task to discuss names, and although it is much more important to know what the names stand for than to quibble about their applicability, still it is of course desirable to have a name that fairly represents the thing named. To meet this demand Bleuler has suggested the name schizophrenia, implying a splitting of the personality, which he thinks is the fundamental symptom. Although this term as used by Bleuler includes a number of conditions that many psychiatrists would object to as being included in the dementia precox concept, still it is generally conceded that the splitting of the personality, as indicated by the name, is fundamental in this group, and the name is coming into gradually more and more general use.

Dementia precox must undoubtedly have always existed and have been observed by physicians, and in particular the grotesque cases of catatonic rigidity and peculiar mannerisms must have always attracted attention. In the early history of psychiatry, however,

few descriptions of cases exist that could be unequivocally said to be cases of precox, as the group had not been defined from other groups superficially resembling it, as for example, imbecility. Willis, the English anatomist, recognized as early as 1672 that many young people underwent deterioration, and Sydenham, a hundred years later, in 1772 describes similar conditions under the description of stupidity, while later on, after mania and melancholia had been more or less defined, many of the excitements and depressions that are incident to the course of dementia precox were undoubtedly grouped under these headings, while at one period, only a few years ago, there was a distinct group supposedly representing a special disease described by the name of catalepsy, where also undoubtedly a certain number of precox cases were arranged.

At the present time three pretty well-defined groups of cases are included in the general concept of dementia precox, namely, the hebephrenic, the catatonic, and the paranoid. Kahlbaum was the first to describe hebephrenia as a disease entity in 1863, and in 1871 his pupil, Hecker, published some excellent descriptions of this disease. In 1869 Kahlbaum described catatonia under the term *Spannungsirresein* or *vesania catatonica*, of which he gave an admirable monographic description in 1874. In 1896 Kraepelin, in the fifth edition of his *Lehrbuch*, arranged dementia precox, catatonia, and dementia paranoides as disorders of metabolism. Clouston, the Scotch psychiatrist, had already described what he termed adolescent insanity and objected to the term dementia precox as being too inclusive. Kraepelin, however, worked over his material with great thoroughness and arrived at the concept that includes the three forms, hebephrenic, catatonic, and paranoid by tracing the life histories of his patients and grouping all these cases, however dissimilar they might appear on the surface, from the standpoint of prognosis. They were cases that had a fairly definite course and outcome, eventuating always in a certain degree of dementia.

Etiology.—The question of *heredity* in precox has been studied, particularly by Wolfsohn,¹ who carefully analyzed the material from this standpoint at the Berghölzi asylum in Zurich. The study of 2215 admissions disclosed 647 cases of dementia precox of whom 90 per cent. showed hereditary taint. Of four factors, mental disease was the most frequent—about 64 per cent.—followed by nervous diseases, alcoholism, and other forms of hereditary taint. Heredity was combined in 34 per cent. The most frequent combinations were those of psychoses and alcoholism, and psychoses and nervous disease. She concluded that a distinct influence of heredity could not be proved in the cases in which the taint was alcoholism, nervous disease, or other forms. The catatonic was the most and paranoid the least affected by the mental taint, while the influence of

¹ Die Heredität bei Dementia Præcox, *Allg. Zeitschr. f. Psych.*, 1907, Band. lxiv, Heft 2 and 3.

the taint had no striking effect on the character of the first symptoms of the disease.

It would seem that there is a certain element of direct heredity, inasmuch as families are found in which several cases of precox occur, just as there are families found in which several cases of the manic-depressive psychosis occur.

Of the exciting causes severe shocks, both mental and physical, are not infrequently found, as for example, severe hemorrhages and infections following parturition. In the latter case precox breakdowns occur as a result of that train of emotional disturbances which follows upon seduction and desertion.

For a number of years there has been a tendency to ascribe the disease to disturbances of metabolism with possible toxic factors and to suppose that its origin might be traced to disorders of the glandular secretions, more particularly of late, of the internal secretions of the ductless glands, and inasmuch as the disease tends to focalize about the period of puberty and adolescence, it has been supposed that perhaps the testicles and the ovaries might be the offending organs. This whole tendency has had its origin largely in the observed physical changes which are so much in evidence in certain cases. More will be said of this matter later when the discussion of the nature of the disease is taken up, but it may be mentioned here that whatever its ultimate nature may be the existence of toxic factors or internal secretory disturbances is purely hypothetical and at the present time it is more useful to formulate the upsetting factors as well as the general symptomatology in psychological terms rather than in terms descriptive of disturbances at physicochemical levels.

The formulation of the disease in terms of the affects or of complexes or, in accordance with Meyer,¹ continued unhealthy biological reactions, or as an outgrowth of a "shut in" character is after all more of an effort of description of what is found. Everyone has complexes, but it is not clear why in certain cases they lead to the development of a precox psychosis, while a "shut in" character might itself in certain cases at least be considered to be an early expression of the disease process, a latent precox perhaps, in the sense of Bleuler.

Symptoms.—*Mental.*—A patient from time to time writes letters appealing for his discharge and his liberty and signing himself "The Emperor." It is this incongruity, this lack of oneness of the individual that for a long time has attracted attention in the symptomatology of this disease. How is it possible for a person so exalted as to think himself an emperor to plead in quite a natural way for his discharge from an asylum? The two positions which the man takes, acknowledging himself as a patient and pleading for his discharge while still proclaiming himself an emperor, show the possibility of maintaining two distinct and mutually opposed trends of thought

¹ Fundamental Conceptions of Dementia Præcox, Brit. Med. Jour., September 29, 1906.

at the same time without the one apparently interfering or serving at all to correct or modify the other.

This possibility is dependent upon a fundamental associational disturbance which has caused Bleuler to see a *splitting of the personality* as the foundation symptom of the disease and to give it the name, therefore, of schizophrenia. This splitting of the personality has been expressed less clearly in many of the theories that have been advanced to account for the symptoms. The symptoms have been said to be dependent upon a disintegration of the personality, a disintegration of consciousness, dissociation, apperceptive dementia, and the weakening of consciousness with the consequent impairment of the function of the real. Reality is unable to correct or adequately modify the delusional ideas, with the result that such bizarre, strange, and apparently unpsychological modifications of conduct are seen.

These unpsychological appearances are dependent upon what Bleuler calls *autistic thinking*, that is, a form of thinking to which Jung would apply the term introverted, in which the individual's interests are withdrawn from reality, and he occupies himself with himself to the more or less complete exclusion of the outside world. This is the field of dream formation, of phantasies, wherein things come true. An analysis of cases of dementia præcox shows that the delusion formations are based upon wish-fulfilling mechanisms which result oftentimes in highly symbolic, and to the observer, non-understandable expressions which are formulated in accordance with the particular complexes which may be operative. These mechanisms are the same as those observed in normal people, in hysterics, and those suffering from the various neuroses, but it is impossible for these individuals to adequately utilize them; they, therefore, result in impairment of efficiency and withdrawal from the world of reality. It would seem, too, that in præcox the regression is very much more profound than in the neuroses and the psychoneuroses, and for some unknown reason involves a serious disintegration of the personality which tends to become chronic and crippling. From the standpoint of this schizophrenic splitting of the psyche, based upon autistic thinking, many points in the symptomatology of the disease become understandable.

The peculiar *emotional dulness* and uncertainty of emotional response of the præcox has long been noticed, and Stransky¹ has particularly designated it by the term of *intrapsychic ataxia*, by which term he means a disturbance of the coördination between the intellectual and the affective attributes of the psyche, which are respectively known as the noopsyche and the thymopsyche. This noo-thymopsychic ataxia gives the appearance at times of emotional dulling and at other times of a senseless emotional reaction. Thus a patient who

¹ Ueber die Dementia Præcox, Streifzüge durch Klinik und Psychopathologie, Verlag von T. F. Bergmann, Wiesbaden, 1909.

receives news of the death of a near relative unmoved, may a little later on laugh heartily at apparently nothing. He is happy when he should be sad, sad when he should be happy, angry without cause, experiencing fear without reason, for the most part cold and impassive, but occasionally showing outbursts of marked and accentuated emotionalism. This is the "April weather" behavior of the affects, in the language of Stransky.

This condition of affairs is understandable upon the basis of the splitting of the psyche. The emotional reactions occur when the complexes have been touched, which the individual is constantly in an attitude of trying to prevent. This method of dealing with the emotions is well known, particularly in the formation of the dream, in which the *mechanism of displacement*, by removing the affect from the constellation of ideas to which it belongs and attaching it to an indifferent set of ideas serves to hide from the patient the realization of the actual difficulty. For example, a patient identifies herself with a schoolmate of her's and then accuses the schoolmate of being "bad" and pregnant. It will be easily seen that in such a case the patient is protecting herself from the realization that she has thoughts to which the term "bad" might be applied, and that the pregnancy which might result from being bad is transferred to her schoolmate. Under such circumstances, she, so to speak, unloads her emotion upon this schoolmate and very easily may produce the impression of indifference toward herself with an unmotivated affective attitude toward the schoolmate.

This *withdrawal from reality*, this looking within, occupying themselves with themselves, no longer subject to the corrective influences of the outside world, produces many surface indications, among which are *failures of voluntary attention, lack of interest, disturbances of orientation, disorders of memory*. The disorders of attention, lack of interest, failure of voluntary attention can easily be seen to be due to the turning of the interests within. The capacity for attention may be as keen as ever, but the patients are not attending to the things going on about them, but rather to the things going on within, and so they apparently take no interest in the people or the events of their environment. They may even express themselves as perfectly satisfied with their confinement in a hospital, and be so manifestly heedless of those about them that it is practically impossible to draw them into conversation. This lack of interest and attention naturally produce what appear to be disturbances of memory and orientation. The patient, who is heedless of his surroundings, may easily not know the day of the week or may have forgotten the events that only recently took place about him, because they were not sufficiently attended to to make any profound impression, while a patient who identifies himself with some great public functionary might easily not give the correct date of his own birth. Such considerations as these show how necessary it is to penetrate beneath the surface indications which

the patient manifests and find out their true meaning. They also indicate unequivocally that what may appear quite unpsychological is really perfectly understandable.

The same type of explanation serves to render clear the meaning of such surface indications as the *shallowness of thinking* and the apparent *dilapidation of thought*. The interests and the energies are occupied with things which are not accessible to casual questioning. In fact, as shall later on be seen, the patient may be wholly inaccessible to any form of approach, and when there is considerable speech productivity what is said may be so incoherent upon the surface as to be quite non-understandable and constitute what has been called a "word salad."

The *delusional formation* of precox is notoriously grotesque and partakes of this characteristic to such an extent in harmony with the grotesqueness of the thoughts in dreams that the similarity has not been overlooked. The mechanisms, too, are probably quite the same, although much more difficult to fathom because of the inaccessibility and lack of coöperation of the patient. If the eye is properly trained, however, to see meaning in the apparently meaningless, there will be little difficulty in seeing how certain expressions may be interpreted, even though in an individual case it may be impossible to verify such an interpretation. For example, an old precox who talked in a thoroughly dilapidated manner but was able with some patience to give a fairly good account of himself, injected into his series of replies to questions that the President was confined in an almshouse and that he had come to Washington to be President. He also stated that he had had something to do with his sister when he was a young boy, that he did not tell the priest and that his shadow was very heavy, that it was black, and that he saw the Devil in it. Here an expression of grandiose ideas, which speaking generally may be considered as compensatory are seen. In his autistic thinking he wishes to be the great man that in his real life he is not. Then one sees the possibility of a serious moral conflict, the result of incestuous relations with, or perhaps only incestuous thoughts about, his sister, while his dark and heavy shadow in which he sees the Devil can be easily seen to be a symbolic representation of the destructive effects which his moral delinquencies have had upon him. Here one also sees that the *hallucinatory experiences*, the *disorders of the sensorium* express themselves as *symbolisms of the conflict* and receive their interpretation with a knowledge of the nature of that conflict.

The delusions are essentially endogenous in origin, that is, ultimately dependent upon factors that are within the individual, and they tend to be colored and determined by complexes which lie at the very foundation of the personality, which have to do with the region of the psyche which has long since been forgotten and to which Freud gives the name "unconscious." It is largely because of their profoundly unconscious origin that they are inaccessible and it is largely

owing to this also that they produce such serious disturbances and such marked departures from conduct that is at all efficient.

Just as the hallucinations are symbolisms of the elements in the conflict so are the various *mannerisms*, *stereotypies*, and *neologisms*. All of these motor disturbances are in effect *symptom activities*, *complex indicators*, and serve in some way to portray the nature of the conflict. In a recently reported case, for example, an old precox was observed to keep pounding one hand with her clenched fist in a rhythmic stereotyped fashion. It was discovered that in her earlier days she had been jilted by a shoemaker. This peculiar action could be seen, in the light of this knowledge, as but the movements of the shoemaker pounding at his last. Many of the old cases of precox have such stereotyped activities which it is often, in fact usually, quite impossible to fathom, but in the light of such a case as this it will be seen that they must all be looked upon as having meaning and that for a complete reconstruction of the psychosis it is necessary to determine what that meaning is.

As an example of what painstaking analysis may disclose may be mentioned a patient of Jung's. She expressed herself in stereotyped and apparently meaningless phrases interspersed with neologisms. One of her statements was: "I affirm a million Hufeland to the left on the last fragment of earth on the hill above." A detailed analysis disclosed this sentence to mean, approximately: "For the bad treatment of the physicians which I have to endure here and with which I am tortured to death I claim a high indemnity."

Negativism, one of the characteristic symptoms of precox, may be expressed passively by the patient's not doing what is expected of him, or what he should do, or by actively doing the exact opposite of what is requested. The passive negativism may show itself in a refusal to attend to the promptings of normal desires, so that the bladder and the rectum are permitted to become overloaded and the saliva to collect in large quantities in the mouth, or, on the other hand, active negativism may show itself in the patient doing the exact opposite of what he is asked to do, for example, if he is asked to shut his eyes he opens them wider and if he is asked to open them he will shut them tightly, and if he is asked to put out the tongue he shuts his lips tightly, and if he is asked to shut his mouth he opens it, etc. This peculiar symptom is dependent upon what Bleuler terms the *ambivalency of ideas*, by which he means that every idea has connected with it by association its exact opposite more intimately than other ideas, and so the patient who is withdrawn from reality and objects to being invaded by the world of real things, who would, in other words, live within himself, finds that in refusing to accord with a suggestion from the outer world the path of the diametrically opposite reaction, is more patent, more accessible than any other. This is a type of reaction which one sees also in other conditions, as in hysteria, and also quite naturally in children.

The *suggestibility* of the precox receives a similar explanation. Some patients find it easier to follow blindly any suggestion which comes to them from any source than to actively initiate contact with reality. One precox patient, for example, had to be fed by placing his food before him and a spoon in his hand and repeating each time the command to take another mouthful. After having responded to the command he remained inert until it was repeated. This form of activity requires no initiative on the part of the patient, no actual effective contact with reality of his own devising and permits him to remain within himself, much as does the negativistic tendency.

The *catatonic rigidity* of the precox is a still more active shutting out of the world to the point of absolute inattention to the environment, but an inattention which is positive and active rather than passive, as in catalepsy and command automatism, while *stupor* still more effectively shuts out the world of real things.

There is a reaction type in dementia precox which is important for an understanding of the nature of the disease. It is the *archaic type of reaction*. In the illustrations that have been given it is seen that when the individual under the influence of mental disease regresses he not only reaches lower levels, but frequently reaches levels corresponding with his early infancy, and so it is frequently found that the delusions and other morbid manifestations only find their explanation when traced back to the infantile period. In the same way regressions may lead back to lower cultural levels so that patients show symptoms that are only understandable in terms of the psychology of more primitive peoples. The materials of experience are used by the patient in an archaic way. One such patient with a very complicated delusional system states that he is the father of Adam, that he has lived in his present human body thirty-five years, but in other bodies thirty million years, and that during this time he has occupied six million different bodies. He has been the great men in the history of the development of the human race; he himself created the human race; it took him three hundred million years to perfect the first fully developed human being; he is both male and female, and identifies all the different parts of the universe with his own body; Heaven, Hell and Purgatory are located in his limbs, the stars are pieces of his body which have been torn apart by torture and persecution in various ages of past history; he is the father and creator of the various races and elements of the human organization, etc. Here is a very primitive type of thinking in which the patient identifies himself with the whole universe somewhat as the baby does and somewhat as primitive man does. He is quite in the position of the chief of some primitive tribes in whom all the forces of the universe center and from whom radiate all of these forces for good or for ill. This is the archaic type of reaction which shows how deep the split of the personality may be, how fundamental it may be, and gives an insight into the seriousness of the disease process.

Physical.—A considerable number of precox patients, particularly the catatonic and the hebephrenic, show marked physical symptoms, and not infrequently have all the outward appearances of being quite ill. They often emaciate during the early period of their illness, suffer from anorexia and insomnia, circulatory disturbances, disturbed cardiac action, cyanosis of the extremities, vasomotor disorders of which dermatographia is not an infrequent manifestation. The deep reflexes are commonly exaggerated, while the pupils in this class of cases are characteristically widely dilated. *Convulsive seizures* of an epileptiform, but more often of an hysteriform, variety may occur.

In the very early stages of the disease physical symptoms which do not lead to the suspicion of mental disturbance are not infrequent. Such symptoms as headache may be in evidence for a considerable time as may also vertigo, and Urstein¹ has called especial attention to the occurrence of gastric disturbances. Other physical disturbances may also of course occur, and if no adequate foundation can be found for them a mental origin should be thought of.

It is this type of physical illness that has led to and maintained the belief in etiological factors at the biochemical level.

Mode of Onset.—The early manifestations of precox vary greatly. As already indicated the disease may remain latent for a considerable period, manifesting itself only in slight disturbances, predominantly of a physical nature, such as headaches and gastro-intestinal disorders. In quite a large percentage of cases a "shut-in" type of character has been found to have existed for a considerable period before the outbreak of the symptoms, in fact to have been a characteristic of the individual before the breakdown. Whether this is to be considered as a symptom of the disorder or an expression of the type of individual in whom the disorder is possible cannot be answered at this time.

In sharp contrast to these latent periods the disorder develops not infrequently with great suddenness. After some emotional shock the patient becomes almost immediately greatly confused or catatonic.

It is quite frequent to have the break-down be a slowly progressive developing condition. During the early period the symptoms may take all sorts of forms and may easily be mistaken for types of manic-depressive psychosis, compulsion neuroses, neurasthenia, hysteria, hypochondria, acute confusion and paranoid states. Anything atypical in these psychoses should make one think of the possibility of dementia precox.

The varieties of dementia precox will be described under five heads: I. Dementia Simplex; II. Hebephrenia; III. Catatonia; IV. Paranoid Forms; V. Mixed and Atypical Forms.

I. *Dementia Simplex.*—In this group of cases the origin is usually insidious, perhaps manifesting itself only by slight physical disturbances, such as headache, gastro-intestinal attacks, by some irri-

¹ Die Dementia Praecox und Ihre Stellung zum manisch-depressiven Irresein, Berlin u. Wien, 1909.

tability, and perhaps a tendency to withdraw from the association of others. The patient may suffer from insomnia, perhaps disagreeable dreams, and there may be passing evidences of hallucinations and delusions. These are apt to be expressed only at times, perhaps under the influence of a little excitement, and show little tendency to organization or progression.

The patient emotionally is more apt to be somewhat depressed, or at least indifferent, lacking in initiative, and presenting outwardly an appearance that frequently leads to the diagnosis of a "nervous break-down," "nervous prostration," or neurasthenia.

Such mild attacks as this may be pretty well recovered from but may recur, and one not infrequently finds a history of one or more such attacks having preceded a more severe break-down.

It is this group of cases of mild abortive forms, "*formes frustes*" of the French, which after recovery from a "flurry" or "excited episode," gives one the impression of "peculiar characters." Many such cases are found in the ranks of the criminals, hoboies, prostitutes, pseudogeniuses, cranks, and eccentrics, and accurate analyses of the histories in these cases would not infrequently show a precox episode which separated a period of relative efficiency in their lives from the period following relative inefficiency, in which latter it might be possible to detect certain precox residuals.

An analysis of the life histories of this class of cases shows quite regularly a constant tendency to slip from under all forms of responsibility, and a lack of capacity for any kind of continuous application and inability to develop the habit of work. They characteristically resort to the hobo type of existence, are unable to adapt, with any degree of efficiency at all, complex conditions of life, and wander from place to place, occupying one position after another from which they are either dismissed because of inefficiency or leave voluntarily, giving reasons for so doing which are totally inadequate. Such cases as these, sometimes by a steady progress become very greatly dilapidated, and it is quite surprising at times to find the amount of deterioration in such cases after their admission to an institution and to realize how long they have gotten on in the outer world in a seriously mentally crippled condition. Of course their continuance in the outer world was made possible only by their having sunk to low and relatively simple social levels that made little or no demands upon them.

When such patients as these get into situations which require continuity of effort and constant adjustment and from which they cannot escape they not infrequently suffer from severe and more acute breaks. This is seen in the military service. The Army and the Navy naturally attract this wandering horde of inefficient who see in the military service only the glitter of brass buttons and the opportunity to see the world. After enlistment, however, when they are required to take up the grind of daily work, their defects soon come into the foreground and manifest themselves either by distinct

psychotic episodes or by minor infractions of military discipline, such as staying away from the post beyond the leave granted, or perhaps more serious offences, such as desertion. Such cases when finally they come under observation in a hospital easily show their defect.

Many women of this type marry, have children and although looked upon as "unique," or "queer," nevertheless get along if there is no economic strain.

II. *Hebephrenia*.—This form of dementia precox is more severe than the preceding. It, however, is not essentially different, being practically the same condition occurring with greater severity, more apt to be somewhat more acute in its onset, and manifesting itself more prominently by hallucinations and delusions.

The onset of hebephrenia not infrequently presents the outward appearances of a depression and so resembles the depressive phase of a manic-depressive psychosis, or perhaps some other form of depression. It not infrequently happens, on the other hand, that the first appearances of the disorder are those rather of mental confusion than of a marked depression. These early manifestations are the method of reaction of the individual to the first outbreak of the conflict. He may become either greatly depressed or may be quite unable to react along any well-defined line and become quite confused. From this acute condition, after a longer or shorter duration, he settles down into a more stereotyped expression of psychotic symptoms.

During these early stages the productivity is not infrequently deliroid in character, accusing voices are heard and rather ill-defined and not well-formulated delusions are expressed which are characteristically of a self-accusatory type and in harmony with the depression. Attempts at suicide not infrequently occur during this period.

After the active symptoms of the onset have subsided there is a settling down into a more stereotyped delusional expression, but not infrequently with a more or less incoherent productivity and with expressions that sound fantastic and silly. One patient complains that the sheets stick to his feet, another than he is the "wandering planet." Such ideas appear to have no adequate reason and are expressed quite disconnectedly from the general train of thought and little or no attempt is made to support them by logic. One patient, for example, says that his enemies are following him, and that he has been killed a number of times. Another complains that other patients are trying to injure him. All of these statements are made without show of emotion in a decidedly matter-of-fact way. Such appearances as these have led to such descriptive phrases as *looseness of the train of thought*, *poverty of ideas*, *emotional deterioration*. From the previous discussion, however, of the meaning of symptoms it is known that the hallucinations are expressions of the conflict, that the delusions are compromise formations, and that the apparent incoherency of the speech does not necessarily imply an incoherency in the thought content. It is usually not especially difficult to find some fairly direct

connection between the accusations of the voices and actual occurrences in the patient's life. Similarly with other disorders of the sensorium. One patient for example, who had seduced a girl, who bore him an illegitimate child, saw visions of his mother and heard the young woman's voice telling him to come home, to go to work, and lead a decent life.

The peculiar emotional reaction, the lack of interest, and apparent emotional dilapidation, as noted, is due to displacement, and so it is perfectly understandable that when an effort is made to gain access to such individuals that they show little interest and characteristically reply to questions addressed to discovering the reasons for their actions by "I don't know." It is really quite true that they do not know the reasons for their actions any more than any person understands the reasons for the different appearances that have come to him in a dream. And then again these patients are so occupied with themselves that they do not care often to be interfered with, and the "I don't know" is as much as to say "leave me alone."

