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Original Articles.

NERVOUS SYSTEM OF THE ECHINODERMATA.

BY GEORGE J. ROMANES, F.R.S.

THE Editors of 'BRAIN' having requested me to supply an abstract of the research which Professor Ewart and myself are at present conducting on the locomotor system of the echinodermata, I think that their wishes will best be met by restricting the abstract to those features of the research which have special reference to the morphology and physiology of the nervous system.

Morphologically, then, the nervous system of the echinodermata is constructed on a simple plan. Taking the common star-fish as a type, it is generally known that the nervous system consists of a pentagonal ring of ganglia and fibres surrounding the mouth, and sending off a trunk into each ray, which supplies numberless branches to the muscles and ambulacral feet, or pedicels. Choosing the echinus, or sea-urchin, for a more detailed account of the character and distribution of the nervous system, we find that it is composed of two parts —one internal and the other external to the shell. The internal part consists of fine radial trunks, which may be traced from the ocular plates along the ambulacral areas external to the radial canals to the oral floor, where they bifurcate and unite with each other, so as to form a pentagonal nerve-ring. This ring lies between the œsophagus and the

tips of the teeth which project from the lantern. Small branches leave the ring and supply the œsophagus, and lateral branches arise from the several trunks to escape with the pedicels through the apertures of the pore plates. Each trunk lies in a sinus situated between the lining membrane of the shell and the ambulacral radial canal; the lateral branches which accompany the first series of pedicels through the oral floor are large and deeply pigmented; the branches within the auricles are small; those external to the auricles gradually increase in size until the equator is reached, and from the equator to the ocular plates they diminish. At the equator the trunk is wider than at either pole, and it is often partially divided for some distance at each side of the equator by a deep longitudinal fissure. When the nerve trunk, after being stained with chloride of gold or with osmic acid, is removed from its sinus, it is seen to be enveloped by a thin fibrous sheath. This sheath contains numerous large pigment cells, and has scattered over it irregular masses of protoplasm which have been deposited from the fluid of the neural sinus.

When the sheath is removed the trunk is seen to consist of delicate fibres and of fusiform cells; the cells consist of a nucleus and a thin layer of protoplasm which projects at each end and terminates in a nerve-fibre.

The lateral branches of the trunk escape along with, and are partly distributed to, the pedicels; the remainder breaks up into delicate filaments which radiate from the base of the pedicel under the surface epithelium. When one of the large branches already referred to as escaping with the inner row of pedicels is traced through the oral floor after sending a branch to the foot, it breaks up into delicate fibres, some of which run towards the bases of the adjacent spines and pedicellariæ, while others run inwards a short distance towards the oral aperture.

Either in connection with, or anatomically independent of these filaments from the lateral branches of the nerve trunks, there is an external plexus lying almost immediately under the surface epithelium and extending from the shell to the spines and pedicellariæ. The fibres of this plexus closely resemble those of the lateral branches of the trunk; but generally they

are smaller in size and have a distinct connexion with nerve cells. The cells consist of an oval nucleus and of a layer of protoplasm, which is generally seen to project in two, or sometimes in three, directions—the several processes often uniting with similar processes from adjacent cells so as to form a fibro-cellular chain or network.

In preparations from portions of Echini treated with both chloride of gold and osmic acid, we have succeeded in tracing the plexus over the surface of the shell between the spines and pedicellariæ; and from the surface of the shell to the capsular muscles at the bases of the spines. Further, we repeatedly observed delicate fibres passing beyond the muscles, apparently to end under the epithelium over the surface of the spines.

In the case of the pedicellariæ, the plexus on reaching the stem runs along between the calcareous axis and the surface epithelium, to reach and extend over and between the muscular and connective tissue-fibres between the calcareous axis and the bases of the mandibles. The plexus, now in the form of exceedingly delicate fibres connecting small bipolar cells, reaches the special muscles of the mandibles. In several preparations, delicate fibres appeared to extend towards the sensitive epithelial pad situated on the inner surface of each mandible, a short distance from the apex. Although this plexus is especially related to the muscular fibres—lying over and dipping in between them—it is also related to the surface epithelium, and delicate fibres often extend from it to end under or between the epithelial cells.

Before proceeding to give an account of the physiology of the nervous system, it is desirable to say a few words on the natural movements of star-fishes and sea-urchins. This is desirable, not only because it is necessary to study the natural movements before we can be in a position to appreciate the results of the following experiments, but also because these natural movements form in themselves a study of considerable interest.

Taking the common star-fish (*Uraster rubens*) as our starting-point, it is needless to dwell upon the well-known mechanism of the ambulacral system. The rate of crawling upon a flat horizontal surface is 2 inches per minute. The animal usually

crawls in a determinate direction, and, while crawling, the ambulacral feet at the end of each ray are protruded forwards as feelers; this is particularly the case with the terminal feet on the ray, or rays, facing the direction of advance. When in the course of their advance these tentacular feet happen to come into contact with a solid body, the animal may either continue its direction of advance unchanged, or may deflect that direction towards the solid body. Thus, for instance, if, while the star-fish is advancing along the floor of a tank, the tentacular feet at the end of one of its rays happen to touch a perpendicular side of the tank, the animal may either at once proceed to ascend this perpendicular side, or it may continue to progress along the floor—feeling the perpendicular side with the ends of its rays perhaps the whole way round the tank, and yet not choosing,[1] as it were, to ascend. What it is that determines the animal in some cases to ascend, and in other cases not, we were unable to ascertain.

When a star-fish ascends the perpendicular side of a tank or bell-jar till it reaches the surface of the water, it very frequently performs a number of peculiar movements, which we may call acrobatic. On reaching the surface of the water, the

[1] It may be as well to explain that in using such words as these, I do not, in the present paper, attach to them any psychological signification; they are used as merely metaphorical terms which serve most briefly, and therefore most conveniently, to express the resultants of those systems of physiological stimuli, the composing members of which we were not able to observe. When one star-fish appears to choose to ascend the side of a tank, while another star-fish, under apparently precisely similar circumstances as to stimulation, seems to prefer walking along the floor, we can only suppose that the circumstances of stimulation, although apparently similar, are not really so, and therefore that the difference in the result is due to some difference in the stimulation. Of course it may be objected to this that the same remark applies to cases in which the psychological element unquestionably enters—choice on its physiological side being merely the resultant of some unobservable system of stimuli. But without here entering on the whole question of the relation between body and mind, it is enough to point out that the only evidence we can have of a physiological determination presenting a psychological side, is by observing that the organism which exhibits the determination is capable of altering it on future occasions, if the determination first made is found by individual experience to be injurious. In other words, the power of learning by individual experience is the only unequivocal evidence we can possess of the presence, in any animal, of a psychological element; and as we have observed no such evidence in the case of any of the echinoderms, we desire it to be understood that we consider all their movements to be of the so-called "reflex" kind.

animal does not wish to leave its native element, and neither does it wish again to descend into the levels from which it has just ascended. It therefore begins to crawl to one side or the other, and while crawling it every now and then throws back its uppermost ray, or rays, to feel for any solid support that may happen to be within reach. The distance to which the rays may thus be thrown back is remarkable; for the animal may hold on with its two lower rays alone, or even with the end of a single ray, and throw back the whole of the other rays with the central disc into a horizontal position—the ambulacral surface of the rays which are thrown back being then of course turned up, so as to face the surface of the water. If the rays succeed in finding a solid body, they will perhaps —though not necessarily—fasten upon it, and when their hold is secure, the rays which hitherto held the animal to the side of the tank relax their suckers, so that the star-fish swings from its old to its new surface of attachment. The activity and co-ordination which the rays manifest in executing these various acrobatic movements is surprising, and give to the animal an almost intelligent appearance.

With slight modifications this description may be taken to apply to all the other species of echinoderms (except *Holothuria*) which we have observed. These modifications are as follows:—

(i) There is a kind of star-fish (*Astropecten aurantiacus*) the ambulacral feet of which have lost their terminal suckers—these being but rudimentary, and the ambulacral feet being therefore pointed tubes. The animal uses these tubes in walking along a flat horizontal surface by pointing all the feet of all the rays in the direction of advance, and then distending them with fluid; they thus become so many pillars of support. The fluid is then suddenly withdrawn, and the star-fish falls forward with a jerk. It is particularly noteworthy that in this mode of progression all the feet of all the rays are co-ordinated in their action for determining one definite direction of advance—those in the ray facing that direction acting forwards, or centrifugally, those in the hinder rays backwards, or centripetally, and those in the lateral rays sideways. This description applies to the animal when progressing on a flat

horizontal surface out of the water; when in the water it appears as if the feet, besides being used as walking poles in the manner just described, are also used to sweep backwards along the floor of the tank, and so to assist in propelling the animal forwards after the manner of cilia. The result of all these movements is to produce a kind of locomotion which would seem more suited to a centipede than to a star-fish, and it enables the animal to travel at a surprising rate—i.e. from one to two feet per minute.

(ii) Brittle-stars (*Ophiuridiæ*) have entirely lost their ambulacral suckers, and the ambulacral feet themselves have become so rudimentary that they are functionally useless. Progression is therefore here effected entirely by the muscular contractions of the rays—the latter alternately shooting themselves forward and curving themselves backwards upon the floor of the tank like the arms of a swimming man—the resulting friction on the floor of the tank causing the animal to advance. This it does with great rapidity—viz. six feet per minute.

(iii) *Echini*, among echinoderms, stand at the opposite end of the series from the brittle-stars with reference to the solidarity of their rays, for these are here all united in a rigid box-like shell. Consequently the rate of their locomotion stands in striking contrast with that of the brittle-stars, being only six inches, instead of six feet, per minute. Looking to the slowness of this rate of locomotion, it must strike us as a curious fact that there is, perhaps, no animal which can properly be said to approach the *Echinus* in respect of the number and elaboration of special mechanisms subservient to the function of locomotion. Careful observation has satisfied us that these special mechanisms are four in number, and each of these displays an immense amount of elaboration. They are the pedicels, which act in the same way as they do in the star-fish, though with more adhesive power; the spines, which by closely co-ordinated movement act as walking poles; the lantern, which by alternate protrusion and retraction, does the same; and the pedicellariæ, which snap at pieces of seaweed and hold them steady till the suckers of the pedicels have time to form their adhesions.

From this account of the natural movements exhibited by

the several groups of echinodermata here considered, it may be observed that we have presented to our view an interesting series of graduated modifications. At one end of this series we have *Echinus* and *Spatangus* with their rays all united into a box-like rigid shell. At the other end of the series we have the brittle-stars with their muscular rays, highly mobile, and indeed snake-like in their well co-ordinated movements. Midway in the series we have the sea-cucumber and common star-fish, where the body is flexible and mobile, though not so much so as in the brittle-stars. Now, the interesting point to observe is, that in correlation with this graduated difference in the function of the rays, we have a correspondingly graduated difference in the development of the ambulacral system. In *Echinus* and *Spatangus* this system is seen in its most elaborate and efficient form—in *Echinus* the pedicels, spines, and pedicellariæ being more highly developed and useful than in any of the other groups, except *Spatangus*, where the spines are even more so. In the common star-fish, sun-stars, and sea-cucumbers, the ambulacral feet are still the most important organs of locomotion, although even here we begin to see that the development of the general muscular system has begun to tell upon that of these specially locomotor organs. Again, in *Astropecten* the still greater development of the general muscular system has told still further upon that of the ambulacral feet, the terminal suckers having become aborted. Lastly, the brittle-stars have altogether discarded the use of their ambulacral feet in favour of the much more efficient organs of locomotion supplied by their muscular rays; and not only the terminal suckers of these feet, but even the whole of the feet themselves, have dwindled into useless rudiments.

Lastly, with reference to natural movements, all the echinodermata that we have observed are able, when placed upon their dorsal surfaces on the floor of a tank, to recover their normal position on their oral surface. The common star-fish does so by twisting the ends of two or more of its rays round, so as to bring its terminal suckers into action upon the floor of the tank, and then, by a successive and similar action of the suckers further back in the series, the whole ray is progressively twisted round, so that its ambulacral surface is applied

flat against the floor. The rays which perform this action twist their semi-spirals in the same direction, and by their concerted action serve to drag the disc and the remaining rays over themselves as a fulcrum. Other species of star-fish, which have not their ambulacral suckers sufficiently developed to act in this way, execute their righting movements by doubling under two or three of their adjacent rays, and turning a somersault over them, as in the previous case. *Echinus* rights itself when placed on its ab-oral pole, by the successive action of two or three adjacent rows of suckers—so gradually rising from ab-oral pole to equator, and then as gradually falling from equator to oral pole. *Spatangus* executes a similar manœuvre entirely by the successive pushing and propping action of its longer spines.

Coming now to our experiments on the physiology of the nervous system, I may conveniently divide them into two classes—those in stimulation and those in section.

All the echinoderms we have observed respond to all kinds of stimulation. The period of latency varies considerably in different species, and in different parts of the same animal. In the holothurians it is remarkably long, and from the seat of stimulation there very frequently starts a wave of strong contraction, which passes with extreme slowness throughout the length of the animal in the form of a deep constriction. Similar waves frequently occur spontaneously.

All the echinoderms seek to escape from injury. Thus, for instance, if a star-fish or *Echinus* is advancing continuously in one direction, and if it be pricked or cut in any part of an excitable surface facing the direction of advance, the animal immediately reverses that direction; or, if it be taken out of the water and a drop of some irritating fluid be placed on any part of the external surface, the animal will endeavour to move away from the source of irritation; whether placed upon a dry table or returned to the water, the echinoderm will at once strike off in a perfectly straight line from the source of irritation, and for a long time will travel much more rapidly than usual. When two points of the surface are thus irritated, the direction of advance is usually the diagonal between them. When a greater number of points are irritated, the direction

of advance becomes uncertain, but if any, even short, interval of time is allowed to elapse between the application of successive stimuli to different parts of the surface, the direction of advance will be in a straight line from the stimulus applied latest. When a star-fish is fastened upon a perpendicular surface, and any part of its body is irritated, as, e.g., by a nip with the forceps, the animal, if a sun-star, will actively run away from the irritation. If, however, the latter be followed up and repeated, the star-fish seems to make up its mind to escape in a still more expeditious manner, for it immediately lets go its hold with all its suckers, and falls to the bottom of the water. A common star-fish will generally resort to this method when first irritated, without waiting for a repetition of the stimulus. An *Echinus*, on the other hand, will not drop off a perpendicular surface unless compelled to do so by serious irritation; it crawls away as quickly as possible, and sometimes rotates upon its axis in a manner afterwards to be described, whereby, without leaving go its hold of the perpendicular surface, it is able to alter its position rapidly. But of all the echinoderms the most curious to observe in this connection are the brittle-stars, for these may be made to leap about in any number of directions with much activity, by gently stimulating different parts of their bodies successively. When any part of the dorsal surface of any star-fish is irritated, not unfrequently one of the arms is doubled over and touches the seat of irritation, as if to endeavour to brush away the offending body.

That the external surface of a star-fish should prove itself to be excitable is what we should perhaps expect *à priori*, although we might not expect to find so high a degree of co-ordination manifested by the nervous system as is implied by its responses to the cutaneous excitations above mentioned. But that the external surface of an *Echinus* or *Spatangus* should be so highly excitable as it is, we should scarcely have anticipated—particularly before our observation of the external nervous plexus; for at first sight it would seem that the numberless long and mobile feet—to say nothing of the spines—would be sufficient to convey all the information that the animal requires concerning the external world, without its

exterior requiring to be rendered sensitive over its whole surface. Yet we find, so far is this from being the case, that the external surface cannot be touched with a needle's point at any part without the whole animal being affected thereby. We have already described the nervous plexus whereby this general sensitiveness of the external surface is secured. We must now enter pretty fully into the functions of this plexus as revealed by sundry experiments on the multitudinous and wonderful system of organs which, either directly or indirectly, depend upon this plexus for their innervation.

These organs are the ambulacral feet, the spines, and the pedicellariæ. That all these organs are in nervous connection with the external plexus is proved by the fact that when any part of the external surface is touched, however gently, all the feet, spines, and pedicellariæ within reach of that point, and even far beyond, immediately approximate and close in upon the point, so holding fast to the needle, or whatever other body may be used as the instrument of stimulation. This simultaneous movement of such a little forest of prehensile organs is a singularly beautiful spectacle to witness. In executing it, the pedicellariæ are much the most active, the spines somewhat slower, and the ambulacral feet very much slower. If the object with which the external surface is touched be itself small enough, or presents edges narrow enough, to admit of the forceps on the pedicellariæ establishing a hold upon it, it is seen to be immediately seized by some of these organs, and held there till the spines and ambulacral feet come up to assist; but if the object is too large, or does not present any surfaces which the pedicellariæ are able to catch—such, for instance, as the point of a pencil—the spines alone are able to hold it with wonderful firmness by forcing their tips against it on all sides.

The area thus affected by an ordinary stimulation, such as that supplied by a touch with a needle, measures in a longitudinal direction about half an inch. The extent of the area affected in a transverse or latitudinal direction depends upon the point stimulated with reference to the ambulacral feet. Midway in an interambulacral area the influence extends as far as the double rows of feet on either side; the feet, however,

of the inner, or nearer rows, moving more decidedly than those of the outer, or further, rows. The spines are rarely affected beyond the area named by a stimulus of mere contact, but in the case of the pedicellariæ the irradiation of the stimulating influence may proceed further, sometimes extending as far as the second double row of feet, or ambulacral area, on either side; the certainty and activity of their movements, however, rapidly diminish with their distance from the seat of stimulation. At and near the seat of stimulation, i.e., within the area first named, the certainty and activity of their movements are very great, and the period of latency very short; in other words, immediately any solid body touches any part of the external surface of an *Echinus*, it is surrounded by all the pedicellariæ in the neighbourhood, while even those which are too far away to touch the object will, perhaps for the long distance round which we have named, bend towards it.

And here we have the proof of the function of the pedicellariæ. In climbing perpendicular or inclined surfaces of rock, covered with waving sea-weeds, it must be of no small advantage to an *Echinus* to be provided on all sides with a multitude of forceps, all mounted on movable stalks, which instantaneously bring their grasping forceps to bear upon and to seize a passing frond. The frond being thus arrested, the spines come to the assistance of the pedicellariæ, and both together hold the *Echinus* to the support furnished by the sea-weed. Moreover the sea-weed is thus held steady till the ambulacral feet have time also to establish their hold upon it with their sucking discs. That the grasping and arresting of fronds of sea-weed in this way for the purposes of locomotion constitute an important function of the pedicellariæ, may at once be rendered evident experimentally by drawing a piece of sea-weed over the surface of a healthy *Echinus* in the water. The moment the sea-weed touches the surface of the animal, it is seen and felt to be seized by a number of these little grasping organs, and—unless torn away by a greater force than is likely to occur in currents below the surface of the sea—it is held steady till the ambulacral suckers have time to establish their attachments upon it. Thus there is no doubt that the pedicellariæ are able efficiently to perform the function which we

regard as their chief function. We so regard this function, not merely because it is the one that we observe these organs chiefly to perform, but also because we find that their whole physiology is adapted to its performance. Thus their multitudinous number and ubiquitous situation all over the external surface of the animal, are suggestive of their being adapted to catch something which may come upon them from any side, and which may have strings and edges so fine as to admit of being enclosed by the forceps. Again, the instantaneous activity with which they all close round and seize a moving body of a size that admits of their seizing it, is suggestive of the objects which they are adapted to seize being objects which rapidly brush over the surface of the shell, and therefore objects which, if they are to be seized at all, must be seized instantaneously. Lastly, we find, on experimenting upon pedicellariæ whether *in situ* or when separated from the *Echinus*, that the clasping action of the forceps is precisely adapted to the function which we are considering; for not only is the force exerted by the forceps during their contraction of an astonishing amount for the size of the organ (the serrated mandibles of the trident pedicellariæ holding on with a tenacity that can only have reference to some objects liable to be dragged away from their grasp), but it is very suggestive that this wonderfully tenacious hold is spontaneously relaxed after a minute or two. That is to say, the pedicellariæ tightly fix the object which they have caught for a time sufficient to enable the ambulacral suckers to establish their connections with it, and then they spontaneously leave go; their grasp is not only so exceedingly powerful while it lasts, but it is as a rule timed to suit the requirements of the pedicels.

We must next consider stimulation with reference to the spines. It has already been said that these organs co-operate with the pedicellariæ in grasping any instrument of stimulation, and this proves that for a certain area round any seat of stimulation the spines admit of co-ordinated action. Further experiments prove that there is no limit to the area within which co-ordinated action of the spines may take place; but that all the spines of the organism may work together to the attainment of some common end. Thus it has already been

stated in a previous part of this paper that a *Spatangus*, when placed upon its back, is able to right itself by the co-ordinated action of its spines alone; and also that an *Echinus*, when taken out of the water and placed upon a table, will walk in a determinate direction by the same means. The very complete co-ordination of the spines implied by these facts is, however, rendered still more conspicuous by experiments in stimulation; for if, while an *Echinus* is walking on the table in the manner just alluded to, a scrape with a scalpel, a drop of spirit, a lighted match, or any other severe stimulus be applied at some one part of the animal's exterior, the spines all over the surface begin to take on an active bristling movement, and the direction of advance is immediately changed into a straight line of escape from the source of injury. And, were it necessary, other experiments could be detailed to show that the multitudinous spines of an *Echinus* are as closely co-ordinated in their action as so many limbs.

It remains to consider stimulation with reference to the pedicels. Taking here the star-fish as a type of the echinodermata, the results of our experiments on this head, and so far as stimulation is concerned, are as follows. When a drop of acid, or other severe stimulation is applied to any part of a row of protruded pedicels, that whole row is immediately retracted, the pedicels retracting successively from the seat of irritation—so that if the latter be in the middle point of the series, two series of retractions are started, proceeding in opposite directions simultaneously; the rate at which they travel is rather slow. This process of retraction, however, although so complete within the ray irritated, does not extend to the other rays. But if the stimulus be applied to the centre of the disc, upon the oral surface of the animal, all the feet in all the rays are more or less retracted—the process of retraction radiating serially from the centre of stimulation. The influence of the stimulus, however, diminishes perceptibly with the distance from the centre; thus, if weak acid be used as the irritant, it is only the feet near the bases of the rays that are retracted; and, even if very strong acid be so used, it is only the feet as far as one-half or two-thirds of the way up the rays that are fully retracted—the remainder only having their

activity impaired, while those near the tip may not be affected at all. If the drop of acid be placed on the dorsal, instead of the ventral surface of the disc, the effect on the feet is found to be just the converse; that is, the stimulus here applied greatly increases the activity of the feet. Further experiments show that this effect is produced by a stimulus applied anywhere over the dorsal aspect of the animal; so that, for instance, if a drop of acid be placed on the skin, at the edge of a ray, and therefore just external to the row of ambulacral feet, the latter will be stimulated into increased activity; whereas, if the drop of acid had been placed a very small distance past the edge of the ray, so as to touch some of the feet themselves, then the whole row would have been drawn in. We have here rather an interesting case of antagonism, which is particularly well marked in *Astropecten*, on account of the active writhing movements which the feet exhibit when stimulated by an irritant placed on the dorsal surface of the animal. It may be added that in this antagonism the inhibitory function is the stronger; for when the feet are in active motion, owing to an irritant acting on the dorsal surface, they may be reduced to immediate quiescence—i.e., retracted—by placing another irritant on the ventral surface of the disc. Similarly, if retraction has been produced by placing the irritant on the ventral surface of the disc, activity cannot be again induced by placing another drop of the irritant on the dorsal surface.

The only other observations that need be mentioned under the heading of stimulation are those which refer to the influence of light. We have found unequivocal evidence of the star-fish (with the exception of the brittle-stars) and the *Echini* manifesting a strong disposition to crawl towards, and to remain in, the light. Thus if a large tank be completely darkened, except at one end where a narrow slit of light is admitted, and if a number of star-fish and *Echini* be scattered over the floor of the tank, in a few hours the whole number, with the exception of perhaps a few per cent., will be found congregated in the narrow slit of light. The source we used was diffused daylight, which was admitted through two sheets of glass, so that the thermal rays might be considered prac-

tically excluded. The *intensity* of the light which the echinoderms are able to perceive may be very feeble indeed; for in our first experiments we boarded up the face of the tank with ordinary pine-wood, in order to exclude the light over all parts of the tank except at one narrow slit between two of the boards. On taking down the boards we found indeed the majority of the specimens in or near the slit of light; but we also found a number of other specimens gathering all the way along the glass face of the tank that was immediately behind the pine-boards. On repeating the experiment with blackened boards, this was never found to be the case; so there can be no doubt that in the first experiments the animals were attracted by the faint glimmer of the white boards, as illuminated by the very small amount of light scattered from the narrow slit through a tank all the other sides of which were black slate. Indeed, towards the end of the tank, where some of the specimens were found, so feeble must have been the intensity of this glimmer, that we doubt whether even human eyes could have descried it very distinctly. Owing to the prisms at our command not having sufficient dispersive power for the experiments, and not wishing to rely on the uncertain method of employing coloured glass, we were unable to ascertain how the echinoderms might be affected by different rays.

On removing with a pointed scalpel the eye-spots from a number of star-fishes and Echini without otherwise injuring the animals, the latter no longer crawled towards the light, even though this were admitted to the tank in abundance; but they crawled promiscuously in all directions. On the other hand, if only one of the five eye-spots were left intact, the animals crawled toward the light as before.

Coming now to the experiments in section, we find that single rays detached from star-fish crawl as fast and in as determinate a direction as do the entire animals. They also crawl towards the light, up perpendicular surfaces, and sometimes away from injuries; but they do not invariably, or even generally, seek to escape from the latter, as is so certain to be the case with entire animals. Lastly, separated rays right themselves when inverted as quickly as do the unmutilated organisms.

Removing the tip of a severed ray does not impair any of these movements, except, of course, the crawling towards light, which it completely destroys. Dividing the nerve in any part of its length has the effect, whether or not the ray is detached from the animal, of completely destroying all physiological continuity between the feet on either side of the line of division. Thus, for instance, if the nerve be cut across half-way up its length, the row of feet is at once physiologically bisected—one-half of the row becoming as independent of the other half as it would were the whole ray divided into two parts. That is to say, the distal half of the feet may crawl while the proximal half is retracted, or *vice versâ;* and if a drop of acid be placed on either half, the serial contraction of the feet in that half stops abruptly at the line of nerve-division. As a result of this complete physiological severance, when a detached ray so mutilated is inverted, it experiences much greater difficulty in righting itself than it does before the nerve was divided. The line of nerve-injury lies flat upon the floor of the tank, while the central and distal portions of the ray—i.e., the portions on either side of that line—assume various movements and shapes. The central portion is particularly apt to take on the form of an arch, in which the central end of the severed ray and the line of nerve-section constitute the points of support (tetanus?). Or the central end may from the first show paralysis, from which it never recovers. The distal end, on the other hand, usually continues active—twisting about in various directions, and eventually fastening its tip upon the floor of the tank to begin the spiral movement of righting itself. This movement then continues as far as the line of nerve-injury, where it invariably stops. The central portion may then be dragged over into the normal position, or may remain permanently inverted, according to the strength of pull exerted by the distal portion; as a rule it does not itself assist in the righting movement, although its feet usually continue protruded and mobile.

The above observations have reference to the common star-fish, but they apply equally to other star-fishes, except that in *Astropecten* single detached rays are not able to right themselves when inverted (owing to the feet not being used by

this species for this purpose, and to the other rays being absent), and that after division of the nerve in a ray of this species, the feet of the proximal portion usually manifest more activity than those of the distal. The destruction, however, of physiological continuity between the two portions is as complete as in the case of the common star-fish. Single detached rays of brittle-stars are able when inverted to right themselves; they wriggle round by means of their snake-like movements, and do not require, as in the case with the less active rays of *Astropecten*, the assistance of adjacent rays to effect the manœuvre. On the whole, then, it may be said, as a general statement, that in all the species of star-fish which we have observed, the effect of a transverse section of the nerve in a ray is that of completely destroying physiological continuity between the pedicels on either side of the section.

The only other experiments in nerve-section to which the simple anatomy of a star-fish exposes itself is that of dividing the nerve-ring in the disc; or, which is virtually the same thing, while leaving this intact, dividing all the nerves where they pass from it into the rays. In specimens mutilated by severing the nerves at the base of each of the five rays, or by dividing the nerve-ring between each ray, the animal loses all power of co-ordination among its rays. When a common star-fish is so mutilated it does not crawl in the same determinate manner as an unmutilated animal, but, if it moves at all, it moves slowly and in various directions. When inverted, the power of effecting the righting manœuvre is seen to be gravely impaired, although eventually it is always accomplished. There is a marked tendency, as compared with unmutilated specimens, to a promiscuous distribution of spirals and doublings, so that instead of a definite plan of the manœuvre being formed from the first, as is usually the case with unmutilated specimens, such a plan is never formed at all; among the five rays there is a continual change of uncoordinated movements, so that the righting seems to be eventually effected by a mere accidental prepotency of some of the righting movements over others.

When entire star-fish are mutilated by a section of each nerve trunk half-way up each ray, and the animal is then

placed upon its back, the tetanic contraction of the muscles in the rays before mentioned as occurring under this form of section in detached rays, has the effect, when now occurring in all the rays, of elevating the disc from the floor of the tank. This opisthotonous-like spasm is not, however, permanent; and the distal ends of the rays forming adhesions to the floor of the tank, the animal eventually rights itself, though much more slowly than unmutilated specimens. After it has righted itself, although it twists about the distal portions of the rays, it does not begin to crawl for a long time, and when it does so, it crawls in a slow and indeterminate manner. Star-fish so mutilated, however, can ascend perpendicular surfaces.

The loss of co-ordination between the rays caused by division of the nerve-ring in the disc is rendered most conspicuous in brittle-stars, from the circumstance that in locomotion and in righting so much here depends upon co-ordinated muscular contraction of the rays. Thus, for instance, when a brittle-star has its nerve-ring severed between each ray, an interesting series of events follows. First, there is a long period of profound shock—spontaneity, and even irritability, being almost suspended, and the rays appearing to be rigid, as if in tetanic spasm. After a time, feeble spontaneity returns—the animal, however, not moving in any determinate direction. Irritability also returns, but only for the rays immediately irritated, stimulation of one ray causing active writhing movements in that ray, but not affecting, or only feebly affecting, the other rays. The animal, therefore, is quite unable to escape from the source of irritation, the aimless movements of the rays now forming a very marked contrast to the instantaneous and vigorous leaping movements of escape which are manifested by unmutilated specimens. Moreover, unmutilated specimens will vigorously leap away, not only from stimulation of the rays, but also from that of the disc; but those with their nerve-ring cut make no attempts to escape, even from the most violent stimulation of the disc. In other words, the disc is entirely severed from all physiological connection with the rays.

If the nerve-ring be divided at two points, one on either side of a ray, that ray becomes physiologically separated from the rest of the organism. If the two nerve-divisions are so

placed as to include two adjacent rays—i.e., if one cut is on one side of a ray and the other on the further side of an adjacent ray—then these two rays remain in physiological continuity with one another, although they suffer physiological separation from the other three. When a brittle-star is completely divided into two portions, one portion having two arms and the other three, both portions begin actively to turn over on their backs, again upon their faces, again upon their backs, and so on alternately for an indefinite number of times. These movements arise from the rays, under the influence of stimulation caused by the section, seeking to perform their natural movements of leaping, which however end, on account of the weight of the other rays being absent, in turning themselves over. An entire brittle-star when placed on its back after division of its nerve-ring is not able to right itself, owing to the destruction of co-ordination among its rays. *Astropecten*, under similar circumstances, at first bends its rays about in various ways, with a preponderant disposition to assume the form of a tulip, and keeps its ambulacral feet in active movement. But after half an hour, or an hour, the feet generally become retracted and the rays nearly motionless—the animal, like a brittle-star, remaining permanently on its back. In this, as in other species, the effect of dividing the nerve-ring on either side of a ray is that of destroying its physiological connection with the rest of the animal, the feet in that ray, although still remaining feebly active, no longer taking part in any co-ordinated movement—that ray, therefore, being merely dragged along by the others.

Under this division it only remains further to be said, that section of the nerve-ring in the disc, or the nerve-trunks of the rays, although as we have seen so completely destroying physiological continuity in the rows of ambulacral feet and muscular system of the animal, does not destroy physiological continuity in the external nerve plexus; for, however much the nerve-ring and nerve-trunks may be injured, stimulation of the dorsal surface of the animal throws all the ambulacral feet and all the muscular system of the rays into active movement. This fact proves that the ambulacral feet and the muscles are all held in nervous connection with one another

by the external plexus, without reference to the integrity of the main nerve-trunks.

If a cork-borer be applied to the external surface of the shell of an *Echinus*, and rotated there till the calcareous substance of the shell is reached, and therefore a continuous circular section of the overlying tissues effected, it is invariably found that the spines and pedicellariæ within the circular area are physiologically separated from the contiguous spines and pedicellariæ, as regards local reflex excitability. That is to say, if any part of this circular area be stimulated, all the spines and pedicellariæ within that area immediately respond to the stimulation in the ordinary way; while none of the spines or pedicellariæ surrounding the area are affected. Similarly, if any part of the shell external to the circumscribed area be stimulated, the spines and pedicellariæ within the area are not affected. These facts prove that the function which is manifested by these appendages, of localising and gathering round a seat of stimulation, is exclusively dependent upon the external nerve plexus. It is needless to add that in this experiment it does not signify of what size or shape or by what means the physiological island is made, so long as the destruction of the nervous plexus by a closed curve of injury is rendered complete. In order to ascertain whether, in the case of an unclosed curve of injury, any irradiation of a stimulus would take place round the ends of the curve, we made sundry kinds of section. It is, however, needless to describe these, for they all showed that, after injury of a part of the plexus, there is no irradiation of the stimulus round the ends of the injury. Thus, for instance, if a short straight line of injury be made, by drawing the point of a scalpel over the shell, say along the equator of the animal, and if a stimulus be afterwards applied on either side of that line, even quite close to one of its ends, no effect will be exerted on the spines or pedicellariæ on the other side of the line. This complete inability of a stimulus to escape round the ends of an injury forms a marked contrast to the almost unlimited degree in which such escape takes place in the more primitive nervous plexus of the medusæ.[1]

[1] See 'Locomotor System of Medusæ,' Phil. Trans. 1875.

Although the nervous connections on which the spines and pedicellariæ depend for their function of localising and closing round a seat of stimulation are thus shown to be completely destroyed by injury of the external plexus, other nervous connections, upon which another function of the spines depends, are not in the smallest degree impaired by such injury. This other function is that which brings about the general co-ordinated action of all the spines for the purposes of locomotion. That this function is not impaired by injury of the external plexus is proved by the fact that, if the area within a closed line of injury on the surface of the shell be strongly irritated, all the spines over the whole surface begin to manifest their peculiar bristling movements, and by this co-ordinated action rapidly move the animal in a straight line of escape from the source of irritation; the injury to the external plexus, although completely separating the spines enclosed by it from their neighbouring spines as regards what may be called their local function of seizing the instrument of stimulation, nevertheless leaves them in undisturbed connection with all the other spines in the organism as regards what may be called their universal function of locomotion.

Evidently, therefore, this more universal function must depend upon some other set of nervous connections; and experiment shows that these are distributed over all the *internal* surface of the shell. Our mode of experimenting was to divide the animal into two hemispheres, remove all the internal organs of both hemispheres (these operations producing no impairment of any of the functions of the pedicels, spines, or pedicellariæ), and then paint with strong acid the inside of the shell—completely washing out the acid after about a quarter of a minute's exposure. The results of a number of experiments conducted on this method may be thus epitomised :—

The effect of painting the back or inside of the shell with strong acid (e.g. pure HCl) is that of at first strongly stimulating the spines into bristling movements, and soon afterwards reducing them to a state of quiescence, in which they lie more or less flat, and in a peculiarly confused manner, that closely

resembles the appearance of corn when "laid" by the wind. The spines have now entirely lost both their spontaneity and their power of responding to a stimulus applied on the external surface of the shell—i.e. their local reflex excitability, or power of closing in upon a source of irritation. These effects may be produced over the whole external surface of the shell, by painting the whole of the internal surface; but if any part of the internal surface be left unpainted, the corresponding part of the external surface remains uninjured. Conversely, if all the internal surface be left unpainted except in certain lines or patches, it will only be corresponding lines and patches on the external surface that suffer injury. It makes no difference whether these lines or patches be painted in the course of the ambulacral feet, or anywhere in the inter-ambulacral spaces.

The above remarks, which have reference to the spines, apply equally to the pedicellariæ, except that their spontaneity and reflex irritability are not destroyed, but only impaired.

Some hours after the operation it usually happens that the spontaneity and reflex irritability of the spines return, though in a feeble degree, and also those of the pedicellariæ, in a more marked degree. This applies especially to the reflex irritability of the pedicellariæ; for while their spontaneity does not return in full degree, their reflex irritability does—or almost in full degree.

These experiments, therefore, seem to point to the conclusions—1st, that the general co-ordination of the spines is dependent on the integrity of an internal nerve-plexus; 2nd, that the internal plexus is everywhere in intimate connection with the external; and 3rd, that complete destruction of the former, while profoundly influencing the functions of the latter, nevertheless does not wholly destroy them.[1]

In order that a more clear conception may be rendered of

[1] These three conclusions, first reached by the experiments described, were afterwards fully confirmed by histological investigation. The internal plexus is spread over the whole interior surface of the shell, and is everywhere in communication with the external plexus by means of delicate nerve-fibres which pass between the numerous plates of which the shell is composed.

the experiments on which these conclusions are based, we shall here quote from our notes one complete observation :—

"*Echinus* was divided into two hemispheres.

"After evisceration one hemisphere was painted over whole of internal surface with HNO_3. (A.)

"The other was painted down one row of ambulacral plates, and also down the inter-ambulacral plates at another part of internal shell. (B.)

"In (A) the spines were 'laid'; spontaneity and reflex irritability almost totally destroyed.

"In (B) similar effects observed above painted areas—unpainted areas unimpaired.

"Three hours after, no considerable recovery where painted; unpainted areas as active as before."

One further point, brought out by further experiments, may here be most conveniently mentioned; it is that a specially great influence, or shock, seems to be exerted on the external plexus by injury of the hypothetical internal plexus *along the lines of the ambulacral pores*. The following observations will serve to show this :—

"Another specimen was divided into two hemispheres. In one hemisphere two adjacent ambulacral rows were thoroughly scraped on internal surface of shell, and then well rubbed with sandpaper and brickdust.[1] The spines along these lines were laid in a very marked way, while spontaneity and reflex irritability, not only along them, *but also in the inter-ambulacral spaces between them*, were completely destroyed. The rest of the hemisphere was normally active.

"Ten minutes after operation the laid spines became more erect, and reflex irritability partly returned.

"Twenty minutes after operation pedicellariæ nearly completely recovered spontaneity and reflex irritability; spines still very imperfectly so.

"Two hours after operation both spines and pedicellariæ

[1] This method of destroying the internal plexus was here adopted in preference to the method of painting with acid, in order to avoid a possible source of fallacy in some of the acid passing through the perforations of the shell, and so finding its way over the external surface. All our experiments with acid were on other specimens controlled by similar experiments conducted on this method.

of the inter-ambulacral area *completely recovered in all respects.*"

If an *Echinus* is divided into two hemispheres by an incision carried from pole to pole through any meridian, the two hemispheres will live for days, crawling about in the same manner as entire animals; if their ocular plates are not injured, they seek the light, and when inverted they right themselves. The same observations apply to smaller segments, and even to single detached rows of ambulacral feet. The latter are, of course, analogous to the single detached rays of a star-fish, so far as the system of ambulacral feet is concerned; but looking to the more complicated apparatus of locomotion (spines and pedicellariæ), as well as to the rigid consistence and awkward shape of the segment—standing erect, instead of lying flat—the appearance presented by such a segment in locomotion is much more curious, if not surprising, than that presented by the analogous part of a star-fish under similar circumstances. It is still more surprising that such a fifth-part segment of an *Echinus* will, when propped up on its ab-oral pole, right itself after the manner of larger segments or entire animals. They, however, experience more difficulty in doing so, and very often, or indeed generally, fail to complete the manœuvre.

We are now face to face with the question—Is the action of the ambulacral feet in executing these righting movements of a merely serial kind, or does it depend upon nervous co-ordination? We have found this question very difficult of solution, and in the end have arrived at the conclusion that both principles are combined—the action of the feet being serial, but also assisted by nervous co-ordination. The experiments which lead us to this conclusion are as follows:—

If an unmutilated *Echinus* be suspended by a thread in an inverted position half-way up the side of a tank, in such a way that the ambulacral feet on one side of the ab-oral pole are alone able to reach the perpendicular wall, these feet as quickly as they can establish their attachments to that wall; the thread being then removed, the *Echinus* is left sticking to the side of the tank in an inverted position by means of the ab-oral ends of two adjacent feet-rows. Under these circumstances, as we should expect from the previous experiments,

the animal sets about righting itself as quickly as possible. Now, if the righting action of the feet were entirely and only of a serial character, the righting would require to be performed by rearing the animal upwards; the effect of foot after foot in the same rows being applied in succession to the side of the tank, would require to be that of rotating the globular shell against the side of the tank towards the surface of the water, and therefore against the action of gravity. This is sometimes done, which proves that the energy required to perform the feat is not more than a healthy *Echinus* can expend. But much more frequently the *Echinus* adopts another device, and the only one by which it is possible for him to attain his purpose without the labour of rotating upwards; he rotates laterally and downwards in the form of a spiral. Thus, let us call the five feet-rows, 1, 2, 3, 4, and 5, and suppose that rows 1 and 2 are in use near their ab-oral ends in holding the animal inverted against the perpendicular side of a tank. The downward spiral rotation would then be effected by gradually releasing the outer feet in row 1, and simultaneously attaching the outer feet in row 2 (i.e. those nearest to row 3, and furthest from row 1), as far as possible to the outer side of that row. The effect of this is to make the globe roll far enough to that side to enable the inner feet of row 3 (i.e. those nearest to row 2), when fully protruded, to touch the side of the tank. They establish their adhesions, and the residue of feet in row 1, now leaving go their hold; these new adhesions serve to roll the globe still further round in the same direction of lateral rotation, and so the process continues from row to row; but the globe does not merely roll along in a horizontal direction, or at the same level in the water, for each new row that comes into action takes care, so to speak, that the feet which it employs shall be those which are as far below the level of the feet in the row last employed as their length when fully protruded (i.e. their power of touching the tank) renders possible. The rotation of the globe thus becomes a double one, lateral and downwards, till the animal assumes its normal position with its oral pole against the perpendicular tank wall. So considerable is the rotation in the downward direction, that the normal position is

generally attained before one complete lateral, or equatorial, rotation is completed.

The result of this experiment, therefore, implies that the righting movements are due to something more than the merely successive action of the series of feet to which the work of righting the animal may happen to be given. The same conclusion is pointed to by the results of the following experiment.

A number of vigorous *Echini* were thoroughly shaved with a scalpel over the whole half of one hemisphere—i.e. the half from the equator to the oral pole. They were then inverted on their ab-oral poles. The object of the experiment was to see what the *Echini* which were thus deprived of the lower half of three feet-rows would do when, in executing their righting manœuvres, they attained to the equatorial position and then found no feet wherewith to continue the manœuvre. The result of this experiment was first of all to show us that the *Echini* invariably chose the unmutilated feet-rows wherewith to right themselves. Probably this is to be explained, either by the general principle to which the escape from injury is due—viz. that injury inflicted on one side of an echinoderm stimulates into increased activity the locomotor organs of the opposite side,—or by the consideration that destruction of the lower half of a row very probably induces some degree of shock in the remaining half, and so leaves the corresponding parts of the unmutilated rows prepotent over the mutilated one. Be this as it may, however, we found that the difficulty was easily overcome by tilting the animal over upon its mutilated feet-rows sufficiently far to prevent the unmutilated rows from reaching the floor of the tank. When held steadily in this position for a short time, the mutilated rows established their adhesions, and the *Echinus* was then left to itself. Under these circumstances an *Echinus* will always continue the manœuvre along the mutilated feet-rows with which it was begun, till the globe reaches the position of resting upon its equator, and therefore arrives at the line where the shaved area commences. The animal then remains for hours in this position, with a gradual but continuous motion backwards, which appears to be due to the successive slipping of the spines—these organs in the righting movements being always used as props for the

ambulacral feet to pull against while rearing the globe to its equatorial position, and in performing this function on a slate floor the spines are liable often to slip. The only other motion exhibited by *Echini* thus situated is that of a slow rolling movement, now to one side and now to another, according to the prepotency of the pull exerted by this or that row of ambulacral feet. Things continue in this way until the slow backward movement happens to bring the animal against some side of the tank, when the uninjured rows of ambulacral feet immediately adhere to the surface and rotate the animal upwards or horizontally, until it attains the normal position. But if care be taken to prevent contact with any side of the tank, the mutilated *Echinus* will remain propped on its equator for days; it never adopts the simple expedient of reversing the action of its mutilated feet-rows, so as to bring the globe again upon its ab-oral pole and get its unmutilated feet-rows into action. At first sight, therefore, this result seems to point to the conclusion that the righting movements are of a merely serial kind; it seems to indicate that the feet are only able to act in one direction, from ab-oral to oral pole, and that there is not sufficient central co-ordination to induce them to act in the opposite direction, when it is found to be useless, from the interruption of the series, to continue the manœuvre in the ordinary direction. But a little closer thought will show that this conclusion is not justified by the facts. For even if we assume that the righting movements of the feet are entirely due to some central co-ordinating influence, it does not follow, when the execution of these movements is interrupted by the highly artificial means of shaving off one-half the feet-rows, that the central co-ordinating apparatus should be adapted to meet so unnatural a state of things. Suppose, for instance, that it is an incipient sense of gravity that determines this central apparatus to work the feet-rows serially, in order to rotate the animal into its normal position; it does not follow that, under any circumstances, the stimulus supplied by this sense of gravity should induce the central apparatus to *reverse* the action of the feet-rows; for to do this would, under any circumstances, be to act in opposition to the stimulus supposed. Only if we were to imagine that

the central apparatus, if present, must possess a true psychological element capable of sufficient intelligence to reflect that by temporarily acting in opposition to the sense of gravity the peculiar exigencies of the situation might be overcome—only then could we fairly argue that the result of these experiments shows the righting movements of the feet to be purely serial, or wholly independent of nervous co-ordination. As a matter of physiology, therefore, the only question in the present connection which we have to consider is this—is the mechanism of the ambulacral feet so constructed as to insure that their serial action shall always take place in the same direction? For if it can be shown that their serial action may take place indifferently in either direction, it would follow that the persistency with which the shaved *Echini* continued reared upon their equators is the expression of some stimulus (such as a sense of gravity) continuously acting upon some central apparatus, and so impelling the latter to a continuous, though fruitless, endeavour at co-ordinating the absent feet. If the righting movements were wholly independent of any such central apparatus, and due only to the serial action of the feet, we should expect that (supposing the feet to be able to act serially in either direction) when the equator position had been attained in shaved specimens, it would not be maintained. For if there were no constant stimulus emanating from any co-ordinating centre persistently trying to induce the absent feet to continue the serial action in the same direction, we should expect, if serial action can take place in either direction, that after a time it should begin to take place in the opposite direction; upon the supposition that the feet may act serially in either direction, there is no more reason why a shaved *Echinus* should remain permanently reared upon its equator than there is that it should remain permanently inverted upon its pole, and therefore the fact that in the latter position the feet set about an immediate rotation of the animal, while in the former and quite as unnatural position they hold the animal in persistent stasis—this fact tends to show that the righting movements of the feet are something more than serial. The question, therefore, that we set ourselves to determine was, whether the serial action of the feet invariably takes

place in the direction of ab-oral to oral pole, or may likewise take place in the opposite direction. We found that it may take place in the opposite direction, as the following observations prove. We have seen a shaved specimen, which after remaining for several hours on its equator was accidentally rolled over into its normal position, forthwith begin to rear itself upon its uninjured feet-rows. Executing this what we may call an inverted righting movement with activity, the *Echinus* was speedily reared into the equatorial position on the opposite side to that from which it had just fallen—and in order to do this, it is needless to say, the feet of the uninjured rows had to be used serially in the direction opposite to that in which they are required to act when executing the ordinary righting manœuvre. We may wonder what the stimulus can have been which induced this *Echinus* spontaneously to rise upon its equator; but it is of interest in this connection to add that, so soon as the equator position had been attained, and so soon therefore as any further action of the uninjured feet-rows in the same direction would have begun to get the animal into a position of ever-increasing difficulty as regards subsequent righting, so soon did the serial action in this direction cease, became reversed, and so again brought the animal gently into its normal position.

We have also seen wholly uninjured specimens when reaching the surface of the water by crawling up the sides of a tank, spontaneously rear themselves upon their equators and remain in that position for several minutes; but we have never observed a case of such rotation carried further than the equatorial line. The fact, however, that such rotation from oral to ab-oral pole can take place over half the whole length of a pair of feet-rows, proves that the feet may act serially in either direction. The same thing is further proved by the fact that single detached rays of star-fish sometimes crawl backwards, and that in entire star-fish the rays opposite to the direction of advance work their ambulacral feet centripetally, while those on the rays facing that direction work centrifugally.

Lastly, as proof that the ambulacral feet of *Echinus* are under the control of some centralising apparatus when execut-

ing the righting manœuvre, we may state one other fact. When the righting manœuvre is nearly completed by the rows engaged in executing it, the lower feet in the other rows become strongly protruded and curved downwards, in anticipation of shortly coming into contact with the floor of the tank when the righting manœuvre shall have been completed. This fact tends to show that all the ambulacral feet of the animal are, like all the spines, held in mutual communication with one another by some centralising mechanism.

Such, then, is the evidence we have to adduce for the purpose of showing that the action of the ambulacral feet is not entirely or only of a serial kind, but is, in part at all events, dependent upon some centralising influence by which all the feet, like all the spines, are rendered capable of truly co-ordinated action. We have next to adduce our evidence to show that the action of the ambulacral feet, although as we have seen in some measure, is not exclusively dependent on this centralising influence.

In order to show this we must first narrate the experiments whereby we succeeded in ascertaining the central apparatus, on the integrity of which both the feet and the spines depend for their co-ordination. Having obtained the definite evidence of co-ordination which has now been fully detailed, we of course sought to localise the centre to which this co-ordination is due; and in searching for this centre our thoughts naturally turned to the only part of the nervous system where we could reasonably expect to find it. This part is the central nerve-ring, and, as we had anticipated, experiment revealed unmistakable evidence of this being the centre of which we were in search.

If a circular incision be made all the way round the lantern of an *Echinus*, at a sufficient distance from the lantern to insure that the connections of the nerve-ring with the rest of the organism shall be severed, the following results are produced :—

1. *Pedicels.*—Spontaneity impaired, though not destroyed. They are protruded, but not in such numbers or with so much activity as in the unmutilated animal; they, however, form their adhesions in the ordinary manner whenever they come

into contact with a sold surface, and therefore their function of anchoring the *Echinus* securely remains unimpaired. They also still continue able to crawl, but they do so feebly and no longer in a determinate direction; the animal therefore advances slowly and in a very uncertain manner, frequently changing its direction of advance, and manifesting a marked tendency to rotate upon its own axis, either without moving from one spot or gyrating round and round some one or more centres in a wholly aimless way. The animals, however, are still able to climb perpendicular surfaces, though in a most uncertain manner. When stimulated strongly the activity of the animal is increased, but its power of escaping from the source of injury is completely destroyed; it crawls indifferently in any direction—as likely as not *towards* the source of injury—rotates upon its axis, and after crawling some distance in one direction may very likely reverse that direction, and so return to the place from which it started. All these movements, standing in such marked contrast to those exhibited by unmutilated specimens under similar circumstances, prove that the co-ordination of the ambulacral feet has been destroyed. On the other hand, the fact that they continue able to act *at all* proves that their activity is not *wholly* dependent upon the nerve-centre; all that the destruction of this centre entails is the destruction of their power of *co-ordinated* action.

When perfectly fresh and vigorous specimens are inverted, a proportion of about three to four remain permanently inverted till they die. As this is never the case with perfectly fresh and vigorous specimens when unmutilated, there can be no question that destruction of the nerve-centre exerts a profound influence on the action of the ambulacral feet upon which the execution of the righting manœuvre depends. On the other hand, the fact that a certain proportion of individuals continue able to execute this manœuvre after destruction of the nerve-centre—although they never do so without much difficulty and after a long time—proves that the integrity of this centre is not absolutely essential to the execution of this manœuvre. Therefore, as experiment has failed to reveal to us any other general nerve-centre in the animal, and as even a

segment of the animal containing but a single row of feet is in many cases able to perform this manœuvre, we conclude, as previously stated, that the action of the feet in performing these righting movements is partly of a serial character, although, for reasons mentioned in the two previous paragraphs we further conclude that in the unmutilated animal these movements are largely assisted by the co-ordinating influence that emanates from the nervous centre.

2. *Pedicellariæ.*—No effect whatever is produced upon these organs by destruction of the nerve-ring.

3. *Spines.*—These organs, on the other hand, are profoundly affected—not, indeed, as regards their spontaneity and the function which they share with the pedicellariæ of closing round any instrument of stimulation, but as regards their other two more general functions. That the particular or local function which they share with the pedicellariæ should not be impaired by destruction of the general nerve-centre is no more than we might expect from those experiments detailed in previous parts of this paper, which proved that this function is performed exclusively by the numberless local nerve-centres (cells) of the external plexus. Thus, for instance, it will be remembered that when a small piece is cut out of the shell of an *Echinus* or *Spatangus*, and the internal surface of that piece painted with acid, its spines and pedicellariæ, although severed from any possible nervous connection save those of the external plexus, will continue to perform their function of localising a seat of stimulation.

As regards, then, the more general function of the spines, we have first to consider what we may term their general reflex irritability—i.e. their power of active bristling response all over the animal when any part of its surface is strongly stimulated, as by burning. Immediately after the operation of removing the nerve-centre this function is found to be in abeyance, or nearly so—strong stimulation of one part of the animal not being followed by any response of the spines in other parts. This effect, however, completely passes off within several hours after the operation, and is therefore to be attributed to shock. The fact, however, that the influence of shock is thus revealed in temporarily suspending this general nervous

communication among the spines, proves that this general communication, unlike the more special one which they share with the pedicellariæ, is itself in communication with the central nervous ring. Further, the experiments detailed in a previous part of this paper prove that the medium of communication is in this case the *internal* nervous plexus, as in the case just mentioned the medium of communication has been proved to be the *external* nervous plexus. And as the effect of the operation in question is only transitory—after recovery from shock the spines being as responsive as ever to severe stimulation—we must conclude that the general communication between the spines is maintained by the direct conductility of the internal plexus, and is not of the nature of a reflex in which the nerve-ring is concerned as a general centre. The only effect of removing this nerve-ring is temporarily to paralyse, through shock, the internal plexus with which the ring is connected.

Lastly, the effect of removing the nerve-ring is that of completely and permanently destroying the general co-ordination of the spines; that is to say, after this operation these organs are never again of any use to the *Echinus* for the purpose of locomotion. When the animal is placed upon a table and a lighted spirit lamp held against one side, although all the spines will manifest their active bristling movements, they will not co-operate to move the animal away from the source of irritation, as is so invariably the case with unmutilated specimens. Removal of the nerve-ring has entirely destroyed the general co-ordination of the spines.

ON OPHTHALMOPLEGIA EXTERNA, IN CONJUNCTION WITH TABES DORSALIS, WITH SOME REMARKS ON GASTRIC CRISES.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and Epileptic.

In the course of the last three years I have had two cases of ophthalmoplegia with tabes under my care in the hospital. One of them, a female, is, I believe, still living, but I have not seen her for some time past. The other, a male, died in the hospital, and an autopsy was obtained. Portions of the hardened nerve-centres were forwarded to Dr. Bevan Lewis, of the West Riding Asylum, Wakefield, who has been kind enough to examine them microscopically, and furnish me with the valuable report and drawing which are appended to the case.

A. B., a female, æt. 25, was admitted into the National Hospital for the Paralysed and Epileptic on January 16th, 1879. The following account of her case is partly derived from notes which were made by Mr. A. E. Broster, then Resident Medical officer.

On the right side there is semi-ptosis of the eyelid, together with paralysis of the recti and obliqui muscles, which appears to be absolute, with the exception that the inferior rectus can slightly move the eyeball.

On the left side there is ptosis of the eyelid with paralysis of all the recti and obliqui, with the exception of the external rectus.

The right pupil is 4 mm., the left 4·5 mm. in diameter. There is no action to light in either, but they act a little during attempted accommodation. (I may say, in general terms, that no muscle of either eye acted properly except the

left external rectus.) There is no affection of cutaneous sensibility or motor power in the range of the fifth nerve.

Taste and hearing are good; the tongue is protruded straight, but with some quivering. The right side of the face does not move so perfectly as the left.

The arms are somewhat weak. The grasp of the right hand measures 52, the left 55. There is slight anæsthesia to touch and pain in the first two fingers of the right hand. When asked to touch the tip of the nose with the right index finger she missed it altogether, but reached it, though with some difficulty, with her left.

As she sits she can raise both knees, but on trying to stand her legs give way, and she needs the help of two persons to walk. Her gait is characteristically ataxic.

She cannot stand by herself with her eyes open; if they are shut the difficulty is greatly increased.

She feels the ground imperfectly with the soles of the feet, and in these she gets acute "pins and needles," which cause her legs to "jump." The muscular sense of the lower extremities is diminished. The knee phenomenon is absent in each leg. There is no foot-clonus.

Besides a general emaciation of the arms and legs, certain muscular groups are picked out by atrophy, especially the right serratus magnus and right interscapular muscles—so that the right scapula tends to drop by its weight—the right pectoralis major, and both sternomastoids.

The trapezii and the muscles of the back generally are thin. There is atrophy of the glutæi, especially on the left side.

The patient suffers occasionally from very severe spasms and pain in the epigastric region together with vomiting (gastric crises).

She complains that she is scarcely ever free from pain. There is a burning pain at the back of the neck and behind the ears, a heavy aching feeling across the forehead, and, in addition, she has very sharp and sudden pains coming and going in the skin at the top of the head.

Her arms feel as if they were being gnawed, and there have been sharp shooting pains in the course of the median nerves, leaving an after-feeling of soreness.

She has shooting pains in the lower part of the abdomen, and what she describes as a "dull" pain in the vagina. There is a feeling of fulness in the abdomen and of oppression over the lower half of the chest, which is so severe at times that it is as though suffocation were imminent.

She has a constant sense of starvation in spite of eating largely. Her own words are, "I always feel hungry—always feel starved. When at home I never seemed satisfied, and ate all sorts of things; in fact everything I could get hold of."

The tongue "feels hard," her throat is dry, and she has a sensation of choking. She can swallow without difficulty. There have been at times severe cramps in the left flank.

She has numb feelings and creeping sensations down the legs and thighs, with aching of the knees. If her legs are allowed to dangle, they feel as though they were being screwed off at the knees.

Seated in a chair she feels giddy and appears to lose all power in the back. A month ago she suffered from what is described as a "starting" pain (so sharp that she was compelled to cry out) in the front of both thighs, and at times a similar pain has come across the dorsum of either foot, causing the feet to start.

At 17 this girl contracted a chancre, which was followed by sore throat and copper-coloured rashes.

At 22, according to her account, she had a bad cold, after which she was always weary and could not rid herself of a tightness in the chest.

She had severe aching pain in the back and vagina, with frequent desire and imperfect ability to micturate. About the same time there was internal strabismus of a passing character.

Next she complained of burning sensations in her loins and her belly seemed large.

A few months later and her right eyelid began to droop, and she had giddiness and pains in the legs, "sharp like needles," causing her legs to jump. The right eyelid recovered its power, and then the left lid drooped. She went into a hospital and her legs began to fail her. She staggered as if drunk, the right shoulder wasted, and the right eyeball

became fixed, as it is now, and the left eyeball began to follow its example.

Next the right lid began to droop again and she gradually wasted. The pains have become more frequent and severe.

She was admitted into our hospital in January and discharged in May.

The treatment consisted in mercurial inunction carried to mild salivation together with iodide of potassium, in doses of 20 grains, three times daily.

Her pains were a good deal relieved by extract of Indian hemp, but beyond this she could not be said to have derived any particular benefit from the treatment, and she quitted the hospital in much the same state as that which she presented on admission.

Before making any comments upon this case it will be convenient if I describe the other one.

X. Y., æt. 36, served for 16 years in the army (10 years in India), and was invalided on account of heart disease.

He was married and had six children. In his family history there was nothing of note except that he had a sister who was paralysed.

For himself he had rheumatic fever ten years before his admission here, and had once suffered from gonorrhœa, but never to his knowledge from syphilis.

He was admitted into the hospital in 1880.

By his own account his illness had only begun in the preceding February, but it appeared on inquiry that for five years he had been subject to dragging "rheumatic" pains which were not sharp or sudden.

Not till six months before he applied here did he begin to have sharp, darting pains in the legs, and also in the fingers. They were very sudden, and described as being like "sparks of electricity" recurring through his limbs. About March he began to stagger in walking.

About two years previously he had begun to see double and had "twitching under the eyes." His sight had got bad since Christmas, but he was not aware that his eyes were fixed till his attention was called to it here. He had rapidly become

very deaf, and for three months there had been difficulty in swallowing. He had nearly choked on several occasions. He had never suffered from attacks of vomiting.

His state, as taken by Dr. Beevor in September, was as follows:—

Patient appears considerably older than his years. He has a dull, heavy, listless look which seems to be due to his eyeballs being motionless. The eyelids droop, the right more than the left. He can close them with equal ease apparently on either side, but in opening them as wide as he can he is unable to raise the eyelids to a normal extent, and the right still less than the left.

The eyeballs are rather prominent. They are completely fixed, the gaze being always directed straight forward, with a slight divergence of the optical axis. The right pupil measures 3·5 mm., the left 3 mm.

They are both insensitive to light, and they do not contract when he tries to look at a near object; but it is to be observed that he is unable to converge the eyes, owing to the paralysis of the external muscles of the eyeballs.

The masseter muscles contract firmly and equally. He cannot whistle, and says his food collects between his cheeks and teeth, especially on the left side. The tongue is protruded almost straight, slightly inclined to the left side.

He can swallow liquids pretty well, but takes a long time to swallow solids, especially if they are dry, like bread. Smell and taste are not apparently affected. Hearing on both sides is so bad that he needs to be shouted at; the degree of deafness varies. There is subjective sensation of booming of bells, especially in the right ear.

By the ophthalmoscope no change is to be noted in the optic discs. Can just read 8½ Snellen with either eye. Colour vision good. Distant objects sometimes appear double; near ones, never.

The skin of the forehead seems rather less sensitive to touch on the left than the right side, but there is no marked impairment of sensibility in the region of the fifth nerve. In the rest of the body it is perfect. The upper limbs are somewhat wasted about the shoulders and forearms, but not to any marked

extent. The movements of the arms are free, and the grasp 35 with the right, 30 with the left, hand.

The lower limbs are rather more wasted, and the calf muscles are especially flaccid. He has free movements with his legs, and considerable power in them when lying down. The wasting of limbs is of the nature of general emaciation, not of progressive muscular atrophy.

When his eyes are closed he staggers, and would fall. In walking he is very shaky, and especially when he turns. He does not bring his feet down with a stamp.

The patellar tendon reflex is absent on each side. There is no ankle-clonus. Sole reflex is present, but small on each side.

Cremasteric reflex absent; abdominal only imperfectly obtained; the epigastric is marked. The faradic excitability of the muscles of the thighs and legs is very slightly indeed below the normal.

There is no delay in the transmission of impressions of touch or pain, and he can localise correctly the slightest touch on the arms and legs.

The lungs are free from dulness. A few râles are heard in the interscapular region. At base of the heart a loud double see-saw murmur, the pulse 68, of water-hammer character. The brachial arteries at the elbow are tortuous, and roll under the finger. The urine contains no albumen nor sugar.

When this man had been about three weeks in the hospital he was taken one day (September 28th) with a fit of coughing, which was followed by vomiting.

He was then seized with intense dyspnœa. The face and lips suddenly became of a livid blue colour; the mouth, opened wide, gave exit to a moaning cry of very distressing character. He was immediately got into bed and propped up, whilst brandy was administered by enema.

After about twenty minutes he was able to speak, having gradually recovered from the dyspnœa. His mind appeared confused for a little time afterwards.

Next day his temperature was 100·6. There was no dulness in the chest, but râles were heard at the bases of lungs.

On Sept. 30th he began to have such difficulty in swallowing

that food was henceforth administered by enema. He gradually got lower. On Oct. 2nd the catheter required to be used. The pulse numbered 100, regular, but very compressible.

Respirations 28 in the minute. There were râles in the chest. On Oct. 3rd his temperature, which in the morning was 102·6, marked 104 in the afternoon, and in the evening 101·2. He became excited and delirious. Urine contained a little albumen. Next morning he appeared almost moribund; but answered questions, and was able to swallow egg and milk with brandy.

His morning temperature was 102·2, pulse 104. Respirations 40, very shallow.

There was no dulness to be noted in the chest, but râles continued at the bases of the lungs. During Oct. 5th he continued to sink, attacks of dyspnœa occurring two or three times in the day, and on Oct. 6th he died.

An autopsy was made nineteen hours after death. There was nothing noticeable about the dura mater or the general aspect of the brain. On removing the cerebrum some of the cranial nerves could not be found. The right third nerve was seen *in situ*, but the left escaped observation.

On dissecting the outer wall of the cavernous sinus, the third and fourth pairs could be made out, but not the sixth. The fifth, seventh, eighth and ninth nerves were seen.

The nerves which could not be discovered had, no doubt, dwindled so much in size as to escape recognition. Section of the spinal cord disclosed no change to the naked eye.

The right side of the heart was full of dark clotted blood. The left side contained some ante-mortem clots. The mitral valves appeared slightly thickened. The aortic valves were healthy in appearance, but the sinuses of Valsalva were much dilated, and the whole of the ascending aorta was dilated.

The spinal cord with the medulla oblongata and portions of the mesocephale were hardened and submitted to microscopical examination by Dr. Bevan Lewis.

The following is the account which he has been kind enough to give me of the results of his examination.

HISTOLOGICAL NOTES ON A CASE OF TABES WITH OPHTHALMOPLEGIA EXTERNA. By Dr. Bevan Lewis.

The Spinal Cord.

Sections taken indiscriminately from any region of the cord exhibit very advanced degenerative changes invariably limited to the posterior column. The lesion, which is of the nature of grey degeneration, is fairly symmetrical in its hemispheric distribution throughout the different regions of the cord; but there is evidence in the distorted marginal contour of these columns in the lumbar cord (due to sclerous shrinking and contraction) that the lower realms have been earlier implicated, as they exhibit a later stage of sclerosis that what obtains in the higher divisions of the cord. To the naked eye all transverse sections when stained display the posterior columns mapped out with extreme distinctness by the far greater depth of tint which they attain in contrast with that of the antero-lateral columns. Aniline staining in this respect may be most favourably compared with the carmine sections, and exhibit with even greater distinctness this morbid coloration due to the preponderance of the connective elements. Similar features are observed under the low powers of the microscope, and the nature of the change indicated by the great increase of intermedullary connective which, invading irregularly the different portions of the tissue, separate whole fascicles of unaffected medullated tubes from one another by the intervening deep-stained sclerous tissue. The latter consists not alone of intermedullary fibrillated connective, but of numerous multi-caudate cells, which differ only in number, size, and readiness of staining from the normal cells of the medullary septa supporting the vascular network of the cord. These morbid elements usually measured ·027 mm. × ·013 mm., were extremely irregular in size and contour, threw out numerous delicate fibrous prolongations, and were generally associated with large, dilated, and tortuous blood vessels, the walls of which were covered by proliferating nuclei. Throughout the whole commissural and radicular zones of the posterior columns, these

vessels appeared both more numerous and more dilated than was the case in the normal cord used for comparison with the morbid specimens.

The spider-like connective cells were far more numerous in the post-commissural than in the posterior radicular zones, and were very scanty or wholly absent in the columns of Goll, where the fine fibrillated intervening connective predominated as the morbid element. In all the minute medullated tubes, the wasting of the medullary sheath was revealed at a glance by the great disproportionate size of the axis-cylinder, whilst in those of wider calibre measurements gave for large numbers an axis-cylinder of ·006 mm., or ·009 mm. for medullated tubes of ·011 mm.—a very appreciable degree of wasting. In the more advanced foci of sclerous change the medullary sheath was entirely absent, and remains of axis-cylinders dotted the embracing zones of connective. The regional distribution of the lesion exhibited the following characters from above downwards.

a. Cervical enlargement.—The most extreme sclerous change was observed in the medullated zone bordering upon the posterior commissure (post-commissural zone), which appeared to have been a focus of the most intense degenerative alteration. To the finely punctuated appearance given by the normal axis-cylinders is here superadded a mosaic of deeper and fainter stained areas corresponding to sites more or less sclerosed, and with this is conjoined a coarse dotting of the field due to the great increase in numbers of deeply-stained connective cells, and the divided orifices of numerous coarse blood-vessels. Immediately behind the commissure the medullated fibres are almost universally reduced in diameter, and although a few tubuli are seen, which attain the average large diameter of this zone (·014 mm.), and a few exceptional ones even rival those of the columns of Turck (·027 mm.), still the vast proportion are extremely reduced, whilst extensive sclerosed tracts embrace the remains of axis-cylinders or the minutest medullated tubes. It was in this region, as before stated, that the connective cell and distended vessel predominated. Next to this post-commissural zone, the radicular zones are the parts most gravely implicated—the anterior series of radicular fibres

suffering more severely than those behind them, the integrity of which may be clearly traced throughout an extensive course. A somewhat large islet of nearly healthy tissue bordered the more anterior segment of these radicular zones (cols. of Burdach), but the posterior segments of the same were sclerosed throughout. The median columns (cols. of Goll) were degenerated to an *equal degree* throughout the whole or greater part of their extent.

β. Dorsal region.—Here, as in the upper realms, the most intense change was concentrated upon the large medullated elements bordering upon the posterior commissure, whence the sclerous tissue fringed the columns of Burdach on either side, implicating to a very serious extent the posterior radicular fibres. Far more universally involved and degenerated than in the cervical cord, these radicular fibres were often completely destroyed, the islets of healthy tissue being very few, and surrounded on all sides by large quantities of inter-medullary connective, branched cells, and dilated, tortuous vessels. The sclerous change has also invaded in this region of the cord the posterior cornua as far as the emergence of the posterior roots, the change being somewhat more marked on one side than the other, a statement which also applies to the posterior radicular fibres. A very limited patch of degeneration is found in the median district, which, on either side of the so-called posterior fissure, is otherwise wholly unaffected by the lesion.

γ. Lumbar enlargement.—Bordering the posterior fissure on either side are two riband-like bands of deeply-stained sclerosed tissue. By subsequent contraction of this tissue the two lateral halves of the posterior columns have been almost completely rent asunder, being connected only by a slight bridge of degenerated tissue near the posterior margin of the cord. These retracted sides enclose an elliptic space betwixt them of some size, so that the area of the posterior columns is greatly reduced in extent. A similar dense sclerous tissue follows out the posterior border of the cord, extending to within a very short distance of the posterior roots. Apart, however, from this morbid fringe along the raphe and borders of the posterior columns, we also find advanced degenerative changes in the post-commissural zone, similar in site and in character to what

was described in higher regions, stopping short at the radicular zones, the fibres of which are less implicated than in any other region of the cord. In the lowest part of the lumbar enlargement, on the right side, a peculiar cyst-like cavity, 1·5 mm. deep by ·75 mm. wide, exists in the posterior cornu. It has condensed sclerosed margins, and occupies the site of a large proportion of the vertical fibres of this cornu, which ascend in front of the substantia gelatinosa. The fibres (horizontal) of the latter arch round it on either side, but not completely surrounding it, and appear compressed together by the former contents of the cavity which have now escaped. A few of the vertical fasciculi still remain intact.

Antero-lateral columns.—A careful examination of these columns in the cervical, dorsal, and lumbar regions was made, and the results enable me to state positively that the (*a*) direct cerebellar tracts, (*b*) the direct and crossed pyramidal tracts, and (*c*) Gowers' tracts were free from any appreciable lesion, the histological elements being well displayed and perfectly normal.

Anterior cornua.—A similar statement may be made for the anterior horns of grey matter. The neuroglia had a perfectly normal aspect; the small ascending fasciculi of minute medullated fibres were well developed; the nerve-cells were plump, slightly pigmented, and exhibited neither quantitative nor qualitative change. Their marginal fibres for the motor roots were beautifully shown in all sections and free from any morbid change, nor in any region was the slightest tendency exhibited to extension of the lesion from the posterior cornua.

To summarise the changes observed:—

a. Most advanced sclerous change in the post-commissural zones throughout all the regions of the spinal cord alike.

b. Extension along the posterior radicular zones of a minor degree of intensity, and implicating the radicular fibres causing their destruction to a slight extent in the cervical, to a still less extent in the lumbar, but to a very grave extent in the dorsal region.

c. The columns of Goll through the cervical region were almost completely sclerosed, but the degree of degenerative change was less than in the above-mentioned regions.

d. Median segment of dorsal posterior columns scarcely affected.

e. Median raphe and posterior border of the same columns in the lumbar cord sclerosed and distorted.

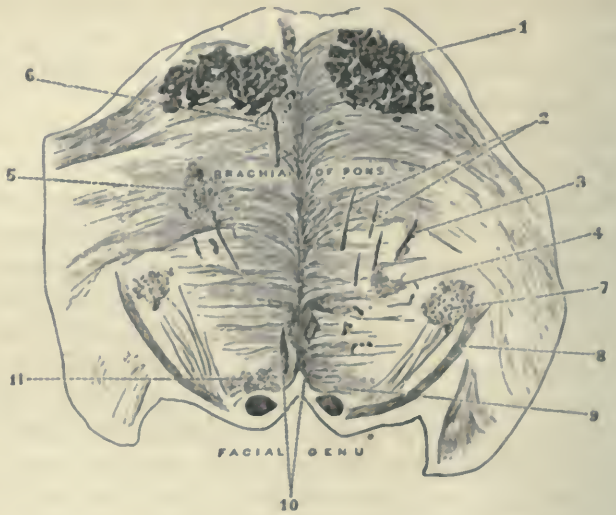
f. Small cyst-like cavity in gelatinous substance of posterior cornua in the lumbar region on the right side.

g. Implication of the posterior cornua by the same grey degeneration, but to a slight degree in both cervical and dorsal regions, extending usually to point of emergence of posterior roots.

h. In the sites of most advanced sclerosis (post-commissural and post-radicular zones) the size and number of the dilated blood-vessels formed a notable feature under a low objective.

i. Numerous amyloid bodies appear scattered throughout the posterior columns of most of the regions of the cord.

The pons and medulla.—Sections were carried through the nuclei of origin of the various bulbar nerves. Of those found along the lower half of the floor of the fourth ventricle—the accessory, hypoglossal, vagus and glosso-pharyngeal—the nuclei as well as the emergent or radicular fibres were found well displayed and perfectly intact. No diseased tract of tissue was anywhere apparent in the transverse sectional areas of this district, and the restiform columns and olivary nuclei and fibres were throughout free from lesion. Upon reaching the higher level of the origin of the sixth nerve, undoubted evidence was obtained of a morbid condition of this tract. The lesion was obviously a vascular one, and had secondarily implicated the emergent fibres from the abducens-facialis nucleus—the root fibres of the sixth pair. The nucleus, where it lies in front of the genu of the facial nerve, was represented by mere remnants of degenerated cells, and a punctated aspect of tissue from the resulting débris. The fibres of origin of the abducens, usually so clearly defined within the field embraced by the band of facial roots, were represented merely by an occasional narrow streak of minute and degenerated fasciculi taking their usual curved course, but lost to view after a short distance. Along the direction pursued by these diseased fasciculi were several large swollen and tortuous blood-vessels perfectly occluded by dark clots, their coats extensively diseased and frequently crowded by heaps of red blood cor-



1. Vertical fasciculi from crura.
2. Remains of roots of sixth nerve.
3. Diseased and plugged vessels covered with blood corpuscles.
4. Miliary extravasation.
5. Large focus of extravasation.
6. Blood-vessel in advanced state of disease, plugged.
7. Posterior nucleus and ascending roots of the facial.
8. Facial nerves.
9, 11. Abducens facialis degenerated on both sides.
10. Diseased and plugged vessel.

puscles. Along the course of the same fibres of the sixth pair are several miliary apoplexies, which have probably induced the degenerative changes here observed. Apart however from these foci of extravasation, we find also innumerable red corpuscles crowding certain parts of the field on either side of the median raphe; in fact the region of the abducens has been the seat of miliary apoplexies extensively induced by plugging of the nutrient vessels of this realm. In the rough sketch appended, I have endeavoured to localise a few of these diseased vessels and extravasations in a section taken at this level.

Level of Lemniscus.—Those vertical medullated fasciculi which limit the tegmentum in front, and are embraced by the most posterior fibres of the brachia of the pons, exhibit a notable increase of their connective elements. These fibres, associated with those from the fillet of the testes as far as the median raphe, have interspersed amongst them numerous islets

of sclerosed tissue, in which the remains of degenerated medullary tubes are well seen. The whole sectional area of the fillet lying in *front* of the tegmentum was thus involved, but the tracts of grey degeneration were not extensive, being scattered apart, not in circular patches like distinct focal lesions.

The upper realms of the pons and medulla had unfortunately incurred such damage during the process of preservation as to preclude an exhaustive examination of the nuclei of origin of the remaining oculo-motor nerves.

In the case of A. B. we have all the symptoms of tabes dorsalis. There are pains sharp and sudden in character, an ataxic gait, absence of knee phenomenon, loss of pupillary reflex, loss of muscular sense, some cutaneous anæsthesia, gastric crises. But beyond these symptoms there is another of rarity and interest. The symmetrical immobility of the eyeballs accompanied by more or less ptosis of the lids is a condition which was first described by Graefe under the name of ophthalmoplegia progressiva. An account of it is contained in the 'Lehrbuch der functionellen Nervenkrankheiten,' by Eulenburg (Berlin, 1871). I can remember that, nine or ten years ago, shortly after reading that account, I saw an example of the disease in St. Bartholomew's Hospital. The patient was a female, under the care, I think, of Dr. Andrew, and she had this symmetrical immobility of the eyeballs typically marked. The disease has more recently received a most searching investigation at the hands of Mr. Hutchinson, who has applied to it the term ophthalmoplegia externa,[1] in contradistinction to that of ophthalmoplegia interna, which he had given in a previous paper, to a state of immobility of the pupil, in which the internal muscles of the eye (the iris and ciliary muscles) are together involved in paralysis. He, however, explains that the former term must be understood as including very frequently, if not usually, the immobility of pupil.

Mr. Hutchinson's description of the condition is so graphic that I cannot possibly do better than quote it here:—"Drooping of the eyelids," he writes, "so as to give to the face a half-

[1] 'Transactions Roy. Med. Chir. Soc.' 1879.

asleep expression, is usually the first symptom, and it is soon accompanied by weakness of all the muscles attached to the eyeball, so that the movements of the latter become much restricted, or even wholly lost. The condition is usually bilateral, though it is not always exactly the same in degree on the two sides. Its symmetry probably denotes that it is of central origin. It by no means always happens that all the ocular muscles are alike affected, or that they are attacked simultaneously, still it is a very marked feature of the malady that the muscles fail in groups and not singly." Seventeen cases of the kind are described in the paper. In ten of these it seemed certain that syphilis was the cause; in eight acquired, and in two inherited. Of the remaining seven Mr. Hutchinson remarks, "it may be said that a reasonable suspicion of syphilis might be entertained in several." In one case an examination of the brain after death was made by Dr. Gowers, who found that degenerative changes precisely similar to those seen in progressive muscular atrophy had implicated the origins of the third, fourth, and sixth nerves as well as, in that particular instance, the optic nerves also. The large nerve-cells had disappeared from the nuclei of origin of these nerves.

Mr. Hutchinson remarks that we have in such cases a very close parallel to the so-called bulbar paralysis—the labio-glosso-laryngeal paralysis of Duchenne—and he thinks it may be plausibly conjectured that the initial lesion is inflammation of the nuclei of the affected nerves, which, in a slowly serpiginous manner, creeps from place to place along certain definite anatomical paths. In my female patient the history of syphilis was clear and certain. On the other hand, there is no evidence that X. Y. had ever suffered from the infection. The condition of his aorta is a suspicious circumstance, but the history of rheumatic fever prevents us from laying stress upon the condition as indicative of constitutional syphilis. In any case, however, the proximate cause of the lesions of nuclei must be sought for in an extensively diffused disease of the vascular system, of which there is evidence in the dilatation of the aorta, the rigidity and tortuosity of the brachial arteries, no less than in the microscopical changes in the intrabulbar arterioles with the resulting miliary hæmorrhages.

In A. B. it will be observed there is a symptom which (as it happens) is not described as occurring in any of those related by Mr. Hutchinson. I refer to the atrophy of muscles about the back and shoulders. This association, however, evidently tends to support the view that the lesion of the nuclei of origin of the cranial nerves is probably of the same nature as that which determines progressive muscular atrophy. In Charcot's lateral amyotrophic sclerosis there is atrophy of the large motor cells in the anterior cornua of the cord. The disease tends, as we know, to travel upwards, and in time spreads to the medulla oblongata. Attacking there the nuclei of the hypoglossal, facial, and trigeminal (its motor portion), it causes death by involving also the nucleus of the vagus. Now, it seems probable that, were it not for the fatality necessarily attaching to the destruction of this nucleus, the disease would also, in those cases, be likely to invade the nuclei of origin of the oculo-motor nerves, and so bring about the condition of the eyes which is to be seen in this girl. Death, in fact, stops the progress of the disease before it invades the region devoted to the innervation of the external and internal muscles of the eye.

The lesion of nerve nuclei would appear, in the case of A. B., to be a more or less continuous one, from the intracranial centres for the higher cranial nerves to the anterior cornua of the cord. On the right side the origin of the facial nerve is involved, though not apparently on the left. The quivering of the tongue shows that the nucleus of the hypoglossal is probably not intact.

In the atrophy of the sternomastoids and left trapezius there is evidence of invasion of the nuclei of the accessory.

I have but little doubt that the gastric crises (as I have some time ago suggested[1]) depend upon irritation of the nucleus of the vagus by sclerosis. This opinion has since received support by the result of an observation of Pierret, who found in a case of tabes, with characteristic gastric crises, sclerosis of the fasciculus gracilis in immediate relation with the nucleus of the vagus. The fibres of the vagus and accessory are in such close proximity at their point of origin that chronic inflammation of

[1] Pathological Society, February, 1880.

the immediately adjacent tissues may be expected to cause both sensory and motor disturbance-pain in the stomach and vomiting.

The symptom of intense hunger, of which this girl complained, is one that I have on several occasions observed in connection with the occurrence of gastric crises in tabes. It was marked in a case of tabes with gastric crises and joint affection under the care of my friend, Mr. Herbert Page.

It is present also in the case of a man, suffering from tabes with gastric crises, who is now under my care in the hospital.

Only a few weeks ago, whilst looking through 'A System of Clinical Medicine,' by Dr. Graves, published in 1843, I chanced upon a description of gastric crises which is so graphic that I cannot refrain from quoting here a rather full abstract of the case narrated. Viewed by the light of our increased knowledge, the case is evidently one of tabes dorsalis, but it occurred long before this disease was differentiated from other forms of paraplegia. The autopsy, which included examination of the brain, spinal cord and nerves, besides the abdominal viscera, naturally failed to throw light upon the cause of the disease; for in those days the methods of hardening preparations of nerve substance, the initiation of which we owe to Lockhart Clarke, had not been devised, and the examination, as far as I can judge, was only made with the naked eye. Dr. Graves appears, however, to have been curiously near the true solution in a remark which he makes. "Are we to attribute," he writes, "this diseased condition of the stomach and bowels, which, from the remarkable periodicity of its occurrence, was evidently functional, to irritation, congestion, or inflammation of the brain or spinal marrow? From the data we are in possession of it appears that this question must be answered in the negative." He speaks of the case as a very remarkable one, which had made a great impression upon him, and, in his view, well worthy of the attention of the pathological inquirer.

The patient, Mr. B., aged 23, was exceedingly strong, and passionately fond of hunting, fishing and shooting. These habits, however, he laid aside after the occurrence of the first attack of his illness, which happened in 1829. From that time

his bowels, previously sluggish, became inclined to looseness, which always increased before the appearance of one of the attacks, accompanied by griping, nausea and inclination to vomit. Each attack was generally preceded by a copious secretion of insipid watery fluid in the mouth, and then the characteristic symptoms of his disease commenced. These consisted in obstinate and protracted nausea and vomiting. He first threw up whatever happened to be on his stomach at the time, and afterwards everything he swallowed, whether solid or liquid, and the quantity ejected in the course of a day varied from three to four quarts of fluid. He complained also of pain, referred to the stomach or lower part of the chest, which continued throughout the attack, being most acute at its commencement; for the last year this sensation had passed into a feeling of painful constriction, which he described as a "contracted feeling of his inside," and compared it to something like the effects of a cord drawn tightly, so as to compress or strangulate his body exactly along the outline occupied by the insertions of the diaphragm. During the prevalence of the attack he had profuse perspirations, particularly towards the termination of each paroxysm. The duration of the first attack did not exceed four or five days, after which he became quite well, and continued so for six or seven months, when his symptoms suddenly returned. He began to reject everything from his stomach as before, but in the course of a few days the vomiting disappeared, and for a considerable interval he had no return of his complaint. In the year 1830 he had three attacks of a similar description; from these he recovered also completely, and without remarking any diminution of power in his lower extremities. In 1831, however, the disease began to assume a more serious aspect; the paroxysms became much increased in severity, lasted longer and recurred at shorter intervals. For one of these attacks he took mercury, and was salivated. In 1832 his symptoms became still more violent, and the duration of the paroxysms more protracted. He had one in March, a second in May and a third in June, each of which was accompanied by some numbness and loss of power in the lower extremities; this, however, was slight, and disappeared altogether as the vomiting subsided. About this time

he noticed that his urine was scanty, and deposited more sediment than usual. He also complained of being very apt to catch cold whenever he got out of bed, and stated that he suffered occasionally from *severe twitches and pains in his legs, thighs, arms and other parts of his body*, which were generally succeeded and carried off by profuse perspirations.

In August, 1832, he had a violent attack, which lasted nearly a month. The vomiting was incessant, continuing night and day, and he suffered severely from the feeling of painful constriction already described. On getting up after this attack his legs suddenly failed him, and he dropped down on the floor quite powerless. The paralysis did not now disappear during the intervals, although it grew somewhat better after each fit of vomiting had ceased; indeed he used to improve in his walking after the paroxysm had entirely disappeared, and, aided by two sticks, supported himself so as to give some hopes of a recovery, until a recurrence of his attack reduced him again to a state of almost total paraplegia. His legs now began to waste sensibly, and he noticed that they had lost their feeling and were remarkably cold. He also complained of severe twitches of pain in various parts of his body, accompanied by profuse night sweats, and turbid scanty urine.

For some months before his death he was completely paraplegic, and continued to be attacked with violent fits of vomiting. The vomiting went on night and day, and he was unable to retain the mildest and most soothing substances for a moment on his stomach. Everything was tried to allay the irritability of the stomach, but in vain. After continuing to resist obstinately every form of treatment for five or six days and nights the vomiting would suddenly cease, the gentleman would exclaim, "Now I am well," and he could then eat with perfect impunity substances of an indigestible character. The transition from a state of deadly nausea and obstinate retching to a sharp feeling of hunger used to occur quite suddenly. One hour he was a miserable object, rejecting everything, and suffering the most painful constrictions across the epigastrium, the next found him eating with a voracious

appetite whatever he could lay hold of, and digesting everything with apparent facility.[1]

Here again we have the symptom of hunger described very graphically. I would submit that it tends to corroborate the view that the seat of lesion occasioning gastric crises is in the neighbourhood of the pneumogastric nucleus.

It may be asked why, if we have to do here with irritation of this nucleus, we should not, in cases of tabes with gastric crises, also meet with symptoms referable to the heart and to the respiratory apparatus. We do, in fact, sometimes meet with laryngeal crises, and rapidity of pulse is of no unfrequent occurrence in the course of tabes dorsalis. It does not seem reasonable, however, to expect that in these circumstances we should necessarily find evidence of impairment in all the functions subserved by the nerve.

It cannot be doubted, I think, that there is a very elaborate differentiation of functions of the nerve-cells constituting a nucleus. In a paper of great interest and importance[2] Dr. Felix Semon calls attention to the proclivity of the abductor fibres of the recurrent laryngeal nerve to become affected sooner than the adductor fibres, or even exclusively, in cases of undoubted *central* as well as peripheral injury or disease of the roots or trunks of the pneumo-gastric, spinal accessory, or recurrent nerves.

The differing amount of paralysis in the muscles of this girl's eyeballs and lids is also indicative of differentiation of function in various parts of the nuclei of the oculo-motor nerves.[3]

I do not think, therefore, that we shall see anything remarkable in irritation of the nucleus of the vagus in a particular case being followed by symptoms entirely referable to the stomach or intestines. Whilst upon this point I would remark that, in certain cases of tabes where the more typical form of gastric crises has not been marked, there has been,

[1] Several cases of typical gastric crises will be found described in my paper on "Certain Little Recognised Phases of Tabes Dorsalis," Transactions of the International Med. Congress, 1881.

[2] See 'Archives of Laryngology,' vol. ii. No. 3, July, 1881.

[3] Dr. Sturge has published some interesting remarks on this subject. "Two cases of simultaneous paralysis of both third nerves."—'Ophthalmological Society's Transactions,' vol. i.

nevertheless, a peculiar tendency not so much to diarrhœa as to abnormally frequent action of the bowels, the evacuations not being necessarily loose. I think it probable that this symptom, like the pain in the stomach and vomiting, may prove to depend upon irritation of the nucleus in question, for it must be remembered that the peristaltic action of the small intestine is largely influenced by nervous impulses passing along the splanchnic and vagus nerves. According to Pflüger, whilst stimulation of the splanchnic nerves tends to check the peristaltic movements, that of the vagus is calculated to excite them.[1]

In reference to this question of the dependence of gastric crises (of the two kinds which I have mentioned) upon sclerosis in the neighbourhood of the nucleus of the pneumo-gastric, I would call attention to a point which, although of purely negative character, seems to me to be of considerable importance.

The man X. Y. did not suffer at all from gastric crises. It will be observed that Dr. Bevan Lewis in this case found the nuclei of the vagus amongst other nerves well displayed and perfectly intact, and he adds, "no diseased tract of tissue was anywhere apparent in the transverse sectional areas of this district." The importance of this observation will be especially evident when it is remembered that the funiculus cuneatus and gracilis, which closely adjoin the sensory portion of this nucleus, represent the continuation upwards to the cerebellum through the medulla oblongata of the posterior columns of the spinal cord.

Let me note here an important difference in these two cases. The muscular atrophy which affects in a marked manner the trunk and extremities of the patient A. B., was entirely absent in the case of the man X. Y. The microscopical findings corresponded with the clinical history. (I ought here to say, perhaps, that I purposely kept Dr. Bevan Lewis in ignorance of the symptoms observed during life, in order that his observations might be quite unprejudiced.) Dr. Lewis reports that the anterior cornua of the spinal cord were entirely free from any morbid change.

[1] 'A Text-book of Physiology.' By M. Foster. 3rd edition, p. 267.

It is interesting to observe that Mr. Hutchinson found "in six of his cases the lower extremities more or less weak, and a condition approaching more or less closely to locomotor ataxy," and he makes the important remark, that "there can be no doubt that ophthalmoplegia externa is sometimes a part of the general malady known as progressive locomotor ataxia, especially when that disease is due to syphilis." The two instances which I have described are examples, at all events, of the first described association. Both patients presented the most characteristic symptoms of tabes dorsalis. In each there were "lightning" or "electric" pains, absence of knee phenomenon and ataxy. No other signs need mention in the presence of these.

ON THE CONDITION OF THE KNEE-JERK, ANKLE-CLONUS, AND PLANTAR REFLEX AFTER EPILEPTIC FITS IN SEVENTY CASES; AND ON POST-EPILEPTIC CONJUGATE DEVIATION OF THE EYES.

BY CHARLES E. BEEVOR, M.D. LOND., M.R.C.P.,

Late Resident Medical Officer to the National Hospital for the Paralysed and Epileptic.

THE presence of ankle-clonus and excessive knee-jerk (patellar tendon reflex) after epileptic fits was first pointed out by Dr. Hughlings-Jackson in a case of unilateral convulsions, which was recorded by him in the 'Medical Times and Gazette' for February 12th, 1881.

While I was Resident Medical Officer at the National Hospital for the Paralysed and Epileptic, Queen Square, I was asked by Dr. Hughlings-Jackson, and also by Dr. Gowers, to make observations as to the presence of ankle-clonus and the knee-jerk after epileptic seizures, and the following results were obtained by personal observation of seventy fits occurring in thirty-one different patients. I have to thank the physicians under whose care these cases were, for allowing me to record the following observations.

To obviate as much as possible any source of error, I took myself, or dictated, notes at the time, and directly afterwards copied them out in full. The knee-jerk was tested with a heavy vulcanite stethescope, with a ring of india-rubber round the disc, as advised by Dr. Buzzard, instead of trusting to the rougher method by striking with the edge of the hand.

The plantar reflex was tested by a sharp quill-pen, and the ankle-clonus was tried with the knee at various angles, as it

is obtained best with different degrees of tension of the calf-muscles.

The cases have been divided into those which were observed *instantly*—where the whole of the fit was observed, or at least the end of the clonic stage was witnessed—and into those which were seen *directly*, where perhaps 1 to 2 minutes might have elapsed, but always before the patient showed any signs of returning consciousness. The time at which the various phenomena showed themselves or disappeared was taken by a watch, and the probable duration was not roughly guessed at.

The cases may be arranged in a table:—

	Seen instantly after Clonic Stage.	Directly after Clonic Stage.	Total.
Knee-jerk increased and ankle clonus present	27	11	38
Knee-jerk diminished, ankle clonus absent	6	7	13
Knee-jerk absent, ankle clonus absent	3	8	11
Knee-jerk normal, no ankle clonus	4	1	5
Knee-jerk diminished, ankle clonus present	2	0	2
Knee-jerk normal, ankle clonus present	1	..	1
Total	43	27	70

In all the above cases the phenomena were obtained on both legs.

In all these cases the plantar reflex was absent instantly after the clonic stage, but it returned in from 3 to 13 minutes, the average time being about 5 minutes; the time at which the ankle-clonus passed off was also noted by a watch, and seemed to correspond to the time of return of the plantar reflex, and in 10 out of 15 cases in which the times of the disappearance of the ankle-clonus and the appearance of the plantar reflex were taken, it was found that the two coincided approximately.

In one of the cases seen instantly, in which the knee-jerk was increased, there was no ankle-clonus.

In one of the cases in which the knee-jerk was diminished

and there was no ankle-clonus when tried instantly, in 6 minutes the former became excessive and the latter was also well obtained; and in two cases where the knee-jerk was diminished, when observed directly after the clonic stage, it was found to be increased in a few minutes.

Of the three cases where both phenomena were looked for instantly and were absent, one was doubtful, and in the eight observed directly two were doubtful, and in one the knee-jerk was present in 5 minutes' time, while in another both phenomena were very well obtained in 8 minutes. The two apparently anomalous instances of the presence of ankle-clonus and diminution of the knee-jerk were observed twice in the same patient.

In all the above cases, the fits were ordinary bilateral epileptic attacks, beginning as a rule with rotation of the head and turning of the eyes towards one side, and in some of the attacks the ankle-clonus and the knee-jerk were decidedly more active after the clonic stage on the side to which the initial rotation of head and eyes occurred.

In the intervals of ordinary bilateral attacks the knee-jerk is usually obtained on both sides more readily than normally, but there is no ankle-clonus.

Besides the foregoing observations on the knee-jerk and ankle-clonus after epileptic fits, I have also had the opportunity of observing another phenomenon, viz., conjugate deviation of the eyes occurring instantly *after* the clonic stage of the fit is over.

A short notice of this phenomenon was given in the 'British Medical Journal' for January 21st, 1882; but I propose to give a fuller account of it in this paper. The explanation of it will, I believe, be better obtained by drawing an analogy between this phenomenon and the conjugate deviation of the eyes which is a known symptom in some cases of recent hemiplegia.

In cases of hemiplegia, after a recent lesion in one hemisphere, the symptom of conjugate deviation of the eyes and rotation of the head to one side is frequently noticed, by which it is meant that the head and eyes are turned towards the same side as that half of the brain in which the lesion is situated, and consequently away from the side of the limbs and face which are the seat of paralysis, so that a lesion in the

left hemisphere causes paralysis of the right side of the body, and conjugate deviation of the eyes and rotation of the head to the left. This symptom has been explained by the theory that the muscles of the eyes on the paralysed side—the right in our supposed case—are unable to antagonise and prevent the healthy muscles from drawing the eyes in a parallel manner towards the unaffected side, i.e. the left. I have not been able to find out that any previous observations have been made with regard to the presence of conjugate deviation of the eyes *after* epileptic fits. My attention was first drawn to the point by a case of Dr. Gowers' that I saw at the patient's own home; the man had had thirty or forty fits in about twelve hours, and when I saw him he had marked conjugate deviation of the eyes and rotation of the head to the left; the fits, his friends assured me, began with turning of the head and eyes to the right and the convulsions were right-sided. As soon as the fit was over the head and eyes turned to the left and remained there till the next fit, when they again turned to the right, to again assume the position to the left after it. It then occurred to me that I ought to find conjugate deviation of the eyes or rotation of the head in ordinary bilateral epileptic fits, for one side of the body is nearly always affected more than the other. I have looked for these phenomena in every case since, and I have taken notes at the time to obviate any source of error.

In an ordinary epileptic fit the tonic stage usually (if not invariably) begins by rotation of the head and turning of the eyes to one side—say to the right—and sometimes with flexion of the elbow and wrist of the same side as that to which the head turns. This tonic spasm is followed by clonic spasm, in which, perhaps, the two sides of the body are convulsed apparently equally. Directly all movement has ceased, and the patient lies with the limbs relaxed, the eyes will be noticed to roll slowly over to the opposite side—which in our supposed case would be to the left—and they remain there from ½ to 2 minutes. In thirteen consecutive cases where this post-epileptic deviation has been looked for, I have noticed it in eleven, and in one case the eyes have simply gone back to their natural position and have not passed to the opposite

side; in only one case have the eyes remained in the same position in which they turned in the primary tonic spasm, and this was a very slight fit. The *head* either remains in the same position as it assumed during the attack, or returns to the front. I have not yet seen the head rotate to the opposite side, after the end of the clonic stage, in universal bilateral fits, though I have witnessed it in a unilateral attack.

It is known that paralysis (either transient or permanent) occurs after some epileptic attacks, and most commonly in unilateral fits; the presence of any paralysis in an ordinary epileptic fit cannot be ascertained, for the reason that, by the time that the patient has recovered sufficient consciousness to use the dynamometer, the paralysis has passed off. I believe that the presence of the conjugate deviation of the eyes shows that the side where there is the primary discharge, and on which, presumably, the spasms are more severe, becomes more exhausted than the side where there is less discharge, and the eyes are drawn to the side where the muscles are the less exhausted. The head does not rotate, for the reason, I believe, that there is not sufficient difference of power between the two sides to affect this, while the eyes being very mobile are very exact indicators of the difference in the paralysis of the two sides.