The general conduct of the patient may also be listless, apathetic and disinterested in character and in harmony with his emotional condition, whereas mild alternations of depression and of excitement with the development of little peculiarities of conduct and speech show the alliance between this group of cases and the catatonics. One patient, for example, who thought that he was ordained to preach and that the bread was impure, would be quiet for months, and then exhibit a certain degree of restlessness by following the doctors and nurses about the wards telling them his troubles for a few days. The alternation may be much more marked as between a state of stupor and a state of excitement in which the patient eats paper, strings, and bedecks himself with all manner of trash and shows outbreaks of violent anger. This condition approaches much closer to catatonia.

Many of the cases, however, do not show that amount of dilapidation which the previous description would imply, while any degree of incoherence may be seen up to the production of a veritable "word salad." On the other hand the patients may be found well oriented, making outwardly a natural appearance, being able to give a very good account of themselves, but presenting a grotesque delusional system, supported by hallucinations the expression of which stands in rather striking contrast to the outward appearances. These delusional systems represent all degrees of coherence and it would seem that the ability on the part of the patient to formulate a coherent and consistent delusional world was one expression at least of his capacity to react to the destructive influences of the conflict.

One such patient complained of illness on a train and told the conductor that she had had a hemorrhage. No sign of any hemorrhage was in evidence, however, and when she reached the sanitarium to which she was removed nothing abnormal was found on examination. When her mother arrived shortly afterward she was found

delirious. Later, upon her admission to a hospital she was very hypochondriacal, talked at length, complained of attacks of hemorrhage from the vagina, which hemorrhage she said was due to an assault while she slept either by a man or some sharp instrument. Later on she claimed to be married to a Mr. O. and wrote long letters giving an account of her troubles and giving an account of the hemorrhages and the supposed operation, showing an extensive delusional system. She claimed to have been the victim of a criminal operation performed by a doctor. She later said that she had been engaged to a Hebrew before she was born and that at twenty she broke this engagement, that at three years of age a Catholic priest requested one of her offspring be given to that church for a clergyman and that the Hebrew family consented. In the midst of a great mass of incoherent jumbled delusional expressions evidences of a compensatory



FIG. 307.—Appearance of patient in catatonic stupor.

wish-fulfilling delusional system are found. Wishing for a child she became impregnated. Being a virtuous woman this has to be accounted for. She therefore has the delusion that she is married to Mr. O. Inasmuch as no child appears a delusion that she has had an abortion performed accounts for its absence, and as this is a criminal operation it was performed without her knowledge while she slept. These delusions are mixed up with all sorts of ideas about the dislocation of her various viscera, numerous hemorrhages from the vagina, and a fractured skull, etc. It is loosely organized and interrupted by violent outbreaks, restlessness and irritability.

She has been unable to adjust to reality, the effort at compensation by delusional formation has also failed, with the resulting progressive dilapidation of coherence in the stream of thought.

III. *Catatonia*.—This variety of precox, like the hebephrenic, may come on suddenly with symptoms of confusion or depression, or may be of somewhat slower onset. It is more apt than the other forms to be of relatively acute onset, in which case it sometimes follows a sudden shock of a highly emotional character. It is characterized more

especially by a predominance of motility disturbances and tends to express itself in alternating conditions of *catatonic stupor* and *catatonic excitement*.

In the conditions of *catatonic stupor* negativism reaches a very high degree. The patients are perfectly immobile, sitting off in corners by themselves or lying in bed without paying any apparent attention to what goes on about them, are quite inaccessible, fail to answer ques-



FIG. 308.—Cataplexy; flexibilitas cerea.

tions, and do not react at all to stimuli from the outer world. They characteristically often refuse to speak at all. This mutism is a manifestation of the negativism. Besides this the patients often refuse food, pay no attention to the promptings of the bladder and the rectum which become overloaded with urine and fecal matter, often to a serious extent. Saliva may be permitted to accumulate in the mouth where, if attention is not paid to it, it may undergo putrefactive changes. The patients quite characteristically show peculiar

theatrical attitudinizing, make grimaces, occupy peculiar positions, and if they speak the productivity is often incoherent and apparently senseless, with a tendency to constant repetition of the same phrases—*perseveration*—which may also manifest itself in the movements of the body, such as a constant swaying movement or the like.

The muscular system may be in a condition of *waxy flexibility*, permitting of the molding of the limbs into any position where they remain indefinitely—*catalepsy*. When this is present the patients tend to show a more or less high degree of suggestibility and *command automatism*, doing mechanically and in a perfectly automatic manner that which they are commanded to do. This suggestibility shows itself further in *echolalia*, the repetition of words or phrases that are addressed to them or that they hear others speak, and *echopraxia*, the repetition of movements which are made in their presence. On the other hand there may be a marked degree of *muscular tension*, the patient maintaining fixed attitudes with the muscles thrown into a condition of rigid contraction. The limbs and body are stiff and immobile and resist any effort at bending. Patients in this condition are positively negativistic, withdrawing from all approaches, refusing any coöperation with the nurse in attempting to dress or undress or feed them, and show a tendency to react by doing the opposite of what is expected of them. In this condition of stupor the patients may appear to be quite disoriented and have no knowledge of what is going on about them. If they are watched, however, they may be seen at times to show evidences of paying attention to their environment, and not infrequently when the stupor passes they are able to give a fairly good account of the things that happened during it, but show no capacity for explaining their strange conduct.

In the opposite condition of *catatonic excitement* there are marked degrees of activity, constant talkativeness and noisiness, sometimes destructive and impulsive tendencies manifested by breaking windows or attacking those about them, but in general showing, as does the speech, a marked lack of coherence. Both the productivity and the activities of the patient fail to show any clear goal, although some patients show what very closely approaches to the flight of ideas of the manic. Catatonic excitement may reach a very high grade, manifesting itself by wildly deliroid reactions, constant motor unrest and sleeplessness, a rapid failure in nutrition, a veritable status catatonicus. Some of these catatonic cases show hysteriform or epileptiform seizures, and death occasionally results in these higher grades of excitement.

The chronic conditions tend to show well-defined mannerisms, such as the peculiar attitudes of the body, especially clumsy ways of holding the spoon or the fork in eating, meaningless grimaces, odd ways of walking, such as sliding the foot back and forth two or three times before starting off, and other ceremonials for initiating movements. They are characteristically stiff, awkward, clumsy, and inaccessible,

and usually indifferent to their surroundings, and apparently emotionally dull, though given at times to emotional outbreaks without apparent cause. All of these psychical symptoms of course must be considered as having some psychological meaning. In harmony with what has been said before both the types of increased suggestibility and catatonic rigidity are ways of shutting out the world of reality, whereas the peculiar automatisms can sometimes be traced to their meanings, as in the case before mentioned of the young woman who kept constantly pounding her hand with her clenched fist.

Physical Symptoms.—The general appearance of the catatonic forms of precox indicate more than in the other varieties the presence of definite physical illness. Exaggeration of the tendon reflexes, lower-



FIG. 309.—Dementia precox; mannerism.



FIG. 310.—Dementia precox; mannerism.

ing of cutaneous sensibility, vasomotor disturbances, cold and cyanosed extremities, widely dilated pupils, disturbances of secretion, and loss of weight are frequently observed. It is this group of cases that have led most distinctly to a toxic theory of etiology and caused the precox cases to be thought of in connection with disturbances of metabolism due to changes in the internal secretions.

IV. *Paranoid Forms.*—In the paranoid cases there is a much more efficient effort at creating a coherent and logically connected series of delusions and associated hallucinations. In these cases there is much less outward evidence of the dilapidation and emotional indifference that have led to the belief in the presence of a permanent mental impairment, a dementia.

These patients, originally, many of them at least, were grouped with the paranoias, but under the influence of Kraepelin that group of paranoid conditions which showed a tendency toward progressive deterioration were included in the dementia precox classification. All degrees of intervening possibilities, however, occur so that one may get a fairly well-knit delusional system in a patient who deteriorates quite rapidly, whereas other patients maintain their intellectual integrity over a period of years. The *déire chronique* of Magnan is generally considered to be dementia paranoïdes, and this group of cases in general have been recently included by Kraepelin under the designation of *paraphrenia*. Some authors today, more particularly Bleuler, are inclined to think that perhaps the so-called true paranoias are only attenuated forms of dementia precox.

The delusional system in these cases must be conceived as a compromise formation and as essentially wish-fulfilling, and is characteristically sexually colored. One such patient, a middle-aged woman, was persistently persecuted by a man who accused her of leading an immoral life to such an extent that she finally went to a physician to be examined to prove her virginity. All sorts of vulgar remarks were constantly made about her, she was referred to by the voices as the widow of this man. The patient was a devout Catholic and the man was a Protestant, a perfectly understandable reason for a severe emotional conflict, which in her delusional system unloads itself upon the man rather than acknowledging its true origin. This whole conflict arose at the time of the death of her brother and it is significant that in her delusions she believed that she had received letters accusing her of incestuous relations with her brothers, of having become pregnant by them, and of destroying the pregnancy. These letters were addressed to her as the wife of her several brothers. Here one sees an infantile determiner for the delusional system in the love which she entertained for her brothers and which later was transformed, under the influence of the conflict, into delusions of an incestuous nature. Finally, there was a whole crowd of people who were conspiring against her and whose object it was to kill her. Such delusional systems can without much difficulty be seen to be expressions in distorted form of the conflict. She has never been able to get away from her infantile attachment to the members of her family and establish herself upon her own feet, and these attachments hold her back, produce a withdrawal from the outer world, a regression into the world of phantasy, with a tendency toward what may be termed psychic death, or an absolute lack of efficient reaction to reality, and this is symbolized by the gang of persecutors who are bent upon her destruction.

V. *Mixed and Atypical States*.—All of these forms of dementia precox so far described are simply variants, in accordance with the present viewpoint, of one disease trend, and so it is not strange that it should be found that there are no hard-and-fast lines separating

one from the other. In consequence it is not infrequent to see transitory or mild motility disturbances in cases that are essentially hebephrenic, or to see fairly well-defined paranoid delusional formations in either the hebephrenic or the catatonic varieties.

As to the atypical forms, there has been mentioned and described under dementia simplex the larvated and abortive types, the "formes frustes." These are probably much more frequent than ordinarily supposed. For example, Wilmanns, in a study of 127 vagabonds, found 66 cases of dementia precox, while undoubtedly it is not infrequent for these abortive forms to be considered as cases of constitutional defect. This difficulty becomes very much more aggravated when it is remembered that attacks of dementia precox may occur in childhood, according to Vogt as early as five years of age, while a little later, nearer to the period of puberty, they are not so uncommon. In these cases of course mental development is interfered with and remaining upon a lower level gives the impression of imbecility. The same difficulty arises in a somewhat aggravated form when dementia precox develops in young persons already of defective make-up. Here, of course, the differentiation is no longer possible, because it must be realized that both conditions of defect and precox are present in the same individual.

Pathology.—There is little that is distinctive in the pathology of precox. It is probable that during the life of the individual disturbances that are shown in the general physical manifestation of the disease occur at the biochemical level. Beginning degenerative changes in the vessels are sometimes found, and tuberculosis is quite frequent, but is easily understandable as being dependent upon the general lowered physical condition and inactivity of these patients, coupled with their bad habits and the necessity of their rather close confinement. Such changes as are found in the nerve cells are of a degenerative character, with evidences of neuronophagia and with perhaps ameboid glia cells. The acute cases of catatonic excitement which end fatally, the so-called catatonic "Hirntod," show a certain amount of evidence of degeneration, more particularly, however, evidences of a chemical nature in the form of certain lipid elements in the cortex, which seem to be products of disintegration.

Southard has found certain anomalies in precox brains which appear to be of the nature of aplasias or ageneses. These agenetic or aplasic areas appear to be grouped more or less in correspondence with the three main types of the disease. The frontal region is characteristically involved, while he has described a cerebellar group corresponding to the catatonic variety, while the profound emotional disturbances he thinks are due to lesions in the deep layers of the cortex which have no direct motor, sensory, or perhaps associational relations.

Nature of Dementia Precox.—From the description of dementia precox up to this point it will be seen that it presents correlations on

the one hand to the more distinctly so-called functional types of disorders, such as the psychoneuroses, and on the other hand to the more distinctly organic diseases. It has been seen that it was possible to formulate all of the mental symptoms in the same way that they are formulated in the psychoneuroses. In other words, the mental symptoms are capable of interpretation solely at the psychological level. On the other hand, for an understanding of the whole disease process it must not be lost sight of that recent investigations are tending to show more and more that there are distinct biochemical disturbances during life and pathological changes are being found after death. In any case the acute cases that lead to death must be conceived as having profound bodily changes correlated with the psychic symptoms.

The formulation of the disease from either standpoint alone must of necessity be unsatisfactory. For example, the theory which makes the etiology toxic fails utterly to give a comprehensive idea of why the mental manifestations take the particular form they do, why for example, in the case already cited, the hallucinatory voice should be that of the young woman that the patient had seduced, telling him to lead a decent life. This is surely the patient's self-critique appearing in hallucinatory form.

It would seem, especially in view of the type of archaic reaction which has been described and which is found quite frequently in precox, that the schizophrenic splitting of the personality is more severe, more profound, than in the so-called functional disorders, in other words, that it goes to deeper levels, that the infantile and the archaic in the individual have been less efficiently sublimated and so gotten away from, and therefore they control more effectively the thoughts and acts.

At any rate, it seems quite evident that the great difficulty of conception here is due in part to the quite arbitrary separation of the individual into two distinct and mutually exclusive parts, namely, mind and body. The dualistic hypothesis that conceives of the mental and the physical going side by side without mutual interference or interac-



FIG. 311.—Phantastic paranoia.

tion is responsible for such a conception. From the medical standpoint such a concept seems entirely unnecessary, for one is met at every turn with the intimate relations constantly maintained between the two, and therefore come naturally to consider the human being as a biological unit presenting types of reactions that at one extreme are predominantly psychic, while at the other they are predominantly physical, but which present every grade of intermediary type. The severe mental disturbance of the nature of anxiety that goes with certain cardiac and pulmonary diseases is well known, as are also the physical upsets, particularly the gastro-intestinal, that are associated with certain mental conditions such as worry. The interrelations between the mental and the physical are a matter of daily observation. Occasionally, however, they are very pronouncedly emphasized. A chronic patient who had been for many years in a hospital, working daily at out-door labor was suddenly seized with a violent impulse in which he attacked all about him. He seized a heavy iron bar, killed two people and injured another, and ran head-long and wildly without direction into the woods. He was finally cornered, and in the process of securing him he was shot by a farmer with a load of buckshot, none of which, however, penetrated further than through the skin and produced no serious wounds. He was brought back to the hospital, incoherent, mumbling and trembling, showing all the evidences of a tremendous emotional upset. The shot were picked out of his skin, the wounds were dressed, and he was put in bed. Up to that time he had been a strong, physically healthy negro. He never left his bed again, and approximately a year afterward he died, having developed an acute tuberculosis. Such cases as these demonstrate the necessity of considering the human being as a unit and not endeavoring to draw hard-and-fast lines of distinction between the mind on the one hand and the body on the other.

In our present state of knowledge, however, we are quite unable to make any specific correlation between the physical findings and the mental symptoms, while on the other hand it is quite possible to express the symptomatology of the disease, to describe it, to, so to speak, reconstruct the psychosis purely in psychological terms. For the present, therefore, the disease must be described psychologically, and the explanation of the mental symptoms must be sought psychogenetically, without, however, forgetting that there are certain somatic changes which are pretty generally attached to the symptomatology of the disease process and which must ultimately be made to fit into the general rubric before a complete understanding of the entire situation is had.

On the psychological side, then, dementia precox is seen to be a certain type of reaction to a mental conflict, resulting in a splitting of the psyche and the outcrop of unconscious mental trends to the surface of the mental life. The patient is confronted with a situation to which he cannot adequately adjust, which is absolutely inaccept-

able and impossible, and he is therefore driven away by his incapacity to assimilate it and cast back upon himself. The battle of the opposing forces produces the disease picture which is the outward evidence of the effort on the part of the individual to reach a solution of the difficulty. The symptoms are the result of the appearance of the unconscious trends distorted and disguised as they are in dream formations. Jung has especially noted this similarity to the dream state and would consider precox as a sort of waking dream or dream from which the patient does not awake, the dream picture being fixed, as it were, by another element in the situation, the toxin, which acts like the fixing agent in the photographic process. It is seen that the conception of the disease as being from the beginning and fundamentally a dementia must be modified, if by dementia is meant a permanent



FIG. 312.—The fetal attitude assumed in severe grades of regression. This attitude was maintained most of the time for years, the patient seeking the darkest corner of the ward.

mental impairment, a mental loss, which is thought of in the same terms as a loss of tissue from a wound. It would appear from the description of the mechanisms that have been given that at least in the early stages of the disease there is nothing at all corresponding to such a permanent loss; that what has happened has been a disintegration, a falling apart of the component parts of the psyche and a shifting of relative positions, more particularly a shifting, or to use the technical term, a displacement of the emotional content of certain idea constellations. The dementia, therefore, which has been described as such, is at least to this extent a misnomer. From the point of view,

however, that this disintegration and resulting impairment is either permanent or tends to be, it has a meaning.

The psychological side of the situation, however, we have seen is not all. There are certain physical changes in the course of the disease, and certain pathological findings. So far as our observation goes, however, the etiological factors lie almost, if not quite entirely, in the mental sphere, and one must therefore conceive of the physical changes as superadded. This is a possibility which was well illustrated by the case already quoted of the man who died after a tremendous emotional explosion during which he killed two people. When the psychic splitting is profound and when it is of considerable duration it is quite understandable that it should unloose bits of physiological mechanism and thus produce the physical changes found. From the descriptions of the mechanisms in the psychoneuroses, taken in connection with

the discussion of the vegetative nervous system, it may be seen that constantly operative psychic disturbances are capable of producing the physical changes. Compare Crile's study of the emotions and Cannon's work on the relation of the psyche to the gastro-intestinal functions where it may be seen that both surgeon and physiologist are forced to put the psychic factor in the foreground.

Ways of Getting Well.—With the concept of the disease process which has been previously elaborated, what is the significance of the three main types of precox which are found clinically? Bertschinger¹ has recently made an admirable study of the process of recovery in precox and his discussion of the nature of the conflict and the way of adjusting is illuminating.

The three clinical types of the disease are the expression of the interplay of the two factors, the conflict and the reaction, the severity of the former and the efficiency of the latter determining the outcome in the individual case. The degree of confusion in the acute onset would then be an expression of the completeness with which the patient was driven back from reality and the dominance of the unconscious trends. Conditions of moderate confusion with capacity for adequate reaction to reality at times, or under the special stimuli of, for example, questions, show that the patient still has a certain grip upon the real world and is making an effort at least to retain it. Certain other cases of quite clear consciousness with complete orientation show a very adequate grasp upon reality, and these patients, to the casual observer, often seem quite natural. In such patients, however, one will notice interference of thought, hesitations in the course of conversations, stutterings and stammerings over certain points, the evidences in other words, of complex interferences, and the patients will complain that from time to time their minds seem to be absolutely blank. These *thought deprivations* and *saltatory associations* are the expressions of reactions to buried complexes, so that in these patients there is a fairly adequate grasp upon reality for ordinary purposes at least, with only spasmodic influences from relatively restricted areas.

In general, then, the hebephrenic type may be seen to be a reaction to the conflict which is essentially inadequate and inefficient. In the acute stages the patient may be absolutely overwhelmed by the conflict, disoriented, and confused. Later on the progressive disintegration and dilapidation of thought indicates the slowly progressive conquering of the capacity for adjustment to reality by the invasion into consciousness of the unconscious trends.

The catatonic type represents a somewhat different form of reaction. Here the patient is oftentimes suddenly overwhelmed by the conflict, as under circumstances of accident or sudden and severe shock. No attempt at adjustment is made at first, but the whole situation is actively and definitely shut out. Here there is an active effort on

¹ Heilungsvorgänge bei Schizophrenen, Allg. Zeits. Psychiat., Band. lxxviii, Heft 2. Translated in Psychoanalytic Review, 1915.

the part of the individual to exclude the offending tendencies, and when this succeeds recovery takes place as the result, so to speak, of the encapsulation of the objectionable material, and its exclusion from consciousness. This form of the disorder is the most acute, and the recovery is equally most apt to be prompt, and it will be seen from this explanation why this is so.

In the paranoid form of the disorder the reaction is much more efficient than in the hebephrenic variety, and in some respects less efficient than in the catatonic. Here the individual takes a flight into a psychosis, and the delusions are the expression of a compromise between the opposing psychic trends. Unable to live in the real world the patient succeeds in inventing a world in which he can live and having invented it he succeeds in getting along fairly well without noticeable deterioration. The conflict in these cases tends to become stationary after the development of the delusional world.

Bertschinger has more especially defined the ways of getting well by pointing out that the patient in recovering may, as the result of his conflict to which he cannot adjust, find a compromise by changing himself and interpreting the world of reality in terms of his morbid phantasy, or by translating the world of his phantasy into terms of external experience. And so one would find on the one hand delusions of grandeur which are a compromise formation and serve to change the individual so that he may be better satisfied with life, and on the other hand one finds delusions of persecution, the delusions of influence from the outer world, that serve to change the outer world in conformity with the patient's complexes. These outside influences are but the reflections back upon the patient of his failures to get from the world what he wants, and they are consequently felt as malign and destructive influences. Another method of getting well is that already described of the catatonic, the shutting out and encapsulation of the conflict in a circumscribed amnesia.

In many cases conversions into bodily symptoms, such as are found in hysteria, are found. Another method of getting well is by living through a series of imaginary experiences which brings the complex to a logical conclusion. For example: A young Japanese woman was overwhelmed by the sad news that five members of her family had been killed in battle. She passed instantly into a dreamy state of consciousness, went on with the work of the household just as if all five were members of it, made their beds, set their places at table and acted in every way as if they were alive and present. Finally she, so to speak, let one of them die and then another and another until finally she had compassed the death of all five, after which she awoke from her dream-like state and was well. She had succeeded in an efficient reaction to the situation by its attenuation, extending it over a considerable period of time. Finally, a certain number of patients get well by the final domination of the reality motive, with a resulting correction of their delusional phantasies.

Course and Progress.—As will be seen from the description of the disease the catatonic form is more apt to be acute in onset and it has the best prognosis, while the hebephrenic form and the simple dementing varieties tend to progressive deterioration, and the paranoid form tends to remain stationary without material deterioration. A few of the hebephrenics get well, more of the catatonics recover, but all of these cases are liable to recurrent attacks. According to a recent study of Zablocka¹ of 515 cases, 60 per cent. proceeded to light, 18 per cent. to medium, and 22 per cent. to severe deterioration.

In the catatonic cases that recover there is usually an appreciable change in the individual. The encapsulation of the conflict means that the recovery has taken place by a sacrifice of a certain portion of the personality. The portion of the personality in which the conflict is resident, so to speak, has had to be cut out or walled off, and the energies from this region are no longer available by the individual. This loss is quite characteristically shown in certain changes of character, indicating that the individual has gotten well, but has paid a certain price for that result.

The cases that do not get well tend to regress to lower levels, and quite characteristically, in institutions at least, they finally reach a level on which they can maintain themselves without sinking further. In this condition they remain stationary for long periods of time. Quite often patients brought to an institution in a very much disturbed condition settle down quite promptly under the simpler conditions of institution life, while in the outer world the precox cases tend to gravitate into the ranks of the hobo, the prostitute, and the petty criminal. Here they finally find their level and get along after a fashion.

Acute and severe grades of regression, in which the splitting goes to the very foundation of the personality, often expresses themselves by suicidal attempts and sometimes by homicidal attempts. The possibility of such attempts may sometimes be foreseen in the dreams.

Remissions are quite the rule and come about under circumstances which reanimate the conflict. Often patients get along very well in an institution, but become upset shortly after going back to the conditions under which the conflict developed.

The course of the disease is irregular and prediction is quite impossible. In a general way, however, one gets clues from keeping in mind the fact that the conflict is between the reality motive and the phantasy formation, and by watching the interplay of these two motives one gets an idea as to whether the reality motive is gaining or losing ground.

The question of a recovery is a mooted one, but in general it is conceded that the patients that recover do present to careful analysis certain residuals. Of course, however, these residuals may be of any degree and may be so slight as to not necessarily impair the individuals

¹ Zur Prognosestellung bei der Dementia Præcox, Allg. Zeit. f. Psychiat., Band. lxy.

in the position in life which they may occupy. It is possible that true recovery may take place with a resolution of all the symptoms, but if this is so it is probably the exception.

Diagnosis.—The diagnosis of dementia precox, while comparatively easy in the well-defined and the advanced cases, becomes a matter of great difficulty during the early history of the illness or in cases in which the symptoms are mild.

The manic-depressive psychosis presents one of the characteristic difficulties. In this psychosis there is usually a history of repeated attacks without deterioration. It must not be forgotten, however, that precox itself frequently presents a similar history and that if deterioration is present it may not be prominently in evidence. The depression which so frequently ushers in a precox attack may easily be mistaken for a depression of the manic-depressive psychosis. The manic-depressive retardation is similar in its outward appearances to the indifference and perhaps lighter degrees of negativism, particularly the inaccessibility of the precox. The manic-depressive is more apt to have delusions of a self-accusatory type than the precox, and the latter's delusions are more apt to be grotesque. They more frequently are evident distortions and symbolisms. The pressure of activity of the manic-depressive resembles the activity of the catatonic. In the former, however, the activity, although rapidly changing as to its object, is characteristically addressed to some purpose, while in the catatonic the activity is more diffuse and has less direction. It is more incoherent.

In the early stages the mild depression of the precox may simulate that of a neurasthenic, or the agitated depression may simulate that of an anxiety neurosis. In both instances the precox is more apt to show grotesque delusions and conduct disorders of a bizarre nature, such as tearing his clothes, self-mutilation, or, on the other hand, characteristic negativistic symptoms, such as retaining the saliva or the urine, withdrawing from efforts addressed to assist him, refusing to coöperate in changing his clothes, the refusal of food and the like. It must not be forgotten, however, that the etiological factors of the actual neuroses may operate in the same person who breaks down as a precox, and that therefore neurasthenic and anxiety symptoms may be present as expressions of these etiological factors. It is important to keep such a possibility as this in mind when it comes to the matter of treatment.

It is still a mooted question whether there are not conditions intermediate between precox and manic-depressive psychosis which partake somewhat of the characteristics of both. We know there are a considerable number of cases in which the difficulties of diagnosis are very great. Flight of ideas may be quite typical in precox, for example, while the depression of the precox may resemble very closely that of the manic-depressive. The difficulties are greatest with that class of manic-depressive psychoses known as the mixed states.

Epileptiform and hysteriform episodes may lead to a diagnosis of epilepsy or hysteria. It must not be forgotten, however, that it is possible to have precox complicated with epilepsy and that in dementia precox all of the symptoms and characteristic mechanisms that are found in hysteria may be found.

In the infection and exhaustion psychoses the differentiation may be very difficult and it is necessary in such patients often to wait for a considerable time until the subsidence of the infection and then see whether the case clears up, as it usually does if it is a simple infection psychosis. One has to be very careful in making a prognosis in cases of this character. Not infrequently cases of so-called puerperal insanity are really cases of precox which have been precipitated by the circumstances of the puerperal period, loss of blood, prolonged labor, infection, or the mental stress incident to an illegitimate pregnancy.

From paresis the differentiation can now be made by the laboratory methods at our disposal. One must not forget, however, that the precox may have syphilis and therefore a positive Wassermann in the blood serum. In fact, not a few do show this reaction, and it is quite readily conceivable that the presence of an uncured syphilis may well be a precipitating factor in the outbreak of the psychosis in a certain proportion of cases.

From the toxic psychoses, particularly from alcoholic deterioration, the differentiation is often quite difficult. It must be borne in mind in this connection that the relatively normal man deteriorates very slowly from the use of alcohol, while one finds in the records of precox cases who have indulged in alcohol that the deterioration has come very much earlier. In addition to this it will be found that the amount of deterioration in the precox case is very much greater than could reasonably be explained by the alcoholic indulgence of the patient. When this discrepancy in the history is found one is justified in suspecting that one is dealing with a fundamentally more serious condition than mere alcoholism. These two types of cases show a characteristic type of defence which serves to separate them, the one from the other. In both instances the patients tend to minimize the true etiological factor and to exaggerate the unimportant one. For example, the precox case will always exaggerate the amount of alcohol he has been taking, while the alcoholic will always minimize it. This is of course only roughly true but it may be helpful in sizing up patients.

The whole question of alcoholic deterioration is by no means settled. Bleuler¹ is inclined to include the alcoholic hallucinosis in the dementia precox group and at least it must be acknowledged that many alcoholic patients after repeated attacks of acute alcoholic psychoses undergo a deterioration which is precox in character.

¹ *Dementia Præcox oder Gruppe der Schizophrenien* Franz Deuticke, Leipzig u. Wien, 1911. This work is by far the most comprehensive and complete treatment of the whole dementia precox problem extant. An excellent review of the work by August Hoch will be found in the *Review of Neurology and Psychiatry*, June, 1912.

The difficulty of differentiating between defect due to precox and congenital defect has already been mentioned in discussing precox in children, and precox on a defective basis. A material help in making this differentiation is an inquiry into the school knowledge. The school knowledge will be reasonably well retained in the precox, while it will not have been acquired in the defective.

It must be insisted upon that in the beginning, the hebephrenic and simplex types frequently complain of numerous minor ailments. These are frequently treated by the general practitioner or the specialist on the basis of their physical rather than their psychical character. Such patients repeatedly go through complicated systems of treatment until the true character of their illness has become evident, when often much valuable time has been lost.

Treatment.—Dementia precox has generally been considered to be a hopeless condition for which little or nothing could be done. This is at least not an attitude with which to approach a patient, and when we bear in mind the considerable number of recoveries that take place in the disease it is hardly an attitude that is warranted. The treatment, however, must of necessity be very difficult, because conditions that have to be met are multitudinous and range all the way from disturbances at the lower physiological levels through distinctly psychological problems to the relation of the individual to his social milieu. These matters may be taken up in their order.

Treatment of Physical Conditions.—Here the treatment must be practically entirely symptomatic. The bladder and rectum must be carefully watched where there is negativistic retention. The mouth must be kept clean, swabbed out with listerine or some other antiseptic mouth wash, if there is retention of saliva or food particles, otherwise putrefactive changes may occur, infections of the gum with ulcerations may result, with possible complications, such as pneumonia and death. Other such conditions as these have to be met in the practical and common sense way and need little special comment; for instance, surgical injuries, such as self-mutilation, bruises, and the like have to be met in the usual way, while it is especially important to get these patients out of doors and not permit their seclusive tendencies to further the development of tubercular disease.

At the present time a good deal of attention is being paid to the internal secretions. These may be investigated in the individual case, but as yet their beneficial action is unproved.

Treatment of the Mental Condition.—The treatment of the mental condition resolves itself into the treatment of disturbances at lower and higher levels. In the disturbances of the lower levels we have characteristically the excitements and the stupors. In general the treatment of the excitements should be by hydrotherapy, the continuous bath or the pack, according to the individual experience of the physician or the convenience of the hospital. Restraint either physical or chemical should be avoided if possible, resorting to drugs only in

so far as may be necessary to produce a sufficient amount of sleep. It must be borne in mind that all of the drugs that are used for chemical restraint, more especially those belonging to the belladonna group, are delirium-producing and therefore tend to interfere with the adjustment of the patient to reality. Small doses of atropin are not contra-indicated to obtain vegetative nervous system control of certain annoying somatic symptoms.

In conditions of stupor the general health has to be carefully looked after. The patient must often be tube fed, bowels and bladder require special attention, and the position of the body should be changed sufficiently so as to prevent pressure upon any portion of the skin surfaces or a tendency to hypostatic congestion of the lungs in weakened patients. Cleanliness and regular and sufficient feeding are the essential things, and it is very desirable in addition to wheel the patient's bed out upon the open porch where he can have abundance of fresh air, if this is possible.

Treatment at the higher psychological levels has the same things to be said for it as psychotherapy has for the psychoneuroses. While it cannot be expected that patients can be cured by psychoanalytic treatment as they are when suffering from the psychoneuroses, still it must be remembered that all of the symptoms of a precox case are not necessarily at the same level, and while the patient may not be susceptible of a cure, still many of the symptoms may be largely relieved, if not dispersed altogether. Psychoanalysis, therefore, should be used and even where the therapeutic effect may not be especially great, still it furnishes that sort of information about the patient which it is essential to have in order to deal with him intelligently. One must know something about the factors that produced the break-down, one must know something of the nature of the complexes, in order to go at the problem of regulating the life of the patient, not only intelligently but with any possibility of accomplishing anything. To approach the problem in any other way is to approach it blindly.

In the old cases of precox that have become considerably dilapidated, that are relatively quite inaccessible, it would seem that the best method of approach was through the agency of industrial training. If an effort be made in this direction intelligently after a sufficiently careful analysis, so far as possible, of the particular condition so that it will appear what is the best method of approach in order to arouse the interest and fix the attention of the patient, a great deal can be accomplished in making this class of patients generally useful about the hospital and in limiting their destructive and filthy tendencies and adapting them to a much healthier series of adjustments than if they are merely left to themselves.

The Treatment of the Social Relations.—The endeavor to modify the environment of the patient must be guided by what has been found as the result of psychoanalysis. The precox splitting goes back to the early infantile situations, the time when the love of the child

was given out in its entirety to the immediate members of the household, father and mother, brothers and sisters. Later on if the child is to become an efficient adult he must emancipate himself from the thralldom of this affection. This, however, is what the precox has not been able to do, and the affection which binds him to his infantile loves is quite truly felt as a destructive force that prevents his onward progress in the world. There frequently results, therefore, all sorts of antagonisms addressed to the members of the immediate household which are variously expressed and variously symbolized and distorted. The love of the little child for the parent or the brother or the sister, when it breaks through from the unconscious into the conscious life of the adult, is not understood at its true value, but becomes a hateful thing, and so characteristically there arise all sorts of incest phantasies. Recently one such patient in the hospital struck and stunned another patient and when called to task about it he instantly protected himself by the statement that "they" were accusing him of incestuous relations with his sister. These are the commonest of ideas among precox cases. Psychoanalysis will orient the physician with relation to these ideas and enable him to adjust the patient intelligently. Such ideas frequently require the removal of the patient to an institution.

Although a wholly pessimistic attitude is not warranted in approaching the problem of precox in an individual case, still it must be realized that after all one can hardly expect a complete recovery. One can only hope to readjust the situation so that the patient may get along comfortably and perhaps lead a useful life, probably upon a slightly lower level. The tendency of the disease is essentially to limit the individual in the mental sphere, and this limitation means of necessity that life has to be led at a correspondingly lower and simpler plane of adjustment. This is exactly what the institution provides for the patient, but there is no reason, in the absence of dangerous tendencies and if it is desirable from other standpoints, why an attempt should not be made in this direction outside of an institution. We know this can be done, for we see not infrequently patients getting along very well under the solicitous care of some relative, for example, and when this relative dies and the care is removed and their world is no longer carefully arranged for them, then they find their way into the hospital.

A careful dealing with all of the conditions surrounding the patient, more particularly dealing with them intelligently as the result of such psychoanalysis as can be made, will enable the physician, in a very considerable proportion of cases, if the means are at his disposal, which of course they frequently are not, to so adjust the situation as to bring about a state of relative calm and quiescence, with the preservation of a considerable degree of efficiency.

Prophylaxis.—The prophylaxis of dementia precox is a most difficult problem, and in the first instance of course should be met from the eugenic standpoint. Marriage should be very carefully supervised

where the individual comes from badly tainted stock. Such general principles may be borne in mind, as for example, the liability to mental disease in children from tainted stock is greatest among the earlier born and falls off rapidly, as Heron has shown, particularly after the fourth child, while Mott in working out his Law of Anticipation has shown that if the individual passes the twenty-fifth year the liability to a mental break-down is very materially lessened.

The possibilities of prophylaxis before the outbreak of the psychosis are not known, yet it would seem that it would be rational to endeavor to deal with those character anomalies that we know favor this type of disorder. The method of approach will of course resolve itself into an attempt to define the lines along which frank, open reactions do not seem possible to the individual, particularly along lines of definite sex conflicts. In this particular the whole matter of sexual education has to be gone over and its value as a prophylactic measure determined.¹

¹ See Jelliffe, *Predementia Praecox*, *Am. Jour. Med. Sci.*, August, 1907, for a study of these early features which antedate the appearance of the disease, also chapters on the treatment of *Paranoid States and Dementia Praecox*, by Meyer and Campbell, *Modern Treatment of Nervous and Mental Diseases*, White and Jelliffe, vol. i.

CHAPTER XXI.

INFECTION-EXHAUSTION PSYCHOSES.

THIS group of infection-exhaustion psychoses is somewhat of a heterogeneous group, including all of the mental disturbances dependent upon the various infections, as well as certain conditions which are tentatively supposed to be dependent upon exhaustion and which give similar clinical pictures. For the most part it contains the psychoses dependent upon all the febrile diseases, for up to the present time at least these different diseases cannot be differentiated solely from the mental side.

In connection with the similarity of the manifestations in this group, although the ultimate etiological factors are widely different, it is worth while to bear a few considerations in mind. In the first place it has been suggested that after the manner of thinking regarding the syphilitic manifestations and the alcoholic psychoses, as already outlined, that the mental picture is not dependent upon the immediate infecting agent or upon the toxin directly elaborated by the infecting microörganism, but on the contrary is due to a general disturbance in the metabolism, the result of the infection; in other words, that there is an intermediate agency at work, a metatoxin. On the other hand it has been suggested that the gamut of symptomatology which the neuron may produce in the process of its destruction is necessarily confined within certain relatively narrow limits and that therefore inimical agencies that act at the biochemical level can only produce relatively few groups of symptom-complexes. This is undoubtedly true. On the other hand, if the individual cases are carefully studied it will be found that the general course of the malady and the mechanisms involved are quite similar in the different cases; in other words, that the patients fall into one of very few groups; still the content of the delirious or delusional experiences and the minor variations in the manifestations in the malady must have another explanation. This explanation is naturally the make-up of the individual. A destructive agency at work in tearing down can only tear down what has before been built up. It is constrained, in other words, to deal with the material at hand, and therefore personal variations must be expected; for example, the delirious patient will weave expressions into his delirious productions that refer directly to his experiences.

Prefebrile, Febrile, and Postfebrile Psychoses.—The same thing may be said regarding fever and infection which has already been said regarding alcohol, namely, that it is a measure of the mental stability of the individual. While some persons may remain mentally

clear with a temperature of a 106° F., others may become delirious with hardly more than a degree of temperature. This difference is a well-known one, and in a disease like typhoid fever, for example, the general consensus of opinion is that the prognosis is most serious in those cases that become early delirious and show from the beginning marked and exaggerated nervous symptoms.

Infection and Initial Delirium.—Under this head are included the mental disturbances which develop early in the infectious diseases before there has been any rise in temperature. In fact the delirium in these cases may disappear when the fever is fully developed, although this is not the rule, the usual course being for the delirium of this period to go over into a fever delirium. This infection delirium is also found in conditions which are essentially afebrile, as for example, rabies, and is there due of course to an overwhelming of the body with toxins, or perhaps to a bacteriemia. This type of mental disturbance occurs characteristically with the onset of typhus, in the period previous to the eruption in smallpox, and has been observed in connection with influenza, acute chorea, especially the chorea of pregnancy, and in malaria.

Symptoms.—The symptoms of infection delirium are the usual symptoms of delirium, which may present any degree of severity, from mild confusion to *delirium acutum*, or collapse delirium, ending in death. The diagnosis of initial delirium previous to the appearance of the typical signs of the disease of which it is a symptom is of course practically impossible.

Fever Delirium.—Fever delirium is the psychosis which accompanies febrile movement and which in general varies in severity hand in hand with the variation in the severity of the fever.

It may be described in four stages according to the degree of its severity: In the first stage headache, irritability, sensitiveness to noises and light, restlessness, and disturbing dreams; in the second stage hallucinations appear, especially in the visual field; the hallucinations are of a dream-like character, and the patient may still be made to react clearly; in the third stage the motor disturbance is greater and takes on the character of jactitation; in the fourth stage there is profound dulling of consciousness, uncertain and ataxic movements, ending in coma and death. Of course this regular progress of the delirium may be interrupted at any point by an improvement in the symptoms and recovery.

The onset and the severity of the delirium, as already set forth, shows to an extent the mental stability of the patient. The delirium develops or becomes severe much more readily in the unstable and poorly organized than in the stable. The course of the delirium may be interrupted by an acute excitement followed by a stuporous condition, which Bonhoeffer¹ describes as an *epileptiform excitement*, which

¹ Die symptomatischen Psychosen.

is followed by a dream state, for which latter he retains the name of Ziehen, *infectious dream state*. With these conditions we may have confusing symptoms which makes the diagnosis difficult for a time. Thus, along with the disorientation there may be *flexibilitas cerea*, confabulation, perseveration.

In certain cases the orientation is less disturbed, while the hallucinations are more prominent, and there is an outward semblance of an hallucinosis.

Here, as elsewhere in psychiatry, the various forms of the psychosis are frequently designated by the prevailing symptoms. Thus, the symptoms found may be epileptiform excitement, dream states, stuporous conditions, hallucinosis, catatonic and confusional states, and the delirium may be designated by using any one of these descriptive terms.

Postfebrile Psychoses.—These conditions either develop as a result of the passing over of the delirium of the febrile state into the period of convalescence, or they may take their origin from the first, during the postfebrile period. In the latter case they would be considered as belonging more properly to the exhaustion psychoses, and yet it must be understood that the term exhaustion is a very vague one and that in all probability it implies at least the accumulation of toxins.

The characteristic picture of this psychosis is associated with the great physical debility which follows the subsidence of the fever in an infectious disease. The patient is weak, tremulous, exhausted, and complains of being tired. The mental state is fundamentally one of weakness. There is no confusion, but the capacity for attention is very much reduced, and in that way there come about apparent memory disturbances. The patient is unobservant of his surroundings, usually somewhat depressed, and may be very much occupied with his own bodily feelings to the extent of having hypochondriacal ideas, and fleeting hallucinations are not infrequent. In more severe cases there may be more evidence of mild delirium or confusion, the mood may be more definitely anxious and fearful and there may be delusions characteristically of the persecutory type. The patient is apt to be irritable, cross, and complaining. Occasionally there may be considerable motor excitement of an epileptiform character, and sometimes actual delirious dream states. Occasionally the picture of a mild expansiveness is found. A certain number of cases present a well-marked Korsakow syndrome.

Exhaustion Psychoses.—The term exhaustion in this connection is not intended to be used in other than a tentative sense. The whole question of fatigue, except with reference to very specific problems, such for example as muscle fatigue, is still little understood. In general, however, it may be said that there are two factors in the problem, the negative and the positive. The former is the result of the actual wearing out of a substance in the body, as for example muscle, while the second is the result of the formation of certain poisonous substances

which result from the breaking down of tissue. In the conditions which ordinarily are observed it would seem evident that both of these factors, the positive and the negative, are in evidence, and exhaustion is considered where they appear to be the predominating factors. For example, exhaustion is spoken of when coming on acutely as the result of a sudden loss of a considerable quantity of blood, or coming on more slowly as a result of the debilitating effects of a chronic disease such as carcinoma. In this latter case, however, it will be easily seen that in all probability the toxic element must enter. Similarly in convalescence from acute illnesses where the fever has been very high and the illness has been prolonged we see conditions accompanied by great physical prostration in which we feel that it is fair to assume that the element of exhaustion very largely is responsible for the mental picture. Under the head of exhaustion psychoses two main types will be described—collapse delirium and acute hallucinatory confusion (amentia)—but it must be understood that these two psychoses are not necessarily peculiar but only conditions in which the exhaustion element appears to predominate. Either or both of them may be found during the period of acute infection in the febrile diseases, and conditions that begin with infection and fever and give the picture of an infection or of a fever delirium, may go over into the severer type as the patient's general condition becomes worse and the organism is overwhelmed with the poisoning.

Collapse Delirium.—This is the *delirium grave*, or the *acute delirious mania* of the older authors.

The disease may present a prodromal period of restlessness, irritability, and insomnia, after which a condition of mild confusion may develop with only a slight degree of perplexity and perhaps fleeting hallucinations, slight clouding of consciousness, disorientation and dreamy delusions; psychomotor excitement is common at this time, the patient being active and perhaps inclined to actual violence and destructiveness. Often associated in the early symptoms are accesses of anxiety, amounting at times to actual terror. This condition gradually becomes worse, and finally the degree of excitement becomes very great, exceeding anything that we usually see in the other psychoses. When this extreme form of excitement is in evidence the outcome is usually apt to be serious, and the older writers believed it to be uniformly fatal.

In these severe cases the incoherence becomes absolute, the disorientation complete, the clouding of consciousness profound, the temperature generally runs high, perhaps as high as 106° F., gastro-intestinal symptoms are common, there is usually almost complete anorexia, coated tongue, offensive diarrhea, a high grade of indicanuria, and rapid emaciation which results in a high grade of exhaustion, with typhoid symptoms. There may be a certain amount of catalepsy, with stereotyped movements, grimacing, and echolalia, and stuporous conditions; coma, and death not infrequently result.

This condition occurs with all degrees of severity, and while the most severe cases die, the milder ones make good recoveries.

It may often be quite impossible to make a diagnosis of the physical condition of the patient while in their excitement. It must be remembered, especially if the patient has temperature, that these conditions are pretty apt to be dependent upon some acute physical illness, particularly an infection, and that not infrequently a deep-seated pneumonia, concealed from the usual approach by percussion, and auscultation, is at the basis of the difficulty, while an infection such as grip, rheumatism, or the like may also be etiological factors.

That the element of exhaustion is after all not the only element, and perhaps not the most important element, will be appreciated if one stops to consider that the great majority of patients do not react in this exaggerated way to acute toxemias or infections. In all probability the fundamental factor at the bottom of such a reaction is the individual make-up.

Acute Hallucinatory Confusion (Amentia).—This psychosis is less acute in its characteristics than the former. The symptoms are those of a mild confusion with incoherence and a considerable degree of perplexity. There are usually fleeting hallucinations in the various sensory areas, sometimes delusions, which, however, are not characteristically fixed, with a more or less changeable emotional attitude varying with the content of the delusions. The patient is characteristically in a condition of mild motor unrest.

The duration of the illness is relatively long. It may be prolonged over several weeks, is usually from one to three months in duration, and may be considerably longer. The course of the disease is not infrequently interrupted by lucid intervals, during which the patient is quite clear. These may last anywhere from a few minutes to a day or two, and then the patient will lapse back into his previous condition of confusion. This is an important point to bear in mind.

A very marked degree of perplexity is rather characteristic with these patients. There is considerable disturbance in their perception of their environment which they do not seem to understand, things about them appear to be changed, they appear to be in some strange place, things are not right, they do not understand the meanings of things, they get mixed up, get into the wrong bed, and act in similarly stupid ways.

Bonhoeffer¹ describes hallucinatory and psychomotor catatonic forms, in the latter of which flight of ideas and incoherence predominate.

In addition to the previously described psychoses, Bonhoeffer² describes, as occurring late in the course of the infectious diseases, and therefore it would seem dependent to a certain extent at least upon exhaustion, a *hyperesthetic emotional state of mental weakness*,

¹ Op. cit.

² Op. cit.

associated with physical symptoms, such as severe headache, parasethias, and pains in the joints, great prostration, oversensitiveness to noise and light, easily frightened, troublous dreams. He also describes an *amnesic variety* resembling Korsakow's psychosis. Acute delirium may also develop and one may see a meningitic form with very severe delirious reaction.

The possibilities of local injuries to the brain, such as brain abscess and local meningitis must be held in mind. When these develop in children they not infrequently leave sequelæ, such as mental defect and epilepsy.

Treatment.—The treatment of all of these conditions is, of course, in the main the treatment of the underlying disease. In general, however, we may say that for the excitement the continuous bath or the wet pack, with perhaps the occasional exhibition of a hypnotic should be chosen, rather than the constant drugging of the patient, so frequently employed.

Where there is marked lack of desire for food and the condition is serious and approaching one of profound exhaustion there should be no delay in resorting to artificial feeding. The rectum, of course, may be used if the stomach is very irritable, but should be a last resort. It is preferable to give small quantities by the stomach frequently, and endeavor in that way to deal with the situation, than to give rectal feeding, with the usual result of being satisfied with the giving of an amount of food which is really very inadequate.