In one patient of Dr. Ramskill's, the head and eyes first turned to the right, she was then clonically convulsed on both sides of the body, and while the clonic spasms were continuing the head and eyes went to the left; after all movement had ceased, the eyes and head rotated back again to the right and remained there for a few seconds; the head then turned straight and then the eyes rotated from side to side, keeping parallel; thus seeming to show that the side on which the discharge last occurred was the more paralysed directly after the clonic stage, and not necessarily the side where the primary discharge took place.

Another phenomenon which I have observed is, that, after the conjugate deviation has lasted for ½ to 2 minutes, the eyes sometimes—for 10 minutes—roll slowly from side to side, keeping parallel, and they also seem to wait longer on the side towards which they first conjugately deviated; during this time the pupils frequently oscillate. Perhaps this may be

explained by an irregular return of power and recovery from exhaustion in the two hemispheres of the brain, the one predominating over the other in turn. I have observed this rolling of the eyes from side to side in about half the cases in which it was looked for.

The foregoing post-epileptic phenomena, I think, seem to show that there is a great similarity between the state immediately following an epileptic fit and the condition which is found in a case of recent hemiplegia, a fact which has frequently been pointed out before in unilateral fits, in which temporary paralysis of the affected side is not uncommon. I believe the same analogy can be applied to *all* bilateral epileptic fits; for in these last there is usually, instantly after the clonic stage, deviation of the eyes, increased knee-jerk and ankle-clonus, and absence of the plantar reflex, and it seems probable that the same condition which produces permanently in cases of hemiplegia these phenomena, viz. excessive knee-jerk, ankle-clonus, and absence of superficial reflexes, is also present, though for a much shorter period, i.e. for a few minutes, directly after epileptic attacks, and when these are bilateral the above phenomena are obtained on both sides, with the exception that the post-epileptic deviation of the eyes occurs towards the side of less paralysis.

It would seem, therefore, that, after an epileptic seizure, the brain and spinal cord are reduced, probably by exhaustion due to the excessive motor discharge, to the condition which is obtained in cases of hemiplegia, for in both there is usually excessive knee-jerk and ankle-clonus and absence of plantar reflex. In reference to the latter, I have had the opportunity of observing, in a case mentioned by Dr. Gowers in his work on epilepsy, where the patient was clonically convulsed on the right side for 20 minutes (by the watch), that the knee-jerk was more marked instantly after on the right than on the left, and the plantar reflex was obtained on the left side in 3 minutes after all movement had ceased, but that on the convulsed side—the right—it was only obtained slightly in 30 minutes' time, and in this case there was paralysis of the right side, from which he did not recover till an hour or two later, speech being also affected.

THE BRAIN OF A CRIMINAL LUNATIC.

BY DAVID FERRIER, M.D., F.R.S.

THE brain figured and described in this paper was put into my hands for examination by Dr. Nicolson, of Broadmoor Criminal Lunatic Asylum.

It was the brain of a woman who in 1871 murdered two of her children, but who, on being arraigned on a charge of murder, was found insane and incapable of understanding the charge against her. She was therefore ordered to be confined during Her Majesty's pleasure.

From the notes supplied by Dr. Nicolson, the following are the main facts which were ascertained in regard to the woman's history and condition:—

She was forty-four years of age at the time of her death, which occurred in November 1881. Her mother states that she was very intelligent as a child and young woman, was well educated for one in her position, and married a labourer when she was thirty years of age.

After the birth of her first child she became paralysed on the right side and aphasic. This was about 1868, the exact date not being obtainable.

In October 1871 she strangled her two children, aged two years and eight months respectively.

In March 1872 she was sent to the Criminal Lunatic Asylum at Broadmoor, under the circumstances already mentioned.

On admission she was hemiplegic on the right side, the right arm being described as almost powerless, but she was able to use the right leg feebly in locomotion without any assistance, though, as a rule, she helped herself forward with the left hand.

The right side had not lost sensibility, but she complained of the whole of this side being cold, and she always kept it specially wrapped up on this account.

For some months after her admission she was subject to flushings, faintness, and occasional convulsions of a slight character accompanied by vomiting.

Towards the end of 1873 epileptiform convulsions became more marked, and she had repeated attacks of dulness and torpor of intellect, with flushing of the face.

Fits continued with more or less prolonged intermission, and she had a series of severe attacks in May 1877. In the severe fits the convulsions were of a general character. Her head and eyes were drawn to the right, and there was complete insensibility. In the slighter fits the eyes were directed to the right, with momentary unconsciousness, but there was no general muscular spasm. From May 1877 up to the time of her death there were no severe fits, but she showed from time to time twitchings of the facial muscles.

She had repeated attacks of pyrexia and flushing, with pain in the head, especially localised in the left temple. The pain was always relieved by a few leeches to this spot, and she usually brightened up after their application, and would signify her desire to have leeches applied when the pain was severe.

Her disposition was very fitful and uncertain. As a rule she was well-disposed and almost cheerful, and frequently exhibited much gratitude for any services done her.

On many occasions, however, she was petulant, irritable and cross with the other patients. When in this state she was quite silent, and sometimes mischievously tried to break windows. Sometimes she was very melancholic and refused food for days. A few months before death she was found attempting to injure herself by dashing her head against a water-tap.

Her conversation was of a jerky, monosyllabic character, and always an evident effort to her. She would frequently forget the word she wished to use, but could indicate by gesticulation when the right word was named to her.

If anything she wished for were granted to her, her face lit

up and she would say, "Thank you kindly, mum," (and then correcting herself), "sir." She could not always correct herself, and then appeared confused. She made use of certain stereotyped phrases, and the usual conversation might be given as follows:—

"Good morning, Mrs. P." *A.* "Goo' mornin', mum,—sir." "Any pain to-day?" *A.* "Yes; pain—always pain." If she did not feel well she would say "bad," and then pointing to her right side, "Cold all down." If more than usually depressed she would say, "Wish were dead!"—"Paralsee long time, 'ome two year, Bo'moor eight." She would then burst into tears and sob piteously.

In July 1881 she became very obstinate and silent, and refused food. At this time her respiration became difficult, and there were marked signs of phthisis in both lungs, especially the right; œdema of the legs set in, and she gradually failed, and died in November 1881.

On post-mortem examination the lungs were found extremely tubercular, numerous cavities existing in the right, which was almost entirely disorganised.

The skull cap was dense, but there was no apparent want of symmetry on the two sides.

Condition of the Brain.

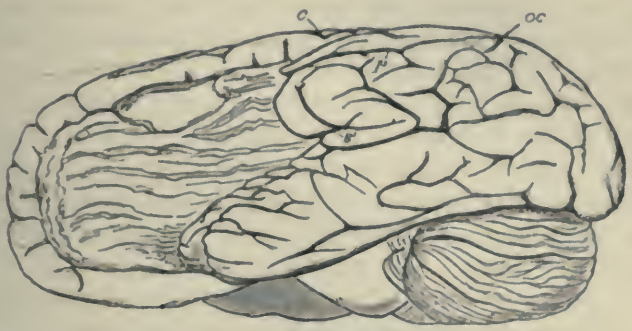
The *right* hemisphere was well developed and normal in the arrangement of the fissures and convolutions.

The *left* hemisphere had a strikingly abnormal aspect.

The general outline was smaller than that of the right, so that viewed from the left, as seen in Fig. 1, the internal surface of the right hemisphere was visible in the frontal, parietal and sphenoidal regions. The occipital surface, however, was entirely covered by the equally well-developed occipital lobe of the left hemisphere.

The anterior half of the left hemisphere was represented by an empty cyst (which was full of fluid at the time of removal), the walls of which were collapsed and puckered. The posterior boundary of this cyst was formed by a line drawn almost perpendicularly from the upper extremity of the fissure of

Fig. 1.



Rolando (indicated on the right hemisphere by *a*, Fig. 1) into the horizontal limb of the fissure of Sylvius, which formed the rest of the boundary. The posterior extremity of the fissure of Sylvius (*s'*, Fig. 1) was still visible. In advance of the line indicated, the whole of the cortex and medulla was entirely deficient, both on the convexity and on the orbital aspect of the frontal lobe.

The island of Reil was also entirely absent.

On the internal or median aspect of the frontal lobe, however, the gyri and sulci were distinct and normal in arrangement, though somewhat smaller than on the right.

The *gyrus fornicatus* was normal from its commencement at the anterior perforated space to its posterior termination in the *gyrus hippocampi*, which with its *uncus* was well developed. Above the calloso-marginal sulcus the marginal aspect of the hemisphere was formed by the median surface of the superior frontal convolution and the paracentral lobule, not so well developed, however, as on the right. The *præcuneus* or quadrilateral lobule, the *cuneus*, the internal aspect of the occipital lobe, and the occipito-temporal convolutions were all of normal appearance.

On the convex aspect of the posterior half of the left hemisphere which remained, the parieto-occipital fissure (*o c*, Fig. 1) was well marked, and extended fully half an inch outwards from the longitudinal fissure. The convolutions of the occipital lobe posterior to this fissure, and its imaginary continuation to the *incisura præoccipitalis*, were well developed.

Between this line and the boundary of the atrophied region, the convolutions and fissures had suffered considerable displacement, owing chiefly to the great diminution of the postero-parietal or superior parietal lobule.

The intra-parietal fissure (Fig. 1) was found to commence at the posterior boundary of the cyst and to pursue a course almost parallel to the median fissure, the greatest distance between the two being only 1 centimetre. The distance anteriorly was rather less, as at this point the lobule tapered somewhat.

The postero-parietal lobule included between the longitudinal fissure and the intra-parietal sulcus was thus, as compared with that of the right hemisphere, greatly reduced in size. On the sound side the greatest width between the longitudinal fissure and the intra-parietal sulcus was 4 cent., and nowhere less than 2 cent.

The posterior or upper extremity of the horizontal limb of the fissure of Sylvius (*s'*, Fig. 1) was situated 2 cent. below the anterior extremity of the intra-parietal sulcus.

The portion of the cortex remaining between these two points corresponded to the anterior extremity of the angular gyrus or portion of the supra-marginal lobule. This was continuous round the extremity of the fissure of Sylvius with the superior temporo-sphenoidal convolution, of which only 2 cent. of the upper extremity remained. The rest was atrophied and its position taken by transverse gyri, which ran obliquely from the middle temporo-sphenoidal convolution into the fissure of Sylvius. The angular gyrus was somewhat smaller than on the right side. The upper or posterior half of the middle temporo-sphenoidal convolution was normal. The lower half was atrophied and sloped obliquely forward into the fissure of Sylvius.

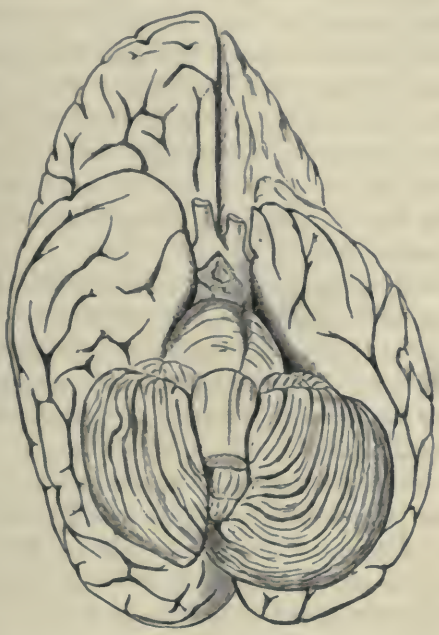
The inferior temporo-sphenoidal convolution was normal.

It will be seen, therefore, that there was *total absence* of the *superior frontal* (median aspect excepted), *middle frontal*, and *inferior frontal* convolutions, the *island of Reil*, the *ascending frontal* and *ascending parietal* convolutions. Also of the *anterior three-fourths of the superior temporo-sphenoidal*, and *anterior half of the middle temporo-sphenoidal* convolutions.

In addition there was *considerable atrophy* of the *postero-parietal lobule*, and to some extent also of the angular gyrus.

The *base of the brain* had a remarkable appearance (Fig. 2).

Fig. 2.



The median fissure was bounded on the left by the internal margin of the orbital lobule, but all external to this looked as if it had been sharply cut off.

The anterior extremity of the temporo-sphenoidal lobe of the left side, instead of the rounded full appearance of the right, was somewhat flattened and sloped obliquely into the Sylvian fossa.

The optic chiasma and optic tracts had a skew appearance, the right being anterior to the left. The corpora mammillaria were similarly displaced, the left being considerably posterior to the right.

The right *crus cerebri* stood out prominently, but the left was sunk and invisible except when the parts were separated. The left optic tract had a greater obliquity than the

right, and was visible to a greater length in its course round the crus.

The foot of the left crus cerebri was atrophied in its inner two-thirds.

The locus niger was only 1 m. from the surface, except at the outer third, where the fibres of the foot formed a layer of 5 mm. The depth of the locus niger of the right crus from the surface was throughout fully 5 mm.

A marked asymmetry was visible in the *pons Varolii*.

The groove indicating the middle line had a tortuous direction, owing to the great flattening and obliquity of the left half. On cross section the pyramidal tracts were of a greyish tint and sclerosed.

The left olivary body appeared more prominent and nearer the middle line of the medulla oblongata than the right.

This was owing to the almost complete disappearance of the left pyramidal tract. The right pyramidal tract at the middle of the olivary body had a breadth of 5 mm., while the left measured only 2 mm.

The left lobe of the *cerebellum* was of normal size, but displaced, so that it projected across the middle line and rested on the under surface of the right occipital lobe. This was due to atrophy of the right lobe of the cerebellum, which was reduced to about half the size of the left lobe.

The cortical laminæ were thin and the leaflets of the *arbor vitæ* smaller than those of the other side.

Microscopical preparations of the cerebellum made for me by Dr. Scott, and taken from the cuneiform lobe of each side, including portion of the nucleus dentatus, exhibited a remarkable degree of atrophy of the cortex of the left side.

The outer or grey layer was greatly reduced in thickness and Purkinje's cells had entirely disappeared, in some leaflets entirely, while in others only portions had become so atrophied.

Dr. Scott's measurements of twenty specimens gave the average thickness of the grey layer as 3·3 thousandths of an inch, while on the sound side this layer measured 15 thousandths of an inch and exhibited the cells of Purkinje in a beautiful manner.

The granular layer was also greatly reduced. The average thickness of this layer on the sound side was 7 thousandths of an inch, whereas in the atrophied regions of the left cortex the thickness was only 1 thousandth of an inch.

The cells of the nucleus dentatus were unaltered in appearance.

The superior peduncles of the cerebellum, and the *corpora quadrigemina* were normal in appearance, and no difference was appreciable between the right and left. The inferior peduncles of the cerebellum were also of a similar appearance on the two sides.

There was, however, a marked difference between the middle peduncles. The right middle peduncle was considerably smaller than the left. At the point of junction with the pons the vertical diameter of the left peduncle was 1·3 centimetre, and of the right only ·8 cent.

On division of the *corpus callosum* in the middle line, this commissure was found to be greatly atrophied, and to form merely a thin membrane along the internal aspect of the hemisphere corresponding to the atrophied region of the convexity. Posteriorly, in the region of the *splenium*, the two sides had a similar thickness.

The pillars of the *fornix* were distinct and equal on the two sides, on the left side almost free from the *corpus callosum* throughout.

The left anterior pillar descended somewhat posterior to the right. Between the two the anterior commissure was of normal size, though slightly oblique.

The posterior commissure, pineal gland, and its peduncles were normal.

The soft commissure was particularly distinct. It crossed the third ventricle somewhat obliquely, the left extremity being a little posterior to the right.

There was no trace of the *nucleus caudatus* or *lenticularis* in the floor of the left lateral ventricle. The floor consisted only of thin membrane forming the orbital aspect of the cyst, which also formed the lateral and superior wall of the ventricle.

The left optic thalamus was smaller than the right.

The anterior extremity of the thalamus began abruptly in a line with the anterior margin of the soft commissure, so that the left seemed smaller than the right by all in advance of this line. The posterior two-thirds of the left thalamus were well developed, and only apparently smaller than the right by the flattening and depression of the left crus cerebri. The *pulvinar* and *corpora geniculata* were equally distinct on both sides.

The left wall of the third ventricle appeared to pass uninterruptedly into the *gyrus fornicatus*.

From the anterior extremity of the left optic thalamus a medullary lamina ·5 cent. width at its junction with the thalamus ran forwards, parallel to the wall of the third ventricle, and lost itself in the marginal region of the frontal extremity.

From the posterior third of the optic thalamus a medullary lamina, measuring 1·5 cent. in width at the edge of the thalamus, radiated outwards towards the occipital and temporo-sphenoidal regions. Between the anterior and posterior medullary laminæ thus marked off, there was a space formed only by the membranous wall of the cyst, entirely devoid of medullary fibres, measuring 1 cent. at the margin of the thalamus, and extending outwards in fan shape towards the atrophied region of the cortex.

Remarks.—This case is in many respects a remarkable one. Though the history is not in all points so complete as could be wished, there is sufficiently clear evidence that up to the age of thirty the woman was to all appearance perfectly well bodily and mentally. Yet such a complete destruction and disappearance of all trace of cerebral tissue had occurred as is met with usually only in congenital cases, and regarded by older writers as due to agenesis or defective development and not to morbid processes. There can be no reasonable doubt, however, that in this case the complete disappearance of such a large extent of the cortex and of the corpus striatum must have been consecutive to some morbid process occurring in adult life, probably embolism of the left middle cerebral artery. The area of distribution of this artery coincides very closely with the regions which have totally disappeared—viz.,

the artero-lateral branches supplying the corpus striatum, and anterior part of the optic thalamus, and the five branches to the convex aspect of the hemisphere, as described by Duret, viz. the external and inferior frontal, the anterior parietal, the posterior parietal, and parieto-sphenoidal arteries.

Considered in reference to the symptoms, the condition of the brain is entirely in harmony with them, according to the doctrines of localisation maintained by myself and others.

With the exception of the postero-parietal lobule, which was greatly reduced in size, and the median aspect of the ascending convolutions, or paracentral lobule and its neighbourhood, the whole of the motor centres of the left hemisphere had disappeared.

In accordance with this, there should have been, theoretically, weakness, but not absolute paralysis, of the right leg, total paralysis of the right hand, with the retention of some of the movements of the upper arm, and paralysis of the right side of the face. This was the condition actually existing.

There should have been also—for a time at least—deviation of the head and eyes to the left, but on this point the history is defective.

And in accordance with the immense majority of instances there should have been complete aphasia or speechlessness. That aphasia existed at first is certain, but the history is defective as regards exact details.

The recovery from the condition of absolute aphasia took place to a considerable extent, though speech was permanently very defective and confined to a few simple phrases and monosyllables. Though in most cases of recovery from aphasia the restoration is due to the fact that the speech-centres of the left hemisphere are not permanently or completely destroyed, yet a case like this seems to bear out the view that compensation may be effected by the speech centres of the other hemisphere. For in this case not a trace of these remained in the left hemisphere. The compensatory action of the right hemisphere was illustrated by a similar case reported by Batty Tuke and Fraser,[1] in which, though Broca's convolution

[1] 'Journal of Mental Science,' April 1872.

in the left hemisphere was entirely destroyed, the aphasic condition, which at first existed, became considerably ameliorated. The present case seems to me thoroughly to substantiate such a compensatory action on the part of the right hemisphere, as always a possibility, if not always an actuality.

That there was no loss of sensibility on the right side shows that the regions destroyed are not the centres of sensibility, and that these, both general and special, are situated in the regions posterior to the fissure of Rolando and fissure of Sylvius, as I have elsewhere demonstrated.

The partial destruction of the auditory centre does not seem to have caused any permanent "word-deafness," as the patient seemed always readily to understand what was said to her. What the condition was in the earlier stages is unfortunately impossible to ascertain accurately.

The mental condition of the patient was profoundly impaired, a condition in which the total destruction of the anterior frontal regions on one side, no doubt, played an important part, as has been demonstrated by recorded cases of lesions invading this region alone. But the case is too complex to allow of an exact estimation of how much is to be attributed to the degeneration of this region.

Considered in reference to cerebral anatomy, the case may be regarded as a functional dissection of the motor tracts and their connections.

Secondary to the destruction of the motor area of the cortex and internal capsule with its nuclei, we have a marked degeneration of the inner two-thirds of the foot of the crus cerebri, which are thus shown to be in direct relation with the atrophied regions.

The remarkable degree of atrophy of the right lobe of the cerebellum is also an important fact. Atrophy of the opposite lobe of the cerebellum has been recorded in many instances of degeneration more or less extensive of one cerebral hemisphere. Numerous cases of this kind have been described and referred to by Cotard,[1] and others have been recorded by Howden,[2] Schroeder van der Kolk, &c. But this case seems

[1] 'Atrophie Particlle du Cerveau,' 1868.

[2] 'Journal of Anatomy and Physiology,' May 1875.

to show with unusual precision atrophy of the cerebellum in relation with degeneration confined to the motor region of the opposite cerebral hemisphere.

Of the peduncles of the cerebellum, the right middle peduncle alone exhibited evident signs of atrophy. In connection with this there was great flattening of the left side of the pons.

This condition is altogether in harmony with Meynert's view, that the middle peduncles of the cerebellum are in relation with the pyramidal tracts of the opposite cerebral hemispheres respectively. Meynert has appealed to such cases of crossed cerebellar atrophy in support of his anatomical views, but their validity has been disputed by some. Gudden has not found that destruction of one hemisphere in animals causes any discoverable atrophy of the opposite cerebellar hemisphere.

But the experimental conditions were probably not such as to allow of time for the atrophy showing itself; for there seems no reasonable ground of doubt for regarding the atrophy of the right cerebellar lobe, so evident in this case, as in direct relationship with the destruction of the motor region of the left cerebral hemisphere.

The atrophy of the cortex of the cerebellum, both the grey layer with the cells of Purkinje, and the granular layer, taken in relation with the atrophy of the middle peduncle, shows the direct anatomical relationship of this peduncle with the cortex, and the functional association of these parts with the cerebral motor centres and tracts.

Only a very small portion of the pyramidal tract of the left side of the medulla oblongata remained, and if the spinal cord had been available for examination, degeneration of the pyramidal tracts would without doubt have been readily demonstrable.

METHODS OF PREPARING, DEMONSTRATING, AND EXAMINING CEREBRAL STRUCTURE IN HEALTH AND DISEASE.

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(Continued from p. 466.)

Staining and Mounting Sections of Hardened Brain.

The staining reagents advocated by different authorities for sections of the nervous system prepared by hardening are numerous, and the list has become so lengthened since the earlier labours of Lockhart Clarke, Van der Kolk, and others, that one great difficulty presented to the student is to make a judicious selection of such as will yield him the best results in the special direction pursued by his work. Amongst those most generally used are carmine and its combinations—picro-carmine, indigo-carmine, and borax-carmine; the aniline series comprising aniline blue-black, aniline blue, methyl-aniline, and rosanilin or magenta; hæmatoxylin or logwood; eosin; picric acid; osmic acid; double chloride of gold and potassium.

Many years' experience has convinced me that a large proportion of these reagents may be safely dispensed with, the following list really comprising all the more essential solutions for staining nervous tissues.

List of Staining Reagents. — Hæmatoxylin, carmine in ammoniacal solution, picro-carmine, aniline blue-black, aniline blue and osmic acid.

In making choice of metallic impregnation by osmium or staining by means of the mineral or vegetable pigments in this list, the student must be influenced by several considera-

tions. Does he require a uniform staining by a simple dye, or does he wish for a differentiation of elements by the use of double pigments, or by double staining? Does he wish to examine specially the cortex and medulla of the cerebrum, or the cortex of the cerebellum; or again, the pons, medulla, and cord? Does he desire to display certain special elements of the cortex, e.g., the nerve-cells and their extensions to their greatest advantage, even at the sacrifice of other details, such as is often required in minute investigations into the histological constitution of the tissues?

Whatever be his object, these questions must receive an answer before the staining reagent can be selected which will yield the effect desired. I propose to give here the composition of each reagent, the method of staining, and its special value as a dye for the various histological elements, and supplement these observations by an analysis of their relative value for different regions of the cerebro-spinal system.

Hæmatoxylin or Logwood Staining.

This reagent has often fallen into disrepute owing to the notable variability in the quality of the dye issued by different makers. It is always advisable to make our own solution, adopting the formula given by Minot, or that employed by Kleinenberg. For the brain and spinal cord I use the former, as it is preferable to that of Kleinenberg, and I find it at all times most trustworthy in its reaction, and the uniformity of results obtained.

Minot's Formula.

Hæmatoxylin (crystals)	3·5 parts
Absolute Alcohol	100 „
Alum	1 „
Water	300 „

First dissolve the hæmatoxylin in the alcohol, and add to it the alum previously dissolved in the water. Keep in a stoppered bottle labelled "Hæmatoxylin Dye" (Minot).

For use—a solution of alum (0·5 per cent.) is poured into a watch-glass or porcelain capsule, according to the size and number of the sections to be stained; a little of the dye is

then dropped in until the solution assumes a light violet tint, when it should be passed through filtering paper before the sections are immersed in it. The degree of dilution will soon be learnt by experience, the depth of tint being our guide.

Kleinenberg's Formula.

1. Make a saturated solution of crystallised calcium chloride in 70 per cent. alcohol, and add alum to saturation.
2. Make a saturated solution of alum in 70 per cent. alcohol, and add 1 to 2 in the proportion of 1 : 8.
3. To the mixture of 1 and 2 add a *few* drops of a *barely* alkaline saturated solution of hæmatoxylin.

Excellent as this logwood solution is for embryonic tissues, I cannot recommend it for the sections of hardened brain such as we are now engaged with, to the same extent that I can that of Minot.

The Staining Process.—Withdraw the sections to be stained from the methylated spirits in which they float by means of a glass rod or camel-hair pencil, and immerse them in the logwood dye contained in a watch-glass or porcelain capsule. The sections should be lightly stained, otherwise they are spoilt, both by too diffuse a staining and by acquiring a brittleness unfavourable to subsequent manipulation. In his first attempts the student should occasionally examine a section on the stage of the microscope, and so judge of the progress made by the staining. When sufficiently stained the dye should be poured off, the sections floated up in a porcelain capsule half full of water, the capsule slightly inclined towards a gentle stream of water from a tap which, falling on the edge of the capsule, keeps up a constant change of fluid, and ensures a most thorough washing of the sections by the currents it produces. Care should be taken that delicate sections are not torn by this means, which is really necessary to ensure removal of all deposit from their surface. Next remove the section to a capsule containing methylated spirit, which, after one or two hours' immersion, will have deprived them of any diffuse staining. They are placed in rectified spirit or absolute alcohol for five or ten minutes for dehydration, transferred to

the centre of a glass slide, floated up by a drop or two of oil of cloves, which is allowed to run beneath them, and, when perfectly transparent, the oil is drained off, and the section mounted in Canada balsam. To ensure successful staining with this reagent, the following points must be carefully attended to:—

Stain only the very finest sections.

Let the sections be perfectly freed from any acidity due to chromic or picric acids, &c., by immersion in spirit, otherwise the staining will be a failure.

Make your own solution of hæmatoxylin.

Employ Minot's solution in preference to others, and reserve Kleinenberg's for embryonic brain.

Never omit filtering the diluted dye prior to use.

Guard against diffuse staining and brittleness from too prolonged action of dye.

Wash the sections *very thoroughly* subsequent to staining.

Remove any accidental diffuse colouration by prolonged immersion in alcohol.

Dehydrate perfectly before clearing up with clove oil.

Mount in a benzole solution of balsam.

Notes on the Reaction of the Dye.—Hæmatoxylin is especially well suited for the display of the various nuclei met with in the brain and spinal cord, e.g., the connective nuclei of the neuroglia, the peri-cellular and peri-vascular nuclei; the nuclei and nucleoli of the nerve-cells. The large nerve-cells of the cortex are beautifully shown in successful preparations, and their primary and secondary branches may be followed for some distance, but never to the extent seen in aniline staining. On the other hand, both neuroglia basis and nerve-structures undergo much shrinking by the use of the dye, and from this cause the smaller cells of the upper layers lose, to a great extent, their normal features. The protoplasmic extensions and nerve-fibre network of the cortex are, indeed, very poorly exhibited in logwood as compared with aniline preparations. The axial cylinders of medullated fibres exposed in transverse section are well exhibited by logwood, and hence sections of spinal cord and medulla stained by this dye show to advantage.

Carmine Staining.

The Dye.—The most useful solution for hardened brain sections is that recommended by Beale, which can be diluted to the required strength, the original solution being far too strong for our purpose.

Beale's Formula.

Carmine (in small fragments)	10 grs.
Price's Glycerine	2 oz.
Strong solution of ammonia	½ drachm.
Alcohol	½ oz.
Distilled water	2 oz.

Agitate the carmine with the liquor ammoniæ in a test-tube gently heated over a spirit-lamp. The solution when completed is carefully boiled for a few minutes, and exposed in the open test-tube for one hour for the escape of excess of ammonia. The water is now added, the solution filtered, and, after the addition of the spirit and glycerine, it must be exposed to the air until the odour of ammonia given off from it is very faint. The clear fluid should be kept in a stoppered bottle, and, should any carmine be precipitated, a drop or two of liquor ammoniæ should be added.

Formula for Borax Carmine.

Carmine	1 part.
Borax	4 parts.
Water	56 parts.
Alcohol	q. s.

Dissolve the borax in the water and add the carmine. Filter the solution, and add alcohol sufficient only to ensure free permeation of the tissue by the dye.

Thiersch's Formula.

Solution A.	Carmine	1 part.
	Liquor ammoniæ	1 „
	Distilled water	3 „
	dissolve and filter.	
Solution B.	Oxalic acid	1 part.
	Distilled water	22 parts.

Mix one part of A with 8 parts of B, and add 12 parts of absolute alcohol. Label "Thiersch's Carmine Reagent."

Modifications of Gerlach's original solution of carmine have been introduced also by Frey and others, but the three above given are all that are really requisite for staining nervous tissues.

The Staining Process.—Give the preference to Beale's solution, although its density from the glycerine renders the action slow. Dilute the strong solution with seven times its bulk of water, and filter. The sections should be placed in a comparatively *large quantity* of this dilute solution, covered with a bell-glass from dust, and allowed to undergo *very gradual* staining—a process which may occupy eight hours or longer. When a section removed on a slide exhibits sufficient staining, the reagent may be poured off, superfluous carmine removed by gentle washing, and all the sections immersed in a very dilute solution of glacial acetic acid (0·5 per cent.) for fifteen to twenty minutes. The acetic acid deprives the specimen of all diffuse staining and fixes the dye, more especially in the germinal centres, whilst at the same time the colour is brightened by its agency. Great care should be taken that neither section of staining solution is very alkaline in reaction, otherwise the staining will prove too deep and diffuse, and the structure itself injuriously affected. A minimum amount of ammonia, however, must be present in the dye, just recognisable by its odour; and if this be not the case, a drop or two of the liquor ammoniæ should be added prior to making the dilute solution. These sections may now be washed and mounted in glycerine or Farrant's Solution, or they may be dehydrated in the usual way by spirit, cleared up by oil of cloves, and mounted in Canada balsam or in dammar. With a few of these carmine-stained sections the student should try the following modified process of mounting, whereby valuable information is afforded. Several years ago I described in the 'Quarterly Journal of Microscopic Science,' vol. xvi., a method of displaying a great wealth of structure in the cortex cerebri by altering the refractive indices of the structural elements, and so producing very remarkable differentiation of structure. I there stated what follows:—"On placing an unstained section of cerebrum or cerebellum in the field of the microscope, saturated with spirit, little or no structure is

apparent, but, if a drop of essential oil be now allowed to run over it, there will be observed at a certain stage of the clearing up, and whilst the spirit is evaporating, a sudden starting out in bold relief of the cells, nerve-fibres, vessels, &c., which again disappear or partially fade on perfect clearing of the section. Now this appearance may be fixed by suddenly dropping over its surface a little balsam, and permanently mounting. Upon this fact depends the process now to be described. Sections treated with Beale's carmine solution (1 to 7 in strength), and washed with the acid wash, are placed, saturated with spirit, upon a slide. When the spirit has nearly all evaporated, a drop of oil of anise is allowed to flow *over* the section (not to float it up), and the clearing process is watched on the field of the microscope; then, just when the appearance referred to above is presented to view, a drop of balsam is allowed to run over the section, and a covering-glass permanently fixed on. In lieu of the oil of anise I frequently employed glycerine with the same results, and mount ultimately in glycerine jelly. . . . I will state my belief that this method will yet prove of most essential service in the estimation of the relative proportions of cell-processes in any individual section, and the most accurate tracing of any existing connections, for not by the deepest aniline staining have I yet succeeded in demonstrating the existence of so thick and numerous a series of processes diverging from the pyramidal layers of the cerebral cortex as by the method described above." These opinions have been confirmed, I am glad to see, by Prof. Stirling in his recent 'Text Book of Practical Histology.'[1]

Notes on the Value of the Dye.—Specially suitable for the large nerve-cells—their contents, their nuclei, and nucleoli. It exhibits well the connective cells and the vascular apparatus. It is less adapted for displaying the cell processes, and wholly fails to exhibit the details of structure in smaller nerve-cells such as those of the upper cortical layers. For the larger

[1] Dr. Stirling says:—"The processes of the cells (cerebrum) are best seen in preparations which are only *partially* cleared up under the influence of clove oil. This is a most important method of investigation. I have often seen in this way delicate fibrils, not unlike elastic fibres, and which are not distinct when the section is completely cleared up." ('Text Book of Pract. Histology,' p. 101.)

ganglionic cells and their immediate environment, nothing perhaps can surpass a successful carmine preparation, whilst it does not appear to produce any further shrinking of protoplasm or connective such as is produced by hæmatoxylin. It has been regarded, even by so eminent an authority as Charcot, as the only reliable dye for exhibiting the morbid conditions regarded as sclerosis of the different columns of the cord. This, however, is not the case, since aniline blue-black portrays the lesion even more distinctly. The more important objections attached to this dye are its variable results, the little definition usually given to the tissue elements from the tendency to diffuse staining; the unpleasant glare of the carmine tint for continuous work, and its unsuitability for examination by artificial light.

Picro-carmine Staining.

The Dye.—This valuable reagent is of special use in the preparation of nervous tissues, and cannot be dispensed with as it certainly ranks next to aniline black for such purposes. Ranvier's formula is as follows:—

Best Carmine	1 gramme.
Water	10 c.c.
Liquor ammoniæ	3 c.c.

Rub the carmine up with the water and add the solution of ammonia, aid the solution of the carmine by heat carefully applied. When dissolved and perfectly cold, add to it 200 c.c. of a saturated watery solution of picric acid. Place the solution in an open vessel, and with gentle warmth evaporate to one-third of its bulk. Filter and keep in a stoppered bottle, labelled "Ranvier's Picro-carminate Dye." Picro-carmine may be purchased in a solid form in a granular and imperfectly crystalline condition—a one per cent. solution is then used. I do not advocate its use, and from personal experience strongly advise the student to make his own reagent.

The Staining Process.—Immerse the sections in a small quantity of the picro-carminate solution for a period of from twenty to thirty minutes. Pour off the dye, draining off most of the superfluous fluid, and then *without washing the sections*

float them up by glycerine, in which they are to be mounted. It will be found advisable to stain each section upon the slide by dropping over it sufficient of the reagent to cover it wholly, and to clear it up with glycerine on the same slide it is to be mounted upon. The object served by this method is to preserve the double dye of picric acid and carmine, the former being very readily washed out by water. Upon the other hand washing freely, dehydrating with spirit, and mounting with balsam in the usual way, will usually yield us excellent carmine preparations, and, so far as my experience goes, better stained specimens than those subjected to the simple ammoniacal or borax solutions.

Notes on the Value of the Dye.—Its special value depends upon the presence of the two pigments, carmine and picric acid, and from the affinity displayed for either dye by different tissue elements, this reagent has acquired a well-deserved and favourable reputation. As a simple carmine dye used in the manner above described, it is also deservedly esteemed for the cerebral cortex, but for the combined staining with osmic acid or with aniline black it forms a most invaluable adjunct to our list of reagents. The merits and demerits of carmine staining pertain to the picro-carminate, but it is decidedly superior to simple carminate of ammonia and its *rôle* as a staining reagent more extensive. As a general dye for the nerve structures, it ranks next to aniline and aniline picro-carminate, and is far superior to logwood.

Aniline Staining.

The Blue-Black Dye.—This, which is by far the most valuable of the aniline series of dyes for the brain and spinal cord, is thus made :—

Aniline Blue-black	1 gramme.
Distilled water	400 c.c.

Dissolve, filter, and keep in a stoppered bottle labelled "Aniline black Dye, ·25% Aq. Sol." It may be convenient, however, to keep a stock solution of 1 per cent. strength, as occasionally a solution so strong may prove serviceable for rapid staining, and it may readily be diluted down to ·25 or

·5 per cent., where the usual dye is required filtering the solution prior to use. The solution as given above can be employed for staining fresh and hardened sections, the aqueous solution being the best dye we possess for the former. The alcoholic solution, as first recommended by Mr. Sankey, was the following :—

Aniline Blue-black	5 centigrammes.
Water	2 cubic centimetres.

Dissolve and pour it into 99 c.c. of Methylated Spirit.
Filter the solution and label "Alcoholic Solution of Aniline Black ·05 per cent."

Prof. Stirling recommends a solution double the strength of the above, thus :—

Aniline Blue-black	1 decigramme.
Water.	4 cubic centimetres.

Dissolve and add 100 c.c. of Rectified Spirit, and filter.
Label "Aniline Blue-black—Alcoholic Solution ·1 per cent."

Of the above dyes the student is recommended to keep always at hand an aqueous solution, ·25 per cent., and the alcoholic solution 0·1 per cent. I much prefer the aqueous solution for most purposes.

The Staining Process.—Sections of hardened cortex or pons, medulla, and spinal cord may be left in the aqueous solution (·25 per cent.) for one hour. In this time they will generally be found stained to a sufficient extent, when they must be removed to a vessel containing water, and well washed from superfluous dye. They are dehydrated by spirit, cleared up by oil of cloves, and mounted in balsam or dammar. *Sections of cerebellar cortex* should be deeply stained by aniline, gently washed by water, and then immersed for twenty to thirty minutes in a 2 per cent. solution of chloral hydrate. Next transfer to the following solution :—

Solution of Chloral Hydrate	2 per cent.
Oil of Cloves	equal parts.
Alcohol	q. s.

to dissolve perfectly and form a clear solution. Add the alcohol by degrees, stirring the solution with a glass rod and avoiding excess of spirit. During use carefully cover the watch-glass containing this solution, so as to avoid evaporation. The chloral removes diffuse staining, whilst the oil of

cloves clears up the section and enables us, by examining one occasionally under a low power, to decide when they have reached a satisfactory stage. When this has been attained, remove your section to a slide, rapidly wash with a little pure alcohol, thoroughly clear with clove oil, and mount in balsam. The alcoholic solution of aniline ·1 per cent. stains the sections of cerebrum, pons, and medulla in the course of a few minutes; they are then cleared and mounted in balsam in the usual way. Glycerine, being a powerful solvent of aniline, must not be made the medium for mounting these sections.

Notes on the Value of the Dye.—Its action is energetic, it is certain and constant in its results, readily controlled, so that any depth of staining may be obtained with ease. The tint given varies from a bluish-grey to a deep blue-black, and is a pleasant one for the eye, causing little or no fatigue. It enables us to obtain the clearest and sharpest definition of elements in a tissue without modifying their structure by shrinking or other change. No other staining agent displays the nerve-cell processes to such a remarkable extent as does this dye, the finest ramifications being followed out with ease. It has a very special affinity for protoplasm—staining the nuclei of the nerve-cells most deeply, then the nerve-cells and their protoplasmic extensions, and to a less intense degree the nuclei of the neuroglia and peri-vascular walls. The finely-formed nerve and connective meshwork forming the framework of the cortex is stained of a pale grey. This dye fails to produce any action upon the medullated sheath of nerve-fibres, affecting only the axis cylinder. Transverse sections, therefore, of nerve-fibres, such as are seen in cross sections of the medulla, cord, and nerve-trunks, exhibit the axis cylinder stained of a dark blue, the white matter of Schwann surrounding it; whilst the inter-tubular connective, likewise faintly stained, differentiates the medullated tubes from each other. For transverse sections, therefore, of nerve fasciculi in any region, this staining reagent will prove of the utmost service. Its value for connective tissue stuctures is very limited, its *rôle* being chiefly confined to nerve elements.

Osmic Acid Staining.—This method we owe to Professor Exner. It is peculiar in the fact that the hardening and stain-

ing proceed simultaneously, the same reagent being employed for both purposes. The preparation of the hardened brain by immersion in osmic acid has already been described, and we have now simply to detail his further treatment of the sections obtained on the microtome. These sections, owing to the deep staining they undergo, must be *extremely thin*, and should be placed in glycerine immediately, since prolonged immersion in alcohol injures them seriously. Transfer a section to a glass slide and add a few drops of strong liquor ammoniæ. Absorb all superfluous moisture by bibulous paper, and after a short time for the ammonia to affect the section thoroughly, drop the cover on without injury to the preparation, which has now become softened by the ammonia. It is at this early period that the section may be examined with greatest advantage. For permanent preparation Exner surrounds the cover-glass with the soluble silicate called "water-glass." Formerly he used to stain with ammoniacal solution of carmine, but, finding the results obtained were attributable solely to the ammonia, he has dispensed with carmine for this purpose.

Notes on the Value of this Method.—The medullated fibres can be traced upwards into the highest realms of the cortex, exhibiting a wealth of structure which no other method displays. The medullated sheath is deeply stained, and can be traced distinctly in the case of the minutest nerve fibres. The nerve-cells, although stained, do not form such prominent objects as by other methods of staining, this process being especially adapted for tracing the ultimate course of medullated fibres through the different layers of the cortex and determining their final destination. Exner states that the staining of the minute fibres occasionally disappears in time, and the student should also be aware that the brain structure swells up under the action of ammonia, so that the section is increased in size to one-third its full diameter.[1]

Double Staining and the Use of Compound Pigments.

Under this head I would include some of the most valuable methods we possess for delineating the minute structure of the

[1] For the results obtained by Exner with this process, see Abstract in 'Brain,' Part XV.

nervous system. Picro-carmine, as consisting of two pigments, would naturally fall under this category; but, since it is employed in the same way as the simple dyes, I have found it more convenient to include it amongst them. The methods of staining to which I now refer are those in which the tissue is first submitted to one dye, and the result subsequently modified by the addition of a second pigment. In these cases either different histological elements assume the tint of each pigment, or the former staining is greatly improved in character by the second pigment combining and modifying the tint acquired; in the former case a genuine double staining is obtained; in the latter, the union of two or more pigments gives but the effect of a single tint, yet one of very valuable quality.

Aniline Picro-carminate Staining.

Three pigments are here used, viz., the aqueous solution of aniline blue-black 0·25 per cent., and Ranvier's compound pigment—picro-carmine. Sections which should be very thin may be stained on a glass slide by dropping over them by a pipette sufficient picro-carmine to cover the section completely. When deeply stained by the reagent, the latter is drained off and the section covered in like manner by a quantity of the aniline blue-black solution. It will be found that the action of the aniline proceeds much more rapidly upon a section stained by picro-carmine; ten minutes usually suffice to produce the desired result. This result is acquired when upon draining off the dye the section is found to have changed from the bright carmine to a deep violet tint. Wash the section well in water, dehydrate by spirit, clear by clove oil, and mount in balsam. In this way the most beautiful effects are obtained, which are specially valuable and instructive. The tint is much less fatiguing to the eye than the bright glare of carmine, whilst in the differentiation of nervous structure in the hardened brain this method of staining stands unrivalled. The deeper the tint desired, the longer immersion in aniline is required; but for the more satisfactory and pleasing effects I have usually found it suffice to stain for half an hour in the picro-carmine and fifteen minutes in aniline solution.

Notes on the Value of the Dye.—This double staining enhances the value of an ordinary piero-carminate preparation in the fact that a better differentiation of structure is obtained, and examination by artificial light rendered as agreeable and valuable as by daylight. The neuroglia or connective element is deeply stained, and its nuclei are prominent objects; the grey matter of the brain is marked off from the central medulla by a much deeper staining of its elements. The nerve-cells are well shown, and their nuclei take the deepest tint. All bundles of medullated nerve fibres cut across by transverse section exhibit their central axis cylinder of a purplish colour, surrounded by the unstained medullated sheath, outside which is seen the intertubular connective and its nuclei stained of lighter hue than the axis cylinder. The vascular channels and their nuclear elements are stained in a satisfactory manner. This staining reagent is very suitable for exhibiting to low powers the coarse structure of the brain, and sections through a whole hemisphere treated by this method are most instructive for naked eye examination or the coarse objectives.

Picro-aniline Staining.

A useful staining of medulla and spinal cord may be obtained by employing Judson's "Cambridge Blue" as the first pigment, washing off superfluous dye, and then by momentary immersion in a saturated solution of picric acid the section acquires a brilliant green staining, which exhibits structural details remarkably well. Since both pigments are soluble in alcohol, the sections should be very rapidly dehydrated, cleared by oil of cloves, and mounted in the usual manner.

Osmic Acid and Picro-carmine.

The Staining Process.—The section is immersed in a solution of osmic acid 1 per cent., carefully covered over so as to confine this very volatile reagent, as far as possible, to the tissue to be acted upon. When the latter has acquired a dark brown tint it should be removed, well washed, and lightly stained with picro-carmine. Such sections must be mounted in glycerine or glycerine jelly. Sections of limited size, such as sections

through the hemispheres of the brain in small animals or the pons and medulla, may be conveniently acted upon by the osmic acid contained in a watch-glass covered with a small glass plate. This is a most valuable method of staining.

Hæmatoxylin with Aniline.

The Staining Process.—Sections are first stained with logwood according to directions already given, and immersed *for a few seconds only* in the aqueous solution of aniline blue-black 0·5 per cent. A solution of chloral hydrate, 2 per cent., will remove any excess of aniline staining. Next, well wash the section, *rapidly* dehydrate by alcohol and mount sec. art. This process is adapted chiefly for sections of cerebellum, and its special value will be referred to later on.

The above are all the methods of staining which I have found to be of especial value, but I append here two methods of double staining for the cortex of the cerebellum, advocated by Prof. Stirling,[1] and of the value of which I have not had personal experience.

Eosin and Logwood.

Stain a section for a few seconds in a very dilute watery solution of eosin (1 part to 1500 of water) until it has a faint red colour. Great care must be taken not to overstain. Wash with water, stain the section with logwood, and mount in dammar.

Picro-carmine and Iodine Green.

Stain a section in picro-carmine. Wash it in water acidulated with dilute acetic acid, and, after washing in pure water, stain again by means of iodine green and mount in dammar.

[1] 'Practical Histology,' p. 100.

(To be continued.)

Critical Digests and Notices of Books.

L'Hérédité Psychologique. Par Th. Ribot. Paris, Germer, Baillière & Cie., 2me éd., 1882.

This "study," forming part of the Bibliothèque de Philosophie Contemporaine, professedly philosophical, is not very strictly scientific. It is, however, extremely interesting and well written, and replete with the results of extensive reading of the now somewhat numerous and distinguished investigators who have observed, speculated, and even experimented upon heredity and its allied subjects. Among them the writings of our own illustrious countrymen, Darwin and Herbert Spencer, are frequently quoted, and Mr. Galton's work upon "The Heredity of Genius" is by no means forgotten. The author remarks of Mr. Galton's book, that it has merits and defects sufficiently common in English works; containing as it does many figures and facts, and but few general ideas; and we may without injustice reciprocate the criticism by saying that M. Ribot's book is sufficiently marked by the opposite faults and merits of French writers, in that it contains a larger proportion of general ideas to the figures and facts which ought to support them, than is desirable in a work of scientific character.

Not that some foundation of facts is not supplied. Indeed, the first half of the volume professes to supply these alone; but they are thrown down somewhat in a heap, like a load of stones, not examined and analysed and sorted as specimens to see how much or how little they prove; which leads us to think that the method of this work may be greatly improved upon by future writers on this most interesting and important subject. Perhaps the question of heredity in man may not always remain more interesting than important in consequence of the difficulty of interfering, at least in civilised society, with the breeding of

men and women. Perhaps in the future a more general knowledge of the heredity of qualities may lead to better results by individual selection than was ever obtained by the will of a despot in Prussia in the selection of females for his grenadiers, or that of a proprietor in Kentucky in selecting males for his dark women.

The author has scarcely justified his choice of a title for his work as L'Hérédité Psychologique, seeing that it deals more fully with the more obvious and distinct facts of somatic than of psychologic heredity. No doubt psychological quality is based upon the hereditary substance, whatever that may be, and, although we may dispute Gall and Spurzheim's notions of intellectual and moral heredity as depending upon the inherited shape of the head, we can scarcely doubt the existence of a true principle underlying their doubtful detail, namely, the principle that moral and intellectual heredity depends upon the inheritance of a similar organism.

But the facts of moral and intellectual heredity, that is to say, those which it is the professed purpose of this volume to elucidate, are extremely difficult to appreciate with any degree of scientific accuracy. It is so easy to observe that some peculiarity of form or feature descends in a family; the thick lip of the Hapsburgs, for instance. But it is quite another thing in point of difficulty to trace the descent of mental qualities, or to account for variations, should the descent be unmistakably broken. Thus, a late Emperor, a man of mild temper and high courage, has been succeeded by a son in whom these qualities would seem to be reversed. Thus the audacious financier of Egypt has been succeeded by a son who is equally timid and honest. Such startling changes may be read by the fierce light which beats upon a throne; but how to account for them, that is the difficulty. M. Ribot would tell us to look to the maternal parents for an explanation; the sons, as a rule, inheriting their qualities from the mother, the daughters from the father. But is this so? A few examples certainly will not prove it, and it is just one of those questions which can only be answered by what M. Ribot describes as the defective method of Mr. Galton, that is to say, by facts and figures, and by using the figures in

strict relation to corresponding notions, a rule of statistical science which is by no means invariably observed. An important example to the contrary may be read in the work before us, where the author attempts to refute the late Mr. Buckle's objections to the doctrine of heredity as applied to mental qualities. Not to risk the probable errors of a retranslation, we give Buckle's words from the original:—

"We often hear of hereditary talents, hereditary vices, and hereditary virtues; but whoever will critically examine the evidence will find that we have no proof of their existence. The way in which they are commonly proved is in the highest degree illogical; the usual course being for writers to collect instances of some mental peculiarity found in a parent and in his child, and thus to infer that the peculiarity was bequeathed. By this mode of reasoning we might demonstrate any proposition; since in all large fields of inquiry there are a sufficient number of empirical coincidences to make a plausible case in favour of any view a man chooses to advocate. But this is not the way in which truth is discovered; and we ought to inquire, not only how many instances there are of hereditary talents, &c., but how many instances there are of such qualities not being hereditary. Until something of this sort is attempted, we can know nothing about the matter inductively; while, until physiology and chemistry are much more advanced, we can know nothing about it deductively. These considerations ought to prevent us from receiving statements which positively affirm the existence of hereditary madness and hereditary suicide; and the same remark applies to hereditary disease; and with still greater force does it apply to hereditary vices and hereditary virtues; inasmuch as ethical phenomena have not been registered as carefully as physiological ones, and therefore our conclusions respecting them are even more precarious."[1]

In answer to this indictment M. Ribot cites Maupertuis's observations upon the inhabitants of a town containing one hundred thousand inhabitants, not with respect to their virtues, vices, talents, or maladies, but as to the individuals among them who had been born with six fingers. None of the objections which Buckle raises to the logic of the usual arguments,

[1] "History of Civilisation in England," vol. i., pp. 160, 161.

as to the heredity of the shifting and indistinct mental qualities which he has designated, can in any way apply to the totally different quality of sexdigitism of which Maupertuis found an example hereditary through four generations, and reckoned that supposing that only five persons in the twenty thousand inhabitants had this physical peculiarity it was 800,000,000 to 1 that the peculiarity would not continue itself by accident through three consecutive generations. But sexdigitism is a quality which can be distinctly defined, virtue and vice are qualities which cannot be; sexdigitism is a rare peculiarity, virtue and vice are common to all men, and to meet Buckle's objections by such a reply is to leave them unanswered.

We can have no doubt now that Buckle was too sceptical as to the moral and intellectual progress of mankind through the operation of heredity in the transmission of the moral and intellectual ameliorations of civilisation; but surely he was right in insisting that scientific methods should be applied to a scientific inquiry, and that the *post hoc propter hoc* argument too often used in these inquiries should not be accepted with facile faith in its efficiency.

A most important illustration of the nature of Buckle's objections may be found in the facility with which heredity is commonly attributed as the cause of mental disease in which ascending relatives have been insane; for it has needed no more than evidence showing that a parent or grandparent has been insane to establish the hereditary character of almost any case of mental disease; the fact being ignored that the accidental causes which operated in the parent may equally operate *de novo* upon the child, and even that the insanity of the parent may cause the insanity of the child indirectly, but subjecting the child to moral and physical evils, such as grief or shame, or physical misery. If there were the same degree of probability that the child of an insane person would be insane from the influence of heredity as, according to Maupertuis, there is that the child of a man with six fingers will have six fingers, the civilised countries of the world would before this have been filled with madmen; unless, indeed, stern laws against the procreation of hereditary madmen were enacted. That no such laws have been, or are likely to be, made is a strong indication that here-

ditary influences have not that certain and constant effect which would justify such laws for the prevention of a dire and overmastering mischief. What we have to do is to find out the marks, if we can, by which hereditary insanity may be discriminated from accidental, and to find out the circumstances, if we can, which favour the transmission of the tendency to it. We are not quite ignorant even now in these matters, but the so-called laws of psychical heredity do not seem likely to lead us far in this intricate investigation as to the effects of inheritance, as distinct from the effects of all other influences, which may be called education, upon the moral and intellectual qualities of men and women.

These laws, according to M. Ribot, are:—

1*st*. That parents have a tendency to bequeath all their psychical characters, general and individual, ancient and newly acquired [law of heredity direct and immediate].

2*nd*. One of the parents may have a preponderating influence upon the mental constitution of the infant [law of preponderance in the transmission of characters].

3*rd*. The descendants often inherit the physical and mental qualities of their ancestors, and resemble them without resembling their parents [atavism].

4*th*. Certain physical and mental dispositions very clearly determined, manifest themselves in the descendants at the same age as in the ascendants [law of heredity at corresponding epochs].

Certainly we must accept as a law that like begets like—a law which was not discovered yesterday; for we have been told in a very old writing that "the fathers have eaten sour grapes and the teeth of the children are set on edge;" and the Roman poet reminds us that—

"Fortes creantur fortibus et bonis."

But the laws of exceptions above expressed in the feeble terms of the potential mood ought scarcely to be called laws until we have succeeded in discovering their positive conditions. We have the one great law of heredity which, from the philosophical point of view, the author considers a fragment of the more general law of the universe, that nothing which has been ceases

to be, and therefore that heredity is the law of life arising from the partial identity of the elements of the organism in the parents and the offspring. But the numerous exceptions to this law, the variations which put on the appearance of spontaneity, can we reduce them to any constant rules or formulas? We venture to think that, in spite of the vast importance of the task, we have not yet acquired scientific knowledge of these exceptions, that is to say, knowledge which will enable us to foretell when they will take place. Heredity is the strongest bond of determinism, yet it allows so much movement that it can never be predicted of the individual to what extent it will press his character into the old paternal mould, or leave it to be influenced by post-natal events. Its tyranny over the race is certainly greater than over the individual, but even in the race it is liable to large and as yet incalculable interference.

M. Ribot, in the eloquent passage which concludes his work, compares the man of nature with the man of civilisation, and remarking that the infinite distance which seems to separate the two extremes has certainly been traversed, he says, "Without doubt this evolution, the resultant of the complex play of numerous causes, is not entirely due to heredity; but we shall have badly succeeded in our task if the reader shall not now comprehend that heredity has largely contributed to it," a modest statement well within the confines of the truth.

JOHN CHARLES BUCKNILL.

The Formation of Vegetable Mould through the Action of Worms, with Observations on their Habits. By CHARLES DARWIN, LL.D., F.R.S. London, Murray, 1881.

THIS work, affording new evidence of the great results attainable by the patient and long-continued observation of small matters, the exposition, as it were, by an eyewitness of a geological formation, gives us the pleasure of again meeting with Mr. Darwin in a department of science which does not often receive patient and cautious observation, namely, in that of psychology, and in a phase of animated nature where a less original observer would scarcely have dis-

covered a fruitful field of psychological inquiry. It is with the mind of the earth-worm, and not with the manner in which it incrusts our fields with a fruitful surface, that Mr. Darwin most interests us in these charming pages. But mind can only be evolved upon the foundation of sense impressions, and worms are blind and deaf and have but a feeble sense of smell. "The nervous system is fairly well developed, and the two almost confluent cerebral ganglia are situated very near the anterior end of the body." To say that worms are blind is perhaps not strictly accurate, for although they have no organs of vision, a strong and continued light causes them to retreat into their burrows, unless such light is shed upon them when they are employed in dragging leaves into their burrows, or in eating them, or whilst they are paired sexually. "When a worm is suddenly illuminated and he darts like a rabbit into his burrow, we are at first led to look at the action as a reflex one, but the different effects which a light produces on different occasions, and especially the fact that a worm, when in any way employed, and in the intervals of such employment, is often regardless of light, are opposed to the view of the sudden withdrawal being simply a reflex action," and is, the author thinks, attributable to attention and to some mental power.

The fact that light is capable of stimulating the cerebral ganglia of the earth-worm through the tissues which cover them, without the intervention of any sense organ, is a fact now resting upon unquestionable authority, which we would fain commend to the notice of the distinguished American physician who has recently published his observations on the transmission of the stimulus of light to the human brain through the skin and bone of the forehead. There is, however, some difference between the diaphanous skin of a worm and the dense and opaque skin and bone of a man's forehead, and there is also some difference between the credibility of Mr. Darwin and that of some other observers.

The sense of touch, including the perception of vibrations, is keen in worms. They do not hear the loudest sounds, but if the pot in which they have been kept for observation be placed upon a pianoforte they withdraw into their burrows

immediately a note is touched. Facts are adduced which show that they possess some sense of smell; and as they appear to like cabbage leaves as food, and to distinguish between different varieties, and as they choose various other substances for food, it is likely that they possess the sense of taste. But it is in the sense of touch that they live and move and have their being, and it is by this they exercise that degree of choice in the selection of materials for lining their burrows, which has enabled the illustrious author to write so much at length upon their habits as displaying intelligence. He may well have been surprised to find that a creature so low in the scale of animal life was able to select and to use the materials of its subterraneous architecture according to the strictest fitness of shape, form and substance. Pursuing his investigations in this matter, the author summarises what he has observed of the mental qualities of his lowly subject thus:—

"*Mental Qualities.*—There is little to be said on this head. We have seen that worms are timid. Their sexual passion is strong enough to overcome, for a time, their dread of light. They perhaps have a trace of social feeling, for they are not disturbed by crawling over each other's bodies, and they sometimes lie in contact. According to Hoffmeister, they pass the winter either singly or rolled up with others in a ball at the bottom of their burrows. Although worms are so remarkably deficient in the several sense organs, this does not necessarily preclude intelligence, as we know from such cases as those of Laura Bridgeman; and we have seen that when their attention is engaged they neglect impressions to which they would otherwise have attended, and attention indicates the presence of a mind of some kind. They are also much more easily excited at certain times than at others. They perform a few actions instinctively, that is, all the individuals, including the young, perform such actions in nearly the same fashion. This is shown by the manner in which the species Pericheta eject their castings, so as to construct towers; also by the manner in which the burrows of the common earthworm are smoothly lined with fine earth, and often with little stones, and the mouth of their burrows with leaves. One of their strongest instincts is the plugging up of the mouths of

their burrows with various objects; and very young worms act in this manner. But some degree of intelligence appears to be exhibited in this work—a result which has surprised me more than anything else in regard to worms" (p. 35).

Why worms plug up the entrances of their burrows, and why they line them with leaves and other substances, the author has not been able to decide. Perhaps to conceal their burrows from scolopenders, their bitterest enemies, perhaps to keep water out, or cold air, as they dislike cold, and the lining also may be for the sake of warmth; but "whatever the motive may be, it appears that worms much dislike leaving the mouths of their burrows open."

But whatever the motive, either for plugging the orifice or for lining the tube, it can scarcely be disputed that the manner in which it is done displays a degree of intelligence surprising in so lowly an animal.

"If a man had to plug up a small cylindrical hole with such objects as leaves, petioles or twigs, he would drag or push them in by their pointed ends, but if these objects were very thin relatively to the size of the hole, he would probably insert some by their thicker or broader ends. The guide in his case would be intelligence. It seemed, therefore, worth while to observe carefully how worms dragged leaves into their burrows; whether by their tips or bases or middle parts. It seemed more especially desirable to do this in the case of plants not natives to our country; for, although the habit of dragging leaves into their burrows is undoubtedly instinctive with worms, yet instinct would not tell them how to act in the case of leaves about which their progenitors knew nothing. If, moreover, worms acted invariably through instinct or an unvarying inherited impulse, they would draw all kinds of leaves into their burrows in the same manner. If they have no such definite instinct, we might expect that chance would determine whether the tip, base or middle was seized. If both alternatives were excluded, intelligence alone is left; unless the worm in each case tries many different methods, and follows that alone which proves possible or most easy; but to act in this manner and to try different methods makes a near approach to intelligence."

A fairer statement of a proposition could scarcely be made, nor can it be thought that its caution is overstrained, although, no doubt, another great biologist is right in saying that Darwin may be reckoned upon to state any proposition most strongly against himself. The answer to the proposition was carefully worked out with leaves of various shapes and textures, single leaves, double leaves with their points cut off, or tied together with silk, or cemented together, and above all with triangles of writing paper, and the conclusion arrived at, that worms do display intelligence in their manner of plugging up their burrows, is shown to be inevitable by the very careful and most interesting details to be found in the work. The questioning experiments were so put that chance and instinct were eliminated, and nothing left to account for the behaviour of these low animals but intelligence, much to the surprise of the questioner, for "many higher animals have no such capacity," ants, for instance; but then an ant has not such a perfect measuring wand as a worm possesses in its sensitive equiform body, and, not forgetting their antennæ, no such means of acquiring a notion of the shape and dimension of things. And it is to be remarked that the most intelligent of the brutes, as the monkey, the elephant and the parrot, are especially gifted with some organ which enables them to ascertain the shape and dimensions of bodies, indicating that the rudiments of intelligence are best obtained through the medium of tactile impressions. As to what touch may be when it is the sole sense, Mr. Darwin says, "it may be well to remember how perfect the sense of touch becomes in a man when born blind and deaf as are worms. If worms have the power of acquiring some notion, however rude, of the shape of an object and of their burrows, as seems to be the case, they deserve to be called intelligent; for they then act in nearly the same manner as would a man under similar circumstances." Mr. Darwin thinks that it will strike every one as improbable that worms should possess intelligence; "but," says he, "it may be doubted whether we know enough about the nervous system of the lower animals to justify our natural distrust of such a conclusion. With respect to the small size of the cerebral ganglia [of worms] we should remember what a

mass of inherited knowledge, with some power of adapting means to an end, is crowded into the minute brain of a worker ant."

JOHN CHARLES BUCKNILL.

Studien über das Bewusstsein. Von Dr. S. STRICKER. Wien, 1879.

Studien über die Sprachvorstellungen. Von Dr. S. STRICKER. Wien, 1880.

Studien über die Bewegungsvorstellungen. Von Dr. S. STRICKER. Wien, 1882.

THE pamphlets, the titles of which are placed at the head of this article, contain, as might be expected from the eminence of their author, a valuable discussion of various philosophical and psychological problems. Taking these pamphlets in the order of their publication, the first deals with the phenomena of consciousness. Prof. Stricker sets out, like other philosophers, with an analysis of human knowledge. He shows that the knowledge which can be presented in the consciousness of any person at one time is only a small part of the whole, much the greater portion being latent. The portion of knowledge which can be simultaneously presented in consciousness he proposes to call *actual* or *living*, and the remaining portion *virtual* or *potential*, following in this respect the well-known division of force or energy in physical science. This distinction is not of much importance, and we pass on to his second division of knowledge, which is of much greater significance.

Knowledge may be divided into that which is direct or immediate, and that which is indirect or mediate. The highest degree of certainty must always be found in immediate knowledge, and Prof. Stricker accepts, without reservation or criticism, the *cogito, ergo sum* of Descartes as our ultimate grounds of certainty and the starting-point of philosophy. But even if the right of the implied *Ego* to be regarded as a direct deliverance of consciousness be allowed to pass unchallenged, we may be permitted to ask the author how he

would vindicate the right of the expressed *ergo* to be included in a statement of *immediate* knowledge.

The relation of the brain to consciousness is next dwelt upon, and two hypotheses explanatory of the connection are stated. According to the one, an independent entity—the soul—dwells in the brain, and plays upon its material substance as a musician plays upon his instrument; while the other, briefly stated, asserts that consciousness is a function of matter. But surely dualism and materialism are not the only hypotheses which have been advanced to explain the connection between the brain, or, more generally stated, matter, and mind. Idealism, for instance, asserts that the matter of the brain, like every other external existence, is a product of mind, while Realism (Indifferentism of Mansel, Monoism of Haeckel, and Pantheism of theologians) assumes that mind and matter are but two faces of one substance which underlies both. But the truth is that Prof. Stricker passes by as unfruitful, as Locke did before him, the discussion of the relation subsisting between mind and matter, and satisfies himself with offering a compromise. He thinks that both dualists and materialists may agree to say that nerve cells are endowed with psychical functions. The dualist, while using this expression, may think that the soul makes use of the nerve cell to form the conception, and the materialist that the process in the cell begets the conception. Personally we have no great objection to this solution, but we prefer to say that mental phenomena are the *correlatives* of certain material changes in the structure of the brain, and *vice versa*. This statement is a mere summing up of facts which can be readily proved, and which are, indeed, admitted by all, and no assumption whatever is involved in it.

Prof. Stricker points out that nerve fibres, as well as nerve cells, are endowed with psychical functions, and very justly remarks that, were it otherwise, the feeling correlated to the function of each cell in the same brain would be as distinct and independent as the consciousness of two individuals. He does not, however, hazard a conjecture as to the nature of the function of the fibre. Mr. Herbert Spencer's theory of the respective functions of cells and fibres is by far the most rational we have seen. He maintains that the activities of the

cells are the correlatives of feelings, and those of the fibres the correlatives of the relations between feelings. And if thinking is the establishment of relations between simple and compound feelings, it will be seen that the structural counterpart of abstract thinking must chiefly consist in the formation of new connections between nerve cells.

Prof. Stricker now proceeds to discuss the origin of our ideas, observation and memory, common emotional or subjective sensations (Gefühle) and special intellectual or objective sensations (Empfindungen), organic sensations, eccentric projection of sensation, Weber's or Fechner's law, the theory of general and abstract names, original and acquired ideas, and various other interesting problems. These subjects are handled with great ability, and even those who are intimately acquainted with philosophical speculations will find in the author's exposition much to interest and instruct them.

Prof. Stricker then deals with the problems of sleep, dreams, illusions, hallucinations, and delusions. It appears to me that he would have been more successful in his explanations of these conditions had he extended his survey to somnambulism, ecstasy and allied conditions, epilepsy, and especially epileptic auræ, migraine regarded as a sensory epilepsy, delirium, and mania. A modification of Dr. Hughlings-Jackson's theory of epilepsy, which may be briefly termed the theory of cerebral cortical discharge, would suffice to explain all those conditions, and the very fact of ranging them alongside one another would represent them in a more familiar light to the mind, and render them less extraordinary if not less incomprehensible.

The author next discusses the theory of a cheerful, gloomy, or irritable disposition, and the nature of the *will*, along with some of the philosophical problems which cluster around it; but we must now pass on to consider briefly the contents of the second and third pamphlets. These tracts are occupied with the consideration of the mental representations which are associated with speech (Sprachvorstellungen) and those associated with voluntary movements (Bewegungsvorstellungen). The word *Vorstellung* is very difficult to translate into our language. It means literally a *placing* of something *before*

the mind, and consequently includes presented as well as represented feelings. The subject-matter of the last two tracts, therefore, embraces the feelings which accompany speech (articulatory movements, vocalisations, and gestures), and other voluntary movements; whether the movements be rendered into actualities (presented feelings), or only revived in thought (represented feelings). But we shall do no violence to the meaning of the author if we briefly term the subject-matter of these two tracts as a consideration of the *feelings* associated with speech and movements. But as speech, whether it be spoken, written, or acted, when it is objectively considered, depends like bodily movement upon muscular contractions, the feelings which accompany the two processes must to that extent be the same, and consequently only one fundamental theory comes under our consideration at present, as indeed the author admits. It is impossible for me to enter into the details of Prof. Stricker's theory, and I must refer those who are interested in these questions to the original papers. Prof. Stricker's theory is, although worked out with great power and originality, practically the same as that of Prof. Bain, which has been common property with English thinkers for a quarter of a century; and allowing for such minor differences as are sure to arise in the mental products of original thinkers working independently of each other, I would not wish for a better epitome of the thesis of Prof. Stricker in 1880 than the words used by Prof. Bain in 1868. "In very lively recollection," says Prof. Bain, "we find a tendency to repeat the actual movements. Thus, in mentally recalling a verbal train, we seem to repeat the very words; the recollection consists of a suppressed articulation. A mere addition to the force or vehemence of the idea, would make us speak out what we speak inwardly. Now, the tendency of the idea of an action to become active shows that the idea is already the fact in a weaker form. But if so it must be performing the same nervous rounds, or occupying the same circles of the brain in both states."[1] It is a matter for regret that Prof. Stricker did not know, or at least does not mention, the cases of amnesic aphasia recorded by Broadbent and Wernicke. The case

[1] 'Mental and Moral Science,' by Prof. Bain; 2nd ed., 1880, p. 90.

described by Broadbent is an example of "word blindness" and the lesion was found in the angular and adjoining gyri (part of visual centre), while Wernicke's case is an instance of "word deafness," and the lesion was found in the temporo-sphenoidal convolutions (auditory centre). He also does not refer to the important fact discovered by Brissaud and Charcot that in old cases of ataxic aphasia a streak of secondary degeneration is to be found in the knee of the internal capsule and in the crusta. In a case of dysarthria under my own care, not yet published, in which the fibres of the knee of the internal capsule were found softened, I traced a streak of degeneration through the crusta, pons, into the anterior pyramid and median raphé of the medulla oblongata. The case of anarthria and masticatory paralysis recorded by Dr. Barlow, in which there was softening of the posterior part of the third frontal convolutions in both hemispheres, and the cases recorded by Magnus, Lépine, Oulmont, Kirchoff, and myself, in which anarthria and the other symptoms of bulbar paralysis were caused by disease of the lenticular nuclei (with probable injury of the knee of the internal capsule) are full of significance to the theory which Prof. Stricker maintains, that the motor centre for speech is also the motor centre for articulation and vocalisation (Lautebildung). Prof. Stricker says that the ruling doctrine of the origin of our mental representations of movements in the external world asserts that the visual impressions produced by the different phases of a moving body blend together to form a mental representation of the movement of this body. If this be true with regard to the ruling doctrine in Germany, it is certainly not true with reference to this country for the last quarter of a century. "The sensations of sight," says Prof. Bain, "are partly *optical*, the effect of light on the retina, and partly *muscular*, from the action of the six muscles."[1] He then states that the optical sensations are light, colour and lustre, and the sensations involving the muscular movements of the eye are visible movement, visible form, apparent size, distance, volume and situation. This simple statement from Prof. Bain is indeed an admirable abstract of Prof. Stricker's thesis, and

[1] 'Mental and Moral Science,' by Prof. Bain, p. 60.

consequently it is not necessary for me to give a detailed analysis of a theory which is so well known to British Psychologists. Prof. Stricker has made some interesting observations with an instrument—the well-known toy sold under the name of the Wheel of Life or Zoetrope—which he designates the Stroboscope. He believes that the illusion of motion in this toy is not due entirely to the persistence of visual impressions, but that it is mainly caused by the intermittent attempts of the eye to retain the rotating images within the field of vision. This is a modification of the muscular slip theory advanced by R. Addams, and more recently by Dr. Emile Juval, to account for the class of optical illusions first noticed by Brewster, and named by him "illusions of subjective complementary motion." The whole of this subject has been so ably handled by Dr. Sylvanus P. Thompson, in a paper "On Optical Illusions of Motion" which appeared in a recent number of 'BRAIN,' that it is unnecessary for me to make any further remarks on the subject. Prof. Stricker brings his discussion of these all-important questions to a close by a consideration of Cause and Effect, the universality of judgment *a priori*, God and Force, and various other problems which are full of enduring interest. His theory of causation is, as might be expected, closely allied to that of Prof. Bain, and is indeed essentially the same as that of M. de Biran, "who," according to Cousin, "placed the type of causality, of the will and of liberty in the phenomenon of muscular effort."[1] But this theory seems to me to have been disproved in advance by the philosophy of Kant; it has certainly been roughly handled by the followers of Kant in this country—Sir W. Hamilton and Mr. Herbert Spencer. With regard to Spencer it may, indeed, be said that the Empirical has as much right to claim his adhesion as the Intuitional school. One of the greatest advances of recent times in philosophical speculation was made by Mr. Spencer when he pointed out what Prof. Max Müller calls the pre-historic genesis of the congenital capacities which are the sources of our intuitive knowledge,

[1] 'History of Modern Philosophy,' by M. Victor Cousin. Translated by O. W. Wright, 1852; vol. ii., p. 346.

and thus effected a reconciliation between the Empirical and Intuitional schools. And I may be permitted to express the opinion that Prof. Stricker would find in this reconciliation the key to the solution of many of the philosophical problems which appear at present to be so obscure and perplexing to him.

JAMES ROSS.

The Physiology of the Nervous System in its relation to Psychic Facts. By Dr. MARIO PANIZZA.

WE learn from the preface that this work contains a critical examination of two postulates of the physiology of the nervous system, viz.—(1) That the nerves are conductors of the sensory impressions from the periphery to the nervous centres, and of the motor impulses of the will from the nervous centres to the muscles; and (2) That external objects, to be perceived, must first make an impression on the sense organs.

The author proceeds to make what seems the startling announcement that neither of these postulates has any foundation in experience, and that they are both entirely contrary to the truth. Physiologists are, no doubt, familiar with this controversy, from a study of the writings particularly of the late Mr. Lewes; but Brown-Séquard, Ferrier and others have discussed it from time to time, and from all points of view. Nowhere, however, probably, have the doctrines in question been attacked with more vigour, or by anyone who has brought to bear on the discussion more physiological knowledge than the present author, or a wider range of reading among philosophical as well as physiological writers. His first point is to attempt to show that science has obtained the idea of the double transmission of sensory impressions and motor impulses of the will, from the speculative ideas of the ancients, and has consequently only subordinated the facts observed to these ideas. The cause of the adoption of the theory, according to this view, was the ancient opinion that the soul occupied a fixed position in the centre of the organism; and this in turn was due to the notion that the soul was an unextended principle. As the soul was at the centre, impressions of sense must, of course, be conveyed to it, and, equally of course, motor

impulses must be conveyed from it. That this view was held by the ancients is proved from the writings of Democritus, Plato, Aristotle, Galen, &c.

The author next shows that anatomy is opposed to the view of double transmission, since (1) The nerves neither converge to a central point in the brain, nor do they diverge from such a point; and (2) Neither do they run isolated and autonomous from one end to the other. Various and differing opinions of different anatomists on these points are detailed, and it is shown that, according to the theory, the spinal cord ought, for instance, to be much more voluminous at its upper than its lower end, whereas, in fact, the more voluminous part is below; and in some animals, as the *Crotalus torridus*, the section of the lower part is eleven times greater than of the upper. The views of modern writers, as Vicq d'Azyr, Cuvier, Bell, Rolando, Gall, are vigorously attacked. As regards the isolated and autonomous course of the nerves, Haller's statement is quoted to show how the theory warped his observation:—"Every nervous filament," he says, "from its commencement to its termination is thus distinct, in order that the sensation of each filament may reach the brain, free from the sensations of every other filament." Henle, &c., wrote to the same effect. When observers, as Roudanowski, in 1865, showed anastomoses taking place between the nerve-fibres, it was assumed that the observations were erroneous; and when later observers again pointed it out, the condition was assumed to be one of very great rarity, and the very researches of anatomists, conducted by teasing the nerve-fibres with needle-points, were so arranged as to destroy the anastomoses. The necessities of the theory compelled observers to overlook the significance of ganglia, and specially of the nervous network found in many places in the body.

As regards the physiology of this question, it is shown that anatomical arrangements and physiological theories were assumed and framed in order to comprise the already accepted theory. With respect to Sir Charles Bell's discovery, our author thinks his merit consisted in having suggested a new process of experiment which he was the first to apply, not to objective researches, but to prove a particular opinion of his

own, viz. that the various functions of nerves, and, therefore, the function of transmission of sensory and motor impulses, had different fibres. His theory, according to our author, presupposed four propositions:—(1) That the seat of the sentient principle and of the will is in the brain. (2) The notion of double transmission. (3) The anatomical disposition imagined by the ancients to adapt the nervous system to this mechanism; and (4) The existence of distinct routes for the transmission of the sensory impressions and of the excitements to voluntary contractions. In fact, Bell's experiments, according to Dr. Panizza, had chiefly the effect of turning the attention of physiologists completely in another direction, viz., to the question, what were the directions taken in the roots and in the spinal marrow by the two opposite currents.

On the other hand, experiments which showed that section of the posterior nerve-roots did not quite abolish sensibility were overlooked; and particularly Arnold's experiments (on which, by the way, Mr. Lewes laid so much stress). This author divided the posterior nerve-roots, and then removed the skin so as to destroy the peripheral apparatus of tactile sensibility, and yet found that the animals perfectly executed movements depending on the perfection of muscular sensibility. Again Marshall Hall and others found they could obtain muscular movements by irritating the posterior nerve-roots.

The author then examines the theories framed to account for these results, contradictory of expectation; but cannot accept any of them as well founded. "Diffusion of the stimulus," "recurrent movement," &c., he sets on one side as unsatisfactory.

In an interesting section on the true signification of reflex movements, Dr. Panizza shows that the ordinary theory takes no account, either of the influence of ganglia, which may act as centres of reflection, or of the inosculations of nerves, which actually occur in plexuses and even in the course of nerve-fibres. The author distinctly maintains, however, the necessity for transmission in the case of voluntary movements, as distinguished from reflex.

The question of paralyses of sensation and motion consequent on division of nerves is then considered, and it is main-

tained that the usual explanation presupposes the theory of double transmission, and that some of the effects of division of nerves are quite opposed to the theory, as for example, when division of a nerve is not followed by loss of sensibility in the part supplied (as sometimes occurs).

The writer spends some time in attempting to establish the thesis, that excitability is a universal property of the nervous system. This he does by showing, that the gradual progress of discovery has resulted in the knowledge that parts formerly believed inexcitable have been shown to be so. Thus the optic thalami, the occipital lobes, and even the frontal lobes have, one by one, been found excitable so as to produce movements. The remarks made on this point are very interesting, and worthy of longer notice, but space forbids.

The law according to which the action of stimuli is propagated in the nervous network is, according to Marshall Hall and the author, this: From whatever point the irritation starts, the effects may be diffused by the nerve-fibres in all directions; and a corollary from this law is the position that a stimulus applied to any point whatever of the nervous system, may determine in various districts of the organism, and in all directions, the most varied disturbances of sensation and of voluntary motion.

The various hypotheses of physiologists to reconcile facts observed, with the acceptance of the theory, are then considered, and all are rejected, the author contending that the large (or, as he terms it, *infinite*) series of hypotheses required is itself an evidence of its incredibility. Even the acceptance of the theory of cerebral localisations seems to him to be upset by those by no means rare cases recorded in pathology, in which pathological destruction of the centres was not accompanied by loss of the movements supposed to be dependent on them.

The second part of the book, dealing with the physiology of sensation, is even more interesting than the former, but must be more shortly dealt with. The author proceeds on the same plan by showing how the notion originated with the ancient writers regarding the unextended character of the sentient principle. The whole question of presentation and

representation is discussed, and the question of vision is considered in all its bearings.

The author's conclusions are—

1. That the postulate of impressions has no foundation in experience.

2. That science, starting from this postulate, has at once been compelled to introduce into the explanation of the facts unextended principles.

3. That the explanation obtained thus, by means of hypotheses, into which unextended principles enter, requires, in the arrangement of the facts, many other hypotheses which are in contradiction to experience.

4. That science solves these difficulties only by entirely abandoning the extended, and betaking herself entirely to unextended principles.

5. That knowledge, built upon such a foundation alone, does not correspond with that which we have of objects, which was the point from which the inquiry set out, the fact whose explanation was sought. We may, therefore, conclude that that postulate is not at all conformable with the truth, and, like that of double transmission, must be absolutely dropped by a positive teaching.

Dr. Panizza's work is highly original, and we should be glad to see it translated. A. Rabagliati.

The Brain of the Cat (Felis domestica). Preliminary Account of the Gross Anatomy. By Burt G. Wilder, M.D., Professor of Comparative Anatomy in Cornell University, U.S., &c. Read before the American Philosophical Society, July 15th, 1881.

This is a contribution towards the supply of a want which the author considers as an opprobrium to anatomy, viz., the exact and detailed description of the brain in typical examples of various mammiferous animals. It is more limited in its aim than the memoirs of Meynert, Krueg and others; but with the object in view of describing certain forms of brain which could be most easily procured, Professor Wilder made choice of the

brain of the cat, not only on account of the readiness with which specimens may be obtained, but also because of its being less subject to variations from differences of race than that of the dog or some others of the carnivora. The memoir is illustrated by four plates, containing in all twenty figures, the most of which are twice the natural size, and have been drawn with great care from a considerable number of specimens, so as to present a fair view of the average forms.

The description is not systematic, but rather in the form of an explanation of the figures; nevertheless it is of such a nature as to bring out all the main features of external cerebral anatomy which they are intended to illustrate. Some new names have been invented for a certain number of the parts; while in regard to the greater number an attempt has been made, by slight changes, to adapt the received names to the author's views of a reformed system of anatomical nomenclature. There is, as is well known to most writers and teachers in anatomy, some risk in this attempt, and Prof. Wilder, in riding his hobby, has not entirely escaped from a tinge of pedantry which is apt to infuse itself into the straining after perfection in scientific nomenclature. But with this exception, on which, if we were disposed to be critical, we might have made further remarks, the account of the coarser anatomy of the cat's brain, which is given in this memoir, must be looked upon as a valuable contribution to the exact and detailed description of an important organ in a particular tribe of animals.

As Professor Wilder's memoir does not attempt more than a simple description and nomenclature of the parts, and does not enter into any comparison of the brain of the cat with that of other animals, or profess any general views, there is no ground for further remark in this notice. But we shall look with interest for the continuation of Professor Wilder's work in the minuter anatomy of the brain in the same animal; and we may hope on some future occasion to go more fully into an examination of that and other works on the comparative anatomy of this organ.

In conclusion, we would remark that there is very little doubt that the scientific knowledge of the structure of the central nervous organs is, in the first instance, mainly to be

advanced by the multiplication of carefully executed descriptions of individual forms similar to that which has now been under our notice, so as to furnish ample materials for their future comparison, and the consequent foundation of that more complete knowledge of cerebral structure which may prove a secure guide in the illustration of its physiological and pathological conditions. ALLEN THOMSON.

Lehrbuch der Krankheiten der peripheren Nerven und des Sympaticus. Von Dr. ADOLPH SEELIGMUELLER. 398 pp. Brunswick, F. Wreden, 1882.

THIS treatise on the diseases of the peripheral nerves and the sympathetic is one of a series of short medical text-books. In a second volume, the author intends to describe the diseases of the brain and cord in a similar style. Dr. Seeligmüller is known as an excellent practical observer, not wanting, however, in a thorough theoretical basis, and therefore it was to be expected that he would write a practical and useful book. The progress of neuropathology has been so rapid that works of ten years' standing have become nearly antiquated, and so the busy practitioner will be glad to find in Dr. Seeligmüller's book a short, but exhaustive and thoroughly scientific account of the present state of knowledge concerning the diseases of the nervous system.

The book is divided into a general and a special part. In the former the author gives an introduction to the study of nervous diseases, and describes the different methods of examining the functions of the nerves (sensibility, mobility, reflex-action); then he speaks of the causes of nervous affections in general; the main subject, however, of this general part is a chapter on the therapeutics of nervous diseases in general. This is subdivided in three parts: physical, chemical, and surgical remedies.

As physical remedies he describes, in the first place, the methods of applying cold and warm water; then follows a very good account of the use of electricity, illustrated by numerous diagrams of electric apparatus, the motor points, &c.

This is followed by a short account of the methods of massage and Swedish gymnastics.

The second division of this therapeutical chapter includes a description of the different drugs used in the treatment of nervous affections. We may mention here that the author has had good results by the use of Cannabis indica in doses of 0·2–0·5 gramme, as a hypnotic. For neuralgic pains he recommends Gelseminum and Butylchloral (croton chloral).

In the second part of his book Dr. Seeligmüller treats of the diseases of the peripheral nerves; neuralgia in its various forms, paralysis and spasmodic affections of peripheral nerves, and the anatomical diseases of the latter, neuritis, atrophy and neoplasms, injuries.

In the chapter on the diseases of the sympathetic, the author is well aware that some of these affections, such as Graves's disease, hemiatrophia facialis, cannot be considered as real diseases of the sympathetic nerve, but may have their origin in other nervous centres; for practical purposes, however, it is convenient to classify them with some other, more or less obscure, affections of the vaso-motor, trophic and secretory nerves, such as: sclerodermia, local asphyxia of the extremities, hydrops articulorum intermittens, &c.

Dr. Seeligmüller's book is an extremely useful, and valuable addition to the literature of neuropathology, and I have no doubt that an English translation of it would meet with great approval among medical practitioners. R. H. Pierson.

General Physiology of Muscles and Nerves. By Prof. Rosenthal (Erlangen). London, Kegan Paul, 1881. 8°. pp. 324.

Physiologie des Muscles et des Nerfs. By Prof. Ch. Richet (Paris). Germer Baillière, 1882. 8°. pp. 924.

Though these works have the same title, their scope is widely different. Prof. Rosenthal's volume (one of the "International Series") is intended to give a semi-popular account of the whole subject; a difficult task, but which could not be entrusted to more competent hands. Electro-physiology is fully treated, in fact occupies one-half of the volume. The

author accepts Du Bois-Reymond's hypotheses, and gives a masterly résumé of the whole question from this point of view. The English translation, though readable enough, contains a large number of peculiar expressions, literally translated from the German instead of converted into the received technical terms. Thus, why speak of muscle "pulsations" instead of "contractions"? Or again of "inductive currents," of "chains" (i.e. batteries), of "ganglion-balls," &c. At page 176 the following curious sentence occurs: "A wire of this sort applied to a conductor traversed by currents is called a *diverting arch* (i.e. a deriving circuit); the ends of the wire with which it touches the body to be examined are called the *feet of the arch*; and the distance between these feet is called the *distance of tension*."

Prof. Richet's stately volume consists of twenty-four lectures, chiefly devoted to the phenomena of nerve and muscle irritability. The electrical phenomena of animal tissues, and still more those of electrotonus, are treated less systematically than might have been expected. The author has studied the phenomena of muscular contraction on the cray-fish; and his results are both interesting and important. Many phenomena are clearly displayed in the lower forms of organisation, which in the higher are far more difficult to single out from the more complex manifestations of their functions. The comparative method in experimental physiology will, no doubt, throw great light upon the nature of the fundamental facts of muscular and nervous action.

The first twelve lectures discuss the alterations of muscular irritability under certain conditions, the phenomena of contraction and cadaveric rigidity, &c., concluding with some subjects of special interest to the physician: muscular sense, atrophy, "tendon-reflexes." We are rather astonished at the author's attributing the latter to a sensory stimulation of the tendon itself. Most of our readers will, however, find still more interest in the perusal of the second part of the work, relating to the nervous system. After discussing nervous irritability and vibration, the author devotes four lectures to the illustration of reflex action, its laws, modifications and manifestations. The great practical importance of a thorough

knowledge of reflex actions becomes daily more evident. They are the means of testing the condition of nerves and nerve centres; and upon them are based many of our most potent therapeutical measures. To them we are impelled to look for the explanation of some of the highest psychical functions of the human organism. Will itself, Prof. Richet believes, is reducible to reflex action, but is it impossible to believe to spontaneous discharges of energy in certain directions fixed by the original constitution and subsequent education of nerve centres? At any rate, however tempting the hypothesis which reduces all actions to reactions, our knowledge concerning nerve-cells, their nutrition and functions, is scarcely ripe enough to justify generalisation on such a scale. Full of interest are the lectures on the conditions of cerebral life, and on the cerebral irritability and reactions. A strong argument in favour of the direct excitability of the cerebral cells is the fact of their latency being longer than that of nerve fibres, and the longer duration of the effects of single stimulation. They are also much more readily exhausted.

The last lecture sums up the author's views on irritability in a comparison between this property as manifested by muscles, nerves and nerve centres.

The book is illustrated with many myographical tracings. Notwithstanding its somewhat bulky appearance, it is sure to be welcomed by all who wish to rest their study of morbid nervous action upon a solid physiological basis.

A Clinical Treatise on the Diseases of the Nervous System. By Prof. M. Rosenthal, of Vienna. Translated by L. Putzel, M.D. New York and London, Sampson Low & Co. 2 vols. 8°. pp. 530.

The term "clinical" must be understood in its German significance. The chief characteristics of the book lie rather in the stress placed on pathological anatomy. With reference to treatment, the English reader will find in it valuable hints with reference to hydropathic measures, a subject far too much neglected here.

Epilepsy and other Chronic Convulsive Diseases. By W. R. Gowers, M.D., F.R.C.P. Churchill. 8°. pp. 308.

This work, based on the Gulstonian lectures delivered by the author in 1880 (see 'Brain,' Vol. II. p. 262), is a conscientious monograph on epilepsy in the light of the most modern facts and theories, and based upon an extensive and well-digested personal experience. The chapter on treatment is well timed, as there is too much tendency now-a-days towards ignoring all drugs but the bromides, and failing these to give up cases as not susceptible of amelioration.

The Diagnosis of the Diseases of the Spinal Cord. By W. R. Gowers, M.D. 2nd ed. Churchill, 1881. pp. 86.

We owe a debt of gratitude to Dr. Gowers for having supplied a great want in medical literature. Within a small compass he has condensed, in a clear and accurate manner, all the important factors of spinal diagnosis. He treats successively of the medical anatomy of the cord, of its physiology in relation to the symptoms of its diseases, of the indications of position and of nature of disease. Histories of typical cases and a number of well-chosen diagrams serve to illustrate the principles laid down. The author's reading and clinical experience are equally extensive, and no better guide to the subject could be found. We hope every practitioner will avail himself of the advantages offered to him in this introduction to the study of spinal diseases.

On Chorea. By Octavius Sturges, M.D., F.R.C.P. Smith and Elder, 1881. Small 8°. pp. 200.

We agree with the author in thinking that the view generally taken of chorea in England is biassed by the exaggerated importance given to rheumatism in its pathogeny. The co-existence of the two diseases is far from being the rule. The heart itself may partake of the chorea movements; hence the cardiac disturbances observed. The fact that fright and

other psychical states are often the existing causes of chorea, and that girls are more subject to it than boys, show that it is rather a "functional" than an organic disease. It has certain points in common with hysteria. The resemblance between choreic jactitation and the purposeless movements of infants makes it appear as if the disease consisted in a temporary suspension of the educational acquisitions in the motor sphere. Dr. Sturges' book is most readable, good in style, and strong in argumentation.

Le Sommeil et l'Insomnie. By A. Marvaud, M.D. Paris, and London, Sampson Low & Co., 1881. 8°. pp. 140.

This little book, which has gained to its author one of the prizes of the "Académie de Médecine," is a useful digest of what is known of the physiology and pathology of sleep. The first part treats of the phenomena of normal sleep; the second, of insomnia, occurring either by itself or as a concomitant of other morbid conditions. The work concludes with a full discussion of the treatment appropriate to the various cases.

Compendium der Electrotherapie. By Dr. Pierson (Dresden). Third edition. 12°. pp. 200.

Guide Pratique d'Electrothérapie. By Dr. Onimus (Paris). Second edition. 12°. 320 pp.

Dr. Pierson's is an excellent résumé of the whole subject of electro-diagnosis and electro-therapeutics. The physical principles, which after all are the most important to have grasped for a successful medical application of electricity, are accurately stated. Electro-diagnosis (which is of purely German growth, if we except the fundamental discovery of Marshall Hall) and the methods of treatment are described with sufficient clearness to be of practical use to the reader.

Dr. Onimus's little book is a *résumé* of the author's teaching, based upon his own physiological experiments and clinical experience. The writer holds curious views, opposed to every physical principle, about "the high tension and low chemical action"

of currents. He also holds the *direction* of currents of supreme importance in therapeutical applications. We believe that the relative positions of the poles on the body is a matter of almost absolute indifference. The chapter on treatment is much enlarged; its dogmatic tone will make it undoubtedly acceptable to those who have to use the galvanic current without any previous knowledge of batteries and their manipulation.

A. DE WATTEVILLE.

Clinical Cases.

A FURTHER CONTRIBUTION ON THE COURSE OF THE OPTIC NERVE FIBRES IN THE BRAIN.

BY J. DRESCHFELD, M.D., M.R.C.P.

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In the last number of 'Brain,'[1] I recorded two cases bearing on the above subject. I beg leave to describe now the history and post-mortem account of another case, which again strongly supports the theory of a semi-decussation of the optic nerve-fibres, and presents also some other interesting neuro-pathological features.

Th. P., æt. 52, married, a plumber by trade, was admitted into the Manchester Infirmary on May 26th, 1881, suffering from hemiplegia, hemianæsthesia, and hemianopsia, all on the left side, and died at the Convalescent Hospital, Cheadle, on November 4th, 1881.

The following is a brief outline of the case as taken by Dr. R. Maguire, then clinical clerk:—

The *family history* is good. Patient does not know of any of his relatives suffering from gout or consumption; he has had five children, one of whom died when young from croup.

Previous history.—Has never had syphilis; his habits have been regular. When a boy had an attack of typhoid fever. He has always enjoyed good health, though much exposed to wet and cold, till four years ago, when he suffered from an attack of gout, since then he has often been troubled with bronchitis and asthma; during last winter he had an attack of lead colic. Twelve months ago he noticed that he had to get up often at night to pass urine, he had no pain in the head, no vomiting, no pains in his loins, or dropsy of any kind. About a month before admission he noticed his eyesight failing; the left eye being the worse; vision then became suddenly worse, so that he was almost blind, and could hardly

[1] Vol. IV. p. 543.

follow his employment. In a few days more he became suddenly paralysed on the left side, without any pain or loss of consciousness.

Condition on admission.—Patient is a spare man, fairly healthy in appearance. There is a little œdema of the eyelids, of both legs, and of the left arm; arcus senilis is well marked.

The patient is much depressed in spirits, and very emotional, but his intelligence is good; he, however, complains of slight loss of memory; he does not complain of any headache or vertigo; there is no aphasia.

There is slight left facial paralysis, affecting the lower facial muscles only. There is almost complete paralysis of left arm and leg; the leg is extended, the foot turned inwards; there are no traces of any contracture, and the arm and hand rest in any position in which they are placed.

The deep reflexes (patellar and ankle-clonus) are absent both from the right and left side.

The superficial reflexes (plantar, cremasteric, abdominal, epigastric and scapular) are absent on the left side, but normal on the right side.

There is complete anæsthesia of the whole of the left side, extending exactly to the middle line of the body. The patient feels tactile impressions very imperfectly, both in the trunk and the extremities of the left side; there is also analgesia of the left side, and when pricked with a pin in the arm the patient feels a peculiar sensation running up the arm towards the neck; if pricked in the leg a similar sensation runs up the thigh. The sense for temperature is likewise diminished on the left side.

The sense of taste and of smell is normal. Hearing is diminished on both sides, but more on the left than on the right. The vision is bad, and there is complete left hemianopsia; different colours are perceived on the right side in their physiological order, on the left there is complete absence of colour vision. Ophthalmoscopic examination of the eye shows swelling of the disc, numerous hæmorrhages, and white patches in the retina, chiefly round the papilla, but not round the yellow spot. The retinal arteries are scarcely visible, the veins over the papilla large and tortuous. The pupils are slightly dilated, but react, though somewhat sluggishly, to light.

There is no paralysis of any of the oculo-motor muscles.

The muscular sense is not affected on the left side. The muscles on the left side feel flabby, but there is no marked atrophy.

The electric contractility, to the galvanic and the faradic current, of the muscles on the left side is diminished.

There is no blue line on the gums. The tongue is large and moist, teeth marked and coated with a thick black fur. Appetite is good, there is no vomiting. Bowels confined. Defecation perfectly under control. The liver dulness and splenic dulness normal.

The voice is rather hoarse, there is a slight cough, with scanty, frothy expectoration of muco-purulent character. There is no dulness over the region of the lungs. A few moist rhonchi are occasionally heard.

Pulse eighty, full and incompressible. The arteries are rigid and tortuous. The heart's apex beat is visible in sixth intercostal space, and is outside the nipple. Heart's dulness increased both in the vertical and transverse diameter. The heart sounds are loud, the first somewhat clangy. The first sound is reduplicated over the ensiform cartilage, the second over the pulmonary cartilage.

Micturition profuse, sometimes involuntary. Urine s. gr. 1009, pale, acid; contains a small quantity of albumen; no sugar. Microscopically, a few granular casts.

Diagnosis.—The case was looked upon as one of chronic Bright's disease, in which cerebral hæmorrhage had taken place, implicating the left thalamus opticus and left internal capsule. The retinal changes, the marked cardiac hypertrophy, the slight amount of albumen, the low s. g. of the urine, and the small amount of œdema and absence of ascites made us look upon the case as one of granular kidney, or chronic interstitial nephritis, which view was also strongly supported by the etiological element in the case (the patient was a plumber by trade) and the history of an attack of gout. The dimness of vision which the patient complained of previous to his apoplectic attack was no doubt due to the albuminuric retinitis. The hemiplegia, hemianæsthesia and hemianopsia were best accounted for by assuming one apoplectic focus situated in the above-given locality. The primary renal disease, the sudden onset, the absence of headaches, vomiting, &c., all pointed in favour of hæmorrhage rather than a tumour.

Progress.—The patient's condition remained unaltered for some time. Experiments were tried with an electro-magnet, but without causing diminution of the anæsthesia; a solenoid was also applied around the left lower extremity for some hours daily, with no better results.

On July 1st, 1881, the patient developed an attack of gout in the left great toe; and there was also diagnosed slight pleuritic effusion on the left side. Some of the fluid was withdrawn from the pleura by a subcutaneous injection syringe, and when treated after Garrod's method to show crystals of uric acid, gave positive results.

During the next few months the patient's condition underwent but little change.

The hemiplegia slightly improved, especially as regards the leg; the patellar reflex, which was noticed as absent before, became slightly marked on the left side, but not on the right. There was no tendency to contraction.

The anæsthesia remained unaltered.

The hemianopsia also remained unchanged.

The optic neuritis and retinitis gradually underwent the degenerative changes, and numerous white patches were visible, whilst the swelling of the disc receded.

The albuminuria remained the same; occasionally the urine contained a small amount of blood.

The œdema of the legs and eyelids disappeared, the gouty toe assumed its natural appearance, and the pleuritic effusion diminished somewhat, though not entirely. In this condition the patient was sent, on September 10th, to Cheadle, where he remained in very much the same condition up to November 4th, when he had another attack of cerebral hæmorrhage, became comatose, and died the same day.

A *post-mortem* was made by Mr. A. H. Young, pathologist to the infirmary, on November 5th.

On opening the skull it was seen that the left cerebral hemisphere felt firmer than the right, but was not otherwise altered. The brain was removed, and on section the interior of the right hemisphere was found to be the seat of a very extensive hæmorrhage, which had considerably damaged and ploughed up the basal ganglia and distended the ventricle, but left the cortex perfectly intact. It was impossible, even after hardening of the brain, to make out the seat of the original lesion; the optic tract and the geniculate bodies, however, were found to be perfectly healthy and quite away from the hæmorrhage. The medulla and cord were taken out and examined microscopically, after having been sufficiently hardened, and no changes whatever were found; there was neither descending degeneration of the lateral columns nor any changes in the posterior columns, either in the dorsal or lumbar region.

The heart showed marked hypertrophy of the left ventricle; the kidneys were small, red and granular, and showed changes affecting chiefly the capsules of the glomerule and the interstitial tissue. (The kidney changes in this case, and in some other cases of Bright's disease from chronic lead poisoning, have some important bearings on the pathology of Bright's disease, and will be the subject of a separate publication elsewhere.)

Remarks.—While the above reported case, owing to the

very extensive second attack of hæmorrhage to which the patient succumbed, does not allow us to exactly localise the exact situation of the lesion which produced the hemianopsia, it yet forms another valuable support for the theory of the semi-decussation of the optic nerves in the optic tract. It shows further that the hemianopsia was not due to pressure or disease on the tract or the corp. genicul., for these parts were found perfectly normal and quite away from the seat of hæmorrhage.

Another interesting feature in the case is the absence of tendon reflexes on both sides (on the left they were slightly marked during the last few months of life) and the absence of any tendency to contracture in the paralysed limb. The spinal cord showed absence of any descending changes. But no changes similar to those described by Westphal (in one case of general paralysis of the insane with absence of tendon reflexes) and situated in the inner side of the post. horn could be made out in this case.

GLIOMA OF THE RIGHT OPTIC THALAMUS AND CORPORA QUADRIGEMINA.

BY DAVID FERRIER, M.D., F.R.S., &c.

Physician to the National Hospital for the Paralysed and Epileptic.

THE following case seems worthy of record, as illustrating the exceedingly complex symptomatology of tumours affecting the *tegmentum* and its ganglia.

Samuel H., æt. 14, was admitted into the National Hospital for the Paralysed and Epileptic on May 13, 1881.

Previous History.—The commencement of the present illness was thought to be a fall on the ice four years ago. Some kind of fit occurred the day after, but there were no other symptoms till September 1880. In this month he had a fit. In October he had a series of fits, with general convulsions and retraction of the head, lasting for about five hours. Next day he had frequent vomiting. In November he had two fits of a similar character. It was observed that after these fits the right eye was turned inwards, and continued so more or less for six weeks. At this time there was diplopia. Several fits occurred, and occasional sickness subsequently, but he was apparently improving and getting stronger, till a month before admission, when it was observed that he began to reel in his gait, and to use the left leg with difficulty. There seemed to be a tendency to reel specially towards the left side. This difficulty in walking had gone on increasing, so that he had not been able to walk alone for a fortnight.

The left arm was also observed to be affected, being unsteady and weak, so that the patient could not use his left hand to button his clothes or hold a fork.

The patient had occasional attacks of headache, and also pain and numbness in the left arm.

State on admission.—Exceedingly dull and heavy-looking, but responds intelligently to questions as to name, age, &c. Says he has pain in the back of the head on pressure or percussion.

Motility.—The ocular movements upwards and downwards are normal, but the lateral sweep is restricted, especially towards the left and a nystagmus tends to occur.

The power of converging the eyes is entirely lost, so that he cannot look at the tip of his nose or at an object brought close up to the eyes. Nor do the pupils show any contraction at the attempts at accommodation. The movements of the face, tongue and jaws are apparently unaffected. The arms retain all their movements, but the left is distinctly weaker than the right. Grasp of right hand = 45 lbs., of left = 15 lbs.

The left hand also oscillates when he attempts to touch or lay hold of anything. Otherwise it remains still and free from all tremors or twitching.

The legs can be moved freely in all directions when he is lying in bed, though the left is distinctly feebler than the right. In attempting to walk he lifts the left foot high off the ground, and throws it forward in a brusque and ataxic manner. Cannot walk without support.

The patellar reactions are very distinct, the left excessive and almost tetanic. Ankle-clonus is readily induced in the left. The plantar, abdominal and epigastric reflexes are obtained on both sides, but better on the right side. The cremasteric reflex was obtained equally on both sides.

Sensibility.—Tactile sensibility is somewhat defective on the whole of the left side, but painful impressions are felt and correctly localised.

Sight is good. He can read 3 Nettleship with the right eye, and 4 Nettleship with the left.

The pupils are equal and react to light, but there is no alteration on attempts at accommodation. Ophthalmoscopic examination reveals well-marked double optic neuritis.

Hearing is better on the right than left side. Hears a watch at 18 in. on left, at 3 feet on right.

Taste and smell good, and no appreciable difference on the two sides.

The subsequent history may be summed up shortly as increasing torpor and mental hebetude, greater weakness, amounting towards the end to complete paralysis of the left arm and leg, and partial of the face, with anæsthesia and analgesia. Fits occurred at intervals, varying somewhat in character, but all tonic rather than clonic.

In one, in which he was carefully observed by the attendant, both arms were rigidly flexed, the legs flexed at the knees, the face drawn to the right, the head retracted, and both eyes widely opened and turned upwards. The spasms lasted about a minute, and were followed by unconsciousness for several hours.

In another attack the arms were rigidly extended, the hands clenched, the head retracted, and the legs extended.

In other attacks, again, there was loss of consciousness with-

out muscular rigidity. In one of these, seen by Dr. Beevor, he lay with the body turned to the left. The face was dusky and covered with perspiration. The right pupil was somewhat larger than left, and insensible to light, while the left reacted slightly. Slight external strabismus existed in the right eye. The breathing was slow, and the pulse sixty per minute. At the end of ten minutes he opened the eyes and moved the right limbs a little. Well-marked ankle-clonus and knee-jerk existed in the right. Plantar reflex was obtained on the right side slightly, but none on the left.

Death occurred on July 27th, 1881.

Post-Mortem Examination.

The skull cap was thin. The dura mater was not adherent, but tense. The convolutions were somewhat flattened and dry, the veins being large and distended. The right parietal region felt firmer than the rest of the brain.

The cranial nerves were intact.

The posterior extremity of the right optic thalamus projected behind the crus cerebri and was in contact with the middle peduncle of the cerebellum,—as seen from the base.

On division of the corpus callosum and exposure of the lateral ventricles and corpora quadrigemina, the right optic thalamus and right side of the corpora quadrigemina appeared, while retaining their normal shape and surface, larger than the left.

The greatest antero-posterior diameter of the right thalamus was 2 inches, while that of the left was 1½ inch. The greatest width of the right was 2 inches, but of the left only ¾ inch.

The right anterior tubercle of the corpora quadrigemina was about ¼ inch larger in all its diameters than the left. The consistence of the enlarged parts was softer than the normal structure, and, on section, the surface presented the characteristic appearances of a glioma shading imperceptibly into the surrounding tissue. The internal capsule and ganglionic structure of the right thalamus and lenticular nucleus were indistinguishable, and the sub-thalamic region and tegmentum of the crus cerebri seemed involved in the growth. The foot of the crus cerebri and locus niger were distinct. The nucleus caudatus, internal capsule and anterior part of the lenticular nucleus were of normal appearance. The enlarged optic thalamus pressed backward against the anterior or quadrangular lobe of the right side of the cerebellum, and was closely appressed and moulded against this and the middle peduncle. This portion of the cerebellum, instead of having the laminæ running parallel to each other, forward and outwards, had an

irregularly convoluted appearance, as if thrown into folds by compression into a smaller space. The pressure on the anterior surface of the right lateral lobe caused the inferior semilunar lobe to be closely applied against the medulla oblongata. The right lateral lobe of the cerebellum was thus somewhat distorted, and the antero-posterior diameter shortened by about half-an-inch as compared with the left.

Remarks.—The diagnosis made in this case, viz., a tumour affecting the right side of the cerebellum and pons, was only approximately correct, nor does it seem possible, even with the actual condition before us, to infer otherwise than vaguely as to the symptoms which should have existed during life.

The fact of the existence of an intra-cranial tumour was indicated by the general symptoms, viz., headache, vomiting, optic neuritis and convulsions; and the reeling gait and tonic or tetanic nature of the convulsive attacks were symptoms which, as has been pointed out by Hughlings-Jackson, are characteristic of tumours of the cerebellum. That the cerebellum was affected here is very clearly shown by the compression and distortion of the right lobe by the backward growth of the tumour; but whether in this, or in the cases where the tumour is actually in the cerebellum itself, the tonic spasms are in reality cerebellar, or, as I am more inclined to regard them, due to irritation of the corpora quadrigemina, is a point not to be decided by clinical facts alone. But the facts of experimental irritation of the cerebellum and of the corpora quadrigemina, which I have elsewhere described, would lead me to regard the affection of the corpora quadrigemina as the essential factor. In this case the corpora quadrigemina were directly implicated by the invasion of the morbid growth, and several of the symptoms seen in this case have been found in connection with tumours in this neighbourhood. Disorders of locomotion, and certain forms of ophthalmoplegia have been specially noted. In this case there was total paralysis of the convergence of the eyes, and weakness of the lateral movements, the others being unaffected. But there was no paralysis of reflex irido-motor action, and there was scarcely any appreciable difference in the power of vision in the two eyes. The right was a trifle more acute than the left. The locomotor incoordination was very marked, not merely disorder of equilibration, but a curious ataxic action of the legs. Cases of impaired co-ordination of locomotion with ophthalmoplegia in connection with indications pointing to disease in this region have important physiological bearings. The partial hemiplegia of motion and sensation on the left side, which might have been the result of a tumour of the right half of the cerebellum pressing on the subjacent

sensory and motor tracts, was no doubt, in this case, due to the direct implication of the right internal capsule which was involved in the growth. The oscillations of the left hand on volitional movements may be regarded also as due to the same cause, in accordance with the observations of Charcot in reference to the lesion in post-hemiplegic choreic disorders.

Abstracts of British and Foreign Journals.

Schultze on recovery from Tabes. (*Archiv f. Psych.* xii. p. 232).—Erb in his article on Tabes dorsalis (*Ziemssen's Encycl.* vol. xiii. p. 598, American edition) writes:—"But even where the disease is fully developed, we may, although very rarely, see recovery take place, or an improvement bordering on recovery. I am in possession of two cases which demonstrate this. In the one, there were lancinating pains, unsteadiness, weariness, and distinct ataxy of the legs, paræsthesiæ in the domain of the ulnar nerve, and vesical weakness. After the malady had lasted for several years, the patient recovered completely with the exception of slight vesical weakness; and for the past few years he has attended to his duties as a civil functionary without suffering any inconvenience."

This same patient came under Dr. Schultze's observation in June 1880, and the only symptoms of spinal disease that he presented were absence of the patellar reflex and a degree of paralysis of the bladder, with vesical catarrh. The gait was normal, and there were no disorders of sensibility. The patient died suddenly of corrosive poisoning, twelve years after the appearance of the initial symptoms, and eight years after the disappearance of the more prominent symptoms of tabes. *Post mortem*, there was found diffuse degeneration of the posterior columns in the lumbar region; degeneration of the outermost parts of Burdach's columns in the dorsal region (least marked in the lowest fourth of the dorsal portion of the cord); partial degeneration of Burdach's columns, especially of their outer portions, and slight affection of the columns of Goll in the cervical region.

This case shows that though most of the symptoms of tabes may disappear, the pathological appearances characteristic of the disease may still be present. It also lends support to the view that, even in the early stages of tabes, important organic changes are to be found, and proportionally discredits the theory of a purely symptomatic stage of the disease prior to the stage of organic change.

From a clinical standpoint there is no doubt that this case may

very fairly be looked upon as a case of tabes with almost complete recovery. And it may be argued that from an anatomical standpoint the absence of granule-cells, which generally appear in the earlier stages of atrophic processes, justifies the same conclusion; in other words, that we have to deal with simply the remains of an old degeneration, and not with an actually progressing degeneration. It must be remembered, however, that the absence of granule-cells is not a very reliable test in this matter, for, if the atrophy of nerve fibres develops slowly, granule-cells may not be found.

Schultze calls attention to several interesting features in this case: (1) Though there was lesion of the outer parts of the columns of Burdach in almost every part of the cord, there was not persistent ataxia. Schultze does not regard it as proved that degeneration of Burdach's columns causes ataxia, and he thinks that the results of this case, and also the fact that where there is extensive degeneration of the posterior columns the ataxia may spontaneously disappear, show that other inhibitory or reinforcing influences are operative in the production of ataxia besides the lesion which forms the spinal substratum. (2) The case teaches us that a moderate degree of degeneration in the posterior columns of the lumbar enlargement need not cause permanent anæsthesia or paræsthesiæ. (3) There was general atrophy of the cord, with diminution in the number of the nerve fibres. This atrophy, which is not usual in cases of tabes, was observed chiefly in the dorsal and lumbar parts of the cord, and was not confined to the posterior columns, but was also found in the lateral columns, and in the anterior cornua of the lumbar enlargement.

Wolff on a case of Tabes.—Wolff (*Archiv f. Psych.* Bd. xii. p. 44) reports a case of tabes with unusual clinical and pathological complications. The patient was a woman, aged 57. Her illness commenced with smarting pains in the lower extremities, and in a few weeks tremors of the upper extremities were observed when voluntary movements were made. Subsequently, ataxia of the lower limbs, paræsthesiæ, loss of the patellar reflex, occasional tension of the muscles of the limbs, incontinence of the urine and fæces, and decubitus, developed, and the patient died after an illness of 14 years. The occurrence of tremors of the upper extremities, along with the symptoms of tabes, rendered the diagnosis difficult. The case might have been a disseminated cerebro-spinal sclerosis in which the posterior columns were specially affected, or a multiple sclerosis with fascicular degeneration of the posterior columns,

or a sclerosis en plaques of the cord alone, with special complication of the posterior columns, or with fascicular degeneration of these columns.

At the necropsy there was found fascicular degeneration of the posterior columns, and in addition two foci of degeneration. The larger focus was immediately below the cervical enlargement, and implicated the anterior columns and portions of the lateral columns and anterior cornua. The smaller focus was at the level of the third cervical nerve, and affected the lateral columns only. There was thus a combination of tabes dorsalis, with the simplest form of a disseminated sclerosis of the cord. Whether the disease, in this case, is to be regarded as primarily interstitial (a primary meningitis) or parenchymatous, Wolff is unable to decide. Many facts point in favour of the former view; e.g. the universal thickening of the pia mater of the cord, the marginal degeneration found in the medulla oblongata and in various parts of the posterior columns, and the thickening of the ependyma in the upper part of the medulla oblongata, where no degeneration was visible. On the other hand, there was no local indication of a spinal meningitis, such as pain along the vertebral column, rigidity, &c. Wolff suggests that the disease of the posterior columns may have been parenchymatous, while that of the two foci was of a meningo-myelitic nature.

The tremor of the upper extremities Wolff attributes to the patch of degeneration immediately below the cervical enlargement.

Schulz and Schultze on Acute Ascending Paralysis.—In Landry's paralysis, or as it is better termed acute ascending paralysis, the electrical excitability of the paralysed nerves and muscles is normal. There are cases, though, which present the general character of acute ascending paralysis but differ from it in exhibiting a rapid diminution of the electrical excitability. In this respect they resemble Duchenne's paralysis (poliomyelitis anterior subacuta); but they are distinguished from it again by the peculiar upward march of the paralysis, and by the fact that disturbances of respiration and bulbar symptoms are regularly present.

An example of this intermediate type is recorded by Schulz and Schultze (see *Archiv f. Psych.* Bd. xii. p. 457) as occurring in a man with a syphilitic history. After a lengthened prodromal stage the disease rapidly developed, and terminated fatally in four weeks. Diminution of electrical excitability and the reaction of

degeneration were only observed towards the end of the illness. There were also slight paræsthesiæ and transitory vesical weakness.

The post-mortem showed acute myelitis of the anterior cornua of the lateral columns (especially of the pyramidal tracts), and in some places, and to a less extent, of the anterior columns. The morbid appearances were found through the entire length of the spinal cord, and in the lower part of the medulla oblongata. Though the evidences of disease were most marked in the localities named, the whole cord showed signs of a degree of inflammation.

It will be seen that this case presents a further point of difference from the typical acute ascending paralysis in possessing such marked pathological features, for in the typical Landry's paralysis the pathological appearances are *nil*. The authors suggest that the acute ascending paralysis may perhaps represent the lightest form of a general spinal and bulbar inflammation, in which the lateral columns and the medulla are specially affected. In some cases the inflammation might be so slight that it would be very difficult of detection even by the best observers; in other cases there would be more decided evidences of inflammation. For example, in Eisenlohr's case of acute ascending paralysis, there were minute hæmorrhagic extravasations and small collections of white blood corpuscles and leucocytes in the medulla, and to a less extent in the cord. The authors mention a case that came under their own observation, in which, shortly before death, the facial nerve became paralysed, and its electrical excitability diminished; and in which the autopsy revealed meningitis and acute myelitis with swelling of the axis-cylinders through the whole length of the spinal chord, particularly in the lateral columns of the cervical and dorsal divisions of the chord. We may mention here another case of the authors' which shows by what gradations we may pass from Landry's to Duchenne's paralysis. Clinically the symptoms were those of Duchenne's paralysis, except that there were symptoms of disordered respiration, and the extent of the paralysis did not declare itself in the first few days. Pathologically there was a very intense poliomyelitis anterior, and a slight affection of the lateral columns, characterised by swelling of the axis-cylinders—the very opposite of the conditions found in the first mentioned case, where the myelitis of the lateral columns was the prominent lesion, the poliomyelitis the less important lesion.

The authors observe that it will aid us in the diagnosis of these cases if we recollect what the first-mentioned case proves, that we

may have an acute poliomyelitis with myelitis of the lateral columns without spastic symptoms.

Senator on Bulbar Paralysis.—Hemianæsthesia alternans has hitherto been regarded as a symptom pathognomonic of lesions of the pons. Recently, however, Senator has reported a case (see *Archiv f. Psych.* Bd. xi. p. 713) which shows that this symptom may be present though the lesion is confined to the medulla oblongata.

A man, aged 56, without losing consciousness was seized with an attack of vertigo. He had the greatest difficulty in swallowing, had a tendency to fall to the left side, had a feeling of cold in the left half of his face, and had an affection of speech which gave one the impression that he was suffering from some obstruction in the pharynx or larynx. There were no symptoms of motor paralysis, except that the tongue was protruded a little to the left, and the left eye appeared somewhat smaller than the right. The temperature was normal, but the pulse beat 120 per minute. Five days later he was seen by Senator. He was then complaining of difficulty in swallowing, of hunger, and of want of breath. Sensibility was almost completely lost in the left half of the face, and in the whole right half of the body, as well as in the right arm and leg. Attempts to swallow either liquids or solids caused hawking and choking, and the substance was returned, sometimes through the nostrils. The voice, once powerful and clear, had become a whisper, and there was still the inclination to fall to the left. The patellar tendon reflex was absent on both sides.

For a week there was little change in his condition. Examination with the laryngoscope showed partial paralysis of the vocal cords. The electro-cutaneous sensibility was either lost or very much diminished in the left face and in the right half of the body. The patient died of putrid bronchitis and broncho-pneumonia, after an illness of fourteen days in all.

The *post-mortem* revealed a small focus of softening in the outer portion of the left half of the medulla oblongata, and thrombosis of the left vertebral and posterior inferior cerebellar arteries. The greatest length of the focus was about 1 cm.; superiorly, it did not extend so far as the pons; inferiorly, it did not reach the level of the plane drawn through the calamus scriptorius. The greatest breadth of the focus was attained a little below the middle of the olivary body; here the restiform body and the contiguous portions of Burdach's column and of the lateral column, the ascending root of the fifth nerve, the motor nucleus of the vagus and a portion of

the fibre of the vagus, were all implicated. The olivary body, the root of the hypoglossus, and the nuclei of the hypoglossus and vagus were quite intact.

Senator remarks that the difficulty in swallowing, the snuffling speech (due to paralysis of the pharyngeal muscles), the altered voice, the rapid pulse, the hunger and the feeling of want of breath were all symptoms indicative in this case of lesion of the vagus nerve. The absence of vaso-motor disturbances (with the exception of a slight and transient lividity of the right arm), of polyuria and glycosuria, is worthy of note; as also the fact that there was no marked defect in the knowledge of the position and lie of the right extremities, notwithstanding the loss of ordinary sensibility in them.

Bernhardt on Athetosis. — The author (*Archiv f. Psych.* Bd. xii. p. 495) reports a case in which there were successively hemiplegia, hemichorea and athetosis. The observation is of special interest, in the first place as showing that hemichorea and other abnormal involuntary movements may develop as post-hemiplegic conditions, and in the second place as confirming Bernhardt's theory, that athetosis is merely a modified chorea.

The patient was a woman, aged 20. The nature of her first illness is unknown, but it commenced with swelling of the feet, and an affection of the speech which grew gradually worse; and in the course of a few weeks there was complete right hemiplegia and aphasia. Seven months after the commencement of her illness she was seen by Dr. Bernhardt, who observed that the right leg was dragged in walking; that the right hand and arm and upper arm were in continual, restless, rapid motion (chorea post-hemiplegica); that there was right hemianopsia; and that there was aphasia, the patient being unable to name objects or to select those named, or to repeat the names after any one.

Iodide of potassium was administered in large doses, and within five weeks a marked amelioration took place. The aphasia had greatly improved, and instead of the choreic movements of the whole arm there were only continual, slow, grasping movements of the fingers. In other words, the hemichorea had subsided into athetosis.

Three years afterwards, Bernhardt saw and examined the patient, and found her condition substantially the same. There was still athetosis and right hemianopsia. The patient continued to drag the right leg a little, and the symptoms of aphasia had not altogether disappeared.

Binswanger on Secondary Degenerations after Lesions of the Cortical Motor Centres.—Binswanger (*Archiv f. Psych.* Bd. xi. p. 727) reports the following cases to show that lesions of the motor portion of the cortex cerebri or of the adjacent portions of the medulla do not always cause secondary degenerations of the pyramidal strands. In each of the cases the basal ganglia and the internal capsule were found intact, and there was no trace of secondary degeneration of the pyramidal tracts either in the spinal cord or the brain.

(1) A man, aged 48, was seized in March, 1880, with an attack of convulsions. He had another attack in a month, and subsequently the convulsions returned about every ten days. They began in the right hand. In September he had three fits in one day. After this he had no more fits, but a weakness, first of the right arm, then of the face and leg, became noticeable, and gradually increased in degree, until, in November, there was complete right hemiplegia and aphasia. The sensibility of the paralysed limbs was carefully examined, and found intact. The man died on November 25. The *post-mortem* showed a small focus of softening at the place of junction of the second frontal convolution with the ascending frontal, and the adjacent part of the upper third of the ascending frontal convolution was soft and pulpy and of greyish-yellow colour. Underneath this softened cortex was a cavity, as large as a walnut, full of clear brown fluid, which was separated from the outer capsule and the roof of the lateral ventricle by a narrow strip of healthy medulla.

(2) A woman, aged 56, who had suffered for some months from headache and vertigo, and had had a slight and transient paralytic attack on the left side, was shortly afterwards seized with a second attack, and died in a few weeks. In this second attack there was complete paralysis of the left face and arm, and partial paralysis of the left leg. *Post-mortem:* the posterior parts of the superior and middle frontal convolutions, and the upper third of the ascending frontal convolution were found occupied by a tumour about the size of a hen's egg; and the cortex here was of abnormally dense consistence. In the medulla, outside the corpus striatum, and somewhat more anteriorly than the first tumour, was a second larger tumour, which was bounded superficially and deeply by a layer of softened brain substance.

(3) A woman, aged 50, who was operated on in April for carcinoma of the breast, soon afterwards had an attack of convulsions which affected the right arm and face, and was followed by

paralysis of the right arm and face and by some aphasic symptoms. The hemiplegia became completed before death. The patient died on June 25, and at the autopsy a tumour, almost as large as a hen's egg, was found imbedded in the middle of the fissure of Rolando. The surrounding brain-substance was softened and discoloured.

Binswanger does not attempt to explain how it is that in some cases of lesion of the motor region there is, and in other cases is not, secondary degeneration of the pyramidal tracts.

W. J. Dodds, D.Sc.

Rumpf on the Treatment of Locomotor Ataxia with the Electric Brush. (*Neurolog. Centralblatt*, 1882, Nos. 1 and 2.)—The results obtained by the author are so striking that we should have felt incredulous had not they been related with full details by so competent an authority as Dr. Rumpf.

The first case was that of a man who, ten years before, had been attacked with the characteristic shooting pains. The usual symptoms manifested themselves in the course of time: extreme ataxia, anæsthesia and analgesia, abolition of reflexes, sense of fatigue, bladder disturbance, sleeplessness, &c. The electric brush was used along the back and legs, and very soon this brought about a considerable amelioration of the subjective symptoms, and the treatment persevered in for two months, when the patient declared himself prepared to resume his work. One year after this, Dr. Rumpf had the opportunity of showing him to the members of the Medical Society of Düsseldorf. The only symptom then present was the absence of the knee-jerk.

Dr. Rumpf stated that he had tried the brush in a series of cases successfully, though the results require time before they can be pronounced permanent. He described one, however, in which the patient had been in good condition for two years after a six weeks' course of treatment. He had had lightning pains, paræsthesiæ and ataxia, impotence and disturbed micturition. The knee-jerk was not abolished. All the symptoms disappeared except the sexual weakness.

Eulenburg on the Latency of the so-called "Tendon Reflex." (*Zeitschrift für Klinische Medicin*, i. 1882.) — The author has measured the latency of the knee-jerk on more than 100 healthy individuals, and finds it to vary between ·0016 and ·032 of a second. These numbers are much smaller than those hitherto given. He calculates that the time necessary for a reflex

act would be longer than ·03, hence concludes to the direct action of the stimulus on the muscular fibres.

Friedreich on Paramyoclonus multiplex. (*Virchow's Archiv*, vol. 86, No. 3.)—Prof. Friedreich relates the case of a man, aged 50, who shortly after a great fright, five years ago, became subject to curious symmetrical clonic spasms in a number of muscles (arms and legs). When quiet the number of jerks amounted from ten to twenty, when excited, to forty or fifty, a minute. During sleep he was free from them, and voluntary movements brought them to a standstill. Except an increase in the skin reflexes and in the knee-jerks, he presented no abnormal phenomena. Galvanism was successfully applied. The author explains the symptoms by assuming an abnormal irritability of certain groups of motor cells in the anterior horns.

Lannois on a case of Paralysis of the Brachial Plexus. (*Revue de Médecine*, 1881, p. 988.)—The author gives a case which is a counterpart of those first described by Erb, and in which, owing to disease of the fifth and sixth cervical roots there is paralysis and atrophy, of the scapular muscles, deltoid, biceps, and supination. In the subject of this observation all the muscles of the hand and forearm, except the supinators, were affected. Sensation was correspondingly impaired. This points to an affection of the cord formed by the seventh and eighth cervical and first dorsal roots. (Cf. 'Brain,' Vol. II., p. 584.)

Charcot on the Galvanisation of the Brain of Hypnotised Hysterical Patients. (*Progrès Médical*, 1882, Nos. 2 and 4.)—Prof. Charcot relates some curious experiments showing that the cerebral centres apparently share in the general hyperexcitability observed in certain hypnotised subjects. Makes and breaks, more especially voltaic alternatives, of a current from four to ten Leclanché cells applied to one hemisphere, gave rise in several cases to muscular contractions on the opposite side of the body. In the waking condition no such effects were ever produced. In other patients the muscular contractions occurred on the same side of the body. Finally, in cases where peripheral hyperexcitability was not well marked, the effects of cerebral galvanisation were but slight. Prof. Charcot expresses himself with great reserve on this subject, which is full of interest, but this requires much careful investigation.

Müller and Ebner on Nerve Stretching. (*Wiener Klinik*, vii. 1881.)—This paper contains a full account of all that had been published up to date with reference to nerve-stretching and its results. The authors then describe very minutely two cases in which the operation relieved the ataxy and anæsthesia, and removed agonising pains to which the patients had been subject 22 and 14 years respectively. They do not, however, look upon the remedy as more than a symptomatic mode of treatment, with (probably) temporary effects only. Nevertheless, with these restrictions its results are often very striking, and unapproached by those of any other therapeutic measure.

They discuss the explanations hitherto given of the "modus agendi" of nerve-stretching, and take the view that it acts less by its coarse anatomical than by its finer molecular effects, which spread to the nerve endings and spinal centres. The experiments of Brown-Séquard on animals, repeated by Scheving, Debove, Quinquaud and others, prove an alteration in the spinal functions consecutive on stretching the sciatic.

The result of nerve-stretching in affections of the sensibility will depend less upon the character of the symptoms (paralgesia, anæsthesia, neuralgia, etc.) than upon the possibility of influencing the spot of the irritative lesion by means of the mechanical stimulus. Hence the doubtful value of the operation when the seat of morbid action is in the brain. In cases of motor hyperexcitability, it is indicated when the lesion is spinal (facial and accessory spasm, dissemitrated sclerosis, late rigidity, etc.). It is useless in paralysis agitans, athetosis, and the like. In traumatic tetanus it ought to be resorted to without delay. It may be of use in certain trophoneuroses.

Gowers on a Case of Loss of Taste from Disease of the Fifth Nerve. (*Journal of Physiology*, vol. iii. p. 229.)—A female, aged 32, after certain premonitory symptoms was seized with sudden anæsthesia and paralysis of the parts innervated by the right trigeminus. No history of syphilis. It was found that on the whole right side of the tongue the sense of taste had disappeared. The lesion probably involved the fifth nerve near the pons; and though the glosso-pharyngeal nerve was intact, yet the nerve of taste had disappeared from those regions usually believed to be innervated by it. The author concludes that in this patient all the gustatory fibres arose from the fifth nerve.

Erb on the Course of the Gustatory Fibres of the Chorda

Tympani to the Brain. (*Neurolog. Centralblatt*, 1882–4.)—The author relates a case which seems to settle this still doubtful point. There was (1) Abolition of the sense of taste on the anterior two-thirds of the tongue on the right side. (2) Integrity of the facial nerve. (3) Lesion of the whole fifth nerve at the base of the skull. This concomitance of symptoms indicates that the gustatory fibres of the chorda tympani reach the brain through the trunk of the trigeminus.

More lately still, Erb has had the opportunity of observing two cases of disease of the fifth nerve. (*Neur. Centralbl.* No. 5.) In the first there was hypæsthesia on the left side; but no paralysis, nor disturbance of the hearing. The sense of taste was considerably diminished on the interior left half of the tongue. In the second there was paralysis of the muscles of mastication on the right side, with complete anæsthesia of the parts innervated by the third branch of the fifth.

Prof. Erb concludes that the gustatory fibres of the chorda tympani run into the second branch of the fifth.

A. DE WATTEVILLE.

N. Bubnoff and R. Heidenhain on Phenomena of Excitation and Inhibition in the Cerebral Motor Centres. (*Pflüger's Archiv*, vol. xxvi. p. 137.)—The above paper contains in the first place observations confirmatory of those of Franck and Pitres on the reaction-time of the cerebral grey matter to electrical stimuli (Trav. du Lab. de M. Marey, 1878–1879, p. 413); it further gives an account of the experimental determination of certain excitatory and inhibitory cerebral effects as indicated by simple spasm, storm of spasms or continuous contraction, or by the arrest of these states.

Franck and Pitres found that the reaction-time is considerably longer when excitation is applied to the grey substance, than when it is applied to the subcortical white substance (e.g. 0·065 sec. in the former case, 0·045 sec. in the latter). Bubnoff and Heidenhain confirm this fact. They find that the muscle-curve obtained by cortical excitation has a longer latency and a greater duration than that obtained by medullary excitation (e.g. the latency in the former case being 0·08 sec., in the latter 0·035 sec.); they observe further that the length of the interval varies inversely with the strength of excitation. All these observations go to show that time is occupied by the excitatory process in the cortex, and contradict the opinion that the grey matter is physiologically in-

different. The authors, however, observed that in the stage of increased excitability as an effect of morphinisation, the reaction-time of cortical stimulation may be as low as 0·02 to 0·025 sec.; the total delay of mere nerve transmission and muscle latency would amount to this, leaving a by no means evident cortical delay. They also observed great prolongation of the cortical reaction-time in deep morphinisation, a prolongation which was at once greatly diminished by substituting subcortical for cortical excitation.

With regard to the experimental determination of action and resolution of action, several significant observations are related.

Munk observed that epileptic convulsions brought about by excitation of a limited cortical area can be abolished by removal of that part of the cortex, unless the convulsions have lasted for some time, in which case extirpation fails to effect quiescence. Bubnoff and Heidenhain observe further that sometimes in the early period of an epileptic attack, extirpation of the cortical centre of a limb may effect quiescence of that limb while the rest of the body remains in strong convulsion; they suppose that the excitatory state has now extended to the whole cortex, but has not yet established itself in subcortical matter. In other cases, after rapid destruction of the motor area on one side, they observed cessation of convulsion not only on the opposite side but on both sides, it being indifferent whether the area removed were the same as, or the opposite to, that by which the outbreak was originally excited; —they suppose that each motor area not only governs the opposite muscles, but that each area also influences the opposite area, and that failing this excitatory factor the excitation soon becomes insufficient to convulse. In other cases they observed that unilateral ablation had no effect on the convulsions; they suppose that the excitatory state had then extended to subcortical motor apparatus. The authors remark also that epilepsy by excitation of the cortex on one side, always commences by muscles on the opposite side; that epilepsy by excitation of subcortical matter (the cortex of that side having been removed) always commences on the same side, and that after bilateral ablation of the cortex, subcortical excitation is never succeeded by an epileptic attack. Admitting the facts, the deductions from them are obvious—experimental epilepsy is a manifestation of cortical activity, which crosses to the opposite side of the brain by commissural fibres.

Turning to other sides of the question, the authors show how centripetal impressions can influence central excitability, e.g. on the one hand, that the muscle-curve of cortical excitation may be

retarded and prolonged by simultaneous excitation of the sciatic; on the other, that submaximal cortical stimulation may be rendered efficient by gentle tactile excitation on the same side. The results depend greatly upon the depth of morphinisation, and it must be added that the latter phenomenon occurs also after removal of the cortex; the enhanced excitability is therefore certainly of subcortical parts, and only *possibly* of cortical substance.

Other phenomena are recorded of a precisely opposite character, viz. the resolution of tonic contraction by gentle tactile excitation, by sudden sounds or blows, or by weak excitation of the sciatic; but here again the part played by cortical matter is not clearly defined, since the phenomena were exceptionally observed in its absence, and could then only depend on subcortical processes.

The authors also observed arrest of action by weak central excitation, increase of action by stronger central excitation applied to the same cortical parts as those from which the observed action proceeded as well as to remote parts.

For the discussion of imaginable representations of the dynamic changes effecting action and resolution of action, the reader is referred to the original paper, in which they are considered at length.

Prof. Christian Loven on the Tone of Muscles excited electrically, and certain Electro-acoustical Phenomena connected with it (with an original Note). (*Arch. für Anat. u. Physiol.* (*Phys. Abth.*) 1881. Heft v. p. 363.)—According to Haughton, the sound of strong voluntary contraction indicates 30–36 vibrations per second.

According to Helmholtz it indicates 36–40, but by consonating strings he concluded that the real vibration-frequency is an octave lower (18 to 20).

Loven affirms that the v.-f. is half this, viz. 9 to 10 per sec., seeing that the spasm frequency of strychnia clonus is about that number (v. Nordiskt Med. Ark. 1879, No. 14). The minimum number of stimuli necessary to cause complete tetanus varies; Loven says that for mammalia generally it is between 20 and 30 per sec.

Listening to the muscle-tone elicited by high interruption frequency, Loven was surprised to notice that the tone heard over the muscle itself is just as well heard in any other part of the body. It was clear that this tone depends on something else than muscle vibration, especially as the observation can be made on dead animals. He first thought of unipolar charging, but he observed that the

tone is well heard when animal and observer are completely insulated.

Seeking to determine the conditions of this electro-acoustic phenomenon, he found it necessary that the *animal and observer should be separated by a moderately thick non-conductor*. The sound is at its loudest when, with one pole on the animal, the ear of the observer is applied thereto; if now a stethoscope is substituted, no sound is heard, provided no part of the observer is near to or touching the dry hairy skin; nor is any sound heard if the observer should happen to touch a wound or the wetted skin of the animal.

Given the above condition, the experiment may be more simply improvised. A wet sponge covered with indiarubber sheeting represents the animal, and is joined to one pole of the coil; the other pole being brought to earth or not (in the former case the sound is rather better). On touching one's ear with the former, one feels at the first light touch a weak pricking and a crackling sound, and on firmer pressure all the peculiarities of the interruption.

The fact may be made evident in several ways: 1. When one holds one pole of a coil, the head being covered by a sheet of thin indiarubber, nothing is felt or heard; but if a second person places his ear against the sheeting the sound is heard by both, and so on for a string of people, providing they be not in conducting chain. The sound is strongest if each terminal person is joined to the coil.

2. One pole is joined to a sheet of metal. On touching the metal, especially if firmly with the moist finger, nothing is felt or heard; if now one covers the metal with indiarubber sheeting (or better, if one superposes on this sheeting a second metal-plate), and touches with the wet finger, sound is heard, most distinctly if the second metal-plate is joined to the other pole of the coil. If the single plate is lightly touched or stroked with the dry finger, one feels as if the plate were rough, though it be quite smooth, and sound is heard; it is best, but not essential, to connect oneself with the earth.

These phenomena evidently depend on static electricity, since an insulating partition is necessary.

At the free pole (i.e. that connected with the sponge or with the metal plate), there is, at a given instant, positive electricity of high tension, at a succeeding instant negative electricity, with each make and break of the primary circuit. If now to this a conductor

is approached, but separated by a thin insulating partition, opposite electric tension is induced by influence, viz., + when the pole is −; − when the pole is +.

The tone may be attributed to (1) actual charging on each side of the insulator, or (2) to molecular vibration in the conductors by the sudden reversal of electrical tension, or (3) to molecular vibration of the insulator, in consequence of the induction action.

Loven inclines to the first of these explanations, and relates this experiment. Two metal plates, ½ cm. distant, joined to two poles of secondary coil, give no sound. A frame on which india-rubber, or, better, gutta-percha paper is stretched, being introduced between the metal plates, a loud sound is heard, when the plates are separated as much as 30 cm. The sound can also be heard with but one metal plate, also when the observer holds one pole and brings the gutta percha close to his ear, and when the sound is absent it can be brought out by electrifying the sheeting by rubbing with the hand.

It might be asked how these facts disclose any source of error in acoustic experiments on muscle, since they are produced in an open circuit with strong currents; but Loven states that he has brought out the phenomenon when the circuit was closed through a short bit of nerve or wet thread.

What small tension differences are sufficient comes out in the following experiments:

1. Both poles are brought to binding-screws a few centimetres apart, joined by a moist thread. A second thread is fixed to the first by a slider, so that it can be pushed along the former, and its other end is joined to a metal plate, or covered sponge. If the slider is near one of the screws, the sound is loud; if it is gradually pushed towards the middle, the sound becomes weaker; when quite in the middle, sound of course vanishes; but it can be faintly heard when the slider is only a few mm. from the middle.

2. When the poles of a coil are joined to a second coil of fine wire, through which the current must therefore pass, and the ear is placed on the latter, sound is heard. That it does not, or at least can only slightly depend on molecular vibrations, is shown by the greater sound heard on a covered sponge or metal plate connected with one screw of the second coil.

That the high tension at the poles of this coil does not depend upon its resistance is shown by the substitution of a rheostat of higher resistance, which brings out no trace of sound. The phenomenon is doubtlessly due to extra-currents generated in the

coil, and which render the latter, in virtue of this fact, comparable with a bad conductor, such as a nerve or a moist thread.

These experiments seem to show that the phenomenon may occur under the ordinary conditions of myophonic experiments. It is specially liable to happen if the currents are strong, if the electrode contact is anywise deficient, if the electrode interval is over 2 to 3 mm. *The way to guard against it is to establish a good conductor between animal and observer.*

Professor Loven relates further the following experiments on the muscle-tone. On frogs he could hear no sound with a Hughes's microphone, when he excited indirectly; with rather strong currents and by direct excitation he heard a sound, but which continued unaltered after complete exhaustion and death of the muscle, or with substitution of WET thread for the muscle. The sound, therefore, depends on vibrations, the immediate consequence of induction. On rabbits, by indirect excitation (sciatic), using an interrupter resembling Bernstein's acoustic interrupter, he examined the questions (1) How far strength of current influences the muscle-sound; and (2) What is the greatest vibration-frequency with which it continues to agree? To the second question, Bernstein's experiments and those of Kronecker and Stirling appear to answer that there is no limit. The first question is hitherto untouched.

Using the coil without its core, and at the max. distance giving complete tetanus, he was surprised to find that a sound was heard at once with its max. loudness, and that on increasing the strength of the current the sound grew weaker till it vanished, "leaving only the ordinary muscle-sound." With moderately high vibration-frequency, 330–380 per sec., and weak excitation, the tone was distinctly an octave lower than the interrupter-note. Often (but only when the vibration-freq. was not too great), after the sound had vanished with increased strength, it reappeared, but in unison; if now the original strength were returned to, the lower octave was heard.

To test the alteration of sound with alteration of stimulus-frequency used, the voice sang into a telephone from g to g′ (198 to 396). The muscle-scale was heard up to C′ (264), d′ was indistinct, e′, fis′, g′ distinct, but belonging to the lower octave.

Exciting by the telephone and whistles, he got no sounds with a stimulus-frequency above 800 per sec., although there was strong muscular tetanus.

According to Bernstein, Kronecker, and Stirling, not only the

pitch but also the quality of the muscle-sound corresponds with that of the interrupter. Loven contradicts this statement, and says the sound has no clang, does not differ whether the contraction were excited by a man's voice or by a whistle acting on the telephone; it only gave the ground-tone or under-tone, never the *over-tone*.

Loven remarks on the peculiarity that it is only in the weaker strin. that the lower octave is heard, and believes it to be a *physiological* phenomenon. Bernstein stated that the muscle-tone becomes weaker when the interruption-frequency passes 300 to 400, and brings this in relation with the duration of the neg. var. Loven never succeeded in observing a distinct decline in the muscle-tone in passing any given stimulation-frequency. He states that its intensity depends, above all, on the strength of stimulation.

Note by Reporter.

The commonly received doctrine, based on the authority of Helmholtz, is that voluntary tetanus is compounded by 18 to 20 discharges per sec. from the spinal cord. Loven, basing his opinion on the fact that of strychninised frogs the spinal clonus initiated by any peripheral stimulus has a rhythm of 8 to 10 per sec., thinks that voluntary tetanus is compounded by 9 to 10 stimuli per sec. viz., by a frequency $\frac{1}{2}$ that of the vibrations of the note heard, and $\frac{1}{2}$ that given by Helmholtz. It has been generally asserted that 18 to 20 induction shocks per sec. give complete tetanus on man, and Prof. Loven states that on mammalia complete tetanus is reached with 20 to 30 interruptions per sec. In relation to this question, I observe that the maximum number of voluntary contractions corresponds with the figure given by Prof. Loven, being generally 8 to 10 per sec.; also that the rhythm of morbid spasms—trepidation, clonus, tremor—is about the same, viz. 8 to 10 per sec. ('Brain,' July, 1880, p. 179). I do not find the strychnine clonus in the frog to be constant spasm-frequency, the rhythm usually becoming slower with fatigue, and I have now under my eyes a tracing showing a vibration-frequency of only 5 per sec. On the other hand, I observe on my own muscles incomplete tetanic fusion, with an interruption frequency of over 50 per sec.; whereas voluntary tetanus (if it be tetanus) gives an unbroken line. This observation is opposed to the current theory of the composition of voluntary tetanus by 18 to 20 spinal discharges per sec. A. Waller.

BRAIN.

JULY, 1882.

Original Articles.

LABIO-GLOSSO-PHARYNGEAL PARALYSIS OF CEREBRAL ORIGIN.

BY JAMES ROSS, M.D., F.R.C.P.

THE first case which I find recorded in medical literature in which a lesion in the hemisphere of the brain gave rise to facial paralysis, and difficulties of articulation and deglutition, closely simulating the symptoms of progressive labio-glosso-pharyngeal paralysis, is the well-known one reported by Magnus,[1] and subsequently quoted by Romberg.[2]

"The subject was a widow, aged 50 years, who had already passed through two apoplectic attacks, causing loss of speech and paralysis of the left side; the first had supervened during a confinement, after violent vexation and the cessation of the lochia, the second in consequence of her menses being suppressed by a cold. After the latter the paralysis of the extremities was removed, but speech was not restored, as had been the case after the last seizure. The face of the patient was perfectly smooth, without either a furrow or an expression. All the muscles of the face were deprived of voluntary movement. She was neither able to contract her forehead nor her eyebrows, to raise the nostrils, nor to move her cheek and chin.

[1] Müller's 'Archiv,' Jahrgang 1837, p. 258.

[2] Romberg's 'Manual of the Nervous Diseases of Man.' Sydenh. Soc. Trans., vol. ii. p. 278, 1853.

She was incapable of closing the eyelids voluntarily. When required to do it she used her finger, or looked on the ground, by which the eyeball was directed downwards, the levator tarsi relaxed in its contraction, and the upper eyelids also moved downwards. On the other hand, the eyelids closed perfectly as soon as a sudden movement was made towards the eye of the patient, if she was suddenly directed into a bright light, or when she sneezed. During sleep the eyes were also perfectly closed." The mouth was constantly slightly open, and the saliva ran out, the movements of the jaw were limited and feeble, the tongue was completely paralysed, deglutition was effected with great difficulty, and articulation was so much interfered with that the patient was only able to utter grunting and inarticulate sounds.

The patient died from an attack of cholera, and at the post-mortem examination, conducted by Froriep, a hæmorrhagic cyst, about the size of a small walnut, was found situated "at the external edge of the right hemisphere of the cerebrum, where the anterior lobe adjoins the middle one." The inner surface of the cavity was invested with a yellowish membrane, and two gyri were destroyed by it.

In 1871 Dr. Friedrich Jolly reported a case of multiple sclerosis which "presented during life all the symptoms of progressive bulbar paralysis without any disease being found after death in the nuclei of origin of the nerves of the medulla oblongata and pons."[1] Extensive sclerotic patches were found in the corpus callosum and walls of the lateral ventricles; a small patch was observed in each crusta, while a limited degeneration, evidently descending in its character, could be traced through the pons, medulla oblongata, and lateral columns of the spinal cord; but the pons, with the exceptions just mentioned, and the bulbar nuclei were completely free from disease.

In more recent years Dr. Barlow[2] has directed particular attention to the fact that the main symptoms of labio-glosso-pharyngeal paralysis may be caused by lesions situated in the

[1] 'Archiv für Psychiatrie,' Band iii., 1872, p. 772.

[2] "On a case of Double Hemiplegia, with Cerebral Symmetrical Lesions, by Thomas Barlow, M.D., B.S." 'The British Medical Journal,' vol. ii. p. 103, 1877.

cerebral hemispheres, and the case reported by him possesses all the more significance inasmuch as the lesions discovered were very definite, and localised in the cortex of the brain. The subject was a boy, aged 10 years, suffering from aortic regurgitation, who had had an attack of right hemiplegia with aphasia, from which he made a good recovery. Four months afterwards he had an attack of left hemiplegia with aphasia, and in addition, the muscles of mastication and articulation, and those concerned in the first act of deglutition, were paralysed. The patient died from the results of the aortic disease, and at the autopsy evidence of an embolus was found in both sylvian arteries. A focus of softening, about the size of a shilling, was found in the cortex of each hemisphere, involving the inferior extremity of the ascending frontal, and the posterior extremities of the second and third frontal convolutions.

About the same time, or very soon after the publication of Dr. Barlow's case, M. Lépine[1] wrote an important paper on the subject, and described several cases illustrative of the affection. The first of these was that of a woman, aged 51 years, who entered the Salpêtrière in 1876 under the care of Prof. Charcot, and whose case was reported by M. Oulmont. The patient had in 1871 a slight attack of right hemiplegia associated with some difficulty of speech, from which she made a rapid recovery. Two years afterwards she had an attack of left hemiplegia, accompanied by difficulty of deglutition and salivation, but without loss of consciousness. The prominent symptoms in 1877, when a full report of the case was taken, were great difficulty of deglutition, paresis of the muscles supplied by the inferior branches of the facial nerves on both sides, paresis, with fibrillary contractions, of the muscles of the tongue, an excessive flow of viscid saliva, and feebleness of the muscles of mastication, while the speech was "slow, drawling, difficult, and badly articulated." The patient, however, was able to pronounce the separate vowels and consonants fairly well. The patient had several epileptiform seizures, accompanied by

[1] "Note sur la Paralysie Glosso-Labiée Cérébrale en forme Pseudo-Bulbaire, par R. Lépine." 'Revue Mensuelle de Médecine et de Chirurgie,' tome i. 1877, p. 909.

coma, from which she recovered. She ultimately had a severe apoplectic attack, and died two days subsequently in profound coma. At the autopsy a recent hæmorrhagic focus was found in the left optic thalamus, which communicated with the lateral ventricles, these cavities being filled with blood. An old hæmorrhagic focus (*foyer ocreux*) was found in the right hemisphere occupying the third or external segment, and a smaller focus situated in the second segment of the lenticular nucleus. Similar foci were found in the third and second segments of the lenticular nucleus of the left hemisphere. The internal capsule was not involved in either hemisphere. The medulla oblongata and pons were free from disease.

The second case, also reported by Oulmont, was that of a woman, aged 58 years, who entered the Lariboisière under the care of M. Raynaud. The main symptoms were double facial paralysis limited to the inferior branches of the nerve, paralysis of the tongue, extreme difficulty of deglutition, anarthria, salivation, and great diminution of the reflex of the palate. At the autopsy an old hæmorrhagic focus was found in the posterior extremity of the lenticular nucleus in each hemisphere. Another focus was situated in the white substance of the occipital lobe, and one about the size of a pea, containing milky fluid, was observed in the white substance of the anterior lobe of the left hemisphere near its base and anterior extremity. The third case mentioned by Lépine is the one recorded by Magnus, which has already been quoted; while his fourth case, although in all probability an example of this affection, need not detain us, inasmuch as the diagnosis was not verified by post-mortem examination.

In 1878 an instance of this affection was described by Dr. C. Eisenlohr.[1] The subject was a man, aged 73 years, who for the three years before his death had suffered from gradually increasing weakness of the lower extremities, and progressive difficulty of speech. The patient on admission presented paralysis of the inferior branches of both facial nerves, the articulation of the consonants was indistinct, and the speech had an explosive and monotonous character, but the general movements of the tongue were retained, and deglutition was

[1] 'Archiv für Psychiatrie,' Band ix., 1878, p. 43.

unaffected. Motor power was diminished in the upper extremities, while there was great feebleness of the lower extremities, which were seized with tremor on any attempt at walking, or on passive movements of them. At the autopsy a cystic cavity was found in the anterior part of each corpus striatum (lenticular nucleus ?) situated immediately beneath the surface of the brain, this portion being depressed and discoloured. A small cyst was also found in the optic thalamus on each side. The pons, medulla oblongata, and spinal cord appeared normal to the naked eye, and no secondary degeneration was found in the spinal cord on microscopic examination. Dr. Broadbent[1] published also in 1878, under the title of "Spurious Aphasia," four cases; and two, if not three, of these appear to have belonged to this category, the fourth being probably an example of apoplectic bulbar paralysis. The affection of speech in one of these cases (left hemiplegia) is thus described: "She answered questions correctly, but very slowly; laryngeal phonation was imperfect, and the voice was a hoarse half-whisper, each syllable requiring a separate effort; the articulation also was indistinct." The absence of a post-mortem examination in Dr. Broadbent's cases renders it unnecessary to enter upon a further analysis of them. Another case of bulbar paralysis of cerebral origin was reported in 1881 by Dr. Kirchhoff.[2] The subject was a joiner, aged 24 years. The disease was ushered in by an attack of dizziness but without loss of consciousness, during which he suffered from clonic spasms of the extremities, difficulty of speech and of swallowing, salivation, and deviation of the face to the left. At the end of four days these symptoms had completely disappeared. Eight days later he fell on the stairs during an attack of dizziness, but he was able to drag himself towards the bed. He was unable to swallow during the whole of that night, but next morning he regained the power of deglutition. Speech was drawling, the lower lip was pendulous, the saliva flowed over it, and the tongue felt heavy to him. On his admission into the hospital in July 1877, four weeks from the onset of the disease, he complained principally of difficulty of speech.

[1] 'Transactions of the Clinical Society,' vol. xi., 1878, p. 37.

[2] 'Archiv für Psychiatrie,' Band xi., 1881, p. 132.

The naso-labial folds on both sides were not well marked, the patient could not whistle, and the angles of the mouth could only be slightly retracted by a voluntary effort. The tongue could only be protruded 1 cm. beyond the mouth, the speech was drawling, the labial and guttural consonants were pronounced with difficulty, while the lingual consonants were pronounced with comparative freedom. The lips moved but little during articulation. Both eyes could be closed, but during closure there were fibrillary contractions of the muscles of the lids, and the right lid offered less resistance to opening than the left. The patient suffered from mitral stenosis. On the 25th of December the patient had an apoplectic attack with left hemiplegia, and died ten days afterwards, and six months from the onset of the disease. At the autopsy the right sylvian artery was found blocked up by an embolus. The right corpus striatum (caudate nucleus) in its posterior two-thirds was of a yellowish-white colour, turbid, and depressed, so that the grey substance was reduced to a thin and softened layer, which was covered by a thickened ependyma. The subjacent internal capsule was of a grey colour, and semi-translucent. The external third of the lenticular nucleus, the claustrum, external capsule, and island of Reil were softened and porous, but not discoloured. A careful microscopic examination of the medulla oblongata and pons, undertaken by Professor Quincke, gave entirely negative results.

A typical case of bulbar paralysis of cerebral origin has been described by myself.[1] The subject was a man, aged 49 years, admitted to the Manchester Royal Infirmary, November 15th, 1880, under the care of Dr. Leech, who kindly transferred the case to me. Eleven months before admission the patient complained of headache, and it was observed that his speech was thick; six months subsequently he had some kind of seizure, which apparently was not attended by loss of consciousness, but from that time up to the date of his admission his speech became more and more unintelligible. On admission there was loss of facial expression, and the patient could not compress his lips or whistle. He could protrude his tongue, but could not curl the tip towards the nose, or roll it up laterally

[1] 'The Diseases of the Nervous System,' vol. ii. p. 626.

so as to render it tubular. He could pronounce the separate consonants with tolerable distinctness, but his articulation, even of words of two syllables, was so imperfect, that his speech was almost unintelligible. Food collected between the teeth, the power of deglutition was greatly impaired, and the viscid saliva which flowed from the mouth had to be constantly wiped away. The uvula was pendulous, but not distorted, its reflex excitability was greatly diminished, and the fauces and epiglottis could be examined with the point of the finger without provoking a cough. The patient complained of great general weakness, but there was no distinct paralysis of any of the extremities. Death took place on the 10th of December from exhaustion, caused by intractable diarrhœa; the lenticular nucleus of each hemisphere was found post mortem replaced by a well-defined cystic cavity, containing a clear straw-coloured fluid, the internal capsules being apparently uninjured. Microscopic examination failed to detect any evidence of disease in the nerve nuclei of the medulla oblongata, or descending changes in the pyramidal tracts in any part of their course.

As the following two examples of this affection, recently under my observation, have not yet been published, and as they present some special features worthy of notice, I shall take leave to report them in detail. I am indebted to Mr. Harris, one of the house physicians of the Manchester Royal Infirmary, for the notes of the following case.

Henry H., æt. 40 years, was admitted into the Royal Infirmary on May 4, 1881, under the care of Dr. Ross. The patient, although much addicted to alcoholic excesses, enjoyed good health until fifteen months previous to his admission. He then had a "stroke" accompanied by partial unconsciousness, and followed by paralysis of the right upper extremity and loss of speech, the latter of which lasted for several days. The right arm gradually regained strength, and at the end of three weeks from the onset he could express himself without difficulty, although his speech remained "thick" and indistinct. He has recently suffered from dizziness, general debility, palpitation and shortness of breath, and it is for these symptoms that he has sought advice.

Present Condition.—The grasp of the right hand is feebler than that of the left, and the tendon reflexes are slightly increased in the right arm and hand. The right naso-labial fold is obliterated, and there is a decided loss of expression on the right side of the face, the difference between the two sides becoming more marked when the patient smiles. There is no deviation of the tongue on protrusion. The patient never appears to be at a loss for the names of objects, or for the proper word whereby to express his meaning, but his utterance is thick and indistinct. He can pronounce the separate vowels and consonants with tolerable distinctness, but his articulation of words of two or more syllables is so wanting in clearness that the patient has some difficulty in making himself understood. There is also some difficulty of deglutition, and fluids find their way into the glottis and cause distressing cough. The action of the heart is forcible, the left ventricle is hypertrophied, and the urine contains a small quantity of albumen. The patient was discharged on May 12, but was readmitted on June 16, on account of a gradually increasing paralysis of the lower extremities. Every form of cutaneous sensibility is diminished in the lower half of the body as far up as the area of distribution of the 7th or 8th pair of dorsal nerves, the sense of temperature being entirely abolished. The lower extremities are extended, the feet are in the position of equino-varus, the patellar tendon reflex is increased, and ankle clonus can be readily elicited in both the lower extremities. There is no deformity or marked tenderness of the vertebral column. The patient has slight dribbling of urine, and the skin over the sacrum is erythematous, but not yet ulcerated. The cerebral symptoms have not undergone any perceptible alteration since last report.

August 7.—Yesterday morning the patient had several distinct rigors and his temperature rose, and now stands at 102.6 F. He complains of pain across the lower part of the chest. About an hour ago severe dyspnœa supervened; the patient's lips are now livid, the breathing is accompanied by a guttural stertor, and although the patient is quite conscious his articulation is so indistinct that his speech is nearly unintelligible. The breathing is entirely abdominal, and loud

bubbling râles are heard over both sides of the chest, but the patient is too ill and helpless to permit of a careful examination of the chest posteriorly.

August 15.—The symptoms noticed in the last report continued with slight fluctuations, and the patient died somewhat suddenly to-day, apparently from asphyxia.

Post mortem.—The post-mortem examination was conducted by Mr. Harris ten hours after death. The brain presented nothing unusual externally. The anterior part of the lenticular nucleus of the left hemisphere presented two small cyst-like cavities, containing clear fluid. These cavities occupied the second and third segments of the nucleus, and one of them appeared to encroach to some extent on the knee of the internal capsule. The right hemisphere was preserved in spirit, and was not examined until a fortnight later, when a small cavity was found in the lenticular nucleus of this hemisphere also. This cavity occupied the anterior part of the nucleus, but did not appear to encroach upon the internal capsule.

On opening the vertebral canal a small sequestrum of bone was found opposite to the bodies of the sixth and seventh dorsal vertebræ, but there was no displacement of the vertebræ. A layer of pus lay external to the dura mater, opposite to the diseased vertebræ. The dura mater was thickened at this spot and the spinal cord itself was softened, although not much compressed by the thickened membranes. The cord appeared normal to the naked eye above and below the lesion in the dorsal region.

The left ventricle of the heart was hypertrophied, and the aorta was atheromatous, but the valves were healthy. The greater portion of the right lung was dense and airless. The capsules of the kidneys were adherent, and on being stripped off the surfaces of the organs were granular; on section the cortex was seen to be diminished in size. The liver and spleen were normal.

A microscopical examination of the crura, pons, and medulla oblongata revealed a streak of descending degeneration, which occupied the positions respectively indicated in the accompanying diagrams (Figs. 1, 2, 3 and 4 *g*), kindly drawn for me by Mr. A. H. Young, Pathological Registrar to the Infirmary. The

patch of sclerosis in the anterior pyramid of the medulla oblongata is considerably less than that in the crusta. A large number of the degenerated fibres must, therefore, have been lost in the pons. The diseased fibres in the anterior pyramid appeared to bend back in the median raphé to reach the nerve nuclei of the opposite side. The bulbar nuclei were quite healthy. The spinal cord exhibited the usual evidences of transverse myelitis, with its ascending and descending degenerations.

FIG. 1.—CRURA CEREBRI.
(*g*. Degeneration of portion of the geniculate fasciculus.)

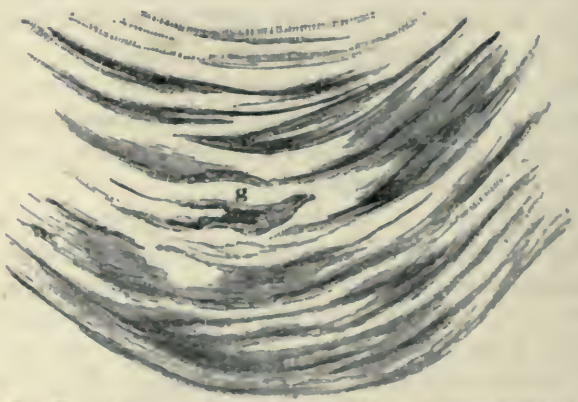

FIG. 2.—TRANSVERSE SECTION OF THE PONS ABOUT ITS MIDDLE.
(*g*. Descending degeneration.)

I am indebted for the following notes to Mr. George H. Williams, my clinical clerk.

Matthew N., aged 34, married, entered the Manchester Royal Infirmary under the care of Dr. Ross, on Jan. 31, 1882.

Family History.—The patient's father and mother are both alive and healthy, each being above 70 years of age. He has three brothers and three sisters, all enjoying good health. No family predisposition to any particular disease can be traced.

Previous History.—The patient has never suffered from any serious disease until about four years ago, when he had an

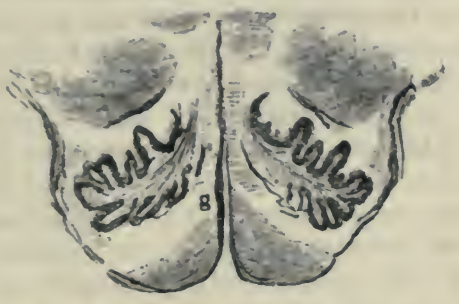

FIG. 3.—TRANSVERSE SECTION OF ANTERIOR HALF OF THE MEDULLA OBLONGATA ON A LEVEL WITH THE MIDDLE OF THE OLIVARY BODIES.
(*g*. Descending degeneration.)

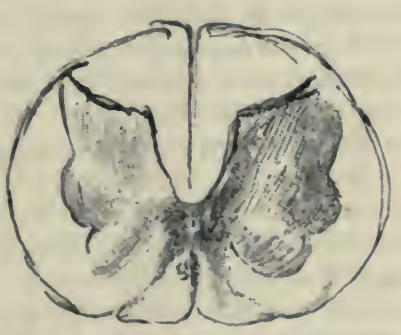

FIG. 4.—TRANSVERSE SECTION OF THE MEDULLA OBLONGATA IMMEDIATELY BELOW THE OLIVARY BODIES.
(*g*. Descending degeneration.)

attack of rheumatic fever, from which he was laid up for three months. At the same time he suffered from an eruption which the medical man who attended him called chicken-pox. One morning last June the patient was writing a letter when he was suddenly seized with cramp in the fingers of his right hand, the sensation of cramp travelling up his arm to his head. He states that he did not lose consciousness during

the attack, but there was partial loss of the power of speech, the patient being only able to utter portions of sentences. Speech gradually improved after a few hours, but complete recovery did not take place until the lapse of three days. During the attack he suffered from intense headache, which lasted half an hour; his right hand and arm were numb during the seizure and for five or six hours subsequently. A few days after this attack he felt quite well and able to attend to his work as usual. Towards the end of October he observed that his water dribbled instead of coming in a full stream, but he did not think this symptom of much consequence. On November 6, 1881, he took a walk of five or six miles into the country, and as he was returning he observed that his foot kept catching the ground so that he stumbled several times. On getting up the next morning he was unable to pass water, but went to work as usual. On walking home at noon to dinner he stumbled and staggered so much that he was obliged to steady himself by catching hold of the rails on the road-side. After dinner and a few hours' rest the patient made another attempt to go to work, and actually walked to the building on which he was engaged, a distance of nearly half a mile from his house, but on arriving there he found himself so ill that he was obliged to return home. He walked back, but with great difficulty, and when he got near to his own house he fell, but his wife came to his aid and assisted him into the house. A medical man was then sent for, and on arriving he drew off his water. This operation had to be repeated night and morning for a week, but at the end of this time the patient was able to pass water himself.

The lower extremities were now more or less completely paralysed, and the patient was confined to bed until about the middle of December; he then began to get up, and was even able to walk a little when supported.

On Christmas Day as the patient was lying in bed he had another seizure. He has an indistinct recollection of feeling a curious sensation in the right arm and right side of the face, but he soon became unconscious. He states that he remained in a dazed condition for two weeks; during this time there were intervals when he knew something of what was

going on about him, but at other times he was quite wild, and then he sang, shouted, and struggled with his attendants. The patient states that the doctor told him that three days after the commencement of unconsciousness he had a fit during the night, which left his mouth drawn to the left side. When he regained his senses he was only able to speak in broken sentences, the right arm was paralysed, the mouth was drawn to the left, and he could not close his right eye. Gradual improvement took place in the symptoms occasioned by the last attack, so that at the time of the patient's admission into the Infirmary his right hand had regained a considerable degree of its former power, he could close his right eye, and the only affection of speech from which he suffered was a certain degree of indistinctness of articulation.

Present Condition.—The patient is a well-made, muscular man, of sandy complexion and healthy appearance. Both the lower extremities are completely paralysed. Some degree of muscular tension is provoked by passive movements of the legs, the patellar tendon reflexes are slightly exaggerated, but ankle clonus cannot be elicited. The muscles of both calves and the peroneal groups are somewhat wasted; they do not reach to a strong faradic current, but respond to a galvanic current from 40 Leclanché cells, the reaction to negative closure being stronger than that to positive closure. The reflex of the sole is exaggerated on both sides and the cremasteric reflex is not entirely abolished, although it is very sluggish. Every form of sensibility is diminished on both sides up to within an inch of the umbilicus. There is constant dribbling of the urine, and the patient passes his stools in bed. There is a small and superficial bed-sore over the sacrum. The grasp of the right is feebler than that of the left hand, and the right forearm is slightly jerked when the lower end of the radius is tapped. The triceps reflex is not perceptibly increased on the right side. The angle of the mouth is on a lower level on the right than on the left side; the right naso-labial fold is obliterated, and there is a decided loss of expression on the right side. The right lower eyelid is slightly depressed, while the upper lid is somewhat retracted so that the palpebral fissure is wider, and the eyeball appears to protrude more on the right than on

the left side. The patient can close both eyes by a voluntary effort, but the resistance which he can offer to the upper lid being raised by the finger of the observer is much greater on the left than on the right side. There is no deviation of the tongue on protrusion, and the patient can turn the tip laterally and towards the nose without apparent loss of power. He can also pronounce the separate consonants and vowels fairly well, but his articulation is indistinct and imperfect, especially on attempting to pronounce words of many syllables, like "Constantinople." The reflex of the palate is very sluggish when the fauces are tickled. The urine is alkaline, and contains bacteria and crystals of triple phosphate. The other organs are healthy. There is no evidence of valvular disease of the heart.

March 8.—The patient has become steadily worse since his admission. The muscles of the lower extremities are much atrophied, and the subcutaneous tissue is soft and œdematous. A large slough has formed on the sacrum, and an erythematous blush, which appeared a few days ago, has spread from the sore upwards as far as the angles of the scapulæ, and downwards below the gluteal folds. The urine is alkaline and contains a large quantity of pus, bacteria and crystals of triple phosphate. There are great variations in the daily records of the temperature, that in the evening being generally above 103° F., and in the morning down to 98° F. The articulation is much more indistinct, but this appears to be due to the dryness of the tongue. The other cerebral symptoms are unchanged.

March 15.—The patient became gradually weaker since last report, and died to-day in a state of great marasmus.

The post-mortem examination was conducted by Mr. A. H. Young fourteen hours after death. Body well developed, poorly nourished, limbs emaciated. Cutaneous hypostasis well marked dorsally. Large bed-sore over the sacrum. Rigor mortis absent. A small cystic cavity was found in the left lenticular nucleus, close to the border of the genu of the internal capsule. No secondary degeneration perceptible to the naked eye in the crura.

There was marked congestion of the meningial rachidian

veins. The membranes of the cord were normal. The spinal cord itself, in the lower dorsal and upper lumbar regions, was abnormally soft, and on section the white matter protruded while the grey matter appeared depressed. The vessels of the grey matter were engorged. The heart weighed 13 ounces; it was hypertrophied and dilated, but there was no valvular lesion, and nowhere were there any indications of previous endocarditis. Multiple abscesses were found in the kidneys, but the other organs did not present anything of special interest.

A careful microscopical examination of successive sections of the crura, pons, and medulla oblongata failed to detect any secondary degeneration of the pyramidal tract. The bulbar nuclei were healthy. The usual evidences of a transverse myelitis in the lower dorsal and upper lumbar regions, with ascending and descending degenerations, was discovered in the spinal cord.

A case, evidently belonging to this group, has just been reported by Dr. Hobson, of Croydon;[1] but I shall only refer to it briefly, as the subject is still living, and the diagnosis is consequently more or less conjectural. A lady, aged 40 years, suffering from albuminuria, had, during a period extending over twelve months, four successive attacks of partial aphasia, with slight implication of the left hand in the third and of the right hand in fourth attack. After the fourth attack, when the patient came under Dr. Hobson's observation, there was complete facial paralysis. The patient was unable to close the eyes by a voluntary effort. "On the other hand, *blinking* occurred, and, when amused, the face would be drawn up into an expression of laughter, but never into a smile." The tongue was paralysed, deglutition was difficult, and the power of speech and articulation was almost abolished, but the jaws were clenched from spasm of the masseters. The clinical picture presented by this case forcibly reminds us of the case recorded by Magnus, although it is probable that in Hobson's case a lesion exists in each hemisphere.

The cases which have just been described are full of theoretical and practical significance. They show, in the first

[1] 'The British Medical Journal,' April 29, 1882, p. 613.

place, that speech, in its objective or expressive aspect, consists of highly special and complex movements, and that ataxic aphasia is a want of power of effecting the combinations of muscular contractions necessary for the production of complex articulate sounds; or, in other words, that ataxic aphasia is essentially paralytic in its nature. If that be so, it may be asked why are not the general movements of articulation paralysed? The reply is, that as a rule they are paralysed, inasmuch as aphasia is usually associated with some degree of oro-lingual hemiparalysis. The special movements of the organs of articulation called into activity during speech are organised in one hemisphere only, usually the left, while the general movements are organised in both hemispheres. And destruction of the centres of articulation in both hemispheres, such as occurred in Dr. Barlow's case, gives rise not only to aphasia but to complete anarthria, along with difficulty of deglutition and bilateral paralysis of the inferior branches of the facial nerves.

It is worthy of note that in Dr. Barlow's case the patient made a good recovery from his first attack of hemiplegia (right) and aphasia, although the autopsy showed that the symptoms were caused by destruction of the cortical centre of speech. The solution of this difficulty appears to be that the special movements of articulation are not so exclusively organised in one hemisphere in young people as in adults; and during youth the brain undergoes structural changes more readily than at a late period of life, so that a new organisation is more rapidly developed in the former than in the latter. The recovery of speech in Dr. Barlow's case must be supposed to be due to a rapid development of new structural arrangements taking place in the oro-lingual cortical centre of the right hemisphere, after the corresponding centre in the left hemisphere had been destroyed. And when the centre in the right hemisphere had become subsequently destroyed, not only the special movements of articulation concerned in speech, but the general movements, were arrested, and the patient suffered from anarthria, difficulty of deglutition, and partial facial paralysis.

Passing over the cases in which the lesion was situated in

the cortex, let us come to those cases in which the lesion was situated in the substance of the hemisphere. It is probable that in one or two of the cases in which the lesion was situated in the substance of the hemisphere the cortical centres were also damaged; such, for instance, as the case reported by Magnus, and the right hemisphere in Lépine's first observation. In the remaining cases, however, the lesions observed were strictly limited to the substance of the hemispheres, and the cortex was uninjured. In all these cases lesions were found in the lenticular nuclei, either in one or in both hemispheres.

The questions which naturally suggest themselves are, Is the lenticular nucleus to be regarded as an independent centre for the regulation of the movements of articulation and deglutition? Is it a ganglion of interruption, as Meynert asserts, between the cortical centres and the nuclei in the medulla? or have the lesions been situated in such positions that the direct conducting paths between the cortex and the nuclei in the medulla oblongata and pons were injured? The first of these questions may be negatived at once, inasmuch as no known anatomical or physiological fact can be adduced in favour of the hypothesis which an affirmative answer would imply. It is somewhat difficult to decide between the alternatives presented by the two remaining questions. The theory that the lenticular nucleus is a ganglion of interruption, and that a lesion of it would interrupt the channels of communication between the cortex of the brain and the pons, medulla oblongata, and spinal cord, was a few years ago generally accepted as an established doctrine. But the anatomical investigations of Wernicke render it very doubtful, to say the least, whether the lenticular nucleus is possessed of radiating fibres to connect it with the cerebral cortex; and pathological observation has proved that the nucleus may be completely destroyed without giving rise to hemiplegia or any other decided symptom.

The supposition that the direct conducting paths from the cortex to the medulla and pons have been injured by the lesions is not free from difficulty, although it is the one we are inclined to adopt. It has been found by Charcot and

Brissaud that secondary degeneration occurs in the fibres of the knee of the internal capsule in cases of long standing aphasia. And a case was reported by these observers in which, along with extensive recent softening of one hemisphere, an old focus of softening was found limited exactly to the knee of the internal capsule (Fig. 5, D).

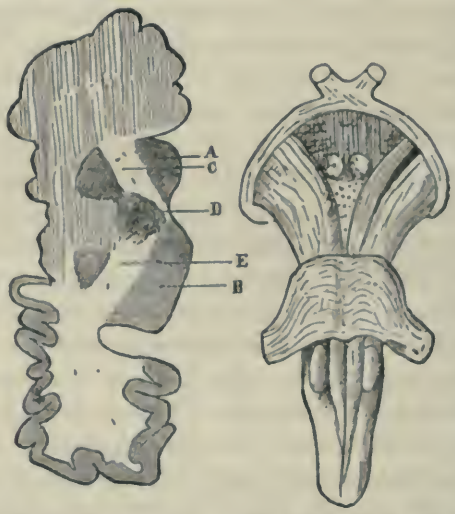

FIG. 5.—(CHARCOT AND BRISSAUD). RECENT SOFTENING OF THE FRONTAL LOBE, THE ISLAND OF REIL, AND MIDDLE THIRD OF THE LENTICULAR NUCLEUS.—D, old focus of softening occupying the knee of the internal capsule; A, Caudate nucleus; B, Optic thalamus; C, Anterior, and E, Posterior division of the internal capsule.

A streak of degeneration was observed in this case lying between the internal and middle thirds of the crusta. It may be presumed, therefore, that the fibres which descend from the cortical centres of speech to join the nuclei in the medulla pass through the knee of the internal capsule, and through the crusta at the point of junction of its internal and middle thirds; and consequently Charcot proposed to call this bundle of fibres the *geniculate fasciculus*. Now, in my second observation, a streak of degeneration was actually observed in the crusta, occupying the position assigned to the geniculate fasciculus by Charcot. But in my first observation (a much more typical example of this affection than the second) and also in the third case the most careful microscopical examination failed to

detect any descending degeneration, although the time which elapsed from the onset of the symptoms to the fatal termination was amply sufficient for a secondary degeneration to have taken place. But in my second and third cases there was, in addition to the bulbar symptoms, a slight degree of paralysis of the right hand, accompanied by excess of the deep reflexes. But notwithstanding this, no secondary descending degeneration of cerebral origin was found in either case in the spinal cord. It must, therefore, be admitted that in a cerebral lesion either slight paralysis of the muscles of the hand, with excess of the deep reflexes, may occur in the absence of any damage to the pyramidal tract or to the cortical centre, or that it is possible for the pyramidal tract to be slightly, but more or less permanently, damaged without being followed by a recognisable descending degeneration. I incline to adopt the latter supposition; and if it be possible to have injury of the fibres of the pyramidal tract which belong to the spinal nuclei regulating the muscles of the hand without descending degeneration, it must be equally possible for the fibres of the geniculate fasciculus to be partially injured without giving rise to a recognisable degeneration. This supposition is still further strengthened by the three cases reported by Binswanger,[1] in which lesions in the motor area of the cerebral hemispheres were discovered at the autopsies, while the patients during life suffered for some months from paralytic symptoms, and yet there were no secondary degenerations of the pyramidal tracts. In my second and third cases the lesions were of very limited extent, and all of them were situated opposite and close to the knee of the internal capsule, while in my first case the fibres of the internal capsule, those of the knee included, in each hemisphere could hardly help being compressed by the distended cystic cavities which replaced the lenticular nuclei. But in Lépine's second observation the lesions were situated in the posterior and external parts of each lenticular nucleus, and consequently away from the knee of the internal capsule. It must, however, be remembered that the spot of discernible softening never represents the whole of the diseased area in the cerebrum, but that an inflammatory condition may spread

[1] 'Archiv für Psych.,' Bd. xi. p. 727.

for a variable and often very considerable distance beyond the diseased focus. It is, therefore, quite possible that the fibres of the knee might have been injured in both hemispheres in Lépine's second case. It is very probable that the cortical centres were injured in the right hemisphere in Lépine's first observation, while the knee of the internal capsule could readily be damaged in the left hemisphere. In the case reported by Magnus the cortical centres of the right hemisphere were injured, and the centres in both hemispheres were probably damaged in the case described by Eisenlohr. The lesions found in the case reported by Kirchhoff demand separate notice. The right sylvian artery was occluded, and the external third of the lenticular nucleus, the claustrum, external capsule, and island of Reil were softened, the softening being probably the result of the embolic closure of the artery. But the seizure which corresponded with the closure of the artery only took place ten days before death, and the bulbar symptoms were in existence for six months previously. It seems clear, therefore, that the lesion in the caudate nucleus was the cause, the indirect cause at least, of the bulbar symptoms. It is expressly mentioned that the portion of the internal capsule subjacent to the lesion in the corpus striatum was grey and translucent, and certainly the knee of the capsule would be subjacent to some part of the posterior two-thirds of the caudate nucleus, the portion said to have been softened. The bulbar symptoms would, therefore, in this case, be directly caused by the secondary implication of the internal capsule.

But if the affection of speech present in my third case, for example, were caused by damage to the fibres of the pyramidal tract which connect the cortical centre with the nuclei of articulation in the medulla, it may be asked why it is that the dysarthria was not associated with aphasia. The reply is that the two symptoms were associated for a time, but the aphasia was only temporarily present. In the first cerebral attack from which this patient suffered the symptoms were very transitory, but some degree of aphasia was present for a few hours. The second cerebral attack was attended by prolonged unconsciousness, but for some time after he regained consciousness the

patient, to use his own words, could only "speak in broken sentences." But why did the aphasia disappear some weeks after the second attack, while the dysarthria remained more or less permanently? The assumption that the right cortical centre had become educated for speech which we made in reference to Dr. Barlow's case is not fully adequate to explain the rapid disappearance of aphasia which takes place in all those cases where the lesion is situated in the basal ganglia. The most plausible explanation of such cases is that proposed by Dr. Broadbent. He suggested that when the usual channel of communication between the centre of speech in the left hemisphere is interrupted, the impulses from the centre make their way through the fibres of the corpus callosum to the corresponding centre in the right hemisphere, and down through the fibres of the right pyramidal tract to the nuclei in the medulla, the nuclei of the two sides being connected by commissural fibres, through which the impulses make their way. In Fig. 6, for instance, let C′ and 3′ 3′ represent the centre and usual conducting path of speech. The conducting path joins V, the nucleus of articulation in the opposite half of the medulla, and the impulses are conveyed through the commissural fibres *c*″ to V′, the nucleus of articulation in the medulla, on the same side as the centre. If the centre C′ is completely destroyed, then the aphasia must either be permanent, or the centre C of the opposite hemisphere must undergo structural development. In adults the aphasia, after complete destruction of the centre, is, as a rule, more or less permanent, but, as we have seen, a new organisation is more readily developed in young people. But when the usual conducting path 3′ 3′ is ruptured in any part of its course, the centre C′ is only temporarily cut off from the nuclei in the medulla. The impulses from C′ make their way through the dotted line *c* to the opposite cortex C, and through the dotted line 3 3 to the nucleus V′, and thence through the dotted *c*″ to the nucleus V.

In my third case, a somewhat exceptional symptom was observed. I allude to the fact that, although the paralysis of the right side of the face was of cerebral origin, yet the upper branches of the facial nerve, which usually escape under such circumstances, were partially paralysed. It has been observed

for a long time that in cases of hemiplegia some of the muscles on the affected side are but little or not at all paralysed; but it was first pointed out by Dr. Broadbent, that the muscles which

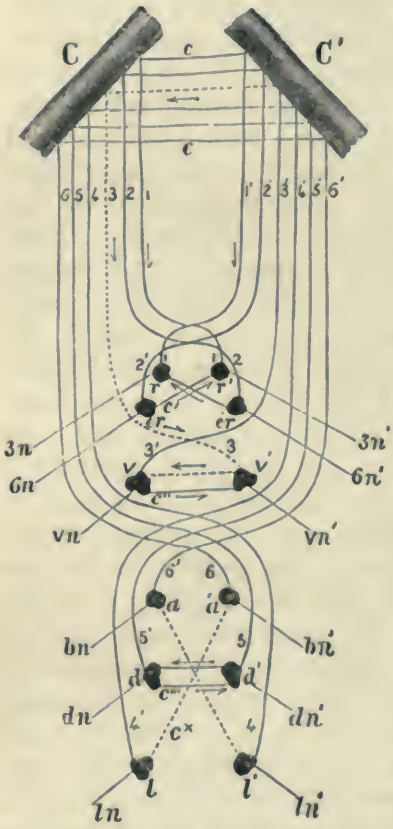

FIG. 6.

C C', cortex of right and left cerebral hemispheres respectively; 1, 2, 3, 4, 5, 6, fibres of the pyramidal tract uniting c, the cortex of the right hemisphere, and *r'*, *e'r*, *v'*, *a'*, *d'*, *l'*, the respective nuclei of the internal rectus and the external rectus muscles of the eye, the muscles of articulation and vocalisation, those of the upper extremity, the dorsal muscles, and those of the lower extremity, all of the left side; 1', 2', 3', 4', 5', 6', fibres of the pyramidal tract, connecting the cortex of the left hemisphere with *r*, *er*, *v*, *a*, *d*, *l*, the nerve nuclei of the right side corresponding to those already enumerated on the left side; *c*, *c*, fibres of the corpus callosum, uniting identical regions of the two hemispheres; *c'*, commissural fibres, connecting the nucleus of the internal rectus muscle of one eye with that of the external rectus muscle of the opposite eye; *c''*, those connecting the nerve nuclei of the muscles of vocalisation and articulation of the two sides; *c'''*, those connecting the nerve nuclei of the muscles of the trunk; *c*×, those connecting the nerve nuclei of the posterior extremity of one side with the anterior extremity of the opposite side. The arrows indicate the direction of conduction.

escape are those which are associated in their actions with the corresponding muscles of the opposite side. Dr. Broadbent further conjectured that the muscles which are associated with the corresponding muscles of the opposite side in their action are innervated from both cerebral hemispheres, so that severance of the connection between the spinal nuclei of the muscles of one side and the cortex of the opposite hemisphere leaves the connection with the cortex of the hemisphere on the same side still intact. In Fig. 6, for instance, *d'* repre-

sents the spinal nuclei of the intercostal muscles, and when the conducting path 5 5 is ruptured, impulses will still reach the nucleus *d'* from the cortex C' of the same side through 5' 5' *d* and C'''. Dr. Broadbent very happily named this principle "the bilateral association of the nerve nuclei of muscles bilaterally associated in their actions," and the association is supposed to be effected by means of commissural fibres between the nerve nuclei in the spinal cord and the medulla oblongata. In facial paralysis of central origin the special and unilateral function of winking is abolished, while the general and bilaterally associated functions of raising the eyebrows and closing the eyelids are retained on both sides. In my third case, however, there was distinct paresis of the orbicular of the eye on the affected side, which was so marked at one time as to have prevented closure of the eye by voluntary effort. It must, therefore, be assumed that in this case the commissural fibres in the medulla, between the nuclei of the upper facial branches, were deficient. But in the case recorded by Kirchhoff, both eyelids appeared to have been somewhat enfeebled, and there was distinct paralysis of the inferior branches of the facial and twelfth nerves on both sides, while the lesion was a unilateral one. A bilateral paralysis of the muscles of the face and tongue from a lesion in one hemisphere was still more markedly present in the case recorded by Magnus. It must be assumed that in these cases, not only the special, but the general facial and lingual movements were regulated exclusively from one hemisphere; and, curiously, in both cases the organisation was localised in the right hemisphere, contrary to what occurs with regard to the special movements concerned in speech, which are usually organised in the left hemisphere. In Dr. Hobson's case the upper and lower branches of the facial nerves were paralysed on both sides, but a lesion probably existed in each hemisphere.

As regards diagnosis, labio-glosso-pharyngeal paralysis of cerebral origin is likely to be mistaken for the progressive form of the disease. The chief points of distinction between them are that the progressive form begins insidiously and advances slowly and gradually, the affected muscles undergo

atrophy, and reflex excitability is abolished in the tongue, palate and pharynx at a comparatively early period of the disease, while in the cerebral disease the symptoms supervene more or less suddenly, and are ushered in, as a rule, by one or more apoplectiform attacks, often followed by aphasia and some degree of paralysis of one or both upper extremities, the reflexes of the palate, tongue, and pharynx are not abolished, although they were greatly diminished in the cases observed by me, and the paralysed muscles do not undergo atrophy.

Bulbar paralysis of cerebral origin may also be simulated by local disease, such as thrombosis, embolism, or hæmorrhage in the medulla. But in local disease the symptoms are usually more pronounced on one side than on the other, while in the more decided cases of the affection caused by cerebral disease the paralysis is symmetrically distributed on both sides. Local disease in the medulla may also give rise to polyuria, or diabetes mellitus, great disturbance of respiration and circulation, or alternate hemianæsthesia, as in the cases described by Gowers[1] and Senator,[2] the latter symptoms never being present in the cerebral variety. In local disease of the medulla the paralysed muscles often undergo atrophy and manifest the "reaction of degeneration," while in the cerebral variety the muscles do not undergo atrophy, and their electrical reactions remain normal.

A case related in Virchow's 'Archives'[3] shows that a peripheral lesion of some of the cranial nerves may give rise to symptoms like those of bulbar paralysis. A man, aged 19 years, suffering from leucocythæmia, developed in the course of a few days, and some weeks before death, paralysis of all the branches of both facial nerves, with complete loss of faradic irritability and the reaction of degeneration in the facial muscles, and a high degree of paralysis of the tongue, soft palate, and pharynx, causing serious difficulty of articulation and deglutition. There was temporary diminution of

[1] Illustrations of the Diseases of the Pons Varolii. 'Brain,' Vol. II., 1880, p. 476.

[2] "Apoplectische Bulbärparalyse mit wechselständiger Empfindungslähmung. Von Prof. H. Senator." 'Archiv für Psychiatrie,' Bd. xi. S. 713, 1881.

[3] See 'Archiv für Psychiatrie,' Band ix. S. 45, 1878.

sensibility in the territories of both facial nerves, and loss of taste. At the autopsy hæmorrhages were found in the sheaths and textures of the facial, hypoglossal, pneumogastric, glosso-pharyngeal, and lingual nerves on both sides, accompanied by a high degree of degeneration of the substance of the nerves. Such a case, however, is not likely to be mistaken for the cerebral affection just described.

THE TREATMENT OF EPILEPSY BY LIGATURE OF THE VERTEBRAL ARTERIES.

BY WILLIAM ALEXANDER, M.D., F.R.C.S.,

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On November 19th, 1881, I published in the 'Medical Times and Gazette' three cases of epilepsy where ligature of the left vertebral artery was followed by a cessation of the epileptic fits. The first case has made a voyage to Calcutta and back. He is now engaged in a permanent situation in Liverpool, and up to the present time has had no relapse. The idiot boy is still in the epileptic ward and constantly under observation, and Williams is in hospital with a swollen limb; neither of these two have had any fits. I think we may take it for granted that the collateral circulation has been established *in these cases* during the ten months that have elapsed since the operation, and that the cure promises to be permanent.

In my paper published in the same journal on March 11th, 1882, I reported five cases in detail. Nicholas M., the slobbering, howling idiot, has had up to May 31st only one fit since last seen. He has had two fits altogether since the second operation, which was performed on December 28th. This child has not a comfortable home. He lives in a very neglected state, and in consequence I should not have been surprised had a complete relapse taken place. It is satisfactory to note that the improvement has persisted in spite of surrounding circumstances. My second case, Margaret O'D., was sent back to Dingle Mount, as she has no home to go to. Dr. Irvine informed me a short time ago, that about the end of March she got into a terrible temper with some of the other epileptics, and had some kind of fits. Since that time she has been free. The right vertebral was tied on December 31st.

During the year 1881, and previous to ligature she had on an average twenty-four fits each month. Two or three fits of some kind after a paroxysm of anger in five months is surely a wonderful improvement. Only one artery was tied, so that should a relapse take place, the resources of surgery are not yet exhausted.

Case 3.—George H. cannot be found. Mr. Maguire, the superintendent of relief, has caused inquiries to be made for him, but without any effect. He promised to come back and report himself, and to return to hospital if the fits recurred. I think we have much reason to take for granted that the fits are not incapacitating him from work at least, or he would have turned up again.

Case 4.—Mary S. is still at Dingle Mount. She had some fits before going there, and it was proposed to tie the opposite vertebral. This she declined, because she thought the fits were leaving her. Dr. Irvine wanted her to come back, but she likes the Epileptic Home so much that she would prefer to have a valid excuse for remaining there all her days. I do not think much improvement has resulted in this case by the ligature of a single vessel.

Case 5.—The imbecile girl upon whom I operated on February 2nd had her fits reduced from 48·5 per month, the average of the previous year, to an average of 11 per month since the operation. Even these are very much lighter. On May 2nd I tied the left vertebral artery. During the remainder of the month she has had three regular epileptic fits and several attacks of epileptiform twitchings. Her mental condition has improved to some extent, but as the imbecility has continued for ten years, and was the result of a fall, it is not likely to be benefited much by the diminution or cessation of the epilepsy. She can, however, now walk about the ward between two attendants, and astonishes them by the apt remarks she makes. These remarks cannot be elicited by questioning. She will sometimes reply to the nurse and talk with the other patients, but to questions of the medical staff she only replies by a stony stare and complete silence. This case can only show the effects of ligature on the most hopeless of all patients.

I will now publish in detail a few more cases.

Case 1.—Mary S. B., æt. 24, a married woman whose husband and only child are alive and well, came into the Liverpool Workhouse on November 26, 1881, with the object of getting rid of her fits, and of getting a severe burn treated which she had received in her last fit. About twelve months ago her menses stopped, and soon after the fits began. She had a good deal of medicine from her medical attendant, but no benefit seemed to result from her taking it. Sometimes she had as many as four or five fits in a day, some of which were slight, and others more severe and prolonged. Previous to December 20th her fits were not recorded. On that date I saw her in a severe one, and told the nurse to obtain a record of her previous history and to report daily the number of fits. During the next four days she had twelve fits, and altogether, from December 20th to January 18th, she had *thirty-one* fits. On the last-mentioned date the right vertebral artery was tied. The wound healed without anything occurring worthy of notice, but the fits still occurred, though in slightly diminished number and severity. Fourteen occurred from January 18th to February 2nd. On that date the left vertebral artery was also tied. This artery was of large size, and the one previously tied was small, which may account for the slight effect of ligature on the previous occasion. On February 8th she had a very slight fit, and on the 12th she had a severe one, followed by two minor ones on the same day. During the severe one she hurt her right bursal patella, which became acutely inflamed and suppurated. On February 18th she had a slight fit, and on the 20th another. From that up to March 16th she had seven fits. Her menstruation is now regular and comfortable.

As the effect of ligature of the vertebrals was not so effectual as I wished, and as there was evidently some external irritation exciting the cord to increased action, I determined to find out what this was. The connection of the onset of the fits with menstrual derangement caused me to examine the uterine organs. She had not complained of any uterine or ovarian symptoms, yet an examination discovered a distinct retroversion of the uterus. The instrumental treatment of pessaries

would probably have increased the irritation, and could not have been tolerated. Fortunately my operation for retroversion by shortening of the round ligaments[1] was especially applicable to this case. This operation was performed on March 16th, and was perfectly successful in bringing the uterus into the normal position and in keeping it there. No inconvenience was suffered during the healing of the wound, and the temperature of the patient never rose above 98·4°. On April 10th she was discharged, having had five very slight fits since the operation. She had improved physically so much, and her state of mind was also so much improved, that I had no hesitation in telling her that her fits would gradually diminish. I saw her on June 20th. She called to tell me that she feels perfectly well and has had no fits since she left hospital. The uterus was in the position in which I placed it. She is to come to see me every month for some time.

Case 2.—Anne G., æt. 18, single, a respectable quiet girl, was admitted into hospital in December last, suffering from epilepsy. About five years ago her father was killed, and when she heard the sad news she fell down in a fit. For four months she had no more, when two or three occurred. In about three months another recurrence took place. The interval gradually lessened until just before admission scarcely a week ever intervened without an attack. Latterly, in addition to characteristic epileptic attacks, she has had daily attacks of petit mal. She knows when these attacks occur, and is never unconscious. They consist in a tremulousness and loss of power both in the arms and legs, so that anything held in the hands at the time drops out of them, and occasionally the patient falls to the ground when the attack is more severe than usual. In general she only staggers, and can manage to sit down or lie down. Immediately after the attacks of petit mal she goes about her work as usual.

On February 15th both vertebral arteries were tied. She slept well during the afternoon of the day succeeding the operation, and her temperature in the evening was only 99°. At two o'clock next morning she felt rather thirsty, and her temperature then was 100·2°. This was the highest tempera-

[1] See 'Medical Times and Gazette' of April 1st, 1882.

ture reached, and the only time when it was reached. On February 20th she had a few jerks of the petit mal. On February 27th she had them again, and she was then ordered moderate doses of ergot and liquor arsenicalis. On March 1st the wound was quite healed. On March 6th she had an attack of petit mal, and slight attacks on six occasions up to March 26th. On that date she had a slight fit. On the 29th she was discharged to our convalescent home at Maghull. The following is the report of the nurse in charge there:—

June 6th.—"The epileptic upon whom I am requested to report has had only two fits since she came under my charge, one on the 3rd of April and the other on the 20th of May. Occasionally she takes a shake, when she will drop anything she is holding, and if standing she will sometimes fall. I believe she intends going home soon."

CASE 3.—Lydia F., 25 years of age, single charwoman. Admitted to hospital on February 17th, 1882.

Family History.—Father died from the effects of a fall, mother from a fit of apoplexy. Two sisters died of convulsions, whilst four sisters and one brother are alive and healthy.

Patient states that she has taken fits since she was 13 years of age. She had a child when she was 17 years of age. It died of convulsions a month after its birth. Since the age of 13 her fits have been almost of daily occurrence.

On February 19th she had a severe fit in the night, and up to March 23rd, when both vertebral arteries were tied, she had sixteen fits, all of which are reported by the nurse as severe ones. Her temperature on the evening after operation was 100·1°; next morning it was 99°, and next evening 99·6°. Afterwards the highest temperature recorded was 98·8°. On February 30th the left pupil was markedly contracted, and she complained of deafness and of singing in the left ear. Next day she complained of pains in her left shoulder. The pain in her left shoulder disappeared by the 17th of April, but the singing in her left ear, the deafness and the contracted pupil continued. The left eyelid also was noticed to droop slightly. She was discharged at her own request on April 28th, without having had a single fit since the operation. I saw her again about the middle of May, when she called to

tell me that she was about to take a situation at Preston as a domestic servant. The buzzing of the left ear has ceased, but some deafness still remains on that side, the left pupil is still contracted, and the eyelid droops. She is now in excellent health, and has promised to let me know, from time to time, whether a relapse has occurred. She is a stout, florid girl, whose education has been sadly neglected, but whose mental powers are otherwise good.

Case 4.—Alice Green, æt. 18 years, single, of no occupation, was admitted to the surgical wards on January 23rd, 1882. Her father and mother are both living, but the one suffers from a "bad leg," and the other from a "bad liver." She has had three brothers and three sisters dead, one of whom died from consumption, another from bronchitis. The cause of death in the others she does not know. She has six brothers and four sisters living and healthy. The patient has suffered from hip disease since she was six years old. No abscesses have yet formed, and the patient can still walk about, except on certain occasions when severe exacerbations of pain compel her to go to bed. Six months ago, when she was scrubbing a floor, she took a fit. Her mother took her to a doctor, who prescribed some medicines, which failed to have any effect upon the fits. Sometimes she has as many as four fits in the day, and she may have no fits for a week. From her admission to hospital, until the time of operation, she has had on an average a fit every other day. They are generally severe, and are followed by stupor for several hours. They are ushered in by mental excitement and extreme irritability, which has, of late, generally produced a quarrel with some neighbouring patient. The pains in her hip are severe during the premonitory stage of her fits. Her menstrual functions are apparently regular. My opinion of the effect of ligature on this case was that it would not be very beneficial. However, at the earnest request of the patient I tied both vessels on the same day, February 15th, 1882. Her temperature followed the same course as in the case of Lydia F. On the 19th the pupil of the right eye was observed to be contracted, and that of the right eye rather dilated. All went well up to the 27th, when she had a fit of hysteria. Next day she had a

very bad temper, and on March 6th she had her first epileptic fit since the operation, characterised by the nurse as a very slight one. On March 16th she had to be sent to a small ward by herself, for using bad language. She had two fits during March, and three during May, one of which is again described by the nurse as a hysterical one. On June 1st she had another slight fit in the evening. The patient is still in hospital undergoing treatment by rest for the hip disease, so that a full opportunity will be given of ascertaining the permanence of the effects of the operation. She is now in a most miserably despondent condition on account of her hip, and I am not very hopeful of a complete cure of her fits.

Case 5.—Anne C., æt. 25, admitted to hospital on March 2nd, 1882, from Dingle Mount Epileptic Establishment.

Dr. Irvine, the medical officer to that Institution, has kindly supplied me with the following table of her fits during the time she was there—

	1881.												1882.		
	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.
1st week	2	1	4	2	1	6	8	8	0	8	0	4	0	10	8
2nd „	0	6	2	0	1	0	2	0	0	0	5	0	5	0	Work-house on 2nd.
3rd „	0	2	0	7	2	5	0	0	2	8	10	2	7	0	
4th „	5	2	3	6	5	16	0	18	8	0	0	12	11	6	
Totals	7	11	9	15	9	27	10	26	10	16	15	18	23	16	8

Total during fourteen months and a day, 213.

The previous history of this case is mislaid, and the patient has gone home. I am therefore unable to give any particulars at present of either her own or her family history.