In the extreme exhaustion of the later stages, especially where there is marked dehydration, hypodermoclysis often gives most excellent results.

Typhoid Fever.—An initial delirium in typhoid fever is generally conceded to be of most ominous import, while an early delirium which is continuous and severe is also of bad prognostic significance. With this disease the good effects of the bath treatment, as used by the Brand method, is especially well seen. Cold baths for the reduction of temperature have as one of their most important results their sedative and calming effect upon the nervous symptoms.

The possibility, in all such diseases as this, of the localization of the infection in the meninges should be thought of, and in cases of marked delirium, therefore the possibility of a meningitis should be borne in mind. Lumbar puncture may be valuable under these circumstances, both for diagnostic purposes and for the relief of pressure.

In the late stages, during convalescence, special efforts should be made to make the dietary as full and liberal as possible.

Erysipelas, the various *exanthems*, and the several types of *malaria* are also not infrequently complicated by mental symptoms, while *grippe* often produces profound depressions associated with great physical exhaustion hanging over during a long convalescence. We must remember in all of these conditions, particularly in the exanthems,

the possibility of meningeal focalization. Grippe sometimes also produces meningeal inflammation.

Acute articular rheumatism is especially important in this connection because of its relation to acute chorea and the so-called *chorea insaniens*.

Pneumonia is frequently associated with mental symptoms. Very many of the cases of delirium tremens owe their severity to a pneumonia, usually a masked form of this disease, either a central pneumonia or one located in the upper lobes, and therefore not so readily diagnosed.

In connection with all this class of diseases it must be borne in mind the many complications which may arise in their course and upon which the mental symptoms may depend other than the meningitides, as for example, the acute types of nephritis in connection with scarlet fever, the middle-ear complications of measles, and the endocardial complications of rheumatism. Not infrequently, too, these various complications may depend upon mixed infections.

CHAPTER XXII.

THE TOXIC PSYCHOSES.

Alcoholism.—It is generally conceded that alcohol is a powerful poison and as such if taken in large quantities or over a long period of time produces serious damage to the individual. There is, unfortunately, associated with this view the opposite view that alcohol is a valuable medicine in certain conditions, particularly that it is a valuable stimulant, while among certain peoples it is generally accepted as an essential article in the daily dietary.

As a matter of fact the toxic properties of alcohol far outweigh any possible beneficent effects that it may have. In fact it is questionable whether alcohol should be considered in any other sense than as a poison. It has no special medicinal properties that are of value, it is not a stimulant, and at most might be considered as a hypnotic, especially in old people with some arteriosclerosis. Other drugs can be used, however, quite as well and more safely. It is probable that the widespread belief in its efficacy for all sorts of conditions is based upon an effort at the justification for its use.

The part that alcohol plays in the production of mental disorders is extremely difficult to express in definite terms. Perhaps 12 to 15 per cent. of the psychoses are dependent upon alcohol as the principal etiological factor, and yet any such figure as this is extremely misleading, for we really do not know how alcohol brings about its results, and especially it is not known whether it is primarily the alcohol which is to blame or whether secondarily the metabolism disturbances which are produced by its continuous use. It is of the highest significance that of the cases that come to autopsy a very considerable number of them in the general hospitals and poor houses are found to suffer from cirrhosis of the liver, while in the hospitals for the insane this condition is of rare occurrence. The indication is very clear that the psychoses are dependent upon some peculiarity of make-up of the individual which is affected in an exaggerated way by alcohol or of which alcoholism is the expression. If this is true, then the alcoholism is only a surface indication, and the true etiological factors lie deeper.

Psychology.—There are many types of persons who drink and there are many reasons for drinking. The usual reasons given refer to social conditions. We are rather taught to believe that the social demands are of such a nature as to require a certain amount of drinking, and that this drinking once started tends to perpetuate itself and to finally become a fixed habit. This way of looking at the situation, like the belief in the beneficent effect of alcohol, is very largely at least an effort

at justification. The social conditions cannot be changed. We have to submit to them, and therefore the drinking is inevitable and not the fault of the individual. This really offers no adequate explanation, and in all probability there is very little truth in the statement. People do not drink simply and solely because they have acquired a habit of drinking. The habit element is the least important in the whole situation, and if that were the only thing to be dealt with the problem of alcoholism and of other habituations would be relatively simple. People drink because of definite returns which they get from drinking. A given number of persons all placed under the same conditions, social, etc., do not all drink. It is only some of them who drink, and those who drink do so not only because they get definite desirable results, but because those results are practically necessary for them. In other words, far more important than the question of habit formation, is the question of the individual psychology. Here again, as has been seen already in dealing with the psychoses, the question of mental conflicts is most important. When the individual is confronted by situations to which he cannot adjust adequately, when the world of reality makes demands which are too great for him to meet, one of the ways in which the individual reacts to such a condition is by narcotizing himself and so withdrawing from the whole situation. Alcohol then becomes an agent which helps the patient to get away from the conflicts thrust upon him by reality, it helps him to withdraw within himself, helps him to live in the world of phantasy where things come true as he wishes them. Under these circumstances it can be seen why what appears to be a habit is formed. The moment the individual, harassed by the absolutely unacceptable demands of the world, finds an avenue of escape in which he can rest from their harassings, finds the possibility of peace, of repose, he finds it equally impossible to resist the temptation to avail himself of it and of course he usually continues to avail himself of it. He is rendered more and more incapable of meeting the conflict efficiently. Therefore a vicious circle is established and the individual is hopelessly involved.

Aside from the class of individuals described above, it must be constantly borne in mind that indulgence in alcohol is oftentimes the expression of a psychosis. For example, the recurrent attacks of manic-depressive psychosis may be ushered in by alcoholic indulgence, and if one is not keenly observant he may easily suspect that he is dealing with an alcoholic psychosis rather than with a manic-depressive. The same thing of course may be said of dementia precox which is often found associated with alcohol, and with paresis, which not infrequently has a history of alcoholism in its early stages. Then it must be remembered that in certain post-traumatic conditions, that is, cases following especially head injury and sunstroke, and in arteriosclerotic and senile conditions the patient may react in an exaggerated way to alcohol and that very small doses may produce very pronounced effects. This is true also in connection with certain other conditions,

particularly imbecility and epilepsy. Here pronounced reactions to alcohol are found, and it is important to evaluate the importance of the alcohol in the entire situation.

Finally, there are certain psychoses which appear to be essentially alcoholic; to depend upon prolonged indulgence in alcohol. Of the acute conditions due to alcohol drunkenness is the most typical, while of the chronic conditions dependent upon alcohol, delirium tremens, alcoholic hallucinosis, and Korsakow's psychosis are the most important. These latter because they appear to be dependent upon something other than simple alcohol, as they never occur as the result of single large doses, but can only occur in a person chronically addicted it has been proposed to call, after the manner of the psychoses due to syphilis, the meta-alcoholic psychoses. With this introduction a short description of each of the mental pictures dependent directly or indirectly upon alcohol will be given.

Drunkenness.—The general phenomena of drunkenness are too well known to require detailed description, except that perhaps the less evident manifestations are not generally thought of. The progressive disturbance of coördination of the motor centers, the higher being thrown out of adjustment first, and the progressive disturbance of the sensory apparatus in the same way produce disturbances, the latter of which are entirely subjective and so are not generally observed. The mood of the drunken man is variable. Each man reacts to alcohol in a way peculiar to himself, but in a general way there may be considered to be two classes into which cases of drunkenness may be divided, the *exalted* and the *depressed*, the former closely resembling the manic phase of the manic-depressive in his extreme loquaciousness and hyperactivity, while in the latter the patient withdraws within himself, being sullen, morose, and disinclined to associate with others.

One of the important considerations under this head is the degree of ease with which the individual is seen to react to alcohol. Alcohol, like fever, may be used to express the measure of cerebral resistance; unstable and defective individuals reacting to both in an exaggerated way.

Pathological Drunkenness.—Drunkenness that exhibits unusual features which lead the individual to perform strange acts or acts of violence or which produce serious physical symptoms are known as conditions of pathological drunkenness. Persons in this condition may develop well-marked hallucinations or delusions to which they tend to react, while convulsive attacks are not infrequent, and the whole period is often obliterated from the memory by an amnesia.

Chronic Alcoholism.—The effects of alcoholic poison may be exhibited in any organ of the body, more particularly the central nervous system, the stomach, the liver, the kidneys, and the blood-vessels. The effects on the nervous system are shown in various disturbances of sensation, such as the paresthesias, amblyopia, amaurosis, dulness of hearing, of touch, etc., while in the motor realm

we find tremor and epileptiform attacks, with general motor enfeeblement characteristically. The mental changes are gradual and progressive, the intelligence is blunted, the judgment is impaired, the moral sense dulled, while actual delusions not infrequently develop.

While all of these changes may occur in chronic alcoholism, it is usual to see in individual cases one organ more especially selected out by the alcohol for its destructive effects. With the beer drinkers who absorb several liters of beer each day, cardiac hypertrophy is quite common. Some patients develop serious kidney complications, while others are able to drink over long periods of years without any material impairment of the kidneys. The same remark applies to the liver, while the effects upon the nervous system and the bloodvessels are equally varied. It is characteristic, too, that the individual as a whole varies in the destructive effect which alcohol has upon him, some individuals apparently being able to use large quantities over considerable periods of time without noticeable impairment. The general outward appearance of health, however, which many indulgers in alcohol show is sometimes rudely dissipated when they are attacked by an acute illness, for however well they may have been able to go on with the routine day's work, their weakness is shown when they are subjected to the special stress of, for example, a pneumonia, and the death rate in this class of persons is much higher than in abstainers.

Delirium Tremens.—Delirium tremens, while an acute manifestation of alcoholism, can only occur in a person suffering from the effects of chronic alcoholic poisoning. It may manifest itself sometimes as the result of a prolonged debauch, sometimes as the result of an illness or injury in a chronic alcoholic. It has often been maintained, and is still believed by many, that the delirium is not infrequently the result of a sudden withdrawal of alcohol, as for example when a patient is taken to the hospital for an injury nothing is thought of his alcoholic tendencies until he develops a delirium. In these cases the delirium is presumed to be due to the fact that the patient did not get his usual supply of alcohol. There is no good reason for this opinion, and it must be remembered that in the prodromal period of delirium tremens not infrequently the patient has experienced a disgust of liquor for a number of days. A pretty effective negation of the theory of an abstinence delirium is derived from the English prison statistics, which show that in the year 1907 there were 63,000 inebriates who were suddenly deprived of alcohol by confinement, and in this number only 246 developed delirium tremens, less than one-half of one per cent.

Symptoms.—The symptoms of delirium tremens may come on slowly, being preceded by several days of general physical upset, with transient sensory falsifications and perhaps delusional interpretation, which show a special tendency to come on at night or under conditions in which accurate perception is interfered with. On the other hand, the delirium may come on very rapidly. A case is recalled of a long-shoreman, a man of perhaps thirty-five, a giant in physique, who walked

into the hospital at noon with a sprained ankle; the ankle was strapped, and he was put to bed. That night he was in the wildest delirium, and the next morning he was dead.

Following these prodromal symptoms the delirium appears in its complete manifestations. It is a typical toxic delirium with multiform disorders of the sensorium. The hallucinations predominate in the visual sphere, although tactile hallucinations are very common. The patient is manifestly very sick, he is greatly depressed physically, he is tremulous, the tremor being so constant as to have given the name to the condition, and his mood is characteristically one of apprehension, anxiety, and fear. He is disoriented, mistakes the people about him; not infrequently his delirium is an occupation delirium and he believes himself back at his accustomed work, but it is characteristically filled with fearsome sights, and he is in constant terror from the invasion of the numerous animals that he may see about him.

In the somatic realm the pulse is rapid, the tremor constant, the skin bathed in perspiration, the tongue foul, the appetite nil, and there may perhaps be a marked grade of albuminuria. The temperature is characteristically either normal or below normal, but it may be high, in which case we have what is known as the febrile type of the disease, which is generally fatal. All sorts of complications may naturally occur. Acute cardiac dilatation sometimes causes death, while one of the most frequent of the complications in severe cases is "wet brain." In this condition the patient sinks into a low muttering delirium, the temperature falls to subnormal, the face is pale and bathed in cold perspiration, the pupils dilated, there may be some rigidity of the neck with a tendency to a bending back of the head; the patient sinks into a comatose condition and dies.

The psychosis runs an acute course of about three days and usually terminates by a long sleep in recovery. Ten to 15 per cent. die.

Treatment.—The treatment of delirium tremens should be supporting, liquid concentrated food, predigested if necessary. The bowels and the kidneys should be freely flushed, heart stimulants are necessary to prevent cardiac failure, and hypnotics often required to produce rest. For the excitement, hydrotherapy in the form of the continuous bath, or if this is not available, cold packs are preferable to drugs. The constant thing to be kept in mind is the support of the strength of the patient, and the logical way to accomplish this end is by feeding. If the patient refuses food no time should be wasted. He should be immediately fed with the tube, preferably as often as three times a day in small amounts, watching the stools and governing the quantities given in each feeding by the amount the patient is able to digest. If the patient is unable to retain the food owing to acute irritability of the stomach and constant vomiting, feeding by the rectum should be resorted to, while if the prostration is extreme and there is emaciation and the deprivation of fluid considerable, hypodermoclysis is a valuable adjunct.

Korsakow's Psychosis.—This psychosis is found typically in association with alcoholic polyneuritis, although the same mental state may be found with a polyneuritis of different origin, as for example the metallic poisons, some of the infections such as tuberculosis and influenza, and some of the endogenous toxins as in diabetes. The syndrome occurs also in connection with general organic changes of the central nervous system as in paresis, arteriosclerosis, and senility. In the latter case it is a part of the clinical picture of presbyophrenia.

While this psychosis occurs typically in connection with polyneuritis, the evidences of a polyneuritis may be very slight. They should be carefully examined for, especially by pressure over the large nerve trunks, which will often elicit painful points, rather typically at the points of exit through the bony foramina. The Lasègue sign should be sought for.

Korsakow's psychosis is sometimes designated in contradistinction to delirium tremens, which is spoken of as an acute alcoholic delirium, as a chronic alcoholic delirium. In fact the attack may begin with a typical delirium tremens which merges into the chronic delirium of Korsakow's psychosis instead of clearing up, although this is not the usual method of onset. Not infrequently, however, a history of previous attacks of delirium tremens may be elicited.

The mental symptoms are the result of a combination of attention and memory disorder. The memory disorder is of the nature of a lack of impressibility. The result of this combination is a special type of amnesia. There is defect in the recording of current events. The patient is usually disoriented, to some extent at least, and the things that have recently happened cannot be recalled. These defects of memory are characteristically supplied by fabrications of all sorts of degrees of probability and grotesqueness, which are usually related by the patient with a composed bearing and with every outward appearance of relating facts, or at least occurrences which he himself believes. These fabrications do not correspond if the patient is asked at intervals about occurrences covering a certain period, and they may often be guided by suggestions from the questioner. Not infrequently the patient in his fabrications invents occurrences which account for his symptoms, for example, a patient who is suffering from a certain amount of pain in his legs as a result of his polyneuritis, even though he may have been confined to his bed for weeks, will say that he was out that morning and climbed a long hill and tired his legs out, and in that way he accounts for their aching.

The experiences which these patients relate are of a delirious character and not infrequently it is rather difficult to distinguish the fabrications from the dreams, and it would appear that the two often merge into one another.

Physically the patient usually presents the signs of a polyneuritis, although these may be very mild. When the neuritis is severe, foot-drop and wrist-drop are characteristic signs, as the nerves of the exten-

sors of the forearm and leg are most characteristically involved in alcoholic neuritis.

The serious complications of the disease are either dependent upon intercurrent affections such as pneumonia or upon the involvement of important nerves such as the vagus, vagus involvement of course being pretty apt to be fatal. A serious complication is a neuritis of the phrenic nerve with paralysis of the diaphragm on one or both sides with resulting tympanitis and serious embarrassment of cardiac and respiratory activity.

Pathology.—The pathology of the disease shows that it is by no means confined to the peripheral nerves nor even to the motor areas of the central nervous system, but that there is a very general involvement of the entire central nervous system, the cord, basal ganglia, and the cortex all showing lesions, although there appears to be a tendency to the focalization of the pathological process. As a result we get certain focal symptoms as aphasia, apraxia, hemianopia, etc. This wide distribution of the pathological disturbances and their tendency to focalization is due to the fact that the disease involves the bloodvessels. The smaller vessels proliferate and present evidences of endo-, meso- and periarteritis with frequent ruptures. When this condition is focalized in the mid-brain and gives rise to various ocular palsies we have the syndrome of *acute hemorrhagic polioencephalitis of Wernicke*. This is frequently observed in chronic alcoholics. The condition is typically ushered in by severe and prolonged vomiting, which is followed by marked confusion with delirium, vertigo, sometimes headache, and somnolence. Optic neuritis may be present. Death usually occurs in from one to two weeks.

With a disease of this character having a pathology so widely distributed throughout the nervous system and dependent upon chronic toxemia we can understand how very many *types* have been described. These types or clinical forms are nothing more than descriptions of the disease in which certain symptoms are especially prominent, and so we have amnesic, confusional, delusional, anxious, demented, delirious, stuporous, hallucinatory forms described by different authors.

Treatment.—The treatment of the condition is of course the withdrawal of alcohol and the treatment of a general toxic state along supportive lines. The deformities which are due to the paralysis should be dealt with by recognized orthopedic measures. It should be remembered that while the patient is in bed and delirious the weakened extensors of the forearm and leg should not be stretched by the weight of the limb, but should be supported, particularly the weight of the bed clothes should be removed from the feet.

Alcoholic Hallucinosiis.—This condition is also an expression of chronic alcoholism and may be preceded by attacks of delirium tremens. It is characterized by hallucinations, auditory predominating, in this respect strongly contrasted to delirium tremens, and delusions of a

persecutory character which harmonize and are explanatory of the hallucinations. It is quite characteristic that the hallucinations and the delusions deal with sexual matters, the patient frequently being abused by "the voices" for committing some sexual crime or is accused of sexual perversions. The delusions are practically invariably persecutory, although occasionally expansive elements may enter the picture. Not infrequently, too, the patient is very much frightened, as when he thinks he is being closely pressed by his persecutors who are determined upon taking his life. There are not a few border-line mixtures of this psychosis and delirium tremens.

The condition is essentially an acute paranoid state and as such its explanation is the same as the explanation of paranoia. In other words, there is in these cases a fixation at the homosexual level, and this accounts for the very great frequency of the sexual character of the hallucinations and the references to sexual perversions. The alcohol which is taken by the individual because he cannot deal efficiently with reality causes him to turn back upon himself and produces a reanimation of this early fixation, and then the mechanism of projection comes in as a distortion to make it appear that the suffering has its origin from without.

The delusional system is rapidly systematized so that in certain cases the patient might give the impression of a paranoiac. The course of the disease is usually prolonged over a number of weeks, and not infrequently a number of months. It generally ends in recovery, but sometimes merges into chronicity. The disease is not fatal and its pathology is therefore the pathology of chronic alcoholism.

Alcoholic Pseudoparesis.—In a few patients prolonged alcoholic intoxication produces a picture closely resembling general paresis. In these cases we find an expansive delirium combined with the signs of alcoholism, such as ataxia, speech defects, and tremor. It must be remembered too that pupillary anomalies quite regularly occur in severe grades of intoxication and even an Argyll-Robertson pupil has been observed. These observations should be taken with a certain amount of reservation because it is quite impossible often to tell, without the aid of instruments, exactly what the pupillary reaction may be and especially whether it is entirely lost, and it is generally conceded that toxic conditions will produce a slowing of the light reflex. On the other hand, it has been thought by some that where these marked pupillary disturbances are found that it indicated the presence of syphilis. Therefore these observations should be correlated with the serological findings. These cases clear up promptly upon the removal of alcohol.

Alcoholic Pseudoparanoia.—In some cases of chronic alcoholism a fairly circumscribed delusional system may develop which characteristically takes the form of delusions of marital infidelity. In endeavoring to interpret this delusion the paranoia mechanisms must be borne in

mind. Quite commonly, however, the delusion when it occurs in man is a defense reaction to impotence, which has been largely produced, probably, by the alcoholic indulgence. Instead of realizing his own impotence, which is an unacceptable thought, he blames his wife for being untrue to him. These cases are essentially chronic and persist at least as long as the alcohol is indulged in, while even when it is removed they may be a long time clearing up and may perhaps graduate into a chronic delusional state.

Alcoholic Epilepsy.—In a chronic alcoholic toxemia it is not strange that epileptiform convulsions should occasionally develop and recur from time to time. The outward characteristics of the convulsion are in every way the characteristics of an epileptic attack. If the person is not essentially epileptogenic the convulsions will subside on the removal of the alcohol.

Dream States.—In conditions of pathological drunkenness it is not infrequent for the patient to have no recollection of the period during which he was intoxicated. If, during this period, he has been engaged in some sort of occupation, business transactions, travel, or what not, or perhaps engaged in the performance of criminal acts, he may still wake up with no recollection at all of this period, although during it he outwardly appeared to be in a normal state. Some people are especially liable to these dream states. It would seem that they present a tendency to the doubling of their personality and that the alcohol helps to produce these somnambulic episodes.

Dipsomania.—Dipsomania is a term applied to a periodical impulse to drink. Drinking is only the outward manifestation of the trouble. It is a recurrent neuropathic attack which demands the narcotizing results of alcoholic indulgence.

Opium.—The habitual use of opium in some form has become common and among all classes in society. The same thing may be said with reference to the reasons for taking opium as has been said with reference to alcohol. The opium habitu  is a person primarily of neuropathic taint, the mere opium taking or the symptoms it produces being but surface indications of the real trouble.

Symptoms.—The general symptoms of its use are well known. In small doses it is mildly stimulating and produces a pleasant euphoria, while if it is taken in large doses and continuously it leads to a mental blunting, a general feeling of malaise, inability to make effort, with marked physical disturbances such as tremor, muscular weakness, constipation, paresthesias, etc. It is not infrequent for opium in some of its forms to be combined with addiction to alcohol or cocaine. Continuous use may simply produce a thoroughly crippled individual who is alternately in a mildly euphoric state or in a condition of stupidity, malaise, and indolence. When a psychosis develops, it is characteristically an hallucinated state usually with paranoid coloring, or it may be distinctly delirious. The prognosis is of course not good unless the underlying neuropathic taint can be dealt with.

The use of opium has much less tendency than that of alcohol to produce tissue degeneration.

Treatment.—The treatment is usually the prompt removal of the large doses. These can be readily withdrawn until the patient is reduced to the actual necessary amount of morphin to get through the day comfortably with, which is about two grains. From this point on withdrawal should be gradual, carefully looking after the symptoms of abstinence as they appear. These symptoms refer particularly to the heart, the nervous system, and the gastro-intestinal system. Sometimes profound collapse may occur with the withdrawal of the drug. Cardiac conditions should be watched and stimulants administered if there is any sign of weakness. For the diarrhea opium should be avoided if possible. Acute withdrawal and treatment by atropin for the vagotonic collapse, may be practised to advantage.

Cocain.—Cocain is taken for the same reasons that alcohol, morphin and other drugs are taken, but it is much more dominating in its influence and more difficult to escape from than either of the others, while it is more disintegrating than morphin or opium.

Symptoms.—The symptoms of cocain intoxication are those of marked stimulation. The patient is extremely active and very talkative, full of all sorts of schemes, ambitious, tells what he is going to do, unfolds plans, and in general is in a manic condition of mind. Following this hyperstimulation there is of course profound malaise and exhaustion. Associated with the habitual use of cocain there is more apt to be marked and persistent sensory disturbances in the form of paresthesias, while in the mental sphere the disintegration of the individual is much more marked than with opium, as a rule. The moral sense is blunted, he lies readily, the judgment is impaired, and not infrequently they develop distinct delusions of which the delusion of marital infidelity is not infrequent. Chronic paranoid conditions, hallucinatory states, and deliria are seen in cases of long-continued habituation. The drug may be withdrawn much more rapidly than morphin.

Miscellaneous Intoxicants.—A large number of drugs are taken habitually, particularly the whole group of pain-relieving and sleep-producing drugs. In general the mental condition due to these drugs varies all the way from delirium as the result of an acute intoxication to chronic paranoid conditions. The main feature of the *drug deliria* is their dream-like character. The patients have all sorts of grotesque experiences which they weave into a more or less consistent description, elaborating the details here and there as may be necessary. The delirious experiences pass like moving pictures before the patient's mind, and they not infrequently are perfectly composed as they recount them, no matter how grotesque or unusual or even terrifying some of them may be.

Bromides.—The possibility of bromide delirium from the taking of large doses of bromides for a long period of time should be borne

in mind. Patients are not infrequently admitted to hospitals supposedly suffering from some psychosis but with a bromide delirium, the bromide having been administered to quiet the nervousness of a neurosis, or perhaps given in the treatment of an epilepsy. The average practitioner does not appear to realize the possibility of chronic poisoning that prescriptions of this sort present.

Carbon Monoxide.—Carbon monoxide delirium is of special importance in connection with the frequent attempts at suicide with illuminating gas that one meets with in our large cities. The principal thing to be thought of in connection with carbon monoxide poisoning is that after the initial effects of the poisoning have subsided and the patient comes out of the coma there may be quite a rapid return to normal and the patient be in an apparently normal condition for a week or ten days and then a relapse occur with marked mental disturbance. The patient should never be discharged from the hospital until after this period of danger has been passed. Delirium is characteristic of this form of poisoning, and an amnesia for the period usually follows, often associated with fabrications and pseudoreminiscences, producing a Korsakow syndrome. Disturbances of speech are also common, as are disturbances in the emotional field, such as, for example, causeless laughter. Pathologically there appear to be thrombotic occlusions and hemorrhages which characteristically are found in the basal ganglia, and in this region may account for the disturbances of mimic.

Lead.—In chronic lead poisoning we may find a condition of pseudo-general paresis or Korsakow's syndrome in addition to the ordinary hallucinatory and delirious episodes.

Mercury.—This poison produces characteristically a condition of great irritability associated with insomnia and anxiety. This condition may become more pronounced and develop into a well-marked delirium.

There are, of course, other poisons, but these are the principal ones.

General Considerations.—With regard to all of the habit-forming drugs, particularly alcohol, opium, cocaine, and the analgesics and hypnotics, it may be said, as already indicated in discussing them separately, that the true cause of addiction lies in the make-up of the individual. This cause would appear to be an auto-erotic or narcissistic fixation. This fixation and its results are by no means simple matters psychologically and differ very materially in different individuals. The only hope for the treatment of this group of cases, however, is an ability to modify this character anomaly. The treatment, of course, therefore, can only be tentative and symptomatic during the periods of acute disturbances, and it is in the interval, when the patient is free from the psychosis, that the treatment should be applied. This is, of course, the psychoanalytic treatment and should be addressed to discovering the underlying etiological factors. Unless something can be accomplished in this direction very little is to be hoped for.

As regards the metallic poisons associated with dangerous trades, of course, the treatment here is purely symptomatic, eliminative, and expectant, while the real effort that is to be of material help should be expended in prophylaxis.

Uremia.—In acute uremia the convulsion is one of the most typical signs, and cannot be distinguished in its outward manifestations from the ordinary epileptic attack. Similarly with epilepsy, too, there may be quite well-defined deliriod experiences or dream states in which there is disorientation and noticeable disturbance of the sensorium, usually associated with more or less constant activity. There may be an anxious affect, or the patient may, as is quite usual in delirium, merely be interested in hallucinatory experiences. Acute uremic symptoms, in the mental sphere, of this character may accompany exacerbations of the physical condition in a patient suffering from chronic nephritis.

In chronic uremia one finds not infrequently marked mental symptoms. The mental symptoms are usually of the character of defect, a general stupidity with a feeling of physical weakness, desire to sleep, with perhaps complaints of headache, associated with irritability. Along with the mental symptoms may go tremor, speech disturbances, eye muscle palsies, pupillary differences, sluggish or failing light reaction, which point to an organic disease of the brain. If along with this condition there are Jacksonian attacks followed by mono- or hemiplegia with perhaps aphasia and disturbances of vision, the similarity to general paresis on the one hand and to brain tumor on the other may be very great. Sometimes along with these symptoms the mood is distinctly euphoric, which still further suggests the possibility of paresis. A certain number of the cases of uremic psychoses show close relationships to dementia precox showing negativism, incoherence, catatonia. Probably this group is more serious in its prognosis.

Diabetes Mellitus.—The mental disorder which is associated with diabetes is usually of a mild depressive type, often with ideas of ruin and sin. Anxiety states have also been described. Occasionally the picture gives somewhat of the impression of general paresis. With the chronic depressed condition paranoid ideas sometimes develop.

In general the severity of the mental symptoms keeps pace with the severity of the physical disease, as indicated by the sugar eliminated. When the intoxication is very great the patient may be irritable, sleepy, stupid, complain of weakness, and headache.

The diabetic state itself is an etiological factor in the production of arteriosclerosis, and evidences of arteriosclerosis may be found associated with the condition. It should be remembered, however, that both the arteriosclerotic condition and the senile state interfere with the metabolism of the sugars and, therefore, may themselves be associated with glycosuria.

Gastro-intestinal Diseases.—In general it is well known that with diseases below the diaphragm there is associated on the mental side a

depressive mood. The relation between diseases of the gastro-intestinal tract and associated glands to mental states is extremely difficult to interpret. Many of the gastro-intestinal disturbances, rather than being causes of mental disease, are effects. This is particularly seen in the realm of the neuroses and the psychoneuroses, and is discussed in the chapter dealing with these conditions.

Certain cases of acute confusion develop associated with profuse and offensive diarrhea, a high grade of indicanuria, vomiting, low fever, and perhaps mild albuminuria. Some of these cases go on to acute delirium, with typhoid state, profound exhaustion, coma, and death. Just exactly where these cases belong and what they mean is not altogether known.

CHAPTER XXIII.

PSYCHOSES ASSOCIATED WITH ORGANIC DISEASES.

Apoplexy.—Immediately following the apoplectic insult the patient is quite commonly unconscious. As the symptoms subside and the patient begins to “come to” there may be marked disorientation to the extent of a mild delirium, especially if fever is present. This symptom of a slight rise in temperature with onset of mild delirium sometimes occurs a few days after the original insult and is then of bad prognostic omen. In severe attacks the unconsciousness may pass on into profound coma and death.

Ordinarily following an apoplectic insult after recovery from the acute symptoms a condition of more or less impairment is left, and when it is borne in mind that the patients who suffer from apoplexy are in the main in the senile or arteriosclerotic period it will be understood that the symptoms of the defect take on characteristically symptoms of these two conditions, which perhaps become aggravated materially following the insult. The defect, however, may appear to be very much greater than it really is, because of the inability of the patient to express himself owing to incident aphasia or apraxia. The emotional attitude of the patients is usually one of irritability, although indifference also enters into the picture. Such patients often lie quietly and apparently indifferent until an attempt is made to communicate with them. Under these circumstances if they have serious aphasic or apraxic disturbance and know, for example, what they wish to say, they may become very much excited and quickly fatigued and emotional as a result of their repeated efforts and failures to enter into communication. This is also quite characteristic in the younger patients with apraxic disturbances and relatively clear intelligence. It is, so to speak, “maddening” not to be able to control any longer the power of expression. Some of these patients, especially those who suffer from sensory forms of aphasia, being out of communication with their fellows, and not being able to understand what goes on about them, may acquire a suspicious attitude which gives a paranoid coloring to their mental condition. It can be understood that the apoplexy which produces disorders of expression and interferes with the patient’s keen contact with his fellows will not only precipitate the deterioration of the senium, but will make that deterioration much more rapid.

The neurological signs of hemiplegia are present, also often the signs of senility, and not infrequently of arteriosclerosis, while a certain few of these patients develop epileptic attacks. (See Chapter XII.)

Traumatism.—The effects of trauma may be divided into the immediate or acute effects and the more remote or chronic results, into which the former may or may not merge. The ultimate results are divisible into changes in constitution and defect conditions, while a certain proportion of cases develop psychoses following an injury without there being any special connection between the two or in which the injury can only be conceived as a precipitating or contributory factor. Oftentimes the injury is the result rather than the cause, as is so frequently seen in paresis, the injury serving to call attention to the presence of a disease up to that time not recognized.

The usual immediate result of a head injury, either direct or indirect, is a certain degree of unconsciousness which may vary all the way from a light stupor to profound coma. When the unconsciousness is not profound the patients are quiet, but if disturbed become very irritable and resistive. Quite typically, associated with the stupor, is a mild delirium of hallucinatory character which is especially pronounced and apt to be more violent in patients who are the subject of alcoholism. Associated with this may be a slight rise of temperature, but if the rise is pronounced meningitis should be thought of. Alternating conditions of apparent clearness and marked confusion may occur. Usually the stupor is of only short duration, but may last several days and finally disappear. In severe cases the patient becomes comatose and dies in that condition. In a few cases the delirious reaction is unusually prolonged and may be associated with Korsakow's syndrome.

After recovery from the immediate effects of the injury certain constitutional changes may slowly develop, the most typical of which is Friedmann's complex, which he attributes to a vasomotor disturbance in the brain. The symptoms of this complex are headache, dizziness, irritability, insomnia, physical and mental fatigability, a certain change of character, and intolerance to alcohol, with which may be associated a memory defect of the type of retrograde amnesia, which, however, is not a part of the complex, although there may be a certain memory defect which is quite common to all highly nervous worried people. The headache is apt to be complained of as a feeling of pain or a peculiar feeling of constriction of the head and is associated with feelings of fulness, especially on stooping over. The dizziness is also characteristically intensified by stooping or by sudden movements. The irritability of temper may be so marked as to lead to explosive outbreaks, which simulate psychic epilepsy. Kaplan has named this condition the *explosive diathesis*. They are much aggravated by alcohol. Sleep is liable to be disturbed, physical and mental endurance is very much lessened, and the patient may become depressed, moody, and in various other ways show a subtle change in character which is appreciated by all of his close associates. The intolerance to alcohol is very characteristic of this post-traumatic constitution. Sometimes associated with the explosive diathesis, but also occurring alone, are

temporary dream states. Various other hysteriform and epileptiform manifestations may also occur.

Finally, certain defects are left as a result of the traumatism, depending, of course, upon the nature, the extent, and the location of the injury. As regards the more characteristic results of head injury of a distinctly psychotic nature, it should be borne in mind that the change in character, transformation of the personality, as it might be called, is often a very subtle process and one extending over a very considerable period of time. It might be practically impossible to evaluate the situation at all if one were dependent upon a cross-section of the patient's mental life. When a longitudinal section, however, is available one finds in typical cases an individual who up to a certain point in life has gotten along well, showing efficient reactions and developing by steady progress in some chosen line of work. Such an individual receives a head injury and from the time of this injury on there will be noted in the history a gradual falling off in efficiency. It may be quite impossible to put one's finger upon any specific thing in the situation and set it down as a pathological type of reaction, but the individual reaction has changed in character, and from efficiency there is evolved inefficiency. Naturally, it is a long time before the true explanation of such a change is reached. In fact it is a long time before it is realized that any change at all has taken place, and it is because of this fact that the older psychiatrists used to speak of traumatic insanity as eventuating many years sometimes after the injury. Not infrequently this falling off in efficiency has going with it a gradual deterioration in morale, and with the heightened susceptibility of the patient to alcohol, which trauma produces, it is natural that alcohol should enter into the picture very largely and often be regarded as the cause of the whole trouble.

Meyer's¹ classification of the effects of traumatism in the nervous system is as follows:

1. The direct focal and the more diffuse destruction of the nerve tissue or of parts of it; and the reaction of the tissues.

(a) The immediate effects—edema.

(b) The scar formation.

2. The distinctly diffuse commotions in which the general reaction and the psychic elements preponderate, including the remote reactive results of exaggerations of vasomotor and emotional responsiveness.

He classifies the psychoses developing as follows:

1. *The direct post-traumatic deliria with the following subdivisions:*

(a) Preëminently febrile reactions.

(b) The delirium nervosum of Dupuytren, not differing from deliria after operations, injuries, etc.

(c) The delirium of slow solution of coma with or without alcoholic basis.

¹ The Anatomical Facts and Clinical Varieties of Traumatic Insanity, Amer. Jour. Insanity, January, 1904.

(d) Forms of protracted deliria usually with numerous fabrications, etc. (with or without alcoholic or senile basis).

2. *The post-traumatic constitution.*

(a) Types with mere facilitation of reaction to alcohol, grippe, etc.

(b) Types with vasomotor neurosis.

(c) Types with explosive diathesis.

(d) Types with hysteroid or epileptoid episodes with or without convulsions (such as most reflex psychoses).

(e) Types of paranoiac development.

3. *The traumatic defect conditions.*

(a) Primary defects allied to aphasia.

(b) Secondary deterioration in connection with epilepsy.

(c) Terminal deterioration due to progressive alterations of the primarily injured parts, with or without arteriosclerosis.

4. *Psychoses in which trauma is merely a contributory factor.*

(a) General paralysis with or without traumatic stigmata.

(b) Manic-depressive and other transitory psychoses, catatonic deterioration and paranoiac conditions, with or without traumatic stigmata.

5. *Traumatic psychoses from injury not directly affecting the head.*

This group of traumatic psychoses includes also a certain number of the *postoperative* psychoses, psychoses following operations upon the eye and residence in a dark room, the so-called *ophthalmic* psychosis and the psychoses of *insolation*. Of course this statement applies only to such psychoses as do not belong in other groups, as the shock of surgical operations, etc., may well be a precipitating factor in the onset of such psychoses. When, however, they are essentially psychoses the result of shock they have a symptomatology and history quite the same as the traumatic psychoses. They can be understood if the hypothesis of Friedmann is borne in mind that the complex named after him is due to vasomotor disturbance. If this be true it can be understood how severe emotional traumas, as well as physical traumas may produce a vasomotor imbalance, although, of course, it must never be forgotten that whatever may in the last analysis be the actual cause, the cause operates upon a certain kind of individual, and the symptom picture is necessarily modified accordingly.

Acute Chorea (Sydenham's).—Quite usually a patient with acute chorea is irritable, somewhat emotionally unstable, fretful, and impatient, a state of mind one would expect from the nature of the illness. While this condition is quite usual it may become somewhat more aggravated, the restlessness and emotional instability be more pronounced, with evidences of transitory disturbances in the sensorium, and perhaps slight apprehensiveness and suspiciousness. The hallucinatory disturbances may become very much aggravated, together with clouding of consciousness and marked delirium in connection with the febrile movement. Under these circumstances, of course, we are dealing essentially with a fever delirium. Along with these

milder manifestations of mental disorder one occasionally finds symptoms of a hysteriform character.

Chorea insaniens is generally considered as a distinct form of acute chorea. It is associated with high temperature and marked mental disturbances, usually beginning early in the disease. The mental disorder is essentially of a delirious character, with hallucinations and clouding of consciousness. The hallucinations are quite apt to be terrifying, and the patient consequently apprehensive and fearful, although the opposite condition of elation has been described. The disease is quite frequently associated with pregnancy, and is not infrequently fatal.

Korsakow's psychosis is sometimes seen in patients suffering from acute chorea as a result of polyneuritis resulting from overtreatment with arsenic. (See Section on Chorea.)

Chronic Chorea (Huntington's). — It has always been recognized that this disease was associated with mental symptoms, and it has generally been considered that there was a tendency to progressive deterioration ending in well-marked dementia. While in a general way this may be true, it is well to be cautious in estimating the mental condition of chronic choreics. They are extremely inaccessible in many instances, especially those patients whose speech apparatus is affected by the disorder, and it requires great patience to make a satisfactory examination of their mentality. On the other hand the patients themselves find extensive explanations so difficult to communicate that they are content with the shortest possible responses, so that they frequently mislead the examiner into the belief in an intellectual poverty which does not really exist.

With these warnings it may be conceded that in general the chronic choreic is of somewhat enfeebled mentality with a tendency to emotional depression and to a mood of suspiciousness. The emotional depression can be readily understood, as the patient realizes quite well his plight, namely, that he is affected with a chronic disease which practically isolates him from his fellows. Not infrequently there is a marked emotional instability and irritability, and such choreic patients may have great difficulty in getting along in wards where they come in contact with other patients. Finally, with the increasing deterioration there is a tendency to indifference, to emotional deterioration. The suspicious mood, which so many of these patients have, we believe has at least in part its explanation in the patient's isolation as a result of the nature of the disease. We find suspicious moods in all persons who are cut off from active contact with their fellows, more especially in people who are closely confined, either voluntarily or otherwise, those who suffer from pronounced varieties of aphasia, which make it impossible for them to communicate their thoughts or to receive communications from others, and predominantly among the deaf. To a certain extent, too, the dementia is probably due to this cutting off of active contact with life by im-

pairing the means of communication. The choreic finds it extremely difficult to talk to others. He may find it equally difficult to read, perhaps because of defect of vision which is liable to be present, as it must be remembered that this is a disease of later life, and in various ways he loses touch with what is going on about him. Finally, when he has long passed any ability to pick up the thread of events he becomes indifferent, and when it is realized that he is frequently in the arteriosclerotic period, often approaching the senium, it can be seen that this removal from acute contact with reality tends to produce inactivity, as it were, and appearances of dementia, if not actual dementia, and so the chronic choreic shows emotional defects, attention disorder, lack of impressibility, defect of recall, and in other ways demonstrates that he has lost interest, as it were, in life.

A certain few cases show distinctly more pronounced psychotic symptoms, occasionally showing well-marked persecutory ideas (See Chapter X.)

Paralysis Agitans.—It is probable that the majority of cases of paralysis agitans show a certain amount of mental disturbance, although in a great number of cases this disturbance is so slight compared with the physical and so easily seen to be dependent upon it that it is quite overlooked. This simple disturbance is in the emotional sphere and for the most part is one of slight depression but rarely one of euphoria. Occasionally deliriod episodes supervene, with marked confusion, but in general consciousness is clear and the patient remains well oriented, except of course, in the terminal condition when arteriosclerotic changes and the deterioration of the senium have set in. Occasionally there is a well-marked psychosis in which instance it is most apt to be of a depressive hypochondriacal character, often with paranoid coloring. The depression may be sufficiently great to result in efforts at suicide. In these cases disorders of the sensorium may also be present.

As intimated above it must be remembered that we are dealing with a disease of later life and that not infrequently arteriosclerotic and senile changes show themselves by changes in the mental sphere.

Multiple Sclerosis.—The outward manifestations of the mental disorder associated with this disease are mainly in the emotional sphere. It has been stated both that the majority of patients were depressed and that the majority were exalted. Both these statements are based upon the observation of very few patients. Although exaltation and depression may be manifest and there may also be spasmodic laughing and crying, due probably to lesions of the basal ganglia, which are unaccompanied by emotion, there is a certain amount of deterioration which is progressive and which depends of course upon the destructive changes. When the distribution of the sclerotic patches is quite similar to the distribution of the lesions in general paresis the outward symptoms of the two conditions may be very

much alike and lead to confusion in diagnosis. The diagnostic problems, however, can be cleared up by the laboratory findings.

Polyneuritis.—See Korsakow's Psychosis.

Pellagra.—There has been very little study of the mental symptoms of pellagra of late years. From the few cases observed and from study with those who have had it under observation there seems to be a variety of symptom pictures.

Many cases present no nervous or mental symptoms at all. In those who do there seems to be a tendency toward a variable localization of the disease process. There seems to be cases in which the spinal cord suffers most and others in which the brain suffers most. In this latter group a condition of very *acute delirium* may be developed running a rapid course to fatal termination and reminding one of the acute forms of paresis. The more frequent condition, of which we have seen a number of cases, seems to be more in the nature of a *simple retardation*. The patient moves slowly, or not at all, and answers questions after a long delay in a low tone of voice and in monosyllables. There does not go with this retardation, however, a corresponding emotional depression as in melancholia. We have also seen *pellagrophobia* in an infected territory.

With this disease, as with many others, it must not be forgotten that it may be associated with various psychoses without having any specific relation to them. This is peculiarly so in this country, as the large groups of cases which have occurred have been in hospitals for the insane.

Perhaps Gregor¹ has made the most careful recent clinical study of the mental symptoms. He considers his cases under the following seven categories: (1) Pellagrous neurasthenia, (2) stuporous group, (3) mental aberration, (4) acute delirium, (5) katatonia, (6) anxiety psychosis, (7) manic-depressive group. It does not seem clear, however, just what is the connection in all of these cases between the pellagra and the psychosis.

Heart Disease.—Depressive states are most in evidence here. Mental symptoms, however, are most apt to be associated with failure of compensation. With precordial distress goes typically a mental state of anxiousness. Transitory confusions with dreamy hallucinations occur with compensation disturbances and edema.

Various other diseases have from time to time mental symptoms associated with them. The great majority of such diseases, if not all of them, have elements of infection, or toxemia and exhaustion combined, with all or part of them. The mental symptom-complex of confusion arises most typically. In some cases, especially the less acute, paranoid conditions occur and hallucinosis is of occasional occurrence.

Head has shown that certain visceral diseases, especially of cardio-

¹ Beiträge zur Kenntniss der pellagrösen Geistesstörungen, Jahrb. f. Psych. u. Neurol., 1907.

vascular and pulmonary origin, often have associated mental symptoms, although they may not appear except on the most careful examination. The symptoms found are: (1) Hallucinations of vision, hearing and smell; (2) moods, either of depression or exaltation, and (3) suspicions usually occurring when a depression has persisted for some time.

These conditions take their origin in part as a result of reflected visceral pains. Each spinal segment has both a visceral and a cutaneous representation. Disease occurring in the visceral area is referred to the cutaneous surface supplied by the same segment. The cutaneous distribution of the fifth nerve corresponds to the visceral distribution of the vagus, so pain occurring in the vagus territory will be referred to the scalp and thus occur points of tenderness in this region with which the hallucinations are associated. The mood of exaltation is essentially transitory and arises as a contrast phenomenon of the depression and as a result of the disappearance or lessening of the reflected somatic pain.

CHAPTER XXIV.

PRESENILE, SENILE, AND ARTERIOSCLEROTIC PSYCHOSES.

THE grouping of the presenile, senile, and arteriosclerotic psychoses together in one chapter is a matter not only of convenience, for obviously one would expect all sorts of admixtures, particularly as between the senile and arteriosclerotic group, but there are many pictures here which are not distinctive, especially in the presenile period, which Kraepelin begins his discussion of by saying it is the darkest region of all psychiatry today.

The Presenile Psychoses.—In the presenile period there are a number of psychotic pictures the exact significance and nosological placement of which is not at all understood. There is no doubt, however, but in this period that depressions are much more in evidence than excitements, the depressions seeming to mount up in frequency during the period of involution. Hübner found, for example, 21 cases of single attacks of melancholia after the fiftieth year, but only 2 cases of single attacks of excitement.

Involution Melancholia.—*Symptoms.*—The Kraepelin school for a considerable time considered that certain depressions of later life, characterized by an anxious apprehensive agitation, with profound emotional depression, composed a nosological unit to which was given the name involution melancholia, or more briefly melancholia, the term melancholia being limited in its application to this particular type of depression.

The symptoms of this psychosis are generally preceded by a considerable period during which there are vague head symptoms, such as pressure, pain, vertigo, together with anorexia, irritability, insomnia, mental insufficiency, and a mild neurastheniform state with perhaps some emaciation. This condition becomes progressively worse and the patients develop an agitated depression, with anxiety, apprehension, fear of impending danger, with quite usually delusions of sin. The depression is characteristically very profound, and the motor agitation may be quite considerable. The patient may go about wringing his hands, moaning and groaning, perhaps repeating over and over again such phrases as "Oh, my God! Oh, my God!" "It is a fearful thing. Good Lord help me!" One patient constantly repeats "Doctor, will I be done away with tonight?" and "Then will I be here tonight just the same as last night, and will I be here tomorrow just the same as today?" Another patient believes herself very sin-

ful, refuses to eat because the food should be used for others, walks about in an agitated manner picking her fingers and attempted suicide because she was afraid she was to be put to death.

Even in these cases of quite extreme agitation and profound depression consciousness remains unclouded, orientation is little if at all impaired, and the form of thought is well maintained.