On March 16th the right vertebral artery was tied. On March 29th she had a slight fit, and three more up to April 12th. The wound healed by the first intention.

On April 12th she had the opposite vertebral artery tied. The wound healed as kindly as before. She had one slight fit on May 14th, and two more up to May 25th. She went

home to her friends on May 27th, having promised to see me once a month and report progress. The reason of my sending her out of hospital so soon will be explained in my remarks.

Case 6.—Elizabeth B., æt. 15, admitted to hospital April 3rd, 1882, suffering from fits.

Father and mother both alive and healthy, and so are the other three children of the family. When she was fifteen months old she had a severe fit during difficult dentition. She had no more till she was about seven years of age, but, from that until the time of her admission into hospital, they have continued constantly.

I have had her under treatment for the last three years, and have given her as much as three drachms of bromide in the day, without producing any permanent effect upon the fits. Just before admission to hospital, the attacks were frequent and severe, and but for the hope of benefit by operation, they would have been compelled to get her into some Home. Lately she had twenty-four fits in a day. Her menstrual functions were regular during the past two years. When I first took charge of the case, the menses began, and by perseverance in the bromide at that time a cessation occurred for nearly three months. Since that time there has been no prolonged cessation.

Both vertebral arteries were tied on April 5th. Her temperature went up to 100 for the first three evenings, when it came down to normal and continued so. During the pyrexia some thirst was experienced. On April 15th and 16th she had two very slight fits, which the nurse could hardly describe as epileptic. She was "out of them" as soon as the nurse got beside her, and quite conscious. On April 24th the wounds had healed, and the patient was taken home by her mother. She has been under my observation ever since. I saw her on June 20th. She has had no fits since she came home; her health is good, her time is spent in knitting, looking after the other children, and in doing other light work. Her menstruation, which was delayed for a fortnight, probably through the operation, has returned a fortnight ago without any disturbance.

Case 7.—Sarah A., æt. 12, admitted to hospital February 22nd, 1882. Her family history is good, and the other members of the family strong and healthy.

Five years ago the fits began without any apparent cause. She had three that day. They continued intermittently until three years ago. She then passed through an attack of scarlet fever, and after that the fits increased in frequency. Latterly she has had as many as fourteen in one night. Until the last three months she could tell when she was going to have them, but now they come without warning, and except for the feeling of prostration, she does not know she has had a fit. Her memory is failing, her speech is limited to monosyllables, her countenance is stupid-looking and "puffy." An examination of the urine revealed nothing the matter there.

She had two fits the evening she was brought in, two next day, four next day, five next day, five next day, two next day, four next day, and five on April 28th.

On March 1st she had the right vertebral artery tied. On March 3rd she had three fits, and afterwards they occurred without any apparent abatement. The wound healed without anything calling for comment. On March 11th the attacks became reduced to two daily instead of four. On March 18th she was sent to our Children's Convalescent Home at Maghull, where she stayed till March 25th. During her stay there the average of four fits daily was again reached. Her stupidity is appreciably lessened.

On March 29th the left vertebral artery was tied. She had two fits during the night subsequent to the operation. The wound trouble was unimportant, and may be dismissed. On March 30th she had two fits, and on March 31st she had two fits. The case was a most stubborn one, and I almost despaired of improvement when I heard that they had come on so rapidly after the second operation.

At the time of writing, June 5th, the improvement has been very decided. Since the 19th of May she has only had five fits, and these not very severe. Previous to that date the improvement began by her passing whole days without any fits, and in her often having one slight fit in the day. Then a day would come in which four or five fits occurred. On

May 11th an ice-bag was applied to the spine, and has been retained there ever since.

Case 8.—Mary McG., æt. 11, was admitted to hospital on March 7th, 1882. Her father died suddenly; her mother is living and strong. Has had a brother who died of scarlet fever, and four sisters who are all living and well.

Four years ago this child was at play when she took a fit. It lasted some time, the mother says a quarter of an hour, and she has had them ever since. General health good, appetite normal, urine free from albumen.

From February 11th until March 1st, when the right vertebral artery was tied, she had nineteen fits. Her temperature that evening went up to 103° 8′, pulse 116, resp. 54. She suffered from headache, and evaporating lotions were applied to her forehead. On March 2nd her morning temperature was 98° 8′. In the evening it was 99°. On March 9th her temperature again rose to 102°, and she had a fit. Next day the temperature was normal, and she had no more fits till the 13th. On that date she had one fit.

On March 15th the right side of her face was swollen, and her right eyelid pale and puffy. Her urine was examined: sp. gr. 1011, no albumen. On March 18th, as she had no more fits, she was sent to Maghull.

Immediately she went out there the fits recommenced, and she had three or four daily up to April 8th. From April 8th up to April 15th she had no more. On April 18th she had one, and from that up to April 26th she had seven. On April 26th the left vertebral artery was tied. The highest temperature was registered on April 27th, and reached 101. On May 7th she had two fits. On May 9th an ice-bag was applied to her spine. Since that time up to the present, June 5th, she has had three fits. The mental condition of both these patients has much improved.

Case 9.—Joseph L., æt. 23, was admitted to Liverpool Workhouse Hospital on September 11th, 1873 (epileptic ward) where, in spite of constant large doses of bromide of potassium daily, he has had, on an average, thirty fits per month. In January, 1882, he had thirty-eight fits; in February, thirty-one; and up to March 29th, thirty-five.

His mother is alive and healthy. His father is dead. He has had the fits since he was five years of age, brought on, it is alleged, by a fright.

On March 29th both vertebral arteries were tied. His temperature went up to 101° 2′ on the 30th, and varied from 99° to 101° up to the 7th of May, when it resumed the normal condition. His tongue was rather dirty, and there was a good deal of gastric disturbance. During the evening of the 30th he had a fit, and during the month of April he had twenty fits; during May he had fifteen fits, and up to June 6th only one fit. On May 6th an ice-bag was ordered to the spine, and was continued up to the 27th. He is now in fair health, and has lost to a considerable extent the stupid look that he formerly had.

Case 10.—Thomas H., æt. 23, had a fit when he was 14 years of age, brought on by a fall. A depressed scar at the back of his head showed where the injury was received. The wound is not much affected, but he is liable to sudden outbursts of anger. His mother died of bronchitis, and his father from "drink." He has a brother and one sister in good health. On July 13th, 1881, he had the left vertebral artery tied. All went on well, and the wound was healed by July 23rd. On July 16th he had a fit, again on the 17th and 19th, and no more up to July 28th. At this time I considered the case a failure, because *some fits* occurred after operation, and tied the right internal carotid on July 27th, thinking that the cause was perhaps more cerebral than medullary in its location. All went on well with the wound. On July 28th he had some twitching without loss of consciousness, and on August 1st, 2nd, 3rd, 4th, 5th, 10th and 13th he had each day a regular fit. During the remainder of August he had four fits. During September he had five fits, and up to October 8th he had two slight ones. He was now sent back to the epileptic ward, and during the rest of October he had only one. Up to the 16th of November he had only another fit. Since that time his average number of fits per month was twelve. During the previous year the average number of his fits per month was twenty. I would probably have tied the opposite vertebral artery before this (as the patient was quite willing), but that the fits have become much milder in character. The patient

is a most useful person in the epileptic ward; his outbursts of anger are leaving him, and he is so much improved in all respects that I am inclined to let him alone for a year, to see how far time will finish the cure. He has had no medicine for the last six months.

Case 11.—Peter McDonough, æt. 22, has had epileptic attacks since he was 11 years of age, brought on by his father beating him. He does not know when the fits are coming on. They are very severe, and produce much stupor and debility afterwards. His average number of fits for the past year were fifteen per month, and during last August, 1881, he had nineteen.

On August 31st the left vertebral artery was tied as well as the left common carotid. His temperature went up to 103° 8′, and his tongue was dirty on September 1st. Temperature normal September 2nd. On that date the left pupil was contracted. On the 3rd the wound was dressed, and looked clean and healthy. On September 10th the fits recurred, and during the month he had six fits. During October he had eight fits. He was then sent to the epileptic ward. During November he had only two fits, and up to December 20th he had two more. In the last one he was found lying on his face suffocated with a bitten tongue. The night watchman who goes about amongst the beds did not hear anything unusual until he saw him lying on his face. On turning him over he was dead. A post-mortem examination showed that the ligatured arteries were occluded; the brain and cord were healthy, and so were all the other organs except the kidneys, which were *large* and *white*. This was a surprise, as no signs of Morbus Brightii ever appeared, nor was the disease at all suspected. The lungs and tongue showed the ordinary signs of death from suffocation.

Case 12.—William Foran, æt. 26. Has had fits since childhood. No cause can be alleged except dentition. He does not know when they are coming on. They are ushered in by screaming and spasmodic twitchings of the muscles. He is sometimes very violent, and even maniacal after they pass off, but generally he falls asleep. His father and mother are dead (drink). He has one brother living and healthy. His average number of fits are ten per month.

On August 17th, 1881, the left vertebral artery was tied. He had no fits till August 24th, and had only two during that month. During September he had four fits. During October he had seven fits; November, twelve; and December, seven. On January 11th the right vertebral artery was tied. On the 16th he had his first fit, and during the remainder of the month he had three other fits. His mental condition had much improved, and the maniacal attacks had completely disappeared. During February he had three fits. He was then sent back to the epileptic ward where, during March, he had ten fits. During April he has had eight, and during May six. His fits are much milder, and are not followed by any mania. Many of the fits are really hysterical ones. They are, however, so intermixed with true epileptic attacks that it is difficult to say in what proportions, especially as we have to depend on the information of mere non-professional witnesses.

Case 13.—Charles Hutchfield, æt. 29, single. Has been the subject of fits since he was 13 years of age. He was then learning to be a wheel turner. The attacks average sixteen per month, and are ushered in by a peculiar twitching in the right hand and arm. The limb is flexed at the fingers, wrist and elbow to such an extent as to render it useless. He can undo the flexures voluntarily to a slight extent, but he cannot grasp anything with the hand owing to the ataxic movements of the muscles. Through long disuse the limb is somewhat rigidly fixed in its position.

On January 25th, 1882, the right vertebral artery was tied. He had a fit during the evening of the day of operation. On the 26th his temperature went up to 103°, next day it was 100° 2', and next day it was normal. His second fit occurred on February 1st, and successive ones were noticed on February 5th and February 7th. On February 8th the left vertebral artery was tied. The temperature ranged from 100° to 102° during the first three days.

On February 9th, to my surprise, the patient called my attention to the great improvement in the paralysis and stiffness of the right hand. He cannot say whether the improvement set in before the second operation or not, but I am inclined to think from the great change noticed to-day that it

could scarcely have taken place since the second operation. He can now move all his joints in a regular way, and pick up a pin. On February 18th he had the first fit since the second operation. He had two more fits during the remainder of February. During March he had seven fits; during April, ten fits; during May he has had six fits. The fits are, however, much lighter. His mental condition has improved, and the power of the right arm is almost normal.

I lost a little idiot girl through septic pleurisy after the operation. She was a hopeless idiot, who tore off the dressings and opened up the wound with her fingers during the momentary absence of the nurse on two occasions. It is the only death from the effects of the operation that has occurred amongst my operations for epilepsy at the Liverpool Workhouse.

I have five more cases at the Liverpool Workhouse whom I have operated on, but too recently for me to say more than that they promise well, and are out of danger.

On nine other cases I have operated in conjunction with Dr. MacDonnel. They are all under his observation, and will, no doubt, be published by him when the results are so tested by time as to become valuable.

Remarks.—I have little to add to my former papers as to the mode of performance of the operation. A linear incision commencing opposite the lower end and on the outer side of the external jugular vein, and about an inch above the clavicle, is carried upwards for three inches along the external border of the sternal mastoid. The layers of fascia are cut through, to the same extent, until the fatty tissue over the anterior scalenus is reached. With the finger the sulcus between the anterior scalenus and the longus colli can be opened up, and the sixth cervical vertebra reached by judicious teasing with a strong blunt probe or director. The artery will be generally easily found, provided no veins are injured. If the vessel is not found there, it will be found running up to the inner side. The sterno-mastoid and the external and internal jugular veins should be well protected and retracted by good retractors during the operation. I generally use the spray during the operation, but I am often compelled to stop it for a little if the

wound is deep or the artery difficult to find. I insert a drainage tube with a phalange outside like that of a tracheotomy tube before the wound is closed, then stitch up the wound, and lay over it a sponge that has been steeped in carbolic acid for twenty-four hours. Over this a dressing of gauze is laid that covers the neck and shoulders. This is retained in position by an elastic bandage. The sponge exercises pressure upon the sides of the deep wound and promotes drainage. The dressing is not changed for three days, when a piece of oiled lint and cotton wool is substituted for the more expensive dressing. No blood is lost during the performance of the operation if it is performed deliberately and skilfully. The danger to life is very small.

The Utility of the Operation.—In the twenty-one cases here alluded to, three have been quite well for nearly a year. Nine others have been so free from fits, and for such a space of time, that it may be said a cure has resulted, or is likely to result; and eight have improved in so many respects, or are improving so steadily, that the operation would be justifiable were no better results ever obtained. The case of the one who died in a fit two months after his discharge from hospital, was an accident such as occasionally occurs in all epileptic establishments. The state of his kidneys was rather surprising, and has caused me to examine the urine in all cases before operating. The diminution of the fits after operation, makes me doubt whether the renal disease had any influence on their production.

I now think that ligature of the vertebral arteries ought to take its place as a recognised operation for the cure of epilepsy, when other means, such as the removal of peripheral causes of irritation and the soothing of irritated nerve centres by drugs, have failed.

The surgical difficulties of the operation can easily be overcome by practice. My method of operation does not involve any important structures. The danger to the patient's life is very small, and should the operation fail to relieve the fits, the patient is in no respect worse off than before. No instance of any deformity or disability on the part of the patient has, as yet, been noticed by me or reported to me by any one.

To afford reasonable hopes of success, the epilepsy must not be allowed to become too chronic. It should, therefore, be performed as soon as it becomes evident that drugs have no curative effect, but that they only diminish the fits by temporarily paralysing the nerve centres. In some of the very chronic cases improvement has resulted, and I am not yet prepared to say how far that improvement may yet progress, or how far it may yet retrogress.

Before performing the operation, every external irritating cause should be, as far as possible, removed. This cannot always be done. Nor is it necessary, provided it be not one that by its nature or severity is the sole cause of the epilepsy. When the sensibility of the epileptic nerve centre or centres is reduced, that cause will have no more effect in producing fits than it has in any other healthy non-epileptic individual. The retroflexed uterus was too powerful an exciting cause, either from its nature or extent, to allow the quieted nerve centres to rest.

It may be said, that had the retroflexed uterus been brought into place, the ligature of the vertebrals might have been unnecessary. Perhaps so. I have, however, a case on hand where the operations were performed in the reverse order. The straightening of the uterus did not cause the fits to cease, but the ligature of the vertebrals promises to do so.

Where the exciting causes of epilepsy are cerebral, and are accompanied or alternating with uncontrollable anger or maniacal excitement, the effects of operation are not so apparent or so permanent. In the case of Alice Green and Wm. Foran, the imperfect success probably depends on cerebral excitement, and the same cause will, I fear, render the operation useless in another case now under my care. The exciting causes are in these cases so powerful and so uncontrollable, that the stimulation of the epileptic centres soon restores the blood supply, on the principle that *ubi stimulus ibi flux.*

In old standing cases of chronic epilepsy the operation mitigates the disease, and I am inclined to think may ultimately cure the fits. In my last case, where the paralysed arm gained its power, the effect upon the cord must have been decided, and was unmistakable. The fits have returned in

diminished number and severity; whether the fits will continue to diminish owing to a gradual improvement in the epileptic region, or whether the fits will resume their normal frequency and severity, and the paralysis return, are questions of much interest that time only can enable us to answer.

A point that I have been led to consider of great importance in the after-treatment, is to turn the patients out of hospital as soon as the wounds have healed. When the fits show signs of recurrence after operation, ice-bags to the spine are valuable. The exercise of the patient's faculties and limbs, promotes the more even distribution of blood through the body. I have had the best results with those patients who could at once go home to the comforts and variety of home-life. With the exception of the idiot boy, I have had the worst results with those who had no friends, and whose only refuge was the epileptic wards. The indolent objectless life promotes sensual irritations and digestive disturbances, prolific causes of spinal and cerebral congestions, that probably upset, in a very short time, all benefit from operation. These are supposed to be the exciting causes of epilepsy. I am more inclined to consider them in many cases the results of the lazy life to which hopeless epileptics are doomed. Epileptics are no more subject to these errors than others who lead a similar enervating and caged life. For these reasons I am afraid that removal of the sexual organ in the male or female will rarely succeed in curing a disease that does not depend on those organs, but that may, and no doubt often is, aggravated by their excessive or depraved functional activity. When epileptics are confined, gastric desires and other excitements are their only pleasures, and the prevalence of these in epileptic wards has caused the opinion to be formed that (instead of these being a result of confinement and laziness, and the absence of healthy mental and physical occupation) they are the cause of the epilepsy.

As to the theory of epilepsy upon which the operation rests, I have nothing to add to my former papers. The epileptic centres may be rendered hypersensitive through some powerful shock. The circulation, is in many cases, restored to the normal, and the hyper-excitability is shown by only one fit, or a series of fits. In other cases the hyper-sensibility exists,

but only produces epileptic attacks in consequence of peripheral excitement caused by worms, gastric or sexual disturbances, or of the cerebral irritation produced by anger. That we can influence for good the nutrition of the medulla, and of the upper, or even the lower, part of the spinal cord by ligature of the vertebral arteries, a paper of mine in the contemporary number of the 'Liverpool Medico-Chirurgical Journal' incontestably proves. That paper should be read in connection with this one, and is supplemental to it.

Lately I have always ligatured both arteries, so that the effect may be as great as possible, and that all that can be done may be done at once. The risk is not any greater, and my experience shows that the effect is greater, and more likely to be permanent. It would be premature to enter more fully into my theories of epilepsy, as modified by my operative experience of it. I have, therefore, contented myself with giving, at the request of Dr. Hughlings-Jackson, a synopsis of the results of this new operation up to the present time.

THE NERVOUS SYMPTOMS OF MYXŒDEMA.

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THE symptoms of myxœdema are now so familiar to the medical profession, that a very brief general description of the disease will suffice for the object we have in view.

Throughout the body there is a solid œdema affecting the skin and connective tissue.

The parts, however, are not anasarcous, or, in other words, they do not pit on pressure. The facies is pathognomonic. The expression is placid and mask-like, the features broad, puffy and coarse, the nostrils swollen, the lower lip thickened, everted and pendulous, the mouth widened transversely. Over the cheek and nostril there is a well-defined red patch, contrasting with the pallid, porcelain-like area beneath the lower eyelid.

These characteristics are so striking that it is possible to diagnose this affection *de visu*, and without further examination of the patient.

The tongue, fauces and palate are also swollen; the speech is slow, nasal, monotonous; the hands and feet are coarse, shapeless and broadened transversely; the skin is dry and scaly, perspiration and the excretion of sebaceous matter being almost suspended.

The thyroid gland is much diminished in size, and the subclavicular fossæ are often filled in with soft masses. The hair, teeth and nails share in the general mal-nutrition.

The urine contains no albumen, except in the later stages; but, as we have pointed out elsewhere, the quantity of urea excreted is remarkably lessened.[1]

[1] 'Progrès Médical,' Nos. 30 and 31, 1880.

When albuminuria occurs it is, as it were, an accident—a frequent accident in the later phase of the disease, but nevertheless an accident pure and simple. It is necessary to lay stress upon this fact, since the idea of a relationship between myxœdema and chronic renal disease has been rather strongly insisted on recently by Dr. Mahomed.[1] This question we do not propose now to discuss, but simply content ourselves with assuming that myxœdema is a disease characterised by a special symptomatology and morbid anatomy. The temperature is almost invariably subnormal, and there is a constant sensation of cold, so that even the hottest weather causes no uncomfortable feeling of warmth. The bodily movements are performed slowly, though usually without imperfection. Falls, however, sometimes occur, and are dependent on the irregular action of the muscular groups, which are physiologically antagonistic.

In short, there is often some degree of incoordination. The muscles, as in ataxy, lack tone, but are never paralysed nor atrophied.

The tendon reflexes are, in our experience, preserved intact.

In a case reported by Dr. Hammond,[2] of New York, the ataxic symptoms were very evident. "The gait was staggering, the feet were not lifted clear of the ground, the grasp of the hands was weak, and the articulation was sluggish and indistinct. There was marked difficulty of co-ordination both in the upper and lower extremities. Although the patient could stand with the eyes shut, she walked with an uncertain step, unless her eyes were directed to the ground, as is the case in locomotor ataxia. She could not put the finger on any given part of the face unless she had her vision to guide her, and even with that assistance she did not readily and with certainty direct the movements of the hands."

No mention is made of the condition of the patellar tendon reflex.

Lightning pains, similar to those experienced in tabes

[1] See Report of Clin. Soc.'s Meeting, 'Brit. Med. Journ.,' Dec. 24, 1881.

[2] "On Myxœdema, with special reference to its Cerebral and Nervous Symptoms." 'Neurological Contributions,' vol. i., No. 3.

dorsalis are occasionally observed; but whether the cause in each case is identical we are not prepared to say.

Common sensation is generally much impaired, apparently never annihilated, but blunted and retarded.

Subjective sensations, such as formication and "pins and needles," are very commonly experienced.

In the late stages of the disease the special senses are liable to become affected. In Hammond's patient there was double optic neuritis, objects appeared blurred and surrounded by a halo, and there was occasional double vision. The pupils were equal, but very sluggish to light. Visual disorders, however, are by no means constant, and may even be absent up to the last.

Deafness is more common. It depends, perhaps, as Hammond believes, on a diseased condition of the auditory nerves themselves.

Subjective auditory sensations, such as tinnitus and "roaring of waves," in both ears (Semon) are described.

As might be expected, the senses of taste and smell are liable to impairment. Hammond found the latter almost absent in his patient, who was unable also to distinguish fish from roast beef. It must be mentioned that a patient of Dr. Ord's presented marked bulbar symptoms. In another case exophthalmos preceded the onset of the disease.

Protrusion of the eye-balls, indeed, has been observed more than once.

To the possible significance of these two conditions we shall allude later.

Fatigue, on the slightest bodily exertion, is a very constant symptom in myxœdema. The most ordinary duties of life, such as dressing, are not only performed with great slowness, but entail an amount of lassitude quite out of proportion to the muscular effort put forth. The patients are consequently inert and sluggish, and indisposed for any exertion, however trifling. Their condition, indeed, may be aptly likened to that of the cold-blooded vertebrates. Headache, sometimes of a very severe character, is very commonly found to be present. Somnolence during the day is a frequent symptom; but sleep at night is generally disturbed by unpleasant dreams, from which the patient awakens much startled and terrified.

The symptoms referable to the cerebrum are very interesting, and at present by no means fully understood.

In addition to the bodily inertia, to which allusion has been made, there is always very marked intellectual lethargy.

The thoughts are evolved slowly, and often a distinctly prolonged interval elapses before the nature of a question addressed is grasped by the patient.

Memory, according to some observers, for recent events in particular, is in most cases impaired, but aphasia, in the strict sense of the term, is of doubtful occurrence. Dr. Semon,[1] however, found that his patient often called things by their wrong names.

We must note the very frequent, almost constant, existence of emotional disturbances, under the influence of which the patchy redness over the cheeks and nostrils is momentarily brought into strong relief.

There is occasionally found in myxœdematous patients an exaggerated degree of what they call "nervousness."

In a case of Dr. Ord's, which we described in the 'Clinical Society's Transactions for 1881,' the condition was that of agarophobia.

The patient's state may be given in her own words:—

"I could always walk across a room, but when out of doors I feel as if there was too much space, and am in constant fear of falling. If I met a child I should stop, as the slightest touch would knock me over; a stone on the path would make me stumble if I trod on it."

In Dr. Semon's case, reported in the same volume, the "nervousness" was of a different nature. "She was afraid that she would jump out of the window, or do something formidable. But still she tried her best to prevent herself thinking of it."

In this instance we see a tendency to impulses which, although not suicidal, are nevertheless suggestive of an unstable mental condition.

True insanity in the later stages of myxœdema has, indeed, been several times observed, and its causation has given rise to a certain amount of discussion.

[1] See 'Clinical Society's Transactions for 1881.'

When mental alienation supervenes, it usually takes the form of hallucinations of hearing and sight.

Hammond's patient heard the voices of Frenchmen abusing and threatening her; but curiously enough she never saw her persecutors. Her visual hallucinations consisted chiefly of apparitions of departed friends. In an interesting *résumé* of myxœdema,[1] Blaise narrates a case in which hallucinations formed one of the most prominent features of the disease.

The patient complained of bad tastes and odours, of insulting and obscene remarks addressed to her by those around. At first she quite recognised the falsity of these impressions; but in course of time she lent them entire credence. She thought her body exhaled a repulsive odour, that she was accused of killing her child, that her face was covered with a mask, and at other times that she had the head of a dog. Ideas of persecution became very pronounced. She mistook the nurses for men, disguised as women, who wished to kill her, and frequently uttered cries of terror under the influence of this delusion. In spite of the severity of the symptoms, the condition of the patient improved to the extent of a partial cure.

M. Blaise noticed that the mental disorder and the swollen state of the integuments underwent amelioration simultaneously. This evident relation between the two lends some support, he believes, to the theory put forward by Dr. Ord.

The question thus opened up has already been discussed by Dr. Savage in the 'Journal of Mental Science, Jan. 1880.' Are we to believe with Dr. Ord that the symptoms of myxœdema depend on the fact that the peripheral nerve-endings are so padded and isolated by solid material that they are partially incapacitated to receive the healthy impressions which, when transmitted to the higher nervous centres, regulate both the bodily and mental functions?

Is this explanation sufficient?

The case given by Blaise certainly lends fresh weight to this hypothesis.

But on the other hand, the observations of Hammond and Semon show that the swollen condition of the integuments may occur comparatively late in the course of the disease.

[1] 'Archives de Neurologie,' vol. iii., Nos. 7 and 8.

In his first autopsy, Dr. Ord was unable to find any lesion in the brain and spinal cord; but subsequent investigations brought to light very evident changes in these parts.

Indeed, it is scarcely conceivable that the organ, to which some of the most characteristic symptoms of myxœdema are referable, should alone remain intact.

In face of the fact that the brain exhibits degenerative changes, it seems to us a forced conclusion to ascribe the cerebral symptoms of the disease entirely to peripheral alteration. Elsewhere[1] we have suggested that the main features of myxœdema may be explained on the basis of a sympathetic lesion affecting the general nutrition.

The principal symptoms of the disease may be summed up under the following heads:—

(1) Slowness of bodily movements.
(2) Slowness of intellectual operations.
(3) Constantly subnormal temperature.
(4) Diminished excretion of urea.
(5) Solid œdema of skin and connective tissue.
(6) Decreased size of thyroid gland.

The remarkable diminution in the amount of urea excreted by the kidneys, to which we first called attention, has since been verified by several observers.

Is the urea actually formed in the system, but retained in the blood?

We think not, because uræmic poisoning would assuredly be experienced more commonly if this were the case. Yet such symptoms were absent in a woman who passed as little as 95·6 grains in one day, and a daily average of 184·3 grains, when placed on a nitrogenous diet.

It must be borne in mind, however, that these patients eat badly, and that on this account alone the formation of urea would be below the mean.

The probable explanation is that the urea is not formed in the system, because the tissues are not subjected to the usual wear and tear of life.

Heat-formation is thus retarded, and the consequence is diminished body-temperature, as shown by the thermometer,

[1] Op. cit.

and the subjective sensation of cold. Every thought and every act is dependent on chemical changes in the tissues, resulting in the formation of simpler products, of which urea is the chief, and a concomitant elevation of temperature. These destructive, but essential changes, upon the active performance of which our healthy being is due, are partially arrested in myxœdema.

It is a matter of common knowledge that the vasomotor filaments of the sympathetic, through their action on the blood vessels, exert a powerful influence over nutrition. It is well known, too, that there are two kinds of fibres having diametrically opposite effects on the calibre of the vessels. Irritation of the *vaso-constrictor* fibres stimulates the excito-motor ganglia contained in the plexus which is distributed to the vessels, whereas irritation of the *vaso-dilator* fibres inhibits these ganglia, the result being vascular dilatation through paralysis of the muscular coat.

The supreme vaso-motor centre is located in the medulla oblongata, but secondary centres probably exist throughout the whole length of the cord.

From what precedes, it is clear that a condition of angiospasm may supervene, in consequence of lesion of either the vaso-constrictor or vaso-dilator filaments. We do not presume to indicate the nature of the lesion, which, of course, can only be discovered by microscopical research, nor, indeed, do we venture to suggest confidently its localisation; but we simply wish to throw out a working hypothesis which, at any rate in our opinion, has a *primâ facie* probability. Assuming then a state of angiospasm universally existing, we have a fair explanation of the main symptoms of myxœdema. As a consequence there would result diminished tissue-waste and heat, together with bodily and mental sluggishness.

And if vaso-motor paralysis be the cause of the enlarged thyroid in exophthalmic goitre, may not vaso-motor spasm, or angiospasm, be invoked on behalf of the opposite condition of this gland in myxœdema?

We have no hesitation in mentioning an apparently contradictory, but nevertheless a highly significant, fact.

In a case of Dr. Ord's, exophthalmos preceded the onset of

the disease. A certain degree of protrusion of the eyeballs has, indeed, been observed more than once. On the theory in question, the solid œdema of the connective tissue is not quite so readily explicable. But angiospasm does not affect the blood-vascular system alone, but involves also the lymphatics. Under such circumstances there would be a generalised lymphatic obstruction, or, in other words, an inability, as it were, on their part to remove the products from the lymphatic spaces within the connective tissue. "The result is the development of what is often termed solid œdema, or leucophlegmasia of the implicated portion of the body. This becomes swollen and tense, and of a pale waxy hue, but does not pit on pressure as in ordinary venous dropsy."[1]

Such a condition is seen in a form of enlarged tongue, described by Virchow, occasionally in the upper extremity, but more often in the lower limb, abdomen, and scrotum, constituting the disease called elephantiasis lymphangiectodes. It is conceivable that these accumulated products should, in the course of time, undergo changes resulting in the formation of mucin; but whether these changes are of an organising or of a degenerative nature in the special case of myxœdema, it is at present difficult to say. In conclusion we believe:—

(1) That in the early stages myxœdema is essentially a disease of imperfect nutrition, dependent probably on generalised angiospasm.

(2) That the solid œdematous condition of the skin and connective tissue is due to a form of lymphatic obstruction, which may also be ascribed to vaso-motor influence; and that the accumulated products undergo changes which result in the formation of mucin.

(3) That the condition of the thyroid gland is also to be explained on the vaso-motor hypothesis.

(4) That the more severe mental symptoms, such as insanity, occurring in the later stages of myxœdema, are due to alterations in the brain itself.

(5) That, although myxœdema is a distinct morbid entity, it is probably intimately allied to certain other disorders, such as sporadic cretinism and scleroderma.

[1] Dr. Bristowe's 'Theory and Practice of Medicine.' Third edition, p. 558.

(6) That the solid œdema, which is universal in myxœdema, may be localised to various parts of the body, such as the tongue and extremities.

(7) That the primary and essential lesion probably exists in the peripheral sympathetic system, and perhaps, too, in the supreme centre in the medulla oblongata, this last supposition being based on the occasional occurrence of bulbar symptoms in myxœdema.

TYPHOID FEVER IN RELATION TO DISEASE OF THE OPTIC DISC.

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It is now many years since my attention was first aroused to the knowledge that patients, having suffered from typhoid fever with cerebral complication, had material diminution of vision shortly after convalescence. I have never yet met with any case of disc mischief after typhoid fever when the patient had passed through the disease without prominent meningitis (cerebral).

The condition of the disc is somewhat curious, for although inflammatory mischief is undoubtedly present, yet it does not take the shape of an ordinary neuritis. If I may so term it, I should say a subacute neuritis in which effusion was sufficiently pronounced to render diagnosis between health and disease a certainty, yet of so slight a character as to deceive any but the most practised in the use of the ophthalmoscope. Exceptions to this condition are sometimes met with.

Unlike an ordinary form of disc mischief following inflammation of the meninges, the visual defect is not noticed until long after the health of the patient is restored. The discs (for as a rule both discs are affected) present an unusual appearance, having a peculiar tint of redness difficult to describe, bordering on a brown, or we might say a brown-red. If considerable attention is paid to the distribution of the retinal vessels, it will be found that the arteries have an appearance of health, but the veins carry too much blood, and in many cases are decidedly tortuous. The contour of the disc is, as a

rule, unaltered, and quite distinct even from the sequela of a slight neuritis, or even from a mild form of neuro-retinitis: yet vision is sadly defective, and in many cases ends in the cancelling of important engagements, and not infrequently causes the ruin of those who depend on perfect vision to earn their daily bread.

The majority of patients suffering from typhoid amblyopia present objective symptoms indicative of acute kidney mischief, and in many cases I used, and before making ophthalmoscopic examinations, to express a very decided opinion as to "Bright's disease" being the cause of the defective vision. As a rule, patients suffering from typhoid amblyopia, have a decided and well-marked appearance of cellular dropsy (facial), with the ordinary pallor peculiar to cases of kidney disturbance. After repeated examination of all cases I have seen (with but one exception) have I failed to detect albumen in the urine.

In this case I was associated with my late colleague, Dr. Chadwick, and my present colleague, Mr. Teale. The case, a typical one of typhoid fever, very early showed marked failure of vision; the urine frequently examined was albuminous, and the ophthalmoscope disclosed effusion into one disc and an abnormal condition of the other. The effusion, although distinct, did not present any typical feature of a model neuritis, although approaching to it more than in any other case I have yet seen. It may be well to state that this is the only case in which I have had occasion to use the ophthalmoscope in an acute stage of the disease. The patient is now living, and has been examined by me from time to time during the twelve years since the disease attacked him. For many months after convalescence no appreciable difference as to the amount of effusion was apparent: the discs—the right one having been more diseased than the left—remaining in an unhealthy condition, presented a raised surface, with a decided and easily-discerned brown tint, as if some artist not satisfied with nature's handiwork had added some unseemly brown paint, rough in application and dreary in colour. Years elapsed ere the patient was fairly fit for work (profession, architect), and indeed it is not by any means a certainty that the disc mischief did not tell so strongly against him as to

effectually and for ever militate against his chance of professional success.

A prominent feature of the disease is a slow but certain invasion of the powers of vision. I have known a patient to be discharged from the fever hospital as convalescent, and who has apparently remained so for six months or a year, but who never was, so far as vision is concerned, anything but diseased. It may be that the early discovery of defective vision in these cases is not easy of detection, as typhoid patients do not usually resume their ordinary avocations till long after leaving hospital; but, be this so or not, it is an indisputable fact that the disease of the optic discs grows progressively worse, and, though not as a rule ending in atrophy, so far as my clinical knowledge of the cases teaches me, terminates in a condition truly lamentable for those who have to depend on perfect eyesight for a maintenance.

Although ophthalmologists have found many of these cases to end in absolute atrophy, yet, with but one exception, I have not known it so to end; and in that case alcohol was so mixed up with typhoid poisoning that it was very difficult to draw the line.

A fact worthy of note is that the disease is much more frequent among women than men, and especially amongst childbearing women—(I now refer to the intra-ocular disease). It has never occurred to me to note one such case amongst the boy population, although I can cite many instances in which the disease has attacked girls.

I have never yet met with a case in which a distinct and very accurate history of meningitis could not be obtained. In many of the more marked cases the mania had been very violent, and reminded me much of the clinical history of a decided alcoholic delirium tremens. I have yet to learn that the meningitis may not spread to the cord, and so give rise to that peculiar form of primary white atrophy so easy of detection by the ophthalmoscope, but the true clinical history of which appears to be enveloped in mystery.

I must trespass, so that I may describe one of the first cases of this disease I ever saw, believing, as I do, that its clinical history may not prove without value.

Elizabeth M., æt. 50, "in August, 1867, suffered from typhoid fever—'My eyesight previous to the fever was remarkably good, for I had not used glasses. During the fever I was very delirious, the nurse assuring me it was one of the worst cases she had seen. I remained delirious (without my senses?) for more than two days, when I awoke without any distinct vision, and could only see the blaze of the fire, and was practically blind for one month. I remember having very much pain in the head during my illness. I also recollect that after leaving the hospital I was a month and could not see to go about. It is now one year since I left the hospital." Ophthalmoscopic examination in 1868, March 10.

The left disc, of which I made a careful water-colour drawing, so far as my lights then allowed, showed a distinct effusion into the apparent outer half with considerable thickening? of the retinal veins (venous stasis).

The right disc showed an anæmic condition not sufficiently pronounced to be atrophic.

General Symptoms. — Very decided nervous prostration, inability for ordinary work, feeble circulation and muscular weakness; very slow contraction of pupils to light, but ordinary contraction for near vision. Three months elapsed after leaving the hospital before ordinary domestic work could be resumed.

Cases I have recorded, and one I will relate, in which the acute meningeal trouble was persistent, and ended not only in loss of visual power but also in the death of the patient. Post-mortem evidence proves beyond doubt that in these cases the inflammatory mischief is distinctly observable in brain tissue proper; and in those cases in which the effusion into the discs has proved rapidly destructive to vision, the optic tracts have presented direct evidence of softening. Undoubtedly the most serious symptom we have to deal with is vomiting, (continued) as a sequela of cerebral neuralgia with increasing loss of muscular power, and impaired powers of speech. Another phase of the disease, not less interesting clinically, is the peculiar change in the general facial expression when the fearful pain (cerebral), which almost hourly is present, has existed for a few weeks. I will not say positively, but I do

believe that I could, from such facial alteration—were I informed that it correlated with intense pain intra-cranial—diagnose a past typhoid in which meningeal trouble had been a prominent symptom. I have seen a fine healthy-looking woman with disc trouble, six weeks after convalescence from typhoid fever (meningeal), become in less than a fortnight after my first examination quite ensanguine, with drooping eye-lids, glassy corneæ, and irresolute gait.

The first class of patients, whose brain tissue proper is suffering, present symptoms in many particulars like those of slowly advancing tubercular disease within the skull, and in the few cases I have watched throughout, so intense is their suffering and so far removed from ordinary medical aid, that the welcome end is a boon hourly desired.

Nothing can exceed the agony produced by the incessant vomiting; a condition over which nothing I have ever seen administered had any control.

In the cases in which cerebral neuralgia is intense, and does not pass into the fatal stage of sickness, nothing is more remarkable than the objective symptoms, which so resemble "Bright's disease," that medical men and students not familiar with these cases insist that the kidneys are at fault. I make the same reply always to these inquiries: "Please, test the patient's urine yourself, afterwards examine with your ophthalmoscope the fundus of each eye." The first examination is always satisfactory; the second not always so, for few are so well versed in the use of the ophthalmoscope as to be able to dissect symptoms at once peculiar and embarrassing. In speaking of disease of the kidneys, I must qualify my statement by saying I have already mentioned an isolated case in which albumen was detected in the urine of a patient suffering from the acute stage of typhoid fever. Here the eye troubles had early shown themselves, and advanced as the delirium became more intense. I have heard it stated on good authority that the urine of typhoid patients in the acute stage is seldom or never free from a trace of albumen, which rapidly disappears during convalescence. The earliest case I find recorded in my note book is one so instructive that I must say a few passing words concerning it. Unfortunately I had then no clinical knowledge

on the subject, and was for a length of time dubious as to the cause and general character of the disease (intra-ocular). During my early days with the ophthalmoscope, a working man asked my opinion concerning a rapid failure of sight. The following short history was all I could at first glean. For six months he had been continually oppressed with a severe and almost unbearable headache, which he described somewhat graphically as "a burden no man could positively bear for a lengthened period without a serious ending;" and so it proved for the poor fellow, for only last evening I saw him perambulating the streets, led by his little daughter, and playing some musical instrument to gain a livelihood. Now, as I had, when I first saw the man, no knowledge of disc mischief after typhoid, I never inquired into that side of the question, and it was only when another case brought the whole thing prominently before me, that closer questioning elicited the fact that typhoid (meningeal) was the real cause. Another case, not less interesting, now stands prominently before me as one in which a mistaken diagnosis was made, and no wonder, for the ophthalmoscope was then in its infancy. I have to-day seen an out-patient, who gives the following history:—"Two years ago I was confined to my bed for six weeks with slow fever (typhoid). I lost consciousness, and was not myself for many days; when I came to myself all objects appeared misty and indistinct (optic neuritis?), gradually my eyesight improved, but has never been good since. Latterly I have suffered from very intense headache, and my sight is again misty." The patient who gives this history is a girl, æt. 18, a mill-hand, who was engaged in weaving. Work is now no longer possible; for on the one hand vision is more or less imperfect, and on the other hand, so soon as she stoops to her work, the cerebral neuralgia, which even during repose is severe, becomes so aggravated as to cause her to at once cease working. The right eye is markedly different from the left: careful examination of the fundus of the right eye discloses a condition not typical of any preceding neuritis. The contour of the disc is normal, the choroidal ring is sharply defined, and the only abnormal condition is a more than usual pallor of the apparent inner third of the disc, with a fulness

and tortuosity of the retinal veins sufficiently pronounced to take cognisance of. Other than these departures from health the fundus was normal. The left eye, examined very carefully, showed a marked irregularity of the edges of the disc, a condition so well known as a sequela of neuritis; the retinal veins were normal, but the arteries were of diminished calibre; with the exception named, the eye was healthy. In this case, although the ophthalmoscope showed us no actively progressive disease, yet vision was much impaired, and the use of the eyes for close work was followed by intense intra-cranial neuralgia: a neuralgia which in degree is ever present. As all know who use the ophthalmoscope how frequently we meet with small hæmorrhages after neuritis; but none ever accompany—so far as my investigations teach me—the disease I am describing. In the clinical history of these cases we must guard against mistake. The cerebral neuralgia of typhoid is in many respects not unlike the neuralgia of syphilis; and the intra-ocular disease may be due to specific mischief, and not to the typhoid poisoning. The evidence on the subject is often so conflicting as to place such cases out of the category of truthful clinical history. It not unfrequently happens that a contracted syphilis shows itself simultaneously with a typhoid fever; and, although there may undoubtedly have been meningeal complications purely typhoid, yet the intra-ocular troubles may be due rather to the poison of syphilis than to the poison of typhoid. I have always carefully avoided adding such cases to my notes of typhoid disc mischief, however (apparently) clear the clinical history may be.

Although I have much yet to say on the subject, the space allotted to me renders it necessary that I should defer further remarks on the subject for a future number.

I had, on my last out-patient day, June 3rd, an opportunity of demonstrating to the students present typical examples of the disease. In each case the age of the patient was seventeen years, and both were females: each of the four discs presented the peculiar brown paint-like patch I have already described, and the history of cerebral neuralgia (in one case associated with vomiting) was very well related by each girl.

PRACTICAL OBSERVATIONS ON ELECTRO-DIAGNOSIS IN PARALYSIS.

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It is generally recognised that success in the art of diagnosis in a great measure bears a relation to our powers of applying physical agents in the investigation of disease. Hitherto, affections of the nervous system have baffled our efforts in this direction, but in comparatively recent times this has in part been overcome. Neurologists now admit that electricity, a powerful dynamic and chemical force, may with advantage be employed for this purpose. Like other novelties with which the profession at large is not familiar, electro-diagnosis is by the majority of practitioners either completely ignored, or denounced as useless; and there can be little doubt that the manipulative dexterity, the labour, and the technical knowledge necessary for its successful performance, have rendered it up to the present unpopular, and have chiefly confined its use to the hands of the specialist. As a matter of fact, the whole procedure is sufficiently simple to warrant its more universal acceptance, and it can scarcely be denied that the advantages to be gained therefrom are various and important. Doubtless, as information on the subject becomes more extended, as the instruments at our disposal are more perfect, and the necessity for accurate investigation of nervous disorders is more urgent, this method of inquiry will, along with others, form part of the ordinary education of the medical man.

It must, however, be noted, that like all other methods of physical research, electricity is not to be depended upon alone as the sole means by which we are to arrive at a just con-

clusion in investigating the nature of diseases of the nervous system. It is only one of the aids we employ, and which, in conjunction with other facts and observations, is a most powerful auxiliary; but we must not expect it by itself to act as a mysterious power, which will give us every information without an inquiry into all the other circumstances of the case. A crepitation heard over the chest by the stethoscope does not by itself diagnose any particular disease, but only demonstrates a physical condition which, in conjunction with other symptoms and signs, enables us with great accuracy to arrive at the anatomical state of the lung. In the same way electrical tests do not specify any form of paralysis, but only indicate certain organic states from which we may infer the morbid lesion. It is from a general knowledge of the facts displayed, and by their intelligent application in practice, that useful deductions as to pathological conditions are to be drawn. While insisting upon this principle, and disclaiming against the formation of two exalted notions as to the exclusive potency of the use of electricity in diagnosis, there appear to be certain general laws, which experience has shown to be useful for our guidance.

Having for some years been interested in this subject, and having had ample opportunity of testing its practical value, I propose to enumerate such broad rules for the employment of this agent in the diagnosis of the different forms of paralysis, as appear warranted from a general survey of the question, and to illustrate the successful adoption of these by a few selected examples which have recently come under my own observation.

In a given paralysis, if the electrical reactions are perfectly normal to the affected nerves and muscles, we may conclude, subject to certain limitations, that it originates in an affection either of the brain, or of the white columns of the cord. This distinguishes it from paralysis from degeneration of the grey matter of the cord, or of the peripheral nerves. Paralysis from cerebral disease is usually hemiplegic, and from a spinal lesion paraplegic in character, which constitutes a general distinction between the two. In irregular forms of loss of voluntary motion, such as in cross paralysis, or in paresis of one limb,

in either case dependent on disease of the brain or of the white columns of the cord, the difference is not to be made out by electrical tests alone, as in both cases these would be normal. Hence, such would have to be determined by the general symptoms, a matter often of great difficulty. Fortunately, paralysis of a single limb from affections of the columns of the cord is extremely rare.

When the responses to electricity are increased, it indicates hyper-excitability of the nervous system, and when in addition reflex muscular contractions are produced in various parts of the body by stimulation of a nerve in the paralysed part, it is further evidence of great irritability of the spinal cord, almost, if not quite, amounting to organic disease. Increased electrical responses may also be due to alteration in the resisting media of the affected limb. On this subject we have little positive information; but it is doubtful whether the modifications thus induced are of great practical import.

Abnormal electrical reactions, especially if combined with qualitative changes, are evidence of disease, either of the grey matter of the cord, or of the peripheral nerves. If these occur in the form of complete paraplegia, the certainty is that the cord is at fault; but if confined to the branches and muscles of a single nerve-trunk, the probability is that the lesion is one of peripheral origin. If one extremity or portion of an extremity is affected, the diagnosis is more difficult. In such a case the *distribution* of the paralysis is of great importance. When a limb has lost its power of motion, as a result of disease of the cord, the abnormal electrical responses may exist in one of three ways: (*a*) They may be uniformly distributed throughout the entire paralysed member, all the muscles being equally affected. This takes place in gross lesions involving a mass of the structure of the cord. (*b*) They may be distributed only to certain muscles forming *physiological* groups, irrespective of their nervous supply. For example, all the flexors of a limb, or its extensors alone, may present abnormal reactions, although they receive their nutritive influences from different sources. This occurs in chronic affections of the multipolar cells of the anterior cornua; and (*c*) Their distribution may be perfectly irregular, affecting the

muscles neither in anatomical nor physiological groups. This often follows acute inflammations of the grey matter.

On the other hand, in peripheral paralysis we never find either the entire limb, or physiological or irregular groups of muscles, presenting signs of degeneration; but these are distributed according to *anatomical* relations, in other words, to those structures only which receive their nervous supply from a special nerve-trunk, totally irrespective of their function.

Hence, in limited paralyses the great distinction between those originating in the cord, or in a peripheral nerve lesion, is that in the former the limbs are uniformly affected, or their muscles are attached in physiological or in irregular groups; while in the latter they are affected according to their anatomical distribution.

Electrical phenomena also enable us to distinguish between the different varieties of paralysis arising from disease of the grey matter of the cord. If these when abnormal are uniform and extend over an entire limb, we may conclude that the lesion is gross, i.e., occupying a mass of its substance as in myelitis. If they are confined to certain physiological groups of muscles, the disease has generally been chronic, and implicates the multipolar cells of the anterior cornua, as in progressive muscular atrophy. And finally, if the degenerated muscles are attacked in an irregular manner, neither according to distribution nor function, the disease has usually been the result of an acute inflammation of the anterior cornua, which has destroyed some of the nutritive centres, and left others intact, as in poliomyelitis anterior acuta.

Again, the electrical reactions may demonstrate degeneration of both nerve and muscle, which, if acute, is evidence of infantile paralysis, and if chronic, of disease of the grey matter, *en masse*, as in myelitis. They may also indicate disease of muscle with healthy nerve, which occurs in chronic lesions attacking the nutritive centres of the muscle only, as in bulbar paralysis. If nerve is found deficient in response, and muscle normal, it shows alteration of the former, the latter remaining intact, as is sometimes seen in the early stages of infantile paralysis.

In cases of limited disease of the grey matter, when the

lesion is confined to one or more foci, as in the so-called mixed or indiscriminate forms of paralysis, the distribution of the abnormal electrical reactions gives an index to the amount, extent, and position of the cord degeneration.

Such general considerations concerning limited electrical abnormalities, as a result of disease of the grey matter of the cord, will usually enable us to distinguish them from one another, or from lesions of the peripheral nerves.

In peripheral paralysis the electrical conditions indicate with exactitude the extent and distribution of the disease. Paralysis with normal reactions is evidence of a very slight and temporary form, the prognosis of which is favourable, sufficient changes having occurred to modify the transmission of voluntary impulses, but not to affect the nutrition of either nerve or muscle. Loss of response to the nerve-trunk with either current points to nerve alteration, and that in proportion to its diminution of action. Loss of response with faradism to the muscle, indicates changes in the intra-muscular nerves without necessary alteration in the fibres. Loss of response with galvanism to the muscle, especially when combined with qualitative alterations, shows modification or destruction of the muscular tissue, and this in proportion to the physical changes induced.

Cases occasionally come under notice in which there is little or no paralysis, and where there are no objective symptoms. In these, early degenerations may sometimes be detected by electrical tests, before there are any other definite symptoms, as will be seen in several of the cases to be subsequently detailed. Even when there are suspicions of disease it may not be readily detected, and the existence of electrical change is a physical fact, which, if demonstrated, establishes an otherwise doubtful diagnosis. Before sufficiently marked alterations have ensued to enable us accurately to measure and compute their locality or distribution, this may frequently be done with considerable accuracy by the same method.

Thus far, I believe, we may generalise with safety on the subject of electricity in diagnosis. To lay down more strict definitions would be too arbitrary. Doubtless, instances will occasionally arise when all our powers of investigation combined fail to determine the true nature of the lesion; we

can only hope that, as our means of research improve, these mysteries will in the future be solved; and that the art of electro-diagnosis, although as yet in its infancy, may by further development constitute one of the means to this end.

In practical illustration of the preceding principles, as well as of the general utility of electricity as an agent in physical diagnosis, numerous examples might be given, which space compels us to suppress. In order, however, to indicate the method of investigation employed, the mode of drawing inferences from the facts elicited, and the actual substantial advantages gained through the procedure, attention is directed to the following selected cases. These are recorded with great brevity, such details only being given as serve to establish the diagnosis.

1. *Electrical reactions in a doubtful case of Monoplegia, suggesting its Cerebral Origin.* W. G., æt. 55, male, sailor. The patient states, that four years ago, without obvious cause, he experienced weakness of his right arm. This began very gradually, and has continued to increase ever since. His general health has been good, there have been no other symptoms, and the other limbs have been normal.

At present the patient is a healthy intelligent man. All the special senses are normal, and, with the exception of the right arm, all the other parts of the body are healthy. This is very weak, the movements can be performed, but feebly and imperfectly; the grasp of the hand is very deficient, and he cannot put his hand behind his head. There is no atrophy, rigidity, or alteration of sensation.

Electrical reactions. — With the exception of very slight comparative quantitative diminution of response in the muscles of the right arm, the electrical reactions are perfectly normal.

The fact of finding in so chronic an affection normal electrical reactions of the paralysed limb, suggested a cerebral lesion. For peripheral paralysis and disease of the grey matter of the cord being thus disproved, it was very unlikely that any morbid condition of the lateral columns would have remained limited for so long a period. The diagnosis arrived at was subsequently proved to be correct by post-mortem examination.

2. *Electrical reactions in a doubtful case of Monoplegia, indicating it to be of Spinal Origin.*—T. C., æt. 44, male, water-colour painter. The patient says that for the last three months he has suffered from pains and weakness in his left upper extremity, otherwise he has been healthy; and he can assign no cause for his present complaint.

At present the patient appears a healthy man, and complains of nothing but weakness and wasting of his left arm. The movements can be performed, but are feeble; he has much difficulty in raising the hand on a level with his head; flexion of the arm can be readily overcome; and the grasp of the hand is extremely weak. The left deltoid is markedly atrophied. The upper arm is smaller than that of the other side, and the muscles are soft and flabby. The forearm and hand of the left side are thinner than those on the right, but not to a great extent. The right upper extremity appears normal. Sensibility everywhere intact.

Electrical reactions.—Both currents applied to all the nerve-trunks on both sides give equal and healthy reactions. Galvanism to all the muscles of the afflicted limb causes in varied degrees vigorous contractions. In most of them the A C C is increased, and in some A C C = C C C. On the right, or apparently healthy limb, the muscles of the hand and extensors of the forearm also show qualitative changes with galvanism, the A C C = C C C.

The point to be decided in this case was whether the paresis of the arm was of cerebral, spinal, or peripheral origin. The nerves being healthy precluded the last, the muscles being diseased showed it was not the first, and the discovery of the affection having extended to the other and apparently healthy limb demonstrated a spinal lesion.

3. *Electrical reactions in a doubtful case of Monoplegia, indicating it to be of Peripheral Origin.*—W. C., æt. 41, male, brass-finisher. The patient states that having been previously in good health, a month ago he fell and hurt his back. This was followed by shooting pains down the right arm, weakness of the limb, and numbness in the fingers. These symptoms have continued since.

At present the patient complains of a dull aching in the

right shoulder and upper arm. The movement of the right wrist-joint and fingers are weak, but not lost; the grasp of the hand is feeble. Numbness is experienced in the fingers, especially the two inner ones. There is no apparent wasting of the muscles. No actual loss of sensibility.

Electrical reactions.—Faradism applied to the trunk of the right ulnar nerve indicates marked diminution, but not complete loss of response, as compared to that of the opposite side. The other nerve trunks are healthy. Applied to the flexor carpi ulnaris, the abductor and the flexor brevis, minimi digiti, the faradic current shows marked diminution of response as compared with the same structures on the other side and the other muscles of the same arm.

In this case of paresis, the fact of finding the abnormal electrical reactions confined to one nerve and to the muscles supplied by it, the remainder of the body being intact, showed the lesion to be a local one, and limited to the nerve in question.

4. *Electrical reactions in a doubtful case of Monoplegia, supposed to be due to injury, but proved to be the result of Progressive Muscular Atrophy.* — A. B., æt. 60, male, farm labourer. The patient states that two months ago he was in good health, when he accidentally fell upon a pitchfork, a prong of which passed through the fleshy part of his upper arm near the axilla. He remained under the care of a local surgeon for some weeks, during which time he kept his arm in a sling, and after which the wound gradually healed. After this had taken place the arm and hand remained weak and got thinner, so that he came to London for advice.

On examination the general health was found good. There was considerable paresis of the left upper extremity from the shoulder downwards. The muscles were somewhat thin and flabby, and those of the hand were distinctly atrophied. There was no loss of sensibility. The right arm and remainder of the body appeared normal.

Electrical reactions.—With both currents, the nerve-trunks of the left upper extremity were normal, and the same as on the other side. Galvanism applied direct to the muscles of the left shoulder and arm caused vigorous contractions, if anything slightly more marked on the paralysed side and

A C C = C C C. The same reactions were discovered in the muscles of the apparently healthy right hand, and here also the A C C was considerably increased.

Several physicians and surgeons, whom this man consulted, came somewhat naturally to the conclusion that the accident had in some way injured the nerves of the brachial plexus, and had, in consequence, induced the paresis and wasting of the left limb. In short, they considered the case to be one of traumatic peripheral paralysis. Electrical investigation, however, demonstrated that the nerve-trunks were perfectly healthy; it showed marked alteration of the muscular structure of the affected extremity; and, finally, it indicated, in what was apparently a healthy limb, that this was undergoing incipient disease, as yet too slight to interfere with function. The result arrived at from the investigation was that the patient was suffering from chronic progressive muscular atrophy in both upper extremities, that the accident had attracted attention to his left arm, in which the disease was furthest advanced, and that he attributed the loss of function to what was in reality an accidental complication of an already existing degeneration. This diagnosis was subsequently proved to be correct by the after progress of the case.

5. *Electrical reactions in a case of Double Facial Paralysis, of Cerebral Origin on one side, and of Peripheral on the other, occurring in the same individual, and showing the distinctive differences of response.* — J. G., æt. 60, male, mason. This patient had a variety of ailments, including diabetes, albuminaria, &c. For the present purpose, it is sufficient to note that about a year ago he received a blow on the left side of his head, which resulted in necrosis of the bone, and which has only recently healed. About a month ago he was suddenly seized with an attack of giddiness, which was followed by loss of power on the *right* side, including the face. This has continued ever since. Prior to this he had suffered from pain in the left ear, and to this succeeded immobility on the *left* side of the face.

On examination the patient presented all the usual symptoms of paralysis of *both* sides of the face, most marked on the left, on which he was unable to close the eye, which he could

do on the right. The whole visage was remarkably immobile, there were slight voluntary movements on the right side, but none on the left.

Electrical reactions.—These were apparently normal, on the right side, to both nerve and muscle with both currents, there being no qualitative changes with galvanism. On the left side the strongest faradic and galvanic currents applied to the facial nerve-trunks produced no response whatever. The former, applied directly to the muscles, was also followed by negative results, but the latter caused markedly stronger contractions than on the other side; these were slow and prolonged, and the A C C > C C C.

This otherwise interesting and complicated case, here given with great brevity, seems to indicate that the paralysis on one side was of cerebral, and on the other of peripheral, origin. the reactions in the former were normal, and in the latter presented a typical specimen of the "reaction of degeneration." The diagnosis made was, that owing to the blow on the left side of the head an abscess or growth had there developed; this by direct pressure causing paralysis of the left facial nerve, and by indirect interference of the voluntary tracts inducing palsy of the right side of the body, including the face.

6. *Electrical reactions in a case of extreme irritability of the Spinal Cord, showing abnormal reflex responses.*—A. B., æt. 20, female, milliner. The patient, of a delicate and nervous temperament, had a year ago suffered for several months from an attack of paralysis of the lower limbs, similar to the present one, from which she completely recovered. About two months ago, without apparent cause, she again suddenly lost the power in her limbs, and this has continued since. At present the patient is in delicate general health, and of a highly nervous and hysterical habit. She has almost completely lost the power of her lower extremities, so that she cannot stand. The arms also are weak and tremulous, and the hands are affected with choreic-like movements. The sensibility is everywhere much diminished, the reflexes highly exaggerated, but the limbs are well nourished. The whole aspect of the case indicated what is usually termed spinal irritation with hysterical paralysis.

Electrical reactions.—Either current, when applied to any portion of the lower extremities, was followed by greatly exaggerated reactions. Apparently these were normal in kind, if excessive in degree. Not only did muscular contraction ensue at the part directly irritated, but it was observed in the neighbouring muscles, and also in those of the opposite limb. A moderate faradic current, for example, applied to the peroneal nerve, caused violent clonic spasm of all the muscles of both lower extremities. This seemed to indicate extreme hyper-excitability of the entire nervous system, and more especially of the grey matter of the cord.

7. *Electrical reactions in a case of supposed Progressive Muscular Atrophy proving the disease to be simple emaciation.*—E. F., æt. 25, male, labourer. The patient states that he was in his usual health till about four months ago. Then he began to suffer from pains and weakness in his limbs. He was unable to work, and consequently to get attention and good food. Two months ago he noticed wasting of the left shoulder and arm, and recently his entire body has become very thin.

On examination the patient is found very much emaciated, weak, and generally in bad health. All the organs are healthy. He complains of occasional pain in the joints, especially the left shoulder, which is stiff. There is nowhere any actual paralysis, but all his movements are very feeble. His gait is unsteady and uncertain. He cannot raise the left hand to his head, and the grasp of both hands are very weak. The muscles throughout the body are small, soft, and flabby, and more especially the extensors of the forearms and the hands, which are very thin.

Electrical reactions.—Both currents to nerve-trunks and muscles everywhere cause normal reactions.

This patient was sent into the hospital as a case of progressive muscular atrophy, and at first sight might easily have been mistaken for that disease. The fact of the muscles being found healthy under electrical examination, in conjunction with other circumstances in the case, suggested that the patient was suffering from extreme simple muscular emaciation, following chronic rheumatism, neglect, and insufficient food. This diagnosis was subsequently found to be correct, and after a

residence of a few weeks in the hospital the patient completely recovered.

8. *Electrical reactions in a case of supposed simple emaciation, the wasting proved to be due to Progressive Muscular Atrophy.*—E. B., æt. 35, male, labourer. The patient states that he was a healthy man till six months ago, when, without apparent cause, he suffered from pains in the head and constant vomiting. This continuing, he entered a hospital, where he remained several months, and where he became greatly emaciated. Finally he recovered from the urgent symptoms. A few weeks after the vomiting returned, and since then he has been unable to retain any food on his stomach. The only explanation offered for this train of symptoms is that the patient was a hard drinker.

At present the patient is a pale, thin, unhealthy-looking man. Now there is no sickness, and the patient takes food fairly well. All the organs and special senses are healthy. The movements of the limbs generally are feeble, but there is nowhere any special paralysis. The movements of the arms and hands are slow, awkward, and weak, otherwise normal. The gait is tottering, shaky, and tremulous, but without other especial peculiarity. The body generally is spare, but no muscles appear specially atrophied. The left arm is slightly smaller than the right, and its muscles are softer and more flabby. The reflexes are somewhat increased. Sensibility is everywhere normal.

Electrical reactions.—Both currents applied to all the nerve-trunks are followed by normal reactions. Faradism direct to the muscles also causes healthy contractions. With galvanism the muscles of both arms are found qualitatively altered. On the left side, with a moderate current, the contractions are everywhere vigorous. In the triceps the A C C greatly exceeds the C C C. In the biceps the A C C=C C C. In the supinator longus C C C>A C C, but the latter is increased. In the extensors carpi radialis longior and brevior A C C>C C C. In the extensor communis digitorum C C C>A C C, but the latter is increased. In the extensor carpi ulnaris and extensor longus pollicis the reactions are normal, as are also all the flexors of the forearm. In the thenar group of muscles the

A C C is increased, and in the hypothenar the reactions are normal. In the right arm all the muscles are healthy except the extensor carpi radialis longior, in which the responses are much more vigorous than in health, and the A C C = C C C. Also in the thenar regions the A C C > C C C.

This case is exactly the reverse of the last one, and contrasts strongly with it. The patient having previously been in good health, suffered for many months from vomiting and inability to retain his food. In consequence he became weak and emaciated. He was accordingly admitted as a case of alcoholic dyspepsia and emaciation. There was no special wasting of any individual muscles, no actual paralysis, or anything likely to suggest a more serious disorder. Electrical investigation revealed the phenomena detailed shortly above, indicating muscular degeneration of both arms. This led to the diagnosis of incipient progressive muscular atrophy, which has been so far justified by the progress of the case, that although the patient remained a considerable time in the hospital under the most favourable circumstances, his malady continued to progress.

9. *Electrical reactions found abnormal in the muscles of a house painter, before there were any other objective symptoms of disease.*—A. B., æt. 40, male, house painter. The patient states that he has been a worker in white-lead for twenty years. He has never been very robust, but has been otherwise in good health. A week ago he was seized with symptoms of dyspepsia, pains in his bowels, and general weakness, which have continued since.

At present the patient is pale and sallow-looking. He is in his usual health, except that he complains of vague pains in the abdomen, occurring paroxysmally; he is bilious, and feels generally unwell. There are no objective symptoms, no blue line on the gums, no trace of weakness or wasting of the arms.

Electrical reactions.—To the nerve-trunks with both currents these are normal. The reactions of faradism to the extensors of the hand and fingers of both sides are much diminished, and require a very much stronger current than the other muscles, which are normal. To these affected muscles the galvanic current acts with greater vigour than to the flexors, and

the A C C = C C C. All the other parts of the body are normal.

In this case the man applied as an out-door patient at the hospital, having suffered for a few days only from indefinite dyspeptic symptoms. His occupation suggested lead-poisoning, but there were absolutely no objective symptoms to permit such a diagnosis being arrived at. The electrical reactions indicated commencing changes in the extensor muscles of the wrist and fingers, although there was no apparent loss of function. This was strong evidence in favour of saturnine infection. The patient subsequently entered the hospital with well-marked wrist-drop.

10. *Electrical reactions in a case of supposed Hysterical Paralysis, proving to be Chronic Poliomyelitis Anterior.* M. S., æt. 21, female.—The patient has been in good general health, although with a tendency to nervous and hysterical attacks. Seven months ago she suddenly became blind of the right eye, but from this in a few weeks she completely recovered. Shortly after this she noticed, for the first time, weakness in the left leg, and this has remained and increased up to the present time. For some weeks past in addition she has experienced a sensation of numbness in the left hand, but without apparent paresis.

At present the patient is a perfectly healthy-looking person. All the functions and organs are healthy. She can walk, but is very lame with the left leg, and the toes drag along the ground. When the limb is raised, the extension of the foot and toes is very weak and imperfect. The movements of the knee and other joints appear normal. There is no marked atrophy, and the remainder of the body is healthy. The sensibility is normal.

Electrical reactions.—Faradism applied to the left external popliteal nerve shows very great diminution of response as compared to the other side, and requires a much stronger current to produce muscular contractions. The other nerve-trunks are normal. Direct to the muscles of the left leg there is slight comparative diminution to all, but to the extensors of the foot and toes the contractability is almost entirely abolished to the strongest current. Galvanism to the external

popliteal nerve also shows great diminution of response, and to the extensor muscles of the foot and toes there is also considerable diminution of reaction, the A C C = C C C, and the contractions are slow and tonic. The other muscles of the leg, as well as those of the other limb, are normal. On testing the arms all the nerves and muscles are found healthy, except the extensor carpi ulnaris, in which the excitability to faradism is almost totally abolished, and that to galvanism is increased, the A C C = C C C.

This young lady had been under the care of several medical men, who unanimously pronounced her disease to be hysteria, and she was treated accordingly. The above investigations seem to have established the fact, that whatever the term hysterical may mean, this was a case of chronic organic disease, and probably of the anterior cornua of the cord, as we find degeneration of both nerve and muscle. A year afterwards the disease had considerably advanced.

11. *Electrical reactions in a case of supposed feigned Paralysis, proving to be Poliomyelitis Anterior Acuta.*—M. A., æt. 31, female. The patient was perfectly well eighteen days ago. Without apparent cause she awoke one morning feeling generally unwell and weak, accompanied with pains in the back, and feverish symptoms. This so increased that, in two days, her legs were almost completely paralysed. After the first day or two the symptoms of malaise passed off, and the general health has since been good.

At present the patient is a perfectly healthy-looking person. Her intelligence is of an unusually high order, she has perfect calmness and control over her emotions, and has no trace of a nervous or hysterical habit. The entire body is normal, except the lower extremities, which are almost completely paralysed. The only movement which there exists is slight flexion of the left ankle and toes. The limbs are limp, and the muscles soft and flabby. There is the faintest trace of plantar reflex in the left foot, but none on the right. The knee jerk phenomenon is absent on both sides. There is no apparent wasting. Sensibility is everywhere normal.

Electrical reactions.—Both currents applied to the nerve-trunks of the right leg fail to elicit any response whatever.

To the left external popliteal nerve a very strong current causes slight extension of the toes. Powerful faradism applied direct to the muscles of both legs is followed by negative results. Galvanism causes vigorous contractions of all the muscles, A C C > C C C in some, and in others A C C = C C C, and the responses are slow and tonic. The intensity of the abnormal reactions are somewhat less marked on the left side.

This young lady was supposed, owing to certain circumstances, to be wilfully shamming. Her friends, relations, and even doctors, treated her with such severity as to cause her great distress. She was banished from her home and sent among strangers, on the ground of finding elsewhere a stricter moral supervision. The electrical reactions demonstrated the existence of serious organic disease of the cord. The diagnosis made at the time was acute poliomyelitis, and the prognosis advanced was unfavourable. The writer, as well as his opinion, was at the time treated with scorn, and the word "quack" was overheard being muttered by some of his medical brethren. At this moment, two years afterwards, the patient cannot stand, the limbs have undergone extensive atrophy, and the entire case has assumed an aspect which does not leave the slightest doubt as to the serious nature of the lesion.

Electrical Reactions in a case of Supposed Bulbar Paralysis, proving it to be one of Peripheral Paralysis.—A. S., æt. 54, female, housewife. The patient states that she was quite well till six months ago. Then she began to suffer from general malaise and muscular weakness. This was accompanied with hesitation in her speech, and dribbling of saliva from the mouth. These symptoms have continued to increase very gradually till the present time.

On examination, the patient is a delicate, nervous woman. She complains of great general debility, so much so that she is unable to perform her ordinary household duties. There is, however, no actual paralysis of the limbs. The special senses are normal, and the intelligence appears intact. At rest the face is not observably altered, except that it is expressionless, and the lower lip hangs downwards, leaving the mouth open. On movement, the under parts, especially about the lips and chin, are distinctly defective in action, and are

drawn slightly to the left side. Food collects in the cheeks, and the saliva frequently dribbles from the corners of the mouth. There is also some difficulty of mastication. The speech is slightly thick and mumbling. The movements of the tongue are deficient, and it is pushed over to the left side. There is no difficulty in swallowing. There is an apparent wasting of the face. The left side of the tongue is distinctly atrophied, it is twisted, puckered, and covered with deep furrows. The right side appears normal.

Electrical reactions.—Faradism to the nerve trunks of the face indicates diminution but not total loss of response, especially on the right side. Applied directly to the facial muscles, a very strong current is required to cause contractions. The right side of the tongue is also very sluggish in reacting, and on the left there is no response whatever to the induced current. Galvanism to the facial nerve-trunks indicates also deficient action, but when applied to the muscles themselves the responses are vigorous, somewhat prolonged, and ACC=CCC. The continuous current to the right side of the tongue causes apparently normal reactions; on the left side they are comparatively increased, prolonged, and ACC=CCC.

This case had visited many hospitals and seen many physicians, and the general opinion expressed was that the patient suffered from bulbar paralysis. This view I shared before applying electrical tests. Then, however, it was found that these exhibited the reactions usually associated with peripheral nerve paralysis, instead of the muscular degeneration alone, ordinarily met with in bulbar paralysis. But the question arose, how could disease primarily attack the facial and hypoglossal nerve-trunks, on both sides, and apparently these nerves only? A post-mortem examination, however, solved the problem. It was there found that the pons and medulla were healthy, the facial and hypoglossal nerves were atrophied, the former chiefly on the right, and the latter most on the left side. These conditions were the result of pressure from extensive disease of the bones at the base of the skull, involving the foramina through which these nervous trunks passed. It is not asserted that in this case the exact diagnosis was determined or expressed before death. In spite of the

similitude of the symptoms to a central lesion, the peripheral nature of the electrical reactions was recognised, but the symmetrical character of the affection did not seem to be explained. In the face of this doubtful problem, an opinion was postponed, as it was not thought advisable to place these electrical phenomena alone, against all the other probabilities of the case. As the facts, however, turned out, if a verdict of peripheral paralysis on both sides had been pronounced in accordance with the reaction obtained, in spite of its apparent impossibility, it would have been correct.

THE PLEA OF INSANITY IN THE CASE OF CHARLES JULIUS GUITEAU.

BY JOHN CHARLES BUCKNILL, M.D., F.R.S.

BEFORE these pages appear, the fate of this notorious assassin will have become irrevocable, and this reflection enables me with the greater freedom to attempt to extricate an opinion upon his mental state from the torrent of argument which has not ceased to accompany and to follow his trial. Unavoidable and unnecessary interference with the course of justice in any country by the people of other countries is to be deprecated, and recent events teach us that the citizens of the United States stand in especial need of example as to this international obligation from ourselves. With regard to political crimes, this rule of national conduct is of still more importance than with regard to common and vulgar murders like that by Lamson, for citizens of the United States so frequently display self-consciousness with regard to those political institutions upon which they delight to think that all the world is gazing with envious criticism, that they can scarcely be expected to bring themselves into the frame of mind of wishing to do simply that which is right and just, without admixture of feeling as to what the world will think of it.

This feeling may well be illustrated by remarks made at the important discussion on Dr. Hammond's paper upon Guiteau which took place at the Medico-Legal Society of New York on the 1st of last March. Dr. Hammond himself said that "to shut our eyes to his (Guiteau's) exact condition, and to try to flatter ourselves that he was of normally constituted mind when he shot the President, is not only cowardly, but it is impolitic. It will lead to the erroneous conclusion

that there was a sane man, a man in the full possession of his mental faculties, capable of killing the President of the United States for the purpose of uniting the two wings of the Republican party." And that excellent surgeon, Dr. Sayre, tried to bandage up the intelligence of his hearers by the still more emphatic assertion that "It would be better that Guiteau should be shown a lunatic than that the idea prevail that any one other than a maniac would dare to shoot the head of the nation in this free and enlightened Republic."

If this argument is admissible, the execution of Booth for the assassination of President Lincoln was a mistake; and if Guiteau be respited, as was demanded at this discussion, to show that "a Republic can do what a Monarchy did when Lord Erskine defended Hatfield," then, indeed, the lives of Presidents will be insecure, and the District Attorney will have good ground for the expression of his fear that, in the easy accessibility of the President to all comers, the simple forms of the American Government contain an element fatal to its permanency.

But these political aspects of Guiteau's case, while tending to warn foreigners from premature criticism, and marking as impertinent and officious such conduct as the circulation in this country by American citizens of petitions to be signed by Englishmen in favour of Guiteau's respite, are, or ought to be, entirely foreign to the medico-legal consideration of the question as to whether Guiteau was or was not insane, and so insane as to be irresponsible.

And this is not a question whether or not the criminal is different from other men, but whether or not he is different from them in a certain manner, and from a certain cause. Every man who breaks the laws must necessarily be different from the men who make and obey them, and therefore Mr. Corkhill well said in his opening speech, "Crime is never natural. The man who attempts to violate the laws of God and society goes counter to the ordinary course of human action. He is a world to himself. He is against society, against organisation, and of necessity his action can never be measured by the rules governing men in the everyday transactions of life."

But this consideration appears to have been very much lost sight of by the many American physicians and lawyers who have written pamphlets and made speeches, apparently with the view of exonerating Guiteau because he was not like other men, and especially other American men who could not possibly entertain the idea of "daring to shoot the head of the nation in this free and enlightened republic;" or because, as Dr. Folsom remarks, "he had lost those faculties of mind which come latest in the evolution of a high state of civilisation, namely, a nice sense of right and wrong, and a free recognition of what is due to others than one's self." He had lived among the Perfectionists without becoming perfect, and it may even be admitted "that he was not normal," whatever that may mean when applied to a man. But was he sane or insane; and if insane, was he responsible or irresponsible? This issue, which was formally tried at Washington, is clearly distinct from the one which has since been informally tried in pamphlets and discussions at New York, Boston, and elsewhere, upon the assumption that any man who is not absolutely sane, and therefore theoretically insane, ought never to be punished. What? Will you advocate or approve the execution of an insane man? seems almost always to have been the *quæstio subaudita*. And the right answer would be—that must depend upon how you define or describe Insanity. Allow me to define or describe its conditions with reference to responsibility, and I certainly would never consent to the execution of any insane offender; but I may very possibly dispute or deny the exonerating power of insanity according to your definition of it. One of Guiteau's medical witnesses did indeed unconsciously expose the argument of irresponsible lunacy to the *reductio ad absurdum* by his estimate of the proportion of his fellow-citizens whom he considered to be insane. I forget the exact fraction, but it was something so little below a moiety of the nation, that if correct, the majority ought to be extremely anxious as to what the insane minority would do with them if they were able to combine and prevail. An exception, however, to this line of thought was afforded by Dr. Hammond's paper above alluded to, and in which he endeavoured to show that Guiteau suffered from the Mania without Delirium of Pinel; strongly, however,

expressing the opinion that the condemned man ought to suffer "the full legal penalty for his crime, and be executed with the distinct understanding that he is a lunatic deserving of punishment." Dr. Hammond argued that "there is no necessary connection between medical insanity and legal insanity;" and if by this he means that medical insanity includes far more than legal insanity, I entirely agree with him, for, strictly speaking, every deviation from the standard of mental health produced by disease is a state of medical insanity. That is to say, it is a symptom of disease which the physician may be called upon to give advice about, or to treat by appropriate remedies, but which might afford no justification for any legal proceedings whatsoever. And this proposition, which is tenable and reasonable, is the opposite of the one which was maintained by Guiteau himself, namely, that he was legally insane, but not medically insane, or rather, as he put it, "I believe that I was insane in law, but not in fact." But insanity in fact includes insanity in law, unless insanity in law is a fiction, which no one can suppose it to be. It may be right or wrong for the lawyers to draw the line through the field of insanity where they have drawn it, and to enact that all on one side thereof shall exonerate a man from responsibility, while on the other side thereof a man shall be liable to punishment. But it would seem that for the practical purposes of the rough justice with which mankind must be satisfied, it is necessary a line must be drawn somewhere, for it is impossible to exonerate from punishment all criminals who deviate from the normal condition of sane and reasonable men. Indeed, if morality is natural, we must admit that no such criminals can exist, since, as a matter of fact, we can find no criminals who are not mentally in disaccord with existing circumstances. Guiteau may be such a criminal, or, as one of his most ingenious defenders has said, he may be "simply an anomaly in the fourth quarter of the nineteenth century, and only a type of an earlier civilisation than ours." That the rules of law should be so elastic and fluctuating as to adapt themselves to all anomalies of character, is impossible, while man's knowledge is so dim and his powers so imperfect that he cannot inflict the same punishment for the same

offences upon apparently healthy criminals without the grossest inequality of suffering. It follows from these considerations that all the discussion which has raged with regard to the punishment of insane offenders has had its origin in the persistent attempt to review and amend legal rules according to medical principles, or, as I have long ago pointed out, from the fallacy of regarding something definite, that is to say, legal insanity, as if it were insanity in general, which is the old fallacy of changing the *argumentum de dicto secundum quid ad dictum simpliciter*.

For there cannot be the shadow of a doubt that *secundum quid*, that is to say according to the law on the matter laid down by the Court, Guiteau was responsible for the assassination of President Garfield, and justly amenable to the capital punishment to which he has been condemned. The most simple statement of the circumstances immediately preceding and attending the crime can leave no doubt upon any reasonable mind that Guiteau did know the nature of the act he committed, and that he knew that it was wrong, and contrary to the law. If he knew this he was legally found guilty and condemned. Having regard to the notoriety of the facts, it seems superfluous to attempt the proof of this limited proposition.

But the wider and more interesting question to medical men may perhaps still be considered unanswered, namely, was Guiteau in any degree or in any manner insane? Could he be said to be insane according to the *dictum simpliciter*? A medical man is essentially a naturalist, and is always anxious to arrange his case in the right class, and to name it correctly. Must Guiteau, therefore, be considered an insane man who was held responsible? One of the American pamphleteers on the subject asserts broadly, "that of all our leading authorities in diseases of the nervous system, not one testified that Guiteau was sane," a statement which can only be considered as having some foundation in truth by a peculiar interpretation of the meaning of the phrase, "leading authorities in diseases of the nervous system," for most undoubtedly a large proportion of the leading authorities upon insanity in America testified with unhesitating directness that Guiteau was sane.

But if Guiteau was not irresponsibly insane, can it be really shown that he was insane in any other degree or manner? Can it be shown that his mind was in any way deranged from the effect of disease, that is to say, in a state of medical insanity? Or failing this, can it be fairly argued that Guiteau suffered from a form of insanity which corresponds to none of the medical types of insanity, and which was not connected with bodily disease, but which was the growth of a lifetime, and the slow development of several influences? Both of these positions seem to have been taken by the defence at Guiteau's trial, and they certainly have both been assumed in articles and pamphlets which have been published since his condemnation. These assumptions or suppositions have been a good deal mixed, but it is desirable that they should be considered as much apart as possible, for if the real presence of medical insanity could be shown, it would not be needful to enter into the more arduous argument as to the existence of social insanity, or insanity of character, or of development, or whatever else the supposed condition may be called. With regard to the first proposition, namely, whether medical insanity, or the insanity of disease, could be established, we are bound to remark upon the fact that the several authorities upon this subject have each attributed some different kind or type of insanity, and that no two of them seem to have been of the same mind upon the subject.

Dr. Hammond[1] considers Guiteau's type or kind of insanity to have been the Mania without Delirium first described by Pinel, which, as he interprets the illustrious Frenchman, "may be continuous, or characterised by the occurrence of periodical accessions. There is no marked change of the functions of the understanding, the perception, the judgment, the imagination, the memory, &c., but perversion of the emotional faculties, and *blind impulsions to the perpetration of acts of violence, or even of sanguinary fury*, without its being possible to recognise the existence of any dominant idea, or any illusion of the imagination to which the acts in question can be ascribed."

But surely this is in no wise a correct description of Guiteau's case, who never displayed the slightest inclination

[1] 'New York Medical Gazette,' March 18, 1882.

to blind impulses to acts of violence or of sanguinary fury, but whose conduct was in a remarkable degree cool, calculating, and foreseeing. Pinel's description, the accuracy of which every experienced alienist must have recognised, applied solely to the cases of *Mania* to which he himself confines it by his designation; and to say that a man who was capable of living in the publicity of hotel society without attracting attention to his conduct is a *maniac*, is a perversion of the term, at least, as it was used by Pinel, and therefore as we are bound to accept his meaning of the term as used by him in his delineations of the types of insanity he had observed. It is true enough that since Pinel's time other observers, or perhaps it would be safer to say writers, have described long-lasting cases without prominent symptoms in which these blind impulses to sanguinary fury have existed without defect of the understanding, and that the attempt has been made to establish such cases as a type of insanity under the designation of homicidal mania. According to the theory of this supposed type of insanity, the blind impulse is to destroy human life without forethought and without purpose, and therefore it does not apply to Guiteau. But it would be obviously unfair to include Dr. Hammond among these speculative pathologists who would certainly not accept the conclusion that a person suffering from such form of disease ought to undergo the penalty legally due to his actions. It seems therefore enough to point out that Pinel's delineation of *manie sans délire* does not apply, as Dr. Hammond supposes, to the case in question. It is curious that while Dr. Hammond, who recognising in Guiteau a distinct form of medical insanity, thinks nevertheless that he ought to suffer the extreme penalty of the law, Dr. Walter Channing and Dr. Charles Folsom, who have more difficulty in deciding upon the distinct form of medical insanity in him, think that he ought to be exempted from the punishment attached to his crime. Dr. Channing[1] says, that "to say how insane Guiteau was, or to what special class of the insane he belongs is extremely difficult." He considers that "he resembles many of the lunatics who possess a mania for writing." He also thinks that "an exhibition in all ways so extraordinary as the

[1] 'The Mental Status of Guiteau.' Cambridge, U.S.

conduct of Guiteau at his trial is not to my knowledge on record; and it is not too much to say that it would be a disgrace to American jurisprudence were it not so explainable on the ground of insanity."

Dr. Channing also thinks that Guiteau had insane delusions, especially the delusion that he, "an insignificant good-for-nothing," was "entitled to one of the most important offices in the gift of the Government;" and another delusion, "that there was a political necessity to destroy the President to secure the country from a civil war." But Dr. Channing thinks: "It was unfortunate that Guiteau's counsel laid such stress on inspiration, as its existence as a delusion could be easily disproved, and thus the most important element of insanity for the defence could be shattered. The real elements of insanity hardly came to the surface, and the prosecution therefore had little to disprove beyond insanity in the father, and inspiration in the prisoner."

There is some likelihood, however, that the more general opinion will be that Guiteau, to say nothing of his counsel, exercised a sounder judgment than this medical advocate when he said, "If the jury believe that I believed, that I had a special inspiration to remove the President, then they must find me insane. I believe that I was insane in law, but not in fact." Clearly the only point on which delusion was debatable was that of the asserted inspiration; while the delusions so called by Dr. Channing are like many common beliefs among ignorant and excited men in countries where the time is out of joint. To stamp them as delusions would shield from legal repression the most dangerous elements of political disturbance.

With regard to Guiteau's conduct in court, "which would be a disgrace to American jurisprudence were it not so explainable on the ground of insanity," the full validity of the excuse can scarcely be expected to be felt in countries where the procedure of American jurisprudence is not admitted to be faultless. Perhaps it may fairly be considered in conjunction with Dr. Sayers' proposal that Guiteau ought to be found a lunatic in order to prove that no sane citizen could take the life of a President of the free and enlightened Republic, as an amusing example of vaunting patriotism. The real explanation,

however, of Guiteau's conduct in court was afforded by Judge Cox in his charge, wherein, quoting the Constitution of the United States, he shows that in all criminal prosecutions the accused has the right "to be confronted with the witnesses against him," and calls to the mind of the jury that the declarations of the prisoner during the trial "could not have been prevented except by resorting to the process of gagging him. Any suggestion that you could be influenced by this lawless babble of the prisoner would have seemed to me simply absurd, and I should have felt I had almost insulted your intelligence if I had warned you not to regard it." The most competent witnesses testified to the opinion, founded upon observation, that Guiteau was playing a part during the trial, by which we suppose they meant that he was endeavouring to impress the jury with the belief that he was insane. If so, he certainly played it badly, for not only did he fail to impress the jury with the belief in his insanity, but he appears to have left the impression upon most of those who have only read the records of the trial, that he was sane during the trial and quite capable of self-control.

The carefully-balanced opinions which Dr. Charles Folsom[1] has expressed with his usual moderation are not more convincing than those already referred to, and would have left any one who might have had to act upon them in a state of complete bewilderment. The crime, he thinks, was the result of criminal motive and insanity in inextricable combination; but the kind of insanity from which the criminal suffered he cannot decide upon. If it were "chronic subacute mania of a recurrent or paroxysmal type, it seems to me that his mental condition at the time of his trial indicated responsibility;" and Dr. Folsom seems to hesitate between this type of chronic subacute mania, and moral affective or impulsive insanity, and lastly between these and a slowly progressing form of general paralysis of the insane, the period of incubation of which he thinks may be the whole previous lifetime. Dr. Folsom also thinks that Guiteau "shot the President under the influence of a delusion," but what this delusion was he does

[1] 'American Law Review,' Feb. 1882; 'Boston Medical and Surgical Journal,' Feb. 16, 1882.

not indicate, although he excludes the delusion of inspiration, as an "afterthought adopted as a means of escape from the gallows, of which he was proved to have made no mention in his early explanations of the murder."

In passing, it may be remarked that there was not one scrap of evidence of any paroxysm of mania or even of ordinary violence, in Guiteau's history, excepting that he once 'raised an axe' against this sister, but without striking at her, and without any words, gestures or circumstances to interpret the action, so that what the action meant is left in utter obscurity. As suggested in these pamphlets, he may at some time or other in his life have had accessions of maniacal excitement, of which there was no observation; but to what tenuity of feebleness is argument reduced when the unrecorded gaps of a lifetime are submitted as probable explanations of its gravest event?

As to the suggestion that Guiteau may have been in the incubation period of general paralysis of the insane, which, according to Morel, may extend during the whole previous lifetime, it would seem that that clear-thinking and precise alienist intended to teach that the whole of a man's life may be such as to lead up to the development of general paralysis; which is certainly true of the incubation period of many types of insanity. But if any signs of general paralysis existed in Guiteau, they must have been observed by some of the numerous medical men by whom he was examined; and the supposition that he might be in that period of the incubation period in which no signs can be recognised, and in which there could been have no effect upon his conduct, is too irrelevant to be seriously considered.

It would seem, therefore, that Dr. Folsom also has failed to adduce valid grounds for the supposition that Guiteau suffered from any one of the forms or types of insanity as they are known to and recognised by medical men. Dr. Folsom does not appear to have made up his mind on the question of Guiteau's punishment, so much were "crime and insanity mixed up in his case." Fortunately the punishment of such men is not decided upon by medical men, and it must be satisfactory to Dr. Folsom as a good citizen that he is able to state that, "as the case stands, he [Guiteau] has impressed the criminal

classes and the country at large as being an unscrupulous, dangerous villain, with a badly-arranged mind, feigning insanity to save his neck. The verdict of the jury has met with almost universal approval." The impression upon the criminal classes is most important, for the great end of punishment is to impress their minds; and it is also well that the public conscience approves the result of the trial, even according to the testimony of a highly conscientious writer who does not wholly or heartily approve of it. And this being so, the vehement objections of another medical writer may well be passed without further remark.

Dr. Folsom, in a pregnant paragraph, has attempted to show that Guiteau's shooting the President was to a certain extent the logical result of bad training, unscrupulous character, self-conceit, self-will, disappointment, cowardice, partizanship, religious delusions or deceit, poverty, love of notoriety, &c. That is to say, taking a man's history, his actions are the result of his character, although we would scarcely say the logical result. But if this be admitted with regard to Guiteau by his most competent medical defender, it follows that the explanation of insanity must be surrendered, for conduct cannot be the result of natural character and also the result of the interference of disease; and on the whole purview of the case it seems to be impossible to escape from the conclusion that the crime of this assassin was the result of his character, as it was formed under social influences to which any sane man might have become subject. It would be tedious and superfluous to go yet again over the details of the many times told tale of this criminal biography; neither does it seem necessary to comment at any length upon the weighty and unanswerable evidence of the alienist-physicians who gave their evidence for the prosecution. There are, however, one or two points upon which some comment may possibly be useful, and the most important of these is the different and extreme opinions which were expressed on the influence of hereditary predisposition to insanity. On the one side it was argued at the trial, and has since been urged by medical writers, that "there was a strong hereditary predisposition in the case;" while on the other side it was as strongly affirmed that the insanity of direct

relatives in the ascending line, i.e. of parents and grandparents, was alone to be considered as of possible importance in influencing the mental health, and that the insanity of collateral relatives was of no significance whatever. And on this principle it was assumed that the insanity of Guiteau's relatives, who were of collateral and not of direct relationship, could not in any way elucidate the question of his own mental state.

This principle, however, cannot be accepted unconditionally, for although the general rule may be correct that the tendency to mental disease can only be inherited from parents or grandparents, yet that which it is desired to show in such a case, namely, a family disposition to disease, may derive as great probability from a large number of collateral instances as from a smaller number of direct ones. Favourable circumstances sometimes enable parents to escape the manifestations of disease in themselves while yet they transmit the faulty organism of the race, so that the insanity of uncles, aunts, and cousins may become an element in estimating proclivities to constitutional disease. I must, however, add that in my opinion the argument in favour of insanity founded upon the supposed transmission of an hereditary tendency to mental disease has of late been used in most absurd and unjustifiable excess, and I do not know that the interests of justice would be damaged if it were to be excluded altogether in judicial inquiries; for if it could be clearly shown that both a man's parents, and all four of his grandparents, and all his uncles and aunts had been unquestionably insane, it would afford no proof whatever that the man himself had been insane. Such evidence would at most strengthen the presumption that he had been so under circumstances which would otherwise be more doubtful. Such evidence can never be a satisfactory substitute for more direct evidence as to the issue, and the small worth it possesses must at once be felt when we consider that only a moderate proportion of the children of insane forefathers ever do become insane.

The procedure by which courts of criminal justice attain to that which is (curiously enough) called the moral probability on which they act is too rough and coarse to encourage the

nicer investigations into psychological heredity, and until the courts are better instructed, one form of insanity among relatives will probably serve as good a purpose as another to give a sympathising jury an excuse for acquittal. Yet it is not the less known to those who have studied this question that the hereditary tendency to mental disease follows certain forms of insanity, or even of nervous disease which is not insanity; and that the forms of insanity so acquired are often distinguishable, so that an experienced mental physician may often form an opinion upon the probability of a given case of lunacy being hereditary, even when he is quite ignorant of the family history. An example of an opposite kind was afforded in the American affidavits sent to this country in favour of the poisoner Lamson, in which the senile dementia of two uncles who died in a New York asylum at an age verging upon four-score, was gravely propounded as evidence of hereditary insanity in that peculiarly cruel, cool, and calculating murderer, whereas it only proved that some of his relations were long-lived. Surely until evidence as to the influence of heredity can be better appreciated at its real worth, it would be better to exclude it altogether, and to insist upon more careful investigation of the real question at issue, which of course is the actual state of the man at the time of the offence.

Another point of the greatest importance which, but for the admissions which the prisoner made during his examination by Dr. John P. Gray, might readily have been the cause of a great mistake and a miscarriage of justice, was the supposed delusion that the prisoner believed he was inspired to the act by the Deity. No other belief at any time attributed to Guiteau could be reasonably construed into an insane delusion, notwithstanding Dr. Folsom's curious opinion that Guiteau's expectation of approval from the President's enemies was an extraordinary delusion. The belief in Divine inspiration is very different, and might easily have been considered a delusion if not explained. The explanation is afforded by the doctrines, and the phraseology in which they are expressed, of the religious community with which Guiteau was intimately associated, and which he had imbibed from his earliest years.

It is surprising that the influence of this curious sect or

community, the Perfectionists of Oneida, upon the mind and conduct of Guiteau was made so little of at the trial, either by the prosecution or by the defence. Probably it was felt to be a double-edged argument dangerous to handle. It would be difficult, however, to over-estimate this influence, and probably it would not be too much to say that the assassination of President Garfield was the outcome of Oneida, for we must not forget that Guiteau's father was an enthusiastic believer in the doctrines of Father Noyes, and diligently impressed them upon his son, indeed upon his sons, for Guiteau's brother expounded in court the creed which sounds so strange in modern ears, of the real battle between God and the Devil, and the part we take in it. "That was my father's theological view, it was my brother's, it is mine." When Guiteau actually entered the community he must necessarily have believed in the main doctrine of his co-religionists, that all actions are directly inspired by God or by the Devil; and after he left the community it is plain, from his letters and papers, that he retained and acted upon that belief. It was by Divine inspiration that he believed himself destined to establish a great theocratic newspaper. If he had been attacked by bodily disease he would have trusted to the Faith Cure as it is used at Oneida, that is to say, its cure by the direct personal intervention of God in answer to prayer.[1] And it is unreasonable to suppose that in the most grave and serious action beyond all comparison in his life he would cease to entertain his most habitual thought. But was this belief an insane delusion? If so, all the world is mad outside each man's little circle of fellow-believers. The inconsistency involved in the belief that God can inspire a wicked act does not make the belief an insane one, for we know that the "devil can quote Scripture to his purpose," and that more devilment has been done in God's name than in any other.

That the belief was not a delusion is evident from the fact that it was derived from the teaching of others; that it was not the result of disease; and that Guiteau attempted to make others believe that it was a delusion as an excuse for his crime,

[1] For a good description of the Perfectionists of Oneida and their creed and mode of life, see Nordhoff's 'Communistic Societies of the United States,' p. 258.

which no one under the insane delusion of inspiration would have done. It was a sane belief, probably as sincere as many other religious beliefs; a belief which may do good or evil in the world as it is entertained and acted upon, with purposes more or less consistent, by good or by wicked men. The answer when such a belief is urged as an excuse for crime is that other men may entertain and act upon it more consistently than the criminal. The judge and jury may say, "We also believe in the inspiration of the Almighty, and we have prayed to Him that He will enable us to give a just judgment upon you, and our judgment, inspired by Him who is the source of all justice, is that you are Guilty, as indicted, and that you must suffer the penalty of your Crime."

Critical Digests and Notices of Books.

Diseases of the Spinal Cord. By Byrom Bramwell, M.D., F.R.C.P. Edin. 8vo. pp. 300. With 42 original chromo-lithograph drawings, and upwards of 100 other illustrations. Maclachlan & Stewart, Edinburgh, 1882.

The work before us is a gratifying evidence of the increasing attention paid to the study of nervous diseases, and an evidence, let us hope, of the increasing demand on the part of students and practitioners for the most recent information respecting them.

Dr. Bramwell's work is well adapted to supply this information with regard to the diseases of the spinal cord, and in as simple a form as is compatible with accuracy.

The first chapter of the work is occupied with a brief account of the anatomy and physiology of the cord, but which is sufficiently full and comprehensive for clinical purposes.

The "tracts" or "systems," of which the spinal cord is composed in its transverse area, as determined by the embryological investigations of Flechsig and others, are described with great care; and the whole chapter is, like the rest of the work, fully illustrated by many admirable diagrams.

In the second chapter Dr. Bramwell deals with the pathological anatomy of the spinal cord, and most of his own original observations appear in this section. The careful descriptions and drawings of the morbid changes in a case of cerebro-spinal sclerosis which came under his observation may be noticed as a particularly favourable specimen of his work. Not only are the lesions of the spinal cord described with great care and minuteness of detail in this section, but the positive and negative symptoms caused by each lesion are also briefly enumerated.

The third chapter is the most valuable in the book to the clinical student. After giving an admirable outline of the method of case-taking, the author proceeds to give specific instructions regarding the precautions which must be taken, and the tests which must be applied in order to give scientific accuracy to the clinical investigation. The part which relates to the examination of the disorders of the motor and trophic mechanism of the cord, is illustrated with Erb's diagrams of the reactions of degeneration and Ziemssen's plates of motor points. The author's explanation of the action of the reflex mechanism presiding over the functions of the bladder, rectum, and sexual organs is particularly deserving of attention. The diagrams by which the action of these complicated mechanisms is illustrated are very effective. This chapter is brought to a close by a general sketch of the diagnosis, prognosis, and treatment, which shows that the author is as familiar with his subject at the bed-side as he is in the pathological room and physiological laboratory.

In the fourth and last chapter, which occupies rather more than one-third of the volume, a very elaborate tabular classification of the diseases of the cord is given, and the organic affections are afterwards separately described. The description of each disease is necessarily brief, but it is always lucid and accurate, and nothing essential is omitted. Dr. Bramwell tells us that it was not until the other three chapters had passed through the press that he determined to add a description of the individual diseases. The working-out of an after-thought of this kind is sure to entail inconveniences. The result in this case is that Dr. Bramwell has had to repeat in the last chapter much of what he had already stated in the sections devoted to the pathology of the cord, and consequently there is a seeming want of harmony and logical consistency between the various parts of the work. Students, however, will readily pardon any defect in the structural arrangements of the book which may have arisen from the cause assigned, and they will only be too thankful to Dr. Bramwell for sacrificing some degree of logical consistency, rather than omit the description of the individual organic diseases.

The work throughout is profusely illustrated by woodcuts

and chromo-lithographs. Several of the woodcuts are borrowed from well-known sources, while a large number—the majority probably—are original; it would be difficult to imagine any more useful diagrams as aids to diagnosis than those which appear in this work. But the chief glory of the book, so far as illustrations are concerned, is to be found in the chromo-lithographs, as many as forty-two of them appearing in a work of about 300 pages. The chromo-lithographs were drawn by the author himself, first with the camera-lucida, and then in lithograph chalk, and are, with two exceptions, representations of his own sections. These illustrations are indeed works of art; the representations of sections stained by osmic acid are quite unique of their kind, and no one who has not seen the original sections, as the present writer has had an opportunity of doing, can understand the extreme faithfulness with which they represent the originals.