Another type of case presents symptoms of retardation. Many of these cases may be so retarded as to be almost stuporous and some of them present an amount of resistance which reminds one of the characteristic negativism of dementia precox. This latter group was originally described by Kraepelin as "late catatonia." Its nosological status is at present in doubt.

Certain cases of anxious depression may be highly agitated with insomnia, refusal of food, rapid emaciation, clouding of consciousness, hallucinations, self-inflicted injuries, attempts at suicide, a rapid course ending in death.



FIG. 313.—Facies of involution melancholia.

In the marked cases of apprehensive and agitated depression there is frequently a considerable degree of precordial distress with perhaps tachycardia and often a sense of oppression over the chest with a feeling of difficulty of breathing. These symptoms are apt to appear in attacks, at which times the depression is more pronounced.

The danger from suicide is very great in this psychosis, particularly because the patient is not so frequently rendered inactive by marked retardation.

The delusional content of consciousness varies widely, but hypochondriacal delusions, nihilistic delusions, and the feeling of unreality are common. Occasionally the delusions take on bizarre, absurd and fantastic forms, and there may be delusions of grandeur late in the course of the disease. There may be a strange mixture of depressive and grandiose ideas, as in the patient of Weygandt, who believed she was going to be boiled in a silver kettle.

The statistics of this disease show that about 40 per cent. got well, the remaining 60 per cent. terminating in various ways: some by

suicide, some by death from intercurrent disease, which they were ill adapted to withstand; some by death from general marasmus or the development of tuberculosis; some lapsed into chronicity; and finally, a few improved sufficiently to leave the hospital and get along at home, though still somewhat depressed. A certain number of this class may get worse under home surroundings and have to be returned to the hospital.

This group of involution melancholia, so-called, was studied with great care by Dreyfus.¹ He studied the life histories of 81 cases, 34 of which were personally investigated. Eight were not personally investigated, and 39 were deceased. As a result of his study he concluded that with the exception of two cases undiagnosed, two cases in which a mistake in diagnosis had been made, and possibly two more doubtful cases, that all were cases of manic-depressive psychosis. This conclusion was reached by finding the fundamental symptoms of this disease present. Of these cases 66 per cent. were recovered or were recovering at the time of death, 8 per cent. developed arteriosclerosis, 25 per cent. died unrecovered of recurrent disease or suicide.

The conclusion reached by Dreyfus that the involution melancholia of Kraepelin is really a form of manic-depressive psychosis has now been accepted by Kraepelin himself. A study of this group of cases shows quite characteristically the presence in the history of periods of affect fluctuation; and quite usually periods of slight depression which were not severe enough to attract attention seriously to the mental condition, or perhaps were not at all understood at the time, being accounted for in various other ways.

Even admitting that this group of involution melancholias really belong to the manic-depressive psychosis, still the problem is not wholly solved and it must be admitted that in all probability the involution period has certain modifying effects upon the psychosis.

In the first place the depressions as noted heretofore are very much more frequent than the excitements and they are very much longer in duration than during earlier life. This of course can be easily understood by the failing resistance of the involution period. A psychosis that has manifested itself only by mild and hardly appreciable evidences throughout the youth of the individual may well get a firm hold when the powers of efficient reaction are failing.

With the recent work that has been done on the ductless glands and with the somewhat characteristic mental pictures that go along with disturbances of the internal secretions one must bear in mind that perhaps many of the changes of the involution period are, in part at least, determined by changes in these glands, particularly as the result of atrophy of the uterus, the ovaries, the prostate, the testicles, and the adrenals, with the possible result of an imbalance being brought about in the relationship between them. This of course,

¹ *Die Melancholic, ein Zustandsbild des manisch-depressiven Irreseins*, Jena, Gustav Fischer, 1907.

if it is so, would only be one of the modifying factors of the involution period which one might expect to see reflected in the diseases at this time of life.

The following case illustrates this type of psychosis: The patient, a woman, was admitted to the hospital at the age of fifty. She was very much depressed, said that she was very unworthy and should be hung, that she had committed the unpardonable sin and would burn forever in the flames of Hell. A little later, in addition to these ideas that she had committed the unpardonable sin and that her soul was lost, she said that she was dead, what existed of her now was her spirit, that her body had passed away, and because she was dead it was useless to attempt suicide. This condition had its origin some two years before, following an operation, when she had cleared up, and as a result, so stated, from overwork, had broken down again and developed these ideas, and subsequently was admitted to the hospital after having made a suicidal attempt. In addition to the delusions she had visual hallucinations, saw spirits and heard them talking to her.

This patient gave the general impression on her admission to the hospital of a patient suffering from involution melancholia. A further inquiry, however, developed the history of numerous previous attacks of depression, which suggested a manic-depressive reaction type. Just before her discharge from the hospital, however, when she had become much more accessible, we learned that in her early life, before she was twenty, she had had a love affair. This love affair had been a very profound emotional experience, particularly because she had found it necessary to give up the young man because of his drinking habits. That this was not willingly done, however, was shown by her subsequent life, which was taken up by constant activities, largely of a social nature, and apparently for the purpose of side-tracking her disappointment. She was an attractive young woman and had many admirers, but did not permit herself to become attached to any one of them. She led a very active life and was able to sublimate the energy of her suppressed emotions until the period of the menopause, when she broke down with self-accusatory delusions and suicidal tendencies. She believed she had committed the unpardonable sin; this sin was having committed adultery in her mind with the young man with whom she had been in love in her youth. The psychosis lasted somewhat over five years, at the end of which she finally succeeded in reaching a compromise by developing tendencies diametrically opposed to her delusional system. She became talkative, humorous, and particularly facetious, and just before her discharge she made the remark that she was born dead, but made it with a smile, and with the added comment that she had gotten along pretty well all her life for a dead person. This condition of relative equilibrium enabled her to go back to her family and in the absence of disturbing factors she will probably get along all right, at least for a considerable

time. Nothing has been heard of her for some years. The compensation, the balance struck, was a fairly efficient one.

Such a case as this illustrates a common type of depression which occurs in the involution period. It is a type which if dealt with solely from the descriptive standpoint would have been considered an involution melancholia, while a little more careful history indicates that it is a manic-depressive reaction type, while a still more profound probing into the facts of the life make the whole thing quite understandable in psychological terms. It is manifestly a psychogenetic depression, but whether it should be called manic-depressive or not is a question. Now that psychoanalytic methods are showing the presence of endopsychic conflicts in the manic-depressive psychosis, such a case as this shows the close connection between a pure psychogenetic psychosis and the manic-depressive reaction type, and the further relation of these conditions to the depressions occurring during the involution period.

Pathology.—An increased neuroglia formation in the deeper layers of the cortex has been described, and in this disease we find most often a termination in the condition described by Meyer as *central neuritis*. This is essentially a parenchymatous degeneration with swelling of the cell body, a disappearance of the chromophile substance, and eccentricity of the nucleus. In rapidly fatal cases there are evidences of extensive destructive changes in the cortex. There are also found a certain amount of disintegration products.

Treatment.—One of the very important considerations in this group of depressions, as shown by the symptomatology, is the prevention of suicide. It is therefore very much more frequently necessary to interne these patients so that they can be properly watched. Insomnia, agitation, failure of nutrition with suicidal tendencies are the conditions that have to be met. For the agitation and the insomnia hydrotherapy is the best means of treatment. As few drugs as possible should be used. For the suicidal tendencies constant watchfulness, which should have as its ideal an unobtrusiveness which does not suggest the suicidal idea constantly to the patient's mind. Failing nutrition must be met by tube feeding if necessary.

An analysis of the patient's mental symptoms with a view to understanding them is of course highly desirable, but this class of cases do not lend themselves easily to analytic technique, at least until they have begun to quiet down, oftentimes not until they are convalescent.

Other Psychoses of this Period.—Various other types of psychoses have been described as belonging in this period. Types of depressive Wahnsinn, some of them associated with anxiety and proceeding to deterioration, while Kraepelin has described a very small class of so-called presenile delusional psychoses, affecting especially women, in which delusions of infidelity are usually prominent, together with hallucinations. The delusions are variable and do not seem to at all logically control the conduct. Farrar has described three types of

psychoses belonging to this period: *melancholia vera*, *anxietas præsenilis*, and *depressio apathetica*.

In *melancholia vera* there is in the main an autopsychosis. The delusions are auto-accusatory, with ideas of sin but with clear consciousness; there is no defect of orientation. The patient believes his soul is lost, that he is to suffer eternal torment hereafter, and about these beliefs there is no doubt, but on the contrary a marked "subjective certainty." There may be some slight tendency to somatopsychic delusions, insight is defective, and slight anxiety may be present.

In *anxietas præsenilis* there is, on the contrary, in the main an allpsychosis. There is very marked "subjective uncertainty" which gives an unreal tinge to the outer world, and out of which grows the fear of things unknown, culminating in the marked anxiety which is characteristic of this form of the psychosis. Remorse or dread of the future are not elements in the depression; on the contrary, it is the great unknown and overwhelming present that seems about to destroy them. These cases occur later in life than the former, show more evidences of senile decay, such as arteriosclerosis, and present such symptoms as verbigeration, rhythmical movements, suggestibility; the prognosis is less favorable.

In *depressio apathetica* there is simply a let-down, a stopping on the part of one who has been leading an active life. Interest abates, the struggle is drawn away from and we have a picture of mild depression with clear consciousness and no disturbance of orientation. There is some "subjective uncertainty" delusions and sensory fabrications play little part. The symptoms are negative rather than positive; the prognosis is relatively good.

Finally one must remember that a true anxiety neurosis may develop, and it not infrequently does, at this period of life.

In conclusion then it will be seen that we have during this period of life a group of psychoses of widely different forms, although tending toward a certain amount of uniformity in their outward expression in that depressions are so much more frequent at this period. In the first place there are frank attacks of manic-depressive psychosis with here as elsewhere marked differences, depending upon the severity of the constitutional taint upon the one hand, or its absence and the presence of a reactive type upon the other. Apprehension and anxiety appear frequently at this period of life, and a true anxiety psychosis is not uncommon. Paranoid conditions occasionally occur and disorders of the sensorium are not infrequent. In a certain group of cases negativistic tendencies are sufficiently marked to make the outward semblance to catatonia quite pronounced.

Certain of the psychoses of this period tend toward more or less rapid deterioration, and in these at least there are evidences of marked physical changes. In other words, the physical side of the disease has to be kept constantly in mind and must be considered to be the

most important prognostic feature. The changes incident to vascular degeneration, while of course not usually prominent in the fifth and sixth decades, may well be in evidence, while the changes in the ductless glands are to be borne in mind. Other changes undoubtedly occur, but their nature and their bearing upon the psychosis are not at all understood. The psychosis must, nevertheless, be expressed in psychological terms and the explanation of the mental symptoms must be sought by psychoanalytic study. We have here, then, diseases which have a marked physical side apart from the mental manifestations, the physical side being more prominent than in dementia precox and less prominent than in general paresis.

The Senile Psychoses.—The normal course of life leads to a certain amount of gradual mental and physical deterioration during the latter years. Whether this occurs or not probably depends upon many factors, for we see some men at sixty as old as they should be at seventy-five, and other people at eighty presenting a wonderful degree of elasticity and enthusiasm without any apparent falling off in mental powers or interests. This variation was wont to be expressed by saying that "a man is as old as his arteries," but it is certain that there are many other factors besides the condition of the bloodvessels that lead to senile deterioration. In fact senile deterioration may take place and lead to very profound dementia without material disease of the bloodvessels at all.

Intermediate Conditions.—A certain number of the involution psychoses continue over into the senile period when the patients undergo the mental and physical changes of senile involution. This is in part, at least, the reason for the termination in dementia of a certain group of involution cases, aside from the added obvious fact that vascular degeneration is also an important etiological moment. The cases that show this outcome in dementia are more especially the paranoid types. This group of cases shows, therefore, that there is a gradual transition from the psychoses of the distinctly involution period to those of the senile period, a perfectly understandable condition if we conceive of the psychotic manifestations as being expressions of mental conflicts that are at the basis of the individual character and which express themselves in the later years of life when efficient reaction is becoming progressively less possible, and which later on become fixed, chronic, and disintegrating at a time when the physical changes of the senium cooperate to this end.

Normal Senile Involution.—The more usual symptoms of senile involution occurring after sixty are in the main a loss of memory for recent events, due to lack of impressibility to the extent even that events of only an hour before are completely forgotten, lack of ability to recognize faces, marked egotism, so that others' wants and comforts are not considered, which may be associated with some irritability on interference. There is developed more and more as the years go on a true misoneism, so that the patient will positively not tolerate

any change in the usual order of things, everything must be done the same from day to day, the same seat is preëmpted, a particular kind of food demanded, and the like with other comforts. With this mis-*oneism* and the lack of memory for recent events goes a marked tendency to reminiscence. The events of youth and the years long past, unlike those of recent occurrence, are vividly recalled and the patient thus really lives in a world of former days, constantly recalling and reiterating things that occurred long ago. This condition becomes progressively worse, the patient leading a vegetative existence almost wholly, no mental initiative, failure of judgment and a progressive loss of comprehension of the environment, so that there is no adequate grasp of the present at all.

With this mental failure goes a corresponding change on the physical side. The signs of age are evident in the wasted muscles, the wrinkled, inelastic skin, gray hair, the raucous voice, *arcus senilis*, senile cutaneous affections, and signs of arteriosclerosis in the superficial arteries. In this condition it should be remembered that the condition of the palpable arteries may not indicate at all the condition of the cerebral vessels. The superficial vessels may show marked arteriosclerotic changes, while the cerebral vessels are in relatively good condition, or, on the contrary, the cerebral vessels may be seriously affected in a person whose radials are comparatively soft and whose temporals are not noticeably tortuous.

Upon this background of dementia there may appear the usual pictures—excitements, depressions, paranoid states, stuporous states, and confusions. All of these conditions, however, must be reconstructed in psychological terms to receive any explanation at all, and it is not difficult oftentimes to make out some logical reason for the particular type of delusional formation.

In the senile dement and in the delusions of this period there is noticed an apparent indifference in the emotional sphere, an emotional poverty. Persecutory delusions, delusions of infidelity, hypochondriacal delusions take on grotesque forms, and their expression is not accompanied by an adequate affect. A patient tells of severe injuries, of having been shot, while perhaps smiling; another patient tells a long and pitiful story about her affairs and about a claim she has against the government, but tells it in a stereotyped, matter-of-fact way without the expression of any feeling. This condition is usually described as one of emotional blunting, emotional deterioration. Bleuler,¹ however, lays great stress upon what he believes to be the fact, namely, that there is no lack of capacity for feeling, but that the affectivity is only disturbed secondarily, that patients with organic brain disease fail to get a sufficiently clear idea and therefore do not react adequately in the emotional sphere. Wherever it is possible

¹ Affectivity, Suggestibility, Paranoia. Translated by Chas. Ricksher, New York State Hospital Bulletin, February 15, 1912.

to get a sufficient comprehension of the situation the emotion of the proper quality is manifested and with commensurate intensity.

Simple Senile Deterioration.—This condition of gradually progressing dementia without marked psychotic disturbances is designated as simple senile deterioration.

The senile dement is apt to be restless and suffer from insomnia, reversing the time of day and sleeping perhaps in the daytime and lying awake at night, perhaps wandering about the house at night in a more or less disoriented condition. This tendency to disorientation, without the development of delusions occurs in the course of the progressive deterioration, and it is where disturbances of the sensorium are marked and confusion becomes very much more in evidence than we have in the condition of senile delirium. This confusion, however, may be only transitory and is quite characteristically in evidence in the intervals between waking and sleeping.

Senile Delirium.—This is merely a form of senile deterioration in which confusion dominates the picture and in which usually disorders of the sensorium are also markedly in evidence. Patients are highly disoriented, they do not know where they are, or what time of day it is, do not know whether they have just had their dinner or whether it is time to go to bed or get up, are frequently quite active and irritable, and are apt to die from exhaustion. The delirium may take the form of an occupation delirium.

Presbyophrenia.—A certain proportion of these patients resemble very closely the Korsakow's psychosis, being disoriented as to time and place and supplying memory defects by fabrications. One old man, for example, who was so feeble he could hardly stand, relates that he had been working for a man, making some sort of a wire affair for the past seven months.

Course.—The course of senile dementia in its various forms is a progressive one. The patients tend to become profoundly demented, wholly disoriented, and die naturally of marasmus. Where the change is somewhat more acute, delirious or confusional episodes may be in evidence from time to time, and of course if the excitement is at all prolonged exhaustion is the result. The patients are naturally susceptible to intercurrent affections, particularly pneumonia, nephritis and cystitis, and many of them die in delirium, the result of a terminal infection.

Diagnosis.—Paranoid conditions may resemble paranoid states of earlier life, but occurring in the senium, usually show evidences of organic brain disease and marked mental deterioration.

Some of the patients also show similarities to dementia precox, especially those that develop negativistic reactions. Just what relation these cases have to the precox of earlier life is not fully known. The marked mental dilapidation and the period of life at which they develop usually give the clue to the diagnosis.

A few cases show a mixed symptomatology, presenting the picture

of senile deterioration on the one hand, together with the various symptoms of arteriosclerotic disease with focal lesions on the other hand. Here are found all sorts of mixtures of pupillary disturbances, disturbances of speech, and various forms of paralysis.

Certain borderland conditions show marked emotional states of either depression or excitement which dominate the picture for the time being. These cases show beneath the emotional state the presence of a defect which shows the basal disorder.

The similarity between presbyophrenia and Korsakoff's disease is very considerable. Nouet¹ has recently made a careful study of the two conditions and has set down the following differential criteria:

Korsakow's psychosis affects persons particularly of adult age; presbyophrenia, on the contrary, belongs to the period of old age, the age of predilection oscillating about seventy years. Presbyophrenia, it is known, is quite uniquely an affection of women, while Korsakow's psychosis is far from being rare among men. The humor



FIG. 314.—Presbyophrenic facies.

of the presbyophrenic is always gay, euphoric and satisfied, quite different from the psychopolyneuritic, who is ordinarily depressed or apathetic, and whose face preserves an invariable immobility. The traits of the presbyophrenic are extreme mobility, they laugh, make grimaces in which the mimic muscles participate. The facies of the psychopolyneuritics, however, are always dull and without expression. Loquacity is a symptom scarcely ever lacking in presbyophrenia; the patients talk without stopping about everything, with equal volubility. This symptom is lacking in Korsakow's psychosis. The presbyophrenic is polite, amiable and cordial, characteristics which one seeks in vain among the psychopolyneuritics. Disorders of consciousness are much more marked in the presbyophrenic. These patients have no understanding of their state of illness and content themselves with laughing when one asks a question relative to their

¹ Presbyophrénie de Wernicke et les Psychopolynéurites, *L'Encephale*, February 10, 1911.

physical or mental health. On the contrary the psychopolyneuritic gives a fairly good account of himself and of his position, and is the first to lament his situation. The judgment of these patients, even in the chronic forms, is less noticeably affected, and their degree of intellectual enfeeblement less marked. The amnesia, finally, is more profound in the presbyophrenic, and besides in this affection the patient does not possess at all a knowledge of this amnesia—the inverse of what one observes in the chronic forms of Korsakow's disease, where the subjects speak spontaneously to their interlocutors of the profound troubles of their memory. Presbyophrenia is rare in its typical forms, but common in the *formes frustes*. Where the symptoms only approach, or where certain of them are lacking, the presbyophrenia has, perhaps, the distinctive characteristics of arteriosclerosis, which are seen much more among these patients than among simple senile dementis. Patients die nearly always of cerebral hemorrhage, and their nervous centers present at autopsy pronounced atheromatous lesions.

Pathology.—Grossly the brain shows signs of atrophy and is decreased in weight, the bones of the skull are thinned, sometimes in well-defined regions, particularly the temporal regions. There is a compensatory external hydrocephalus as a result of the atrophy of the brain. The convolutions are shrunken and the fissures correspondingly widened. The atrophy is not uniformly distributed and may be very much more marked in some areas than others. Arteriosclerosis is a frequent finding, but is not a necessary part of the picture. In fact the two processes are quite distinct in every way, although frequently associated. When arteriosclerosis is present there may of course be found its results in such lesions as softenings.

The architectonic of the cortex is greatly disturbed. The nerve cells show advanced degeneration with large quantities of degenerative products of a lipoid nature within them, amounting to a severe grade of fatty degeneration. A very characteristic picture are the basket formations about the nerve cells. The neurofibrils appear to be thickened and produce whirls and loops about the nerve cell. It is thought by some that these basket formations are due to incrustations upon the neurofibrils of perhaps neuroglial origin. The condition is particularly well seen in presbyophrenia.

The miliary plaques are perhaps the most distinctly pathological findings in senile dementia. In fact they are distinctive as much as any pathological finding can be distinctive of any one condition. Their presence in considerable numbers practically makes the diagnosis of senile dementia, while their absence or extreme rarity would negative such a diagnosis. They are small spots of necrosis which are not dependent upon softenings and are in no way associated with the bloodvessels.

There is a great deal of neuroglial proliferation throughout the brain and marked evidences of disintegration and the presence of scavenger cells and the like.

Aside from these changes the usual changes of this period of life are found, particularly in the heart, the kidneys, and other internal organs.

Treatment.—The mild cases, especially those that maintain their orientation fairly well, can be cared for at home. Those with marked confusion, especially with a tendency to wandering, need an attendant to be with them. There is danger of their becoming lost and coming to grief, or if they wander about the house at night they are apt to meet with some accident, more often to fall down stairs and sustain fractures. Patients who are very resistive, present surgical troubles, are filthy in habits, or show a tendency to commit sexual offences, should be cared for in an institution.

As regards the more special treatment, little is to be said. Hygienic surroundings, a simple diet, looking after the emunctories, and if insomnia is present the occasional exhibition of a hypnotic constitutes about all there is to be done. In this class of cases, more perhaps than in any other, is the use of alcohol as a hypnotic indicated. A little whisky and hot water, or a glass of beer or ale acts very nicely. It should be given, however, strictly under medical authority and supervision, as these patients are apt to be susceptible to its influences. In the earlier stages of the disease potassium iodide is the drug par excellence for its general alterative properties and its effect on the arterial tension.

As soon as evidences of mental deterioration appear relief from business worries, cares, responsibilities, and mental stresses of all sorts is indicated, with the general hope of limiting disintegration as far as possible. Whether this is of value or not is pretty difficult to state, for it must be remembered that there appear to be certain hereditary tendencies involved even in this condition and that arteriosclerotic disease and senile dementia both appear to be more prevalent in certain families.

Alzheimer's Disease.—This disease was first described by Alzheimer in 1906 and since that time a number of cases have been reported. The disease occurs usually in the fifth decade, although cases have been reported in the early part of the fourth decade and as late as the beginning of the seventh. The symptomatology is one of a gradually, often of a rather rapidly progressing dementia, interrupted perhaps with episodes of a certain amount of excitement and anxiety. A rather rapidly progressing dementia in a man of about forty, or a little over, is in itself a rather unusual picture, and in the absence of signs of brain tumor or syphilis, Alzheimer's disease should be thought of. The dementia is marked by a considerable degree of disorientation. The symptoms otherwise are very largely neurological and particularly focalized about the function of speech. Various aphasic and apraxic symptoms are prominently in evidence. Paraphasia and asymbolia are quite frequent. There may also be spasticity and convulsions, although there are never well-marked paralyses.

The pathology of this condition throws almost the only light upon its nature. There is marked and extensive degeneration of the nerve cells with disturbances of cortical architectonic and the presence of large quantities of disintegration products, while there are found numbers of basket formations and typical miliary plaques. The condition of the bloodvessels is quite normal. The pathological picture thus resembles closely that found in senile dementia. In general, the disease is considered as a presenile dementia, although some believe it to be a distinctive disease.

The presence of this disease, if it be considered a presenile dementia, offers another one of those warnings to us not to be too dogmatic. It would appear that the senium is by no means a clearly defined period of life, and that the pathological changes which are usually thought of as dependent upon old age, may occur within wide limits and perhaps represent failure of special tissue resistances.