This book must greatly enhance the already well-earned reputation of Dr. Bramwell as a neurologist, and it cannot fail to prove an acceptable and safe guide to all those who wish to obtain a scientific knowledge of the diseases of which it treats.

James Ross.

Sur les fonctions de la Vessie. By Prof. A. Mosso and P. Pellacani. (Reprint from the 'Archives Italiennes de Biologie.') Turin, 8vo, 1882, pp. 66.

The investigations related in this pamphlet were conducted by means of the plethysmograph, both on animals and on the human subject. The importance of the results obtained by the authors can scarcely be overrated; they throw light not only upon the various vesical symptoms in nervous affections, but also upon the functions of unstriated muscles generally. The methods employed by previous observers were too crude to give either accurate or complete information on the subject; and Profs. Mosso and Pellacani's contribution constitutes a great advance in our knowledge concerning the important subject-matter of their experiments.

By registering the varying pressure of the liquid within the bladder they were able to determine the slightest changes

(dilatation or contraction) of its walls. This they did by connecting the registering apparatus with a catheter within the bladder, taking care to eliminate the effects of extraneous pressure (such as that due to contractions of the abdominal muscles). The details of the method are fully described in the Memoir. The shortest contractions of the bladder last six or seven seconds, and are thereby easily differentiated from compression of the organ through contractions of voluntary muscles.

Experiments on the human subject show that *every psychical act*, intellectual, emotional, volitional, as well as every sensory perception, is accompanied by a contraction of the bladder. Moreover, they prove that the bladder can be made to contract by a pure act of the will, apart from any sensory reflex, without the concomitance of abdominal pressure. (That no abdominal pressure is required for normal micturition is clearly shown by the vigorous expulsion of urine by curarised animals.) There is therefore an unstriated muscle, the bladder, which is under the direct influence of volition, as there are several striated muscles which are beyond that influence.

The influence of psychical states on the bladder fully coincides with what we know to occur in blood-vessels. Prof. Mosso in his classical researches on cerebral circulation[1] has put this point in evidence. The question arises, however, whether the bladder under any conditions undergoes dilatation or contraction without the accompaniment of similar changes in the blood-vessels. Simultaneous tracings of the respiratory movements, arterial pressure, and vesical pressure clearly show that the latter may vary without any corresponding variation in the former, nay, that it may increase whilst the former diminishes (as, for instance, when the pneumogastric is cut).

Experiments made on thoroughly curarised animals, with the bladder exposed, confirmed the results of the experiments made on man, and showed that every psychical act, every excitation of a sensory nerve, is accompanied with a contraction of the bladder. "The movements of this organ," say the

[1] See "Brain," April, 1881.

authors, "demonstrate the painful fact that sensibility and consciousness remain intact in curara poisoning"; and they affirm that the bladder constitutes a more delicate æsthesiometer than the blood-vessels, in no way inferior to the iris itself.

Contractions of the bladder give curves which cannot be reduced to any type, owing to their irregularity, and are always protracted, as we have previously stated. They may extend over several minutes, and during that time undergo many variations in their intensity. It is obviously difficult to distinguish between such contractions and changes in the tonus of the organ. Nor is it of any importance that we should do so in the case of the unstriated muscular fibres which differ so widely in their properties from the striated fibres. We may therefore speak indifferently, at least for the present, of changes in the tonus, or of very slow contractions and relaxations of the bladder.

The authors have made many observations on the variations in the tonus of the bladder during normal and artificial sleep; but we shall not dwell here upon these, and merely summarise their results with reference to the vesical tension. The elasticity of the bladder is considerable, and under the same pressure the organ may contain different quantities of fluid. The want to micturate occurs whenever a certain pressure is reached independently of the mere amount of fluid. Considerable distension leaves after it a condition of dilatation of more or less duration.

We reach the interesting subject of the influence of the cord on the bladder. Preliminary experiments showed that sensory impressions, even powerful, did not produce contraction of the bladder in animals whose cord had been cut through high up, unless large doses of strychnia had been administered. The main task was, however, to determine in what part of the cord the motor supply to the bladder is contained. This the authors did by the method of spinal hemi-sections, but did not use the very deceptive method of Budge (faradisation of the cut end of the cord) for testing the effect, but applied the facts elicited previously with reference to the response of the bladder to sensory stimuli. A series of experiments and counter-

experiments gave them very uniform results. We refer the reader to the original memoir for the details of, and the precautions taken during, these delicate manipulations. Their conclusions are: That motor-fibres of the bladder are contained in the posterior columns of the cord, or in the extreme posterior bundle of the lateral columns; that the portions of the cord anterior to these do not contain any vesico-motor fibres.

With reference to the sympathetic nerve, we find that the excitation, central or peripheral, of the cut filaments produces in both cases movements of the bladder; hence that it has sensory as well as motor functions. On the other hand, it cannot be the sole source of supply to the bladder, for its complete extirpation—an operation undergone by dogs without any notable ill-results in other respects—left the vesical functions unimpaired.

The authors give a history of the various views held by physiologists, from Galen to Goltz, on the mechanism of micturition, and give their own experiments and conclusions concerning it. They lay special stress (1) upon the sufficient power of the vesical muscle itself, removed from the influence of abdominal pressure, to raise a column of water of considerable height (1·5 to 2 metres for a dog); (2) upon the reaction of the bladder to volitional impulses. These points have been alluded to already as established by various experiments. The authors further show that the act of micturition is entirely independent of the respiratory movements, and can begin whilst the abdominal muscles were either contracting or relaxing for inspiration or expiration. Any attempt to interrupt the act, however, is accompanied with a deeper *inspiratory* effort. The abdominal pressure, moreover, can readily be registered by means of a small india-rubber bag introduced in the rectum; it may thus be demonstrated that this pressure is not sufficiently powerful to overcome the resistance of the vesical sphincter and urethra, even as measured post-mortem.

The general conclusion to be drawn from all these facts is that any respiratory change observed during miction is only a phenomenon of inhibition, similar to those observed during cerebral activity for instance, and not as an act directly contributing to the compression of the bladder.

Another point brought to light by Profs. Mosso and Pellacani is that there is no antagonism between the actions of the detrusor and sphincter muscles; every contraction of the one is accompanied by a contraction of the other. The expulsion of the urine therefore takes place only when the contraction of the former is sufficiently powerful to overcome the resistance of the latter. The phenomenon of involuntary micturition, under the influence of emotions, &c., is not due to any paralysis of the sphincter (which can be shown under such conditions to be contracted), but to the direct action of the detrusor; and the frequent want to micturate felt under many psychical conditions depends upon increased pressure within the bladder, due to a persistent increase of the tonus.

During sleep the vascular and vesical tonus is diminished, and remains low as long as we remain warm in bed. Hence the want to micturate is not felt to any degree until we leave the bed, and expose the skin to the colder air. Then the tonus is at once increased, as becomes equally manifest by the contraction of the cutaneous vessels, and desire to relieve the bladder.

This fact illustrates the general law, that the desire to micturate depends not upon the quantity of liquid contained in the organ, but upon the amount of pressure exerted upon its walls—the truth of which has been established by the authors in a great variety of experiments; they show that the converse holds, i.e. that when the desire disappears, the pressure diminishes. It is within everybody's experience that the feeling of plenitude of the bladder frequently disappears if the desire to evacuate it is resisted for a short time. Under such circumstances, though the distension of the bladder goes on increasing, the pressure within is relaxed through a diminution of the tonicity.

We have still to mention briefly the results reached by Profs. Mosso and Pellacani with reference to the influence of certain respiratory influences on the condition of the bladder. Experiments on man, and animals with the cord cut or entirely destroyed, show that arrest of respiration or asphyxia produces a contraction of the bladder. The same effect follows a deep inspiration. Apnœa following a series of deep inspirations causes a diminution of the vesical tonus.

The bladder contracts when the action of the heart is arrested. It first contracts, then rapidly dilates, on destruction of the medulla oblongata; and though in this case the vascular tonus did not participate in these fluctuations, it may be said in a general way that "under conditions which excite the vaso-motor and the respiratory centres, there is at the same time stimulation of the muscular walls of the bladder."

Profs. Mosso and Pellacani promise us further contributions, destined to elucidate more completely several of the results recorded in their already very valuable contribution to the physiology of vesical functions. The interest and practical importance which their labours have for the neuro-pathologist are too evident to need any comment on our part. We need only express to them our thanks for their past and best wishes for their future labours.

Handbuch der Elektrotherapie. By W. Erb, M.D., Prof. of Medicine in the University of Leipzig. (Part I., 300 pp.) 8vo. Leipzig, 1882.

The great expectations raised by the announcement of Prof. Erb's Treatise on Electrotherapeutics have been fully realised, at least with reference to the part of the subject treated in the first instalment of the work. Within 300 pages the author gives a short history of the applications of electricity to medical purposes, then discusses electrophysics and electrophysiology in so far as they concern the subject in hand. As might be expected, electrodiagnosis, which may be said to owe its existence to the untiring zeal of Prof. Erb, is treated in a masterly fashion. Finally, we have an excellent exposé of the general methods of electrical treatment, and their rationale. Though the author has no new discoveries to impart, yet it may truly be said that this volume marks a new era in the development of electrotherapeutics. In Germany, where the influence of his teaching was directly felt, his principles have already found their application in the hands of many able followers and fellow-workers. But even there the organization

of those doctrines into a continuous exposition must have a powerful effect in furthering the progress of the theory and practice of electrotherapeutics; whilst in other countries the apparition of a clear rational book on the subject will be welcomed as a new revelation. The majority of the treatises on medical electricity are mere sketches, or, if they have any pretensions to completeness, consist often of painful compilations of undigested, useless matter, interspersed with narratives of wonderful cures. The gross ignorance of physical principles displayed by certain authors make a curious contrast with their fantastic ease in evolving physiological data from the depths of their inner consciousness. The systematic silence preserved with reference to cases where the panacea has failed to cure, or where the results have been temporary only, give to such writings that appearance of ex parte pleading which has brought the whole subject of electrotherapeutics into suspicion and contempt among us.

We hail the appearance of Prof. Erb's book (of which, we understand, there is a hope of seeing an English translation) as one written not only by a master of the special branch of medicine with which it is concerned, but also by a representative of modern neurology—hence as a book calculated to command respect and disarm prejudice.

Nothing could be happier than the unerring certainty with which the author knows how to deal with the troublesome questions of electrical treatment, always keeping in view both the reality of results obtained empirically, and the almost absolute want of experimental data whereby to explain them. For him, as for us, the first duty of one who wishes to make a rational use of electricity in medicine is to become thoroughly master of the physical principles which govern the application of the current to a composite electrolyte, such as the human body. The unipolar method, the basis of all that is sound and durable in electrotherapeutics, of diagnosis especially, is itself an encouraging fruit of the increasing knowledge of those principles.

It would be impossible to enumerate all the excellencies we find in Prof. Erb's lectures; but we feel no regret at our inability to give even a short résumé of the volume, for we

feel quite sure that all those who take the slightest interest in the subject of electrotherapeutics will be anxious to read it for themselves. And having read it, they will appreciate the difference between the new science and the old.

Experimentelle und Kritische Untersuchungen zur Electrotherapie des Gehirns. By L. LÖWENFELD, M.D. Munich. 8vo. pp. 146.

THIS book may be divided into two parts. The one, historical and critical, contains a copious list of references, and does credit to the author's industry in ransacking the medical records from the time of the earliest therapeutical applications of galvanism down to the present time. The various opinions held on the effects of electricity applied to the human brain (through skin and bone) are recapitulated and criticised. In his anxiety to be complete, Dr. Löwenfeld does not respect the rules of perspective, and takes great pains to analyse and refute at full length the "physiological" theories of certain electrotherapeutists—views which carry much the same scientific weight as that which their notions about hydrostatics or spectral analysis may have. Still we are grateful to the author for having brought together so much matter, and thereby facilitated the task of future investigators.

The main phenomena produced by transverse galvanisation of the human brain are subjective giddiness, an objective tendency to fall towards the anode on closing, towards the kathode on opening, the circuit, certain nystagmus-like movements of the eyes. To these we may add a sensation of cerebral oppression and of sickness. Hitzig has studied some of these effects with much care, and according to him the rotation of the eyes is of the nature of "forced movements," and the loss of equilibrium must be considered as consequent upon them.

This theory is not free from objection, and perhaps Erb's suggestion satisfies the conditions of the problem more completely. He says that the equilibration of the body may be considered as depending upon a centripetal, bilaterally symmetrical, influence. If this influence is disturbed, as would be

the case by a transverse galvanic current, and its unequal polar influence on either hemisphere, the equilibration will be destroyed. The movements of the eye may be secondary to this influence, and be the consequence, not the cause, of the disturbed sense of equilibrium.

Hitherto every attempt to excite the motor cortical centres through the skull has failed, a fact which does not seem to discourage certain authors from assuming, under the same conditions, a stimulation of more or less problematical and deeply-seated centres, even by weak currents. Thus the vaso-motor, vomiting, convulsion centres may all be brought into play! Nothing can be simpler than the explanation of the functions of an organ of a highly complex structure, in which every phenomenon depends upon the co-operation of several parts, each depending for its normal function upon mutual influences which escape every means of estimation—nothing can be simpler, we say, than to explain those functions by referring every single action to the excitation of a "centre," or to the dilatation of an arteriole. Unfortunately, what is simple is not always true.

But to return to Dr. Löwenfeld's book. The original part of it consists of the description and discussion of experiments made by him on the influence of the electric current on the circulation of the brain. It is strange that the author should apparently be ignorant of Mosso's researches, especially with regard to the methods used by physiologists. Mosso did not use electricity in his experiments, and it would have been interesting to test the effect of this agent under the same conditions. Dr. Löwenfeld simply looked at the superficial vessels of the exposed brain through a lens, and estimated the changes in their calibre by the eye.

This method cannot possibly yield very reliable results. Control experiments of no kind whatever seem to have been made by the author; a point which detracts still further from the value of his results. But we do not intend to criticise experiments which have clearly to be repeated from a strictly scientific point of view. We grant, in the meanwhile, the correctness of Dr. Löwenfeld's conclusions,[1]

[1] See 'Brain,' April, 1881.

which may be summed up that the vessels of the brain contract according to the pole applied to either side, or to the forehead and occiput. But we cannot see of what assistance the knowledge of such facts can be in the explanation of, and in the indication for, therapeutical applications of galvanism to the head.

It is, no doubt, desirable to ground empirical data upon experimental results; but it does not promote the advance of medicine to force complex facts into a narrow formula. Two objections at once present themselves against the indiscriminate application of Dr. Löwenfeld's conclusions to electro-therapeutical problems. First, he does not sufficiently take into account the strength of the currents necessary to produce the alleged vaso-motor changes in the brain of the animal experimented upon. But granting that he never used these currents stronger than is admissible in the case of the human subject, he overlooks the fact of the difference in size between the brains, and consequent difference of electrical density in the two cases makes it impossible to argue from one to the other. Secondly, in how many cases of brain-disease is the anæmia or hyperæmia the cause instead of the effect of the disturbed nervous action? And in cases where vaso-motor changes are the cause, why should the weak and transitory vasomotor effects of "medical" currents act more favourably than those, much more marked, of nitrite of amyl and the like?

The last chapters of the book contain the views of a number of authorities as to the curative results of cephalic galvanisation, and the best methods to obtain them. Here, as usual, doctors differ as to the strength, duration, and direction of the currents to be used, and Dr. Löwenfeld has but one more opinion to add to many others. But we prefer not to dwell upon such an uncongenial topic, and conclude with stating our conviction that Dr. Löwenfeld's great industry, literary and experimental, will not have been spent in vain if his book were to induce a physiologist to take up the question and apply to it the resources of modern science.

A. DE WATTEVILLE.

Teoria fisiologica della Percezione. Del Prof. GIUSEPPE SERGI. 1881.

THIS work is a scientific attempt to elaborate a doctrine of perception from observation of its fundamental facts, and deduction thence of the laws they obey. According to the author, the difference between sensation and perception depends on this, that the former implies no localisation or space-relation, while the latter does. In tactile perception and in taste perception the localisation is to the surface of the skin or mucous membrane; in sight, hearing, or smell it is to the outside world. The former, the author terms adherent-localisation, the latter, projective-localisation. After a full criticism of the English and German views on the subject, the author goes on to explain his views. Chapters IV. and V. contain an account of the nervous process in perception, the author holding the doctrines of localisation and of transmission as strongly as Dr. Panizza rejects them. Chap. V., on the proofs of the perceptive wave, is interesting and ingenious, the author contending for a centrifugal wave in perception, and quoting as an instance the case in which, when one eye is examining a microscopic appearance, the other, being open, perceives the same image also. Unless there were a centrifugal wave present, this, he says, would be inexplicable. After localisation, the next step in perception is movement of the sense-organs, which may be of two kinds, viz. of direction or of accommodation. The latter exists strongly marked in the case of vision, to a less extent in that of hearing, and least of all in smell. The former is also well marked in vision, the eye having six muscles for the purpose. In other sense-organs they are absent; but the muscles of the parts to which they are attached perform the analogous function.

The movements of organs are associated to the centrifugal wave of perception, and produce complete localisation, both in direction and distance; so that the clear and distinct perception of an object is obtained by its form in space combined with its sensational quality. Physiologically we find to be associated (*a*) the movement of direction, (*b*) sensitive excitation. Psychologically there are combined (*b*) extension (form power),

(*a*) sensational quality (colour, sound, resistance, smell). Hence there arises objectivity which is projection, or the externality of subjective forms, which come to be considered as real qualities of things.

Physiological time is that occupied from the peripheral excitation of the organ to the occurrence of localisation.

Perception reaches the maximum of clearness in attention. Localisation, therefore, is most complete when concentrated in a special site, particularly if at the same time there is insensibility of the other perceptive centres, and temporary paralysis of the different motor centres.

Hallucinations are formed when the centrifugal course of the sensitive excitation occurs, without the occurrence of the centripetal. The localisation represented is mistaken for the actual.

All the processes take place unconsciously. Consciousness is only the revealing form of the phenomenon which is taking place.

Professor Sergi's work forms the 29th volume of the International Scientific Library, and is undoubtedly worthy of taking its place there.

A. Rabagliati.

Clinical Cases.

CASE OF GLIOMA OF THE RIGHT HEMISPHERE.

BY J. E. SHAW, M.B.

Physician to the Bristol Royal Infirmary.

S. H., æt. 19, domestic servant, was admitted to the Bristol Royal Infirmary, November 17, 1881, complaining of loss of power in the left arm.

History.—Two months ago she observed a twitching or jerking of the index and middle fingers of the left hand, which came on for a few minutes at a time, several times a day. This twitching was not attended by any pain or other abnormal sensation, and did not interfere with the voluntary control over the fingers when the twitching was not present. About a week afterwards the same phenomenon was observed in the thumb, and two or three days later in the ring and little fingers. The jerking then affected the wrist-joint in a day or two, and a day or two later again the elbow-joint became involved; but the muscles moving the shoulder-joint remained exempt. In about three weeks from the commencement the jerking ceased, and she began steadily to lose power in the limb, being able to move the arm, but not to grasp anything in her hand. She placed herself under medical treatment until her admission to the Bristol Infirmary, but did not observe any particular alteration in her symptoms beyond the increasing weakness in the arm, and some headache.

On admission she presented the appearance of a well-nourished, healthy young woman. The left arm is weak; the fingers cannot be quite extended, especially the inner ones; they can be flexed so as just to touch the palm; the movements of the thumb are almost abolished; wrist can be flexed and extended slightly; elbow and shoulder-joints can be moved more freely, but the movements can easily be restrained; the hand can just be raised to the top of the head; the intrinsic muscles of the thumb and little finger are a little smaller than those of right hand; no grooving on back of hand, and left forearm measures

fully as much at various points as the healthy one. No evidence of joint disease. Says there is a *feeling* of numbness in the left hand; but on experiment, there does not seem to be any actual difference in sensibility between the two sides. Face symmetrical, and movements of leg unimpaired. No abnormality in heart or other organs; sight good.

Previous History.—Has always had good health, and has had no illnesses; has never had any kind of fit, nor blow on head that she can remember. During the last three weeks she has had a good deal of headache, unattended with vomiting. Temperature normal. No history or marks of syphilis.

Family History.—Father died of consumption. Mother alive and healthy. Only one brother and one sister, both alive and healthy.

Subsequent History.—Was kept in bed for four days after admission, and then allowed to get up. On attempting to walk she noticed a stiffness of the left leg, and a day or two afterwards she could not walk without dragging the toes along the floor; there was marked weakness of the limb also, even when lying in bed. Plantar skin reflex retarded on left side, and less marked than on right; knee reflexes are well, and about equally marked on the two sides. On one occasion a slight amount of ankle clonus was obtained in the left foot. She had not experienced any kind of numbness, pain, or other sensation in the leg previous to this partial paralysis, and was not aware of anything being the matter with it until she attempted to walk on it. At this time, six days after admission, the arm had become weaker than on admission, so that the hand could not be raised to the top of the head. On examining the face it was still symmetrical in appearance, but on her attempting to draw back the angles of the mouth, it was found that the left angle of the mouth was only very slightly retractable; this paralysis, however, applied to voluntary motion only for the automatic action of the left zygomatic muscle, as seen brought into use in smiling, for example, remained unimpaired. How long this condition may have been in existence it is impossible to say. Neither the buccinator, nor the orbicularis, nor any other facial muscle was affected. On ophthalmoscopic examination, the margin of the left disc was observed to be shaded off; but there was no swelling of the disc, and there was possibly some hyperæmia of the right disc.

Dec. 1*st.*—Complained a good deal now of headache, which from the first onset of it has generally been on the right parietal region, though sometimes it was situated equally across the two sides of the forehead; the pain is of a dull, aching character, like a bad headache.

The tendon reflexes of the left knee and forearm now excessive. The disc of right eye decidedly hyperæmic, and it is somewhat swollen also. The shading off of the margin of the left disc is more marked.

Dec. 4th.—Complained very much of headache, which is now like "rheumatic," rather sharp and darting, located generally in the right parietal region, but sometimes in the right frontal region. The scalp is tender on combing the hair. For the last three or four days she has vomited in the morning on rising from bed, the vomiting being unattended with nausea or retching.

The headache is less severe by night than by day, being worst in the morning, but never quite absent.

Sensation and power of localisation in left hand are now quite normal, nor is there now any subjective anæsthesia.

Dec. 7th.—Remains about the same, but says she "sees double" at times. There is no squint nor paralysis of the muscles of the eye-ball perceptible on testing her power of moving her eye-balls. Sickness and headache continue.

Dec. 15th.—To-day, on approaching her, I noticed that her eye-balls were moving independently of one another, their motions not being co-ordinated until her attention being given to fix her gaze upon a definite spot, she was able to regain control over the motions of the eyes. The double vision comes on frequently, and lasts several minutes.

Can now only move fingers very feebly; no power of grasping; slight elbow movements; cannot raise her arm, but habitually carries her left hand supported by her right. Paralytic symptoms of face and leg remain unaltered. Headache often, but no impairment of mental faculties, nor of speech. Vomiting occasionally.

Some slight improvement during the next fortnight.

Jan. 4th, 1882.—Is now very drowsy and dull by day, but does not keep her bed. Is perfectly clear in her mind. Very restless at night, and cries out in her sleep. The pain in the head is very severe; vomits every morning.

Jan. 14th.—Headache still worse, almost entirely referred to right parietal region; no vomiting. The absence of co-ordination in the movements of the two eyes is now very marked; but the movements are still quite under her deliberate control. Optic neuritis is now well marked and equally present in the two eyes. Being much worse in herself she now remains in bed altogether.

Medicinal treatment having quite failed to check the progress of the disease, a consultation of the surgeons was held to determine whether it was advisable to resort to operative interference—to remove the growth, if that should

be possible, from the cortex of the brain, or if that were not possible, to relieve the pressure upon the brain by trephining. Having held a consultation the surgeons decided that, though they would not urge upon the patient, or her mother, the desirability of performing any operative measures, at the same time they were quite willing to make the attempt if it was wished. The grounds of this decision were, that though the patient's present condition was practically hopeless, it was probable that the lesion had extended over too large an area of the brain for its successful removal to be possible.

Jan. 17*th.*—The patient decided not to have any operation performed. Headache about the same; no vomiting; no ptosis nor other paralysis of ophthalmic muscles. Remains in bed heavy and drowsy, but not at all insensible; no kind of convulsion.

Jan. 18*th.*—Passed a very restless night. Has been screaming out this morning from severe lancinating pains in the head, very much more acute in character than it has ever been before; is quite sensible, and able to talk perfectly. At 10 A.M. had a hypodermic injection of ¼ gr. of morphia and $\frac{1}{60}$ gr. of atropia; two hours afterwards she was sleeping, and snoring rather loudly. At 4 P.M. had a severe attack of hiccough, which did not, however, completely awake her. At 9 P.M. she was still sleeping; right pupil still remained dilated, but left was contracted. At 11 P.M. had another attack of hiccough; slept afterwards, and died quietly shortly before midnight, without any convulsion.

Post-mortem examination 12 hours after death.—Body well nourished. Only head examined. On removing skull-cap, which was not abnormally adherent to the dura mater beneath, a yellowish-coloured patch was seen lying beneath the dura mater, in the centre of the parietal region of the right cerebral hemisphere. On removing the dura mater, which was not adherent to the pia mater, some yellow-coloured fluid escaped from the interior of the growth, which was now fully brought into view. The tumour was soft, and had broken down in the centre; its superficial surface was nearly circular in shape, situated apparently on the site of the fissure of Rolando, but there was great difficulty in tracing the convolutions of the right hemisphere. On microscopical examination, the fresh tumour exhibited the structure of a glioma.

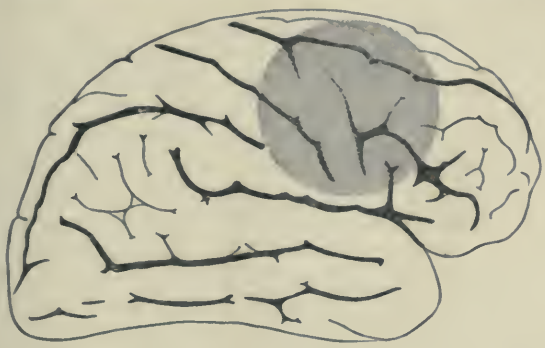
Measurements.—Cerebral hemisphere from before backwards, 6½ in. Tumour from before backwards (maximum), 2 in. Tumour transversely (maximum), 2¼ in.

Note by Dr. Ferrier on the Condition of the Brain.

The brain which was sent to me by Dr. Shaw, after hardening in spirit, presented no abnormal appearance, except on the right hemisphere, where the tumour was visible, and of the exact dimensions indicated by him. The surface of the tumour coincided almost exactly with the surface of the hemisphere.

On careful examination it was seen that the growth had essentially only displaced and compressed the convolutions, so that the arrangement of the gyri and sulci was considerably altered.

The centre of the tumour, which was soft and easily broken down, coincided almost exactly with the centre of the præcentral sulcus. The ascending frontal or præcentral convolution, instead of pursuing its usual direction from above, downwards and forwards, made a sweep round the posterior aspect of the tumour. This convolution was much flattened and hollowed out anteriorly, and pressed backwards against the



ascending parietal or posterior central convolution, which, with the intervening fissure of Rolando, made a sweep round the tumour posteriorly. The bases of the frontal convolutions were compressed forwards in irregular gyrations by the anterior margin of the tumour, and the lower extremities of the ascending convolutions similarly downwards towards the fissure of Sylvius.

The tumour could be shelled out of its position more or less easily, and when it was followed in this way it was found to have the shape of a pear or wedge, the apex penetrating through the centrum ovale almost horizontally inwards to the roof of the lateral ventricle, where the corpus callosum begins to radiate into the hemisphere. It did not extend to the

cerebral basal ganglia, which were of normal appearance and consistence. The position and relative size of the tumour are indicated on the accompanying figure, and the mode in which the convolutions were displaced can be ideally represented by supposing the tumour to have grown out on all sides from a centre in the præcentral sulcus.

This case illustrates very clearly the fact pointed out by Hughlings-Jackson, that the most volitional and independent movements are the first to suffer from any cause affecting the motor centres generally, the thumb and index finger before the rest of the hand, and the distal before the proximal movements of the upper extremity, the arm before the leg, &c.

It is doubtful how far operative interference might have succeeded, owing to the depth to which the tumour penetrated the centrum ovale. But it is not impossible that enucleation might have been effected, or at any rate, the compression relieved by the operation of trephining. There is reason for believing that much may yet be done towards the relief of these and similar cases by surgical interference under proper antiseptic precautions.

MELANCHOLIA WITH LEFT HEMIPLEGIA, AND DEFECTIVE VISION OF LEFT EYE; DESTRUCTIVE LESIONS OF RIGHT ASCENDING CONVOLUTIONS AND GYRUS ANGULARIS.

BY JAMES SHAW, M.D.

Haydock Lodge Asylum.

A. G., housewife, aged 46, was admitted November 24th, 1879, in feeble physical health, with symptoms of phthisis in an advanced stage.

The "Statement" appended to the "Order for Admission" contained the following information: first attack; duration, four months; is suicidal; supposed cause, not known.

The "Medical Certificate" stated:—"Very depressed and melancholic in character. Has attempted to commit suicide, and says she will do so again, as she is tired of life."

Condition on November 28th, 1879:—Expiratory murmur over left apex prolonged and interrupted; pain in left subclavicular region; cough; sibilant râles; had not menstruated since September 1879; left hemiplegia of face, tongue, and extremities, but no contracture; vision of left eye defective; the tendon reflexes were not investigated; she had been troubled, according to her own statement, with muscæ volitantes and tinnitus aurium, but had had no definite hallucinations, either visual or auditory; said she had had strange dreams; low-spirited; memory defective, especially for recent events; power of calculation feeble.

December 5th.—Less low-spirited. Improved as to her physical health.

December 19th.—Stupid and depressed.

January 26th, 1880.—Confined to bed. Very weak. Has just recovered from an attack of diarrhœa. Hemiplegia more pronounced.

February 1st.—Great tendency to contract bedsores; especially on the paralysed side. Very weak. Temperature normal in the forenoon.

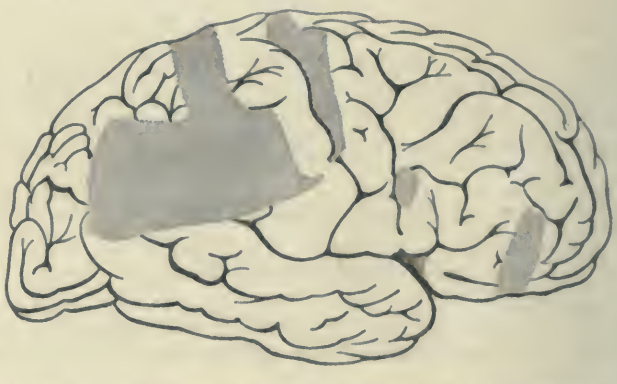
February 15*th*.—Rational and quiet, but very weak and helpless.

February 20*th*.—Very pale and feeble, with loud râles.

Died February 21st, 1880.

Autopsy :—

Left cerebral hemisphere, apparently normal.



Right cerebral hemisphere, arachnoid adherent over the occipital lobe. Several foci of softening, viz. :—

1. A slight superficial softening of the third frontal convolution, about a quarter of an inch in diameter, near the junction of the inferior frontal sulcus with the præ-central.

2. A superficial patch of yellow softening, measuring about three-quarters of an inch by half an inch, anteriorly where the third frontal becomes the external orbital.

3. The ascending frontal convolution, which was rather narrow, was affected with yellow softening in its superior half, the lesion extending the whole depth of the cortex ; and, inferiorly, near the level of the inferior frontal sulcus, encroaching on the ascending parietal convolution.

4. A large, irregularly-shaped focus of yellow softening, the anterior border of which corresponded with the posterior border of the ascending parietal convolution for two-thirds of its extent superiorly, encroaching on the convolution inferiorly, and almost meeting the lower portion of the focus extending from the ascending frontal ; posteriorly, the diseased portion (4) extended as far as the second temporo-sphenoidal fissure ; inferiorly, it passed round the termination of the fissure of Sylvius ; superiorly, a portion, about half an inch broad, extended as high as the superior extremity of the ascending parietal, the remainder nearly reached the inter-parietal fissure ; the cortex in this focus also was destroyed to the whole of its depth.

This last lesion (4) included nearly the whole of the gyrus angularis and gyrus supramarginalis, the anterior portion of the superior parietal convolution, a small portion of the ascending parietal near its junction with the supramarginal, and the posterior extremities of the first and second temporo-sphenoidal convolutions.

The central ganglia were intact.

Remarks.—Here we have sinistral hemiplegia with lesion of the opposite hemisphere, the motor area being affected with softening in three distinct places, viz.:—

1. In the third frontal near its origin.
2. In the superior part of the ascending frontal.
3. In the ascending and superior parietal convolutions.

With regard to the sensory symptoms, the defective vision on the left side coincident with lesion of the opposite gyrus angularis would tend to support Dr. Ferrier's views as to the functions of that convolution. The patient had no ocular disease visible to the naked eye, but no ophthalmoscopic examination was made.

The tinnitus aurium might have been caused by the lesion, when commencing, of the first temporal. The relative acuteness of hearing on the two sides was not ascertained.

The muscæ volitantes and strange dreams might be the result of commencing lesion of the pli courbe or of the adhesion of the arachnoid over the occipital lobe, where, Dr. Hughlings-Jackson says, irritative lesions, especially on the right side, give rise to coloured vision and other subjective ocular spectra.

To account for the melancholia there were two lesions; viz., an irritative one of the occipital lobe, and a destructive one of the right first temporal convolution. In 'L'Encéphale,' No. 3 (September 1881), M. Luys has reported several cases (Recherches Nouvelles sur les Hémiplégies Emotives), from which he draws the conclusion that emotive hyperæsthesia is, as a rule, the result of destructive lesion of the superior part of the right first temporal convolution. He finds that left hemiplegia, generally speaking, is almost always accompanied by excitation of the emotive faculties, rarely by troubles of sensibility, and less frequently by contracture than is right hemiplegia. He considers the coincidence of the destructive lesion with the emotive over-activity a new argument in favour of the theory of inhibitory phenomena.

CASE OF SYMMETRICAL SYPHILITIC DISEASE OF THE THIRD NERVES, WITH ARTERIAL AND OTHER LESIONS.

BY J. A. ORMEROD, M.B., M.R.C.P.,

Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen's Square.

[HEADACHE; attack of somnolence with delirium, lasting three days. Nine months later, left hemiplegia, drooping of both eyeballs and of left eyelid; extension of weakness to right leg; drowsy condition. Four months later, increase of coma; death with febrile symptoms.]

Frederick W., æt. 50, commercial traveller, was brought by his wife to Queen's Square Hospital on August 31st, 1881. His condition was then as follows: He can just walk when supported, but appears to have little control over his legs. The grasp of the hands is feeble, equal on both sides. The tendon reflexes at the knees are exaggerated, equal on both sides; at the carpal end of the radii present, equal on both sides. There is slight nystagmus. Neither eyeball moves freely in the upward direction; on the left side, movement in that direction seems quite lost, and that eye is constantly directed downwards. There is said to be failure of vision in the left eye. The right fundus oculi is normal; of the left I cannot obtain a satisfactory view. He is in a drowsy semi-comatose condition; puts out his tongue; moves any of his limbs. There is no actual paralysis, except that mentioned of the ocular movements.

The history was obtained from his wife, who appeared intelligent and trustworthy. His illness began in November, 1880 (nine months ago); before that he had had good health, with the exception of much headache. He woke one morning looking very ill, rambled "about trains and coaches," and for three days slept constantly, waking only to take his meals. He had no fits or paralysis, but after the disappearance of the somnolence his memory and mental faculties did not perfectly return for three months.

After that he remained fairly well, but seemed "weak," and did not go back to work. His employers dismissed him in July, 1881, and this was a great shock to him. A week afterwards, he returned home from a walk, dragging the left leg, and weak in the left arm; the left eyelid drooped, and the eyeballs were directed downwards, as now. The right leg became weak in a few days, though less so than the left. There was no affection of sensation. He seemed always drowsy and sleepy. His wife further stated that he had always been a healthy man; she knew of no nervous disorders, and of no consumption in his family. She had been married to him twenty-two years, had five healthy children, and had never miscarried. His occupation had entailed much mental work and anxiety.

The hospital being closed for repairs, he could not be then taken in. Mercury and iodide of potassium (the latter in increasing doses) were administered. No change of importance was reported till

December 28th.—He has been weaker and more drowsy during the last fortnight, and during the last two days much worse; always sleeping, perfectly helpless, unable to speak or understand when spoken to. As seen now in the outpatient-room he is in a semi-comatose condition, but can be roused sufficiently to enable him to put out his tongue or open his eyes. He cannot stand or walk; the eyes are still directed downwards; the tongue is protruded slightly to the right side; the patellar tendon reflex is absent on the right side, and obtained once only on the left; the plantar (cutaneous) reflex present on both sides.

He was admitted, and after being taken to the ward became cold and comatose; temperature (rectal) 95·6°; pulse feeble; respirations shallow; deglutition almost impossible; pupils small, inactive to light, but conjunctival reflex normal.

Brandy was administered freely.

December 29th.—Temperature normal. Has spoken and taken food, but easily goes off to sleep.

Evening temperature, 102·6°.

December 30th.—Much worse; breathing rapidly; quite unconscious; apparently moribund. Has vomited frequently in the night.

Temperature (10 A.M.), 102·2°; (2.30 P.M.), 103·6°.

Died at 3.30 P.M.

I am indebted to Dr. Beevor for the notes taken after admission, including the details of the history, and also for making the post-mortem, the account of which follows.

[Post-mortem nineteen hours after death; small tumours on cervical dura mater; thickening of the other membranes,

and softening of the cord in that region; disease of the basilar artery; symmetrical enlargement of the third nerves.]

Calvaria and dura mater of skull normal; arachnoid and pia show slight thickening and opacity; and in the sulci about the ascending parietal convolutions, a few opaque bodies the size of a pin's head.

(Subsequent microscopical examination showed that there was here no tubercle, that the vessels of the pia mater were quite normal, and that the cortical substance of the brain beneath was natural.)

Cerebrum and Cerebellum, both surface and interior, normal.

Basilar Artery decidedly thickened, the thickening being not uniform, but taking the form of nodules, projecting from the sides of the artery. No thrombosis found anywhere.

The third Nerves of both sides present a fusiform enlargement, soft, reddish-gray in colour, commencing a few lines beyond the superficial origin of the nerves, and extending nearly ¾ inch along them.

Pons and Medulla.—On the anterior surface of the pons, in its left half, are two small reddish-gray elevations, extending a little way into the substance of the pons, each about the size of half a pea: the one situated just to the left of the middle line, near the origin of the sixth nerves, the other about the level of origin of the left fifth nerve, half-way between it and the middle line. All the nerve roots, except the third, appear normal. Posteriorly the membranes covering the back of the medulla, from the point of the calamus downwards, are thickened, granular, and adherent.

Spinal Cord.—On opening the bony canal, nothing abnormal; on opening the dura mater its inner surface is seen to be studded with warty excrescences, flattened, reddish-gray, and soft-looking, size of half a pea and smaller; there is no effusion, and the membrane, except in the immediate neighbourhood of the tumours, is smooth; the tumours do not extend below the cervical region, and are limited to the posterior aspect of the cord. The arachnoid and pia mater seem thickened, and cut harshly. The cord itself is swollen and soft. In the dorsal region the arachnoid and pia still seem hard; in the lumbar region normal.

Right Lung.—Lower lobe congested; upper lobe adherent to the chest wall, containing near its apex an old quiescent cavity, about the diameter of a threepenny piece, filled with caseous putty-like material.

All the other viscera quite normal.

I have made a microscopical examination of the diseased tissues, with the following results—

Third Nerves.—Sections through the thickest part of the

swelling consist of small cells or nuclei, aggregated together without definite stroma, staining deeply. Examined in glycerine under a high power, they are seen to be irregularly rounded, granular, without definite nucleus, having the size and appearance of lymph corpuscles. The tissue is permeated by numerous pervious blood-vessels, whose walls seem to be formed simply by an aggregation of the cells. No part of the section is free from disease, though in some places the cells are less thickly distributed, and here a very few nerve fibres are visible. Sections made where the nerve is regaining its normal size, show much less of the cell infiltration, and further, that it affects principally the periphery of the nerve. Thus the periphery, consisting principally of cells, takes up logwood or carmine: the central part, where a large proportion of healthy nerve fibres are seen, takes up osmic acid.

Basilar Artery.—Transverse sections, not passing through the nodules mentioned above, show that the adventitia is normal for the most part, though in some places infiltrated by the small round cells: the muscular coat is quite normal; the elastic layer of the intima (fenestrated coat of Henle) is well marked and normal. Between this and the endothelium intervenes a layer of new formation, attaining perhaps twice the thickness of the external coats, and consisting of a fibrillated ground substance, interspersed with numerous nuclei; many of these nuclei are spindle-shaped, others rounded (possibly spindle-shaped, but seen in transverse section): they stain less deeply, and are more irregular in shape and distribution than the nuclei of the muscular tissue in the media. In the most internal part of this layer rounded and more deeply staining nuclei become more abundant, till it merges into the endothelium. The lumen of the artery remains of a very fair size.

Sections involving a nodule have a different appearance. The nodule projects from the outer part of the adventitia with which it blends, and in which it sends out prolongations extending round the vessel, so that the disposition of the disease is in the shape of a signet ring. The structure is exactly that of the diseased part of the third nerve. The media and intima are normal.

The first of these descriptions tallies with the account given by Heubner[1] of syphilitic arterial disease (viz. a spindle-celled growth in the tunica intima between the fenestrated membrane of Henle and the endothelial layer; originating in his view from the endothelium). The second implies a different lesion, viz. an inflammation or growth limited to the adventitia.

Cervical Dura Mater.—The warty excrescences have just the

[1] 'Die Luetische Erkrankungen der Hirn-Arterien.' Ch. iii. and fig. 1 of the illustrations.

same structure as that described in connection with the third nerve, except that the blood-channels are less abundant, and a fibrillar stroma is visible in some parts, especially at the base of the tumour, where it blends with the normal fibrous structure of the dura mater. The tumour sends out spurs along the inner surface of the membrane, which correspond with the roughenings noted post-mortem.

Cord and Pia Mater.—Throughout the cervical region, there is slight infiltration of the pia mater in its whole circumference. The periphery of the cord, in its posterior parts especially, is unduly vascular. In the upper cervical region patches of infiltration extend from the pia mater into the cord in the region of the posterior median fissure and of one posterior nerve root. Again, opposite the sixth cervical nerve there is a patch the size of a pea, situated at the mouth of the posterior median fissure, and sending prolongations into it along the coats of a vessel. The central parts of the cord easily fall away from the sections.

In the upper dorsal region, the cord between the posterior nerve roots was unduly vascular; in the lower dorsal region, the white matter round the central gray portion was opaque, structureless and ill-stained; and there was a patch of infiltration extending from the pia up to one posterior cornu. In the lumbar region again there is a similar, but even larger patch, though the rest of the cord is here healthy.

To summarise: the lesions in the spinal cord were limited to, or originated from, the pia mater, and affected principally, and in the lower parts of the cord entirely, its posterior aspect; lastly, there was no systematic degeneration.

Pons and Medulla.—Of the two superficial tumours, that near the sixth nerve had the structure already detailed. I could not decide whether it interfered with the sixth nerves within the medulla, but externally they seemed healthy. A third still smaller superficial tumour lay just behind one of the olives, it differed in that it contained, in the centre of the cell growth, dimly staining fibrillated tissue; i.e. it approached more nearly to the ordinary type of a gumma.

At the point of the calamus scriptorius there was a patch of disease, distinct but very limited in extent, and not involving the adjacent nuclei. No other disease was found in the medulla or pons, and it was specially noted that the oculo-motor nucleus was normal.

The diagnosis made during life, and sustained post-mortem, was syphilitic disease of the cerebral arteries. It was based chiefly on the peculiar semi-comatose state of the patient and the history of transient hemiplegia. The absence of thrombosis does not preclude us from referring these symptoms

to the arterial disease; for, as Heubner observes, the infiltration of the intima deprives the arteries of their elasticity, and thereby interferes with the cerebral circulation apart from actual obstruction. The immediate cause of death must be sought, I imagine, in the cervical region of the cord where the pia mater was most uniformly infiltrated, and the cord swollen and soft.

There can be little doubt that the disease was syphilitic. The wide distribution of it and the symmetrical invasion of the third nerve (putting aside the arterial disease) would scarcely be found in a simple inflammation. Looking to the character of the cells, and to the fact that in the third nerve and pia mater the disease was diffuse, it would perhaps be better called a syphilitic inflammation than a new growth; but in the dura mater and the pons there were distinctly isolated tumours, one of which at least was becoming fibrous in the centre; which indicates that the formation of typical gummata might have followed.

As regards the localities of the lesions there are some points of interest: First the symmetrical character of the lesion in the third nerves, to which I shall allude presently. Secondly, with regard to the cord and its envelopes: Though in the cervical region the pia mater was to a certain extent thickened all round, yet both here and down to the lumbar region definite patches of infiltration, extending from it into the cord, were found only on the posterior aspect of the cord (chiefly involving the posterior nerve roots and the posterior median fissure). Now although there was no systematic sclerosis, it seems probable that, had the patient lived, secondary ascending degeneration of the posterior columns would have set in. The possibility of a locomotor ataxy thus originating from a syphilitic meningitis is a matter of some interest.

Considerations of interest are also raised by the lesion of the third nerves in connection with the loss of the upward movement of the eyes. Paralyses in general may be roughly divided into three classes according as the lesion is situated—(1) on the nerve after it leaves the cord or medulla; (2) in the lowest (spinal or medullary) nucleus of the nerve; (3) in some higher (mostly cerebral) centre. In the first class of cases the paralysis follows the anatomical distribution of the nerve; the second class, as a rule[1], differs little in this respect from the first; in the third class the paralysis need not follow the distribution of any one nerve, but may affect movements presided over by individual branches of one or more nerves—

[1] For exceptions see Buzzard, 'Brain,' Vol. V., 'On Ophthalmo-plegia Externa in Conjunction with Tabes dorsalis;' and Sturge, 'Transactions of Ophthalmological Society,' vol. i. p. 176.

movements connected by a functional rather than by an anatomical association. In no case is the functional association of movements better exemplified than in that of the eyes, and for the classification of paralyses affecting these movements I would refer to a paper by Dr. Sturge.[1]

Now, seeing that in the present case the paralysis of the eyeballs was symmetrical, and that it affected the upward movement chiefly,[2] it was not unnatural to diagnose a central lesion. I remember to have seen, when casualty physician to St. Bartholomew's Hospital, a woman with fits of an uncertain nature, who could not raise her eyes above the horizontal plane, the other movements of the eyes and eyelids being perfect. Dr. Gowers[3] showed to the Ophthalmological Society a woman with ocular paralysis, limited to the upward movement of the eyes, associated with optic neuritis and other symptoms of intracranial disease, in which case he suggested a cerebellar lesion. Nevertheless, in the case now under consideration, the lesion turned out to be peripheral; symmetrical indeed, and therefore explaining the bilateral character of the paralysis, but leaving in some doubt the question why the upward movement of the eyes should have been so particularly affected. From the fact that the disease seemed to spread along the peripheral parts of the nerve, it might be surmised that the fibres, which go to form the branch to the levator palpebræ and superior rectus,[4] run in the periphery of the nerve trunk; but I do not insist upon this supposition, seeing that at the thickest part of the enlargement of the nerve there were very few fibres left at all. It seems better to fall back upon the analogy of paralyses in another organ, viz. the larynx. Dr. Felix Semon has shown[5] that here, whether the seat of the lesion be central or peripheral, the *abductor* fibres of the recurrent laryngeal nerve are most likely to be affected. Why this should be so, when the lesion is upon the nerve trunk, does not appear easy of explanation. Dr. Ferrier[6] surmises that in general "the extensor and abductor nerves and muscles have less vital resistance, and are sooner exhausted than the flexors," and that "a generally enfeebling cause will show itself first in the extensors." He instances the paralysis

[1] 'Ophthalmological Society's Transactions,' vol. i., "Two Cases of Simultaneous Paralysis of both Third Nerves."

[2] Originally there had been ptosis of the left eyelid, sufficiently marked to attract his wife's attention. The droop of the eyelids when he came to the hospital was less marked than the downward direction of the eyes.

[3] 'Ophthalmological Society's Transactions,' vol. i. p. 117.

[4] Unfortunately the subdivisions of the third nerves were not specially dissected out. But the nerves had regained their normal size and appearance before entering the orbit.

[5] 'Archives of Laryngology,' vol. ii. No. 3.

[6] 'Brain,' Vol. IV. p. 311, "The Localisation of Atrophic Paralyses."

of the extensors in lead poisoning, and the experiments of Onimus upon the electro-irritability of muscles post-mortem. But whatever the explanation, the present case shows, I think, that a lesion of the third nerve trunk so complete as to leave (where at its worst) very few nerve fibres visible, may be expressed by a paralysis of the upward movement of the eyes, or at most by that and an incomplete ptosis.

TWO CASES OF PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

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THE elder patient, E. P., is the second child, and the younger patient, G. P., the fourth, in a family of eight. Except a baby, a few weeks old, they are the only males. The sisters are all healthy. The parents are both 36 years of age, and were married at 19. The father is healthy, and is not aware of any similar affection ever occurring in any relative on his side. The mother is also healthy, but she had a brother, the eldest male of the family, who is said to have been affected in the same way as the patients, and died at 17.

E. is stated to have been a fat and heavy child. He cut his teeth late, and did not walk until he was eighteen months old. At the age of twelve months he began to lose power in his right upper limb, and at two years, although he had been urged to use it by the expedient of tying up the other hand, this limb was noticed to be smaller than its fellow. When he came to be taught writing he had to use his left hand for the purpose, and he has written with his left hand ever since. He had a "swinging" gait from the first, and could never run. At 5 he began to tumble about, and was so unsteady in his walking that, as his father expressed it, "a puff of wind would blow him down." When down he could not get up again without some help; this given, he would assist himself by putting his hands on his knees. In going upstairs he would crawl if without banister or other object to hold by, and in descending he would slip himself down on his buttocks. At 7 he could not bring his heels to the ground. Until 9 he walked on his toes, and since then he has been unable to walk at all. At no period had his calves attracted attention. Except a mild attack of scarlet fever twelve months ago, he had had no acute illnesses.

G. walked at fourteen months, and he could walk and run well until four years old. He then began to "waddle and turn

his toes in," and he soon showed the same peculiarities of gait and locomotion that his brother had done. At 7 his calves were noticed to be large and "on one side." He is said to have had about this time an attack of fever, from which he did not recover for two or three months; but it was without any apparent influence upon the course of the paralytic affection. After having been for a while in a Cottage Hospital near his home, he was admitted an in-patient into the Bath Royal United Hospital on August 10th, 1880.

The description of G. P.'s state at this period was to this effect: He was an intelligent, healthy-looking boy, 9 years of age, 4 ft. in height, 3 st. 12 lbs. in weight; was only now cutting his upper central incisor teeth. He walked with a peculiar rolling gait, moving his body from side to side, with his shoulders and head carried back and legs apart, as if to maintain his equilibrium. On stripping him it was observed that he had an incurvation of the lumbar spine, which disappeared in the sitting posture. His calves were enlarged, measuring 9¾ inches each in circumference, and they were remarkably firm, especially the right one. He had difficulty in bringing his heels down to the ground. When placed on his face on the floor and requested to get up, he raised himself first on his elbows and knees, then, with both his hands, pushed his body backwards, extending his knees until it was supported by his hands and feet. He then placed one hand on the one knee and the other on the other, and gradually worked his hands up to his hips, and thus he at last gained the upright posture. Besides the sural muscles the vasti externi were enlarged, the glutæi were prominent, and the erectors of the spine stood out. On the other hand, the latissimus dorsi and serratus muscles were atrophied, causing apparent projection of the inferior angles of the scapula, so also was the sterno-costal portion of the pectoralis major. The deltoid muscles were not appreciably wasted, but all the other muscles of the arm were slightly so. The muscles of the forearm were not affected. His sensation was nowhere impaired, but to the tap on his patellar tendon there was no responsive jerk.

E. P. was admitted an in-patient a month later. He was 14 years of age, about 5 feet in height, and 4 st. 8 lbs. in weight. He had the look in his face of robust health, and was of fair intelligence, but was so helpless that he could not raise himself in bed, nor when set up could he maintain the sitting posture without support. His dorso-lumbar spine in this posture was observed to be distinctly curved, the convexity being backwards, and there was in addition a slight lateral deviation of it to the right. Excepting the erectores spinæ, which projected somewhat below, all the muscles of the

back were seen to be in a more or less advanced state of atrophy, and on the right side particularly; the upper dorsal region was remarkably flattened, and the transverse processes of the vertebræ could be readily felt through the integuments. The weakness of the neck muscles was such that his head constantly tended to fall forwards. The latissimus dorsi, as apparent from the thinness of the posterior fold of the axilla, was all but gone, and the anterior fold and front of the chest bore similar testimony concerning the pectoral muscles. The sterno-mastoids were of fair size, but their clavicular portions were indistinct. The masseters were enlarged. The muscles of the abdomen seemed to be normal. His thoracic respiratory movements were diminished, his abdominal increased. The deltoid on the right side appeared to have gone, the head of the humerus being seen and felt as though quite subcutaneous. On the left side the shoulder was slightly more rounded, and the posterior fasciculus of the deltoid was distinct. Notwithstanding this difference he could elevate the right upper limb a very little, but the left not at all. The triceps of the right arm was much atrophied, and the biceps, which itself was thin, was somewhat contracted, so that the elbow could not be straightened. The extensors and supinators of the right hand and wrist were wasted, and the power of movement of the same was correspondingly impaired. Of the left upper limb the triceps was not so attenuated, and the muscles of the hand and forearm were in rather better condition. The right hand was smaller than the left, but there was no shortening of the right humerus or bones of forearm. As before mentioned, he was left-handed; but he did knitting well, and this was his chief pastime; his penmanship was indifferent. He could just extend in the slightest degree his left leg, otherwise he was almost totally unable to move his lower limbs. The thighs and legs were fixed in flexion, the left thigh, in his usual posture, being adducted towards its fellow, and the feet were contracted in talipes equino-varus with prominence of astragalus. The quadriceps extensor muscles of both limbs were much atrophied, but the adductors of right showed fair size. The glutæi were moderately plump. The hamstrings and Achillis tendons were in tense contraction. The calves measured 10 inches each in circumference. Irritability to faradisation was more or less impaired in the affected muscles, also voltaic irritability, except in the biceps of the right arm, where it was increased, contraction taking place first at the positive pole on closing the circuit. The patient's sphincters were unaffected, so also was his sensibility, whether general or special. He had some tendency to mottled appearance of his arms and legs.

The two lads remained in hospital between four and five months, but their condition varied so little that there was really nothing of sufficient importance to record. They took phosphorus in pill for some six weeks of the time, but in neither one case nor the other was there any manifest result. They came again under observation, indeed, were readmitted into hospital, in July 1881. G. had gained two inches of stature, but he was now distinctly unable to bring his heels down to the ground; he was only 3 st. 11 lbs. in weight. E. also had lost some weight, being only 4 st. 7 lbs. It was at once remarked of the latter that he sat up better. They now took cod-liver oil, and, while the general condition of both improved, that of E. seemed especially to do so; his skin became sleek and more free from mottling on his limbs, and he developed signs of puberty. By November 15th, when they were again discharged, some noteworthy changes had occurred. G.'s weight was now 4 st. 6 lbs., his right calf measured $11\frac{3}{8}$, and his left $11\frac{1}{4}$ inches; his inability to bring his heels down to the ground was such that he walked tiptoe; he could not now get up from the floor without help; his erector spinæ muscles had become indifferent in size; he had now decided wasting of his deltoids, and the muscles of his forearms were affected to a slight amount. E.'s weight at this date was 5 st. 3 lbs., his height being still about 5 ft. (he could be measured only part by part); he could now in some degree maintain the sitting posture without support; his head did not tend so much to fall forward, though if it only slightly lost the position in which he kept it he could not prevent its dropping; on the other hand, he could not now elevate either upper limb, and the biceps of his right arm was more contracted, also he could no longer extend his left leg, but was quite powerless in both his lower limbs; his calves measured right 10, and left $10\frac{1}{4}$ inches. E.'s photographs were taken at this time, but G.'s at the period of his first admission.

P.S. March 1882.—I am informed that G. can now no longer walk. The male child last born is sixteen months old, but as yet presents no evidence of being similarly affected. I hope to have the opportunity of seeing these cases from time to time, and will take the liberty of supplementing the foregoing record should occasion arise.

Abstracts of British and Foreign Journals.

Monakow on Excision of the Cortex Cerebri in new-born Rabbits.—Monakow, in the *Archiv f. Psych.* vol. xii. p. 141 and p. 535, gives the results of some experiments he made with the view of determining the relations between different parts of the cerebral cortex and the basal ganglia. He followed Gudden's method, excising circumscribed portions of the cortex in new-born animals, and carefully noting the resulting atrophy in the adult animals.

Gudden had already shown that the removal of a hemisphere in new-born rabbits and dogs caused arrest of development in the optic thalamus and the corpora geniculata externa and interna, while the corpus striatum remained unaffected. Monakow's experiments confirm these results, and demonstrate further that extirpation of circumscribed portions of the cortex—and it matters not whether these portions have a motor or a sensory significance—is followed by localised atrophies of the nuclei of the optic thalamus and the adjoining parts. Thus we have a zone of the cortex, destruction of which causes atrophy of the corpus geniculatum externum, and it may, for convenience sake, be called the zone of the corpus geniculatum externum. In the same way we have the zone of the corpus geniculatum internum, the zone of the external nucleus of the optic thalamus, the zone of the stratum reticulatum (Gitterschicht), the zone of the tuberculum anterius, or superior nucleus, and the zone of the internal nucleus. We shall now indicate the position of these cortical zones, and shall give more fully the results that follow their excision.

The *zone of the corpus geniculatum externum* (A in the figs.) occupies the postero-superior part of the cerebrum. It is a large area, forming almost a third part of the hemisphere. Removal of it caused atrophy of the posterior third of the internal capsule, the corpus genic. ext., the lateral layer of the optic thalamus, the optic tract, and in a slight degree the tractus pedunc. transv., the anterior tubercle of the corp. quadrig., and the optic nerve.

This area corresponds with Munk's visual sphere. The *zone of the corp. genic. int.* (B) is situated below the former, and occupies the postero-inferior portion of the hemisphere. There is reason to believe that it corresponds with Munk's auditory sphere, in which case the corp. genic. int. would bear the same relation to the auditory nerve that the corp. genic. extern. does to the optic nerve. Extirpation of it caused atrophy of the inferior and posterior portion of the internal capsule, the corp. genic. int. and in a less measure the posterior portion of the stratum reticulatum. The *zone of the external nucleus* (a) lies in front of A. Destruction of it caused atrophy of the third fifth of the internal capsule, the external nucleus, the outer portion of the crusta, and to a less extent the laminæ springing from the external nucleus, the formatio reticularis, the middle cerebellar peduncle, and the corp. trapez. The *zone of the stratum reticulatum*, or Gitterschicht (b), is on the outer side of the previous zone. Extirpation of it caused atrophy of the third fifth of the internal capsule, a part of the lateral portion of the crusta, the anterior portion of the stratum reticulatum, and to a less degree the posterior portion with its continuation into the tegmentum. The animals in which this zone was removed did not live longer than four weeks. Monakow is unable to assign the functions of zones a and b, though he surmises that they are sensory. The *zone of the tuberculum anterius* (c) is situated in front of zone a. Extirpation of it caused atrophy of the tuberc. ant., the anterior portion of the internal capsule, a part of the pyramidal tract, and a less considerable atrophy of the laminæ med. of the anterior tubercle, and the bundle of Vicq d'Azyr. The *zone of the internal nucleus* (e and f) occupies the most anterior portion of the brain, and is bounded posteriorly by zone c. Extirpation of it causes atrophy of the most anterior portion of the internal capsule, and partial atrophy of the pyramidal tract and the internal nucleus. The zones e and f have a motor function. External to zone c is a zone d, extirpation of which causes atrophy of the anterior portion of the internal capsule, and of a tract that runs direct to the curs. and, in a less marked degree, of the anterior stratum reticulatum. The direct tract, according to Monakow, contains fibres of the facial and hypoglossal nerves.

The atrophies observed in the above experiments may be classed under three heads, atrophy of fibres leading to the nuclei of the optic thalamus and of these nuclei themselves, atrophy of fibres leading direct to the periphery, and atrophy of fibres arising from the implicated nuclei and passing downwards to the tegmentum.

Strümpell on the Pathological Anatomy of Tabes dorsalis.—Strümpell, in a lengthy paper in the *Archiv f. Psych.* vol. xii. pp. 723–771, discusses the pathology of tabes dorsalis, and comes to the conclusion that it is a combined systematic disease. We shall first give an abstract of the cases he reports, noticing any special points of interest that present themselves, and shall then briefly analyse his remarks upon the general pathology of the disease.

Case I.—A woman, aged 43, while suffering from tabes in its earliest stage—the symptoms being lancinating pains in the limbs, absent patellar reflex, and spinal myosis—was cut down by an attack of typhoid fever. At the necropsy the meninges of the cord were seen to be perfectly healthy, a fact which should be a death-blow to the theory that tabes has its origin in a chronic meningitis. Microscopic examination showed in the *cervical* region a small strip of degeneration to the outer side of Goll's column, and another strip to the inner side of the posterior cornu, where the posterior roots enter the cornu. There was also in the lower sections slight degeneration of the anterior part of Goll's column, the degeneration not extending quite up to the posterior commissure. The cord gradually assumed a healthy appearance as the medulla oblongata was approached. In the *upper dorsal* region there was a thin strip of degeneration bordering the posterior fissure, and another strip, similar to that described in the cervical region, but broader, to the outer side of Goll's column. In the *lower dorsal and upper lumbar* regions these two strips gradually broadened and approximated, and ultimately formed one large patch of degeneration occupying the middle and anterior parts of the posterior column. The posterior part of the column was quite normal, as was also a small zone to the inner side of the anterior portion of the posterior cornu. The degenerative changes were most marked in the upper part of the lumbar enlargement. In the *lower lumbar* region the degenerated area diminished, and was found chiefly in the middle portion of the column, being bounded anteriorly and posteriorly by an intact region. There was a small zone of healthy tissue in the middle line in the lower sections, which, with its fellow on the opposite side, formed an oval-shaped area. It is of interest as corresponding to a group of fibres, which Flechsig's developmental researches enabled him to differentiate. It is important to mention that in the upper part of the cord the lateral limiting layer (seitliche Grenzschicht) was not quite normal. The changes we have described were found in both posterior columns, and were remarkably regular and symmetrical.

Strümpell compares this case with a case of incipient tabes reported by Westphal, and points out how closely the localisation of the degenerated areas agrees in the two cases. And he contrasts it with a case of combined systematic disease (See 'BRAIN,' Vol. III. p. 563), in which the areas of the posterior columns that remained intact are the very areas that are degenerated in these cases of tabes.

The author attributes the absence of the patellar reflex, and also the lancinating pains, to the lesion of the middle portions of the posterior columns in the lumbar region; for, on the one hand, cases have been reported in which there was lesion of the posterior portion of the column without loss of the patellar reflex, and, on the other hand, lesion of the anterior portion is rather rare in tabes. Strümpell concludes his remarks on this case by observing how inaccurate it is to say, as is often said, that 'Tabes commences in the outer portions of the posterior columns.'

Case II.—This case was somewhat more advanced than the first, but was still in an initial stage. The chief symptoms were lancinating pains, absent patellar reflex, vesical weakness, and a degree of ataxia of the limbs. The anatomical changes presented a striking resemblance to those just described. In the lumbar region, for example, the main area of degeneration was in the middle portion of the posterior column, the anterior portion was intact, the posterior portion only slightly degenerated. In the dorsal region there were the same medial and lateral strips of degeneration, but in addition there was a degeneration of the posterior part of Goll's column, which extended upwards to the cervical region. The degeneration of Goll's column was more marked in the upper dorsal and cervical portions of the cord than in the lumbar region, a fact not easy to explain if we hold that the degeneration of Goll's column in tabes is a secondary degeneration. It is far more likely that the degeneration is primary.

Case III.—This case is remarkable for the almost apoplectic suddenness of the appearance of ataxia. The patient improved under galvanisation, and for two or three years was almost free from ataxia. Subsequently mental symptoms showed themselves, and the patient died of dementia paralytica. Strümpell comments on the unusual sequence of the diseases in this case. As a rule, it is tabes that occurs in the course of a general paralysis; here the order was reversed. The degenerative changes were similar to those described above, but more advanced. The postero-external field of the posterior column was intact in the cervical and the greater part of the dorsal region; so also was the anterior

portion of the posterior column in the lumbar region, with the exception of a small strip in the middle line. Both these conditions Strümpell regards as characteristic of tabes.

Case IV.—Here the disease was still more advanced. There was degeneration over the whole of the posterior columns in the dorsal region, but the changes were less marked in the outer part of Goll's column, and in the postero-external field of Burdach's column than elsewhere. These regions, it will be remembered, were intact in the initial stages.

Case V.—This was an advanced case of the disease. In the dorsal region the posterior columns were, with the exception of some fibres immediately behind the posterior commissure, completely degenerated. In the cervical and lumbar regions, there were intact areas in the antero-lateral and anterior portions of the posterior columns, respectively. The postero-external field was degenerated, but only slightly; in this respect contrasting with the remaining portions of the column.

Case VI.—This case presents a beautiful example, in the knee and hip, of the so-called tabic arthropathies. Anatomically there was simply an arthritis and an ostitis deformans. Strümpell thinks the anæsthesia of the joints and the disorderly movements are important factors in the causation of the disease. The anterior cornua were healthy. The localisation of the degeneration in the lumbar region agreed with that described in the previous cases; in the dorsal and cervical regions some differences were noticed. In the former there was a triangular zone of intact tissue in front of the posterior root-zone, in the latter Goll's column was in an advanced state of degeneration, and the posterior parts of Burdach's column were diseased.

Case VII. resembles the previous cases so closely that it is unnecessary to describe it. *Case VIII.* was remarkable on account of the degeneration of the most anterior portions of the posterior columns, the almost universal degeneration of Goll's column, and the normal condition of the remaining parts. There is some doubt, however, whether this was rightly considered a case of tabes. In *Case IX.* there was only slight degeneration of the middle portions of the posterior columns in the lumbar region. In the dorsal region the only lesion was a thin strip of degeneration on the inner side of the posterior cornu. In the cervical region there was an incipient degeneration on the outer side of Goll's column. In this case there had been the characteristic neuralgias of tabes for twenty-five years, and yet the anatomical changes were comparatively slight. The

last case was a case of tabes complicated with paralysis (see 'BRAIN,' Vol. III. p. 566). This and Case I. are the only cases of tabes in which Strümpell has found degeneration of the lateral as well as the posterior columns.

In the second part of the paper, the author examines shortly the various views that have been taken of the pathology of tabes. That the changes in the cord are secondary to a chronic meningitis, Case I. disproves. Strümpell protests against the custom of attributing the increase in the connective tissue of the pia mater in tabes to a chronic meningitis; it is really a result of the atrophy of the cord. Another view is that tabes originates in a disease of the posterior roots, whence the degeneration spreads upwards to the cord. This view is both true and false. It is true in so far as it recognises the fact that the posterior roots are always implicated in tabes, generally even in the earliest stage. It errs in regarding the posterior roots as the only starting-point of the disease. The fibres of the posterior roots down to the spinal ganglia and the fibres in the posterior columns are really the same fibres, and the atrophy is equally in each case a primary atrophy.

A third view is that tabes is a chronic degeneration of the connective tissue, which advances along the course of the arterial supply. We should therefore expect to find the degenerative changes most marked where the connective tissue sends septa into the posterior column, which, however, as Strümpell points out, is in many instances not the case. A fourth view, which we need only mention, is that tabes is essentially a brain disease, and that the changes in the cord are secondary to it. According to a fifth view, tabes has its origin in the peripheral nerves. This theory has been propounded in consequence of the effect of nerve stretching in tabes.

These various theories being set aside, there remain two which compete with one another for our approval; viz. (1) that tabes is a chronic myelitis of the posterior columns, affecting primarily the connective tissue, and spreading like any other inflammation; and (2) that tabes is a systematic disease, a primary degenerative atrophy of the nerve fibres that spreads in directions that are determined by the physiological function and anatomical distribution of the fibres first attacked.

Strümpell adopts the latter view, and argues that both the clinical and pathological facts are in its favour. Clinically, tabes is characterised no less by the peculiar combination of a number of nervous symptoms than by the almost constant absence of a whole

series of symptoms, of frequent occurrence in the most diverse nervous diseases (e.g. paralysis, muscular atrophies, changes in the electrical irritability, spastic phenomena). These two facts bespeak something more than a mere hap-hazard distribution of the causal lesion. The clinical postulate is satisfied by the theory of a systematic disease, but not by a chronic myelitis.

The pathological facts that Strümpell advances in favour of the view that tabes is a systematic disease are the following:—(1) The strict limitation of the disease to the posterior columns. Other authorities, for instance Erb, state that the morbid process often extends to the lateral columns; but this has not been Strümpell's experience. The only exceptions he has met with were Case I. (lesion of lateral limiting layer), and the last case he describes (degeneration of the pyramidal and cerebellar tracts). This latter case, however, was not a typical case of tabes. The so-called marginal degeneration (*Randdegeneration*) is mentioned in this connection, but is dismissed with the remark that its origin and significance are not yet known. (2) The remarkable symmetry of the disease in both posterior columns. The symmetry often extends to the minutest details. (3) The peculiar localisation of the degeneration in the posterior columns. There are areas which become diseased in the early stages of the malady, and there are others which rarely become diseased at all, or if they do, only in the later stages. The order of degeneration of the different areas is as follows:—In the dorsal region, first a thin strip bordering the posterior fissure, and another strip to the outer side of Goll's column, then Goll's column, the posterior earlier than the anterior parts, then the postero-external field, and lastly, the remaining portions of the column. In the lumbar region, first the middle portion of the column, then the posterior portion (a small oval-shaped area in middle line is the last part of it to degenerate), and finally, the anterior portion. In no case of undoubted tabes has Strümpell seen degeneration of this anterior portion. In the cervical region, first a thin strip in Burdach's column, then Goll's column, then the posterior root-zone, and subsequently the postero-external field, and the antero-lateral field. (4) The changes in the grey matter of the posterior cornua. In old cases of tabes there are always atrophic changes in the area between the anterior and posterior cornua. That the disease does not spread, *per continuitatem*, is shown by the fact that the cells of Clarke's columns remain normal while all around atrophies.

Tabes is not a systematic disease in which one system of fibres

only is implicated, it is a combined systematic disease, cerebral as well as spinal systems being involved. The various systems, though differing greatly in their physiological functions, have this in common, that they are affected by the same morbific influence. Toxicology presents many examples of a similar action; a very pertinent example is the action of ergot on the nervous system. Tuczek has lately shown that this drug is able to produce many of the symptoms of tabes, and in four cases microscopic examination demonstrated the existence of degeneration of the posterior columns. What the morbific influence is in tabes we know not; in some cases it is probably the syphilitic virus.

Westphal on Disease of the Posterior Columns of the Cord in General Paralysis.—Westphal, in a paper published four years ago in the *Archiv f. Psych.* (vol. viii. p. 519), stated that in every case of general paralysis in which the gait is affected, and in which there is constant absence of the patellar reflex, there will always be found degeneration of the posterior columns of the cord, extending down to the lumbar region. In a later communication (*Berl. klin. Wochenschr.* 1881, No. 1) he goes further, and asserts that this lesion may be safely predicted, even though the lower extremities show no motor or sensory symptoms, if only the patellar reflex is absent. The present paper (*Archiv f. Psych.* vol. xii. p. 772) consists of two cases which go to support this statement.

In the first case the gait was rather stiff and at times somewhat reeling, but there was no ataxia. There was a slight diminution in the sensibility of the limbs to pain. The patellar reflex was absent. Microscopical examination revealed degeneration of the posterior columns in their entire length. It is worthy of mention that in the fresh state granule-cells were found in the posterior part of the lateral columns, while after hardening in potassium bichromate and chromic acid no granule-cells were visible, and the lateral columns appeared quite normal. In the second case there were no sensory symptoms and the gait was not ataxic, but the patellar reflex was absent. There also the microscope showed degeneration of the posterior columns throughout their entire length.

Westphal observed that the degeneration was not everywhere equally intense. Thus, in the cervical region there was in both these cases an area of well-marked degeneration bordering the median fissure; outside this there was an area of very slight degeneration; outside this again an area of intense degeneration, which was bounded externally by an area of slight degeneration. At the inner

side of the posterior cornu, especially opposite its anterior portion, there was a strip of normal substance. As we pass downwards to the dorsal and lumbar regions these distinctions become less and less distinct, and we have ultimately one area of intense degeneration (situated about the middle of the column), and another of slight degeneration. These areas are perhaps to be looked upon as indications of the existence of separate systems of fibres in the posterior columns, but the solution of this question Westphal thinks must be decided primarily, not by pathologico-anatomical, but by developmental investigations.

The absence of ataxia in these cases of general paralysis with lesion of the posterior columns is thought to be due to the comparatively early death of the patient, the atrophy of the posterior columns and posterior roots not being sufficiently advanced to produce ataxia. Ataxia does not show itself unless a considerable number of the fibres of the posterior columns or roots have been destroyed.

Westphal on the Localisation of Hemianopsia and the Muscular Sense in Man.—Westphal (*Charité-Annalen*, 1882) reports a case which throws some light on this subject. The patient was a man, aged 38, who in July, 1879, suffered from delirium tremens. From this he recovered, but on Aug. 7 he became aphasic without losing consciousness. On Aug. 14 and again on Aug. 19 he was convulsed on the right side and had symptoms of the so-called atactic and sensory aphasia, but did not lose consciousness. And lastly, on Nov. 13, he was unconscious for a short time, and was temporarily paralysed in the right arm. In all of these attacks the motility of the right extremities seemed deeply impaired, but it is difficult to say how far the appearances were due to actual motor weakness, and how far to the inability to originate a suitable nervous impulse, in other words to a defect of volition. There was undoubtedly some motor weakness, but it was not permanent, and in a short time the patient always recovered his muscular strength.

But in addition to these motor symptoms there were sensory symptoms, and these were permanent. They were observed with great minuteness from Oct. 1879 to Dec. 1881 (when the patient died somewhat suddenly), and were never found to exhibit much variation. There was a marked impairment of the muscular sense of the right upper extremity. The right hand was used very clumsily, and the patient seemed unconscious of the position of the upper extremity and of the movements it made. In the right

lower extremity there was scarcely any affection of the muscular sense. Sensibility was diminished in the right side of the body and in parts of the face. There was bilateral right hemianopsia.

The *post-mortem* revealed a lesion implicating the whole of the parietal lobe (including the ascending parietal convolution), the greater part of the occipital lobe, and a small portion of the temporo-sphenoidal lobe. Here the pia was adherent to the cortex, the grey matter was softened, and of a yellow colour, and the convolutions were atrophied. The atrophy was less noticeable in the posterior part of the angular gyrus than elsewhere. The cortex only was involved. The rest of the brain was normal. This condition of the brain reminds us of general paralysis, but Westphal is of opinion that the two processes are essentially different.

Westphal attributes the hemianopsia to the lesion of the occipital convolutions. He is unable to say whether the loss of muscular sensation is dependent on the lesion of the ascending parietal convolution or on that of the remaining parietal convolutions.

Bernhardt on Sensory Symptoms and Hemianopsia in Cerebral Lesions.—Bernhardt (*Archiv f. Psych.* vol. xii. p. 780) reports several cases that have an interesting bearing on the question of the sensory functions of the cerebrum, though their value is lessened by their being as yet unchecked by *post-mortem* examination.

Case 1.—A woman, aged 53, without losing consciousness, suddenly lost power in the right half of her body. Speech was affected for a short time during the apoplectic attack. Fourteen days afterwards she was able to walk as well as ever, and ten months afterwards she was examined by Bernhardt with the following result:—The patient complained of the dimness of the vision of the right eye, and of the awkwardness of the right hand. The sensibility of the right upper extremity was decidedly diminished. Passive movements of the fingers were not perceived. The muscular sense, as tested by her ability to discriminate weights, was impaired. If small objects were placed in her hand, she could not tell what they were by simply feeling them. The limits of the visual field were normal, but there was a sector-shaped area of indistinct vision occupying the upper three-fourths of the right half of each eye. The perception of colour was impaired over this area, and very careful perimetric investigations showed a diminished acuity of vision here.

Bernhardt compares this case with a case reported by Samelsohn,

where the patient after an apoplectic seizure became hemiplegic on the right side. At the time of Samelsohn's examination the hemiplegia had almost disappeared, there was only slight paresis of the right arm and leg. There were no sensory disturbances, but there was *left* hemianopsia, the perception of colour being affected, but not the acuity of vision. A case (*Case* 2) very similar to this was reported by *B.* some years ago. There was left hemiparæsthesia and right hemianopsia. The affection of sensibility was chiefly of a subjective nature; the patient felt that his left arm and leg did not belong to him. In both these cases, there were probably lesions of each side of the brain.

Case 3.—A man, aged 50, while dressing, was suddenly conscious of a peculiar feeling in the left half of his body, particularly in his arm and face. He felt as if this half of his body was asleep or dead. He could not recognise with certainty small objects placed in his hand. Motility was unimpaired. There was complete left hemianopsia. The affection of sensibility soon disappeared, but the hemianopsia was permanent. Some months afterwards his left arm became convulsed, and Bernhardt again saw him. The hemianopsia was unchanged, and there was a feeling in the left arm "as if it had been beaten with a stick;" but with these exceptions nervous symptoms were absent. *Case* 4.—A woman, aged 45, seemed to gradually lose power in the left limbs, without being actually paralysed. Sensibility was diminished over the whole left side. Her knowledge of the position of her left extremities was much impaired. She could not recognise small objects merely by their feel. There was left hemianopsia. We should mention that there was some mental weakness, which complicates the interpretation of the case.

In all the cases we have given above there has been hemianopsia. But that this is not a necessary symptom in the class of cases we are describing the following case (*Case* 5) will show:—A man, aged 39, awoke one morning with a feeling of weakness in the right leg, and burning in the right ear. These abnormal sensations gradually extended to the right arm and the right side of the trunk. Four months later he was examined by Bernhardt, who found peculiar paræsthesiæ in the ear, face, neck, arm, and part of the trunk of the right side, ataxia of the upper extremity when it grasped at objects or was engaged in fine movements, and general impairment of tactile sensibility. There was no hemianopsia.

If we compare these cases we shall find that they closely resemble one another, and evidently belong to the same type of

disease. The course of the disease is generally as follows: A man, previously in good health, is seized with an apoplectic attack, but does not become unconscious. Any symptoms of paralysis that are present speedily disappear, but there remains a characteristic awkwardness in the use of the limbs of one side of the body, especially of the upper extremity. This awkwardness depends not on a general motor weakness, but on an impairment of the various tactile sensations,—the sensations of touch, of pressure, of muscular movement, &c. The limbs may feel as if they did not belong to the body, and the delicate touch of the fingers on which we are so dependent for our knowledge of things, is lost. The awkwardness and uncertainty of the patient are further augmented by the hemianopsia, which is a frequent symptom in these cases. The senses of hearing, taste, and smell are not involved. The sensory affections may become greatly ameliorated or even disappear, but the hemianopsia, according to Bernhardt's experience, never disappears. The patient may remain in this condition for weeks or months without exhibiting serious symptoms.

Bernhardt finds the anatomical substratum of the symptoms we have described in a lesion of the cortex cerebri and the adjacent medulla, the symptoms varying with the portions of the cerebral superficies involved. The parts most frequently involved are the tactile centre for the upper extremity, and the visual centre.

W. J. Dodds.

Homen on Secondary Degeneration of the Medulla Oblongata and Spinal Cord.—Dr. Homen (Virchow's *Archiv*, April 1882) contributes the notes of eight cases in illustration of the histological relations of this pathological condition. Full clinical history of each case is given, together with the results of microscopical examination, of which the following is a brief summary.

In the first case there had been repeated apoplectic attacks. Softening was found on both sides of the pons, with obliteration of the basilar artery and its branches. On the left side (attributable to an attack dating two years previously) the pyramidal strand was implicated. Descending degeneration was traced, with atrophy of the left pyramidal column and its continuation into the spinal cord. On the right side, likewise, a lesion, dating about five weeks, was followed by descending degeneration of the pyramid. Wasting of the axis cylinder of the nerve fibres was observable, also grey degeneration of the posterior columns of the cord.

In the second case, concurrent with an apoplectic seizure, three years before, with right hemiplegia, there was almost total grey degeneration of the left pyramid, descending into the cord with atrophy of the nerve fibres; the fillet was involved in the descending degeneration.

The third case was one of hæmorrhage into the central cerebral ganglia on the left side, three weeks before death, with right lateral palsy; there was descending degeneration of the pyramidal strands, wasting of the axis cylinders of the nerve fibres, and proliferation of nuclei in the connective.

The features of the four following cases had much in common with the preceding. The last case related by Dr. Homen was one of myelitis of the dorsal region of the spinal cord, of seven months' duration, attended with ascending and descending degeneration, atrophy of the axis cylinders, and incipient atrophy of the nerve-cells and nuclei.

With reference to these histological lesions, it was observed that in the first case, where a double lesion existed, there was an important difference in the secondary degeneration on the two sides. The first attack was twelve years before death, the second occurred ten years subsequently.

In cases of the shortest duration, after the apoplectic attack, there was already apparent a wasting of the axis cylinders, a granular degeneration of the medullary sheath, with increase of connective tissue, and proliferation of its nuclei. Those parts which have undergone degeneration are noticeable even on microscopic inspection, as they take the colouration of the Müller's solution more markedly than the adjacent healthy parts.

Dr. Homen appends the following conclusions as the results of his investigations :—

1. That the earliest degenerative changes are found in the axis cylinders, not in the medullary sheaths.

2. That degeneration of the spinal cord may be found as early as three weeks after the occurrence of the lesion in the brain, and that proliferation of nuclei will then be present.

3. That a descending degeneration of the fillet (Schleifenschicht) of the pons and medulla oblongata also occurs.

4. That, subsequently, slight atrophy of the anterior cornua of the grey matter, and some degree of degeneration of the anterior nerve roots on the affected side will supervene, but not necessarily a change in the nerve cells of the anterior horns.

Davida on the relation of Spinal Nerve Centres in a case of Perobrachia (arrested development of the forearm). (Virchow's *Archiv*, April,[1] 1882.)—A very interesting example of arrested development of the forearm and hand, which presented only a rudimentary stump, was found on the examination of the body of a railway labourer, aged 40 years.

The upper arm was of the same length as in the sound limb, and was only as much less firm as might be expected from want of use. At the upper epiphysis of the forearm was a short stump, which could be flexed or extended, as the elbow joint was normal, admitting also of the movements of pronation and supination. The stump was rounded off, smooth, and its skin thin. On the median surface of this stump was a soft supernumerary structure, which had some resemblance to a rudimentary hand, viz., five short cylindrical projections with interspaces, as with fingers—looking like the hand of a four or five months' fœtus, with embryo finger-nails.

It was, as the author observes, a point of interest to notice the relative conditions of the nerves at their centres on the two sides. This is shown in the following Table.

No difference was observable as far down as the sixth cervical nerves—below these:

	Left Side (Normal).	Right Side (Perobrachia).
Posterior Cervical—		
6th nerve contains . .	11 cords	10 cords.
7th do. „ . .	9 „	7 „
8th do. „ . .	8 „	5 „
Posterior Dorsal—		
N. 1	6 „	5 „
Anterior Cervical—		
6th nerve contains . .	8 „	7 „
7th do. „ . .	5 „	4 „
8th do. „ . .	5 „	3 „
Spinal Ganglia –	Length. Breadth.	Length. Breadth.
Cervical 6	5·5 Mm.[1] 6·0 Mm.	4·0 Mm. 5·5 Mm.
Cervical 7	5·5 „ 6·5 „	3·5 „ 4·0 „
Cervical 8	5·0 „ 6·0 „	3·0 „ 4·0 „
Dorsal 1	4·5 „ 5·5 „	3·0 „ 4·0 „
	Thickness.	
Anterior Cervical 6	4 Mm.	3·0 „
Anterior Cervical 7	4 „	3·0 „
Anterior Cervical 8	4 „	2·0 „
Anterior Dorsal 1 . .	3 „	1·0 „

Dr. Davida enumerates authors who have recorded cases of Perobrachia in both dead and living subjects, and notes that in none have the differences in the nerve and their trunks been accurately ascertained. With reference to causation the writer observes with force, that the decrease in the number and size of the nerve-roots on the side of the Perobrachia is not explained either by the action of a too short umbilical cord, nor by the influence of maternal imagination in pregnancy.

In the case now recorded the author considers that there was primarily an arrest of development of the nerve-centres, and that the perobrachia was secondary thereto—leaving still the question how the arrest of growth of either the one or the other has been occasioned. W. B. Kesteven.

Westphal on the Knee-phenomenon. (*Archiv für Psychiatrie*, vol. xii. part 3, *May*, 1882.)—Prof. Westphal relates three cases in which the knee-jerk would be produced by excitation of the skin over the patellar tendon. 1. A patient, aged 19, suffering from atrophic paralysis of the legs, suddenly showed the knee-jerk after it had been absent for some time. It was found, however, that exciting the skin produced a localised contraction of the extensors of the leg, or even of both legs. A more energetic excitation was followed by contraction in the flexors also. There was complete cutaneous anæsthesia; but the muscular excitability to direct percussion, previously present, disappeared when the skin-reflexes showed themselves.

2. Rapidly invading poliomyelitic paralysis of the four extremities. Percussion over the tendon is often followed by a contraction of the extensors; this contraction occurs at a notable interval of the excitation; the latter may be confined to the skin, which is not anæsthetic, as in the first case. It even once occurred before the blow had actually been delivered. These reactions suddenly disappeared after lasting some time.

3. Case of spondylites in the lower dorsal region. Here the ordinary knee-jerk was exaggerated. Excitation of the skin over the tendon on the left side produced a contraction also; but this contraction was longer in its production (half a second) and ran a more protracted course. Exciting the skin at the malleolus was followed by contraction of the triceps extensor only, or of all the thigh muscles, according to the strength and repetition of the excitations. On the right side similar phenomena were observed, but not so regularly. Later, the retardation in the skin-reflexes was no longer noticed.

Prof. Westphal lays stress upon the practical importance of these observations. It is evident that in testing the knee-jerk we must keep in view the possibility of being deceived by true skin-reflexes. Cases of sudden appearance of the knee-jerk have been described, which possibly will find their explanation in the way pointed out by the author.

Prof. Westphal mentions the results of some experiments made in order to discover whether the administration of strychnia to animals, whose lumbar posterior roots have been cut (and the knee-jerk thereby abolished) could restore the tonicity of the muscles upon which he believes the knee-jerk to depend. The results were negative. In an instructive instance, however, the section had accidentally not been complete; and here the knee-jerk at first abolished could be restored by strychnia. The author, with much reason, believes this result to show how delicate a mechanism is the one which regulates the knee-phenomenon; and explains how it may become such a valuable index to pathological conditions.

The rest of Professor Westphal's paper is taken up by a restatement of his views with reference to the real nature of the knee-jerk. From the beginning he interpreted it as being due to the direct excitation of muscles in a state of tonus. When the tonus is increased, the jerk is increased, and *vice versâ*. He does not lay much importance upon the arguments drawn from the short latency of the contraction to prove that it is not a spinal reflex. At the same time he reminds us of Prevost and Waller's experiments, which tend to show that the so-called crossed reflex is merely the result of the mechanical stretching of the tendons, owing to the transmission of the mechanical impulse through the femur to the pelvis.

[The Reporter, in a paper published in the *British Medical Journal* (May 10th, 1882), also expresses a doubt as to the cogency of the argument drawn from the short latency of the knee-jerk as compared with the known rates of travelling of the nerve-vibrations. He therefore has thought it desirable to compare the latencies of a true reflex contraction with that following percussion of a tendon by measurements taken upon the same individual and by the same method. For this purpose he has adopted Waller's method ("BRAIN," 1880), and recorded the results of his experiments on the latency of the plantar reflex. He shows it to be at least three times as long as that of the knee-jerk, which therefore must be looked upon as "a pseudo-reflex." The latency of the "crossed-reflex" contraction is identical with that of the direct knee-jerk—a fact which shows that both phenomena are of the

same nature. The latter is easily explained by the consideration that percussion of one knee produces a vibration transmitted to the pelvis, through the femur, so that the tendons of the opposite leg are suddenly stretched. This view is supported by Prevost and Waller's experiments (*Revue Méd. de la Suisse Romande*, June 15th, 1881), and by a simple clinical test devised by the Reporter. If one leg be held out in a state of complete extension, at a right angle to the pelvis, percussion of the heel causes a lively contraction in the opposite leg (adductors chiefly); a slight bending of the leg, which prevents the transmission of the mechanical impulse to the pelvis, at once abolishes the effect of the percussion. Reporter has also found in some cases that the knee-jerk is double, the second contraction being only brought into evidence by the myographic record. To the eye the two contractions are fused into one. Upon investigation he finds that the first contraction has the usual knee-jerk latency (·02–·03 of a second), the second contraction has the true reflex latency (·08–·1), whence he concludes that the latter is a sensory-reflex phenomenon, probably cutaneous—a view which fully coincides with Professor Westphal's results. The practical outcome of the facts just recorded is that in certain cases a true reflex contraction may occur which masks the absence or diminution of the "pseudo-reflex" knee-jerk, the latter alone, of course, constituting the valuable clinical test known as "the tendon-reflex."—A. DE WATTEVILLE.]

Cerebral Localisation.—Cha. Leegaard (*Nordiskt Medicinisk Arkiv;* Trettonde Bandet, Tredje Häftet, 1881) reports a case of embolic softening of the cortex of the right hemisphere, in which the symptoms were—first, a sudden attack of paralysis of the left side of the face, with slight embarrassment of speech, paresis of the left arm, and almost inappreciable weakness of the left leg. The paresis of the left arm and leg disappeared in eight days, that of the left side of face and tongue continuing. A week afterwards the left arm and leg became paralysed. A few days afterwards the paralysis of the leg disappeared, that of the face and arm continuing. Death occurred five weeks after the first seizure.

After death a yellow softening was found occuping the lower half of the ascending frontal and parietal convolutions, and the base of the third frontal convolution of the right hemisphere. The softening was limited to the cortex, and did not invade the centrum ovale. Besides this there was a minute focus of softening, the size of a pea, in the caudate nucleus of the right side.

D. FERRIER.

BRAIN.

OCTOBER, 1882.

Original Articles.

FOLIE À DOUBLE FORME
(ALTERNATING INSANITY).

BY DR. ACHILLE FOVILLE.

FOLIE à double forme, Alternating Insanity, is the name proposed to be introduced into medico-psychological science for one of the latest subdivisions of mental disease. This designation appears to have been accepted by the majority of Alienists at the present time, and it may be said that Folie à double forme has acquired a definite right of citizenship in the medico-psychological domain. Two recent facts demonstrate this. The Academy of Medicine of Paris has opened a competition on the following subject for the yearly prize founded by Dr. Falret, to be awarded in 1880—"On Insanity under the names of Folie Circulaire, Folie à double forme, and Folie à formes alternes." In 1880 the English Committee in charge of the arrangement of the programme of the Section of Mental Diseases of the International Medical Congress held in London in August 1881, enrolled among the clinical questions proposed for discussion that of Folie à double forme. It will be noticed that it was under the French name that this disease was entered, both in the English and German programmes—a proof that it was in France that it had been studied and described as a new malady, as an independent morbid entity.

It was, doubtless, because too many subjects had to be taken up in too brief a time that no communication with reference to Folie à double forme was brought before the London Congress. That part of the programme fell through entirely.

We have thought that it might be to the point to fill up this blank by here presenting a *résumé* of the present state of French science on this interesting question. We do not offer this as an original article, and lay no claim to having discovered Folie à double forme, or to the formation of the theories in reference to it; but, taught by the first publications of our masters of mental disease, we have always studied the subject with predilection. By experience and personal examination of a great number of patients we have arrived at the conclusion that Folie à double forme is a very distinct and natural (*naturelle*) kind of insanity. On this account it is a pleasure to us to summarise our knowledge by completing and developing what we have already done in regard to this disease in the new Dictionary of Medicine and Practical Surgery.[1]

Quite recently, August 2, 1881, the Academy of Medicine decided on the Report of Dr. Luys to award the prize for 1880 to Dr. Ritti, one of the physicians of the Charenton Asylum. Our esteemed colleague will soon publish the result of his labours; but he has, in the meantime, kindly placed at our service his interesting MS., so that we have been able to review the most recent work in connection with this subject, and hope to omit nothing.

Historical.—The constitution of Folie à double forme is a contemporary fact; and it is all the more necessary to trace its history carefully, as it has been the occasion of sufficiently violent personal discussions, or rather of urgent claims of priority, between two French Alienists, who have both acquired a celebrity justified by important original work, and by numerous publications. They are—Dr. Falret senior, who died about twelve years ago; and Dr. Baillarger, one of the present veterans of psychology in France. Both have been for a number of years physicians to the Salpêtrière, and as a result of studies prosecuted by each on this common ground, they de-

[1] 'Dictionnaire de Médecine et Chirurgie pratique,' lxv. pp. 322, 187.

scribed almost at the same period and in very analogous terms this new disease. Hence the dates have acquired great importance, and must be quoted not only by years, but even by months and days.

It is not the case that no one, before the above-mentioned physicians, has within recent times stated the existence in the same patient of symptoms of maniacal excitement and of melancholic depression. The succession and, to a certain degree, the mixture of those two morbid conditions are facts so frequent in many cases of insanity, that all the physicians who have described mental diseases have more or less alluded to such states. Several had even indicated, in passing, diseases in which those two classes of symptoms habitually succeeded one another, but without insisting on the successive alternation of opposite phenomena, and without attributing to it special theoretical importance. The work of M. Ritti contains all the necessary bibliographical references in connection with this question.

A more precise opinion, the first that may be considered as really significant, is found in Griesinger's 'Treatise on Mental Disease,' published in Germany in 1847. We there read the following passage: "The transition from melancholia to mania and the alternations of these two forms are very common. It is not rare to see the whole disease consisting of a cycle of these two states, which often alternate very regularly."

Then occurs the first French notice of this disease. It is found, in embryo, in a clinical lecture by Dr. Falret, published in the 'Gazette des Hôpitaux' for 1851, and afterwards in the same lecture republished with further details in 1854, of which the following is the most salient passage: "The transformation of mania into melancholia and of melancholia into mania has been hitherto pointed out as an accidental fact; but it has not been sufficiently observed, or at least it has not been stated expressly, that there exists a certain class of lunatics in whom this alternation is manifested continuously and in an almost regular manner. This fact has appeared to us important enough to serve as a basis for a particular form of mental disease that we have named *Folie circulaire*. For a patient so affected spends his existence in

an unchanging circle of morbid conditions, that are continually reproduced, as it were inevitably, and are only separated by lucid intervals of rather short duration."

It will be noticed that, in this passage, Falret speaks of a particular form of mental disease, which he calls *Folie circulaire*: but he only mentions it incidentally whilst treating of the course of insanity. He does not think of assigning to it a special place as a distinct morbid entity in the classification of the various species of mental diseases. It is, moreover, always when treating of the progress of insanity that we find the same paragraph in the volume in which Falret senior collected in 1864 his various antecedent writings under the title of "Maladies mentales en Asiles d'Aliénés."

This indication of a new variety of mental disease, "laid down confusedly in the columns of a periodical" (Morel), had not drawn the attention of the medical faculty; so an original memoir, read on the 31st of January, 1854, before the Academy of Medicine in Paris by M. Baillarger, was received as a novelty. It was delivered under the title of a "Note on a kind of Insanity, the attacks of which are characterised by two regular periods, one of depression, the other of excitement."[1]

The following are the essential terms of this paper, in which little is said of the history, and in which the preceding passage of Falret is not quoted. "By bringing together and comparing a certain number of observations, it is seen that rather numerous cases occur in which it is impossible to consider separately and as two distinct affections the excitement and depression that succeed each other in the same patient. This succession does not take place at random, and I have been able to make certain that there is a connection between the duration and intensity of the two conditions, which are evidently only two periods of one single attack. The result of this opinion is, that those attacks properly belong neither to melancholia nor to mania, but constitute a special kind of mental disease, characterised by the regular existence of two periods—one of excitement, and another of depression. I will here attempt to indicate the leading characteristics of this

[1] 'Gazette Hebdomadaire,' pp. 263-279.

form of insanity, and will provisionally designate it Folie à double forme."

This provisional name has, as is well known, become permanent.

M. Baillarger then gives a few individual observations on patients, so as to give an exact idea of the attacks, and he demonstrates that in such cases there is no question either of remissions or of interruptions, properly so called. He indicates several of the chief characteristics of the disease, such as the following: the maniacal excitement is the more violent as the melancholic depression has been more severe; the principal point of diagnosis of Folie à double forme consists in the regularity of the succession of the periods; the disease, which is usually prolonged for several years, may be compared to a long chain, of which each attack would be one of the links; the duration of the attacks varies according to the cases observed; the frenzy is much more frequently characterised by impulses, than by delirious conceptions, properly so called.

He concludes thus, "Such are the observations that I have thought it my duty to make on Folie à double forme, the existence of which as a specific kind of insanity cannot be doubted."

Such is an abridged form of the description given by Baillarger, a description which will be considered by the majority of specialists as the revelation of a series of pathological facts not recognised till then.

However, one of those present at Baillarger's lecture believed that he recognised in it a picture already traced by himself, a species of disease the existence of which he himself had already indicated by giving a first sketch of it.

Therefore, as early as the following sitting of the Academy of Medicine (Feb. 7, 1854) Falret asked leave to speak on the occasion of the official report, in order to draw attention to the fact that the form of insanity described by Baillarger in his memoir was not a new one, and that it had been pointed out by himself as early as the year 1851. At the same time he requested permission to read a long memoir, in which the diagnosis, progress, and prognosis of *Folie circulaire* were described.

The question immediately before the meeting did not allow of this communication being read at that time, and it was postponed till Feb. 14.

In this paper Falret begins by eliminating the well-known and numerous cases in which mania succeeds melancholia, and vice versâ, in an accidental manner. He reserves the name *Folie circulaire* for those cases in which depression and excitement succeed each other during a long time, in an almost regular manner, in never varying order, and with a lucid interval which is of rather short duration compared to the length of the attacks. The disease, characterised by the regular alternation of maniacal excitement and melancholic depression, lasts sometimes for a short and sometimes for a long period, without this difference of duration at all changing in its nature. It is a specific form of mental disease, since it consists of a complete series of physical, intellectual and mental symptoms. These are always identical with one another in the various periods, and succeed one another in a fixed order which it is possible to foresee. In the same patient the various periods of each attack resemble one another not only in their entirety, but even in most of their maniacal manifestations.

Then follows a succinct description of each of the three periods—excitement, depression and lucid interval—and the advantage that is gained by the determination of this new form of mental disease, from the threefold point of view of nosology, therapeutics, and forensic medicine.