Arteriosclerotic Psychoses.—These psychoses are dependent, on their physical side, upon arteriosclerotic changes in the cerebral bloodvessels, and this condition is due in turn to the general causes which produce arteriosclerosis. In the main the two factors are chronic toxemia and high blood-pressure. It must be borne in mind, however, that an advanced degree of arteriosclerosis may exist, particularly in the peripheral vessels, and the cerebral vessels retain their elasticity, while on the contrary the cerebral vessels may be severely sclerosed while the peripheral, palpable vessels show little change. Arteriosclerosis is essentially a regional disease.

The psychoses of arteriosclerosis form another one of the connecting links which join the period of involution and the senium. Many of the involution psychoses merge into arteriosclerotic deterioration, and arteriosclerosis is frequently combined with the changes incident to the senium.

Aside from the usual causes of arteriosclerosis there would seem to be certain hereditary factors at play. Certain families show a high incidence of death dependent upon arteriosclerotic disease.

There are four fairly well-defined varieties of this disease based upon both clinical and pathological findings as follows:

1. **Arteriosclerotic Brain Atrophy.**—This occurs in two forms: a mild form with severe arterial sclerosis but an absence of focal brain lesions. The symptoms are easy fatigue, slight failure of memory, dizziness and headache. The severe type may resemble the mild at first but is progressive, leads to profound dementia and presents in its course apoplectiform and epileptiform attacks and focal symptoms.

2. **Subcortical Encephalitis (Binswanger).**—In this condition the white matter is largely involved as a result of disease of the long medullary arteries. Apoplectiform and epileptiform attacks occur and also transitory attacks of confusion, aphasia and paresis, disturbances suggesting focal lesions. Focal lesions are not found extensively but areas of softening often occur in the basal ganglia.

3. **Perivascular Gliosis.**—In this condition there is a disappearance of nervous elements about the diseased vessels and replacement by neuroglia.

4. **Senile Cortical Devastation.**—Here extensive destruction of cortical areas in the vascular territories of the diseased vessels is found. In this condition the arteriosclerosis is localized largely in the small cortical vessels which come off from the pia. The basal vessels remain relatively normal.

Pick has very thoroughly described certain large atrophies involving whole lobes or portions of lobes. The occipital lobes may be involved, producing blindness, or the temporal, producing deafness, for example. The atrophy, however, does not always follow a vascular area, and so, while it is generally supposed to be due to arteriosclerotic disturbances in the irrigation of these territories, the cause is not always altogether clear.

Symptoms.—In the main the symptomatology of cerebral arteriosclerosis is one of gradually progressive mental deterioration, to which are added the evidences of focal lesions which are the results of thrombotic softenings. (See Chapter on Hemorrhage.)

The prodromal disturbances of the arteriosclerotic psychoses are very apt to extend over a considerable period of time, and manifest themselves in the main as nervousness and irritability, with headache, dizziness, insomnia, associated of course with the special signs of the vascular disease, more particularly as a rule, high blood-pressure. With this series of symptoms there may be of course associated a certain amount of deafness, with sclerosis of the drum membrane, cardiac attacks with the Stokes-Adams syndrome and evidences of interstitial nephritis. Quite frequently, too, these patients show the signs of arteriosclerosis of the vessels of the spinal cord, with perhaps some sclerosis of the palpable vessels. On the mental side the patient may have a feeling of growing inefficiency, at least his work shows a falling off in efficiency, which characteristically manifests itself at first in a failure to do the creative things. Pick has called particular attention on the emotional side to a lack in the finer modulations of the emotions. Bleuler's suggestion must be remembered, that patients with organic brain disease fail to show a natural emotional reaction, not because of any defect of emotion, but because of a lack of grasp of the situation, and when the situation is fully appreciated an adequate emotional response issues.

These prodromal symptoms gradually merge into symptoms of greater severity and may be punctuated from time to time by attacks of excitement or of depression, and delusions may develop which are characteristically of the paranoid type. The following case illustrates this paranoid trend very well: A man who had been a successful business man in his younger days began to fall off in efficiency as he entered the arteriosclerotic period of life, and finally entered one of the Soldiers' Homes as he was unable to adequately support himself.

Here, because of his training as an accountant, he was employed in the office. While employed here the characteristic series of paranoid ideas developed. In the first place he developed exalted ideas of his own ability which were distinctly defensive in character, compensating him for the real fact, his failing ability. Alongside of these exaggerated ideas he had delusions of a persecutory character. He was interfered with by those about him who were envious of him because he had secured such a good position in the Governor's office. This is another defense reaction in the opposite direction and serves to explain to him how a really efficient man after all can turn out such poor work. It is not his fault, but the fault of those about him, and so he is again saved from the realization of his failings. Removed from this situation and taken to a hospital where he could no longer indulge in alcohol, which had been a factor in bringing about this condition, he recovered from these distinct psychotic manifestations, but without, however, a full insight into what his condition had been. This lack of insight probably had its basis in the organic changes. The man was no longer capable of adjustment, except within narrow limits, and while removal from the painful conditions relieved the situation, he was unable to fully understand it.

This case shows very well how even in a psychosis dependent upon organic brain disease the mental symptoms as such must receive a psychological interpretation.

From time to time these patients show periods of confusion and bewilderment, with disorientation. These periods may be of considerable duration and some of them at least are dependent upon thrombotic attacks which, when they do not occur in the motor area, are easily overlooked. Even though they do occur in the motor area the disturbance of consciousness may not be very great, or at least there is nothing comparable to the unconsciousness and coma of cerebral hemorrhage; there is, perhaps, only a slight confusion, and the paralysis, if it exists, is only very slight and is often explained by the family as the result of some inconsequential cause. Then again the patient may be so blunted mentally as not to complain of a slight impairment of function.

These patients are especially susceptible to alcohol and become easily very badly confused from small quantities.

One of the characteristic features with regard to this group of psychoses is that the so-called "nucleus of the personality" is well preserved. The patient preserves all of the outward appearances of his old self until the mental dilapidation has reached an extreme degree. There is not, as a rule, that marked "change" in the individual that is seen in some of the psychoses.

The areas of softening produce focal lesions and these focal lesions are most prominent in the motor areas, producing various upper motor neuron paralyzes, and in the speech areas producing various forms of aphasia and apraxia. The marked focal lesions, particularly

those which lead to well-defined speech disturbances hasten very greatly the dementia. An aphasia which puts the patient out of actual touch with his fellows hastens the tendency to mental deterioration in this class of patients who are no longer fluid, but are well along on the down-hill path of life. They need every stimulus of mental activity to even hold their own, and as soon as an important function like speech is destroyed they are quite apt to lapse promptly into a serious deterioration.

It is in this class of patients that, as a result of the focal lesions, epileptiform attacks develop late in life—the so-called “late epilepsies.”



FIG. 315.—Extensive thrombotic softening, the result of arteriosclerosis. Patient aged eighty-three years. There were also softenings on the right side in the lower portion of the cuneus, and upper portions of the lingual and fusiform lobules. There is general atrophy of the convolutions.

Their significance in the main is in pointing to the focal disorder, and as an indication that the patient needs careful oversight.

Pathology.—The pathology shows the arteriosclerotic process in the cerebral vessels in various stages of progress. There may be miliary aneurisms of the smaller vessels and other vessels may be completely occluded with resulting areas of softening. These areas of softening usually show entire disintegration of the nervous elements with large numbers of scavenger elements about. The nerve cells show various grades of degenerative change dependent upon deficient nutrition as a result of decrease in size of the lumen of the vessels. There

is neuroglial overgrowth about the vessels and in the degenerated territories.

Diagnosis.—The disease most apt to be mistaken for the arteriosclerotic psychoses are paresis and the psychoses of cerebral syphilis, which should be considered together now that the etiology of paresis has been so thoroughly established. The reason for the possibilities of mistake is dependent upon the fact that both processes are more or less diffuse, producing a progressive mental deterioration, and that both processes tend to be more severe at certain points and thus produce focal symptoms. Syphilitic and metasyphilitic disturbances



FIG. 316.—Widely distributed arteriosclerotic softenings; patient aged seventy-two years. There were also numerous softenings in the basal ganglia. Brain greatly shrunken. It can be seen from the distribution of these lesions how the clinical picture might simulate paresis.

occur as a rule at a much earlier age than the arteriosclerotic, generally not later than the fourth decade, while the arteriosclerotic period is not entered until the fifth decade. When an arteriosclerotic has had syphilis also the differentiation becomes more difficult. The Wassermann of the cerebrospinal fluid, however, would be negative, while with large areas of softening on the surface of the cerebrum there would be considerable evidence of disintegration products in the cerebrospinal fluid. On the mental side the preservation of the "nucleus of the personality" is much more in evidence than in paresis.

Treatment.—In a general way the treatment should be prophylactic as far as possible. With the first symptoms of prolonged and intract-

able high blood-pressure the individual should be removed from the influences of physical and mental stress, and the usual means should be employed to keep the blood-pressure down and to reduce or do away with any toxemic condition present. A carefully regulated diet from which alcohol and tobacco are excluded, hydrotherapy intelligently applied, possibly a visit to some watering place, and in some instances a visit to a somewhat higher altitude, with moderate outdoor exercise, and the exhibition from time to time of drugs to reduce the pressure and, especially where there are kidney complications, the drinking of considerable quantities of mildly alkaline water are in general the things to be depended upon. Insomnia has to be dealt with, and in cases where the patient is under absolute control a small amount of alcohol at night will sometimes produce the desired result. Unless the patient is under absolute control, however, it is best to give hot milk, or such simple hypnotics as veronal.

CHAPTER XXV.

IDIOCY, IMBECILITY, FEEBLE-MINDEDNESS, AND CHARACTEROLOGICAL DEFECT GROUPS.

IN drawing a distinction between dementia and idiocy Esquirol said: "The demented man is deprived of the good that he formerly enjoyed; he is a rich man become poor; the idiot has always lived in misfortune and poverty." In other words the idiot, the imbecile, and the feeble-minded lack something; the psychotics are suffering from a disorder of that which they possess.

This definition, so admirably worded by Esquirol, adequately expressed the distinction between the insane and the idiot and the imbecile according to the knowledge of his day, and has been used continuously since to express such distinction. It can no longer be said to be a valuable formula except it be used with many reservations and explanations.

In the first place, from the standpoint of this book there is no such thing as insanity, at least in a medical sense, as Esquirol used that term and as other psychiatrists in the past and the present have used it. Insanity is purely a legal and sociological concept, and as such does not imply anything more than the judgment of a man's fellows on the desirability of having him live in the community. Defectiveness, under which term the various grades of idiocy, imbecility, and feeble-mindedness are included, is quite as generic a term, and while it refers to a condition the fundamental characteristic of which is lack of development, it includes a great variety of states, and in its application is a relative term only.

It is quite as illogical to group all defectives together and endeavor to draw a conclusion from their study as a whole, as it is to group all of the so-called insane together and endeavor to draw a conclusion from the study of all of them. There is obviously very little similarity between the cretin and the defective as a result of cerebral hemorrhage during a prolonged and difficult labor, and therefore any principles or conclusions which are reached as regards the whole defective class without an adequate appreciation of the multitude of different conditions that are found under this generic caption must be avoided. It must be remembered, too, that with any defective patient it is possible to have associated a psychosis, so that the picture may be still more complicated. Therefore in other parts of this book various forms of what are grouped in this chapter are treated of from an etiological standpoint.

In considering the etiology of various defect states the important general principle that should be borne in mind is the location of the defect; first, as to whether it is in the germ plasm or acquired, and second, as to the time of development of the individual when it became operative. It is only those defects which are the result of a lack of some element in the germ plasm that are truly of an hereditary nature.

The process of development may be interrupted at any time from the beginning of the growth of the child in the uterus to the attainment of its complete adult development, and as this interruption may take place during intra-uterine life it is in such instances that it is important to make the distinction between a truly inherited and an acquired defect. In both instances the defect is congenital, that is, exists from birth.

Disease or injury may affect the child during intra-uterine life or during the process of birth or after birth and during the processes of extra-uterine development. Previous to birth, illness or injury to the mother are the common etiological factors; during birth asphyxiation and injury by the forceps are common causes, while after birth the infectious diseases and direct injuries enter largely into the etiology. Finally, there are relative conditions of defect which are due to lack of the opportunity to develop, such defect, for example, as results from the deprivation of the important sense organs, as the eyes and ears. If the patient be born blind and deaf, under ordinary circumstances he will be very defective mentally, because he has not the opportunities for learning which the ordinary child has. On a still different plane relative defect due to sordid and unsanitary conditions and lack of educational facilities is found. Children are ignorant who have no opportunity to go to school and learn, and may even lack the ordinary brightness that comes about spontaneously if they have lived under insanitary conditions that impaired their general health and energies, especially if these conditions be complicated by the presence in the child of some such debilitating factor as infected tonsils, adenoids, high degree of myopia, otitis media with deafness, and such other like things which impair the general health and vigor of the child and interfere with the patency of the avenues through which he gains his information of the outer world. This general enfeeblement of the child may also be the result of debilitating habits, such as masturbation and the use of narcotics and alcoholic drinks, the latter of which especially are an extreme expression of a vicious environment.

Classification.—The general group of defectives is a very wide one and includes not only the idiots and the imbeciles and the frankly feeble-minded, but the still higher grades of backward children, and then a whole borderland group of ne'er-do-wells in whom the defect is much less clearly defined than in the lower grades.

David Starr Jordan has said that "A good citizen is one who can take care of himself and has something left over for the common welfare." This saying represents in a general way the basis on which

until recently the classification of this group has been based. The defectives as a class may be said not only not to have anything left over for the common welfare, but only in the highest grades, and then under the most favorable circumstances, can they care for themselves. The general basis of classification, therefore, has been their educability and their capacity to care for themselves, according to some such scheme as follows, for example:

Feeble-mindedness.—A condition of mental defectiveness capable of much improvement by educational methods. The afflicted individual may ultimately take a place in the world and be self-supporting under favorable circumstances.

Imbecility.—A condition of mental deficiency which can, however, be materially improved by training, but not sufficiently for the subject to take a place in the world.

Moral Imbecility.—A condition of mental defectiveness which is shown predominantly in the absence of the highest functions, particularly the moral; capable of training to a considerable degree, but always a menace to society.

Idio-imbecility.—A condition midway between idiocy and imbecility.

Idiocy.—A condition of profound mental defectiveness. The lower grades are unteachable, while the higher may be trained slightly in self-help, *i. e.*, to attend to the calls of nature.

More recently an attempt has been made to define with greater accuracy the different grades of defect, and this effort has taken the form of an attempt to correlate the psychological development of the defective with the psychological development of the average child, so that the defective as a result of this correlation is said to correspond to the development of the average child at such and such an age. In other words, age has come to have a psychological rather than a chronological significance, and an individual who may be forty years old, but who is only developed to the extent that an average child is developed at the age of seven, is spoken of as having the psychological age of seven years.

Until the use of this scheme of classification all people were classified among other ways, in accordance with their chronological age. For example, in this country a person attains his majority and can cast his vote at the age of twenty-one. This refers of course entirely to the chronological age. It can easily be seen that if there are a material percentage of persons in the community who are defectives, although they may have acquired the chronological age of twenty-one they have not the mental development and the judgment that it is expected goes with such an age, and therefore from the psychological standpoint are not twenty-one and ought not on that basis be permitted to vote. The psychological classification of the age as outlined by the Binet-Simon test is a far more accurate way to standardize the mental development of the individual than the chronological age method, and although there are objections which may be urged against it and

although it is concededly not a perfect method and is at present undergoing gradual modifications, still it is so much better than the chronological method that it should be used in its place, and is by far the best standard which we have for designating the development of the defective. The classification, according to this method which has recently been adopted by the American Association for the Study of the Feeble-minded, is as follows:

Mental age.	Capabilities.	Class.	
Under one year	Helpless	Low	} Idiot.
1 year . . .	Feeds self. Eats everything	Middle	
2 years . . .	Eats discriminatingly	High	
3 years . . .	No work. Plays little	Low	} Imbecile.
4 years . . .	Tries to help		
5 years . . .	Only the simplest tasks	Middle	
6 years . . .	Tasks of short duration. Washes dishes		
7 years . . .	Little errands in house. Dusts	High	
8 years . . .	Errands. Light work. Makes beds	Low	
9 years . . .	Heavier work. Scrubs, mends, lays bricks, cares for room with simple furniture		} Moron.
10 years . . .	Good institution helpers. Routine work	Middle	
11 years . . .	Fairly complicated work with only occasional oversight		
12 years . . .	Uses machinery. Cares for animals. No supervision. Cannot plan	High	

The classification, according to the psychological age, while it is practically useful for defining the stage of development which the individual has reached, is also of some value in differentiating hereditary and acquired conditions. Somewhere from 65 to 80 per cent. of defectives have feeble-minded parents, and therefore in most of them the condition is hereditary. In these hereditary cases it would seem as if the child developed quite normally up to a certain point and then stopped rather suddenly, and that this stopping was a pretty general one, so that the development ceases at a certain level without many irregularities. A patient, for example, will test to seven years of age, will do practically all of the tests for seven years, and fail completely with all of the tests above that. In other words, the age is very accurately seven. The defect is a clean-cut one. When this condition of affairs is found there are reasons to suppose that the trouble lies in the germ plasm.

In acquired conditions the stoppage is not so abrupt as a rule. For example, with the deterioration that goes along with epilepsy in childhood, the tests show much greater irregularity, a much more uneven development, and this might be expected of any condition in which deterioration was going on. In numerous deteriorating conditions such results are found and therefore it can be seen that one is dealing not only with a stoppage of development, but with a disease process which, while it is in operation, is producing symptoms of its own.

The most useful classification of the feeble-minded is a clinical one.

It would be quite as absurd to enter into a discussion of the general psychology or the general pathology of defectives as it would be of the so-called insane, and therefore in this chapter the different well-defined forms will be briefly and separately described.

Clinical Varieties.—**Amaurotic Family Types** (Tay-Sachs Disease).—This is a disease which generally affects more than one child in the family and appears to be confined in its incidence to the Jewish race. Its etiology is unknown. Its pathology is in general a degeneration of certain elements of the brain, more particularly the cortical elements and pyramidal tracts, including particularly the optic nerves. It has been described in three stages:



FIG. 317.—Adenoma sebaceum. The skin affection associated with tuberous sclerosis of the brain, a profound degree of mental defect and various tumors of the viscera, especially the kidneys. (Courtesy of Dr. Martin W. Barr.)

First Stage.—The infant is usually all right at birth and the disease does not make itself manifest for some few months thereafter, usually at about the fourth month. At this time the first symptoms observed are some weakness in the neck muscles and indications of dimness of vision. If the fundus is examined during the fourth or fifth month there will be found a whitish-gray symmetrical patch, oval in shape, with a horizontal axis occupying the macula lutea. In the center of this patch is seen the fovea centralis which appears as a dark cherry-red spot. Optic atrophy follows, and later total amaurosis. *Second stage:* In this stage the weakness of the neck muscles is more marked and the head falls backward if unsupported, and while lying on the back the infant is unable to turn over to either side. The hand

grasp is noted to be feeble, objects are dropped and the infant is generally apathetic. The vision is materially reduced in this stage, but the senses of taste and hearing are preserved, the sense of hearing appearing to be unusually acute. *Third stage:* In this stage the affected muscles are atrophied, and later the atrophy extends to all of the muscles of the body, emaciation becomes marked, the reflexes exaggerated, and late in the course of the disease the extremities become rigid, and there is retraction of the head.

Spasmodic contractions and convulsions have been noted. There is at no time any rise in temperature, and the thoracic and abdominal viscera remain normal. Death usually occurs in less than two years.

Sclerotic Types.—Certain types of mental defect are seen associated with a condition of the brain which in general may be said to be due to an overproduction of neuroglial tissue and corresponding atrophy and disorientation of nerve elements. The exact nature of this process is not definitely known. It is not improbable that there are a number of different conditions comprised in this general picture.

The sclerosis may be diffused pretty generally or it may be localized in patches and the affected portions may be atrophic or they may be hypertrophic. When the process is hypertrophic and involves a large portion of the brain one finds what has been called a hypertrophied brain. The brain is much larger and heavier than the normal, and is much firmer in consistence after being hardened. It does not look like a normal brain, but the surface has a cauliflower appearance. The condition is usually associated with grave degrees of mental defect and with epilepsy. The localized varieties are more apt to be associated with convulsions than the diffuse, while there may be also noted marked tremors.

This disease has been recently correlated with other changes than the local cerebral changes. Kufs¹ in a valuable extensive article, including autopsy material, has accumulated the evidence for a distinct disease entity which comprises various manifestations. In the first place, besides these changes in the brain, which have briefly been referred to, there is a distinct cutaneous affection of the form of adenoma sebaceum, which affects more particularly the face and the back. Along with this condition is frequently found associated mixed tumors of the kidney—tumors made up of various elements of which the smooth unstriped muscle tissue is the most prominent.

These three symptoms, then, adenoma sebaceum, mixed tumor of the kidney, tuberous sclerosis of the brain, are the triad which constitute the most important evidences of this disease entity. In addition to this pathology it is to be noted first, about the sclerosis that it involves sometimes the cerebellum, that tumors of the ventricles

¹ Beiträge zur Diagnostik und pathologischen Anatomie der tuberösen Hirnsklerose und der mit ihr Kombinierten Nierenmischstumoren und Hautaffektionen und über den Befund einer akzessorischen Nebenniere in einen Ovarium bei derselben. Zeitschrift für die gesamte Neurologie und Psychiatrie, Band. xviii, Heft 3.

are quite characteristic, and that relatively commonly there is associated with this triad of symptoms rhabdomyoma of the heart. Myoma of other organs, such as the stomach and the uterus have also been observed.

While on the mental side, along with this condition there usually is associated a marked degree of mental defect with epilepsy, still Kirpiczniks has reported a case occurring in a man twenty-eight years old without mental impairment.

Cretinism.—This disease is endemic in certain parts of Europe, but so far as we come in contact with it in this country is sporadic.

The disease is due to a defect in the secretion of the thyroid gland. All degrees of defect may be present, from athyroidism through the various degrees of hypothyroidism. The several degrees of cretinism which are described and which correspond with different degrees of defect are three, namely, the lowest grade, in which the defect is greatest, the cretins, the middle grades, the semicretins, and the higher grades, the cretinoids.

The disease usually begins quite early in the life of the child, sometimes during the first year, although it may be delayed for several years. The general symptoms are those, *first*, of retarded development; the child appears less bright than he should be, walking is learned slowly, speech is delayed in development, the anterior fontanelle is late in closing; and *second*, the characteristic cretinous appearance. The body is dwarfed, the head relatively large, and the legs short and bowed, hands and feet stumpy, and the ossification of the bones is delayed. The appearance of the face is typical, the nose is broad and flattened, the lips are thick, the tongue thick and often protruding from the partly opened mouth, the eyes widely separated, the eyelids often heavy and swollen, and the hair coarse and scanty. The skin is cachectic in appearance and dry and thick, resembling the skin of the myxedematous patient, the neck is short, the abdomen protuberant, with sometimes umbilical hernia. The signs of puberty are late in making their appearance, there is often a failure in the complete development of the genital organs, and many of these patients are sterile. The pulse and respiration are slow, the temperature may be subnormal, and the movements of the patient are usually very deliberate. Mentally the cases show various grades of defect, from the lowest grades of idiocy through the various degrees of imbecility. In general cretins are quite good-natured, pliable individuals who are easy to get along with and care for.

Diagnosis.—In the matter of diagnosis the principal conditions which have to be differentiated are rickets, achondroplasia, and mongolism.

Rickets can usually be differentiated by the characteristic beading of the ribs, the symmetrical enlargement of the epiphyses, and the absence of the typical signs of cretinism.

Achondroplasia should not be mistaken for cretinism. There is an

imperfect development of the long bones, but none of the signs of cretinism, particularly none of the impairment of mental development.

Mongolism is at times extremely difficult to differentiate. It is very important that this condition be not mistaken for cretinism, particularly with reference to the matter of treatment, as treatment is capable of modifying the cretin, but not the mongol. The following table of differential signs taken from Shuttleworth and Potts,¹ will serve to point out the detailed characteristics between these two conditions:

MONGOLISM.

1. Characteristics noticeable from birth.
2. Skull brachycephalic: contour rounded or short oval; longitudinal and transverse diameters nearly correspond.
3. Forehead usually smooth.
4. Palpebral fissures "almond-shaped," and more or less oblique upward and outward. Frequent epicanthus. Strabismus common. Ciliary blepharitis frequent.
5. Cheeks chubby, often florid. Complexion mottled.
6. Lips often transversely fissured. Lower lip may be pursed up over upper.
7. Tongue large and coarsely papillated if not fissured. Tongue frequently protruded and drawn back.
8. Skin smooth in infancy, but furfureaceous later; not redundant or "baggy."
9. Hair "wiry," often "mouse color," but sometimes blonde. Downy growth common on forehead and cheeks.
10. Thyroid gland palpable to greater or less extent.
11. No fatty tumors (pseudolipomata) in posterior triangle of neck.
12. Long bones somewhat shorter than usual, but slender.
13. Hands broad; thumb and little finger short, the latter often curved toward ring finger. Finger tapers at ends.
14. Feet large and flat. Fissure between great and next toe often seen.
15. Abdomen often distended; occasional umbilical hernia; often inguinal hernia.
16. Expression more or less vivacious and mobile, observant and imitative.

CRETINISM.

1. Characteristics often not noticeable till sixth or seventh month.
2. Skull dolichocephalic: flat at top (fontanelles close late), expanded laterally; broad behind, often asymmetrical.
3. Forehead usually wrinkled.
4. Palpebral fissures horizontal, but appear small, owing to pseudo-edema of eyelids. Strabismus and ciliary blepharitis less common.
5. Often circumscribed malar flush; complexion ashy or waxy.
6. Lower lip often everted. Mouth open. Drivelling common.
7. Tongue large, but not coarsely papillated or fissured. Tip of tongue thickened, and constantly protruding.
8. Skin dry and scaly; forms folds here and there, being redundant and "baggy."
9. Hair harsh, coarse, and scanty. Usually of darkish tint (Bourneville says brown); scalp often eczematous.
10. Thyroid gland impalpable to most thorough examination.
11. Fatty tumors (pseudolipomata) frequently found in posterior triangle of neck, etc.
12. Long bones shortened and thickened, in some cases bowed.
13. Hands, broad thick, and stumpy, with wrinkled skin. Fingers square at tips.
14. Feet squat; skin redundant about ankles and dorsum of foot.
15. Abdomen very bulky and prominent with folds of skin; umbilical hernia common.
16. Expression dull and immobile; unobservant and apathetic.

Deficient stature, flattened bridge of nose, with expanded *alæ*, late and irregular dentition, deferred closure of fontanelles and retarded puberty are similar in each variety.

Treatment.—This has been taken up in Chapter IV on Diseases of the Internal Secretions, or Endocrinopathies.

¹ Mentally Deficient Children, London, 1910.

Mongolism.—The Mongolian or Kalmuc type of defectiveness is so-called because of the resemblance of the patient to the Mongolian race, particularly the slant of the eyes and the general facial expression.

The present belief with reference to this condition is that it is congenital and not hereditary in the true sense, that is, not dependent upon a condition of the germ plasm. The condition is believed to be the result of some abnormal state of the mother which may be produced by injury or shock, but which in general is believed to be the result of a



FIG. 318.—Mongolian type, aged eighteen years, apathetic idiot, semimute, speaks only a few words, enunciation imperfect. Learned to feed himself and is cleanly. Dwarfish, brachycephalic, forehead flat and wrinkled transversely. Eyes oblique, photophobia, chronic conjunctivitis. Tongue very large, filling mouth completely; deeply fissured, papillæ enlarged. Hands broad. Fingers short and thick. (Courtesy of Dr. Martin W. Barr.)

worn-out reproductive capacity, the mother being unable to bring the child to complete development in the uterus. Corresponding with this assumption the Mongol is generally the last child born in the family, and not infrequently comes from good stock.

There are three prominent physical signs of this condition which are sufficient when present to make a diagnosis. They are the form of the skull and the peculiarities of the palpebral fissures and of the tongue. The skull is brachycephalic, diminished in its antero-posterior diameter, flattened on the face and occiput, but without

recession of the frontal and supraoccipital regions, as in the microcephalic. The tongue is large, the circumvallate papillæ are hypertrophied, and there are marked irregular transverse fissures. This condition of the tongue is characteristic for this type, and is not found in any other varieties. Thompson has suggested that the fissuration is dependent upon two factors: an extreme vulnerability of the mucous membrane, and the habit of sucking the tongue, commonly present in these children. The hands and feet are broad, clumsy and spatulate, while an incurving of the little finger has also been described as of frequent occurrence in these children. In addition to these quite characteristic symptoms the children are clumsy, joints loose, the skin rough, the abdomen protuberant and there is a tendency to chronic inflammatory conditions of the mucous surfaces. The circulation is generally poor, congenital cardiac anomalies may be present, such as imperfect closure of the foramen ovale, vital resistance is very low, and these patients are quite apt to die relatively early in life from tuberculosis.

Mentally these children are usually at a very low grade of development, generally gravitating about four years of age. They may be less or they may possibly reach the seven-year limit, but rarely go beyond it. As a rule they are good-natured and easy to care for by those who are understanding and sympathetic.

A special form of cortical aplasia has been described as belonging to this condition.

Thyroid does no good in these conditions. It is therefore necessary to carefully separate them diagnostically from cretinism, a condition which resembles it very closely on casual observation. For differential diagnosis see cretinism.

Hydrocephalic Types.—Various degrees of mental defect may be associated with hydrocephalus. Hydrocephalus may of course occur previous to birth, but is rarely congenital, as a child with any marked degree of hydrocephalus could not be born alive. The condition may be relatively acute, in which case it leads rapidly to death, but it may come to an arrest or be extremely slow in progress. In this latter group of cases we find patients sometimes who live to a fairly advanced age, although as a rule this disease terminates life before the patient has passed middle life and generally much younger. The symptoms in the slowly progressive cases are the symptoms of gradual obliteration of the mental faculties, and are undoubtedly dependent upon pressure effects. These symptoms are in general loss of intelligence, gradual loss of vision, hearing, the function of language, and gradually the sinking into a semistuporous condition, and death. The causes of hydrocephalus are probably numerous, syphilis, tuberculosis, brain tumor, and meningitis are among them.

Microcephalic Types.—Mental defect associated with extreme smallness of the head. No definite rule can be laid down as to what size of head shall be considered microcephalic. Ireland gives the

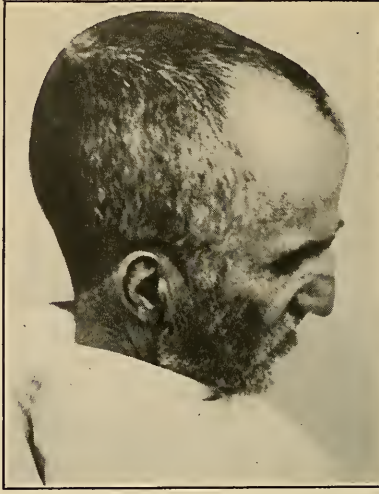


FIG. 319.—Hydrocephalus, anteroposterior diameter $9\frac{1}{4}$ inches; transverse diameter $7\frac{3}{8}$ inches. This man for many years made a living as a bell-ringer. Previous to death he became profoundly demented and his head so heavy that he could not lift it from the pillow. Autopsy showed the brain to be literally little else than a bag of fluid.



FIG. 320.—On left, high-grade imbecile, aged thirteen years; mentally, six years. On right, microcephalic imbecile, aged nine years; mentally, three years.

general rule that heads below seventeen inches in circumference (431 millimeters) may be so considered. This rule is not absolute,



FIG. 321.—Erma. Front view.



FIG. 322.—Erma. Side view.



FIG. 323.—Mary. Front view.



FIG. 324.—Mary. Side view.

Microcephalic twins, aged eight years. (Courtesy of Dr. A. C. Rogers, Faribault, Minnesota.)

and when it is considered that these small-headed varieties may probably be the result of various etiological factors it will be appreciated that the term microcephalic had better be used purely as a descriptive term rather than as a term to apply to a definite class of defectives even though that class be considered solely from a morphological standpoint.

It is probable that two sets of causes may produce the extremely small skulls which are found in the microcephalic types. The old theory that the condition was due to premature synostosis has long since been discarded as has also the operation of craniectomy based upon that theory.

The characteristic condition of the microcephalic brain is its extreme smallness, more particularly pronounced in the temporosphenoidal, parietal and occipital regions. The posterior lobes of the cerebrum do not cover the cerebellum. The convolutions of the cerebrum are more simple in pattern than in the normal brain, and in addition there



FIG. 325.—Paralytic type. Athetoid movements of hands and arms.

may be localized agenesis with resulting microgyria. There may also be associated morbid processes such as encephalitis. The hypoplasia usually also involves the spinal cord.

The general appearance of these patients is quite characteristic. The conformation of the skull is "sugar loaf," or as it is technically termed "oxycephalic." This is characterized by a rapidly receding forehead with a flat occiput. Along with the receding forehead there is usually also a receding chin which gives a pointed aspect to the face, which associated with a small stature gives a quite characteristic general appearance to these patients and has led to their being characterized as "bird-like" in appearance.

The mental development of these patients varies between wide limits, although the tendency is for them to belong to the lower grades of defect. They are usually well disposed, good-natured, affectionate, and not difficult to care for. They generally do not live to advanced years, but die at rather an early age.

Paralytic Types.—There are a large variety of cases in this group. The paralyzes may involve any portion of the body or be of almost any extent. Monoplegias and diplegias are common, but localized palsies and hemiplegias are also not infrequent. They depend either upon lack of development of certain portions of the brain or more frequently upon injuries and sometimes new growths. Hemorrhage is the most frequent injury that produces the various palsies. This may occur as the result of prolonged labor or injury by forceps delivery, or may be the result of injury during the early months of life. The amount and the character of the mental defect varies within wide limits and no description which would apply to this whole class would be possible, principally because the class is not a homogeneous one.

Porencephalus.—Within this group of paralytic types one of the conditions which is found and which has been frequently described is porencephalus. This condition is consequent upon gross cerebral lesions such as a lack of cerebral substance resulting in a cyst connected with the ventricle—*true porencephalus*, or due to cysts not connecting with the ventricles and resulting from softening, hemorrhage, or inflammation—*false porencephalus*.

Traumatic Types.—Arrest of mental development may occur as the result of an injury to the brain during the developmental period. The most frequent type of injury producing this result is prolonged labor with instrumental delivery. Where the injury produces a lesion in the motor pathway with a resulting paralysis the patient is generally included within the paralytic group.

Epileptic Types.—Epilepsy occurring early in life is one of the causes of lack of mental development. Just how this result is brought about by the epilepsy is not altogether clear. In part it is due to the fact that the child has to be differently treated from normal children, is often deprived of the same educational advantages because of its disease, but apart from this there seems to be a direct relation between the lack of development and mental deterioration, and the epilepsy itself. This is probably dependent upon the fact that the epilepsy is a disease which strikes very deep, close to the foundation of the

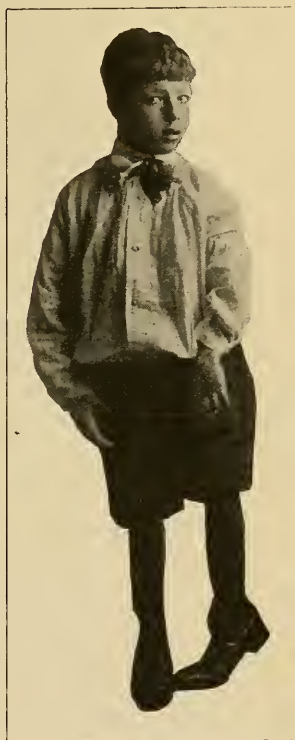


FIG. 326.—Hemiplegic, aged eleven years. (Courtesy of Dr. A. C. Rogers, Faribault, Minnesota.)

neurological mechanisms. Its manifestations are evidently in part, at least, dependent upon disturbances at the physico-chemical level. Disturbances which are as fundamental as this necessarily are difficult to deal with and also necessarily impair the superposed levels. (See Chapter on Epilepsy.)

Epileptic attacks are found in many of the other forms of defect, more particularly in those defects associated with gross cerebral lesions such as are found in the paralytic and traumatic types. It has generally been assumed that the localized lesion was the cause of the epileptic manifestations. While this may be so in certain cases, still in a general way it must be borne in mind that epilepsy is presumably



FIG. 327.—Sensorial type, imbecile by deprivation, middle grade. Male, aged about thirty years. deaf-mute. Taught himself to talk and to read and write in a fashion. Often cruel to children but devoted to animals. Appropriates things not his own. Very keen and observant. (Courtesy of Dr. Martin W. Barr.)

an hereditary disease dependent upon a defect in the germ plasm and that perhaps only those children develop convulsions as a result of injury or localized cerebral lesions in whom hereditary conditions are favorable for the outcrop of epilepsy. At least the hereditary factor should not be neglected in the study of the patient simply because a localized lesion has been found. Perhaps the localized lesion could not have produced such a result without the hereditary factor—the spasmophilic tendency.

The epileptic type of defective, besides the symptoms of his defect, characteristically manifests the symptoms of the epileptic character and is therefore quite a difficult problem with which to deal.

Inflammatory Types.—This group includes those conditions resulting from inflammation of the meninges and of the brain, found most frequently as a result of acute infectious fevers such as pneumonia, typhoid, and the exanthemata. Local areas of meningitis or meningo-encephalitis are not infrequent in connection with the specific fevers, and when extensive or severe and occurring early in life produce an arrest of development, to a certain degree, of the mental faculties together not infrequently with epilepsy. Strümpell's polioencephalitis superior belongs in this group.

Sensorial Types.—The defect in this group is the result of deprivation. Here there is no defect of the germ plasm or no defect in the structure of the brain, but owing to injury or disease which has destroyed



FIG. 328.—Sensorial type, imbecile by deprivation, middle grade. Boy, aged ten years, deaf-mute, no ears. (Courtesy of Dr. Martin W. Barr.)

the patient's vision or hearing or both the child is cut off from communication with the outside world to such an extent that mental development is impaired thereby. Theoretically these patients are of course educable, but it is only very rarely that one is found with the capacity, the patience, and the ingenuity to develop such a child by educational methods. The cases of Helen Keller and Laura Bridgman stand out as illustrative of what can be done.

Syphilitic Types.—Syphilitic types of defect have always been known in that group of cases presenting distinct evidences of hereditary syphilis, particularly the Hutchinson teeth, linear scars about the mouth and nose, and keratitis. Until, however, the discovery of the *Treponema pallidum* and the elaboration of the Wassermann reaction there was no comprehension of the frequency with which syphilis

entered into the etiology of the various types of mental defect. One of the best-known authors, who just antedated this period, says that the number of cases of defectives due to syphilis is quite insignificant, probably not more than one or two per cent., and comments upon this in the face of the frequency of syphilitic disease in the fully developed brain and the frequency of so-called inherited syphilis.

There is one distinct type of disease which only recently has come to be recognized with any degree of accuracy which used to be classed among the defective states and which is due to syphilis, namely, juvenile general paresis. It is probable that still in many places this disease is not recognized, but is put down probably as some form of progressive defect. The number of juvenile paretics is not very large.

The application of the Wassermann test to the defectives as a class shows that not far from 20 per cent. of the patients taken indiscriminately show a positive Wassermann reaction. Of course it is quite another question as to just what the relationship is in these patients between the syphilis and the mental defect. In one case it may be that the syphilis has produced vascular disease and the defect is due to a vascular lesion. In another case it is the typical effect of a general paresis, or perhaps a meningo-encephalitis, and still further probably syphilis has a decidedly deleterious effect upon development in ways that we do not at present understand, so that it may well be that certain of the types of defect which do not present characteristic syphilitic lesions may be due to subtle nutritional changes, which, in the last analysis, have their origin in syphilis. The great part that this disease plays in this class of cases is yearly becoming more and more evident.

Idiot-savants.—These are rare cases, who, although often deeply defective, still have some special ability wonderfully developed. It may be music, calculation, or memory for some special class of facts, etc.

The calculators can name the answer to mathematical problems almost instantly; the musical prodigies often play well and may even improvise; one patient under the observation of one of the authors would instantly name the day of the week for any date for years back. Many of these patients have a capacity for mimicry and buffoonery, and from this class undoubtedly were recruited in the old days many of the court fools.

The psychology of these individuals is not understood and they themselves are quite unable to give any explanation of their special abilities. Their abilities, however, are really not so great as they appear. They appear exaggerated because they stand out upon a background of pronounced defect, also because they are unusual in the sense of not being the common possession of mankind. The calculators, for example, do very wonderful calculations in the way of adding up long series of figures very rapidly. There are, however, well-known devices for increasing the rapidity of the ordinary old-fashioned method of adding, and many of our experts at figures today

could compete with some degree of success with these calculators. The patient, for example, who could name the day of the week for years back upon merely being given the date, and do it instantly, spent most of his time in studying calendars. It would not be surprising if almost anyone could accomplish such a feat if he spent any such commensurate degree of effort upon it.

Other descriptive terms are used to describe certain types of defectives, for instance in addition to the so-called Mongol or Kalmuc types there are described American Indian and Negroid types because of the obvious resemblances. Then again, for purposes of practical classification the lower grade of defectives are spoken of as either *apathetic* or *excitable*, and certain of these latter who keep up certain definite and characteristic movements almost continuously are described as



FIG. 329. — American Indian type. (Courtesy of Dr. Martin W. Barr.)



FIG. 330.—Negroid type. (Courtesy of Dr. Martin W. Barr.)

rhythmic idiots. Other motor disturbances of course also occur. Aside from paralysis and epilepsy, probably one of the most common is athetosis.

Mild Grades of Defect.—A systematic examination of large numbers of children has disclosed the fact that a great number of the disorders of conduct and types of inefficiency which are manifested among them are dependent upon some degree of feeble-mindedness. Systematic examinations of school children, for example, have resulted in showing a not inconsiderable percentage of the general school population so far behind in their mental development as only to be described by a term indicating an inherent defect. These defects range all the way from well-marked imbecility among the younger children up through the grades of the so-called backward children. For the most part, the

different grades of the moron, of the feeble-minded, and the lighter measuring rod which has been used for determining these conditions has been the Binet-Simon scale of intelligence tests, and the treatment which has been applied has been the segregation of these defective children from the general school population into classes and sometimes whole schools devoted particularly to them, thereby gaining both the advantage of the application of special educational efforts to these children and the relief of the normal child from the drag back to which he was subjected by having the defective in the same class with him.

Many of the mental defects, it must be borne in mind, are only relative affairs and are dependent upon general conditions of ill health,



FIG. 331.—In center a moron, aged twenty-four years; mentally, ten years. At right, moron, aged eleven years; mentally, eight years. At left, imbecile, aged nine years; mentally, six years.

such, for example, as are due to adenoid vegetations in the posterior pharynx. Under such conditions of ill health development is impaired and does not proceed at a normal rate. With anemia, impaired digestion, and infected tonsils, which produce a constant toxemia, the child cannot be expected to proceed in his development with normal rapidity. In addition to such conditions as this it is found that the defect is often due to high grades of myopia which make it impossible for the child to learn, because he cannot see to read or even see the blackboard. In the same way deafness and other quite gross lesions have been found to account for many of these conditions. It should be remembered, too, that the child may have a neurosis or a psychoneurosis,

and that these conditions may produce conduct disorders which show a moral obliquity which, if they are not carefully investigated, may too easily be classified as due to defective conditions. Such psychic states are susceptible of psycho-analysis, and often of cure.

Psychopathic Constitution.—There are many anomalies of character which, because normal or usual to the individual, cannot be said to properly constitute a psychosis, but because they lead to a rather inefficient type of adjustment of the individual to his environment, and because persons exhibiting these peculiarities often become actively disordered, may be considered as borderland conditions.

Something of the *hysterie* and *epileptic* characters has already been discussed. The general type of inefficiency of the *neurasthenic* and the *psychasthenic* character has been described, and attention has been called to the *unresistive* and the *post-traumatic* types with their intolerance of alcohol and fever. There also may be said to exist a *manic-depressive* type and lately attention has been directed to the “*shut-in*” type as the type par excellence found in those cases which develop dementia precox.

Besides these there are the “*cranks*,” who with some pet scheme, closely approach the paranoiac type and that host of *ill-balanced*, *eccentric* individuals who may be superficially brilliant but lack continuity of purpose and capacity for the continuous expenditure of effort in any one direction. Their life, to use the well-chosen words of Régis, is one “long contradiction between the apparent wealth of means and poverty of results.”

There are also the *pathological liar*, or *pseudologia phantastica*, and certain types of *swindlers*.

Constitutional anomalies of mood are seen, those who are always depressed—*psychopathic depression*—and the opposite state—*psychopathic exaltation*. Others never seem to be quite able to successfully cope with conditions; they are the failures of life, the cases of *constitutional inferiority*.

Conditions of *psychogenic depression* occur quite often in psychopathic individuals. Many weak characters who are led into crime develop a symptomatic depression when caught and sentenced. More pronounced defects of character are seen in the *criminal* classes, many of whom lack the ordinary moral inhibitions and are properly *moral imbeciles*.

There are many psychogenic states that occur in psychopathic individuals—*deviates*. The so-called *prison psychoses* are types and come about as the patient's reaction to the difficulties in which he finds himself. They may be hysterical, catatonic, paranoid, according to the type of individual. They clear up when the stress is removed—pardon, expiration of sentence, commutation of sentence, etc.

ANOMALIES OF THE SEXUAL INSTINCT.

Quantitative Anomalies.—These are *frigidity* or lack of desire for sexual congress—*sexual anesthesia*—or *eroticism*—*sexual hyperesthesia*.

Qualitative Anomalies.—These are *inversions* and *perversions*. Inversion consists of a lack of harmony between the physical and the psychical sex and leads to *homosexuality* or desire for persons of the same sex. Various physical anomalies are often found in these persons. For example, the general conformation of the body, pilosity, etc., may indicate one sex, while the genitalia are of the other.

Sex inheritance is *alternative*. That is, both male and female characters are present in the germ and only one normally develops. Sometimes there seems to be an uncertainty as to which will develop and the result is a certain mixture which may take place either in the bodily or psychic sphere alone or in both.

The perversions are many and include the various anomalous means of gratifying the sexual appetite.

With respect both to inversion and perversion it must be remembered that in the young child the sexual instinct has not developed and later as it develops and comes into prominence it differentiates and tends to specialize by centering its aims in a special direction, *i. e.*, toward the opposite sex and normal coitus. The child, before this takes place is, to use a term of Freud's, *polymorphous-perverse*. He may be developed in any direction by appropriate influences or he may stay in the undeveloped, infantile stage.

The most important of the perversions are:

Masturbation.—Masturbation is very frequent among psychopaths and very often a result rather than a cause of mental anomalies, though undoubtedly an important factor in some cases of acute psychosis. A transient period of onanism in infancy is probably normal and serves to focalize the sexual sensations on the normal erogenous zones.

Active Algolagnia (*Sadism*).—The gratification of the sexual feeling by the infliction or sight of pain—real or simulated. In the latter case the sadism is *symbolic*. As the male is normally the more active and aggressive in the sexual relation, as might be expected, this anomaly is more frequently found in men.

Passive Algolagnia (*Masochism*).—The gratification of the sexual feeling by suffering pain—real or simulated. In the latter case it is *symbolic*. The female, being the more passive of the two sexes in the sexual relation, so an exaggeration of this passivity is more frequently found among women.

Homosexuality.—Sexual desire for the same sex.

Narcissism.—A form of auto-erotic sexuality in which a person is in love with himself—his own body.

Fetichism.—Sexual excitement and gratification by the sight, contact or possession of some object or part of the body. The object

is usually some wearing apparel, such as shoes, handkerchief, petticoat, or a part of the body other than the sexual organs.

Bestiality.—Sexual relation with animals.

Exhibitionism.—Sexual gratification by exposing the genital organs.

Necrophilia.—The desire to have sexual congress with a dead body.

Most of these conditions stand for what was normal at a certain stage in development but should have been left behind in the progress; or else they are the result of aberrant development from these lower points when there has been a stagnation of the developmental process and so are included in this chapter. The higher psychic ramifications are fully discussed in the chapters on the neuroses, psychoneuroses, epilepsy, and certain psychoses, notably dementia precox and manic-depressive psychosis, in all of which disturbances of psychosexual development are present.

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