The analogy that we have just pointed out between the two memoirs read within a fortnight before the Academy by Baillarger and Falret is not merely of historical interest. Its object is to show what have been the pathognomonic characters attributed to this disease from the beginning, by the two physicians who claimed its discovery. In truth, their descriptions coincided in a good many points, and were also within a little of being complete. It seems to us that it is impossible to solve the question of priority which each has not ceased to claim for himself, and that an equal right of paternity may be assigned to both, and no wrong or reproach attach to either.

It is evident that the original memoir read by Baillarger

on the 31st of January, 1854, was not a plagiarism of the short quotation that was half-lost in a medical journal in 1851; and it is equally certain that the paper brought forward by Falret eight days later was not a plagiarism of Baillarger's memoir of the previous week. Even supposing that the material fact of the drawing up of Falret's paper was quite recent, it cannot be doubted that the observation of the cases and the conception of the theory had long before attained maturity in the mind of the author, whilst he waited for the chance that was to bring them to light.

Let us therefore set aside all paltry rivalry between two distinguished *savants*, and let us take the coincidence of discovery, of which there are numerous similar examples, as an additional proof of the reality of the facts and the probability of their scientific interpretation.

Moreover the publication, by different Asylum physicians, of individual observations on patients labouring under Folie à double forme, soon demonstrated that the type of this mental disease was met with wherever lunatics were under care. Since that period, therefore, all writers who have treated of insanity in general have been obliged to devote more or less space to the consideration of Folie à double forme. Besides, it has been the subject of several original memoirs and of various inaugural theses.

Among the early writers, Morel[1] and Dagonet[2] were content to speak of Folie à double forme as a secondary variety, without recognising that it possessed value as a distinct morbid entity. On the other hand, this pathological individuality has been recognised by Marcé,[3] Foville,[4] Ritti,[5] and Luys.[6]

Among special writings we may mention those of Billod,[7]

[1] 'Traité des Maladies mentales,' Paris, 1859, p. 477 *et seq.*

[2] 'Traité élémentaire des Maladies mentales,' 1st ed., Paris, 1862, pp. 114, 276; 2nd ed., 1876, p. 171.

[3] 'Traité pratique des Maladies mentales,' Paris, 1862, p. 329.

[4] 'Nouveau Dictionnaire de Médecine et de Chirurgie pratique,' tome xv. 1872, p. 371.

[5] 'Dictionnaire encyclopédique des Sciences médicales.' Quatrième série, tome iii., Paris, 1878.

[6] 'Traité pratique et clinique des Maladies mentales,' Paris, 1881.

[7] "Mémoire sur les diverses formes de Lypemanie." 'Annales Médico-psychologiques,' 1856, p. 333.

Jules Delaye,[1] Geoffroy,[2] Falret junior,[3] and Gérard.[4] We shall not undertake to name all the individual observations published in the various periodicals. Nearly all of these will be reproduced in the forthcoming edition of Ritti's memoir, to which the prize has been awarded.

The labours of French physicians in regard to Folie à double forme were unknown for long, and tardily accepted abroad. At length, however, they attracted attention.

In England, Maudsley,[5] in his book on crime and insanity, has given a short but very accurate description of the phases of excitement and depression of Folie à double forme, which he has connected with moral insanity.

In Germany this disease has been studied with great care during the last few years, and several important works have been devoted to it, by Meyer,[6] Kirn,[7] Krafft-Ebing,[8] and Karrer.[9]

In America, Spitzka[10] has given a good clinical description of it.

These are the names that have come to our knowledge, but we do not pretend that they are the only ones.

During the last few years, cases of general paralysis have been noticed in which delirium, added to muscular agitation and dementia, assumed alternately the form of maniacal excitement and melancholic depression, and it has been proposed to class it as a form of general paralysis à double forme. *Vide* Fabre,[11] Essian de Lamaistre,[12] Garne Lafitte.[13]

We consider the inconstancy of the mania to be a very

[1] "Etude sur la Folie à formes alternes." 'Journal de Médecine de Toulouse,' 1860.

[2] "De la Folie à double forme." 'Thèses de Paris,' 1861.

[3] 'Archives générales de Médecine,' 1878 et 1879.

[4] "De la Marche circulaire de la Folie." 'Thèse de Montpellier,' 1880.

[5] 'Le Crime et la Folie.' Edition française, Paris, 1874, p. 166.

[6] "Des Maladies mentales circulaires." 'Archiv für Psychiatrie,' 1874.

[7] 'Die periodischen Psychosen.' Stuttgart, 1878.

[8] 'Lehrbuch der Psychiatrie.' Stuttgart, 1879, 1880.

[9] 'Zeitschrift für Psychiatrie,' Band 37, Heft 6.

[10] 'New York Medical Gazette,' May, 1880.

[11] "Folie paralytique circulaire." 'Annales Médico-psychologiques,' 1874, tome i. p. 196.

[12] 'Congrès Internationale de Médecine mentale, tenu à Paris du 5 au 10 avril 1878,' p. 131.

[13] 'Archives cliniques; Annales Médico-psychologiques,' Mars 1882.

frequent, we might say a general feature in the evolution of general paralysis; independently of the delusions of grandeur and of hypochondriasis that have characteristics so strongly marked that they may almost be considered as pathognomonic. Sometimes depression, sometimes elation is observed in such patients; very often these opposite states alternate with great rapidity, or are even combined in so intimate a manner that they may be said to be simultaneous.

When, in certain of these cases, maniacal excitement and melancholic depression succeed one another in distinct periods, and a congestive attack seems to be the occasional cause of the alternation, we must look upon it as an interesting series of facts. It adds to our knowledge of the unequal and essentially variable progress of the intellectual aberrations of general paralysis. Our thanks are due to the authors who have described it; and we hope that future observations, going as far back as possible over the history of the patients, will cause it to be recognised—if the latter do not belong to the so numerous class of those hereditarily predisposed, which might partly explain the progress of their malady.

But these cannot be separated from the category of general paralysis, of which they only form a group with a few special characteristics. They remain, in our opinion, absolutely distinct from Folie à double forme, properly so called, and such as Falret and Baillarger have conceived, and such as we shall now describe.

Definition.

We will here guard against re-entering upon the long discussions regarding the words *Mania* and *Melancholia*. They have been employed from the times of the earliest medical literature, according to the widely different interpretations of the varying ages of the science; thus great confusion has been introduced.

It will be sufficient for us to say that, in the present state of our theoretical and clinical knowledge of mental diseases, in our opinion it will bring order and clearness into psychiatric nomenclature to admit that there are:—

Firstly.—Two diseases, characterised by symptoms, a special

evolution, and termination; and for these a separate place in nosology must be reserved under the names of mania and melancholia, or rather *lypemanie.*

Secondly.—Two symptoms, maniacal excitement and melancholic depression, which are met with not only in mania and *lypemanie,* properly so called, but also as accessories or transient manifestations in the greater number of the other species of mental diseases.

This preliminary distinction, to which, in our opinion, much importance cannot be attached, once established, we define Folie à double forme by saying that it is:—

"A species of insanity characterised by a prolonged succession of periods of maniacal excitement and of periods of melancholic depression usually alternating in a regular manner. The duration, intensity, type of the excitement and depression, the mode of transition from the one to the other may greatly vary; but their alternate recurrence is constant and pathognomonic. This species of insanity is almost always owing to the development of an hereditary predisposition; and most often it is incurable. It constitutes one of the plainest (*plus naturelle*) and least defined groups of mental disease."

Etiology.

We have already said that Folie à double forme is almost always owing to the development of an hereditary predisposition. This is in our opinion a most interesting fact, and one of great clinical importance. Falret senior, in his paper read in February 1854 before the Academy of Medicine, pointed out that *Folie circulaire* is very hereditary. Since then, all those who have written on this subject have expressed the same opinion, or have brought forward observations confirming it. In 1872 we said: "The fact of the well-established existence of Folie à double forme in a patient alone constitutes a presumption bordering upon certainty, that in him mental disease is chiefly due to the development of an hereditary or congenital germ. There is abundance of facts in our paper to support this opinion, and there is a total absence of evidence to the contrary."

We have nothing to change in the expression of our opinion on this point, and it is justified by the statistical data derived from various authors, collected by M. Ritti in his last work. In fact, of 29 cases recorded by him, proofs of direct or indirect heredity are noted in 23. They exist in all of the seven cases which came under M. Ritti's personal observation.

The cases that seem to be exceptions are therefore comparatively rare, and there is always reason to ask oneself if they are not explained by the insufficiency of the investigation instituted, or of the information obtained. For instance, in saying of any one that he is a healthy (*naturel*) child, it is far from being established that he is not predisposed to insanity.

Besides this transmitted predisposition, there might perhaps be ground to give a place to acquired predisposition; notably to that which a cerebral injury received in youth might develop. Professor Lasegue has indicated the part played by those traumatic accidents in producing a far-reaching cerebral diathesis;[1] the effects of which are only manifested at a more or less advanced period of life, in the shape of attacks of physical, intellectual, motor, and intermittent disorders. Several observations, recorded in Germany by Kirn and Von Krafft-Ebing, and reproduced by Ritti, are calculated to give the impression that Folie à double forme might be one of the ulterior manifestations of this neuropathic diathesis acquired in consequence of traumatic injury. This is an interesting point to put to the test of research, which, to be of value, should be long-continued and patient.

The female sex is evidently more subject to Folie à double forme than the male. Besides assertions made without figures to support them, the following proportions have been indicated by various French authors:—Falret senior, one man to three women; Foville, five to fourteen; and Ritti, one to six. In Germany also the predominance on the female side is generally admitted; but in the New York Asylums the reverse has been stated. The periodical which refers to this fact, however, adds

[1] Lasegue, 'Congrès International de Médecine mentale.' Paris, 1880, p. 227. "Les Cérébraux :" 'Archives générales de Médecine,' 1880, p. 29.

that the figures upon which the assertion is based seem to be wanting in exactness.[1]

The disease generally manifests itself after puberty, and it is very rarely developed after the age of 30—characteristics that permit of the classification of the malady under the head of hereditary insanities.

As it occurs especially among young women of nervous diathesis, it is quite natural that it should be observed concurrently with symptoms of hysteria. Hence the latter is occasionally mentioned as one of the causes of Folie à double forme; but it seems to us more correct to consider the hysterical symptoms as a parallel effect of the organic predisposition, rather than a special cause of the disease.

Among the various classes of the hereditarily insane, Folie à double forme occurs almost exclusively in those cases in whom predisposition was not indicated at birth by physical malformations or evident mental anomalies. On the contrary, before the outbreak of insanity, the patients may have presented the appearance of good bodily and mental organisation. During the respite the evolution of their disease afterwards leaves to them, they often enter upon the enjoyment of those advantages more fully than any other class of lunatics.

Having given a full account of acquired neuropathic diathesis, little need be added regarding the exciting causes. The latter, both physical and mental, may be those which are usually enumerated for the production of mental disease in an often commonplace, and not very demonstrable manner. In certain cases it is very difficult to discover any of them, and the evolution of the disease seems to begin with the single fact of the natural development of the pre-existing germ. In other cases those that are cited—pregnancy, lactation, intellectual fatigue, mental trials—have only a determinating influence; for the mental equilibrium is very unstable, and little suffices to destroy it.

[1] 'Journal of Nervous and Mental Disease,' October 1881, p. 796.

Symptomatology.

There is no lack in the world of people who, without being exactly considered fools, at least pass for originals and eccentric folk, on account of the alternations in their conduct and whole manner of being that succeed one another in a prolonged and often regular manner. For one period, lasting several weeks, months, or even years, these individuals are active, talkative, self-confident, full of initiative enthusiasm. They work with great zeal, they are at every one's service, undertake the greatest affairs, see everything from a favourable aspect, and know of no obstacle. For another period, on the contrary, they are inert, silent, and indifferent; they are distrustful of themselves and of others, avoid society, feel themselves incapable of undertaking anything, look on everything in the most sombre manner, and see in everything insurmountable difficulties. And thus during the whole course of their lives they do not cease from passing alternately from one extreme to another. Animation is the characteristic of all the acts of one phase of their existence, the other bears the impress of inertness. The disposition of the mind is never in equilibrium—for their life is alternately all rose-coloured or one complete black.

This condition may not pass the limits, sometimes so difficult to define, that separate sanity from insanity. Retained within bounds moderate enough, it is not sufficient to constitute a really characteristic mental disease, but it is surely an outline, a sketch, a miniature of it.

But when the alternating characteristics above indicated are more strongly marked, when the conditions of development, progress, evolution, and periodicity, remain the same, their traits are more defined, and their shadows more intense—the sketch is replaced by a morbid picture, by that of Folie à double forme.

The latter itself may present several degrees. In the cases that are perhaps most numerous, animation becomes maniacal excitement; inertness, melancholic depression; and these two classes of symptoms are reflected more or less violently in the conduct and bearing of the patients without there being in

them any intellectual delirium, properly so called; for the delirium of actions and feelings suffices to fill up the whole outline of the disease.

In some rarer and graver cases there are added to the excitement and to the general depression intellectual disturbances that constitute a real delirium, alternately assuming the form of melancholy and mania.

Naturally between those new extreme forms of the disease there are a number of gradations serving as a transition from one to another. It seems to us useful to separately describe the phase of depression and that of excitement, and to consider both in their psychic manifestations and somatic phenomena. After that we shall say a few words about the trophic disturbances that accompany them. We shall then indicate the manner in which the different morbid elements are connected by succession and alternation—that will be the study of the evolution of the disease.

Period of Depression.

Psychic Symptoms.—Whether depression succeeds a period of excitement or a normal intermission, it usually begins slowly and progressively becomes worse. The intellectual action diminishes, the patients grow sombre and silent; they keep apart from their neighbours, and generally occupy the same place. They employ themselves with nobody and with nothing; at least, if they still do something of trifling importance, they work with extreme slowness and without any liking for it. When these patients live in their own houses, or with their friends, they sometimes remain in bed for weeks or months together, and require that the blinds of their rooms be drawn. It is a real hybernation. In Asylums, where they are obliged to rise, they submit passively, as it were mechanically, to the arrangements made for them, hours of meals, occupations and recreations. They do not take the initiative, but are interested in nothing, and appear complete strangers to their surroundings. It has been frequently observed, during periods of excitement, that this appearance is merely deceptive, and that in reality they hear, see and retain everything. They

show no desire for anything; if they have any money at their disposal, they are avaricious, spend nothing, and refuse to make even the most necessary purchases. They never think of complaining, or offering any objections; their humility is without limit. They take no care of their personal appearance, dress in the simplest manner, and refuse everything in the shape of an ornament calculated to attract attention. Their affections seem lost; those at other times most dear to them are treated with indifference; there is no longer any expression of tenderness or indication of affection.

Such is the first stage of depression in which there does not seem to be any intellectual disturbance, properly so called. But things may go further, and this may appear and assume the diverse forms found in melancholia (*lypemanie*). The work of M. Ritti contains a detailed analysis of these different varieties of melancholic disturbances (*délire*), and reports cases of Folie à double forme divided into these four classes, according as they are:—

1. Under the influence of *tedium vitæ*, and possessed by ideas of suicide that may end them in death:

2. Tortured by continual anxiety, groundless fears, remorse for imaginary faults, ideas of damnation, or fear of coming want:

3. Tormented by hallucinations of hearing or of other senses, giving rise to the usual procession of ideas of persecution, fears, poisoning, and so on:

4. Or finally a prey to hypochondriasis more or less intense.

Of course these different varieties of melancholia are not always clearly distinct. Several may be united and intermingled in varying proportions; but the result of all is to make the patient extremely unhappy.

Finally, there are a few rare cases in which depression reaches its furthest limit, so as to plunge the patient into a state of stupor or dulness. Inertness is then absolute, and may extend to a total forgetfulness of the most essential things. Food is only taken on compulsion, the saliva is not retained, and no control is exercised over the excretions. This stupor may, as in ordinary melancholia, conceal two very different intellectual conditions: either a real suspension of all

mental activity, an abolition of thought; or a mental disturbance of so intense an activity that it paralyses the whole being, congeals it, so to speak, and suppresses all external signs, although the mind may not be at rest, but, on the contrary, highly active.

Somatic Symptoms.—Whatever may be the degree of melancholic depression, the physical condition of the patients corresponds to their mental state. The attitude is constrained and most frequently motionless, the movements of the body are slow and as limited as possible. The patients change their place only when obliged; they walk, as it were, reluctantly; it seems as if their feet cannot leave the ground; the shortest distance may appear enormous to them (*Ritti*). The eye has lost its brilliancy and is continually cast down. The face is without expression, it bears the stamp of indifference, prostration, or the most profound sadness. The voice especially is completely altered. Some patients cease to speak; others still answer when plied with questions, but with few words, and in a low voice. The words must be dragged out of them; and are painfully murmured, or, as it were, strangled.

There are disturbances of the general sensibility, especially connected with internal organs—precordial anxiety, epigastric pains, cephalalgia, or a feeling of emptiness in the head, intercostal or other neuralgias. We find sometimes either analgesia and anæsthesia, or cutaneous hyperæsthesia, especially in women who at the same time present hysterical symptoms. Photophobia and dulness of hearing have also been noticed.

It is to be remarked that sleep is almost always calm and regular, often lasts a long time.

The organic functions are not less usually trammelled than the functions of relation. The appetite often fails, the patients do not eat spontaneously, they must be forced to sit down to dinner, their food must be put into their mouths. Sometimes recourse must be had to the stomach-pump. The digestion is slow and painful; constipation is almost universal and sometimes very obstinate.

The respiration is retarded both in the nature and extent of the chest movements. It is the same with the circulation,

the beats of the heart being feeble, and lowered in some patients to 25 or 30 in a minute. Under this double influence the blood is insufficiently oxygenised, and the skin may be strongly cyanotic. More usually, however, it is pale and dry. Perspiration and other secretions are poured forth more slowly. In women, menstruation is sometimes completely suppressed, and almost always more or less thoroughly out of order.

Of course the physical instincts appear extinguished; there is no erotic tendency, and no taste for stimulants is displayed.

When to simple depression are added the delirium and stupor of melancholia (*lypemanie*), the physical disturbances increase proportionately to the mental. The inward anxiety may be manifested by a pantophobic attitude and by continual sighing. Certain patients tear their faces and scratch their hands. Finally, in the condition of profound stupor, the body, having become completely inert, no longer seems to suggest to the mind any desire or need. Even the evacuations are involuntary, cleanliness can only be maintained by the care and control of those in charge, and thus only can existence be supported.

Period of Excitement.

Psychic Symptoms.—This period is in every respect the reverse of the period of depression above described; and its most usual characteristic consists in the disorder of the emotions and actions much more than that of the purely intellectual faculties. So in many respects it has been confounded with what has been described under the names of Moral Insanity, Insanity of Action, Impulsive Mania, Reasoning Mania, &c.

The intellectual activity is continually on the alert; the patients are lively, confident in themselves, continually move about, and cannot take a moment's rest. They are ready to undertake all kinds of business at the same time, taking an interest in whatever comes to their knowledge. Nothing is indifferent to them. Their loquacity is extreme, and they talk on all subjects. They are seldom actuated by friendliness; more frequently the most biting irony and the keenest spitefulness dictate their utterances. Some of them possess a diabolical cleverness in saying to every person what is most

calculated to ruffle them. They utilise for that purpose, with an astonishing tenacity of memory and with great adroitness, whatever they have seen or heard during their period of depression, when they had appeared incapable of paying attention to anything whatever. The feelings of affection undergo the same metamorphosis; whether in point of friendship or hatred nothing in them is kept within the bounds of moderation. Everything is violent, exaggerated and implacable, and at the same time equally mobile, so that the most opposite states succeed one another without transition and without motive.

Their behaviour is excessive in everything. When they are their own masters, they become lavish and enterprising; buy all they have a fancy for—and they have a fancy for everything. They rush into business, and engage without prudence in risky transactions, and it sometimes happens that their blind rashness is crowned with success. If they own property, they stop at nothing to adorn their houses, to embellish their estates. They overturn their land, displace their trees, and want to change everything at the same time. Others are seized with a desire to travel, must see the world, and spare no expense. Sometimes they disappear from their homes, and wander about at random without giving information as to their whereabouts. One fine day you learn that they are far from home, and in a locality where they have no business.

They prove aggressive and quarrelsome, and are unconscious of duty; but are not disposed to admit that there is any limit to their rights; therefore the slightest pretext causes an outburst of insult, acts of violence and altercation. The instinctive impulses are over-excited and run to every excess; and so there are often observed in these patients incorrigible depravities which at one time were considered separate diseases, —such as destructive monomania, dipsomania, nymphomania, satyriasis. Nothing is better calculated than the study of Folie à double forme to demonstrate that those morbid impulses have no other value than that of a symptom. It is the same with kleptomania. Certain cases without any scruple appropriate whatever is within their reach.

The erotic instinct is almost always one of those most de-

veloped, whether it shows itself merely by coquetry, or elegance of apparel, or finds utterance in the most obscene language, the most abject lewdness, the most outrageous practices of masturbation. It is to be noticed that the forms of genital perversion described of late years by Westphal and some other German authors, under the name of Perverted Sexual Inclination (*die Contrāre Sexualempfindung*), are sometimes observed in patients afflicted with Folie à double forme. (*Ritti.*)

When the patients are confined in Asylums, the field of their activity is forcibly limited, but the latter is not the less overexcited and strives to find means of exercising it. They excite the patients against one another, they invent the most treacherous calumnies against the officials, and know how to clothe their lies with the semblance of truth. To use a popular expression, they would set mountains at loggerheads. "One must have lived," says M. Jules Falret, "with similar patients to form an exact idea of the infernal stories that they are capable of inventing, of the domestic strife and trouble that they spread around them." Very frequently also they attempt to escape.

All this disorder may be produced in their actions without being accompanied by any manifest disturbance in the purely intellectual domain, or by incoherence of language, so that when inexperienced people see these patients only incidentally, they may not recognise the form of their disease, may attribute their conduct to other causes than insanity, and imagine that they are wrongly confined.

There are other cases in which this error is impossible, a real maniacal frenzy being added to the excitement. The patient may then experience numerous hallucinations, succeeding one another with great rapidity. Their actions bear the impress of a truly furious violence; they break the furniture, smash the window-panes and destroy everything that comes in their way. Among the most usual delusions are those that resemble the notions of grandeur in general paralysis. Sometimes they are moderate, and limited to a general optimism, or to the hope of a large fortune; or, on the other hand, they flatter the patient that he has an extraordinary aptitude for

all the arts, that he excels at the same time in poetry, music, and painting. Or, again, they suggest to him a number of discoveries or inventions, every one more marvellous than another. Or, finally, reaching the utmost limits, they transport him to the summit of power and wealth, and lavish honours and millions of money on him.

Somatic Symptoms.—The physical condition faithfully reflects the degree of mental excitement. The patients carry their heads high, walk with a firm and rapid tread, their movements are impetuous and their gestures abrupt. They cannot keep their seats, and show great exuberance in whatever they do and say. The voice is full and sonorous, sometimes shrill, like a flourish of trumpets. Though their words follow one another in the most rapid succession, they do not succeed in giving expression to all their thoughts. The eye is bold. The most expressive mimicry describes the mental tumult, but it is especially by their deportment and dress that they are distinguishable from those about them. Most frequently the eccentricity of their appearance would be sufficient to point them out; the women arrange their hair pretentiously, or let it hang down. Everything in them breathes provocative coquetry. They dress in a showy incongruous manner that clashes with their age and position. Or passing those still comparatively modest limits, they attire themselves ridiculously, put their petticoats over their dresses, continually don and doff their shoes, stockings, and hats. They unsew their clothes to refashion them, to add as ornaments the most vulgar finery, to lengthen them here and shorten them there, and so on. Men show analogous symptoms. Some introduce the greatest affectation in their toilette, pomade and perfume. Others put on everything the wrong way, or pull off their buttons, and rend their garments in shreds.

The organic functions operate with an activity which produces a general sense of physical comfort. Never have they felt better, more fit for work, and more capable of doing great things. The respiration is ample and frequent, the circulation full and rapid, the number of pulsations nearly always considerably increased. All the secretions are abundant, the complexion is clear and bright, the skin is supple and of a

healthy colour. The appetite is good, and digestion prompt and easy. Sleep is short and light, frequently disturbed by dreams. Sometimes there is a total absence of it. In a few patients those signs of functional abundance may be accompanied by other physical symptoms, not unlike those at the onset of general paralysis. In this way, activity on the one hand and superabundance of ideas on the other, may modify the speech and the voice by imparting to them a wavering or a shaking; thus the over-activity of the circulation may determine towards the head with a more or less intense congestion, even pupillary phenomena or epileptiform seizures have been observed.

Trophic Disturbances.—Although these are closely connected with the state of depression or excitement, it seems to us appropriate to make special mention of them. Everyone who has written on Folie à double forme has pointed out that there are remarkable alternations of stoutness and spareness in patients who suffer from that malady. These correspond to the periods of depression and excitement. Meyer, especially, considers the increase in weight during the excitement and reciprocally as a constant and characteristic fact. He therefore does not hesitate to apply the term *trophoneurose*. We cannot adopt this opinion, for observation has taught us that the variations in weight are far from being always produced in the same order. One of the patients mentioned in 1854 by Baillarger got very stout during the period of excitement, and got thin during the depression, as Meyer says. It is even stated that once during the melancholy stage she lost twelve pounds (*livres*) in a fortnight. On the other hand, we ourselves have observed a young man who got thin during the excitement, and stout when depressed. In taking a great number of cases, we have found several of them in which modifications in fatness were noticed in conformity with those two opposite types; so there seems to be a contradiction here, but in reality it is only apparent: the disturbances in the nutrition are in correspondence with the intensity of the symptoms proper to each period of excitement or depression. When the latter is profound enough to be accompanied by refusal of regular and

sufficient nourishment, spareness naturally follows, as in the case of Baillarger's patient. When, on the contrary, the patient, having become calm and sad, vegetates in absolute inertness without ceasing to take regular meals, the organic receipts far surpass the expenditure, and stoutness obtains as in our case above alluded to. The reverse takes place during the period of excitement, according as the development of the appetite and the digestive perfection make the patient grow fat; or as the general over-activity, the absence of sleep, and the over-work that makes him grow thin, gain the upper hand. We can therefore see nothing pathognomonic in those alternations of body-weight.

Progress of the Attack—Development of the Disease.

We have said nothing as yet of the manner in which the two opposite periods of depression and excitement are connected, and of the lucid intervals, which together constitute the complete attack of Folie à double forme. This is, however, a point which until now has given rise to serious controversies, and which has played in our opinion too large a part in the history of this disease.

Baillarger, notably, has done his best to establish a well-marked distinction between Folie à double forme, such as he conceived it, and Folie circulaire, such as Falret described. According to him, the latter had committed the mistake of representing the evolution of Folie circulaire as proceeding in the following manner: mania, intermission; melancholy, intermission; mania, &c.; so he saw in it only intermittent insanity with alternate forms. Baillarger, on the contrary, represents the mania and the melancholia as succeeding each other without interruption, and as constituting together a complete attack, separated from the next seizure by an intermission or lucid interval, so that the formula would run: mania, melancholia (attack), intermission; mania, melancholia (attack), intermission; and so on. This makes Folie à double forme a distinct morbid entity.

For our part we must confess that, from personal observation of patients, we can allow to these formulæ neither

absolute precision nor any considerable nosological value. The important fact, is that certain lunatics hopelessly revolve within an unchanging circle, and alternately present the symptoms of mania and melancholia. This is what causes these patients to form a very distinct group, which ought not to be confounded with any other in mental pathology. Another fact that we consider established is, that in certain of these patients the disease is not continuous; that is to say, between the termination of one attack and the commencement of another, they may remain for a longer or a shorter time, (for months, and even years,) in a condition which constitutes for them a state of normal sanity, or which comes very near to it. Such are real remissions.

But in the cases in which these long remissions are absent, whether or not there be between the two morbid states a short period of calm, of relative or complete lucidity, is in our opinion an accessory fact, which is, moreover, almost always very difficult to determine.

In some cases the transition is abrupt, and immediately concluded. It is then most usually produced during sleep. A patient has gone to bed a maniac, to waken in a state of melancholia. This is rare, but it happens. It is evident that there are cases where we cannot discuss if there be an intermission, and at what moment it is produced.

Much more usually the transition is slow and gradual, the patient appears less excited than he was before. He then becomes more and more calm, all aberration ceases, all vehemence subsides, each one of the functions remains in order, but no equilibrium appears. A little prostration sets in, the sadness gradually increases, and then the patient enters on the period of depression.

The interval between decided mania and confirmed melancholia can only have lasted a few days, but it may have lasted a few weeks or even months. In that case the intermediate gradations are numerous and undiscernible. Has there really been during that time a period of perfect sanity, a moment in which the equilibrium was perfect—a real remission? We would not venture to say so, and do not see what importance it would have from a nosological point of view.

The duration of the attack is most variable in different patients, but in the same person it is generally sufficiently regular. Sometimes the circle is traversed very rapidly, and each period of excitement or depression lasts only for three weeks. Some writers have spoken of stages of very brief duration, two or three days or even a single one; we have never noted any so short, and would be inclined to doubt if it were really Folie à double forme.

It is much more common to meet with attacks that last for several months, and it is exceptional to find them extending over a year.

Griesinger was the first to indicate a certain connection between the alternations of the seasons and the opposite phases of this disease, viz. that the depression takes place in winter and the excitement in summer. Baillarger confirmed this remark, and we ourselves have verified it in a few patients, but it is far from being constant.

As to the respective durations of the periods, sometimes they are equal, sometimes one is longer than the other. Thus certain patients remain for months in a state of depression, afterwards to traverse a period of excitement of only a few weeks' duration.

One very remarkable peculiarity of Folie à double forme is that in the same patient the different phases of melancholia and mania are very like each other. During each of the periods of depression the humility of attitude, the mask of sadness, the timidity of the look, the resistance at meal times, the carelessness in dress, the imaginary fears, are reproduced in a perfectly identical manner. It would seem that the patients take the greatest care in recopying themselves; but what is still more striking is the absolute similarity of the actions and of the extravagance during the period of excitement. If one observes a patient so affected only on a single occasion and cursorily, everything in his case appears absolutely unsettled; but if he is seen during several attacks of excitement, it is recognised that all his disorder is subject to exact and constant laws.

When after a longer or shorter interval he is about to become elevated again, it is perceived beforehand by the same signs

of awakening, by the same modifications in his bearing and dress, by the same inflections of his voice. The storm bursts forth and displays itself exactly in the same way. We have the same extravagant acts, the same spirit of quarrelling, the same impulses to drink to excess, the same erotic profligacy, &c. It is at the same point in the evolution of the attack that one man starts without warning on purposeless journeys; that another is arrested for having fought without motive or having insulted the police; that one woman disappears from her home to follow at random the first man that turns up; and so the circle continually revolves, till one might say that the repetition of the same phenomena corresponds to each of the divisions of its circumference.

M. Baillarger had already shown that there is a certain equality between the degrees of intensity of the two classes of phenomena in the same patient. Quite recently, M. Gérard has specially insisted on the constant harmony between the two periods, "so that a stage of stupor will never succeed a simple intellectual exaltation; nor a vague state of melancholia a violent attack of mania." We are willing to grant that this is often the case; but it is far from being constant, and, notably, the period of depression is frequently more moderate in its manifestation than the period of excitement. Not unfrequently patients enter lunatic asylums only at the moment when the latter begins, to leave them when it abates. They may stay at home without inconvenience during the remission and the period of melancholy, when the latter is not very severe; but they could not be kept there during the period of mania without danger.

By reason of its constitutional character, transmitted by heredity or more rarely acquired by traumatism, Folie, à double forme is essentially chronic, and when once the rotation is well established, the disease usually continues during the remainder of life without very perceptible modification. It even most frequently escapes the usual fate of manias that have become chronic, viz. an ending in dementia. Through these perpetual losses of equilibrium, sometimes in one direction and sometimes in another, the faculties, instead of getting worn-out and disappearing, preserve their vigour and relative

integrity. We have known patients suffering from Folie à double forme of a very advanced age, who during remissions, or at the onset of periods of excitement, gave proofs of much refinement of mind, and of great freshness of memory.

Diagnosis.

As soon as it has been possible to verify, by observation or exact information, the alternation of opposite symptoms constituting one or several attacks with or without intervals of remission, the diagnosis of Folie à double forme may be confidently formulated. When its type is well characterised, it cannot be confounded with any other variety of mental disease.

But what would be desirable, as was truly said by Baillarger as early as 1854, would be to find clinical features recognisable on the first attack. Do we possess those clinical indications? We do not think so, and in our opinion the almost regular return of several alternate phases of depression and excitement marks the only pathognomonic character of the disease.

Can one not, however, by direct examination, limit this absence of absolute certainty by watching the evolution of the attack, and establish grave presumptions? It is not, we think, when the patient is depressed that it can be done, for then it would be difficult to distinguish him from the crowd of ordinary cases of melancholia (*lypemanie*). Can he not, in fact, be confounded with those cases, presenting as they do the various features of sadness, from simple melancholy up to absolute stupor? The difficulty is less great when we have to distinguish between ordinary mania and the period of the excitement of Folie à double forme. In this latter the delirium of the actions predominates in a remarkable manner, without being accompanied by any evident incoherence of speech or perceptible disturbance of the purely intellectual functions. It may happen that the ill-natured and caustic language of the patient, the disorder of his dress and behaviour, the deviation in his conduct, a certain *ensemble* in his whole manner, difficult to describe but recognisable by observation, all accompanied by an absence of hallucinations, of senseless

vagaries, lead one to suppose that we have not to deal with an attack of ordinary mania, but with Folie à double forme. Nothing less is required, before we can categorically pronounce upon the case, to reconstitute, as completely as possible, the previous history of the patient. It has happened more than once that we, by the special extravagance of the actions, were put upon the track of a diagnosis which has turned out to be more or less completely confirmed.

But the chance of error that we must often meet with, and against which it is most difficult to be upon one's guard, is the possibility of confounding the period of excitement of Folie à double forme with the beginning of the expansive variety of general paralysis. The resemblance may be very great, both as regards the mental and bodily symptoms.

As for the first, we have already stated that when intellectual disturbance is added to the maniacal excitement of Folie à double forme, the first frequently assumes the form of delusions of grandeur so common in paralytic patients. Even when there is no mental disease, properly so called, the resemblance may be very great. The mind deranged with enterprises; the opinion of self in the intellectual, artistic, and poetical domain exaggerated; the optimism generalised, in a word accompanied by impulses to theft, to excess of all kinds, to the most compromising actions might present the appearance in the two diseases of almost identical characteristics.

How are we to distinguish between the two maladies? Can we not depend upon the physical phenomena, that is to say, on the symptoms of paralysis, or rather the muscular ataxia, present in one series of cases and absent in another? Unfortunately we cannot invariably. On the contrary, it is well known that certain patients at the beginning of their excitement may not present any apparent derangement of speech or of motion, and who yet are suffering from general paralysis. The progress of their malady soon furnishes proof of it. On the other hand, as we have already said, in certain cases of Folie à double forme the close connection between the emotions and the cerebral activity may impart to the speech a degree of tremulousness very difficult to distinguish from that of general paralysis. Finally, what makes the question still

more embarrassing is the fact that in a few cases of Folie à double forme pupillary derangements, congestive cerebral attacks, and even real attacks of epileptiform convulsions have been noticed. (*Jules Falret.*)

A psychic element, which would be of great value if it could be invariably established, would be the inception of dementia, which is common enough in the beginning of general paralysis, to be considered constant. But is it not known that this partial weakening of certain intellectual faculties is often so disguised, in consequence of the general state of excitement, that it is almost impossible to make sure of its existence?

In a quite recent work, M. Regis,[1] after having shown the difficulties of the question, thinks he has found a criterion in the nature of the patient's feelings. According to him, the paralytic would be really kind-hearted, generous, even prodigal, desirous of being agreeable to everybody, and spreading around him the treasures of a common benevolence. The patient suffering from Folie à double forme, on the contrary, during the period of excitement would be wicked above everything, cantankerous, ironical, clever in injuring everybody approaching him. We are far from denying that it is often so. We even add that, from our observations of M. Regis's patient, extending over twelve years, we altogether share his opinion of the character of the pathological wickedness which forms the most salient feature of the periods of excitement of that case. But can we see in this a sufficiently constant sign to make it the theoretical basis of a difficult differential diagnosis? We are not inclined to admit it. We have known cases of general paralysis who were caustic and mischievous, and we believe that patients suffering under Folie à double forme might be found who, by way of exception, might be generous and benevolent.

After having recognised from this special point of view the untrustworthiness of the various classes of symptoms of the intellectual faculties of the moral feelings and of the psychic functions, may we find the solution of the problem in general conditions, such as age, sex, or heredity? Evidently not. These states may furnish the elements of a

[1] 'L'Encéphale,' Dec. 1881, p. 681.

presumption, more or less approaching to certainty, but will not be certainty itself. To conclude: before making the diagnosis of Folie à double forme with any surety, we must repeat that it is essential to know positively that the patient has furnished, by a greater or smaller number of repetitions, the series of phenomena constituting alternate periods of mania and melancholia—with or without intermediate remissions. This, with our knowledge of mania, is the really pathognomonic character, the only certain basis of a differential diagnosis. In expressing this opinion we do not think that we are "avoiding the clinical difficulties or seeking the elements of the distinctions to be made elsewhere than in a comparative and profound study of the two diseases themselves."

Prognosis.

The prognosis of Folie à double forme must be looked at from a threefold point of view; of the influence of this malady on the general health, of its chance of cure, and of its ways of terminating.

Although the nutrition and the whole organic functions at the time of each crisis of mania and melancholia undergo the modifications that we have described, Folie à double forme is not a disease that seriously endangers life. Apart from the rare chances of suicide during the period of melancholia, and those of the serious accidents due to imprudence during the period of mania, the longevity is not perceptibly modified. The congestive attacks and those of apoplectic character are too rare to perceptibly vary the preceding statement. Sometimes in Asylums, such patients are met with in a vigorous old age.

Folie à double forme presents only a slight chance of recovery. Inasmuch as it is almost always hereditary, it forms, so to speak, an intrinsic part of the condition of existence of the patient. It is essentially chronic and constitutional. When the rotation has been established, it sometimes happens to undergo accidental modifications, but they do not last long, and soon it hopelessly returns in its alternations. It has even appeared to us that when the onset was delayed longer than usual, this remission was balanced by a more lengthy duration and greater

intensity of the following attack. So even if the remissions are complete, they should inspire a very limited hope, and the approaching relapse must be considered very probable.

It is true that a few cases of recovery are cited as having occurred after one or two attacks, but we consider such quite exceptional, even if the diagnosis of Folie à double forme were correct.

We have already said that this disease is distinguished from other forms of insanity, inasmuch as it less regularly ends in dementia.

Treatment.

The really efficacious treatment would consist in preventing a return of the attacks. It must therefore have appeared quite natural to have recourse to antiperiodics—by preference to sulphate of quinine. Some cases are quoted said to have been cured by this means. But we must remark that we have to deal with facts published anterior to the discovery of Folie à double forme as a special disease, and retrospectively associated with it; or with observations published shortly after the original paper of Baillarger and Falret. It is very remarkable that since Folie à double forme has become better known, and clinical experience of it more complete, no more cases have been mentioned as having been cured by sulphate of quinine, or any other medicine. We cannot be surprised at these negative results when we consider the constitutional and chronic nature of the malady.

Baillarger has mentioned the case of a young girl in whom, by monthly bleeding, he obtained the disappearance of the maniacal phase, and reduced the disease to periodic attacks of melancholia. Dittmar, quoted by Krafft-Ebing, is said to have obtained an analogous result by forcing the patient to remain in bed. Those results must be partial and exceptional, and the general opinion is that, in what concerns the nature and the alternating return of the disease, Folie à double forme is nearly always beyond the resources of therapeutics.

In default of preventive or curative treatment, we still have the palliative treatment of the most salient symptoms, and here the indications are the same as in ordinary mania or melancholia.

Legal Medicine.

Little has been written, to our knowledge at least, on the connection between Folie à double forme and forensic medicine. Ritti alone has briefly treated of this side of the question. It may be of great importance, especially during the period of excitement. Certain authors have proposed, not without reason, to apply to the initial period of general paralysis the name of "medico-legal stage." The same denomination would be equally applicable, and for the same cause, to the period of excitement of Folie à double forme. This cause is, as is well understood, the predominance of disorders of the actions and the impulses with little or no intellectual disturbance. The patients, by their very disease, are induced to commit such acts as bring them within the reach of justice, and their speech has not that stamp of incoherence that at the first glance indicates a state of insanity. Difficulties of appreciation may tax the skill of the specialist, and may be very embarrassing even to him if he has not the means of being completely informed as to the antecedents of the accused.

We have already said that a certain number of cases of Folie à double forme may live at liberty during the period of depression; but that the return of excitement renders it necessary for them to be placed in a special Asylum. There are some who wander far and commit extravagant acts, which cause them to be arrested by the police. Each attack, therefore, is a new problem for the local authorities, all the more difficult as the individual and his history are totally disregarded. So the result is that they are usually sent to prison, there develop their disease, and thence are sent to an Asylum.

The morbid impulse to commit thefts, whether it be isolated or accompanied by vagrancy, has still the frequent result of bringing those patients into contact with the authorities. It may be the same with their troublesome and aggressive temper.

These patients, when placed in an Asylum, become a scourge to the staff by their insubordination and their diabolical cleverness in accusing everybody, and giving to their most mendacious imputations the aspect of reality. Their

complaints to the magistrates charged with the supervision of Asylums may be the starting-point of medico-legal enquiries and inspections, in which an exact knowledge of the disease is necessary to allow the nature and value of the facts to be properly established.

In other cases, justice may have to estimate the value of a will, the validity of commercial transactions, &c., carried on by those patients when in the state of furious activity. The verdict to be given must deal cautiously with all legitimate interests, and is a very delicate question.

The period of depression may also raise medico-legal questions, by having provoked to suicide, or attempted or completed misdemeanours and crimes. Renaudin has published a case of this kind, in which a young girl in a paroxysm of melancholia had cut off the head of a girl of two years of age with a kitchen-knife.[1]

These general indications, though brief, will suffice to show the importance and the difficulties of the forensic aspect of this disease. The fundamental principle by which they must be resolved has been clearly formulated by Falret senior (1854), when he said that "in such a case the judgment must be based not upon an observation of the patient at a given moment, but on the clinical study of the natural progress of the disease."

Anatomical Modifications.

It cannot be doubted that conditions so different as maniacal excitement and melancholic depression must be in close connection with certain modifications of the anatomical elements of the brain, and that certain organic changes must correspond to the functional derangements. But it has not been, up till now, determined if there be an anatomical lesion proper to mania or to melancholia.

An attempt at the theoretic systematisation of the anatomical lesions of all mental disease, much more complete than any other, has recently been made by M. Luys,[2] and the undertaking

[1] 'Annales Médico-psychologiques,' 1858, p. 111.

[2] 'Traité clinique et pratique des Maladies Mentales.' Paris, 1881.

is too meritorious, and the question too interesting, not to attract the most serious attention.

M. Luys starts from the principle generally adopted now-a-days that in the normal condition of our organisation the degree of activity of the circulation of the blood exactly corresponds to the degree of activity of the function; so that hyperæmia is a cause of exaltation, and anæmia a cause of depression.

Applying this principle to the nerve-cells, he regards it as established (pp. 383–4) that "localised or general hyperæmia becomes the initial phenomenon on which the activity of the nerve-cell depends," and that, inversely, "the degree of intensity of the innervation is susceptible of falling below its normal conditions by the fact of the rarefaction of the currents of the blood."

Hence he comes to the following conclusion, that serves as an axis to his whole theory: "These relations between the condition of the circulatory channels and the functional derangements ascertained during life are so closely connected that we have not hesitated to take them for a basis of scientific classification of mental diseases. And as the various derangements of mental maladies are always more or less comprised in the phenomena of irritation or nervous torpidity, general or local, we have been led to state that they have been more or less subordinated to a diffused or partial condition of hyperæmia or anæmia of the parts concerned. On these grounds, we have valued the natural elements designed to establish the classification of the different groups of mental maladies."

As the general theory has been formulated in terms so categorical and exclusive, it is easy to foresee the application of it to Folie à double forme, which M. Luys states, as we do, to be one of the best established types of mental pathology. It forms a special class, that of mental diseases characterised by the alternation of the hyperæmic process (excitement), and the anæmic process (depression), that succeed one another in the same subject at periodical intervals (p. 513).

To put the anatomical theory in accordance with the symptomatological evolution of the disease, it is enough for

M. Luys to say that, "on the one hand, the phenomena of alternate depression or excitement inevitably succeed one another by a series of imperceptible gradations, regulated by the fatality of the laws of the capillary circulation; and on the other hand, that the depression and excitement are only apparent dynamic variations of the condition of anæmia or successive hyperæmia through which the nervous course passes."

Of course, so that the symmetry may be complete, M. Luys admits that, if certain functional derangements be partial, it is because the circulatory disturbances also only affect certain cerebral regions (p. 96). If, especially, in many cases of Folie à double forme, there be a preponderance of the disturbance of the actions or of the emotions over the purely intellectual functions, it is because the melancholia, or the anæmia, is limited to the sensitive zones or the matrices of the hemispheres, and does not extend to the frontal convolutions.

It is seen that the harmony admitted by M. Luys between the functional derangements and the anatomical changes leaves nothing to be desired. If this theory of so simple and regular a law were demonstrated to be exact, many of the unknown facts that still block the way of the study of insanity would be eliminated.

But has it been demonstrated? and is it sufficient to say that in principle there must be a parallelism between the function and the circulation for the difficulties of the problem to be removed? That would certainly be very convenient; we therefore regret very much that we have to make very serious reservations.

When a patient is in a state of melancholia, more or less bordering on stupor, and it is seen that the skin is congested, that the mucous membranes are cyanotic, and that everything indicates a state of stagnation of the capillary circulation—can it be admitted, without any proof, that the brain is in a general or partial state of anæmia? Are we not rather to believe that the internal organs are congested as well as the external? We know that it might be said that the congestion would be passive instead of active, but this condition would greatly differ from anæmia.

Is it correct to represent profound melancholia and stupor as conditions during which the intellectual phenomena are lessened and abolished? That there is a suspension of these phenomena in certain cases of stupor is very likely; but is it certain that in other cases, behind the mask of an absolute physical torpidity are hidden considerable mental activity, mental impression, ability, and phenomena of sensation? How can we believe in such a law as that, in the period of depression of Folie à double forme there may exist in a constant manner both cerebral anæmia, and suspension of the intellectual faculties?

We still have to observe that, were the harmony admitted by M. Luys recognised as exact, it would remain to be demonstrated that the anatomical phenomena are certainly the cause of the psychic phenomena, and not the effect. Everybody will admit that when we are busily engaged in an interesting and serious intellectual labour, our arterial cerebral circulation is more active and rapid; but is it because the circulation is accelerated that we work better? Is it not, on the contrary, because we work, that the circulation is accelerated?

In the erectile organs, is it the afflux of blood that causes the orgasm, the erythism? Is it not, on the contrary, the appetite or orgasm that determines the afflux of blood?

This rapid review seems to us sufficient to show, without going beyond the general data of physiology, that the anatomico-pathological theory of insanity propounded by M. Luys, however attractive by the simplicity of its principles and the symmetry of its proportion, lends itself with difficulty to the explanation of all the phenomena; and that, what more specially concerns Folie à double forme, it seems to have no connection either with the state of the circulation nor with that of the psychic function—at least during the state of depression. For the present it cannot be admitted; and the problems of the relations that must exist between the dynamic modifications and the anatomical alterations of the cerebral organs have, in our opinion, not yet been sufficiently solved.

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FURTHER OBSERVATIONS ON CHRONIC MORPHINISM.

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I PUBLISHED in the January number of 'BRAIN,' 1880, a short paper on Chronic Morphinism. I there stated my opinion that it is by no means advisable to confine oneself to the 'weaning cure' in all cases. I have rather insisted upon the frequent uselessness of this cure, which in addition is the cause of very great sufferings to the patient. The conditions in which it is likely to be durably successful, are when the habit is of not too long standing, when the daily quantity consumed is not too great, when especially the cause which has led to the abuse is no longer existing, and the physical and psychical condition of the patient is satisfactory.

The cases of morphinism are very numerous; but if we consider only the nine cases described in my previous paper, it is apparent that the prognosis is far less favourable than is generally stated.

Perhaps only one of these cases, at most two, may be considered as examples of complete, permanent, recovery.

In all the others the treatment either had no result, or had to be suspended; or relapses occurred, and in the worst instances mental disturbances made their appearance. Hence I must again call attention to the fact that the slighter forms of morphinism are not to be taken into account here. I would again insist, therefore, first upon the fact that the weaning cure must be resorted to only when there is reason to expect a successful issue; in the other cases, one inflicts useless pain upon the patients, or even jeopardises their life.

A second point, which I have briefly alluded to previously, is that mental disturbances are frequently connected with morphinism. Indeed the starting-point of the habit may lie in such a disturbance. I can now state with greater certainty that a man who consumes large quantities of morphia during a number of years will display many nervous symptoms, and that the continued intoxication attacks the psychical much more constantly than the somatic life. Most physicians are still of opinion that nothing can be simpler than to take away a morphinist from his bad habit, either by the gradual or by the sudden removal of the drug.

With the unfortunately ever-increasing frequency of morphinism, every physician can surely recall to himself a number of instances. I shall therefore confine myself to record a few selected cases from my own practice, which offer some points of interest, and give some confirmation to the views just stated.

Case 1.—L., aged 25. The description of the antecedents of this interesting patient are in a great measure derived from an excellent history of the case which I owe to Dr. Hölscher. Patient's mother, to whom he had a physical likeness, died of apoplexy. As a child he frequently had spasmodic fits; at puberty syncopal attacks occurred also. At school, and still more during his law studies, he was frivolous, and often got intoxicated.

On the 1st of October, 1876 (he was then about 21), he came home in the evening apparently drunk, was very excited, cried, wanted to jump out of the window, &c. These psychical disturbances lasted three days; he then returned to his senses, but became subject to fits of a hysterical nature. These were preceded by an aura, consisting of pains along the spinal column, and assumed the form of generalised tremors, accompanied with violent cutaneous and muscular (back) pains. When the fit attained its climax, there occurred a choking, like globus, and occasionally left facial spasms of short duration were observed. He groaned deeply, and spasmodic backwards and sidewards bending of the body followed; sometimes he would spin with great rapidity on his own axis, sometimes he raised his whole body and then threw his head

violently from side to side. Then occurred spasms of the diaphragm, the tongue was protruded, the mouth opened to the point of dislocation of the lower jaw; there was frequently excessive meteorism. The fits lasted from 30 to 45 minutes, and were repeated as often as twenty times in one day. After the fit, the patient was exhausted, perspiring. Consciousness was not notably diminished during the attack. It was only at the end of December of that year that the fits became milder, and occurred with intervals of some days. During one of the attacks, contracture to a high degree of the flexors of the left extremities, with anæsthesia of these parts, had set in.

Patient was persistently sleepless, had fearful visual hallucinations, complained of great pain along the spine and in the region of the stomach. He was highly sensitive to light and sound, melancholic, with suicidal ideas. The reflex excitability on the non-contractured side was increased.

The bodily condition of the patient became materially impaired; he became thin and anæmic. At the same time morphia injections began to be used, to combat the spasms and contractures.

The patient's mind was very excitable. A fit was brought about even by slight impressions. New phenomena and exacerbations always appeared on Monday, which day the patient always saw arrive with great terror. Objects at 12 inches distance appeared double.

In the course of 1877 the patient spent four months in a sanatorium without showing any new symptoms. The previous condition persisted. Boulimia of great intensity was developed. One point deserves notice; the excessive sensitiveness to atmospheric changes. Storms, for instance, were felt one or two days in advance by an increase in the spasms and hyperæsthesia.

Attempts to deprive the patient of his morphia (0·15 gramme daily) failed utterly. His want of it, on the contrary, rapidly increased, so that on his return home he arrived to 0·5 gramme (8 grains) daily. At the same time he was again rarely one day free from fits.

On the 1st of November, 1877, the contracture suddenly disappeared during a fit, and in three days he had regained full and normal motility in the left extremities.

The spasms became less frequent. There was a temporary relapse in April 1878, after which they disappeared altogether. Nutrition and sleep improved; on the other hand, numerous abscesses formed in the left leg, independently of the injections. The use of morphia became more abundant and uncontrollable, and *pari passu* the patient's moral nature fell off gradually: tendency to lying, quarrelsomeness, indisposition to any serious occupation, &c. Physically, there was great muscular weakness, tremor, nocturnal sweats.

In 1879, after the death of his father, which made but little impression upon him, he went to reside by himself, and thereby was removed from any control which his relatives and physicians might have had upon him. It appears certain, however, that he consumed then about 1 gramme of morphia daily. His moral and intellectual decadence was more marked; for instance, he would keep his fæces, contained in various vessels, in his cupboard, &c.

On the 18th of February he allowed himself to be sent by his sister and relations to my sanatorium, in order to break off his morphia habits. The patient was, at the time of his admission, physically reduced, with a greyish complexion. The left leg dragged slightly; there was no anæsthesia. He wishes himself to make the cure, and willingly conforms himself to our prescriptions. On the 19th he received about 0·1 gramme morphia; on the 20th none. There occurred diarrhœa, sacral pains, feeling of cold, vomiting, and fear of contractures. Hydrocyanic acid, chloral-hydrate, tepid baths, and wine were administered. 21st. The same; œdema of the face. 22nd. He feels very exhausted, and complains of cramps about the heart. Pulse 100, often intermittent. 23rd. Marked hyperæsthesia of left side of body; breathing superficial, without the co-operation of the diaphragm, black excreta. The formerly contracted pupils are wide, re-act sluggishly. Still feeling of cold, appetite returning. 24th. Slight spasms in various muscles (especially wide opening of the mouth) occur transitorily. 27th. Sleep pretty good, no diarrhœa, appetite good. No desire for morphia; much stronger. 28th. Goes for the first time into the garden; feels well-pleased with the result. Pulse 108. Again puffiness about

the face. During the night he complains suddenly to the attendant of præcordial anxiety. The physician, called instantly, finds the body already lifeless; all attempts at reanimation proved useless.

No post-mortem examination could be made.

I have given this case with some detail, because it is a good example of hysteria in the male, chiefly because there have been hitherto but few cases recorded of death during the "weaning cure."[1]

In my case, notwithstanding the cardiac weakness, a general increase of strength had taken place before the death on the 11th day of the cure; the patient felt so well that he sent home his sister, who had come to reside in the neighbourhood on account of him. One could therefore hope that in spite of the unfavourable initial prognosis—we had expected to see the old hysterical attacks return on the removal of the morphia—that the further course would be an easy one.

In such occurrences, where cardiac weakness exists, it is obvious that, as Levinstein points out, one should give the patient a small injection of morphia. But in our case the circumstances were so unfavourable that, in spite of the immediate arrival of medical aid, life could not be saved.

Cases 2 and 3.—One day a thin, pale gentleman came to me, stating that his wife's mind was affected in consequence of abuse of morphia and alcohol. He hoped that on removing the morphia from her the symptoms would abate, and in consequence wished me to visit her. At once I saw from his appearance that he himself was suffering from morphia-poisoning; but I had no time to enter then into that question. A few hours later, before I had visited the wife, he returned in a state of anguish, covered with perspiration, saying that she was in a state of furious excitement, and had driven him from his house, so that he had been obliged to take a room in a neighbouring hotel. He requested that the woman be as soon as possible taken into the establishment; that the

[1] Fiedler, 'Vortrag gehalten in der Gesellsch. für Natur- und Heilkunde in Dresden,' Jan. 1876. Levinstein, 'Die Morphiumsucht,' 2nd ed., p. 233. Burkart, 'Die Morphiumvergiftung,' p. 111.

morphia was ruining her physical energies; that she was a burden to all her neighbourhood, &c.

On my visiting her, I found a strong, blooming, well-nourished lady, of fresh complexion, with whom the pale decrepit man made a strong contrast. I shall shortly give the result of a consultation with two medical men in attendance.

The patient, aged 30, had been married for fifteen years. The husband, who possessed landed estates, and had already begun to use morphia injections, easily persuaded her, almost a child, to do the same as a joke, since everything was to be in common between married people. Since then this matrimonial relationship had been kept up, and the two agreed on no point better than on the common enjoyment of their morphia. It is impossible to estimate the quantity consumed daily, for they obtained it wholesale from manufactories of chemical products.

The physical peculiarities of the couple have been already alluded to; their psychical conditions differ considerably from the normal.

The husband has a mania for buying the most expensive pianofortes from Vienna, Paris, &c., and to change the corresponding portions of the Viennese with those of the Parisian instruments; and so work at them all—chiefly during the night—until they become worthless. He starts on journeys without saying anything about it; and in every particular leads a crazy life. His behaviour is shy, uneasy; he has a marked tendency to lying.

The wife is very excitable, has suicidal ideas; she is occasionally aggressive towards her neighbours; the servants find their condition unbearable. She drinks a good deal of wine, and has no wish to get rid of her morphia habit; she only wishes her husband would behave in a sane manner.

I scarcely need mention that I declined to undertake any measures against morphinism in their case. It is not probable that both could have been cured; and owing to the mutual propensities of this couple, the healthy one would have been sure to return to the syringe out of affection for the other.

Owing to the circumstances that morphinists are prone to insist with other people upon the advantages and pleasures of the drug, it is conceivable that such married couples, addicted to its use, are not rare. Another consideration which made me mention this case here is the fact that both presented psychical abnormities. I might easily adduce further examples in which mental aberrations of a much more marked description were manifest. But this would lead me too far. The point I had in view was to place in evidence the relationship between the abuse of morphia and mental disturbances, especially with reference to how far morphinism may be considered as an etiological factor of disease of the kind.

Levinstein[1] has certainly not put it clearly, for he places the question thus: "Is Morphinism a mental aberration?" He must necessarily come to false conclusions; the differences he draws between the two are in a great measure untenable. I must particularly demur to the statement, that the psychical disturbances caused by morphia cease within a few hours, and affirm that mental diseases arising in the course of morphinism are of the most intractable kind when once fully developed. Not only do they not disappear on depriving the patient of morphia, but they then usually get worse.

Burkart[2] on the other hand lays much more stress, and rightly so, upon the mental alteration displayed by morphinists.

The degree of mental aberration arising from protracted use of the drug is very variable. There may indeed be individuals who retain their power of mind in spite of it; but the number is much greater of those who betray a marked alteration of their intellectual and moral life; and in not a few cases finally the point of distinct aberrations is reached. This usually consists of a depressed state with suicidal tendencies, occasionally with violent excitement and hallucinations. I have never seen a case of progressive dementia paralytica developed during morphinism. Individuals of nervous habit, or with hereditary predisposition, fall an easier prey to mental disease, than those who offer a greater resistance to the influence.

[1] Loc. cit. [2] Loc. cit.

The following two propositions sum up the views developed in the foregoing remarks:

1. A real and persistent result after the "weaning cure" is very often doubtful, or is not obtained; the cure itself may under certain conditions endanger life.

2. In most cases the protracted use of morphia in large doses is followed by psychical alterations of a lasting nature, which may amount to decided insanity.

ON THE CONDITIONS OF THE NERVOUS DISCHARGE.

BY CHARLES MERCIER, M.B. (LOND.), F.R.C.S.

THE endeavour to fix with precision the conditions of the nervous discharge, is encountered at the outset by the almost overwhelming difficulty that there are no means of making a direct estimate of the discharge. The only way in which it is objectively manifested is in the resulting muscular contraction; and all reasoning on the subject, in fact the whole science of neurology, is founded on the assumption that the muscular contraction represents quantitatively the nervous discharge which gives rise to it. In spite of the exigencies of scientific method, which forbid us to postulate that which is capable of proof; and in spite of the immense, the fundamental, importance of this principle, it still remains a pure assumption. The first step in the investigation of the nervous discharge must be, therefore, to discover whether there is indeed any constant adjustment between this discharge and the resulting contraction, and to determine if possible the relation between them.

Now the muscular contraction is a very complex phenomenon, comprising electrical changes, changes of configuration, thermal and chemical changes, besides the mechanical effect which is the main element; and this mechanical effect may manifest itself in various ways. It may take the form of velocity of movement, or that of overcoming resistance, both of which may however be estimated by the Work Done; or it may take the form of sustaining a weight without movement, in which case it cannot be so estimated. The whole of the changes in acting muscle cannot be expressed in any term less general than that of energy. The changes are all mani-

festations of energy, and in almost every case there is a liberation of energy by the muscle.

Of the nervous discharge, on the other hand, it is as yet undetermined whether it is an electric change, or a chemical change, or some other change of molecular configuration, or whether it is a combination of some or all of these, or whether indeed it is not some form of energy with which we are as yet unacquainted. All that can be positively stated is that the nervous discharge is a liberation of energy in some form. Hence it is obvious that the connection between the nervous discharge and the muscular contraction can at present be formulated in terms of energy only, that being the highest common term. And since, while we postulate some connection between the nervous discharge and the resulting contraction, we are so far ignorant of the manner in which they are connected, we are warranted in formulating the most general term in the most general manner only. Such a formula may be expressed as follows:—The amount of energy liberated in the nervous discharge is, *cæteris paribus*, proportionate to the sum of the different forms of energy liberated in the muscular contraction. It is not said that the amount of energy liberated by the nervous discharge is *equal* to the amount liberated in the resulting contraction. It is certain that they are not equal. The statement is that the amounts of energy liberated in two nervous discharges bear an equal *ratio* to the amounts liberated in the resulting contractions; or if a and b represent two discharges, A and B the contractions respectively resulting; then is $a : b$ as A : B. It is evident that the formula will be true only under the restriction of other things being equal, for a feeble or exhausted muscle will need a greater nervous discharge to elicit a given mechanical effect than one that is fresh and vigorous.

It has been approximately proved by physiologists that in similar contractions the thermal, chemical, &c., effects bear a constant ratio to the mechanical effect, and as the mechanical effect is the one which alone is directly open to observation, and moreover with which alone we are directly concerned, the formula above given may be rendered a degree more precise by substituting the mechanical for the total effect of the

contraction. It will then run as follows:—*The amount of energy liberated in the nervous discharge is* cæteris paribus *proportional to the amount of energy manifested in the mechanical effect of the resulting contraction.*

This being the most precise formula which it is possible to construct synthetically, let us now apply it to observed phenomena and see if they are consistent with its negation. Take the simplest possible case of determinate movement, of movement that is, which is determinate in direction, in extent and in force. Every such movement requires the co-operation of at least two muscles. It may at first sight seem that the simplest movements, such as the raising of the upper lid, the turning of the eye to one side, or the depression of a finger, are due to the action of a single muscle only. But this is not so. It has been shown by Duchenne that in order to produce a determinate movement, not only must that muscle or set of muscles act which tend directly to produce the movement, but that muscle or set of muscles which tend to produce the opposite movement must also at the same time be in action, in order that the movement may be even, orderly, and not excessive. It is evident that were the external rectus muscle of the eye, for instance, to act alone and unchecked, it would have but one movement,—to evert the eye to the extreme extent and within certain narrow limits of rapidity. For all intermediate rates and positions, and for the orderly attainment of the position of extreme eversion, it must be balanced and regulated by the simultaneous action of the internal rectus, the visible result being the difference between the actions of the two muscles. This arrangement was aptly called by Duchenne the Principle of the Harmony of Antagonism. It is perhaps most obviously displayed when a muscle or set of muscles is paralysed, the antagonist set being intact. If a patient under such circumstances make a violent effort to perform the movement which would normally be made by the paralysed muscles, the movement which results is the direct antagonist of the movement attempted. If, for instance, a patient with double facial paralysis make a vigorous effort to shut the eyes, the result is that the lids are more widely opened. The reason is, of course, that the contraction of the

antagonists, which under normal circumstances would moderate and render even that of the primary set, is now unopposed and produces actual movement; and not only do the antagonists produce a movement instead of balancing its opposite, but the movement so produced, being unbalanced, is tremulous and irregular. Another proof of the necessity of the action of two opposing sets of muscles to produce a determinate movement is seen in the inability of a patient with dropped wrist to clench his fist. This striking illustration of the principle of Duchenne has been often demonstrated to me by Dr. Hughlings-Jackson.

It may be objected that the arrangement in question, by which a movement cannot be produced by a single contraction, but must be the differential result of the actions of two muscles pulling in opposite directions, necessitates a waste, a squandering of force, which is quite out of harmony with the rigid economy generally prevailing in natural operations, and which is *primâ facie* evidence against it. There would be much cogency in this objection were the above a complete statement of the process; but it is not. It is of course evident that two opposing sets of muscles cannot contract simultaneously. When movement occurs, one set of muscles contracts while the antagonist set elongates, and what the principle requires is not that a contraction of the antagonists shall be overpowered by a stronger contraction of the primaries, but that the elongation of the one set shall be proportionate to the velocity, force, and extent of the contraction of the other. Now physiologists have proved that when a muscle is weighted to a certain extent—over-loaded—and its nerve is then stimulated, the muscle, so far from contracting, *actually elongates*, and the stronger the stimulus sent through the nerve, the greater the extent of the elongation. This, which occurs when an over-loaded muscle receives a powerful stimulus, may well occur also when a muscle loaded to a moderate extent receives a very faint stimulus, and should this be found to be the case, it will be a strong corroboration of the truth of the principle of the harmony of antagonism. Meanwhile that principle is the theory in possession, and the onus of disproof lies with its opponents.

Every determinate movement, then, requires the co-operation of at least two muscles, and each of these muscles must act to a determinate degree, with determinate force, for a determinate time; and the amounts of the force, degree, and time of the action of the one muscle must bear a determinate ratio to the amounts of the force, degree, and time of the action of the other. But these determinate actions in determinate ratio are themselves determined by the nervous discharges to the several muscles. Therefore if these nervous discharges are not in determinate ratio, then any two discharges whatever to the two muscles will determine the same movement, the same in degree, force, and direction. In other words, only one movement can possibly result from the combined action of two muscles. But, as a matter of fact, the number of movements that do so result is infinite. For to consider extent only, the number of positions intermediate between the two extreme positions to which the part can be moved by the actions of the two muscles or sets of muscles, is infinite. Therefore the ratio which the amounts of energy in the nervous discharges to the two muscles bears to the amounts of energy in the mechanical effect of the muscular actions resulting is not indeterminate, that is, it is determinate.

This reasoning, cogent as it is in the case of muscles which are directly opposed in action, becomes much more evidently unanswerable when applied to the cases of muscles and sets of muscles *both* of which contract, producing movement in force and direction the resultant of their contractions.

Taking as a foundation this principle, that the energy liberated in the nervous discharge is proportionate to the mechanical effect of the resulting muscular contraction, it follows that the contraction of a large muscle will require and be set up by a greater liberation of energy in the nerve centre than the contraction of a small one. The centre which represents the movements of the larger muscle must therefore contain a greater store of energy than that which represents the movements of the smaller, and to contain a greater store of energy it must, if both are similarly constituted, be of actually greater size. It is of course not implied that any centre represents a single muscle, nor that the size of the represented

muscles, though probably the chief, is the only condition determining the size of the centre, but, generally, that the larger the muscles, the larger the centre representing their movements.

Speaking generally, the largest muscles, and those the mechanical effect of whose contractions is the greatest, are the most bilaterally acting, and, save in the face, the muscles situated most centrally with reference to the mesial plane of the body, and progress from the mesial plane down the extremities to the periphery, progress to muscles successively smaller and smaller, and more and more capable of differentiated movement. It is natural to inquire whether a corresponding arrangement of the gradations of size does not obtain among the representing nerve centres; and a little consideration will show that it must do so. For the order observed by the sets of muscles in their position from the centre to the periphery, in their sizes and in the massiveness of their movements, corresponds also with their order in the process of evolution. In the lowest vertebrates, the fishes, the muscles of the lowest order in the series, i.e. those which are symmetrically disposed immediately about the mesial plane, and have simple and massive bilateral actions, are almost the only ones existent; and as the appearance of a limb precedes the differentiation of the limb into segments, so must the development of the muscles which move the limb as a whole, precede the development of those which move the segments on one another; and similarly the development of the muscles appertaining to the primary segments must precede the development of those pertaining to the secondary segments.

Pari passu with the differentiation of separate muscles out of pre-existing muscular sheets, must go on the differentiation of nervous centres representing the movements of the new muscles, from pre-existing nervous centres representing the movements of the parent sheet; and when the muscular system is complete, the development of finer movements and more delicate adjustments necessitates the further differentiation of yet smaller nervous centres. And as the new muscle or muscular group will always be smaller than the primary muscle from which it is derived, the new nervous centre

corresponding will always be smaller than the old one, since the size of the centres is proportional to the size of the represented muscles. Now what will be the position of the new centres and series of centres thus differentiated? Three positions with respect to the parent centre are possible;—the central, the collateral, and the peripheral. The position of the centre cannot be central with respect to its parent, for, setting aside more direct reasoning, were it so—were the new centre to be interposed in the path of the discharge of the parent centre to the muscles—then the discharge of the larger centre must pass through the smaller, which would in consequence also be discharged, hence the larger centre could never discharge without the smaller discharging also, or in terms of muscular contraction, the head could never turn without the eyes turning also. Neither can the position of the new centre be collateral, for the space within the cranium being limited, the existing centres must be in apposition, and as the lateral budding of each centre would be opposed by, and meet with resistance from, the lateral budding of surrounding centres, the motion in this, as in all other cases, being in the direction of least resistance, the outgrowth of new centres must in all cases be towards the periphery. Hence, as the order of evolution is from massive movements to fine movements, and from large centres to small, so the gradation in the central nervous system must be from large centres at the base to small centres at the periphery, with intermediate centres intervening.

Of two centres, the smaller will have the greater surface in proportion to its size; that is to say, it will have a proportionately greater number of points at which disturbing agents can act upon it; and hence the same disturbance will act with greater energy upon a small centre than on a large one. That is, the smaller the centre the more unstable is it, other things being equal. But the smaller centre represents the smaller muscle. Hence, when a uniform wave of disturbance passes through the grey matter, the smaller centres will be first discharged, and the movements produced will be those of the smaller muscles. Such a uniform wave is that which accompanies an emotion, and it is an established fact that "it

affects the muscles in the inverse order of their sizes and the weights of the parts to which they are attached." Now suppose a wave of a greater intensity. Its effects will differ from those of the former in several ways. The centres that were discharged by the former will be discharged by the latter, and they will be discharged more completely. For in each centre there must be some elements more exposed to disturbance than others; and again, some must be more highly charged, in a state of greater tension than others. These two states being compounded in various proportions in the different elements of the centre, it will result that certain elements will be more prone to discharge, that is, will discharge under a feebler disturbance than the rest, and that there will be all degrees of proneness to discharge according to the strength of the disturbance and the direction from which it comes. A feeble disturbance will discharge only those elements which are the most highly charged of the most peripherally situated. A stronger disturbance will discharge these, together with the less highly charged of the most peripherally situated, and also the most highly charged of those elements next in order from the periphery. So that a strong disturbance will discharge more completely the same centres that were discharged by the weaker disturbance. This I call the spread of the discharge in intention. Next, it will be evident that the same reasoning which applies to the elements in a centre will apply to the centres in a region, and that while a feeble disturbance will discharge those centres only which are the most favourably situated with respect to the disturbance and most prone to discharge, a stronger disturbance will discharge, in addition to these, certain other centres of *the same order* which are either less highly charged, or at a greater distance from the focus of the disturbance, or both. So that a second particular in which the effect of a stronger disturbance will differ from that of a weaker, will be that the stronger disturbance will discharge a greater *number* of centres of the same order. This effect I propose to call the spread of the discharge in extension. Thirdly, a stronger disturbance will discharge centres of the order next below those discharged by the feebler disturbance. For it has already

been shown that the larger centres, having a less area in proportion to their size, will remain stable under a disturbance which will discharge a smaller centre. Hence the third difference will be that the major disturbance will discharge centres of larger size than those discharged by the minor disturbance. I propose to call this effect the spread of the discharge in depth. Summing up these three important differences, we may say that as the disturbance increases, the discharge becomes more powerful, wider and deeper.

This being the process in the nervous centres, when translated into terms of muscular contraction, the phenomena will be as follows. The least discharge will produce feeble movements of a few small muscles, and as the discharge increases, the contraction of these muscles already affected becomes stronger, to them is added the contraction of other muscles of the same order, and this combined effect will be further complicated by the addition of contractions of the muscles next in order of size. It will be apparent that the conclusion thus deductively reached expresses the identical process established long ago as an observed fact by Dr. Hughlings-Jackson in cases of epilepsy, and set forth at length in a previous paper, "On the Phenomena of Convulsion."[1]

The foregoing reasoning gives an adequate explanation of the Origin and March of clonic centripetal convulsions; and since the conclusions reached follow necessarily upon the assumptions made with regard to the constitution of the nervous centres, and harmonise completely with observed facts, the grounds are extremely strong for believing, first, that these assumptions are true; and second, that convulsions of tonic quality and centrifugal march depend on the discharge of centres arranged in a different manner. Before passing on to this portion of the subject, which will involve the consideration of the extremely complicated question of Pitch, it will be well to touch on certain other elements in the class of convulsions already considered.

The Extent of a convulsion was defined as the degree to which the threefold march had extended, and its index was expressed as the order of muscles which the spasm had reached.

[1] 'Brain,' October, 1881.

Translated into terms of nervous processes, it is evident that the extent of a convulsion depends on its depth, or the spread of the discharge downwards from stratum to stratum of the superposed series of centres. The convulsion of least extent involves only the highest order of centres; that of next greater extent involves this order, and that next below, and so on. Now what determines the limitation at any given stratum in the series, of the process of successive discharges? To answer this question it is necessary to return to the evolution of the nervous centres, and consider an aspect of the process which was for the sake of simplicity not insisted on in a previous page. The nervous centres represent, as is well established, not so much muscles as movements, that is, certain combinations of muscles; and the movements of the great bilateral muscles differ from those of the small peripheral muscles in many respects besides the massiveness which is the most notable distinction. They differ also in precision, in variety, in generality, in complexity and in rapidity, and each of these differences must correspond with and depend on some difference in the structure and arrangement of the corresponding nervous centre, or otherwise we must believe that an effect ensues without a cause. Neglecting for the present the consideration of the other differences, it is manifest that, since each nervous centre represents a movement, variety of movements must necessitate plurality of nervous centres; and the increase in the variety of movements of the muscles from the centre to the periphery of the body must therefore correspond with and depend on increase in the number of centres in the successive strata from the lower to the higher, or, in other words, the smaller the nerve-centres, the more numerous are they. Again, the smaller secondary centre being differentiated out of the larger primary centre, and placed peripherally with respect to it, it follows that the discharge of the secondary centre *passes through* the primary centre on its way to the muscles. Speaking generally, the arrangement of the nervous centres which are concerned in clonic centripetal convulsions may be diagrammatically represented by a compound umbel. Now supposing one of the higher centres, represented by one of the small groups of florets of the umbel, to discharge. The

liberated stream of energy flows in the direction of least resistance,[1] which is the direction in which it has most often escaped, which is downward through the primary centres to the muscles; or, recurring to the diagram, through the successive stalks of the umbels to the stem. If the liberation of energy be very slight, then it may be entirely dissipated before reaching the muscles. In this case no movement will ensue, but the nervous process may have a subjective accompaniment. An "idea" of the movement may be formed in the consciousness. If the amount of energy liberated be somewhat greater, then it will pass through the subordinate centres to the muscles, and set up in them the movement which the centre represents; but the discharge, being still slight, will pass through the subordinate centre without upsetting its equilibrium. Let the discharge become a degree more powerful, and in its passage through the subordinate centre it will act as a disturbing agent, and set up a discharge therein, resulting in movements of muscles of a lower order, as well as of those of the higher. And so with each increase in the draught of energy liberated from the topmost centre, and flowing downwards through successive subordinate centres to the muscles, a centre situated lower in the series is discharged, and the effect of its discharge is added to the muscular contractions previously evoked.

To give a concrete example, movements of the wrist become added to those of the hand, and to these again are successively added movements of the elbow and shoulder. Now suppose that in place of the gradual and orderly discharge which results in normal movement, a rapid and tumultuous liberation of energy takes place in a topmost centre, such as from the severity of the muscular contraction we must suppose the discharge of an epileptic fit to be. In what way will the results of this sudden and excessive discharge differ from those of the normal? The stream of liberated energy will flow downwards towards the muscles as before, and the extra quantity reaching the muscles in a unit of time will produce the excessive contractions of convulsions. Further, the discharge being of high intensity from the first, will rapidly discharge the subordinate

[1] See Herbert Spencer's 'Principles of Biology,' vol. ii. p. 350.

centres through which the liberated energy flows. But this is not all. If the liberation of energy is very rapid, it will not all be able to find an escape downward. The downward channel will be choked by the excessive flow, and the energy, still accumulating above, must pass off by the channels of next less resistance. It will pass off laterally, and if the liberation in the centre is rapid, and the intensity of the laterally spreading energy considerable, it will set up discharge of the collateral centres lying around the original focus in the same plane. Reference to the process of evolution and the diagram of the compound umbel, will show that the discharge of these collateral centres, which of course will primarily flow off downwards towards the muscles, must pass through the same subordinate centre as the discharge of the original focus passes through. The consequence will be that this subordinate centre will receive simultaneously on its peripheral surface the combined impact of the discharges of all its superposed centres, and the resulting discharge of this subordinate centre also will therefore be excessive. It is obvious that the further extension of the spasm must depend on the further extension of this process of excessive discharge. It has already been shown that the extension of the excessive discharge from the original focus to the collateral foci and the subordinate centre, depends on the rapidity with which the energy is liberated in the centre forming the original focus. It is obvious that the rapidity with which the discharge spreads must be due to the same circumstance; hence, if the above reasoning be correct, the Rapidity and the Extent of the discharge must bear a direct ratio to one another. In the paper "On the Phenomena of Convulsion" it was stated as an observed fact that the Rapidity and Extent of convulsions do vary together.

ON THE SPASMODIC PARALYSES OF INFANCY.

BY JAMES ROSS, M.D., F.R.C.P.

THE various forms of paralysis which result from organic disease of the nervous system may, from the clinical standpoint, be divided into two chief groups, named respectively, *atrophic*, and *spastic* or *spasmodic* paralysis. In the atrophic group the affected muscles are flaccid and undergo progressive wasting, the faradic contractility of the nerves and muscles is diminished or lost, the galvanic contractility of the muscles manifests quantitative and qualitative changes, technically called "the reaction of degeneration," and all the reflex actions are abolished. In the spasmodic group the affected muscles are in a state of tension or spasm, their nutrition is maintained, the electrical reactions are normal, and the reflex actions are increased, the deep reflexes being especially exaggerated. If the characteristic features of each form of paralysis were always well pronounced, there could be no possibility of mistaking the one form for the other. But these features are not always well pronounced. In the slighter degrees of atrophic paralysis the muscles do not undergo any perceptible wasting, the electrical reactions are not much altered, and the reflex actions may not be wholly abolished or not available as a test; while in the first few weeks of a spasmodic paralysis muscular tension is not established, and the reflexes may not be exaggerated. But although it is not always easy to decide whether the paralysis in a particular case of organic disease of the nervous system belongs to the atrophic or spasmodic group, yet the distinction is a fundamental one, and ought to be kept steadily before the mind of the observer in every case of paralysis. If we now turn from

the disorders of functions characterising these groups of paralysis to the diseases of structures which underlie them, the distinction between the two is no less clear and trenchant. Atrophic paralysis is caused by disease of the ganglion cells of the anterior horns of the spinal cord, and the upward continuations of these horns in the medulla oblongata, pons, and crura cerebri, or of the efferent fibres which connect these cells with the muscles. Spasmodic paralysis is caused by disease of the large ganglion cells in the third layer of the motor area of the cortex of the brain, or of the fibres of the pyramidal tract which connect these with the ganglion cells of the anterior horns of the spinal cord, and the homologues of those horns in the medulla oblongata, pons, and cerebral peduncles. The two groups of paralysis, named *atrophic* and *spasmodic*, when regarded from the functional or clinical standpoint, may therefore be named respectively *spino-peripheral* and *cerebro-spinal* when regarded from the structural standpoint. But if this nomenclature be adopted, it must be remembered that the spinal axis embraces not only the spinal cord, but extends through the medulla oblongata, pons, and crura cerebri. The fundamental principles of the pathology of both the atrophic and spasmodic forms of paralysis are now well ascertained, but there are several minor but still important problems with respect to them which are still open for discussion.

Both atrophic and spasmodic paralyses are peculiarly liable to occur in infancy. The pathology of the atrophic paralyses of infancy, especially that of the spinal variety, is now well ascertained; but much greater obscurity hangs over the nature of the lesions which underlie the various forms of the spasmodic paralyses occurring in childhood. My object in the following paper is to describe a few more or less typical examples of the spasmodic paralyses of infancy, with the view of clearing up the pathology of these affections, so far as our present knowledge of them will permit.

The spasmodic paralyses of infancy may, from the clinical standpoint, be divided into (1) paralysis of hemiplegic, and (2) paralysis of paraplegic distribution. How far this clinical division corresponds to the anatomical division of cerebral or spinal disease respectively remains to be seen, but we may

say in anticipation that it will hereafter become apparent that many cases of paralysis of paraplegic distribution are of cerebral origin, and ought to be regarded as bilateral hemiplegiæ rather than as belonging to the group of true paraplegiæ.

(1.) *Spastic Hemiplegiæ of Infancy.*—As the pathology of the spastic affections which result from cerebral disease is better ascertained than that of those which may be presumed to be of spinal origin, we shall deal with the former first.

The following case, kindly sent to me by my friend Dr. Cullingworth, may be taken as a good example of the affection known as the spastic hemiplegia of infancy.

Case I.—Mary Ann A., aged 17 years, has been for a long time under my observation, but the following notes were taken on March 2, 1880. The patient is well-developed and healthy. She has been taught to read, but she cannot write. She herself states that she was healthy until she was 9 years of age, when she had a fit, followed by weakness of the left side of the body; but her mother had previously informed me that she had a convulsion, with loss of consciousness, when she was between 4 and 5 years of age, and that the left half of the body has been paralysed ever since. When the patient was 12 years of age she began to suffer from convulsive seizures, which have recurred regularly since that time. The mother stated that the spasms were at first most marked in the left, or paralysed arm, but now the whole body is convulsed during the attack. The fits frequently recur now, the patient having sometimes as many as two or three a day. At other times she may go as long as a whole week without a fit, but in the week following such a free interval they are apt to recur with unusual frequency. Each fit lasts from two to ten minutes.

Present Condition.—The patient looks a strong and healthy young woman. Her head measures 20 inches in circumference, and 7 inches in the longitudinal and 5 inches in the transverse diameter. Her forehead is narrow, but the head is symmetrical, although the right side of the face, including the features and the superior and inferior maxillary bones, is somewhat larger than the left half. There are, however, no distinct traces of left facial paralysis. The left arm and leg are more or less paralysed. The forearm is usually held in a state of

semi-flexion on the arm and strongly pronated, the hand is maintained in a line with or slightly flexed upon the forearm, and the fingers are in a state of semi-flexion, while the thumb is bent inwards under the semi-flexed fingers. The patient can move the left arm freely at the shoulder-joint, but she cannot fully extend the forearm, and flexion of it is limited, while she cannot supinate in the slightest degree. On passive extension and flexion of the forearm some degree of muscular tension is provoked, the triceps reflex is increased, and there is a lively jerk of the forearm when the lower end of the radius is tapped. During repose the left hand and fingers are maintained in fixed positions, but they become the seat of choreoid movements when the patient makes a voluntary effort to grasp an object with the left hand. The choreoid movements consist chiefly of extension and flexion of the fingers, occurring in an uncertain and irregular manner. The patient is unable to abduct the thumb, or to oppose it to the tip of the index-finger. She grasps small objects between the pulp of the thumb and the second phalanx of the index-finger, and during this action the last phalanx of the index and the remaining fingers are strongly flexed. It is observed that the left arm is smaller in all its dimensions than the right. The following are the comparative measurements :—

Left upper arm . . .	9½ inches	—Right . . .	9¾ inches.
Left forearm	9 „	—Right . . .	9¼ „
Left hand	7½ „	—Right . . .	8¼ „
Length of left forearm .	8 „	—Right . . .	8¾ „

Even the left clavicle is one half-inch shorter than the right. The muscles of the left forearm appear to be better developed than the corresponding muscles of the right side, the former being hypertrophied from the constant spasm. The comparative diminution in the circumference of the left forearm appears to be due to a diminution in the size of the bones; this view is confirmed by a comparison of the wrists of the two sides, a great relative diminution of size being observable in the circumference of the left wrist.

The various segments of the left lower extremity are extended upon one another, but the patient can partially flex the leg upon the thigh. The foot is maintained in a

position of extreme talipes equinus. The instep is strongly arched and protruding, the sole is hollow, and the toes are spread out and hyper-extended, the big toe being at right angles to the metatarsal bone. Muscular tension is provoked by passive movements of the limb, the patellar tendon-reflex is exaggerated, but ankle-clonus cannot be elicited. The left foot is cold and blue, as compared with the right one, and the left limb is also smaller in all its dimensions than the other, as the following measurements testify :—

The left thigh measures	16 inches	—Right . . .	17¾ inches.
Left calf	„	11 „	—Right . . .	13 „
Length of the left leg		13¼ „	—Right . . .	14 „

The electrical reactions of the muscles of the left leg are normal.

The patient had an epileptic attack in my presence, but the convulsive movements were general, and I could not perceive that they had any unilateral character. She states that she has no warning of the attack; but she volunteered the statement that when she began to have the fits for the first time, each attack was preceded by a creeping feeling in the left leg and arm.

Tactile sensibility is diminished in the skin of the palm of the left hand, but this is probably due to want of exercise.

There are no other sensory disturbances. The patient's memory has recently failed very much, and she has a stupid and apathetic look.

Remarks.—The clinical history and morbid anatomy of cases of this kind are now tolerably well known. In the majority of cases the onset of the disease dates from the age of from two months to four or five years of age. At this period the child, either with or without a previous illness of a few days, is suddenly seized with convulsions and unconsciousness, the spasms during the seizure being often limited to one half of the body. When the child recovers consciousness it is observed that one half of the body is paralysed; the hemiplegia pursues the usual course, contractures become established, and the limbs are maintained in fixed positions by tonic spasm, or, more frequently, are agitated by choreoid movements.

When the patient arrives at the age of 10 or 12 years epileptic attacks supervene; the convulsions at first assume a unilateral character, the spasms being limited to the paralysed half of the body. After a time the convulsions become general, and it may even be impossible to discover the existence of a unilateral aura. Another interesting feature of these cases is that there is an arrest of development of the paralysed limbs and half of the face, but the electrical reactions of the muscles remain normal. When the limbs are subject to violent choreiform movements, the muscles may even undergo hypertrophy, but in these cases careful measurements show that the bones of the affected limbs are smaller than those of the healthy side.

Spastic hemiplegia of infancy is usually associated with some degree of idiocy. In some cases the mental defect appears to become developed, as in the case just described, as a sequel to the repeated epileptic attacks, while in other cases the intellectual powers are markedly defective from the onset of the disease.

In the variety of the spastic hemiplegia of infancy which is acquired after birth, the lesion is situated in the motor area of the cortex of one of the cerebral hemispheres. The primary lesion appears to consist of a local hæmorrhage or softening, or of a local encephalitis, the latter being sometimes set up by an injury. As a general rule, a puckered cicatrix forms at the seat of the primary lesion, and the surrounding portion of the cortex undergoes a diffused sclerosis with retraction; the motor area of the cortex of the affected hemisphere thus becomes much diminished in size, and consequently the spastic hemiplegia of infancy is sometimes named *unilateral atrophy of the brain*.[1] In addition to the changes occurring in the cortex, the fibres of the pyramidal tract connected with the diseased area undergo a descending sclerosis, and consequently the anterior pyramid of the medulla oblongata

[1] See 'Atrophie partielle du Cerveau,' par J. Cotard, 'Thèse de Paris, 1868; 'Iconographie Photographique de la Salpêtrière,' par Bourneville et Regnard, tome deuxième, 1878, p. 1; and 'Leçons sur les Maladies du Système Nerveux,' &c., par J. M. Charcot, tome ii., Paris, 1877, p. 335; 'Atrophie cérébrale: Hémiplégie; Epilepsie partielle,' par Bourneville et Poirier, 'Progrès Médical,' Paris, 1878, vii. p. 224; 'Atrophie partielle de l'hémisphère cérébral gauche: Épilepsie jacksonienne à forme hémiplégique; mort en état de mal,' par H. d'Olier, 'Progrès Médical,' Paris, 1881, ix. p. 39.

on the side of the lesion is generally found atrophied, while a microscopical examination of the spinal cord reveals a patch of sclerosis in the lateral column on the side opposite to the lesion.[1]

In other cases, instead of a cicatrix with diffused atrophy, a distinct loss of substance has been found on one or both hemispheres of the brain. This condition, named Porencephalus by Heschl,[2] will be subsequently considered at greater length.

Closely allied to the spastic hemiplegia of infancy is the condition described by Hammond[3] under the name of *athetosis* when it occurs in childhood. Unilateral athetosis is, according to Oulmont, a disease of mature age, although a few cases are recorded in which the affection had become established after a hemiplegic attack occurring in infancy. In these cases sensory disturbances have been observed on the affected side, and it is probable that the lesion is situated, not in the cortex, but in the basal ganglia, and that the posterior portion of the internal capsule is implicated. In the bilateral athetosis of idiotic children described by Clay Shaw,[4] Oulmont,[5] and Dreschfeld,[6] sensory disturbances have not been observed; there is an entire absence of a history of an apoplectic attack or convulsions having occurred during infancy, and consequently the affection is probably due to a congenital defect of the cerebral hemispheres, involving the cortex of the brain. The following case, however, shows that bilateral athetosis, or bilateral post-hemiplegic chorea, as it may be called, is not always due to a congenital defect.

Case II.—Ellen L., aged 14 years, came under my obser-

[1] See 'Beitrag zur Kenntniss der "psychomotorischen Centren" im Gehirn des Menschen,' von Dr. F. Neelsen. Deutsches Archiv für Klin. Medicin, Bd. xxiv., Leipzig 1879, s. 483.

[2] See 'Die Porencephalie, eine anatomische Studie,' von Dr. Hans Kundrat. Graz, 1882.

[3] A 'Treatise of Diseases of the Nervous System,' by W. A. Hammond. New York, 1871.

[4] 'On Athetosis, or Imbecility with Ataxia,' by T. Clay Shaw, M.D. St. Bartholom. Hosp. Reports,' vol. ix. 1873, p. 130.

[5] 'Étude Clinique sur l'Athétose,' par le Dr. Paul Oulmont. Paris, 1878.

[6] 'Sur quelques cas d'Athétose,' par J. Dreschfeld. 'Rev. Mensuelle de Méd. et de Chir.,' Paris, 1878, ii. p. 766. See also 'On Athetosis and post-Hemiplegic Disorders of Movement,' by W. R. Gowers, M.D., 'Medico-Chirurgical Transactions,' vol. lix., 1876, p. 271.

vation towards the end of 1881. Her father died a few years ago somewhat suddenly. The mother had nine children, but all of them are dead except two. Four of the children died of cholera in St. Louis, in America, and two died of scarlet fever. No neurotic tendency can be discovered in the families of the parents, and there is no evidence of congenital syphilis. The patient was a fine, well-nourished infant, and was in every respect healthy until she was two years of age. The mother states that at this age the child was very intelligent and quick, and could talk much better than is usual with children of that age. At this time she fell out of her perambulator, and struck the left side of her forehead on the ground. The skin was not cut, but the part struck was swollen and contused. The child was supposed to have completely recovered from the results of the accident, but it was observed that on being frustrated she was liable to have an attack of what seemed to be an exhibition of temper. During these attacks she would hold her breath, her hands were clenched, and her face became of a blue colour. Each seizure lasted about five minutes, and on recovery, the child's hands were noticed to be flaccid and powerless. Eight months subsequent to the fall the child was sitting at the table, and, on being refused some ham, her mother observed that she seized hold of a knife which lay beside her; immediately afterwards her hand became clenched round the handle, while the blade of the knife was clutched by the other hand, and twenty minutes elapsed before the knife could be extricated from her hands. During this time the body was stiff, the eyes were drawn, the teeth were clenched, and the mouth was covered with froth. The first seizure is said to have lasted five hours. The convulsions continued to recur for thirty-two consecutive days, and the interval between the fits did not average more than ten minutes. The mother states that between the attacks the child was conscious, and could utter such short sentences as, "Ma; I want to drink;" and it was not until three months afterwards that she lost the power of speech completely. It was nearly two years after this attack before the child began to walk again, but the opinion of the medical attendant was that the legs were not paralysed, and that the want of

power of walking was due to general debility. The mother is unable to give a good account of the condition of the upper extremities, but it is evident that they were not observed to be completely paralysed after the attack, although she thinks they were decidedly feeble. On the cessation of the general convulsions the spasms became limited to the upper extremities, recurring in them two or three times each day, but it was not until the child was five or six years of age that the clonic spasms became more or less continuous during waking hours.

Present Condition.—The patient is a strong and healthy-looking girl of fair complexion. Her height is 5 feet. She has not yet menstruated, but the mammæ are well developed. The lower extremities are in every way of normal appearance,

FIG. 1.

and the muscles are well developed, but the patellar-tendon reflexes are absent. Both the upper extremities are subject to choreoid movements associated with great muscular tension. The right forearm is usually held slightly flexed on the arm; the movements of pronation and supination, flexion and extension of the right forearm are awkward, executed with difficulty and only to a limited extent; the fingers are usually held flexed at the metacarpo-phalangeal, and extended at the

phalangeal joints, but the relative positions of the forearm, hand, and fingers, are constantly changing. The left forearm is usually held at right angles to the arm, the hand is strongly flexed on the forearm, and the fingers are held, like those of the right hand, in the interosseous position. The choreoid movements are even more exaggerated in the left than in the right upper extremity, and with the view of restraining these movements to some extent the patient habitually carries the left arm behind her. In this position the upper arm is abducted and retracted, the forearm is bent at right angles to the arm, and is thus carried across the back, the back of the wrist is firmly held against the vertebral column in the lumbar region, and the palm of the hand is directed backwards, while the fingers are still agitated by choreoid movements.

The face is variously contorted by a more or less constant clonic spasm of the muscles supplied by the upper and lower branches of the facial nerves. The expression of the face is thus continually changing. While under examination, the expression of the face is frequently indicative of grief or pain, but even this expression, which is probably the predominant one, is often transient and succeeded by a gay and lively expression, so that the effect is by no means unpleasing. The muscles of the neck, especially those of the back of the neck, and the muscles of the back in the dorsal and lumbar regions are implicated in the spasm, and consequently the body is frequently arched backwards as in tetanus, the lumbar curve being very marked.

The patient is unable to speak. The mother states that her daughter can sometimes say "Ma," and that she occasionally attempts to say "Yes," "No," and "Good-night," but that is the extent of her spoken language. She can, however, read with facility; she has taught herself to write, and wrote her name in my presence fairly well, notwithstanding the choreoid movements with which her hand was agitated. She is very fond of romping with other girls, dances well, has a taste for music, has a good idea of time, and she has even taught herself, the mother states, to play the piano. By the aid of facial expression and pantomime she can make her mother

and sister understand all her wants, and indeed she has very little difficulty in making her ordinary wants known to any intelligent person.

When examining her for the first time in the out-patients' department in the Infirmary, I asked her mother to undo her dress, so that I might examine the upper part of her chest. The patient was surrounded by several students, and two or three of the out-patients were standing in the doorway of the apartment, the door being open at the time. As her mother proceeded to obey me, I noticed that the girl had a distressed expression of face. I then observed her nudge her mother with her right elbow, and when she thought that she had attracted her mother's attention, the right angle of her mouth on the right side, which was nearest the door, was drawn downwards and outwards, the lower jaw and chin were also depressed, and the tip of the tongue was thrust out between the compressed lips near to the depressed angle of the mouth on the right side. The patient at the same time glanced at the door, this movement being accompanied by a significant arching of the eyebrows. I immediately called out, "Oh, she wants the door to be shut," and felt mentally reproved for not having ordered it to be closed before requesting her to be undressed. I have frequently observed a minor degree of the same expression in women under circumstances calculated to induce a feeling of shame.

Remarks.—In the case just described, there can be little doubt that a lesion exists in each hemisphere, that the two lesions occupy more or less symmetrical position, and that they became established when the patient suffered from convulsions at two years of age. A lesion involving the operculum on each side would embrace the centres of speech, of the movements of expression, and of the special movements of the hands; these being the functions chiefly disordered. It is also likely that a descending degeneration of the pyramidal tracts would be found in the spinal cord. The following case of aphasia occurring in infancy may be worth recording here, even although the patient is not the subject of any form of spastic paralysis.

Case III.—Lillie H. at eight years became an out-patient

in the Manchester Royal Infirmary under my care, on May 4, 1882.

The parents of the child are healthy, and there does not appear to be any tendency to nervous disease in the family. The mother has had eight children, only four of whom are living and healthy; the remaining four died in infancy, but the causes of death are not well known. The mother had a tedious labour when she was confined of the subject of this notice, and was delivered by instruments, but the child did not suffer any external injury. The day after birth the infant was seized with convulsions, and these continued to recur for the next six days, during which little or no food was taken. On the seventh day after birth she took a little of the breast; but the mother states that she did not suck with the same strength as the other children, and that the milk frequently dribbled from the mouth as if she had some difficulty in swallowing. During the fits the infant's countenance was fearfully contorted, and she screamed violently, but the mother never observed any indication of the face being drawn or unsymmetrical after the convulsions ceased. During the early years of childhood the saliva dribbled very much, and even now the patient is never quite dry about the mouth, although she has improved very much in this respect during the last twelve months. The patient never had convulsions at any period subsequent to the first week after birth. The parents did not observe that there was anything seriously wrong with the child until it was time for her to speak, and although she is now eight years of age and quite intelligent, she has never been able to speak a single word. The mother never noticed that the movements of the hands or fingers were awkward, but it was observed in cold weather that her hands were unusually liable to become cold and blue, and to break out with chilblains, so that they had always to be kept carefully wrapped up in winter.

Present Condition.—The subject looks a fine healthy girl, 49 inches in height, and stout in proportion. Her head is well-formed, but the forehead is somewhat narrow. The lobules of the ears are large but well-formed, the roof of the mouth is not excessively arched, the tongue is well-formed, and the

patient can move it in all directions. The senses of sight, hearing, and smell are perfect. The child is liable to fits of temper, and during those times she is very strong, struggles violently, and screams loudly. There are no indications of facial paralysis or spasm; the movements of her hands and fingers are well co-ordinated, and there is not a trace of tension in the muscles of any of the extremities. The child's face is bright and intelligent; she can sing, is proud of joining the Sunday School scholars in singing hymns, and has a good notion of time. She is very fond of playing with other children and is a great favourite with them; she is, however, quite unable to speak a single word, but is able to make her wants known to some extent by pantomime and facial expression. Her hands are of a deep red colour and very cold, but there are no trophic changes.

Remarks.—In this case the infant suffered from convulsions and unconsciousness for the first few days after birth, this being the condition known as asphyxia neonatorum. In such cases labour is usually tedious, and the mother is frequently delivered by the aid of instruments; there can be little doubt that in many such cases the brain suffers some injury during delivery.[1] It is probable that in this case a localised hæmorrhage had taken place in the cortex of each hemisphere, and that the damage done was limited to the posterior parts of the third frontal convolutions. It is doubtful whether any degeneration of the pyramidal tracts is present.

In the following case the patient was also the subject of asphyxia neonatorum, and in all probability the paralytic condition became established at birth.

Case IV.—James W., æt. 7 years, has been under my observation for the last five years, but the following notes were taken on May 16, 1882. The mother had a natural but very tedious labour during the birth of the child, and immediately on being born the infant was seized with convulsions. The convulsions

[1] See "On the influence of Abnormal Parturition, Difficult Labours, Premature Birth, and Asphyxia Neonatorum on the Mental and Physical Condition of the Child, especially in relation to Deformities," by W. J. Little, M.D. 'Obstetrical Transactions,' vol. iii., London, 1862, p. 293.

continued with scarcely any intermission for the first three days after birth, and during this time the infant took no notice of surrounding objects, and no food could be administered. On the third day the convulsions ceased, and a little milk and water was administered in small teaspoonfuls at a time, but it was observed that the infant had difficulty in swallowing, and dribbled very much. It was not, however, until he was six months of age that the mother became fully convinced that the child's power of swallowing was unusually defective. When the child was five months of age it was observed for the first time that his arms and hands were held in unnatural positions; the forearms were at that time dragged behind the body, and the legs were stiff and powerless. At three years of age the child suffered from a slight convulsive seizure, but the mother thinks that he was not any worse after that attack than he was before it.

The parents of the child have had four other children, and all of them are alive and healthy; there is no history of syphilis, and it does not appear that there is any special tendency to nervous disease in the families of either parent.

Present Condition.—The patient is a well-developed boy for his years, he has a fine ruddy and healthy complexion, and his internal organs are all healthy. The boy is unable to walk without support, his lower extremities being partially paralysed. The various segments of the lower extremity are, with the exception of a slight degree of permanent flexure at the left hip and knee-joints, usually extended upon one another, and the feet are held in the positions of extreme talipes equino-varus, the deformity of the left being the more marked of the two. When the boy is supported in the erect posture, the distortions of the feet become more marked, the heels being drawn upwards, so that only the tips of the toes touch the ground; and when he attempts to walk, the adductors become strongly contracted, so that the advancing leg crosses over to the opposite side and the foot is always planted on the ground in front of the other. During attempts at station and locomotion, the right heel comes close to the ground, but the left heel is three inches removed from it, and the foot is held in an almost vertical position. The

muscles of the lower extremities are very tense, and become tenser during attempts at passive movements of the limbs. The patellar tendon-reflex however, is not markedly increased, and ankle-clonus cannot be elicited. The upper extremities are stiff, and maintained in comparatively fixed positions. The elbows are habitually held removed from the body. The forearms are semi-flexed on the arms, and the patient is unable to fully extend either of them, and it is even difficult to extend them fully by passive movements, and during such efforts considerable muscular tension is provoked. The left hand is slightly flexed on the forearm, and the fingers are held in partially flexed positions, although these positions are constantly changing, owing to choreoid movements. These movements are not altogether absent during repose, but they are much increased during voluntary efforts to grasp an object. The muscular rigidity is not so pronounced in the right as in the left upper extremity, and choreoid movements of the hand and fingers are only present when the child makes an effort to grasp a small object. The chin is directed to the left and upwards, and the head inclines to the right, the right sterno-mastoid muscle being more prominent than the left. The child appears to have some obstruction in the nose, and he breathes with his mouth open. His saliva dribbles slightly, he has considerable difficulty in swallowing, and fluids are specially apt to enter the glottis, causing paroxysms of distressing cough. The face is symmetrical, and the expression is bright and intelligent, but the patient cannot protrude the tongue beyond the lips, and during efforts at articulation the tongue may be seen to be contorted in various directions by muscular spasm. The boy goes to school, and keeps up with his class, and his memory is said to be particularly good. His command of language is as copious as that of any other boy of his age, but he stammers so much that it is difficult for a stranger to understand a word he says. During his attempts at articulation the muscles of the tongue and the orbicularis oris are the subjects of clonic spasms. During sleep the tension of the muscles of the extremities and the choreoid movements of the left hand completely disappear.

Remarks.—The question of the localisation of the lesion in this case presents many difficulties. The severe convulsions which had occurred during the first three days of extra-uterine life would seem to indicate that the lesion was situated in the cortex of the brain. It must, however, be remembered that the affection of speech present in this case is a difficulty of articulation, or a dysarthria, and not a true aphasia, as in the last two cases reported. It is, therefore, probable that there exists in this case either a lesion in the lenticular nucleus of each hemisphere, involving the internal capsules, or a single lesion in the pons, partially injuring the pyramidal tracts in their passage through it.

The spastic hemiplegia of infancy, however, is not always established at birth or in early childhood, but it is sometimes congenital. The following case is a very good example of the congenital variety:—

Case V.—Mary F., aged ten years, was admitted into the Manchester Royal Infirmary under the care of Professor Lund.

The patient is a well-developed and healthy-looking girl. Her height is 49 inches, and she is stout in proportion. She has been brought up in the Withington workhouse, and has been sent to school; she knows the letters of the alphabet, but cannot read. The child's mind is not quite bright, although she answers questions about herself with readiness and a fair amount of intelligence.

There is a defect in the right parietal bone (Fig. 2), said to have been in existence since birth, through which the brain can be seen to pulsate. The defect in the bone is situated immediately above the most prominent part of the parietal eminence. It constitutes a lozenge-shaped space, which is 3 inches in the antero-posterior, and 2 in the vertical or transverse diameter. The edge of the bone forming the margin of the opening stands out clearly and well-defined under the scalp, but the whole of the floor of the space is not uncovered by bone. It would appear as if the inner table of the skull had grown inwards over the space to a much greater extent than the external table. The consequence is that when the finger of the observer is placed over the depression it is

found that there are only one or two small spots over which the brain can be felt to pulsate, and which are completely uncovered by bone. The visible pulsation, however, appears to be co-extensive with the area of depression.

The patient has a fairly intelligent expression, and no difference can be detected between the two sides of the face. The left upper extremity is partially paralysed, and is rather smaller in all its dimensions than the corresponding extremity of opposite side. The following measurements were taken :—

Left arm, about its middle, measures 7½ inches in circumference, and right, 8¼ inches.

Fig. 2.

Left forearm measures 7 inches, and right 8 inches, one inch below elbow-joint.

The length from the margin of the acromion process to the external condyle of the humerus, is 9¼ inches on the left, and 10¼ inches on the right side. The length of the left forearm is 7½, and of the right 8 inches.

The left lower extremity is also somewhat feeble, and mea-

sures less than the corresponding parts on the right side. The circumference of the left calf at its thickest part is 9¼ inches, and of the right 10 inches; that of the left thigh at its thickest part 14 inches, and of the right 15 inches.

The left arm is freely movable at the shoulder-joint, but the forearm cannot be fully extended at the elbow. The forearm is usually held at right angles to the arm and strongly pronated. The patient cannot supinate the forearm, nor can it be supinated by passive movement. The extensors of the wrist and fingers are feeble, and consequently the hand and fingers usually hang in a helpless position, as if they were depending from the forearm, just as occurs in lead paralysis. The grasp of the left hand is feeble as compared with that of the right hand. The left forearm jerks strongly when the lower end of the radius is tapped. The patient drags the left foot slightly in walking, although there is no deformity of the foot, but the patellar-tendon reflex is slightly exaggerated on the left as compared with the right side.

The skin over both the left extremities is always of a blue colour, and the surface is cold, just as occurs in infantile paralysis. This condition of the skin begins in the upper extremity, about the middle of the arm, the normal skin being separated from the congested portion by a sharply-defined line which slants downwards and inwards towards the elbow.

The muscles of both the left extremities react to a weak faradic current. This test was specially applied to the extensors of the wrist and fingers, which were the most paralysed, and reactions were obtained to feeble current.

The patient enjoys very good health, and is able to eat and sleep well, and there is no evidence of any disease of the internal organs.

Remarks:—A somewhat similar case to the one just described has been reported by Meschede.[1] The subject was a woman, aged 27 years, who had left hemiplegia with contracture from early infancy; the affected extremities were of a cyanotic colour, and smaller than the corresponding healthy limbs; epileptic seizures became established at puberty, speech was stammering,

[1] Virchow's Archiv, Bd. 34, s. 305.

and the patient was idiotic. A defect, 3 inches long and from ½ to 1 inch broad, of the right parietal bone was present, the floor of the opening being closed by a fibrous membrane. At the post-mortem examination a cavity, 3 inches long, 2¼ broad, and 2½ in depth, and filled with a clear serous fluid, was found in the right hemisphere immediately underlying the defect in the bone. This cavity communicated with the lateral ventricle, the septum ventriculi was defective, and both ventricles were so much distended by the fluid, that the hemispheres and basal ganglia were greatly compressed. The cerebellum was smaller than usual, the right hemisphere being smaller than the left. The walls of the cavity were found on microscopical examination to consist of a soft, wavy, fibrillary tissue, in which a yellowish-brown and rust-coloured pigment was accumulated, thus showing that the cavity was caused by some destructive process, and was not simply due to an arrest of development. No mention is made of the condition of the medulla oblongata and spinal cord, and consequently it is not known whether any descending degeneration was present. But in a case of congenital paralysis with contracture of all the extremities in a child 6 months of age, reported by Kundrat, in which a porencephalous defect was found in the left hemisphere of the brain in the neighbourhood of the Island of Reil, the crura, pons, medulla oblongata, and spinal cord were carefully examined. The left crus cerebri and the left half of the pons were smaller and flatter than the corresponding parts on the right, but this condition might have been caused by the compression of the fluid which distended the cavity in the hemisphere and the lateral ventricles. The spinal cord appeared nearly normal to the naked eye, but on microscopical examination it was found that the lateral columns were completely undeveloped.[1] Portions of the walls of the cavity were formed of cicatricial tissue, showing that the cavity itself was caused not simply by an arrest of development, but by some destructive process. It is interesting to observe that under such circumstances the pyramidal tracts were simply undeveloped instead of being, as one would expect, in a state of sclerosis. These considera-

[1] 'Die Porencephalie, eine anatomische Studie,' von Dr. H. Kundrat. Graz, 1882, s. 45 und 101.

tions, therefore, render it probable that there is, in the case which I have just reported, a porencephalous defect in the right hemisphere, immediately underlying the defect in the bone, and that the fibres of the pyramidal tract which would have sprung from the cortex of that situation in a healthy brain are absent.

(*To be continued.*)

LOCALISED CONVULSIONS FROM TUMOUR OF THE BRAIN.

BY J. HUGHLINGS-JACKSON, M.D., F.R.S.

Physician to the Hospital for the Epileptic and Paralysed, and to the London Hospital.

THIS is the case of a patient who for twelve years was subject to occasional convulsive seizures, beginning in his right foot. Post-mortem, there was a tumour in the cortex of the left side of the brain, as shown in the two drawings. For these drawings and much more I have to thank Dr. James Anderson. The case belongs to a class which was described by Bravais in 1824. Their pathology has been greatly cleared up by Bright, Todd, Wilks, Ferrier, and others in this country; and by Hitzig, Charcot, Lepine, Landouzy, and others abroad. A valuable case is reported by Dr. Franz Müller of Graz.[1]

On October 15, 1872, the patient, then 30 years of age, was admitted under my care in the London Hospital. He had had fits about two or three times a week over a period of two years; their duration, he said, varied, lasting five or ten minutes, but sometimes an hour. Perhaps when, as he said, he had a fit for an hour, there was a succession of convulsions, but it is quite certain that one fit may last many hours—may affect the whole of one side for hours, the patient remaining conscious all the time. The patient gave the following account of his attacks. After some dizziness his right big toe begins to "cramp," and gets drawn towards the sole of the foot, then he feels a twitching of the leg, chiefly of the calf; the leg and knee are drawn up. Most of his fits stop here. He called these "weak fits," others he called "strong fits." In the latter the arm is affected, but always

[1] 'Trans. International Medical Congress,' vol. ii. p. 15.

after the leg; the spasm begins in the fingers and passes up the arm; his arm and hand may be drawn above his head; his mouth works about. During this further range of convulsion the leg continues working. After a severe fit he loses consciousness: he knows nothing about affection of the left side, possibly because he is unconscious.

He remembers his first attack; he was astonished and amused, so he said, by the lifting of his right leg when standing talking. Soon he fell to the ground, both right limbs "working;" in this attack he lost consciousness. Some patients suffering from seizure of this class are subject to quasi-trifling limited seizures before they have one which they think severe enough to make them consult a doctor.

He had many attacks of the two degrees when in the hospital in 1872, but none were witnessed; he lost consciousness in the stronger seizures. Occasionally also he had attacks of dizziness, with apparent movement of objects and dimness of sight, but with these there was no limb affection.

I saw him several times in 1872 when he was an out-patient; he always gave the same account of the starting-point of his seizures, and so he did on a second admission, September 1875, and on his third, January 28, this year.

Second Admission.—In 1875, my then house physician, Mr. Richard Atkinson, saw several seizures, and noted some important facts as to the condition of the limbs when the seizures were over. On his admission he was examined carefully and in great detail for paralysis. None was discovered. The man said he was weak on the right side after his fits. One was seen, September 1, by Mr. Atkinson just after an examination revealing no paralysis. There was first hurried respiration, and then tonic movements of the right hand and arm and right side of face, then clonic spasm. Paralysis of the arm was very marked after the fits; the convulsion began in the leg, but nothing is said of its movements in the notes, as the fit so described occurred, as I suppose, when the patient's legs were covered in bed. Mr. Atkinson saw an attack on September 2; it set in during a conversation. The patient began to answer vaguely, and ceased speaking; his legs were seized with tonic spasm; their muscles felt hard and

rigid; the right leg was the one more affected, the left in a much slighter degree. In about half a minute the clonic spasm began, the contractions became fewer and fewer, and in about a minute from the onset ceased. In this fit the legs only were engaged. The patient denied that his left leg was affected, and said it appeared to be so from movements of his body. But affection of the left leg along with greater spasm of the right was observed four times. On another occasion the man said the stiffness of the left leg was voluntary. Probably Mr. Atkinson was right. In another patient[1] I saw such a fit; the spasm, starting in the left foot, affected the whole leg and slightly the left arm; the right leg vibrated rhythmically.

To return to the present case. Observations as to the leg in which the spasm does not begin are important. It is quite certain from the facts of "descending Wallerian wasting," that each side of the brain sends fibres to both sides of the cord. It is not very rare for both legs to be affected in convulsions of this class starting in one arm. Gowers has recorded such a case. But since the fibres sent from one side of the brain into the cord lie in different columns of the cord, one would *à priori* expect some difference in the seizure on the two sides. On one occasion the spasm, Mr. Atkinson observed, was such that the right leg was flexed at the knee and the left at the ankle. On Sept. 3, Mr. Atkinson saw another fit just the same as the one detailed, and after this he found more loss of power in the right leg, as he did after others; the patient was not unconscious in this seizure. He had many fits of this kind. He had twelve in one night, of the exact range of which we know nothing, and one night so many that he did not count them; the morning after these numerous fits the only movement of the leg remaining was slight flexion of the knee; he could not lift it from the bed; sensation was unimpaired. Often his fits began by inability to talk, probably aphasia. On one occasion he was unable to speak for three minutes and a half, but no spasm was then observed; the patient, however, felt "twittering" in his right great toe. He had at other times fits in which, as in the one on Sept. 1, the arm became affected after the leg; but on their cessation Mr. Atkinson found only

[1] 'Medical Times and Gazette,' Feb. 12, 1881.

paralysis of the leg, not of the arm, as on Sept. 1. It is not, however, stated whether the man's hand was tested in delicate operations.

I remember nearly making a mistake in saying there was no paralysis of the left hand, after a seizure beginning in the left leg, because the patient seemed to grasp as strongly with the left as with the right hand. But I found, a little later, that he had great difficulty in using the left hand in delicate operations.

To return to the case, the subject of the paper. Facial paralysis was never noticed. He had fits daily, sometimes twenty or thirty. On Sept. 11, having had but two fits the day before, and none since, he could move the leg better, but could not walk unaided.

On Sept. 17 he ceased to have fits; his paralysis passed off, but unfortunately there is no note about the date of its disappearance. On Oct. 15 there is the statement that there was no paralysis at all. On the 20th he went out. On Dec. 21 he came to see a surgeon for fistula; he had had no fit for ten weeks; there was no trace of paralysis.

Third Admission.—He came in again Saturday, January 28, 1882, hemiplegic of the right side. Mr. Coates, my present house physician, got just the same account of the starting of the fits as we did in 1872 and in 1875. The patient said that he rarely became unconscious; but when conscious in them, could not speak; he could at the same time recognise people, and understand what they said. He was not paralysed on Thursday the 26th, having on that day helped his father, an undertaker, in a funeral. He was found paralysed on Friday morning, the 27th. Whether he had a fit or a series of fits in the night or not was not ascertainable. His face and tongue were normal; his speech good; the right arm was quite paralysed, and the only movement of the right leg was that he could, when lying in bed, draw it up slightly. Superficial reflexes (in front) were not obtained on either side. There was ankle-clonus on the right side only; the knee, wrist, triceps and biceps' jerks were greater on the right side. So much for his nervous condition. His temperature was 104½, resp. 40; and there were the physical signs of right-sided pneumonia. He died on Monday, January 30.

At the autopsy the right lung was found to be consolidated. A tumour was seen on the surface of the right cerebral hemisphere.

The following is Dr. James Anderson's report of his examination of the brain :—The brain has been preserved in spirit. Part of the dura mater has been left in the middle line opposite the fissure of Rolando. Both this and the rest of the meninges present a normal appearance, except in front of the upper half of the fissure of Rolando, where the pia mater is dark brown in colour from extravasated blood. Opposite the posterior extremity of each superior frontal convolution in the longitudinal fissure is a marked depression corresponding to a Pacchionian body. The pia mater detaches readily from the surface of both hemispheres, including the area of extravasation mentioned above

After removal of the pia mater the surface of both hemispheres presents a perfectly normal appearance, except an area on the upper surface of the *left hemisphere*, including *the posterior half of the superior frontal convolution* and *that portion of the ascending frontal convolution from which it arises*, viz. here *the upper half*. About half an inch of the upper extremity of the ascending frontal convolution is normal in appearance. In the centre of the above area is a hæmorrhage about the size of a sixpence (Fig. 1, E), and round this to the area of a crown-piece are numerous miliary hæmorrhages. The whole area has a wrinkled (from numerous blood-vessels grooving it) nodular aspect, while the fissures are almost obliterated (by adhesion of their walls) and the convolutions are depressed. The area is sharply limited by the *fissure of Rolando* posteriorly, the *superior frontal fissure* externally, and by the *longitudinal fissure* internally; while anteriorly the above appearances shade off gradually and as stated, the anterior half (in length) of the superior frontal convolution is to all appearance normal. The fissure of Rolando and the superior frontal fissure are both deep, the latter being prolonged back into the former. The portion of the cerebrum thus defined is harder to the touch and more resistant, especially at and internal to the central hæmorrhage, than the rest of the brain. *In the longitudinal fissure* the

same wrinkling, nodulation and flattening of the convolutions exist opposite the above area *down to the level of the calloso-marginal fissure*, not however so marked as on the superior surface. The area included by the tumour (Fig. 1, A, B, C, D) is manifestly larger than that corresponding to it on the right side. The length of A, B (Fig. 1) is $2\frac{1}{4}$ inches, while that of A′ B′ is $1\frac{1}{2}$ inches.

A transverse vertical section through the central hæmorrhage (E), that is, through the posterior extremity of the superior frontal convolution, shows the tumour to be limited in the deeper parts also to the area above defined. The tumour cuts stiffly, and about an inch below the central hæmorrhage is a calcareous mass about the size of a pea (Fig. 2, F). The

FIG. 2.

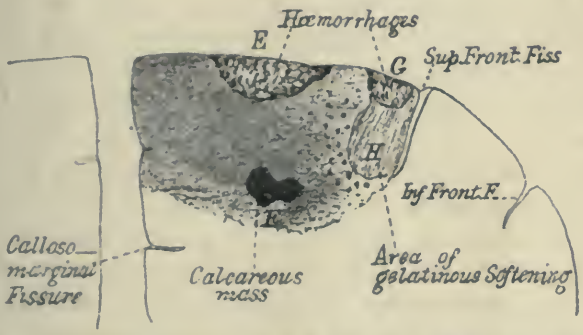

FIG. 2. TRANSVERSE VERTICAL SECTIONS OF LEFT HEMISPHERE THROUGH THE JOINT MARKED E IN FIG. 1. THE DEGREE OF HARDNESS IS APPROXIMATELY INDICATED BY THE DARKNESS OF THE SHADING.

great bulk of the hard part of the tumour lies above and internal to this calcareous nodule, and is of a dull grey colour. Throughout the cortex are numerous miliary hæmorrhages; the central hæmorrhage (Fig. 2, E) extends to the depth of half an inch; a second smaller hæmorrhage (Fig. 2, G) lies about 1 inch external to it, and below this is a patch of gelatinous softening (Fig. 2, H) in the inner wall of the superior frontal fissure. The outer wall of that fissure, and the neighbouring brain-tissue outside the area defined on the surface, presents in successive parallel transverse vertical sections a perfectly

normal colour and consistency. On laying open the interior of the brain in the usual way, the ventricles were found of normal size, the choroid plexuses and velum interpositum were gorged with blood. The ganglia at the base of the brain, as also the pons, medulla oblongata, and cerebellum, were to all appearance perfectly normal.

Microscopic examination shows the tumour to be a glioma composed of round and spindle cells, but varying much in the amount and arrangement of these at different parts. At certain points, especially externally, there is extensive fatty change, and in that part beginning external to the central hæmorrhage the tumour contains numerous calcareous particles ("brain sand"). The tumour shows numerous recent hæmorrhages. Microscopic examination of the tissue of the ascending parietal convolution shows that the tumour does not extend behind the fissure of Rolando.

Remarks.—This man's fits always began, so far as I was told, in the foot, and affected the leg first and most. But he was several times under the care of Dr. Stephen Mackenzie, who tells me that the man declared in 1877 that the fits usually began in the right hand. Dr. Mackenzie feels confident that for the last two years of the man's life the fits began as frequently in the right hand as in the right foot. That the disease was in the mid-region of the brain (the so-called motor region) is in accord with many observations. The difference in the starting-points of the spasm, sometimes foot, sometimes hand, renders the case of comparatively little value for precise localisation. Even supposing that the fits had begun either always in the hand or always in the foot, the locality of the tumour is neither in accord with other observations I have made, nor with the recent experiments of Hitzig and Ferrier. It is of little use saying anything on localisation in such a case, and therefore I omit what I said when reading the case before the Medical Society of London before I was acquainted with the facts Dr. Stephen Mackenzie supplied me with. Besides, the damage by tumours is mostly widespread, coarse and indefinite. The experiments of Hitzig and Ferrier, and the researches of Charcot on cases of monoplegia from limited cortical softening, are more definite for purposes of localisation than anything

we can conclude from the doings of most cases of cerebral tumours.

Now for clinical facts.

The duration of the case, considering that we found a gliomatous tumour post-mortem, is noteworthy—about twelve years from the first attack. That there was tumour all that time I cannot know. One of the cases of fits beginning in the right leg I have recorded was that of a man who was under my observation three years, and he had had fits of just the same kind seven years before. Ten years from the first fit we found cerebral tumour post-mortem. Moreover in that case there were none of the common symptoms of tumour until about a month before he died (when he had double optic neuritis), not even severe headache. Dr. Young, of Aldershot, diagnosed tumour of the cerebellum in a boy whom he sent to me in 1870. This patient died from meningeal hæmorrhage the result of a fall seven years later; there was a tumour of the cerebellum.[1]

Now I speak of the ordinary symptoms of local gross brain-disease.

On the first admission (1872), the patient said that since the fits, that is, for two years, he had been subject to severe headache; for some time the pain was over the left temple, but mostly all over the head. But I do not think it was very severe, not of severity enough to warrant the diagnosis of local gross organic disease as the cause of his convulsive seizures, and on his next admission (1875) he said he did not often suffer from headache. At none of the three admissions was anything found wrong ophthalmoscopically. Nevertheless it is possible that some time during the six years or so I did not see him he had double optic neuritis, and got rid of it. I have known a man who had fits beginning in his left hand who got rid of optic neuritis entirely, and of his fits too, for ten years at least. I saw him to-day. This was a syphilitic case. But neuritis has come on and disappeared in the case of a patient who had fits affecting mostly one arm, and who died with gliomatous tumour of the brain.

The question comes, What would one think of the patho-

[1] 'Brit. Med. Jour.,' May 17, 1873, and March 24, 1877.

logy of the case of a patient who had only fits of this kind and no headache, or no severe headache, and no affection of his optic nerves? Perhaps we may say that, as a matter of fact, most seizures of this kind are owing to syphilitic brain-disease; but supposing there to be no evidence of syphilis, as in the case I relate? What should we conclude? There was no history of a blow—which Virchow believes to be one cause or starting of gliomatous brain-tumour—and no ear disease. There was nothing definite except the fits, which are only localising—do not point to the nature of the disease.[1] I do not think we could conclude with certainty as to the pathology of such a case. I suppose I thought in 1872 and 1875 it most likely that there was not tumour; that there was some cortical lesion, I felt sure. I have had two autopsies on patients who had epileptiform seizures beginning very locally, and yet I found no local central disease; there was universally spread atrophy of both sides of the brain. Such widely-spread disease would not account for localised fits, and besides, atrophied brain-tissue is for function nothing at all, and could not cause anything. No doubt in each of these two cases a minute local lesion existed, which I failed to discover.

In some cases of cortical syphilis I believe the gumma is absorbed or disappears, leaving a sort of scar, as syphilitic liver-disease does. I have published[2] the case of a man who had fits beginning in the right hand; we found adhesions of the dura mater over convolutions of the left cerebral hemisphere, where the grey matter had disappeared, leaving an œdematous tough scar. There was a large ordinary gumma in the opposite cerebral hemisphere. But I do not believe that I overlooked any such changes in the two cases of cerebral atrophy I have just mentioned.

Now that we have had the autopsy, we feel sure that the

[1] The abnormal physiology of such cases is quite certain; there is highly raised instability of some nerve cells. The question in pathology is, "By what process does this great local instability result?" And part of this question is "Is the instability the indirect result of gross organic disease?" If local gross organic disease be decided for, then arises the further question, "Is it syphilitic or not?"

[2] 'Medical Times and Gazette,' Dec. 28, 1872.

patient was always, so long as a large tumour existed, on the brink of death; for at any time a large hæmorrhage might have occurred from the tumour and killed him. It is well known that patients seemingly well, or not very ill, die suddenly in this way by hæmorrhage from cerebral tumour.[1] Hæmorrhage from such tumour is one cause of hemiplegia. It is hard to believe, however, that the small hæmorrhages found post-mortem in the case I have narrated had anything to do with the patient's hemiplegia on his last admission. The nature of this hemiplegia is a very difficult clinical problem. It could not be due to the destruction effected by the tumour. I think it possible that it was a sequence of some seizures in the night, and that the patient would have recovered from it had he not died of pneumonia. Of course I cannot know that he had fits in the night. Before (in 1875) he had had almost perfect paralysis of the leg; he told us (in 1882) that he had been hemiplegic several times. I think this likely, but I must add that the patient was mistaken as to another matter, the date of his last hemiplegia; he was too ill to be thoroughly trusted; his father said his son had never been hemiplegic. I will allude only to the paralysis in 1875. This was clearly a sequence of his seizures. The most reasonable explanation of paralysis after such convulsions seems to me that given by Todd (who first described what he called epileptic hemiplegia, and what I call post-epileptiform hemiplegia) and Alexander Robertson—exhaustion of nerve fibres by the excessive discharge in the paroxysm. I have seen perfect hemiplegia come on after convulsive seizures, and pass off again several times in the same patient; a cortical lesion was found post-mortem in that case.

It is very important to note the condition of the reflexes in cases of paralysis after local convulsive seizures, but I say nothing about them in this case; they were only tested for when the patient was hemiplegic on his last admission.

For many reasons it is important to note the condition of the deep reflexes in cases of local paralysis after epileptiform

[1] Hence a young person with double optic neuritis is to be considered in danger of sudden or rapid death, not on account of the neuritis, of course, but because in young people it often results somehow from vascular glioma.

seizures. The matter is somewhat complicated. I have known foot-clonus of the side affected by fits, when there was no paralysis, and several weeks after the last fit. But we sometimes find temporary foot-clonus and temporarily exaggerated knee-jerk on the side temporarily paralysed after a convulsive seizure affecting that side, in a patient also before his seizure had normal reflexes.

Critical Digests and Notices of Books.

The Brain and its Functions. International Scientific Series, vol. xxxvii. By J. Luys, Physician to the Hospice de la Salpêtrière. With Illustrations. London: Kegan Paul, Trench & Co., 1881.

The Author assures us that this work is an abstract of his personal experiences, and of the ideas which for many years he has been attempting to popularise by his public lectures; but it is not made quite clear whether by experience he means observation, and intends it to apply to that "system which he has found it necessary to adopt, which consists of making regular stratified sections of the cerebral tissue, in the faithful reproduction of these by means of photography, and in the employment of successively graduated powers for the representation of certain details." "I have," he says, "been able by these new methods of investigation to penetrate further into the still unexplored regions of the nervous centres, and like a traveller returned from distant lands, to bring back correct views and faithful representations of certain territories of which our predecessors caught scarcely a glimpse."

These new methods, however, are not very new, and their introduction is not due to the author, nor their use peculiar to him; and we are somewhat surprised to find that the name of Dr. Decke, who has long made a speciality of cerebral photography, is not mentioned. In this respect, however, the American pathologist suffers in goodly company, for the work of other men is so far ignored throughout this volume, that the popular reader might very well arrive at the conclusion that most of the discoveries announced have been the results of the new methods of investigation which the author has found it necessary to adopt. It is true that the author does not

frequently refer in a definite manner to his own discoveries, so that when he does so the exception may be well worth notice. For instance, at p. 18, in describing the cerebral cell, he says: "I have lately discovered, in some researches as yet unpublished, that this substance which we call the protoplasm of the cell is formed by a true tissue organised in a special manner; that this tissue, consisting of very delicate fibrillæ interlaced like the wicker-work of an osier basket, has a tendency to agglomerate towards the nucleus of the cell, which thus becomes a true point of concentration, and that the nucleus itself is not homogeneous; that it is endowed with a special structure, radiated in appearance; and that, lastly, the nucleolus, considered as the final expression of the unity of the nerve cell, is in its turn divisible into secondary filaments."

One cannot but wish that the author had given a woodcut of these fibrillæ interlaced like the wicker-work of an osier basket, and with a tendency to agglomerate towards the nucleus of the cell, of which he has lately discovered the protoplasm of the cerebral cell to be composed. But, alas! a woodcut would but very coarsely reproduce the photographs with which, like a traveller returned from distant lands, he brings back with him correct views of his anatomical discoveries, and to judge by the woodcuts which he has given, it would be wonderfully and fearfully made; for the illustration of the cerebral cell at p. 19 is terrific in its resemblance to that *pieuvre* or octopus which nearly swallowed Victor Hugo's chief *Misérable;* while, to make amends, the cerebral section at p. 15 is exactly like the beautiful foliage of the major convolvulus. If photographs of cerebral sections are of the value which the author supposes, why not give them, or at least some fair attempt at their reproduction by the engraver's art? Such a representation might lift us out of the unsatisfactory mental state in which the author is content to plunge us by the contemplation of the wicker-work fibrillæ of the cell protoplasm, namely that of "imagination confounded when we penetrate so far into the infinitely little."

The author's own imagination would seem to be less confounded when he passes from the experiences of observation into the domain of the ideas which for many years he has

been endeavouring to popularise. In this ideal world he is at home, and from the scientific point of view these popular or popularisable ideas have this value, that many of them are not unlikely to be more or less consistent with the truth of fact, when the latter has been really discovered. The objection to his method of statement, and a most serious objection it is, is that he puts forth his ideas as ascertained facts, which in great measure they are not. For instance, a fundamental idea with him is that the mental functions are the result of vibrations in the sensory nerves, causing vibrations in the cerebral cells. This is, indeed, the hypothesis which David Hartley promulgated in the beginning of the last century, and which, as Lewes remarks in his 'History of Philosophy,' "is historically curious as the first attempt to explain the physiological mechanism of psychological phenomena." But, as Lewes further remarks, "so entirely aloof is the hypothesis of vibrations from any psychological process, as explained by Hartley, that when Priestley abridged the work he omitted the hypothesis altogether, and it was never missed."

It would be impossible to omit this doctrine of vibrations from the work of Dr. Luys, for if that were done, the keystone of the arch would be removed and the structure would collapse. For there is this noteworthy difference between Hartley and our author, that whereas Hartley's dissertation contained the precious psychological principle of the Association of Ideas, the notion of cerebral vibrations being put forward as an hypothesis merely, Dr. Luys describes these mind-producing vibrations under a variety of terms and phrases, and as if their existence were a well-ascertained fact, affording the true explanation of the manner in which cerebral activity becomes mental function.

But before proceeding to the exposition of this science of mind-producing vibrations, we may briefly record what really does seem to be a new idea, namely that "the nervous elements, like bodies which have received the vibrations of light, preserve for a long period traces of the excitations which have in the first place set them in action, thus storing up within themselves phosphorescent traces, which are records of the received impressions" (p. 81). This quality,

which the author designates Organic Phosphorescence, he classifies as one of the three fundamental properties of the nervous elements, the other two being sensibility and automatism.

In another chapter (p. 133), Dr. Luys declares more explicitly what he means by this one of his three fundamental properties of the nervous elements.

"I have proposed to apply the term Phosphorescence to that curious property the nervous elements possess, of remaining for a longer or shorter time in the states of vibration into which they have been thrown by the arrival of external excitations—as we see phosphorescent substances, illuminated by solar rays, continue to shine after the source of light which has illuminated them has disappeared."

In passing, we may remark that true phosphorescence, which is a state of luminous decomposition, is in no way dependent upon illumination by solar rays, and that the curious phenomenon described by the author is quite incorrectly called phosphorescence. It would more accurately be called fluorescence; and while phosphorescence is intelligible to the chemist, the nature of this luminous property of substances which are not in process of decomposition is at present quite unknown. There is, however, this difference between the brain and luminous paint; brain absorbs the vibrations of light and sound, and gives forth that which is neither light nor sound; while fluorspar and other substances absorb light and give it forth. We are unable to perceive even the similitude of mental acts to phosphorescence, and much less to admit their identity; but perchance this inability may be caused by our cerebral cells having become old and lazy, and incapable of erecting themselves. For in elderly persons the author says that "the cells of the *sensorium*, altered in their essential constitution, have become lazy, and incapable of erecting themselves in the presence of recent external impressions; and that this state of torpidity of the elements of the *sensorium* for new excitations, leaves the field free to the older ones, which, not being obscured by more lively impressions, continue to vibrate without opposition, and thus perpetuate the last phosphorescent gleams of a far-off past, which is dying" (p. 164).

The Chapter on Attention commences with a description of the manner in which this mental state is produced.

"We have already several times insisted upon the different phases of evolution of the phenomena of sensibility, and shown that this simple physical impression produced by the external world is transformed, as it becomes incorporated with the organic tissues, into nervous vibrations, and that these nervous vibrations, passing through successive agglomerations of cells, undergo the action of the different media through which they pass, until they arrive, transformed and purified, in the plexuses of the cortical substance, which are set in motion, impressed and vivified by them alone."

What is meant by the purification of a vibration we are left to surmise, but the purified vibrations having arrived, the plexuses of the cortical substance undergo another change. We have already heard of lazy cells; but now the cell becomes *attentive*, and there is a chasm of ignorance between the idea of a vibrating cell and that of an attentive cell which the author in no way attempts to bridge. He says: "It is necessary that the impressed cerebral cells shall, like the cell of the sensorial plexuses, be endowed with a certain special retentive power, and with a certain energy for supporting fatigue; for it is at the expense of its substance that it produces movement, vibrates, enters into erethism, and becomes attentive."

Thus we are getting on very comfortably in our "psycho-intellectual sphere." Memory is organic phosphorescence, mind is vibration of the cerebral cells; vibration, however, which must be purified, concentrated and transformed. Purification, concentration, transformation, read like the processes of chemistry, although it is rather puzzling to conceive how these terms can fitly apply to vibration, which is a physical and not a chemical condition. But then the cells are also said rather promiscuously to become erect, or to become erethised, and these are physical terms.

And even this is not the end of it, for "these same sensorial excitations are *incarnated* in the living cell," and their "sensitive impressions are again *reverberated* in the sensorium," which brings us back to physics.

But, after all this elaboration of the mechanical and

chemical conditions of the genesis of mind, we arrive at last at the vulgar old notion of cerebral secretion, or rather of cerebral excretion.

"We may say then, in a general manner, that some of the peripheral excitations which arrive at the sensorium in the form of a vibratory impression, of a living force in activity, remain there stationary, stored up in one place. They develop then a series of secondary reactions, of energies regularly co-ordinated, which are incessantly distributed in the direction of the apparatuses of organic life, and represent the continuity of the primary movement, and, as it were, the *modes of excretion of the living forces*, implanted in the organism, which here and there effect their physiological discharge" (p. 316).

This excretion of living forces applies, it is true, to what is called the reflection of the motor processes, in which the cells of consciousness may be supposed not to be erethised; but the presence or absence of consciousness would not seem to make much difference to the value of the idea that a mandate to the muscles is *excreted* from the sensorium. The idea is rendered more curious, if not more valuable, by a further comparison.

"Henceforth the mental process has made one more step in the intricacies of the cortical substance. It opens up a new path, that of the motor regions proper. *A living automatic Pianoforte from this moment comes into play*, and in various forms expresses the sensitive keys it is bound to interpret faithfully. It is the instrumental part of our organism that vibrates, and the process tending more and more to emerge from the plexuses of the cortical substance, becomes concentrated within certain circumscribed limits, in certain psycho-motor regions, and hence, in the form of rapid intermittent stimulations, effects its discharge directly upon the different territories of the *corpora striata*" (p. 324).

Does this mean that the sensorium excretes co-ordinated motion as a pianoforte excretes harmonised music? If so, the comparison needs no refutation, and it may well be left as a fitting climax to this remarkable scientific treatise, which does read not unlike the views "of a traveller returned from distant lands;" but as described by the Brothers Verne, by whom so much of the marvellous has been aptly told as it might be, or

rather as perhaps it might seem that it ought to be, but certainly not as it is known to be. It may be that mental action consists in waves of vibration of the neural substance, but hitherto not a particle of evidence is adducible in support of the hypothesis. It may be that some of the conditions attributed by the author to the cerebral cell in a state of activity are real. It may be phosphorescent, erect, or erethised, or it may receive purified or concentrated, or reverberated or incarnated vibrations; but up to the present moment there is not only an absolute failure of all evidence on these points, but it has not yet even been proved (although from many accepted facts the inference is highly probable) that the cerebral cell is the factor of the mental processes. Moreover, if it could be proved that the cerebral cell is the main factor of mind, as we do in truth believe it to be, what a vast difference must we acknowledge between any idea we can form of a vibrating cell and that of a cell which is attentive!

We heartily wish, as some counterpoise to the bewildering treatises of psychological guesswork, for which there would seem to be some popular demand, that some candid and well-instructed psychological biologist would take the trouble to tell us faithfully what is not known on the subject of his research. Meanwhile, to write thus of these purified vibrations of the erect, erethised, and attentive cerebral cells is as scientific as to descant upon the "Aspirations of the Soul," and the judgment which Dr. Luys himself passes upon the hypothesisers of old times applies with equal fitness to himself.

"The controversies of philosophers and metaphysicians which have been taking place for time immemorial have succeeded in arriving at but one thing—the expression in sonorous language of their ignorance, more or less complete, of the fundamental characters of psychical life" (p. 322).

JOHN CHARLES BUCKNILL.

BUZZARD *on Diseases of the Nervous System.* (8vo., pp. 466. London: J. and A. Churchill, 1882.)

THE able work Dr. Buzzard has done in Neurology is so well known that it is not necessary to write an ordinary review of the contents of this volume. He has achieved a position such that a reviewer's praise or blame is to him of little import. Saying, once for all, that we believe the book to be one of the most valuable contributions to the diagnosis and treatment of diseases of the nervous system we know of, we pass on to give some account of it.

There are twenty-five lectures. The first two are devoted to the subject of so-called "tendon-reflex," the study of which Dr. Buzzard approaches almost entirely from the clinical side. In these chapters, which occupy the first forty pages, there is a very full description of the conditions of "tendon-reflex" in various morbid conditions affecting the cerebro-spinal nervous system. There are frequent references to this subject throughout the book. Several sketch-diagrams serve to illustrate the various points referred to.

Lectures 3 and 4 are upon acute anterior polio-myelitis in the infant and adult. A number of cases are brought before the reader, and the clinical and pathological points graphically illustrated. Reference may be made to some original observations by Dr. Buzzard. He tells us that in this disease a certain amount of voluntary power will often return before the nerve-trunk shows excitability to electrical currents, and suggests the following explanation of this seeming anomaly. According to Huguenin, the anterior root of a nerve is composed partly of fibres which are practically prolongations from the large ganglionic cells in the anterior horns, and partly of fibres which are not so formed, but pass down in the lateral column without direct connection with the ganglionic cells. The trophic centre for the former is the ganglionic cell, which is destroyed, or more or less seriously damaged, in acute anterior polio-myelitis, with consequent loss of voluntary power and electric excitability. But the trophic centre for the latter would be somewhere higher up, and these nerve-fibres

may readily therefore, Dr. Buzzard thinks, be supposed to escape all but a temporary obstruction of function.

Dr. Buzzard thinks it is not uncommon for the hemiplegic form of infantile paralysis to be confounded with hemiplegia of cerebral origin. The tendon-reflex will, he says, easily differentiate the two conditions In the former, this is lost; in the latter, if there be any change at all, it is in excess. Another point of interest to which Dr. Buzzard draws attention is the fact, that if we examine cases of old infantile paralysis, when the patient only complains of a defect in one limb, we shall very often indeed find that the exemption of other limbs is only apparent and relative. He remarks that it was "from applying the test of the patellar tendon-reflex that I have been led to discover how very generally in a patient affected with old infantile paralysis those limbs which were supposed to be sound are not by any means normal. "I feel sure," he adds, "that monoplegia in this disease is far less common than it is usually thought to be, and that limbs are very frequently supposed to be sound which are in reality only comparatively so. The suggestion is made that many cases of sudden or very rapid death which occur in children, and the cause of which is often left to conjecture, may really depend upon this disease (acute anterior polio-myelitis) attacking the medulla oblongata with the same kind of suddenness with which it ordinarily attacks the anterior grey matter of the spinal cord." In a foot-note, Dr. Buzzard mentions that Dr. Hale White was led by these observations to examine microscopically the medulla oblongata of a child who died suddenly in the Evelina Hospital, and found extreme vascular dilatation over a considerable extent of the bulb, with several hæmorrhages, one of which occurred at the margin of the nucleus of the vagus.

The 5th lecture, "on the differential diagnosis between certain hysterical conditions and myelitis" abounds with information of an important kind in reference to a subject of peculiar interest at the present time. In this lecture Dr. Buzzard describes several cases in which the nature of the ailment had been mistaken, and considers in a very able manner the points available for diagnostic purposes and the pitfalls

for error which abound. In this lecture also, as well as in those upon infantile paralysis, the subject of electro-diagnosis is carefully gone into. We would draw especial attention to one remark which cannot be too forcibly endorsed. "The accurate testing of electrical reaction is a more difficult matter than is commonly supposed. It is not safe to attach importance to alleged alterations in reaction, unless these are reported by one who is known to be a competent observer."

Lectures VI., VII. and VIII. are upon "Tabes Dorsalis." In Lecture VI., after a sketch of the anatomy of the spinal cord and the pathological changes observed in sclerosis of the posterior columns, various symptoms of the disease are discussed, especial attention being given to two, on the sensory and motor side respectively, viz. pains, and Westphal's symptom (the absence of the patellar tendon reflex), along with a fairly normal condition of the quadriceps extensor muscle. Lecture VII. deals with the Argyll-Robertson pupil, in which Dr. Buzzard's method of testing with the ophthalmoscope is described and the generalisation first made by Dr. Buzzard and subsequently by Professor Erb is suggested, viz. the movement of the iris during accommodation—the equivalent of a voluntary movement—is preserved; whilst the reflex movement, which should succeed the stimulus of incident light upon the retina, is abolished or impaired. The cephalic symptoms of tabes next claim attention; the pains in the region of the fifth nerve and occipital nerves, and the anæsthesia apt to occur in the region of the trigeminus, are especially considered. Pierret has suggested that the lesion in the district of the latter nerve is to be explained by extension upwards of the sclerosis affecting the posterior root-zones of the cord, so as to involve the descending root of the fifth; this explanation is adopted by Dr. Buzzard, the relations of the parts in question being explained by a diagram. In reference to the pains in the head in tabes, Dr. Buzzard suggests some observations in reference to their diagnosis from ordinary neuralgia. He writes, "Flying, so-called neuralgia pains in the head, when they attack both sides and do not map out the district of one or other division of the fifth nerve, should lead to attentive examination for symptoms of tabes." Cases bearing upon the distinction

between amaurosis from cerebral tumour and tabes with optic atrophy, conclude this chapter. In Lecture VIII. lesions of the fifth nerve in tabes are further discussed by the help of cases, one of which bore a superficial resemblance to diphtheritic paralysis. Dr. Buzzard suggests that in this instance the condition of the soft palate is probably not one of paralysis. It is symmetrical in the two sides, as the Argyll-Robertson pupil. The difficulty lies, Dr. Buzzard thinks, on the *sensory* side. Touches are not felt, and do not produce reflex contraction. Besides the dysphagia, the patient's voice was nasal in character. Dr. Buzzard remarks: "This peculiarity is known to depend upon the circumstance that the soft palate is not drawn up and applied, as it should be, to shut off the nasal cavities. It would appear, then, that this action, which takes place in health without our consciousness, is a reflex process, the afferent part of which depends upon branches of the fifth nerve." A patient whose case is considered, had facial paralysis succeeding very severe electric shock-like pains in the same side of the head. The paralysis had subsided in a month. The author refers the paralysis in the district of the portio dura to the exhaustive discharges in what may be looked upon as the sensory root of the portio dura—the fifth nerve. He refers to a case, lately in hospital, of a female subject for years to paroxysms of facial tic, recurring every fortnight, and concentrated in the ophthalmic division of the right fifth nerve. Within the last year or so each attack has been followed by partial paralysis of the oculo-motor, the right eyeball being turned outwards and the eyelid dropped. "Pierret reminds us," Dr. Buzzard says, "that the fifth nerve (its sensory portion) comports itself like a spinal nerve, and that not only its motor portion, but in effect *all* the intracranial motor nerves—the oculo-motor, fourth, sixth facial and hypoglossal, must be considered as practically constituting the anterior roots of the nerve.

The subject of ataxy is considered at great length. Trophic lesions of the skin, herpetic, bullous, and ecchymotic, are illustrated by cases of interest. Lecture IX., "Ophthalmoplegia externa with tabes dorsalis, gastric crises," appeared so recently in the columns of this Journal, that we need do little more than

mention it, except to say that the view originally suggested by Dr. Buzzard, at the Pathological Society in February 1880, as to the gastric crises being occasioned by sclerosis invading the neighbourhood of the roots of the vagus, is countenanced by the observations of Pierret, and more recently by those of Emile Demange.

Dr. Buzzard has gone through some change of opinion in reference to the supposed association of tabes dorsalis with syphilis, the subject of Lecture X. In 1871, writing upon the subject of syphilitic affections of the nervous system,[1] he included progressive locomotor ataxy amongst the nervous affections belonging to the tertiary stage of syphilis. But the remarkable absence of success in treatment upon this basis induced him, he tells, to exclude the disease from consideration in his work on syphilitic nervous affections, published in 1873–74. He writes: "The connection between nervous disorders and syphilis was then not generally recognised, and I was anxious to avoid weakening the force of that which was to be said on a very important subject by the introduction of debatable material." Subsequent consideration leads him to the conclusion that though there is a remarkable frequency of association between syphilis and tabes dorsalis, the time has not arrived as yet for us to draw safe inferences as to the precise nature of the relation. He is not disposed to draw a hard-and-fast line between cases so called primary and secondary tabes dorsalis, and thinks that a case of the disease which is typical in its features as it comes before us may prove on investigation to have arrived at such a condition through a stage of subacute meningo-myelitis with paraplegia. He is not able to subscribe to the view prominently supported by Vulpian and the French school, that in sclerosis it is the nervous element proper contained in the nerve-tubes which is first affected by an irritative lesion, the connective tissue suffering secondarily. "One cannot conceive," he writes, "an atrophy of axis-cylinders without some antecedent changes in the carriers of trophic material for the axis-cylinders, *i.e.* in the blood-vessels and the tissue in which they lie." "On the other hand," he says, "if a vascular

[1] 'Lancet,' March 11, 1871.

change be the initiatory stage, it does not seem difficult to understand that syphilis, which is prone to occasion meningitis, may sometimes lead to sclerosis through inflammation of the soft membranes of the spinal cord."

A considerable part of the book is devoted to the important subject of the osseous and articular lesions in tabes dorsalis, of which the author has had large personal experience. He considers that the frequency of the association of gastric crises with these affections justifies him in suggesting the hypothesis that there is in the medulla oblongata, in the neighbourhood of the nucleus of the vagus, something of the nature of a centre concerned in the nutrition of the bony skeleton. The circumstance that we are able to exclude various parts of the spinal cord as seats of the lesion, he considers, lends support to the view. The fifteenth lecture deals with "Certain little recognised phases of tabes." Pointing out that the prominence given to the symptom ataxy by both Romberg and Duchenne is not sustained by experience, and that ataxy may be absent just as optic atrophy, diplopia, or anæsthesia, Dr. Buzzard thinks that in many cases a symptom which, as a general rule, is slightly expressed, may be so dominant as to overpower and dwarf all the others, so as to lead to erroneous diagnosis. This subject is illustrated by many interesting cases. He believes that stone (of a local, not of constitutional origin) may be due to tabes, and may by the severity of its symptoms conceal those which would indicate the original source of the disease. He says that imperfect expulsion of urine in tabes may be a very easily and strongly pronounced symptom, and that in the mucus of the bladder, accumulated by the cystitis which results from retained urine, a stone may be formed.

Lecture XVI. deals with cases of prolonged somnolence in cerebral syphilis, a condition which the author attributes to narrowing of cerebral arteries by syphilitic endarteritis. Neuritis, rheumatic and syphilitic, are the subjects of Lecture XVII., which contains many points of importance in reference to diagnosis and treatment. Lecture XVIII. is upon cases of "rapid and almost universal paralysis," of which the author has seen several examples. They belong, he thinks it probable, to

the category of Landry's acute ascending paralysis, though divergent in some details. Syphilis is thought by Dr. Buzzard to be the cause of some of these. Incidentally the subject of diphtheritic paralysis receives attention in this chapter. The lecture on "Paralysis Agitans" (Lecture XIX.) was recently published in 'Brain.'

Lecture XX. deals with spastic paraplegia, secondary to a transversely localised myelitis, as distinguished from that form described by Seguin, Erb, and Charcot, in which the disease is supposed to be protopathic. Some interesting results of treatment are stated in this lecture. Lecture XXI. is upon "Cervical paraplegia," and is an extension of a paper which our readers may remember to have perused in this Journal. "Syphilitic paraplegia" is considered in Lecture XXII. Some examples of Tetany form the subject of Lecture XXIII. Lecture XXIV. is devoted to the "Phenomena of Transfer produced in Epileptic patients by the application of encircling blisters." It is fourteen years since Dr. Buzzard published in the 'Practitioner' a paper showing some very remarkable results which he had found occurring after the application of encircling blisters to the limb which was the seat of a marked epileptic aura. The substance of this paper is here reproduced, and further cases are given.

The author, relying on the evidence that strong peripheral irritation, artificially employed, brings about some kind of change in a nervous centre, has been trying to turn this to account in the treatment of aphasia. Lecture XXV. is concerned with the important subject of diagnosis of lead palsy, in which some interesting illustrations are introduced. Here again there are important remarks on the subject of Electro-diagnosis.

J. Hughlings-Jackson.

Clinical Cases.

CASE OF ALLOCHIRIA.

BY DAVID FERRIER, M.D., F.R.S.

Physician to King's College Hospital, and the National Hospital for the Paralysed and Epileptic.

In Part XIV. of this Journal (July, 1881), Obersteiner published a short memoir on a form of perversion of sensibility to which he gives the name Allochiria, characterised by the erroneous reference of sensory impressions to the corresponding part of the other side of the body.

The following case, which I have recently had under my care, is rather a remarkable example of this condition, and appears to me worthy of record, though up to the present it does not throw much light on the pathology of the affection. The case was so anomalous, that I for some time doubted the veracity of the patient's statements, and endeavoured in every way I could think to involve him in contradictions; but the phenomena were so uniform and precise, that I was obliged to admit the facts, however unable to explain them.

The history is one of severe cranial injury, causing prolonged unconsciousness, followed by motor disorder of a combined ataxic and hemiplegic character, from which the patient had only partially recovered at the time of his discharge, together with the temporary remarkable perversion of sensory localisation. There was no anæsthesia, the slightest touch being readily perceived, but unhesitatingly referred to the corresponding point on the other side. This confirms Obersteiner's view that allochiria must be regarded as a symptom not necessarily associated with anæsthesia.

But a still more remarkable fact than the mere transposition of the sensation was the transposition of the reflex reactions, as detailed below, in the notes of the case which was taken for me by Mr. Rabbeth, HousePhysician of King's College Hospital.

The pathology of the case is one on which at best only speculations might be offered, and these I forbear.

James S., aged 29, came to my out-Patient Clinic at King's College Hospital, and was admitted under my care on December 15, 1881.

History.—He had enjoyed good health, and had lived temperately, and never had venereal affection of any kind. Last September, while engaged in his work as carpenter, a scaffold-pole fell on his head from some considerable height. He was knocked down and rendered unconscious. He remained in a state of insensibility from 6 o'clock in the evening till 10 next morning. He was confined to bed some days, and had some difficulty during this time in micturition and defecation. When he got up he felt rather unsteady on his legs, but not to the same extent as at present.

For the last month he has been complaining of headache, and difficulty in standing and walking.

State on Admission.—Patient is a healthy-looking, well-nourished man. He has a peculiar expression of face, the eyes having a fixed stare and looking in the distance. The power of convergence seems entirely lost. He cannot maintain his equilibrium with his feet together, and when he walks he staggers from side to side, his legs tending to cross each other. He says the floor seems to rise up towards him when he walks. He can move his legs freely when he is seated on a chair, but the left is weaker than the right. There is no unsteadiness in the movements of the arms and hands, but the left hand is distinctly weaker than the right. Measured by the dynamometer, the grasp of the right = 110 lbs., of the left = 25 lbs. only.

The facial movements are equal on both sides, but the tongue tends slightly to the right.

Sensation.—Tactile sensibility is throughout normal as regards delicacy and quickness of response, but there is a most remarkable condition of allochiria, or localisation of impressions on the wrong side, as well as a similar reversal of reflex response to tactile stimuli, affecting the legs from the groins downwards, and also the tongue, inside of the mouth, and the nostrils.

The unhesitating precision with which the patient referred the impression to the corresponding point on the side not touched, led me at first to suspect deception; but the absolute uniformity of the phenomena under every precaution against the patient knowing what was being done to test the condition, together with the reversal also of the reflex reactions, compelled me to abandon this hypothesis.

A touch on the right big toe was at once referred to the same spot on the left, and *vice versâ;* a similar condition existing on every point of both legs up to the groin. Above

this, impressions were exactly localised on their proper side. Sensation was also correctly referred on the conjunctivæ, and on both sides of the face and head generally.

But a feather or spill of paper thrust into the one nostril immediately caused retraction of the head and lachrymation, while the patient vigorously rubbed the other nostril where he felt the irritation. It need scarcely be remarked that the patient was always securely blindfolded during these tests.

So on the tongue; a touch or prick on the one side of the tongue was at once immediately referred to the corresponding point on the other. The same condition existed also as regards gustatory sensibility, salt on the one side being distinctly tasted on the other, as indicated by the position the patientpointed to. So far as could be judged, the patient's sense of smell not being very acute, there was no reversal as regards smell proper, but only as regards the common sensibility ofthe nostril.

On the mucous membrane of the mouth there was also a reversal of tactile sensibility.

But, as has been stated, the reversal was not confined to perception of impressions, but extended also to reflex reaction. Tickling the sole of the one foot caused retraction of the other, while the foot actually tickled remained perfectly still. So also tickling of the inside of the one thigh caused flexion of the other. Conjoint tickling of the sole of the one foot and the inside of the other thigh caused crossed reflex reactions of the foot and thigh.

Sight was normal. The pupils reacted normally to light. The ophthalmoscopic appearances were normal. There was scarcely any power of convergence of the eyes to near objects, but all the other ocular movements were carried out freely.

Hearing (tested by Dr. U. Pritchard) was defective in the right ear.

For ticking of watch—Left ear = 50 per cent. of normal.
„ „ Right ear = 16 „ „
For tuning-fork on nose—Left ear—5″
„ „ Right ear, not heard at all.

The patellar reactions were exaggerated, the left being greater than the right.

The cremasteric reflexes were obtained on both sides equally, but the stimulus causing retraction of the testicle also caused movement of the opposite leg, sometimes of both. The abdominal and epigastric reflexes were also obtained on both sides equally.

The organic functions were normal. The patient complained greatly of headache, referred to the forehead and occiput. Percussion of the occipital region, specially of the right, always elicited or intensified the pain.

Progress of the Case.—The patient continued to complain of headache and pain along the spine, the headache being always worse at night. On January 2 his gait was observed to be more impaired than before, and he could not stand without support. There was especial difficulty in planting the left foot, which crossed the middle line when advanced, and the leg was thrown into rapid tremor when the weight of the body was rested on it.

At this time sensation was correctly referred in the thighs, but just above the patella stimulation of the one leg was felt in both legs simultaneously, and caused reflex movements of both. The condition of allochiria existed below the patella, and in the nostrils and tongue, as before.

On January 9 the allochiria had disappeared in the legs in all parts above the ankles. Here stimulation of the one caused sensation in both at corresponding points.

On January 16 the patient vomited or coughed up about an ounce of blood, and this was repeated on several occasions to a less extent for two or three days subsequently.

Walking was somewhat improved, the legs did not cross so much as formerly.

Sensation is now correctly referred in the foot touched, but is felt also to some extent at the corresponding point in the other as well. Plantar stimulation excites reflex action mostly in the same leg, but sometimes in the other, and occasionally in both.

Sensation is still crossed in the nostrils and tongue.

On Feb. 6, sensation was correctly referred everywhere, both on skin and mucous membranes. The reflexes are confined to the side stimulated. The reaction seems to be considerably slower than in normal conditions.

The patient cannot stand or walk without support. When he is on his feet, objects seem to be moving laterally, and he is giddy, especially when he looks towards the right. The left arm and leg are still feebler than the right.

During the next month there was little or no alteration. The pain in the head was less, but occasionally so severe at night as to require the administration of morphia subcutaneously.

On March 22 the patient was much better. The pain in the head was much diminished, and he could walk alone for a little way. The legs are not crossed, but there is great hesitation in planting the left leg, which trembles greatly when extended, apparently from clonus of the quadriceps.

With the aid of a stick he can, however, get about the ward. From this time onward to June 31, when he was discharged, the patient gradually improved. The left leg, which was

especially feeble, improved under the local application of the faradic current, but was still weak at the date of discharge, and the quadriceps tremor still existed more or less.

Sensation and reflex action were everywhere normal.

Deafness, however, still continued in the right ear. The face had lost its peculiar staring expression, and the power of convergence of the eyes had been regained.

I saw the patient again on July 24. He was still feeble in the left leg, and his gait was somewhat tottering. No new symptoms had appeared.

CASE OF HYSTERO-EPILEPSY.

BY W. H. DOBIE, M.B.

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THE following case was under the care of Professor Grainger Stewart, to whom I am indebted for permission to publish it:—

T. M., aged 15, a general servant girl, was admitted to the Royal Infirmary, Edinburgh, on June 29th, 1881, suffering from fits of a hysterical character. Her family history was unimportant, save that a cousin was said to have been subject to epileptic fits in childhood.

The patient, an illegitimate child, was brought up amid poor and not very favourable surroundings. When ten years old, she used often to complain of pain and tenderness in the crown of the head, the least touch causing her to cry out. This subsided gradually under simple medical treatment. Shortly afterwards she became subject to fits, which, from the description given, were apparently epileptic of the petit mal character, recurring at intervals of a week, afterwards less frequently, and liable to be brought on by any unusual excitement. She had no such fit for eighteen months previous to her admission.

In disposition she was cheerful and intelligent; it was remarked, however, that at an early age she showed a propensity for telling lies, not as a rule for her own advantage or any adequate reason—a propensity which has increased with her years. Latterly also she had become possessed by certain delusions which it was almost impossible to dispel, for instance, that her grandmother was dead.

In April, ten weeks before admission, she became general servant in a lodging-house amid unfavourable surroundings. She was much over-worked, and this, together with undue sexual excitement during the same period, would seem to be the immediate cause of her present illness.

On the morning of June 29th the patient left her mistress's house on an errand, apparently in her usual health. She did not return, and afterwards had no recollection of how she

spent the day; but about eight o'clock in the evening she was found by a policeman at the corner of a street where she had fallen. She appeared to have had a fit, but told her name, and complained loudly of pain in the back. She was accordingly conveyed to the Royal Infirmary as a supposed surgical case. After admission she went through a series of hysterical attacks, such as she has since suffered from at varying intervals.

A typical seizure may be described as follows:—

The patient was perhaps lying comfortably in bed, talking rationally with those about her, when suddenly she complained of a violent pain in the back or side, and putting her hand to the spot, was immediately thrown into a state of tonic spasm, in which the features were fixed, the back arched, the head thrown back, the limbs rigid, the fingers clenched over the thumbs, the eyes staring, and the eye-balls turned up.

From this, which lasted a few seconds, she would pass into a stage of violent contortion, throwing herself from side to side, so that she was with difficulty held down; at one moment arching her back as in tetanus, at the next springing forwards, as if to throw herself out of bed.

The limbs were usually flung about irregularly in all directions; but sometimes the arms were repeatedly circumducted, or if possible thrown against some hard object, from which bruises now and then resulted. This violent stage generally lasted from half a minute to a minute, and was succeeded by one of comparative quiet, but was liable to recur at any time during the progress of the fit, being at once set up if any of the sensitive spots, to be afterwards mentioned, were pressed upon. The fit could be checked to some extent by firm pressure over the region of the ovaries, or to a less degree by pressure above the mammæ, but not completely arrested by either.

During the next period she would lie quiet for the most part, no restraint being necessary. Her intellectual faculties became active, although she was unconscious of what was going on around her, or if imperfectly conscious, only associated it with her delusions. Her pulse and respirations were slightly quickened, and there was occasional jerking of the muscles of the face. She was seized with vivid hallucinations, imagining she saw persons and things not really present, and mistaking the identity of those around her, if she could be induced to notice them at all. Usually she made no response to questions, but sometimes her hallucinations were influenced by what was said to her.

To give an instance or two:—She, perhaps, fancies herself back in her place as general servant; she is looking after the children of her late mistress, and calling them by their names;

if now she be suddenly and loudly told it is time to get up she will start up in bed with a confused look, as if afraid she has overslept herself.

Now, perhaps, she sees "Maggie," an old friend, is transported with delight, calls her by name, beckons or whistles to her. The next minute she is crouching beneath the bedclothes, with an expression and attitude of terror at a supposed new presence; and then, as she throws her head back, her features become frightfully distorted, the left side of the face and mouth drawn down, the eye-balls rolled upwards and to one side, the brows knit, while the breathing becomes stertorous, and there is usually more or less recurrence of the stage of contortion.

During the latter part of the emotional stage there were often prolonged fits of yawning, symptoms of choking, stoppage of respiration for long periods, alternating with rapid and forced breathing, or protrusion of the tongue, which, however, was not bitten.

She usually regained consciousness rather suddenly, recognising those around her, complaining of dizziness, sometimes of headache, and either dropping off to sleep, or gradually engaging in conversation. She would become at once perfectly rational, and had then no recollection of her hallucinations, or knowledge that anything out of the way had occurred. Shortly after each seizure she would drink a large quantity of water.

This being the course of the fit itself, the next point of interest is the condition of the nervous system, especially as regards sensory functions.

1st. In the interval between the fits.

2nd. During the fit itself.

1. In the interval between the seizures the patient often complained of pain in the back and right side, of coldness of the feet, dizziness, and headache.

Her sensibility to touch and pain was acute in all parts of the body, her perception of hot and cold substances good, and her localisation accurate. There were, however, certain areas, varying at different periods, which were over-sensitive. At the time of her admission to hospital this hyperæsthesia was best marked over the spinous processes of the lumbar vertebræ and part of the sacrum, together with an indefinite area to the right of this, notably over bony prominences, the iliac crest and the lowest ribs. In this area hot substances were recognised only as painful, while the slightest pressure, heat, or electrical stimulation, caused her to cry out, and sometimes threw her into a fit. In addition to this, slight ovarian pressure caused pain, and might produce a fit.

2. When these points were examined into during the progress of a fit, however, the condition was found to be very different.

Complete anæsthesia was found to exist on both sides of the body; needles might be thrust into the skin without her exhibiting the least expression of sensibility; pricking the forehead, however, caused blinking of the eyelids. The previously hyperæsthetic areas were now for the most part anæsthetic; but firm pressure there would often reproduce the violent stage. The part of the body in which sensation was first regained appeared to be the soles of the feet, the repeated pricking of which sometimes served to dispel the fit; but her first idea of the sensation thus produced was pain referred to the back, or at least to some part of the body not actually pricked. Her power of localisation then was defective during the period of returning consciousness. This was first observed two days after her admission, when a painful abscess in the pulp of the thumb was incised during the quiet stage of the fit, the pain of which caused wincing, but was referred to her back, causing her to sit up and look for a stone in the bed. (She had afterwards no recollection of the abscess being opened.)

The same fact was brought out in a curious manner by pricking repeatedly some portion of the upper extremity in the last stage of the fit. As consciousness was returning, she complained of the prick of a pin, but indicated the corresponding point on the opposite limb; as consciousness further returns she suddenly indicates the point correctly. [Since these observations were made, it has been pointed out to me by Dr. Hyslop, Royal Edinburgh Asylum, under whose care this patient afterwards came, and who observed the fact independently, that this is an example of the condition described in 'Brain,' July, 1881, as "Allochiria," by Professor Obersteiner, of Vienna.]

With regard to our patient's special senses, nothing was found abnormal in the intervals between the fits, except a considerable degree of myopia. There was no colour-blindness. Ophthalmoscopic examination showed staphyloma posticum, and some atrophy of the choroid. During a fit she had hallucinations of sight as above mentioned, and her sense of hearing was abnormally acute; at one time, about a week after her admission, she started at the slightest noise, as a footfall in the passage, even when she was quite lucid, and during a fit she would mistake the creaking of the door for a noise under her bed.

The condition of the *motor* functions during a fit has been described; at the time of her admission she could support

herself fairly well on the left leg, but not on the right. On attempting to walk she sank down on her knees, and was often thrown into a fit. This continued for about three weeks, when she gradually regained the power of locomotion, and afterwards showed no sign of muscular enfeeblement.

Tendon reflex was well marked, but not excessive, and the organic reflex functions, with the exception of occasional retention of urine, were normal.

The patient's growth and sexual development were apparently unaffected by her complaint. She continued strong and stout, slept well, and ate heartily as a rule.

Menstruation began six months before admission, and was irregular, the periods frequent, and the flow scanty.

The circulatory and respiratory systems were normal.

The urine was pale straw colour, acid in reaction, of specific gravity 1014. The quantity per diem 50 to 60 ounces.

The prodromata of an attack consisted in disinclination to work, drowsiness, dizziness, headache, and often pain in the back. The fits, when she was in hospital, usually occurred at night and in the early morning, and she often had one immediately after a period of sleep. When this was the case, she would groan, grind her teeth, and become restless, then waking up, she would stare vacantly around her, and immediately pass into the stage of tonic contraction. Occasionally during the daytime she could tell that an attack was coming on, and tried to prevent it by an effort of the will, but without success. As a rule she had no time for this; but she never fell as in a true epileptic fit.

The attacks were more numerous during her menstrual periods, at which time she became especially drowsy and listless.

The duration of an individual fit varied from ten minutes to two hours, and the number per diem from one to six. The series usually continued three or four days, and she was seldom free from them for an entire week.

The patient's peculiar temperament was further shown by a craving for men's society, by lying propensities before referred to, and by other attempts at deception, of which the secret mixing of milk with her urine may be mentioned as an example; also by the occurrence of periods of melancholy and religious fervour, and by sudden transition from mental depression to exaltation.

Effect of drugs.—1. Chloroform.—On July 9th the patient was put fully under its influence during an attack. After its administration was stopped she attempted to vomit, but appeared to be still in the fit. She presently became conscious, and complained of violent headache.

A smaller quantity of the drug, sufficient to quiet the

spasms, threw her into a state of mental exaltation, which would continue for an hour or two until she fell asleep.

2. Nitrite of amyl.—On one occasion this drug was inhaled during a fit; it caused blushing, choking, and apparently some interference with respiration, but did not arrest the fit.

The application of the faradic current gave uncertain results.

Treatment.—Besides freedom from excitement and wholesome discipline, which were secured as far as possible, the following treatment was adopted:—Bromide of potassium gr. xx., with tincture of digitalis ♏ vijss three times a day, together with counter-irritation by repeated fly-blisters, applied over the back and ovarian regions, and cold shower baths. Valerianate of zinc was also tried, but no marked benefit seemed to result from medicinal treatment of any kind.

Progress.—Improvement to some extent took place after she had been a month in hospital; but at the end of three months there was very little change in her condition. When she was sent into the country she again improved for a time, but soon afterwards the fits became as frequent as ever.

Remarks.—Any comment on the above case seems unnecessary. I have ventured to record it as an interesting example, occurring in this country, of a disease fully described by Charcot, Richer, and others.

CASE OF SPASMODIC RHYTHMICAL CONTRACTIONS OF DIAPHRAGM, RECTI AND OTHER MUSCLES.—RECOVERY.

BY R. SHINGLETON SMITH, M.D., M.R.C.P.,

Physician to the Bristol Royal Infirmary and Lecturer on Physiology in the Bristol Medical School.

In 'Brain,' January, 1882, was published by Dr. E. Buchanan Baxter a case of paroxysmal clonic spasm of the left rectus abdominis, the reading of which recalls to my mind the case of which I now publish the details. My case differs notably from Dr. Baxter's in the absence of all evidences of localised nerve lesion; it differs also in the permanent recovery of the patient. It is doubtful whether any one drug may be credited with the ultimate result: the improvement commenced whilst the valerianate of zinc was being administered, but was much more marked after the iodide of potassium had been given.

Laura F., æt. 20, single, servant, of good general health, but with a tendency to faint, and occasional globus, was admitted to the Bristol Royal Infirmary on September 10th, 1878, for peculiar choreic jactitations, which came on in the night of the 9th, kept her awake all the night, and persisted in the morning.

A similar attack, six months previously, subsided after two nights and one day.

Patient was well nourished, but anæmic, sedate-looking and not easily excited. Temperature 97⅗. Pulse 80, of good quality. Heart sounds healthy. Tongue protruded well and remained steady. Bowels and catamenia regular. The body was being constantly jerked by spasmodic contractions of the recti abdominis and diaphragm, the contractions being regular and almost synchronous with the pulse at about 70 per minute.

The sides of the thorax were tucked in deeply with each contraction, and the recti stood out prominently, showing their tendinous bands; the head moved forwards in a kind of nodding movement, and each contraction was accompanied

by a little grunting sound proceeding from the larynx. The movements ceased entirely during sleep, but recommenced immediately on waking. They could also be arrested by making the patient draw a succession of deep inspirations, but mental pre-occupation produced no effect. The movement was equal on both sides, and there was no evidence of any localised nerve-lesion. Hypodermic injection of curara, gr. $\frac{1}{20}$, produced no manifest effect, and on September 17th patient was ordered one-ounce doses of succus conii, to be taken every four hours. On the following day the dose was increased to two ounces, and this dose was continued till the 21st, when no improvement had manifested itself, and there were no physiological symptoms of conium.

A fresh specimen of conium juice was then obtained, and one ounce was given in the morning; this gave rise to great muscular lassitude and a feeling of exhaustion which continued nearly the whole day, but the spasmodic movements continued as before, not in the least degree modified by the conium, although the patient was so prostrate as to be unable to get up, and scarcely able to raise her arms from the bed.

Bromide of potassium was then given, and with an equally unsatisfactory result.

On September 28th the contractions were more vigorous than before, and had extended to the sterno-mastoid muscles; the rate also had increased to 140 per minute. Deep inspiration caused temporary cessation, and the movement varied in frequency and intensity from time to time. It was remarked by the nurse that it was always worse whenever the patient was watched, but that it ceased entirely only during sleep. Chloral hydrate was then given, ten grains every four hours, and the constant current was passed through the recti muscles night and morning.

October 3rd.—Spasm of sterno-mastoids had ceased: there was marked hypnotic effect from the chloral, and the tongue was dry, but the spasm recurred whenever the patient was roused from her stupor, and continued actively even after the patient seemed prostrated. The chloral was discontinued and liq. arsenicalis was given, five minims *ter in die*.

October 8th.—The spasms, continuous up to this time for twenty-eight days, ceased suddenly after tea without manifest cause, but four days later recommenced in the recti, and afterwards became more general, the recti, sterno-mastoids, scaleni, pectoral muscles, and the diaphragm were all affected, as also the biceps and triceps brachii. Bromide of zinc was then given, one grain, *ter in die*; afterwards bromide of potassium, increased up to sixty grains four times daily. On November 14th patient was unable to stand, and was very

drowsy though easily roused; when awake the movements continued with unabated vigour. The spasm of diaphragm was more marked than that of the recti, the "grunt" was very decided, and there was occasional hiccough. Was then ordered liq. atropiæ, m. ij. hypodermically night and morning.

November 19th.—Spasms, and the patient, were getting worse, the latter weak and emaciating. Pulse 84, small. Chapman's ice-bag was then applied to the spine, and chloral was re-ordered in combination with cinchona.

December 5th.—The ice-bags had not modified the spasms; the general health had not improved. Patient was drowsy from chloral, and had no appetite. Valerianate of zinc, gr. ij., was given thrice daily, and chloral hydrate, gr. xxx., at night.

December 15th.—There was distinct improvement. Patient could sleep without chloral; appetite and nutrition had improved. The spasms were not so severe or so frequent; there was less jerking of the head, and speech was better.

January 6th, 1879.—Improvement had continued. The jerking had now ceased, and patient had improved in nutrition and colour.

January 22nd.—Since the 17th of December patient had taken iodide of potassium, three grains three times daily, together with sp. ammon. aromat. and infusion of calumba. The improvement had commenced before the iodide was given, and the dose of the salt was not increased beyond three grains; nevertheless the patient continued steadily to improve, and on January 22nd she was practically well, and was sent home. She continued to attend as an out-patient for several weeks, and manifested no return of the old symptoms.

July 31st, 1879.—Patient again appeared in consequence of symptoms of dyspepsia. She stated that she still felt a little jerking at epigastrium every night after going to bed.

May, 1882.—The patient has been seen recently: her general health continues good, there has been no return of the old symptoms, nor have any new nerve symptoms manifested themselves.

CASE OF GLOSSO-LABIAL PARALYSIS, WITH PROGRESSIVE MUSCULAR ATROPHY AND LATERAL SCLEROSIS.

BY CHARLES E. BEEVOR, M.D. (LOND.), M.R.C.P.,

Late Resident Medical Officer to the National Hospital for the Paralysed and Epileptic, London.

THE following case was admitted into the National Hospital for the Paralysed and Epileptic, Queen Square, on September 18, 1879, under the care of Dr. Ramskill, to whom I am indebted for permission to publish the case.

The early part of the notes were taken by my friend and predecessor, Mr. A. E. Broster.

R. R., a waiter, aged 36, married, had no children, wife had one miscarriage. Patient had lived well and drunk freely, and had smoked much. Had scarlet fever when aged 10, but no rheumatic fever, and no syphilis.

Hereditary History.—A first cousin was insane; father's sister paralysed, and also her daughter; his father and two paternal uncles died of phthisis.

Present illness began in March 1879. Six months before admission he had had much worry, and three months previous to onset of illness he had walked through some water in the winter time, after which he never seemed free from rheumatism. He first noticed in March 1879 that his hands began to feel weak, a month later his speech began to be affected, and about the same time he had some difficulty in swallowing.

Present state in September 1879, as taken by Mr. Broster, was as follows:—He is very low-spirited, and complains of pain in the back of his head; he has the characteristic sad inanimate expression. *Face.*—In closing his eyes, the left side of face moves more than the right; has some difficulty on both sides in showing the upper teeth; there is much tremor about his chin and upper lip. Masseters act well. Complains of food collecting between his teeth and gums. Movements of eyes and eyelids normal, pupils equal.

Tongue protruded about ¾ inch beyond margin of teeth, and

deviates to the left, very tremulous, and much wasting on both sides.

Speech is an indistinct monotone, no inflexion, and not affected by closing anterior nares.

Swallowing is difficult, fluids sometimes regurgitate through nares. *Soft palate* seems wasted, uvula points to left.

Neck.—Head tends to fall forwards, and he moves it back stiffly; he can, however, turn it horizontally very well.

Trapezii weak, and left worse than right.

Supra- and *infra-spinati* wasted. *Serrati* and muscles of back not affected.

Upper limbs both very much wasted, and the muscles are very flaccid. The interossei and balls of thumbs are almost gone, and on the right more than the left; marked wasting of extensors of wrists, right also more than left; the right deltoid is, however, less wasted than the left, and both pectoral-maj. are somewhat affected. He can extend the left fingers, but not the right; can flex and extend both elbows, but cannot raise the arm higher than on a level with the shoulder. Has constant fibrillar tremor of the muscles of the arms and of the sterno-mastoids, and also especially in the pectoral-maj. and the deltoids.

Lower limbs feel feeble to him, and he thinks he walks stiffly, and his legs give way under him; the knees shake much and seem to "shoot him up;" cannot run, but can walk uphill better than down. He has some twitching in the muscles of his legs.

Sensibility and organs of special sense normal, and heart not affected.

He was discharged on January 15, 1880, but was re-admitted on October 14, 1880, when I found that he was generally weaker; his tongue was more wasted, and the mucous membrane thrown into folds; could protrude it only just beyond the teeth; the soft palate contracted fairly well in phonation. The upper limbs were more wasted, especially the small muscles of the thumbs and interosseous spaces, which were very hollow. He could not move the thumbs by their small intrinsic muscles, nor separate the fingers.

Both hands were flexed at the proximal phalangeal joints.

Forearms are also very thin, but he can just flex and extend the wrist-joints. Grasp of either hand by the dynamometer = 0. Can pronate fairly well, but not supinate forearms. Can extend the elbows, but not flex them so well; both upper arms wasted. Cannot quite flex the elbows, owing to the forearms being fixed in the pronation position. Deltoids are so wasted that he cannot abduct either arm from the trunk; he can, however, put the hand on to the opposite shoulder by action of

the pectoral maj. Can shrug his shoulders well. Supra- and infra-spinous fossæ are very marked and hollow. He can put his hands behind his back by the latissimus dorsi, and draw his scapulæ forwards by the serrati.

The fibrillar tremor is so marked that the fingers are continually being involuntarily moved. The muscles most affected are those which are not absolutely but only somewhat paralysed, the movement being most visible in the muscles of the arm, forearm, the pectorales majores, the sterno-mastoids and trapezii; while the muscles of the palm of the hand and the deltoid are not affected, or perhaps the latter are to a very slight degree; the quivering is more marked when the muscles are slack, and after they have been used.

He cannot lift the head forwards when lying on his back.

Lower limbs are somewhat wasted, the calves measuring 11½ inches in diameter. He has all the movements of the joints of the legs, but with diminished power. In walking he scuffs very much, dragging his feet along the ground. All the muscles of lower limbs, except the gluteus maximus, have fibrillar tremors, but less than in the arms. The muscles of the back are also affected in the same way.

The joints of lower limbs are rather rigid.

All the muscles, and especially those of upper limbs, are very sensitive to direct percussion, but the contraction is fibrillar, and not of the whole muscle as in health.

Knee-phenomenon (patellar tendon reflex) is very excessive; right and left ankle-clonus present on both sides.

Plantar reflex readily obtained on either side; also the *abdominal* and epigastric reflexes.

Electrical reaction to faradisation; the intrinsic muscles of the left thumb do not react to the strongest current of a Stohrer's battery; the left extensor digitorum and right and left interossei require a strong current; the muscles of the right thumb, the right biceps and extensor digitorum, react to one degree of the scale weaker; both flexor digitorum muscles and right deltoid respond a little better; while the triceps of both sides and the left deltoid and biceps are somewhat below normal. The pectoralis major and the trapezius on both sides, however, react readily to a normal stimulus.

Patient was discharged from the hospital in about the same condition, on January 1, 1881.

In July 1881 he was again readmitted, and said he had not been able to walk for three months—since April.

Swallowing is now more difficult, and speech worse; soft palate moves only very slightly in phonation, and he says "ngrum" for "grub;" but pronounces better when anterior nares are closed. Cannot blow with his mouth unless the

nostrils are closed. Soft palate moves better to reflex tickling with a pen.

Left forearm weaker than right. He cannot extend the fingers or wrists, though he can flex them a little; can just flex distal phalanx of thumbs, but cannot extend them. Can just flex the left but not the right elbow. Can just put either hand to opposite shoulder, but the left with more difficulty than the right.

Can use trapezii and serrati much less; otherwise the movements are as before, only weaker. Movements of thorax very slight, breathing being chiefly diaphragmatic. He cannot bring the head forwards when it is thrown back, without bending the whole body forwards. Quivering of muscles more marked.

Legs much more wasted; calves measure only 10½ inches. He cannot walk, and can only just stand by holding on to a rail; while lying down can lift the heels off the bed, but moves the knees and ankles very slowly and with very little power.

Rigidity, which is absent in the arms, is very marked in the joints of the legs.

Knee-phenomenon is very excessive, and *ankle-clonus* very well marked.

The plantar, abdominal, and epigastric reflexes are all well marked, the first two being excessive, the epigastric less pronounced.

The cremasteric reflex is also slightly obtained.

Oct. 10*th.*—Patient to-day, while eating, suddenly choked from impaction of food in his larynx, and before medical assistance could be given, he was dead.

For the following examination I am indebted to my friend Mr. Horsley, Surgical Registrar at University College Hospital.

EXAMINATION OF THE SPINAL CORD.

BY VICTOR H. HORSLEY, M.B., B.S. LONDON.

The cord was not in a good state of preservation, proving so brittle that it was unfortunately impossible to examine the dorsal region; this apparently was not due to any pathological softening.

Spinal Cord.—Membranes apparently normal, but the pia mater unusually adherent; microscopic examination showed, however, that the pia mater was considerably sclerosed, presenting on section an almost homogeneous appearance, from

the dense fibrous tissue of which it was composed, and containing very few nuclei corpuscles.

Vessels.—The middle coat of the smaller arterioles presented the same change, only much less marked, and the tunica adventitia had an unusually close texture; the internal coat was quite normal in every way. In one section a concentric calcareous mass of "brain sand" was found.

Nerve-roots.—Not diminished in size, nor altered in appearance (latter after hardening).

On microscopical examination, some sections of the nerve-roots showed slight destructive changes in the myelin sheaths; but these changes were post-mortem, rather than ante-mortem and pathological. The axis-cylinders showed no alteration at all. The perineurium partook of the general thickening of the fibrous tissues.

Spinal Cord. Neuroglia.—The trabeculæ passing in from the pia mater were everywhere strongly marked, and the neuroglia throughout the cord was increased, and stained readily with anilin black; but in the whole motor region of the right side and left descending lateral tract in the cervical enlargement, and in both descending lateral tracts in the lumbar enlargement, the increase of the neuroglia amounted to well-marked sclerosis, and in some places consisted of dense fibrous tissue. This increase in the connective tissue was simply an excessive fibrosis of the neuroglia, which (much thickened) showed a differentiation into fibres in those places where the lesions were most marked. The capillaries were surrounded by a dense ring of the same tissue, its thickness generally being equal to the diameter of the vessel, and here and there the lumen of the perivascular lymphatics was occupied by a delicate nucleated connective tissue. There was no trace of any inflammatory process, in fact the cord presented a markedly anæmic appearance. The neuroglia in the cornua of grey matter was also condensed, and filling up the gaps in the tissue formed as the cells degenerated (vide infra). The walls of the vessels in the grey matter were especially thickened, and connective tissue was noticed in the perivascular lymphatics, just as described above. In addition to these changes there was found a bright substance which stained moderately with anilin blue-black, and was of homogeneous appearance, with here and there a nucleus of a leucocyte. This substance was formed in the posterior commissure between the fibres, and forming a long mass stretching the length of it; it was also formed in the perivascular lymph-spaces and scattered in the grey substance, but most usually in the lymph-spaces. Besides these situations, it was present in the cervical region at the bottom of the anterior fissure, and resting on the pia mater. It

probably was of albuminous nature, it differed from myelin in amount of refractive power, and is perhaps identical with that substance lately noted in a case of tetanus (Doran).

Nerve Structures.—1. *Corpuscles.*—The large motor cells in the anterior cornua had undergone extensive degeneration and destruction. The degeneration was not so marked, however, as simple atrophy of the whole corpuscle, which was very obvious; the cells remaining having but few processes, and the protoplasm of the whole corpuscle not staining readily with anilin. The degeneration was pigmentary, and in the right anterior cornu of the upper third of lumbar enlargement two corpuscles were completely degenerated, appearing as masses of orange-yellow granular pigment. No vacuolation was observed in any of the remaining corpuscles. The whole process resulted in the total destruction of the majority of the nerve corpuscles; thus in the cervical region there were left scarcely any (opposite the level of the 6th cervical nerves), and the surviving cells were only scattered remnants of corpuscles. In the lumbar region, however, there were more to be seen, and in the upper third of the lumbar enlargement there were three or four partly degenerated corpuscles in the left anterior cornu, the cells belonging to the large infero-external group. On the opposite side there were three cells with two or three different processes, and belonging to the upper half of the similar group. This condition varied, so that at a point a few millimetres lower there was only to be seen one corpuscle of the left anterior cornu, and then at another point the large group of cells was fairly well represented, especially on the left side.

2. *Fibres.*—The fibres throughout the "healthy" parts of the cord appeared to be fairly normal, and there was no swelling or atrophy of the axis-cylinders, which, however, did not stain readily with anilin. In the sclerosed portions the fibres simply had undergone atrophy, and there was no increase in the axis-cylinder here at all; the fibres evidently had wasted and disappeared. There were of course all degrees of the process to be seen.

Central Canal in the cervical region was almost obliterated, and the epithelium multiplied. In the lumbar region it was flattened from before back, and filled with epithelium degenerated to a considerable extent.

Nothing abnormal was found in the medulla oblongata. The rest of the brain was not examined.

Remarks.—The above case was under observation from a few months after its commencement until the death of the patient, a period extending over two years and seven months, and it

was therefore possible to make clinical records throughout its course.

The disease began with "rheumatic pains" following exposure to wet and cold, and the first marked symptom consisted in weakness of the hands.

A month after the hands were first affected, he began to suffer from well-marked symptoms of disease in the medulla oblongata, such as: difficulty in speaking and swallowing; and when he first came under observation—six months after the first symptoms showed themselves—the bulbar symptoms were very characteristic, as: pain in the back of the head; difficulty in showing the upper teeth; a sad expression, from depression of the angles of the mouth (it may here be noted that the temperament of patients suffering from this disease is usually remarkably cheerful and sanguine); tremor of chin; food collecting between the cheeks and gums; tongue tremulous and wasted; speech, a drawling monotone, and uvula wasted and deviating to the left.

The muscles of the neck were wasted and weak, and there was atrophy with paralysis of certain groups of muscles in the upper limbs; but this was unattended by any rigidity.

The lower limbs were stiff about their joints and feeble, so that he walked with difficulty.

There was also marked fibrillar tremor in the affected muscles, which was powerful enough to cause involuntary extension of the finger; this tremor was subsequently noticed as being most marked in those muscles which were considerably affected, but to be absent in those which were so much destroyed as to be powerless. The tremor was also produced by direct percussion, and took the place of the contraction of the muscle as a whole, as occurs in health to direct percussion.

The subsequent course of the disease was progressively worse.

The soft palate failed to act in deglutition, but still reacted reflexly to tickling with a quill-pen, and the patient was thereby unable to inflate his cheeks or pronounce explosive consonants, unless the anterior nares were first closed.

The muscles of the neck also lost power, so that he could not bring the head forwards by the sterno-mastoids. The groups of muscles in the upper limbs became more affected and the legs more rigid, so that two years after the onset of the illness he was unable to walk, and the knee-phenomenon (patellar tendon reflex) became very excessive, ankle-clonus also being very readily obtained in both legs.

At the end of his illness respiration became chiefly diaphragmatic. The sphincters were unaffected throughout.

This case would seem to be one of progressive muscular

atrophy, followed by lateral sclerosis—amyotrophie spinale deutéropathique of Professor Charcot—and not one of amyotrophic lateral sclerosis, in which the first symptoms are due to primary lateral sclerosis, and consist of weakness, usually of the arms, with spasmodic permanent contraction and rigidity of their joints; atrophy *en masse*, and not muscle by muscle—the patient being, however, able to raise the arm, but with difficulty, due to the rigid condition.

In this case, on the other hand, the atrophy was primary and attacked individual muscles; loss of power followed the atrophy, and there was no rigidity in the arms. The rigidity of the legs, however, occurred after the wasting of the arms, and was the first symptom noticed in the lower limbs. In cases of progressive muscular atrophy the cells of the anterior cornua are affected most in the cervical and least in the lumbar enlargements, and this was found in the microscopical examination of the present case. Sclerosis of the lateral columns was, however, very well marked in the lumbar regions; so that though the joints of the legs were noticed to be somewhat stiff six months after the onset of the illness, inability to walk, coupled with increased knee-phenomenon, and well-marked ankle-clonus, did not supervene till two years after the onset. The legs, though small, did not present excessive atrophy of individual muscles, the appearance being probably due to lateral sclerosis more than to atrophy of the anterior cornua; and when unable to walk, he would still move all the joints of the legs, though with great difficulty, owing to descending sclerosis, which was perhaps antecedent to the wasting of the cells of the anterior cornua of the lumbar enlargement (?).

The absence of rigidity in the arms, notwithstanding the well-marked sclerosis in the lateral columns, was perhaps due to the extreme destruction of the motor cells in the cervical region.

The group of muscles most affected in the upper limbs comprised the intrinsic muscles of the hands, the extensors, and, to a rather less degree, the flexors of the fingers and wrists, the supinator longus, the flexors of the elbows, the deltoids and the supra- and infra-spinati; while the pectoralis major, latissimus dorsi, triceps, teres major, and subscapularis were less affected.

According to the experimental researches of Professors Ferrier and Yeo,[1] the segment of the spinal cord at the level of the 4th and 5th cervical nerves supplies the muscles contained in the first-named group, with the exception of the flexors of the fingers and the intrinsic muscles of the hand, which are supplied by the 8th cervical and first dorsal segment respect-

[1] Proc. Roy. Soc., No. 212, 1881.

ively; while the second group is supplied by the 6th, except teres major and subscapularis, which are supplied by the 7th cervical. The flexors of the fingers are also supplied by the 6th and 8th, and the triceps by the 8th cervical nerves, and the flexors have not suffered to the same extent as the extensors. The pronators supplied by the 8th cervical were also much less affected than the supinators. The above grouping is also borne out by the faradic reaction, where the flexor digitorum reacts better than the extensor; and, on the right side, the triceps better than the biceps and deltoid; while the muscles of the hand and the interossei react very feebly or not at all; the pectoralis major and trapezius acting about normally. Clinically, the above grouping is illustrated by the fact, that in January 1880, the patient could pronate, but not supinate the forearms; could extend, but not flex elbows; and though he could not abduct either arm from the trunk, he could put the hands to the opposite shoulders, and place the arms behind the back.

From the above grouping of the affected muscles, one might expect to find that the motor cells at the level of the 6th and 7th and perhaps the 8th cervical nerves would be less affected than the other parts of the cervical enlargement; but post-mortem examination, made three months after the patient was last examined, failed to reveal any difference.

The relative affection of the various muscles is in favour of the disease being progressive muscular atrophy and not a primary sclerosis of the lateral columns. With regard to the former disease there are two theories; the one, maintained by Friedereich and Lichtheim, that muscular atrophy is primarily a myositis; and the other, that it has a primary spinal origin. According to Dr. Ferrier,[1] the atrophy of the groups of muscles in accordance with the functional relations of the segments of the cord, as is also probable in this case, is strongly in favour of the spinal origin of primary muscular atrophy, and this is also borne out here by the bulbar paralysis and the attendant lateral sclerosis.

This case agrees with Dr. Ferrier's observations,[2] in that the disease commences from one end—in this case the lower—of the cervical enlargement, afterwards involving the upper end, while the middle part, represented by the 6th and 7th cervical, is much less affected. The 'main à griffe,' which is produced by invasion of the 1st dorsal and 8th cervical roots, did not occur, owing to the extensors of the wrists being involved in the lesion of the 4th and 5th cervical.

[1] 'Brain,' Part XV. [2] Ibid.

CASE OF MULTIPLE CEREBRAL TUMOUR.

Reported by

DAVID A. KING,

Late House-Physician, St. Bartholomew's Hospital.

R. D., æt. 16, was admitted under Dr. Andrew's care, November 24, 1881, and died February 3, 1882.

Previous history.—Had been on a training-ship for three and a half years, till six weeks before admission, when he took to brick-making. Had always enjoyed excellent health, though just before his discharge from the ship the surgeon had treated him for "sore-throat," which lasted a week.

While brick-making he had a fall upon his head which, however, appeared to have done him no harm.

He had never had any fits.

Family history.—Father died young of "consumption." Mother "consumptive." One sister out of four died of fits; the rest were in good health.

Present illness began about Oct. 20, with slight diplopia, frontal headache, and staggering gait. On Nov. 20 he vomited four times, but this had not continued. His appetite was good, and he was able to swallow well. His bowels were regular. Beyond complaining of headache and double vision, he had seemed well in himself, and had never kept his bed.

On admission.—He was a well-nourished boy, with a heavy expression and flushed face. Internal monocular squint of right eye. Secondary squint greater than the primary. [Paralysis of right external rectus.] Pupils dilated, the left one rather more so than the right. Both fairly sensitive to light.

The ophthalmoscope revealed nothing abnormal in either disc or fundus. Vision very much impaired; could count fingers at three feet. Character of diplopia could not be ascertained. Partial right facial palsy. Closure of the right eye less perfect than of left.

Mouth drawn slightly to the left.

Tongue moist, thinly furred, deviated to the *left* when protruded.

Agustia to aloes on the right side of the tongue. Taste good on the left.

Complete anosmia to asafœtida in both nostrils. Hearing fairly good. No otorrhœa. Fauces quite anæsthetic. Epiglottis can be felt without causing discomfort. Tonsils enlarged and congested; no exudation upon them.

Voice guttural and rather hesitating. Swallowing slow and difficult, the patient appearing to experience most difficulty in getting a bolus to the back of the fauces, but even after succeeding in that, its descent was slow.

No cutaneous anæsthesia could be detected. Chest carinate. Heart and lungs natural. Abdomen not retracted. Abdominal reflexes equal and natural.

Micturition and defecation natural.

Urine loaded with lithates. Sp. gr. 1032.

No albumen or sugar.

Pulse 52, regular, small and soft.

The temperature was normal throughout. His limbs appeared natural; no bed sores, no muscular wasting.

Slight exaggeration of right plantar reflex.

Slight ankle-clonus on both sides.

Some exaggeration of the knee-jerk on both sides, especially the right. No rigidity.

Patient experienced no difficulty in maintaining his equilibrium, when he stood with his eyes shut and his feet close together.

In walking the right leg drags, like that of a partially hemiplegic person, and his gait is jerky, and marked by some propulsive tendency.

The left arm was distinctly weaker than the right, though all its normal voluntary movements could be well executed.

The diagnosis of the cause of these various partial paralyses was at first sight doubtful, and, considering the anæsthesia of the fauces, coupled with the history of sore throats, Dr. Andrew suggested the possibility of diphtheritic paralysis. The progress of the case cleared up these doubts, and the presence of multiple cerebral tumour was recognised long before the diagnosis was verified.

The course of the case was marked by gradual emaciation, frequent vomiting, retraction of the belly, increase in the extent and degree of the paralysis, with retention of urine and of fæces, and by the occurrence of spells of a semicomatose condition.

These last occurred at intervals of two or three weeks, their onset was gradual, and they lasted from two to four days.

They passed off as gradually as they commenced, and their severity increased with each successive attack. In the fifth fit the boy died.

As a description of one of these attacks, I give the note of the fourth, taken on January 18:—Lies on his left side with his head bent forward, the legs drawn up, and the arms either between his legs or crossed over his chest. Pupils contracted (from opium in the nutrient enemata). No corneal reflex. Constant dribbling of long ropes of viscid mucus from his half-opened mouth. Respirations 27, regular and shallow. Moans occasionally, but for the most part is quite silent and motionless. Opens his mouth slightly when told to show the tongue. Takes but little notice of questions. Puts his arm under the clothes if it is left lying out of the bed. Pulse 84, regular, small and soft. Heart's impulse feebly felt through the thin thoracic wall.

Abdomen retracted. No plantar reflex.

Knee-jerk exaggerated on both sides.

Front-tap contraction more marked on the left side than the right.

Achilles jerk exaggerated, both sides.

Slight ankle-clonus.

Patient moves both legs and both arms voluntarily. The paralysis of the right side of the face remains as on admission.

On the day of his death the note is: Comatose, lies on his back, his face on its left side, with purulent viscid matter dribbling from the mouth. Eyes half open, glazed, roll from side to side occasionally. Can swallow nothing and retains no enemata. On attempting to feed him with a catheter in his mouth, last night, respiration became so embarrassed as to compel the abandonment of the attempt. During the first two comatose fits there was retention of urine requiring the use of the catheter, and the peculiar condition was noticed, that, after a certain amount of urine had been drawn off, no more came till pressure was made over the pubes, and after the bladder had been emptied in this way it was noticed that on withdrawing the hand from the hypogastrium, as the abdominal wall filled out, so air was sucked in through the catheter, and not only air, but some of the urine could be made to re-ascend the catheter. An example, this, of the "retentive power of the abdomen," upon the existence of which Dr. Matthews Duncan has insisted.

Optic neuritis was first observed after the first attack of coma, and soon progressed to general neuro-retinitis, but there was never much swelling of the discs, and never any patches of white effusion over the vessels.

A corneal ulcer formed in the right eye, but under fomenta-

tions and atropine drops it completely healed a fortnight before death.

Great fæcal accumulation several times occurred and was marked by abdominal tumour, and required the frequent use of the scoop.

Treatment.—For the first few days he took liquor strychniæ mjij. and tinct. cinchonæ. Afterwards he was put upon potassii bromid. gr. xv. three times a day, and for the last fortnight upon potassii iodid. gr. v. three times a day.

Frequent purgatives, calomel, croton oil and enemata, were required.

At first able to take milk diet and bread and butter, he was soon unable to take any solids, and was fed with nutrient enemata. For a week before his death these were not retained, and he was fed by a catheter passed through the mouth.

Post-mortem Examination, made twenty-two hours after death.

Rigor mortis well marked. Signs of commencing putrefaction about the belly.

Calvarium healthy. No sign of injury to the skull. Dura mater natural. The veins, especially those on the right side of the vertex of the cerebral hemispheres, were very full, and this venous turgescence was as obvious over the frontal convolutions as over the occipital. Convolutions much flattened, sulci nearly obliterated, and subarachnoid fluid absent or very scanty.

On opening up the longitudinal fissure with the fingers, its anterior third was obliterated, so that on separating the hemispheres with the handle of a scalpel the pia mater was torn from the inner aspect of one hemisphere. Surface of brain appeared natural, but the posterior half of the corpus callosum bulged up somewhat, and undue resistance to pressure was felt there. On lifting up the frontal lobes preparatory to removing the organ, no fluid was seen in the optic sheaths, which were dry and filled with air as the optic nerves were cut.

No tubercles anywhere along the arteries, the distribution of which was normal. On making the centrum ovale majus a large quantity of clear fluid escaped from the lateral ventricles, which were much distended. The fluid measured ʒvij.

The third ventricle also contained much fluid, but the fourth was empty. There was a large commissura mollis. The corpora quadrigemina were more prominent than natural.

On turning the brain over to inspect the base, a ragged, sloughy-looking blood-stained mass, the size of a small Tangerine orange, was seen occupying the right side of the pons Varolii, extending up the middle peduncle (crus ad pontem) nearly as far as the cerebellar substance, but not invading the

left half of the pons. This mass broke down under the finger, and could be washed away by a gentle stream of water.

There was no capsule to the tumour, the substance of which merged gradually into that of the surrounding healthy parts. On making a section into this, its thickness was about equal to that of the healthy tissue above it, i.e. the corpora quadrigemina. The left side of the floor of the fourth ventricle bulged up somewhat, and the olivary body and left half of the medulla also appeared swollen. On cutting into it, a rounded mass, half an inch in diameter, was found imbedded in the white matter. This was sharply defined from the surrounding tissue, and showed no signs of infiltration. It was of light chocolate or dark fawn colour, appeared spherical, did not cross the middle line, nor did it pass up the peduncles or down the cord. Of firm consistence, it could not be shelled out of its position. No visible degeneration of any of the columns of the cord below was found.

For the microscopical examination of these tumours, I am indebted to the kindness of Mr. Bowlby, the Curator of St. Bartholomew's Museum. Dr. Klein examined one of Dr. Bowlby's sections, and entirely agreed with him as to the nature of the growth. The following is Mr. Bowlby's report: "The two tumours were of almost identical structure, the only difference being that one was firmer than the other, and that into the latter some hæmorrhage had occurred. The new growth was sharply defined from the surrounding brain substance, which was stretched over it in a thin layer.

"The new material consisted entirely of perfectly-formed neuroglia, in which were found numerous nerve corpuscles, both branched and unbranched. In some parts these corpuscles appeared rather smaller than normal, and had lost their branched processes, but in the greater part of the growth their structure was perfectly natural. The tumours were abundantly supplied by blood-vessels, many of which were extremely thin walled, and appeared to be new-formed.

"The nerve fibres had almost entirely disappeared.

"The growth appears to have commenced in a hyperplasia of the neuroglia, hyperplasia in which it is probable that the blood-vessels also shared; for if such had not been the case, it is unlikely that the nerve cells would have preserved their normal structure as they did almost throughout the tumours, and that the neuroglia itself should have been so perfectly formed, and not shown any tendency to thickening or contraction.

"The nerve fibres, however, do not appear to have been able to exist together with the growing tumour, and it is in their absence alone that the tumours differ from the normal structure of the surrounding healthy brain substance. The thin-walled

vessels had in some places, in the larger, softer tumour, given way, and small extravasations had occurred."

The case is of interest as much from the peculiar histological characters of the morbid growths, as from the accuracy with which they could be recognised before death. The frequent attacks of partial coma, taken in connection with the seat of disease, seem analogous to the similar conditions occasionally noted in diseases affecting parts of the brain, other than the hemispheres, e.g. Disseminated and Lateral Sclerosis, &c. In most such cases the medulla has been found to be affected.

Abstracts of British and Foreign Journals.

Report on Visceral Neurology.—The Heart. *Effect of Galvanism in the Human Subject.*—The effect of electrical excitation of the heart in the human subject has been very carefully investigated by Professor Ziemssen in a woman with a large defect of the anterior thoracic wall, the result of operation. A considerable portion of the præcordial structures had been removed in the excision of a tumour; and the ventricular and left auricular surfaces, the root of the pulmonary artery, the left coronary artery, and the left phrenic nerve could be distinctly made out, lying at the bottom of a deep hollow. (*Deutsch. Archiv f. Klin. Med.* xxx., S. 270.) The normal movements having been carefully ascertained, as well as the effect of mechanical irritation, the influence of the induction current, and of the continuous current variously interrupted or non-interrupted, was thoroughly investigated.

The results appear to have been perfectly definite, and may be summarised as follows:—

1. The *induction current*, of whatever strength, in whatever situation and in whatever fashion applied, produces no effect either upon the action of the heart, or in the way of pain or other sensation.

2. The *galvanic current*, on the contrary, affects the frequency and rhythm of the cardiac action, as well as the character of the individual contractions.

a. Each individual galvanic irritation is followed by a visible and palpable contraction of both ventricles, when powerful currents are used. The effect of cathodal closure was acceleration of the beat from 80 to 140, and elevation of the apex of the cardiographic curve.

b. A strong galvanic current produces a *sensation* of tearing behind the lower part of the sternum, but no pain, beyond some "feeling" in the left arm.

c. A strong galvanic current, uninterrupted by opening or

closure, applied to certain parts of the ventricular surface, raises the frequency of contraction to two or three times the normal, the rhythm remaining remarkably regular. Unquestionably the nervous structures, and not the muscular substance, must be considered the irritable elements. For, first, the irritable area extended from the auriculo-ventricular groove 2 cm. downwards upon the ventricle—upon the left side, behind the descending branch of the coronary artery; on the right side, immediately in front of and upon the phrenic nerve. Secondly, repeated irritation increased six or eight times the irritability of the centres. Thirdly, when a muscular contraction was induced, it was equally well-marked, whether the irritation was slight or powerful.

d. To *reduce the frequency* of the heart, very powerful galvanic currents are necessary, and in these instances the rhythm always becomes irregular.

3. Galvanisation of the heart *through the chest walls* (as in ordinary individuals) produces acceleration, the force and the rhythm being also influenced; this result was confirmed upon other subjects.

Accelerator Nerves.—A case of injury of the neck under the care of Dujardin-Beaumetz appears to illustrate clinically the situation of the accelerator nerves of the heart in the cervical portion of the cord. A man fell on his head, and sustained a contusion of the four lower cervical vertebræ and the spinal cord, the result being paresis of both arms, with pain along the course of the brachial plexus. Even for months after the accident, the pulse exhibited a remarkable difference in frequency, according to posture, being, for example, 49 in recumbency, 73 in the sitting position, and 100 in standing. (*Bull. gén. de. Thérap.* &c., 1882, Feb. 28; and *Centlbt. f. d. Med. Wiss.* 1882, p. 432.)

Vaso-motor System. *Tracts in the Cord.*—An investigation by Nicolaides, in the Physiological Laboratory at Leipsic, contributes somewhat to our knowledge of the course of the vaso-motor fibres in the spinal cord. By unilateral section of the cord in the lower dorsal region, and galvanisation of the cervical portion, with necessary precautions, Nicolaides determined that the vaso-motor nerves of the kidney are derived from both sides, although the kidney corresponding to the undivided half is more irritable than the other. In other words, the vaso-motor nerves of the kidneys, in their course down the cord, lie chiefly in the corresponding half of the cord, but partly in the opposite half. Observations upon the carotids appeared to indicate that the same anatomical disposition

holds good for this part of the vaso-motor system also. (Du Bois-Reymond's *Archiv*, 1882, S. 28; and *Centlbt. f. d. Med. Wiss.* 1882, S. 466.)

Peripheral Mechanism.—The mechanism by which the blood vessels react to the influence of change of temperature has been shown by Lewaschew to be peripheral in its seat. The hind leg of a dog was completely removed from the body by the thermocautery, and the local vitality maintained by means of an artificial circulation of defibrinated blood, which could be done with perfect success for 8 or 10 hours. It was then found that the vessels of the part contracted on exposure to moderate cold, and dilated on exposure to heat and severe cold. Digitalis and nitrite of amyl even more distinctly influenced the size of the vessels. An important observation was to the effect that the dilating influence remained unaffected by degeneration of the sciatic and crural nerves, showing that the nervous influence must reside in peripheral centres; for organic muscular fibre, on being tested by similar experiment, was found to become relaxed by cold and contracted by heat. (Pflüger's *Archiv*, xxvi. S. 60; and *Centlbt. f. d. Med. Wiss.* 1881, 918.)

Respiration. *The Vagus in Respiration.*—The effect of electrical stimulation of the vagus on respiration continues to occupy the attention of physiologists, without removing much of the difficulty which surrounds this subject. Very different results are obtained from time to time by different observers, and much ingenuity is exhibited in attempting to reconcile or otherwise account for the conclusions.

It will not have been forgotten that Rosenthal, as described in 'Brain' for October 1881, p. 418, has recently re-examined the conclusions at which he had arrived more than twenty years ago, upon the mechanism of respiration, and which have formed the foundation of all physiological and clinical teaching upon the subject ever since. Rosenthal adheres practically to his former views; whatever modifications may appear in his plan of the respiratory mechanism being in respect of the manner of stating it, rather than of matters of fact. The publication of these results of Rosenthal's has naturally caused a certain amount of disappointment to the younger physiologists who had been working at the subject of respiration for the last few years, and who had been publishing from time to time, as we have had occasion to notice, the results of elaborate investigations, more or less at variance with Rosenthal's. We are accordingly beginning to receive replies

to Rosenthal's recent paper; and a prolonged and elaborate discussion may doubtless be expected for some time, before the matter is allowed to rest. Gad of Würzburg, whose observations on the vagus have been more than once referred to in these pages, has opened the discussion by questioning the soundness of one of the fundamental principles of Rosenthal's theory of the regulation of respiration. According to the latter, as is familiar to all, stimulation of the vagus accelerates the respiratory movements, but weakens them at the same time; stimulation of the superior laryngeal nerve slows the respiratory movements, but deepens them at the same time. In other words, stimulation of the vagus does not increase the activity of the medulla; *it only distributes the energy* (or the muscular contraction) *in a different manner.* It will be seen that the principle involved here lies at the bottom of inhibition in general; the problem being whether inhibiting impressions alter the amount of force liberated within a given time, or whether they merely distribute the force differently, the frequency and the amount varying inversely with each other, whilst the total effect remains the same. Gad maintains that the latter is not the case with the respiratory mechanism, any more than it is the case with the heart, and that stimulation of the vagus distinctly influences the amount of force liberated by the respiratory centres in the medulla, the result being acceleration with diminished depth of the individual respiratory movements, in association sometimes with distinctly increased, sometimes with distinctly diminished, respiratory efforts. The proofs offered in support of this contention consist not in any new observations, but in a restatement of previous experiments, which have been already recorded in these *Reports* (Du Bois-Reymond's *Archiv*, 1881, vi., S. 538).

Wedenskii has also obtained very different results in the rabbit, according to the strength and duration of the faradisation, and the period of the respiratory rhythm at which the current was admitted to the vagus. The strength of current being gradually increased, transient irritation caused shortening of inspiration; the strength being further increased, an effect in expiration also made its appearance in the form of shortening. To explain this result, which agrees neither with Rosenthal's nor with Gad's view (1880), Wedenskii suggests that the same irritation diminishes the excitability of the respiratory centre in inspiration, and increases it in expiration, the inhibitory effect being the first to appear. The former effect is the result of sensory irritation of a centre during its motorial activity; the latter, of sensory irritation of a centre

during its inactivity. Bubnoff and Heidenhain have found that the same law holds for cerebral (motor) centres, and the sensory tracts corresponding. When the irritation of the vagus was protracted, inspiration alone was shortened; and this result, in Wedenskii's opinion, strengthens his view. (Pflüger's *Archiv*, xxvii. S. 1.)

Meanwhile Meltzer of Berlin has offered a curious explanation of the discrepancy of these and similar results. He observed that the results of faradisation of the vagus vary in different animals, not in the same individual; and an analysis of his subjects revealed the interesting fact that female animals gave one result, male animals another and a different result. He frankly states that the observation is of value only as far as it goes. In eight *female* rabbits the *expiratory* effect of faradisation of the vagus was unquestionably the more marked. In seven *male* rabbits the effect was purely *inspiratory*. This result reminds one of the male and female types of respiration in man. (*Centlbt. f. d. Med. Wiss.* 1882, S. 497.)

The Respiratory Centres.—Our readers may remember that twelve months ago an account was given of the novel conclusions arrived at by Langendorff with respect to the "respiratory centre," *par excellence*, in the medulla; this physiologist suggesting that it is a *regulating* centre for the much more widely diffused respiratory centres in the cord and brain, and that its function is chiefly *inhibitory*. A fresh series of experiments, performed by himself and Gürtler, appear to confirm Langendorff's theory (Du Bois-Reymond's *Archiv*, 1881, vi., S. 519). Electrical stimulation of the medulla, carried out with the greatest care, yielded an astonishing variety of results, both as regards the frequency and the type of respiration; the expiratory phenomena on the whole predominated, however. There would thus appear to be both an inspiratory and an expiratory centre in the medulla, and these with a distinctly *inhibitory* function. The conducting, innervating, centres are, according to Langendorff, not situated in the medulla; and he describes a method by which he was able to mark out the limits of these, as distinguished from the inhibiting centres. It is important to note that the latter pass for some distance into the cord also. Langendorff gives further an interesting account of the manner in which the inhibitory centre in the medulla could be *stimulated* by slight pressure on the part, with the effect of arresting respiration.

Cheyne-Stokes Respiration.—"Periodical" respiration is a subject of the greatest possible interest to the physician, who is still in

search of a satisfactory explanation of "Cheyne-Stokes respiration." Many elaborate experiments, and observations of other kinds, have been made from time to time, and various theories propounded with more or less plausibility, respecting the cause of the ascending and descending rhythm and the periodic arrests in this remarkable form of breathing. The chief importance has naturally been attributed to an intermittent (insufficient) supply of oxygen to the respiratory centre, and consequent inequality of the nervous discharges, the depth of the respirations steadily increasing until a sufficient amount of oxygen is admitted to lower the irritability of the centre, whereupon the descending rhythm commences. Manifestly there are difficulties in accepting this explanation as sufficient. Langendorff (Du Bois-Reymond's *Archiv*, 1881, S. 241 and 331) has recently made what would appear to be an important suggestion in connection with the subject. He has found that, in frogs, periodical breathing may be set up by cutting off the blood-supply to the medulla, whether by tying the aorta, by rapidly bleeding from the heart, or by arresting the ventricle with digitalis; that restoration of the blood-supply restores the natural rhythm; and—which is of special importance—that the disturbance does not occur under these circumstances in the "salt-frog," *i.e.* if the circulating blood be replaced by a "physiological" salt-solution. It is obvious from these observations, that in constructing a theory of the cause of "Cheyne-Stokes respiration," we must attach more importance to the *nutritive* function of the blood supplied to the medulla, and somewhat less importance to its *respiratory* function. We appear to have laid too much stress upon the insufficient supply of oxygen to the centre, and to have comparatively overlooked the insufficiency of the supply of plasma, by which alone the nutrition of its irritable tissue can be maintained.

The Spinal Cord.—The localisation of the spinal centres in clinical practice is illustrated by a case under the care of Fürbringer. A man of 69 sustained a fracture of the spine, with complete paraplegia and paralysis of the bladder. Priapism supervened thirty hours after the accident, and the urine contained spermatozoa; thereafter there was a steady discharge of semen, until death on the third day. Post-mortem, a laceration of the cord was found opposite the fourth dorsal vertebra, indicating the limits of the genital centre. (*Berl. Klin. Wochen.* 1881, No. 43; and *Centblt. f. d. Med. Wiss.* 1882, S. 396.)

"Trophic Nerves."—*Skin Diseases.*—Several attempts have recently been made to discover actual anatomical evidence of lesion of

the nerves or nerve-centres in certain diseases of the skin which have long been believed to be of "nervous" origin. Herpes especially has been carefully studied in this connection. Thus, Lesser has recorded in Virchow's *Archiv* (lxxxvi. S. 390) two cases of herpes in which he found disease of the corresponding spinal ganglia; as well as two cases in which curvature of the spine was associated with zoster on the side of the convexity.

Leloir argues in favour of the same connection in the case of vitiligo, ichthyosis, ecthyma, pemphigus, and gangrene of the skin. Having shown that parenchymatous degeneration of the nerves can be demonstrated in these forms of disease, he points out that the nerves are free from similar degeneration in severe diseases of the skin, such as lupus and epithelioma, and that the nervous lesion is therefore most probably the primary change. This circumstance, as well as the fact that such degeneration is found precisely in diseases which appear to be of nervous origin (herpes, vitiligo, &c.) suggest their true pathology. The most plausible theory of the origin of these lesions is, according to Leloir, the trophic one, whether they be referable to disturbance of the trophic nerves, or of the trophic centres by reflected irritation (*Centblt. f. d. Med. Wiss.* 1882, S. 621).

Letulle relates an instance of herpes in the region of the first division of the right trifacial nerve, commencing with severe neuralgia, and followed in three weeks by facial paralysis of the same side; but he appears to attribute the whole to the local influence of chill (*Arch. de Phys.*, &c., 1882, No. 1). Joffroy describes in the same journal a case in which herpes of the right shoulder was followed, in the course of three months, by atrophy of the intrinsic muscles of the corresponding hand, with degenerative electrical reaction. Similarly, herpes of the left forearm was followed by severe pain in the region of the circumflex nerve of the same side, and weakness and atrophy of the deltoid.

Locomotor Ataxy.—Fresh observations continue to be made of disturbances of nutrition in association with locomotor ataxy. Besides the familiar diseases of the joints and bones, and perforating ulcer, there have recently been recorded peculiar affections of the teeth and nails, ending in their shedding. Thus Demange (*Revue de Méd.* No. 3, 1882) describes a case of tabes in which all the upper teeth fell out in a short time, without pain of the usual kind, but with darting "lightning" pains through the face, and disturbance of sensibility in the region of the trifacial nerves. Post-mortem, the floor of the fourth ventricle presented sclerosis, which involved the nuclei of the ninth, tenth, and eleventh nerves, the restiform bodies, and

some of the neighbouring parts. Sclerotic change was distinctly seen in sections of the trigeminus where it leaves the pons. In another very similar case, the loss of teeth was confined to the left side of the upper jaw. The post-mortem appearances were almost exactly like those just enumerated, but more marked on the left side. One of the instances of loss of the toe-nails is given by Joffroy (*Arch. de Physiol.* &c., 1882, No. 1). The great toes were the members affected, and there was no accompanying pain; the nails simply looking dark, with subungual ecchymosis. The part was soon restored. Pitres relates in the *Progrès méd.*, No. 8, 1882, somewhat similar cases, in which, however, both great-toenails were shed repeatedly, after deep-seated dull pain, and a sensation of creeping in the affected parts for several weeks. There was no ulceration or suppuration in these cases, any more than in Joffroy's; and the new nails were in every instance perfectly formed, with the exception of slight superficial irregularities. An interesting instance of necrosis of the phalanges of the two great toes in a case of locomotor ataxy is described by Dr. Russell, of Birmingham, in the *Med. Times and Gaz.*, August 19, page 210.

J. Mitchell Bruce.

Schwalbe on the Relations of the Calibre of Nerve Fibres. (*Leipzig*, 1882.)—The author points out the error of supposing that there is no law regulating the size of the nerve-fibres. Although apparently fibres of different sizes are heterogeneously mixed together, there are reasons for the variations. A basis of classification was found in Remak's observations upon the gelatinous fibres, and a physiological distinction, *e.g.* motor or sensitive, was regarded as influencing the size of the fibres. Schwalbe adopts the law of M. Pierret that the size of the cell and thickness of the nerve-roots are regulated by the distance at which its nervous influence is exerted, and identifies this with Deiter's remark that the size of the cell is in proportion to the thickness of the proceeding axis-cylinder. The variations in size of the nerve-fibres, the author adds, are less striking in the brain than in the spinal cord. These variations, Schwalbe states, are influenced by the size of the animal, by the extent of distribution, by the physiological attributes of the nerve, and by the amount of usage it ministers to. These differences, the author concludes, cannot be accidental, as shown from the numerous and elaborate tables of the dimensions of nerve-fibres in man and in the lower animals, with which he has illustrated his observations.

Laura on the Structure of the Spinal Cord. (*Archives Italiennes de Biologie*, tome 1, fasc. 2, April 1882.)—Dr. Laura commences his remarks by the observation that the acquisition of the medullary sheath is the distinctive character of all processes from nerve-cells that become nerves. His object has been to trace these processes or nerve-fibres from their cells to their ultimate distribution. His researches apply more particularly to the cells of the anterior cornua, of the Stilling's nucleus, or Clarke's posterior column, and of the posterior cornua.

The descriptive details are illustrated by explanatory plates, showing the course of medullated nerve-fibres.

The following summary places before the reader the author's conclusions:—

1. The cells of the anterior cornua send their nerve-processes, in the greater number of instances, into the anterior nerve-roots.

2. Fibres from different points of both of the anterior and posterior cornua contribute to the formation of the anterior commissure.

3. The cells of the nucleus of Stilling (posterior column of Clarke) supply nervous prolongations which pass at first inwards, then after a long course in the same direction fold backwards, and go to form a large bundle passing into the lateral column.

4. The lateral column receives fibres from different points of both anterior and posterior cornua.

5. The cells of the posterior cornu furnish processes which pass in various directions; *a*, into the anterior commissure; *b*, directly to the anterior roots; *c*, into the lateral columns; *d*, into the posterior columns; *e*, across the middle line behind the central canal into the opposite cornu.

6. Cells are found in the cord which send nerve-processes in opposite directions, and act intermediately in the change of direction of the fibres.

A copious bibliography concludes this essay, which embodies a series of observations, modestly spoken of by the author as not very numerous, but which, considering the complexity of the investigation, may be regarded as an important contribution to the histology of the spinal cord.

Edinger on a Case of Congenital Absence of a Forearm.—(Virchow's *Archiv*, July 1882, p. 46 *et seq.*)—The small number of histories of such cases, as the above-named, induces Dr. Edinger to place the present on record, at the same time that he gives a slight

sketch of the literature of the observed condition of the nervous centres after amputation of the limbs.

C. K., a male, aged 52 years, died of disease of the heart in the hospital, at Riegel. At his birth he was deficient of the left hand and greater part of forearm. A stump of about the size of a child's fist was found at the elbow-joint, and terminated in a prominence of about the size of a bean, on which were two small tubercles. He had considerable power of movement of the stump, and as a dealer in poultry, could carry heavy baskets by means of a hook attached to the stump of his arm.

On examination, after death, the muscles of the left arm were thin, but normal in structure, and ended in a tendinous expansion inserted into the integuments. The nerves of the forearm were thin, and distributed to the muscles.

On opening the vertebral canal there was, to outward appearance, no difference between the right and left side of the spinal cord in the upper part of the cervical region, but at the point of exit of the sixth and seventh cervical nerves the left side of the cord was distinctly smaller than the right. After immersion in Müller's solution, the following appearances were noticed. The posterior nerve-roots from the fifth to the eighth cervical were thinner on the left than on the right side, only in the fifth, sixth, and seventh anterior roots the difference was equally striking. The anterior root of the eighth cervical nerve was only half as thick as on the right side.

A microscopical examination of the cord from the second to the eighth nerve was carefully made by a series of successive sections. It was at the bend of the fourth and fifth nerves that atrophy of the structures on the left side became most marked. The anterior appeared cut short and bent backwards, and was deficient in nerve-cells. The white matter was also smaller than on the right. A similar change was here observable also in the posterior horns. The highest degree of atrophy was reached about the region of the sixth and seventh cervical nerves, so that it was almost impossible to trace the cells or their processes on the left side. No trace of any other form of lesion than simple wasting of the structures was observed. The further examination presented much the same anatomical changes as observed after amputation, so that the present case is considered by Dr. Edinger to be one of intra-uterine amputation. In those the condition of the cord is that of atrophy of the cornua, their cells and nerve-roots on the side of the amputated limb. The wasted nerves and their roots could be traced to wasted portions of the horns.

The brain having been hardened in Müller's solution, it was observed that the convolutions were well marked; but on the right side the two central convolutions were distinctly smaller than on the left, in their lower half. It would seem that in this, the first case that has been examined, the atrophied region in the spinal cord has a corresponding wasted zone of the cortical substance of the brain, an observation lending support to the established facts of localisation of function. Dr. Edinger sums up the lesions observed after amputation as consisting in atrophy of the nerve-roots and white substance of the cord, and of the anterior horns, with degeneration of their cells, whence diminution of the white columns from decrease in the number and size of fibres. This occurring during growth, the motor zones of the brain will suffer arrest of development.

Schultze on Fissures, Cavities, and Gliomata in the Spinal Cord and the Medulla Oblongata. (Virchow's *Archiv*, March 1882.)—Notwithstanding the large number of recorded observations of these lesions, there is as yet, Dr. Schultze observes, no approach to uniformity of opinions as to their source and nature. Westphal traces the fissures to degeneration of the neuroglia; Simon, to abnormal congenital development of the central canal; while, more recently, they have been attributed by Langhaus to congestion in the cerebellar spaces. Dr. Schultze, for the elucidation of the question, gives the record of several cases in which these lesions were found.

Obs. 1. A case of chronic arthritic disease, with contraction of the upper limbs and agonising pains of the lower extremities. There was found a fissure in the medulla, and a transverse fissure also in the cervical region, extending to the posterior horn. In the dorsal region, degeneration of the lateral columns was observed, with fissures and cavities in the posterior horn. The central canal was obliterated by excessive growth of the ependyma. *Obs.* 2. Neuroses of the upper extremities, originating in typhus; hyperæsthesia of all the limbs, with severe pain in the pelvic region more especially. In this case fissures and glioma (sclerosis?) were found in the posterior columns. *Obs.* 3. A case of imbecility from hydrocephalus. Partial atrophy of the centre horns and columns of the dorsal region. Degeneration of the Goll's columns in the cervical region. A cancerous growth was connected with the brachial plexus, causing peripheral paralysis. *Obs.* 4. Atrophy and paresis of the upper extremities, diffused analgesia and partial

anæsthesia, bulbar phenomena supervening. There was seen almost total destruction of the grey matter of the cervical region, fissures in the medulla oblongata and partial atrophy of the olivary bodies. *Obs.* 5. Case of amyotrophic lateral sclerosis, with spreading glioma in the cord.

These observations suffice to show that other morbid conditions than that of hypertrophy of the neuroglia concur to produce the fissures and cavities that are found in the cord, and that in some cases a veritable glioma is observed. In some sections various stages of softening may be traced in the columns of the cord and medulla. This variability of consistence is regarded by Dr. Schultze as evidence of there being degeneration of the neuroglia. Macroscopically, the newly formed fissure is soft, spongy, or gelatinous and of a grey colour. Microscopically, degenerated cells and fibres may be discovered in these. In answer to the question—how are these fissures and cavities to be explained?—their concurrence with glioma shows that they cannot be accidental. Dr. Schultze considers that there is direct evidence that they depend upon disintegration of the neuroglia. The condition of the neuroglia, both near to, and at a distance from the fissures, may vary in one and the same case, and may present differences which may seem to be accidental but are not so. The growth of the neuroglia bears some relation to the age of the individual, as does also the obliteration of the central canal by the so-called ependyma. From this structure proceeds a development of cells and fibres into the surrounding structures, the anterior and posterior commissures.

W. B. Kesteven.

Facial Monospasm.—Dr. H. J. Berkley (*Medical News*, July 15, 1882) reports a case of spasm of the left angle of the mouth which had existed in a patient, without other symptoms of motor disorder, for a period of two years and a half. Death occurred suddenly.

Extensive cardiac disease was found after death. The cerebral arteries were atheromatous.

The only lesion found in the brain was a calcareous nodule of nearly circular shape, measuring only three-sixteenths of an inch in diameter, situated quite superficially in the right ascending frontal convolution an inch and a half above the fissure of Sylvius —corresponding with the position of the centre for the zygomatic muscles as defined by me.

Blindness and Deafness with Bilateral Cerebral Lesion.—Dr. J. C. Shaw, of Brooklyn, reports (*Archives of Medicine*, Feb. 1882) an important case of a woman, aged 34, who, two months before her admission into the asylum, complained of loss of power in the right arm, and soon after had a sudden attack of loss of consciousness, loss of speech, and deafness. The condition as to vision was not noted at this time. The loss of power in the right side soon passed off. She became incoherent, more or less maniacal at times, and demented. On admission she was found to be perfectly deaf and blind. The pupils were slightly dilated and regular. Tactile sensibility and smell were normal, and, with the exception of slight paresis of the right hand, there was nothing beyond the deafness, blindness, and demented condition described. She suffered from repeated epileptiform convulsions, chiefly on the right side, and died of pneumonia a year after admission.

Post-mortem examination showed *complete atrophy of the angular gyri and superior temporo-sphenoidal convolutions* of both hemispheres, almost exactly symmetrical, as indicated by the figures which illustrate the text.

On microscopic examination of the atrophied regions, it was found that the grey matter had entirely disappeared, leaving the outer layer attached to the pia mater. Between this and the medullary fibres there was a cavity formed at the expense of the grey matter. The cranial nerves were normal in appearance. The microscopic examination of the optic nerves, however, indicated an increase of the connective tissue septa and atrophy of the nerve fibres, with areas filled with a colloid-like material.

The author, in his comments on the case, is inclined [and in this view we agree with him] to regard the blindness and deafness as primarily dependent on the destruction of the visual and auditory centres which are specially implicated, and the atrophy of the optic nerves to be secondary. An ophthalmoscopic examination during life could not be made, on account of the patient's restlessness and screaming when interfered with. The passing motor disturbances, specially of the right arm, are explicable by some degree of implication of the ascending parietal convolution.

Blindness and Localised Cerebral Atrophy.—Mickle (*Med. Times and Gazette*, Jan. 28, 1882) reports the condition of the brain in a case of blindness which had existed over twenty years. Some months before death the patient had become maniacal, and death occurred with symptoms of septicæmia.

The cerebral vessels were more or less atheromatous.

Atrophy was found in the optic nerves and tracts, the upper part of the supramarginal gyri on both sides and to some extent of the angular gyri. In the first and second occipital convolutions there were a few patches of red softening, with dilatation and infarction of the smaller vessels.

There was slight wasting of the left corpus striatum, both of the thalami, and the corpora geniculata of the right side.

Cerebral Tumour and Unilateral Deafness.—Strümpell (*Neurologisches Centlbt.*, Aug. 15, 1882) records a case of tumour of the right hemisphere, in which along with the general symptoms of cerebral tumour, and left hemiplegia with some degree of anæsthesia, deafness developed in the left ear, with a total inability to localise the origin of sounds.

After death a tumour was found of the size of an apple, situated in the middle of the ascending parietal convolution. Round the tumour the cerebral tissue was in a state of white softening, which extended forwards as far as the fissure of Rolando, backwards throughout the parietal lobe, and beneath almost into the central ganglia.

He is of opinion that the deafness was probably due to lesion of the parietal lobe; and that the localisation of sounds in space is a function of binauricular hearing.

Word-Deafness and Blindness.—D'Heilly and Chantemesse (*Gaz. des Hôpitaux*, Aug. 5, 1882), demonstrated before the Société Médicale des Hôpitaux on July 28, 1882, the brain of a young woman who had suddenly become aphasic, and who died of tuberculosis. She was unable to comprehend what was said. She could neither read nor write, and could utter only incoherent words. Sight, hearing, mobility and general sensibility were unaffected.

A thrombus was found in the fourth branch of the left Sylvian artery, causing softening of the upper margin of the superior temporo-sphenoidal convolution in its posterior half, extending into the lower half of the inferior parietal lobule, the lobule of the pli courbe, and the anterior half of the pli courbe or angular gyrus.

Luys (*L'Encéphale*, No. 4, p. 647) describes a case of deafness of long standing, in which, after death, atrophy was observed in the convolutions in the region of the *cuneus*. The paper is illustrated by figures of both hemispheres.

Without concluding from a case of this kind as to the exact position of the auditory centres, he thinks a causal relationship between the central distribution of the auditory nerves and the particular atrophy is indicated, and he refers to a similar case reported by him in 1876, in which there was atrophy in the same regions.

Regional Diagnosis and Trephining.—Wernicke and Hahn (*Virchow's Archiv*, Feb. 1882, p. 335) report a case of tubercular abscess of the left occipital region diagnosed from the symptoms, which were right hemiopia, proceeding to motor and sensory paralysis of the limbs on the right side. The skull was trephined (under antiseptic precautions) at the upper posterior angle of the left parietal bone, the dura and cortex incised and an abscess evacuated. The abscess was about the size of a hen's egg, and about three teaspoonfuls of pus was removed from it. The symptoms of paralysis and the general condition were at first greatly improved, but again returned before death, which took place a fortnight after the operation. The post-mortem examination showed a tubercular abscess in the left parietal and occipital region which had recently opened into the lateral ventricle. There were a few smaller softened tubercles in the neighbourhood of the abscess. There were a few scattered patches of chronic tubercular pneumonia. The unsuccessful result was attributed to the unusual character of the abscess, and the absence of any capsule preventing the further extension and perforation into the ventricle.

D. Ferrier.

BRAIN.

JANUARY, 1883.

Original Articles.

SUGGESTIONS AS TO THE ÆTIOLOGY OF SOME OF THE SO-CALLED SYSTEM-DISEASES OF THE SPINAL CORD.

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Among the most salient facts in clinical medicine and pathology that recent investigation has brought to light are certainly those relating to the differential diagnosis of some diseases of the spinal cord. There is no doubt that clinical study, stimulated and enlightened by anatomical research, has enabled us to discriminate affections which really can lay claim to individuality, and are found to be associated with a definite lesion when examined *post mortem*. Till quite lately, as is well known, many of these affections were classed together under the general term of paraplegia, or myelitis. Though doubtless existing for an indefinite time they have remained unnoticed in a confused mass of morbid symptoms, till, like the statue set free by the sculptor from the marble block, they have come into being through the labour of the clinician and the pathologist. Much more can now be learnt than heretofore of the life-history of this class of affections, and an improved prognosis ought necessarily to follow in the wake of this diagnostic advance.

Yet as we contemplate the progress we have made, we cannot but be struck with our abiding ignorance concerning the origin of these diseases, not to mention the absence of any line of treatment, which, of course, to be rational, must largely depend on ætiology. On every ground this question is of commanding interest to the pathologist and the physician. The post-mortem study of such cases of spinal cord disease as end in death, and can be thoroughly examined, has at present given us but little direct help towards tracing their causation from the beginning. When we thus examine the body the mischief is already wrought; destructive lesions at most are demonstrated to us; though we may be led by a kind of natural inference to suppose some change in the cord, antecedent to the symptoms and to the structural lesion before our eyes.

But in face of these facts, and though the morbid anatomist has made no general claim to teach us anything regarding the ætiology of the diseases to which we refer, it would seem that many are led to regard too exclusively the lesion found in the cord as the primary source of the malady in question, and so incidentally to limit the field of inquiry as to how these special diseases may be brought about. But little attention is paid to the possible ætiological antecedence of peripheral disturbance, or what may be called abnormal function, which, by its continuance, may set up the ultimate change in the structure of the cord, entailing all the morbid symptoms of the established disease.

In speaking of the possibility of functional disorder producing structural change, it must be acknowledged that at first sight the natural order of things is apparently reversed, and the behests of a truly materialistic pathology seem to be too lightly regarded. But it must be remembered that the word "functional," when applied to disease, does not in any sense exclude the notion of a material cause. We mean by the term such disorder as is not preceded by any structural change detectable by our present means of research, and further, because in many cases such disorder tends to recovery, in all probability not often followed by any permanent alteration. In such a case the word "functional," though theoretically pro-

visional in character, has a practical value, and denominates a real class of affections. Such diseases as chorea and the many disorders of function shown by the various forms of hysteria are instances in point. There are, as Dr. Wilks has said, but few forms of organic nervous disease (i.e. nervous disease demonstrably associated with structural change) that do not find their copy among the hysterical. Does not this close correspondence of so-called hysterical or functional disease with disease that is called "organic"—a correspondence sometimes so close that almost the only differentia seems to be that the one tends to recovery, the other to permanent disablement or death—give us at least a hint of some possible common element at work which may underlie in some cases the beginnings of both affections? In other words, may we not conceive of the more central nervous structures suffering from a continuance of undue excitation from the periphery, so that what at first may have been, for instance, a temporary derangement of the circulation, may come to be a permanent alteration in structure due to chronic congestion or inflammation?

In applying this view of a functional origin to some of the system-diseases of the cord, we must bear in mind that it can only be regarded as a suggestion, to be used or not as it is found to be fruitful, and not contradictory to established facts. Even if it be looked upon as a "*vera causa*," it could with difficulty be demonstrated, the morbid procedure being ex hypothesi in progress during life, and causing thereby an impossibility of tracing continuously the working of the primary peripheral disturbance towards the ultimate structural change. But it may be of some use to consider the subject for a while from this standpoint, and to inquire whether this view derives any support from analogical instances, or falls in with established facts or received theories regarding the physiology of the nervous system.

The study of the working of the nervous system, both in health and disease, is confused and hampered at the outset by an enormous difficulty. We have to deal with the functional aspect of the nervous system; with nervous action, or, as we often term it, nervous force—a force of whose nature

we are ignorant, uncorrelated as it is at present, but which nevertheless we must recognise and provisionally describe. The functional activity of nervous structures is therefore quite a different thing to discuss from that of other organs. Dilate as we may upon the action of the nervous system, whether normal or abnormal, we can as yet do but little towards explaining the phenomena we are grappling with. The words we use, such as "nervous force" and, more notably, "discharge" —of late so frequently occurring in writings on nerve-pathology —are at best but analogical and indefinite in character, in no degree explanatory in the true sense of tending towards simplification, or of expression in terms of the known. We are indeed almost obliged to call in the aid of non-physical terms when speaking of some nervous phenomena, so intimately are they blended with factors which, for the sake of clearness, must be called mental. The unknown element in this field of study is too great for us to institute as yet a scientifically consistent nomenclature. We cannot intelligibly express in definite terms the simplest voluntary movements of our muscles, for we know not the nature of the functional relation subsisting between the brain-cells and the periphery; and we can hardly do more than attempt to give a metaphorical account of the higher workings of the cerebrum, or of the well-known processes whereby a so-called mental impression, though involuntarily arising, is followed by an obvious physical result. We are, in fact, constantly confronted in our study of nervous phenomena with the deepest problems of life, the relation of brain and mind, and we must, in our ignorance, of necessity confuse (albeit with conscious acknowledgment) the terms of psychology and physiology. This unexpressed vital factor is an ever-present "crux" in our endeavours at explanation, and a defiant enemy of post-mortem discovery.

It is necessary to bear in mind this difficulty and source of inaccuracy in our terminology, when listening to any ætiological speculation in nerve-disease. If physiology gives an uncertain sound, pathology may be expected to do the same, or, at least, must not be forced, if it be heard at all, into narrower and stricter limits of diction than those granted to physiology. It may be legitimate and even useful to assume

an undemonstrated pathological process as a tentative explanation of any given phenomena not otherwise understood, provided such a process be not contradicted by accredited knowledge, and be in nowise deemed to be established just because, if assumed, it appears to be adequate to the production of its alleged result. This justification there seems to be for inquiring as to how far a functional origin might serve to account for certain diseases of the cord (as, for instance, locomotor ataxy or spastic paraplegia), without contravening undoubted facts. For there is an especial difficulty while considering these diseases from the standpoint of the *structural* lesions observed, in the view of the *primary* occurrence of anatomical change in certain *functional* or *physiological* paths.

The pathological aspect of these affections to be discussed, while appearing at first sight out of harmony with the ordinary method of study, seems more reasonable when the complexity and peculiarity of the nervous system is borne in mind. The arrangements and mutual relation of the elements of the nervous system, the elaborate structure of the nervous centres, as well as the question of the action and reaction of the individual and the environment, in the development of the higher centres and functions, may all, to some extent, bear on the pathological study of nerve-disease. Perhaps we are all too apt, especially in medical practice, to regard the human individual in his aspect of independence rather than as an integral part of the universe, and to forget that the manifestations of both normal and abnormal action in man may be partially explained by reference to how and whence he came, and to the influence on his organism, both through heredity and present action, of impulses from the external world.

The current manner of speaking of nervous phenomena tends to present a one-sided, or rather, probably, an incorrect view of the mutual relations in structure and function of the several parts of the nervous system. When we speak of the nervous centres, meaning more especially those in the brain and cord, we are apt to regard them in our minds as in every sense antecedent to and causative of, both retrospectively and actually, the rest of the nervous system and its phenomena. We use, for instance, the term "nerve-force" (though more as an exigency of nomenclature than as conveying a very definite

idea), and almost irresistibly regard the nervous centres as the "fons et origo" of this force. We tend to represent the completely developed nervous system, as we see it, as antecedent to the display of nervous action. Generally speaking, in relation to the individual, it is sufficient for all practical purposes to describe the nexus between structure and function in the ordinary manner, and to regard all function or activity as dependent for its existence and character on the structure of its proper organ, without regard to the evolutional history of the organ itself. For this mode of regarding it is, as a rule, scientifically accurate *as regards the individual*. We may thus rightly infer a primary alteration of structure in various organs of the body from an ascertained abnormality of their function, and we find that definite lesions are constantly followed by definite sets of symptoms. Ample instances of this are afforded us by the study of visceral pathology. But when we come to the nervous system, the matter and manner of investigation are not so simple. Instead of a comparative homogeneity and simplicity of structure and function, we find the greatest complexity and diversity. The differentiation of structure is here so great, and the nature of the functions so various and, as it were, unique, their study deriving so little proportionate help from direct experiment, that we must carefully consider the subject in all possible aspects before using a terminology and method of description which cannot be without its influence on future work, and may possibly obscure, instead of tending to advance, the true understanding of the problem.

It is the developmental aspect, then, that we cannot afford to ignore in relation to the physiological and pathological working of the nervous system. For here especially does there seem to be some indication of certain practical bearings on the individual of developmental influences, or, in other words, a different ratio of value to obtain between the hereditary and acquired elements of the organism. Though the hereditary factor may still be quantitatively preponderant, the acquired factor asserts itself visibly, and is obviously of great and characteristic importance.[1]

[1] This is clearly seen of course in those so-called psychical functions which are more especially characteristic of the human species. The notable modification of an individual by circumstances, or the irresistible effect of what Spencer calls the

But little is known by direct observation of the details of the development of the nervous system in man. Certain main points, however, regarding the evolution of nervous structures generally should be borne in mind. And chiefly does it concern us here to remember that "embryological evidence shows that the ganglion cells of the central part of the nervous system are originally derived from the simple indifferentiated epithelial cells of the surface of the body; while the central nervous system itself has arisen from the concentration of such cells in special tracts."[1] Complexity of the nervous system is a characteristic of later evolution in the animal series, and some of the structural arrangements of the nervous centres existing in the higher animals are not found in the embryo, or even soon after birth, but increase in a demonstrable manner during the life of the individual. Apart from the probable fact that the sharp distinction between the white and grey matter is a feature acquired in the higher vertebrata, we know that the grey matter of the cerebrum does not exist in the human embryo, and that its thickness continues to increase through a long period of years. There is then an increase of structural complexity during the period of active cerebral life. Remembering, then, the admitted influence of external stimuli on the elaboration of the nervous apparatus, and its probable effect on the individual, we cannot but think that some different consequences from what we see in the case of other organs may be entailed by this view on nervous pathology. The lung of the new-born infant is essentially similar to that of the adult; and preparations of its liver or its kidney present no structural difference from those of the man. But when we consider that some of the most pre-eminent functions of the human individual—the functions of the nervous system which

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super-organic environment, is a striking example. In illustration of this it may be remembered, as quoted in another context by Mr. Henry George in his recent work on 'Progress and Poverty,' that the special form of articulate speech with which we express our ideas (i.e. our national language) is entirely the result of impressions made on our individual brains, though the hereditary capacity for articulate speech is one of the most important *differentiæ* of our species. A child of any country could learn with equal ease the language of all, if placed in similar native surroundings.

[1] See Balfour's 'Comparative Embryology,' vol. ii. ch. xv.

tends so largely to the differentiation and predominance of man in the animal series—are correlated with structural arrangements which are not complete at the beginning of life, it appears most probable not only that modifications and interferences with the developing organism may take place, but also that their significance, occurring as they do in such important material, will be proportionately great. If nervous structure becomes organised abnormally, we may then expect in turn a more or less permanent aberration of function as a result. The structure of the nervous centres, in fact, is to be contrasted, rather than compared, with that of other organs. The fact, again, of the least stable part of the organism, or that part which is most lately developed, being the first to undergo dissolution or degeneration, is exemplified in the study of the nervous system both in health and disease. The grey matter of the brain in old age tends to waste, and its functions to approximate to those of early life; and the tremors of senility are comparable to symptoms which are referable, before the period of natural decay, to cerebral degeneration.

Similar influences, then, to those which in the normal course of things tend towards the development and complexity of the nervous centres, such as an increasing variety of impulses and stimuli from without, or the multiform contact of the environment with the organism, acting through the peripheric nerves, may in altered conditions produce pathological results. The normal complex nervous centres may be best regarded not as primary originators of "nerve force" and antecedent to its display, but rather as automatic registers of those actions which, at first vaguely exerted, were by frequent repetition conveyed along the "lines of least resistance," and have at last come to be organically marked in definite structure. From this point of view we may roughly liken the nervous centres rather to a calculating machine which, with great complexity of structure, can automatically produce elaborate results, though set in action for every process at a given moment by the simplest means, than, as is so commonly done, to the electric battery of the telegraph system, which is the source of telegraphic phenomena. The calculating machine represents, or, it may be metaphorically said, contains stored up in

its structure the brain-work of the inventor. So the nerve centres are the permanent and complex registers of often-repeated peripheric impressions from the external world, and we may conveniently regard the whole nervous system of man as a reflex apparatus derived from and dependent upon sensory impulses.

It is easy to understand, when these nervous paths are once formed, and closely associated by degrees with their increasingly elaborated central organs, that nervous action may proceed along these paths alone, and that the activity of the central cells in the matured organism is involved in every expression of so-called nervous force. It is as if a central government devised and brought into existence by a community had received in complete surrender, for the purpose of the work being carried on with the greatest efficiency and the least friction, the entire control of the action of that community. Such a government, though really the consolidated expression of many individual wills, would appear at first sight to be the mainspring of the action of all those who had so completely given themselves up to its authority.

On the pathological side, then, this method of presenting the genesis of the nervous centres has its application. As certain peripheric stimuli have gradually contributed in the healthy body (regarding both the hereditary and acquired elements) to a normal organisation of the nervous system, so certain stimuli, not so favourable in their nature, and acting moreover perhaps on an organism with hereditarily weak points, or "unstable" in character, may result in the production of a badly organised nervous structure. And when the nerve-centres become thus mis-organised, their indissolubly associated functions must be abnormal too. Thus, for instance, while we rightly attribute the morbid symptoms which we call a disease of the spinal cord to the structural change observed therein, as proximately causing the symptoms, we may consistently regard the structural derangement of the cord as resulting from primary untoward action from the periphery, preventing or marring the normal course of development. And we may compare the diseased cord which spoils the working of its associated nerves to the calculating machine which is out of

order, and whose maker is not to be found. The complicated nerve-structure, once formed, is absolutely necessary to all action; the contributory constituents have long ago surrendered their initiatory powers. But by a reference to the maker of the machine the cause, at least, of its disorder may be manifested; and thus may the consideration of the development of the nervous centres tend possibly to throw some light on the processes of their derangement.

It is obvious that the more stable or earlier-organised parts of the nervous system, being hereditarily comparatively perfect, may (relatively to the individual) be regarded, both in physiology and pathology, as antecedent to functional action. But the more unstable parts irresistibly remind us of how and whence they came, and to duly understand some aberrations from the normal in the case of nervous disease, we should perhaps have this ever before our eyes, regarding some parts of the nervous centres as dependent for their characters on functional actions in the past, and capable of being modified by its continued influence. For this view is probably true of the whole nervous system when taken in relation with the biological series.

It does not concern us here to trace the first appearance of a nervous system in the animal world, any more than it does to know what order is taken by the nervous link in the vital chain of each individual. But given in man a highly differentiated nervous system, it is impossible but that he will suffer materially from its derangement in any degree. The complexity of its arrangement and the importance of its manifold functions are sufficient evidence of this. By it man is brought into relation with the external world. It is at once the highest in function, and the most liable to derangement of all the systems. Nature's "last work which seems so fair" is open to the evil influences of what seem to be ordinary causes. The highest functions of all may be irreparably damaged by influences which are traceable by inference or conjecture alone, and which reveal no morbid stamp when their associated structures are examined in the light of our present knowledge. But we are none the less sure that, though it eludes our search, some "havoc in the tender cells" of the

brain has yet been wrought. And coming to subordinate departments of the nervous system, less subtly constituted than those whose structure is involved in the manifestations of insanity, we should not be surprised to meet with cases of disease in which some peripheral disturbance, owing to interference with the nervous paths, had caused an alteration, either temporary or permanent, of the central nervous structures—an alteration sometimes to be detected and demonstrated by comparison with the normal structure of healthy development.

Again; as in the individual the most recently differentiated and most complex structures are the most likely to suffer from undue stimulation, so too we might expect to find certain cases where the organism had a special liability to such derangement, or an hereditary tendency to fail on the side of the nervous system, even on the provocation of such slightly undue stimuli as might be difficult to detect or impossible to demonstrate. We may have to deal with an inherited "nervous constitution," the vagueness of which much-used and much-abused term may be to some extent diminished by the present point of view. That a certain so-called "neurotic" tendency is found in certain families is admitted by all clinical observers, and is in accordance with what we think we know of the physiology of the nervous system. There is a further point, again, which these considerations illustrate. Although the neurotic tendency is often inherited, it is not a matter of common observation that this is the case with special neuroses or definite "nervous" diseases. We may have many different morbid expressions of nervous action occurring in the same family, such as epilepsy, or chorea, or perhaps various forms of paralysis. And hysteria in its multiform aspect (though no more multiform than might well be expected if we duly regard it as want of proper systemic nervous control) is often a notable mark of many closely-related persons; but the form which the hysterical display shall take is largely determined by the physical and psychical surroundings of each individual case, or, in other words, by the nature of the untoward stimulus.

It is of course obvious that the application of this point of view to the ætiology of any of the so-called "system diseases"

of the cord is beyond the region of demonstration. We are at once confronted with the difficulty or impossibility of showing how such post-mortem lesions as are observed in the cord can have resulted in the manner suggested; and with the further obstacle, that in many cases we cannot prove the existence of any obviously undue functional action at all. The hypothesis must be regarded as merely tentative, perhaps to be completely discredited by future knowledge, and must be granted the full measure, due to all hypotheses, of *à priori* assumption. Thus, in the case of any given specialised disease of the cord, although in some instances obvious functional disturbance, whether in a presumably predisposed individual or not, may irresistibly direct our attention to its probable ætiological importance, yet in many others we can demonstrate no probable exciting cause. Then we may call to mind the inference from other considerations which teaches us the presumable existence of hereditarily unstable nervous structure, and the probable fact of this instability being transmitted more as a tendency than in any specialised form or expression. The patient whose case puzzles us may have been the inheritor of a nervous system thus characterised, and hence requires a much slighter exciting cause (though that cause, be it remembered, must be of a special nature) to bring about an ultimate central change similar to that which, with an obviously great exciting cause, may be presumably found in a better constituted individual. It is because the so-called system diseases, especially those connected with changes in the white columns of the cord called respectively *posterior* and *lateral sclerosis*, or "locomotor ataxy" and "spastic paraplegia," seem so difficult to explain ætiologically from direct observation, and yet from their peculiar course are so interesting and important from this point of view, that some hypothetical discussion of their cause seems excusable. As careful anatomical examination has progressed, so has it come to light that in certain diseases, clinically well marked, there are constant demonstrable post-mortem changes, and, moreover, that these changes take a *physiological* direction, or follow a tract of *special function*. It is the question why this change should start into being in a definite functional region that arrests the attention and de-

mands inquiry; the progress of the change once so started, mainly along the same tract, presenting no great difficulty.

The disease clinically known as *locomotor ataxy* is characterised by the well-known sclerotic change in the posterior root-zones of the cord on both sides; and more recently it has been shown with great probability that a definite set of symptoms, now best characterised by the name of *spastic paraplegia*, but commonly called "lateral sclerosis," is associated with a similar morbid process, symmetrically affecting a part of the lateral columns of the cord known as the crossed pyramidal tract. The anatomical recognition of the latter disease does not rest upon a large number of facts; but the clinical picture is very complete. The post-mortem change cannot be often seen in the unmixed cases, as they run a long and indefinite course; but there is at least one case on record[1] which has almost all the value of an "experimentum crucis."

It is this physiological or functional direction taken by the morbid process that suggests some explanation other than the haphazard occurrence there of a primary inflammatory or degenerative change. The peculiarity of the localisation of the mischief would meet with some explanation if it could be admitted as possible that some morbid influence, or irritation, —call it by what name, or express it how we will—working molecularly, or otherwise (for in our ignorance it matters but little what metaphorical term we use), can be transmitted from the periphery to the centres, and bring about the ultimate changes which are now recognised. We could then conceive with some consistency that an over-excitation of motor or sensory functions of certain parts might account for observed changes in the tract or centres in immediate connection therewith. In such a tract or centre the motor apparatus is gathered, as it were, into a focus, and might represent in its morbid state the result of untoward stimulation from the periphery, just as in its healthy state it is the result of a series of normally conveyed impulses.

Have we, then, any justification in fact or analogy for the

[1] A case brought by Dr. Dreschfeld, of Manchester, before the Pathological Section of the recent International Medical Congress. See 'Transactions of Congress,' vol. i. p. 407.

hypothesis of a functional origin for these definitely marked diseases? Considering the peculiarities which specially mark the nervous system, it cannot be expected that we shall find any closely-related facts in illustration of this hypothesis, which for its chance of life must mainly rest its defence on the absence of facts in positive opposition to it; but the following considerations appear to have some relevancy.

The clinical study of nervous diseases suggests several examples of more or less permanent disorder, which may find at least a not unlikely origin in functional disturbance. Leaving out of the question for the moment the existence or not of structural change demonstrable *post mortem*, we can call to mind in this context the innumerable examples of confessedly physical states, brought about and modified by influences acting on some part of the nervous system; these results being tangible and demonstrable, though the so-called central organ whose action takes part in the phenomenon must certainly be looked upon as being affected in a very different way from what we are in the habit of calling "structural" change. The varied field of the so-called action of the mind on the body is an illustration of this — where often a stimulus to some of the peripheral nerve organs, applied either directly or indirectly through the medium of memory of analogical states in the past, or *mental action*, causes an altered physical state easily recognisable by the observer. Thus a word, or a thought, will cause a blush; the blush being proximately dependent on disturbed vaso-motor mechanism. The same condition may be brought about by the purely physical experiment of dividing a nerve. It is not stretching a point too far to surmise that a constantly repeated stimulus of the first order, could it be attained, would bring about a state of more or less permanent congestion of capillaries.

The influence of various sights, smells, and even thoughts, on appetite and digestion is well known. Undoubtedly, a change in the physical state of the organs concerned causes the nausea and indigestion that are so well known, in certain kinds of temperaments, to result from the untoward stimuli alluded to. Atrophy of organs, of course, is obviously due in many cases to their functional disuse; while hypertrophy is a

consequence of their over-use. Hypertrophy of the heart is known to result from an undue strain upon this organ, dilatation being probably the first stage in the process. But as a further result we often find fatty degeneration, accompanied by an increase of the connective tissue. Here we have an example of structural change occurring in ascertained lineal descent from functional over-work. In some cases, as in pregnancy, the cause of hypertrophy of the heart being not continuous, or, in other words, the functional demand being intermittent, the structural change is not permanent, and does not go on to degeneration; the whole process here may be called physiological. But in most other cases the cause is more continuous, and the morbid result is permanent. Thus we see how narrow a line may divide a physiological phenomenon, demonstrable by even structural change, from a pathological process which we call disease.

To come now to what is more proper to the subject in hand—the diseases referable to the changes in the spinal cord. The most common and best-described example of the system-diseases of the cord is the malady known as locomotor ataxy, or tabes dorsalis, which, by ample post-mortem observation, has been connected with sclerosis of the postero-external columns of the cord. As the observation of this affection has been made with increasing attention, so the manifold nature of its symptoms has become more and more obvious. The symptom of ataxy which, when the disease was first described, occupied such a prominent position, is now rivalled or outrivalled in importance by a host of sensory and even of cerebral disturbances which must necessarily be taken into account when considering the probable origin of the affection. Some authors, as is well known, regard the disease as essentially one of the sensory system, including the symptom of inco-ordination. Doubtless in most cases sensory symptoms of a pronounced character mark its onset. The prominence of the sensory symptoms in locomotor ataxy has lately been especially insisted on by MM. Pierret and Rougier of Lyons, whose writings on this subject are very suggestive.

Various exciting causes of this disease have, from time to time, been put forward. Before the post-mortem condition of

the cord had been so carefully studied as it now is, a theory of the functional origin of tabes enjoyed a much larger share of attention than at present. It was a wide-spread opinion that excessive sexual intercourse was its frequent or general antecedent. This opinion, not traceable to any theoretical bias, but presumably to some extent founded on a basis of observation, has almost disappeared from the schools, owing to the proved connection of the symptoms with structural disease of the cord. This definite sclerotic change, whether primarily inflammatory or not, and wherever the exact histological seat of its origin may be, now alone arrests the attention; and every known cause of such a change is called into requisition to explain it. It is not here my intention to discuss this matter in detail; but this much may fairly be said, that no reasonable theory of the origin of this definite and localised change in the cord has as yet been started. Syphilis, that inexhaustible cruse of ætiological oil, may be regarded as having been weighed in the balance of causation and found wanting. Recourse was doubtless had to it, with that natural longing for finality and logical rest which characterises the minds of most of us; but both on account of the wide prevalence of syphilis, and the known habit of its morbid deposits, *which do not make choice of functional tracts*, its supposed causative *rôle* in locomotor ataxy cannot be at all satisfactory to the serious inquirer. The suggestion, in order to remove a difficulty from the syphilitic theory, that the morbid process may have been first diffused over the cord, and afterwards become localised in the posterior columns, is somewhat gratuitous, and certainly quite out of accord with the clinical history of most cases of ataxy.

But though the origin of this disease is confessedly obscure, there would appear to be some points of probability in the older notion that it may lie in what is to the affected individual relatively excessive sexual excitement. The far greater frequency of tabes in men than in women agrees with this view; and a very strong impression prevails both among practical medical men and others that its causation is of this nature.

I am unable to give adequate details in support of this

position, having been in the habit, in common probably with many others, of to a great extent ignoring this point in cases which have from time to time come before me, owing to the prevailing influence of modern teaching. But as I deprecate these remarks being regarded as claiming any demonstrative value, or as being anything else than barely suggestive, and as I am not attempting here to discuss fully the pathology of tabes, this omission is less inexcusable than it may seem. Long before I was led to theorise at all on the subject, I had difficulty in rejecting from my mind the probable influence of sexual intercourse in the production of this malady. Some cases that I have in my memory strongly impressed me at the time of seeing them; the patients themselves, without previous knowledge of the name or nature of their disease, being confident of its cause, and stating their opinion unasked. These patients' accounts of their excesses were not equivocal; one man especially, with a previously clean bill of health, giving a history where, during two or three years, increasing indulgence had been accompanied by weakness and darting pains in the legs, these symptoms having been relieved by occasional short periods of abstinence. But a typical and rapidly progressive case of ataxy soon developed itself as a sequence, if not as a result, of what all would admit to be a course of extraordinary venereal excitement.

A case is also within my knowledge of a young man of unrestrained desires, who says that after frequent and excessive sexual intercourse he often suffers from symptoms which appear to be temporary attacks of inco-ordination. A commoner complaint, however, after undue indulgence, is that of neuralgic pains in the extremities, more especially in the legs.

It may be said here that Romberg, one of the earliest students of this disease, taught that loss of semen, when combined with that hyper-stimulation of the nerves to which sexual abuses give rise (i. e. to the exclusion of mere spermatorrhœa), not unfrequently favours the origin and encourages the development of tabes dorsalis.[1]

Now if this malady be regarded as one especially affecting

[1] See Dr. Sieveking's Translation (Sydenham Society) of 'Romberg on Nervous Diseases,' s. v.

the sensory system, may not some little light be thrown upon it by supposing the first step in the morbid process to be due to abnormal stimulation of sensory organs; and, assuming for the sake of argument the frequency of relatively excessive sexual intercourse as a forerunner of tabes, may we not reasonably consider the intense and widely diffused sensations accompanying the sexual act as an adequate source of disturbance of an hereditarily unstable, or in some cases even of an approximately normal sensory apparatus, provided that it be admitted that structural alterations in the cord may result from prolonged peripheral irritation? And from this point of view the greater frequency of tabes than of a system-disease of the *motor* apparatus proper derives some little explanation. The possible cause that will be presently suggested for the disease called spastic paraplegia, or lateral sclerosis, viz. over-work of the muscular system, is one that might be expected on this hypothesis to be less frequently active. For the natural demands for action on the motor apparatus are more constant than those on the sexual organs, and in a normally constituted cord we should look for a greater margin of functional activity to be allowed to the motor tracts without risk of over-stimulation taking place.

It may at least be said that this view of the possibility of a peripheral origin of the disease known as tabes seems to harmonise to a considerable extent with all the characteristic clinical facts. I am fully aware that the opinion of many experienced, and deservedly high, authorities on diseases of the nervous system is opposed to any close connection between tabes and the hypothetical cause under discussion. And were I defending a thesis, instead of inquiring, I should not venture to criticise existing views without bringing at least an equal amount of experience and labour towards the solution of this question. But it may be pointed out that just as in chorea, as ably argued by Dr. Sturges in his recent works, the apparently ordinary emotional causes which may produce it are necessarily often overlooked, so, in this instance, what is really relative excess of sexual indulgence might well be passed unnoticed, quite apart from the number of cases where it might be suspected, but purposely denied Of course if this sequence

of events could be satisfactorily substantiated, the question of the causation of the disease would not be set at rest entirely. But on the principle, "Causæ non sunt multiplicandæ præter necessitatem," this hypothesis, serving to cover a large number of cases, would deserve at least a front rank in the investigation of the matter.

A few words may be said here on the arguments used by those who, partly from certain clinical data, partly from theoretical reasoning, deny the sensory factor in the production of inco-ordination, and whose position would, by implication, be opposed to the probability of the origin of the entire disease in disturbance of the sensory apparatus by peripheral irritation. It would seem that the use of the term co-ordination often causes and implies considerable mental confusion. Because a certain tract of the cord is found to be diseased in cases characterised by a want of co-ordination, the minds of many are satisfied with the conclusion that this tract is in some special way the seat or the conductor of "co-ordinating impulses." Now apart from the distraction which has been caused by the pathological study of locomotor ataxy, the question of a special seat or tract for co-ordination in the cord would never have struck the philosophic neurologist. Physiology tells him nothing of it; but, rather than suggest a search for it, would lead him to regard co-ordination as only a functional aspect of the nervous system. Co-ordination simply means the harmonious working of the nervous system as expressed in muscular action, whether considered as a whole or in its parts; and is implied in the very notion of a nervous *system* at all. That this harmonious working is dependent on the proper conduction of afferent and efferent impulses in connection with organised centres, or in other words implies a certain relation between the sensory and motor apparatus of the nervous system, would appear from a physiological point of view fairly obvious; and an impairment of co-ordination, or want of harmonious action in any part, would be referred to derangement of part of the apparatus involved, causing a want of proper conduction of either sensory or motor impulses. A special tract for the conduction or localisation of "co-ordinating" impulses is as little wanted in

pathology as it is hinted at by physiology. But those who argue against the influence of the afferent factor on the production of the *ataxia* of tabes, regarding it purely as a motor morbid symptom, seem at least unconsciously prepossessed with the idea that some special seat must exist for what they appear to treat as the co-ordinating "function," omitting in thought to analyse it into its simpler elements. Forgetting moreover, apparently, the fact that such a localisation has been suggested only by the observed connection between *ataxia* and disease of the posterior columns, and bearing in mind that physiology has as yet given only sensory functions to those parts of the cord, some have endeavoured to find in the lateral column the true seat of what they regard as co-ordination, believing it *must* have a seat somewhere; so that, on this showing, the well-known and accurately defined lesion in the posterior root-zones of the cord does not, after all, account for one of the most important symptoms of the disease in question.

The clinical evidence called in to support this view is based on the quotation of cases where inco-ordination is observed without loss of sensation; or where anæsthesia takes place without ataxia. Quite apart from the fact that the cases are, to say the least of it, very rare and questionable where some degree of anæsthesia does not obtain in connection with ataxia, the confessedly difficult subject of the due investigation and valuation of the symptom of anæsthesia should prevent us drawing any very important conclusion from the little we know of the matter. We learn but very little from direct physiological experiment on sensations, and especially of sensory conduction in the cord. The subject is beset with obvious difficulties; and though investigators speak with almost one voice regarding the motor functions, there is palpable discord among them respecting sensory phenomena. But when we remember (to quote from Foster's work on Physiology) "that an afferent impulse passing along an afferent nerve may in certain cases, simply produce a change in our consciousness, unaccompanied by any visible movements, and in other cases may give rise to reflex movements, or modify existing reflex or automatic action without causing any change

in our consciousness," we shall at least see that it is premature to deny the afferent or sensory factor in the production of inco-ordination, on the strength of such cases as we have alluded to. This question is argued at some length by Professor Erb, in his article on "Tabes," in Ziemssen's 'Cyclopædia,' where I think, in his advocacy of the exclusively *motor* nature of the ataxic symptoms, there is betrayed the confusion I have referred to concerning the *word* co-ordination, and this partly gives a bias to his arguments and quotation of cases against the possible part played by the sensory side of the nervous system in the production of inco-ordination. The consideration of this question illustrates the danger there is in attempting to make physiology too soon—to build up a physiological theory on pathological facts or clinical observations alone. The lines of physiological and clinical investigation, though often giving mutual help, must be kept distinct; the inferences of the latter being never looked upon as the dicta of the former. In the case we have been glancing at we have scientific warrant in maintaining only the *concomitance* of certain symptoms with a certain observed change. This fact is established. But we may not thereupon invent a function to plausibly occupy an insufficiently explained structural locality. This fallacy and confusion, although confessedly not put forward as a theory, or baldly stated by any writer, nevertheless seems to lurk deeply in many discussions on the subject.

To take now another example of the system-diseases of the cord, which may serve on the motor side as a counterpart to tabes on the sensory side. The disease now known as *primary lateral sclerosis*, or *spastic paraplegia*, must be regarded, owing to the admirable work of Professor Erb, as a clinical entity; and, though in all probability less common than tabes, is being recognised with increasing frequency. It is true that the evidence of the associated cord-change rests upon very few observations; but taking into consideration what has been indubitably made out, and what is known, moreover, regarding other forms of sclerosis in the lateral columns, in conjunction with the concomitant symptoms observed—this disease must challenge the attention of those interested in the ætiology of

the system-diseases of the cord. It is believed that certain tracts symmetrically placed in the lateral columns of the cord are especially occupied in the conduction of motor impulses towards the periphery; the normal conduction of these impulses implying, too, the control, or exhibition, of reflex action. Difficulty of motion, stiffness of the legs, and an exaggeration of the reflexes, are the prominent clinical symptoms connected with this form of sclerosis. Is there any clinical justification for supposing that the cord-change existing here may be secondary to undue stimulation of the motor apparatus: granting, for the moment, the possibility of a pathological effect being wrought on the concentrated nerve masses of the motor tract in the cord by undue irritation of the motor nerves of the periphery? Certain points, both negative and positive, seem to favour such a supposition, though they may be not sufficiently obvious to claim recognition. There is no theory, even vaguely accepted, as to the ætiology of this affection, definite as it seems to be. The syphilitic explanation has not been seriously broached, and by most authors is rejected, to fall back on "cold," possible injuries, and such-like general hypotheses—so general, as almost to defy either proof or disproof. One positive point, however, is noticed by those who have written on this disease: it occurs comparatively frequently in men—and in muscular men. It is perhaps in this class that we may expect to find the material for a disease hypothetically producible by excessive demands made on a possibly unstable nervous apparatus: for here it is more likely that muscular overwork might take place, and the possessor of the strongest muscles is not necessarily the subject always of the most perfect nervous control. It must be remembered, too, that this disease, anatomically characterised by sclerosis, most marked in the lower part of the cord, begins generally in the lower extremities, which are more liable to continuous overwork than other parts of the body. In some cases of this disease, moreover, excessive action of the muscles can be clearly established. One case especially,[1] which has lately been under my care, may, I think, be taken as supporting this view. It is that of a young acrobat or contortionist,

[1] See 'Brit. Med. Journal,' Dec. 9, 1882.

aged twenty-one, who about eighteen months ago began to complain of weakness and tremulousness of his legs. When he first came under my observation he had not developed ankle clonus or the spastic gait, and the knee-phenomenon was not markedly exaggerated. With rest he made some marked improvement for a time; but he again took to his work, and gradually became, what he is now, a typical specimen, as far as clinical observation alone can demonstrate, of a "spastic" paralytic. For six months previously to June in the present year he had been performing, against my advice, literally nightly in Berlin and Paris, being obliged more and more to favour his legs by confining most of his "business" to his arms. This youth had been an acrobat ever since four years old. With the exception of having been a "delicate" child, his health appears to have been very good up to the time of his legs beginning to fail. The rest of his family were always strong, and are following the same occupation as himself. I satisfied myself without doubt that he had not had syphilis.

A case of this kind irresistibly turns our attention to the possibility of its functional origin, though of course much more observation is wanted. Taking into consideration the history of the case, the mode of its onset, the amelioration of the early symptoms by rest, and the extreme difficulty of suggesting any other plausible explanation for this typical case, it may be said that the hypothesis of its functional origin covers the facts before us; and, provided it be not shown to be contrary to others, may possibly, if tentatively held, with full readiness to drop it in the light of further knowledge, be found of some service in the study and treatment of paraplegic symptoms.

Other affections ranked under the head of system-diseases of the cord I do not propose to discuss here. Progressive muscular atrophy, and the disease long known under the name (now proved inaccurate) of infantile paralysis, whose morbid anatomical concomitants are localised in the grey anterior cornua of the spinal marrow, present some differences in character from the affections connected with lesion of the white matter that we have been discussing. The lesions here

do not take a physiological course in the disease known as polio-myelitis anterior; and the existence of a primary inflammation, especially affecting the grey matter of the cord, and occurring, as it is believed, in the cervical and lumbar enlargements, is not out of harmony with the clinical facts or the anatomical conditions of the case. The chronic change in the anterior cornua observed in progressive muscular atrophy, corresponding as it does in position to the entry of the nerves supplying the affected muscles, is not contradictory to the theory of the functional origin of the disease. That the mischief begins in the periphery is held by several observers, both clinical and anatomical, and many facts in the history of these cases seem to favour this point of view.

Certain affections of more or less permanence, characterised by paralysis or spasm, but which are not as yet connected with any observed central change, may be here alluded to, as apparently of functional origin, and illustrative of the point under discussion. The frequent great chronicity, for instance, especially when not sufficiently regarded at the outset, of scrivener's palsy, or writer's cramp, would lead us to suppose that some central change had taken place. The marked failure of co-ordination seen in this disease seems to imply some kind of disorganisation of the central structures immediately concerned in the previously harmonious action of the muscles. Yet the apparently functional origin of such an affection as this is very striking, and widely believed in. There is, moreover, reason to believe that it is most often seen in members of "neurotic" families.

The prognostic and, possibly, the therapeutic aspect of this method of regarding some of the system-diseases of the cord deserves perhaps a few words in conclusion. The obvious physical result of overwork (whether the overwork be regarded as absolutely primary in causation or not) in such a disease as scrivener's palsy calls loudly on Rest for its cure or relief; and if taken in time, the disease frequently yields to this rational treatment.

With respect to the prognosis, and perhaps the prophylactic treatment, of spastic paralysis and tabes, a possible hint may hence be derived. The established disease of course implies

the structural central change; and treatment here can do little or nothing. But the study, when possible, of early untoward symptoms which, though known often to precede the well-recognised disease, yet, according to the hypothesis of functional disturbance, may not portend the necessary sequel of the full-blown malady, might help in some cases to prevent the hopeless and dogmatic diagnosis that is in danger now of being given, even on the strength of a single, and especially a newly-observed, symptom; and perhaps in some few cases might hinder the occurrence of the incurable malady. It is possible that in these days of minute and microscopical research we may sometimes fail to hold ourselves off, as it were, and take a look at our facts from a little distance. To get a true notion of our object we must vary our focus and our points of view. And just as the clinics and pathologies of pulmonary consumption are in some quarters regarded, with apparent satisfaction to the mind, as already entirely revolutionised by the discovery of the tubercle *bacillus*, and the accumulated experience of past observers in the field seems to be overlightly, or at least prematurely, set at nought, so in neurology the recently-studied changes in the cord may tend to engross the attention of observers too entirely as ultimate facts, and thus perhaps prevent us from reaping the advantages that sometimes result from merely changing our point of view.

DEAFNESS IN HYSTERICAL HEMIANÆSTHESIA.

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Among the perversions of sensibility to which hysterical patients are subject, that of hemianæsthesia is one of the most common, and the study of its various phenomena well illustrates the fact, that this disease is as subject to given laws and as worthy of scientific investigation as any other; a fact which, if not at present universally accepted, is rapidly becoming so, thanks in great measure to the efforts of Professor Charcot and his pupils. It is at his suggestion that I have examined the hearing of the patients (numbering thirteen) with this affection in the Salpêtrière, the result of which examination it is the immediate object of this paper to report.

Before, however, entering upon this branch of the subject, it may not be out of place to devote some space to the general characteristics of the symptom under consideration. A knowledge of these characteristics is especially important, inasmuch as the diagnosis must sometimes be made between hysterical hemianæsthesia and that due to an anatomical lesion.

Hemianæsthesia is one of the more common of the lasting symptoms exhibited between the attacks by hystero-epileptics, to which class belong the majority of the cases examined, all conforming more or less closely to the classical type. There exists in these cases a loss of sensibility, occupying the whole of one side of the body, generally limited sharply by the median line, though not always so well defined, as for instance in the case reported by Féré,[1] in which a sensitive spot, the size of the hand, was found behind, between the dorsal and lumbar regions, and another of similar size near the navel. The

[1] "Notes pour servir à l'histoire de l'hystéro-épilepsie;" 'Archives de Neurologie,' Nos. 8 and 9, 1882.

anæsthesia may be complete, involving loss of sensation of touch, pressure, heat, cold, and pain; or it may be incomplete, in which case analgesia, with or without thermo-anæsthesia in the more common variety. The anæsthetic side is generally paler and colder than the other, and blood flows less easily from the prick of a pin or other instrument. This condition of ischæmia, which is, however, rarely found existing to such a degree, can explain, after a certain allowance for exaggeration, the so-called miraculous cases in the Epidemic of St. Médard, in which strokes of the sword failed to produce hemorrhage.[1]

The anæsthesia is sometimes limited to the integument, and sometimes extends to the deeper parts, muscles, bones, articulations, mucous membranes. The abdominal viscera, however, do not seem to share in the anæsthesia; on the other hand, the so-called "ovarie" generally exists on the affected side; that is to say, an extremely sensitive spot is found in the region of the ovary, pressure on which, if the patient is subject to hystero-epilepsy, can induce one of the attacks, the nature of which has been so carefully studied in the wards of the Salpêtrière, and which has been made the subject of an admirable volume by Richer.[2] Forcible pressure over the same region is capable of cutting short an attack once commenced, and for this purpose various belts have been made, resembling trusses by means of which constant pressure may be applied over the ovary. That this sensitive region really corresponds to the ovary is placed beyond a doubt by the fact that it gradually rises during pregnancy with the rise of the uterus, and falls again after parturition.

The hemianæsthesia is more commonly located on the left half of the body, but it may change to the opposite side in the same patient, together with all the attendant symptoms of special sense which will be presently mentioned. This change may also be effected at the will of the observer by the use of one of the many so-called æsthesiogenous agents. In some

[1] Charcot, 'Leçons,' &c., p. 303. Jolly quotes a characteristic case of ischæmia described by Rosenthal: 'Allg. Wien. med. Zeit.,' 1876.

[2] 'Études cliniques sur l'hystéro-épilepsie ou grande hystérie.' Par Dr. Paul Richer. Paris, 1881. (See review in 'Brain,' Jan, 1882.)

cases the anæsthesia occupies both sides of the body, though not always in the same degree, and these cases are particularly favourable for examination with regard to the symptoms of sight, hearing, &c., as the affection of the special senses corresponds in degree with that of the general sensibility. Contractures, when present, occur as a rule on the anæsthetic side, and the motor force, as shown by the dynamometer, is generally found somewhat diminished. The loss of sensation, far from inconveniencing the patients, is often not noticed by them until the attention is called to it, and they sew, knit, and perform other duties without the least disturbance.

With regard to the share which the special senses take in this affection, I cannot do better than to quote verbatim the account given by Féré:[1]—

"In the hemianæsthesia of hysterics the insensibility extends not only to the skin and mucous membrane, but generally also to the organs of sense; the sight, the smell, the hearing, the taste, are equally diminished on the same side. There is generally present, in one word, a sensitive sensorial hemianæsthesia. Still, the organs of sense may be unaffected, or only slightly involved. The eye, for example, which in the case of complete anæsthesia is the seat of the characteristic disturbances, advancing sometimes to total loss of vision, shows more often a concentric retraction of the visual field, corresponding generally with achromatopsia, more or less pronounced.

"The colour-blindness is sometimes complete, but is frequently partial; certain patients lose, for example, only the perception for violet,[2] and in general there is a correspondence between the degree of retraction of the field of vision and the degree of achromatopsia.

"It is a fact which has attracted little attention that the sensibility in general of the eye, the sensibility of the conjunctiva and cornea, bears a close relation to the special sensibility of that organ. The patients whom we have been able to

[1] "Notes pour servir à l'histoire de l'hystéro-épilepsie;" 'Archives de Neurologie,' Nos. 8 and 9, 1882.

[2] "Violet is the colour for which the field of vision is physiologically the least extended, therefore the first to disappear on retraction of the visual field."—Landolt, 'Leçons sur le diagnostic des maladies des yeux,' p. 155.

observe, who have presented no retraction of the field of vision, and no achromatopsia, have preserved the sensibility of the conjunctiva; those who have lost their vision for one or more colours, and who have a retraction of the visual field more or less regularly proportional, have lost not only the conjunctival sensibility, but also that of the cornea. In the latter case, if, while the patient directs the gaze fixedly at some object, one advances a little roll of paper, one sees the eye and eyelids remain immovable as long as the foreign body does not come into the field of the pupil. The oculo-palpebral reflex is obtained quickly if, instead of advancing the object to contact with the membranes, one only advances it part way in the median line. The reflex is then determined solely by the stimulation of the retina, which, though it has lost the power of perceiving colours, can distinguish light and darkness. In the pure hemianæsthetics, or in the total anæsthetics with predominance of one side, one can by the magnet, the static electricity, or the other well-known procedures, transfer the anæsthesia, and make a counter-demonstration with constant results. There is then in hysterical hemianæsthesia a constant relation between the cutaneous insensibility and the sensorial insensibility. And this constant relation exists not only when the anæsthesia extends over the whole of one side of the body, but also when it is more or less limited. When one causes the disappearance of the hysterical anæsthesia by static electricity, one sees the anæsthesia return at the end of a certain time, the return commencing in a localised region, varying in different subjects, and having in general no relation to the distribution of the nerves. In one of our patients the insensibility returns in the first place over a limited zone, which includes the eye, the conjunctiva and cornea, and the sensorial anæsthesia returns with that limited tactile insensibility."

The same author mentions the unilateral deafness which appears in these patients, and states that the general sensibility of the external auditory canal (not the tympanum) varies with the acuity of hearing, just as that of the conjunctiva varies with the field of vision for colours. He draws the conclusion that there exist in the brain sensitive centres,

which supply in common the organs of special sense and the integument covering them.

Jolly [1] remarks that functional deafness is sometimes noticed in severe cases of hysteria, remaining after violent attacks, and combined with other anæsthesia, adding that it may, like hysterical blindness, suddenly disappear, and be replaced by other hysterical symptoms.

Uspenski [2] has lately reported two cases of hysterical deafness, accompanying anæsthesia of the face and external meatus, in one case complicating lesion of the ear, while in the other the physical examination revealed no defect. In both these cases the functional deafness varied with the cutaneous anæsthesia, and in both cases a marked improvement appeared on the application of the galvanic current to the cervical sympathetic.

Gellé has made measurements respecting the distance necessary for distinct audition during the "transfer" of hemianæsthesia, confirming the law that cessation of anæsthesia on one side of the body involves its appearance on the symmetrical part on the other side of the median line.

Urbantschitsch has reported a case of hysterical hemianæsthesia with the "transfer." The subjective noises, and hyperæsthesia for loud tones which existed previously in the non-anæsthetic, were transferred to the anæsthetic ear. The deafness, which was complete, passed meanwhile to the previously hyperæsthetic ear. Before the transfer the strongest galvanic current (40 Siemens-Halske elements) produced on the affected side neither auditory nor optic symptoms, while six elements on the unaffected side produced such violent vertigo as to prevent the establishment of the auditory formula. This relation was reversed by the transfer.

With regard to the genuineness of the anæsthesia in these cases. It must be remembered that the psychological state of hysterical patients is such as predisposes to deceit, in view of which the explorations are conducted at the Salpêtrière with

[1] Ziemssen's 'Handbuch,' Bd. xii. 2, 1877, p. 534.

[2] "Taubheit complicirt mit hysterischer Neurose und hysterische Taubheit." 'St. Petersburg med. Wochenschr.' 1882, No. 8. Reported also in 'Neurologisches Centralblatt,' Oct. 15, 1882.

all the precautions necessary to its detection. That simulation played a part in any of the cases here reported was out of the question, for apart from the various precautions taken, the very regularity of the results precludes deceit.

The inadequacy of "expectant attention" in explaining these results becomes evident on considering the cases in which the loss of hearing for sounds conducted by the bone was lost, while that for sounds in the air was retained. It is absurd to suppose that a number of patients, hitherto unexamined in this respect, on being separately questioned should be found to coincide with regard to this symptom, and in a degree proportional to their several grades of anæsthesia.

Coming now to the special division of the subject, we may divide the patients for convenience into three classes, and consider the hearing in each class separately. The *first class* comprises the patients with *complete* anæsthesia on one side, the other side remaining intact. The *second class* includes the patients with *incomplete* anæsthesia on one side, the other side remaining intact. The *third class* includes those patients with anæsthesia more or less complete on both sides. The degree of anæsthesia in these cases is rarely the same on the two sides.

In the *first class*, as in all, the anæsthesia extends to the deep part of the ear. The anæsthesia being complete in this class, the tympanum may be touched without any acknowledgment of sensation, and without the least reflex movement. In such a case the patient will be unable to tell with the eyes closed whether there is an instrument in the ear or not. Touching the deep parts of the opposite ear in these patients produces the usual disagreeable sensation and reflex movement, generally indeed exaggerated. That the anæsthesia extends to the middle ear is seen by the fact that insufflation by the Politzer air douche produces no sensation in the ear of the affected side. This is a fact to be borne in mind in the examination of these cases, for if the patient's sensations were relied on, the Eustachean tube might be supposed impermeable.

In this class of patients neither the watch, the voice, nor the tuning-fork is heard by the affected ear, and no tuning-fork is

heard on this side when vibrating against the skull. The tuning-fork vibrating in contact with the forehead or teeth is heard *only on the healthy* side, in direct opposition to the cases in which the loss of hearing is due to defective conductibility in the ear, and in which the tuning-fork so placed is heard more distinctly on the affected side.

In the *second class* of patients, those with incomplete anæsthesia on one side, the loss of sensibility in the ear corresponds as a rule to that of the body in general. A common form consists in analgesia with thermo-anæsthesia and diminution of tactile sensibility. In patients exhibiting this degree of anæsthesia, the tympanum of the affected ear may be touched without producing any unpleasant sensation, the touch being only faintly perceived, and being followed by no reflex. The air douche produces a slight sensation. The degree in which the hearing is affected varies within certain limits, but has been found lessened in every case examined. In some of these cases is found a *diminution* in the hearing for sounds conveyed by the *air*, and a *diminution or loss* of hearing for sounds conveyed by the bone. In these cases the watch, voice, and tuning-fork, all of which are heard at a normal distance, or at a distance abnormally great, by the unaffected ear, are heard at a half or a third of that distance by the affected ear, or perhaps only in the immediate vicinity; while the watch or tuning-fork placed on the bone behind the ear on that side is heard faintly, or not at all, when the ears are closed. In these cases the sound of the tuning-fork vibrating on the forehead or teeth will predominate on the healthy side if the hearing for sounds conveyed by the bone is diminished in the affected ear, and solely there if it is lost. The hearing may be approximately or absolutely normal for sounds conducted by the air, and lost entirely for sounds conducted by the bone, as in the case of one patient who can hear all tuning-forks distinctly in the air, and can hear the watch, normally heard at 80·100 ctm., at a distance of more than a metre, while no tuning-fork is heard on the affected side when vibrating against the skull at any point, as long as the ear is closed. In the normal condition, closing the ear, as is well known, intensifies the sounds conducted by the bone.

In regard to the *third class* of cases, in which the anæsthesia is total, the completeness of the anæsthesia is rarely the same on both sides, a common form being complete hemianæsthesia on one side, and analgesia on the other. These cases are particularly interesting as regards the study of the hearing, for the degree of deafness always corresponds to that of the anæsthesia. To illustrate; one patient has complete hemianæsthesia on the left, and analgesia with diminution of tactile sensibility on the right. This patient cannot feel the contact of an instrument with the tympanum on the left; on the right she feels the contact more or less distinctly, but it gives rise to no unpleasant sensation. On the left she hears neither watch, voice, nor tuning-fork, even in the immediate vicinity, and no tuning-fork vibrating against the skull. On the right she hears all with greater or less distinctness by the air, but perception for sounds conveyed by the bone is absent. A tuning-fork vibrating against the forehead or teeth is not heard at all when the right ear is closed, though with the right ear open the same tuning-fork may be heard by the air quite distinctly.

Another patient has complete hemianæsthesia right; incomplete, left. She feels nothing when the right tympanum is touched, but a slight sensation when the left is touched. The air douche produces a faint sensation in the left, none in the right. This patient hears no sound on the right, whether conducted by the air or by the bone. On the left, the watch is heard on contact only. A tuning-fork placed on the bone behind the left ear is heard faintly when the ear is open, and still more faintly when the ear is closed. No tuning-fork vibrating on the forehead or other part of the skull is heard at all.

In this case the hearing, which is completely lost on the right, is greatly diminished on the left, both for sounds conducted by the air and those conducted by the bone.

These two cases will serve to illustrate this class, in which an infinite variety may be imagined, the general character remaining the same.

A noticeable feature in all these cases, and one which throws some light on an important physiological subject, is the uni-

formity with which the deafness for sounds conveyed by the bone exceeds that for sounds conveyed by the air. This is probably due to the fact that the vibrations conveyed to the ear by the air are better adapted for the irritation of the peripheral auditory apparatus than those conveyed by the bone.[1] When then the receptive power of the auditory centres is lessened, as is probably the case in the hysterical patients, the hearing for sounds conveyed by the bone disappears before that for those conveyed by the ear. This enfeeblement of the auditive centres in hysteria is quite analogous to that in old age, in which, as is well known, the perception for sounds conveyed by the bone disappears before that for sounds conveyed by the air, the former being sometimes completely lost before the age of sixty.

Some authors attribute this condition in age to a lessening of the conductibility of the bones of the skull, which is a very improbable explanation, for if the conductibility of these bones for vibrations becomes altered as age advances, it is probably rather increased than decreased with their increasing solidity. The ears, meanwhile, of the large majority of persons undergo such changes as age advances, thickening and indrawing of the tympanum, closure of the Eustachean tube, &c., that comparatively few persons at sixty preserve the normal conductibility of the tissues of the ear. As far, then, as the conducting media are concerned, the deafness for sounds conveyed by the bones should never be expected to precede that for sounds conveyed by the air. When the hearing for the former sounds disappears first, it must rather be attributed to the gradual enfeeblement of the auditory centres in the brain. In the hysterical patients there can be no question of a change in the conductibility of the bones, but all symptoms must be referred directly to the nervous system, and in these cases, as has been seen, the hearing for sounds conveyed by the bone always disappears first. On this question, as on many others in the physiology of the nervous system, these hysterical patients point the way to the most satisfactory solution.

[1] That this is really the case is evident from the simple experience that a tuning-fork, feebly vibrating, is heard much longer through the air than through the bone.

It need hardly be added that the condition of the ear itself may modify the cases to a certain extent, a fact not to be forgotten in the analysis of the cases, for distinction must be made between deafness due to a cessation of function of the auditory centres, and that due to an accumulation of cerumen, or to a chronic catarrh of the middle ear. The examination by osseous conductibility is here of the greatest value, for whereas obstruction in the ear itself intensifies sounds conducted by the bone, such sounds are the first to disappear in hysterical deafness.

The following cases, typical of all, will serve to illustrate the three groups of patients mentioned.

Case I.—Mlle. Bl——, 20 years old. Complete hysterical hemianæsthesia, left.

Senses of *taste* and *smell* lost on the left. With the left eye patient distinguishes only the colour red.

Examination of the ears shows membranes normal. Touching the tympanum is neither felt nor followed by a reflex movement on the left. On the right the sensibility is normal. Politzer air douche not felt on the left.

Examination of hearing.—The patient hears neither the voice, the watch, nor the tuning-fork on the left; the two latter, whether in the air or on the bone. The watch (heard normally at a distance of 80–100 centimeters, is heard on the right at 85 centimeters. On the right the voice, whispered and murmured, is heard at a normal distance, and all tuning-forks are well heard, whether vibrating in the air or against the bone. The tuning-fork placed on the teeth or forehead is heard only on the *right*.

Case II.—Mlle. Card——, 16 years of age. Incomplete hemianæsthesia, left; the sensations of pain and of cold are lost, and that of touch lessened. Retraction of the visual field, left. No achromatopsia. Sense of taste entirely, and that of smell nearly, lost on the left.

Examination of ears showed right tympanum normal; the left indrawn and opaque. Left Eustachean tube impermeable. Touching the left tympanum produces only a faint sensation.

Examination of hearing.—Patient hears the watch at 130 centimeters on the right; only at contact on the left. The

whispered voice (using the word "einq") heard normally at about 10 meters, is heard on the right at 14 meters; on the left at 20 centimeters. All tuning-forks are heard in the air on the left, but none on the bone. Neither the watch nor tuning-fork placed on the bone with the ear closed is distinguished on the left. *A tuning-fork placed on the forehead or teeth is only heard on the right.*

Case III.—Mlle. Lim——, 19 years old. Incomplete hemianæsthesia, left.

Special senses.—Patient distinguishes all colours on the right; only the red and green on the left. Retraction of visual field, left. Taste and smell are entirely absent on the left; slightly diminished on the right. V. = $\frac{2}{7}$ right; $\frac{5}{10}$ left.

Examination of ears shows the membranes approximately normal. Touching the tympanum on the left is felt, but produces no reflex movement nor disagreeable sensation. The deep parts of the right ear preserve their normal sensibility.

Examination of hearing.—The watch is heard on the right at 56 centimeters; on the left at 13 centimeters. The whisper is heard on the left at 30 centimeters. The tuning-fork is heard normally on the right, both by the air and by the bone. The tuning-fork placed behind the ear is not heard when the ears are closed. *The tuning-fork vibrating on the forehead or teeth is heard on the right.*

Case IV.—Mlle. Mig——, 16 years of age. Complete anæsthesia, left; analgesia, right, with diminution of tactile sensibility. *Muscular sense* preserved; *odour* lost on the left, diminished on the right. The same is true for the taste. On the left, sees only the colour red. On right, all colours. Visual field greatly contracted left. V. = 1 right; $\frac{1}{7}$ left.

Examination of ears shows the membranes normal. Touching the membrane is not felt on the left, slightly on the right.

Examination of hearing.—Patient hears the watch on the right at 120 centimeters; on the left, not on contact. The voice is not heard on the left. The tuning-forks are not heard on the left, either in the air or on the bone; on the right they are heard in the air, but not on the bone with the ear closed. The largest tuning-fork, placed on the forehead, is unheard when the right ear is closed.

The second and last cases illustrate a condition not uncommon in hysterical hemianæsthesia, i.e. an unusual acuteness of hearing on the unaffected side. The watch, heard normally at 80–100 centimeters, was heard in one case at 120 centimeters. This acuteness of hearing was transferred, without diminution, to the other ear in Case IV. (in which the membranes were normal) in the experiment to be immediately described.

The transfer.—The fact that hysterical hemianæsthesia, with all its symptoms of special sense, may change its seat, and that by means of certain agents this change may be brought about at the will of the operator, has been known for some time. This change has been known as the "transfer." In 1878 Charcot called attention to the so-called "oscillations," which are merely a continuation of the transfer. Rumpf[1] has demonstrated, by a series of careful experiments with the compasses, that similar oscillations of sensibility from side to side take place under physiological conditions. The oscillations in hysteria have been made the subject of careful study by Richer, the result of whose researches were published in the 'Progrès Médical' (Nos. 46 and 47).

To quote several of his conclusions:—

"III. The oscillations take place in the same way, whether the æsthesiogenous agent be removed or maintained.

"IV. The oscillations exist as well for the special as for the general sensibility.

"VI. The duration of the oscillations is very variable; we have observed oscillations of several seconds, and others of twenty minutes. They may last even longer. One may say in a general way that the oscillations are the shorter and the greater in number, the more easily they are produced.

"VII. When the oscillations are finished, the patient remains, as far as sensibility is concerned, in a state of relative stability. We have observed three varieties. 1. The patient recovers, for some time, total sensibility. 2. The patient remains in a state of transfer, i.e. if she was hemianæsthetic on the left before the experiment, she remains for

[1] "Ueber den Transfert;" 'Berliner Klinischer Wochenschrift,' 1879, No. 36.

some time hemianæsthetic on the right; perhaps one should consider this condition merely a prolonged oscillation of which the end has not been observed. 3. Finally, the patient finds herself in the same condition as before the experiment."

The study of these oscillations is especially interesting in connection with the study of special sense, and observation of the several varieties mentioned above shows that the same rule holds good in all, for the hearing as for the other senses, i.e. that displacement of anæsthesia on one side is accompanied by its appearance on the other, and always to a corresponding degree. Given a patient who hears perfectly by air and bone on the left, but who has complete deafness on the right, after the transfer she will hear perfectly (there being no obstruction in the ear itself) by air and bone on the right, while she will have completely lost the hearing on the left. This condition will change from side to side during the oscillations, until the patient arrives at one of the states of relative equilibrium mentioned by Richer. The sensitiveness of the tympanum will oscillate in a corresponding manner.

Given, again, a patient who has lost on the right perception for sounds conveyed by the bone, but preserved that for sounds conducted by the air, the same condition will be found on the left after the transfer.

If obstruction exists in the ear itself, the hearing by the air will only return after the transfer to the degree which is to be expected with such obstruction. This was well illustrated in the case of Mlle. Card——, whose left tympanum was normal, but whose right indrawn and opaque, while the Eustachean tube on the right was impermeable. The transfer being made in her case, while the hearing by the bone is lost on the left, and that by the ear reduced to such a degree that the watch is only heard on contact, the hearing on the right is recovered for sounds conveyed by the bone, while that for sounds conveyed by the air only returned to a slight degree. That is to say, the watch, which before the transfer was only heard by the right ear, on contact was heard after the transfer at a distance of 10 centimeters. This recovery must be in her case considered a perfect one, as far as the auditory nerve is concerned. Both membranes being normal in any case, the same degree

of hearing is recovered by the previously anæsthetic ear, which existed before the transfer in the unaffected ear. This may be seen in the following experiment, the only one of which the details need be given, as it is typical of all, the variations between the different cases being such as are pointed out by Richer, and quoted above.

Experiment.—Mlle. Mig——. (Case IV. above.) Complete anæsthesia left, including the tympanum. Analgesia on the right. Loss of hearing by the bone on both sides; loss of hearing by the air on the left (does not hear the watch on contact); preservation of hearing by the air on the right to a high degree (hears the watch at 120 centimeters).

A large horse-shoe magnet was so placed that one of its poles was within a few centimeters of the left, or completely anæsthetic ear.

In a few minutes the sensation appeared in the left ear and the whole of the left half of the body. The watch, previously not heard on contact, was heard first on contact, then at a distance of a few centimeters, and at the end of seven minutes from the commencement of the experiment was heard at a distance of 115 centimeters. At this time a tuning-fork placed on the bone behind the left ear was not heard when the ear was closed. Touching the deep part of the ear was felt, but produced no unpleasant sensation. This ear, then, had now reached exactly the same condition of the right before the transfer.

At this time all sensitiveness to the touch had disappeared in the right ear, as well as over the whole right half of the body. The watch was not heard on contact, nor the tuning-fork against the bone. This ear had then lost its general and special sensitiveness, while the other had recovered exactly the amount which this had lost.

At the end of ten minutes from the commencement of the experiment the return oscillation commenced, and was completed in three minutes, at the end of which time the two sides stood in the same relation to each other as at the beginning. After an interval of three minutes the second oscillation commenced and lasted two minutes. The return commenced in three and a half minutes, and lasted a minute and a half.

Measurements taken during this return oscillation of the distances at which the watch was plainly heard, gave the following results:—

Time.	*Distance* (R.).	*Distance* (L.).
0	Not on contact.	115 mm.
30 sec.	6 mm.	40 mm.
60 sec.	58 mm.	Contact.
90 sec.	120 mm	Not on contact.

This table illustrates admirably the regularity with which the disappearance of function on one side is compensated for by its appearance on the other.

The third oscillation commenced in five minutes, and lasted only thirty seconds; the return commencing in four minutes, and lasting forty seconds.

The next four changes ranged from thirty to forty seconds, and the intervals were of about the same length. The character of the oscillations was always the same, and at the end of the fifth the experiment was discontinued.

The patient was found at the end of several hours in her original condition, completely anæsthetic on the left, and incompletely so on the right.

As it seems hardly necessary to give further details, I close with the following principal

Conclusions.—1. The sensibility of the deep parts of the ear, including the tympanum and middle ear, disappears in hysterical hemianæsthesia with that of other parts of the body, and in the same degree.

2. The degree of deafness corresponds with that of the general anæsthesia, being complete when the latter is complete, and incomplete when the latter is incomplete.

3. When the loss of hearing is incomplete, the deafness for sounds conveyed by the bone exceeds that for sounds conveyed by the air.

4. When the transfer is made, the hearing as well as the general sensibility of the deep parts of the ear improves on one side (allowance being made for accidental lesions in the ear itself) in exactly the same degree in which it disappears on the other.

ON THE SPASMODIC PARALYSES OF INFANCY.

BY JAMES ROSS, M.D., F.R.C.P.

(Continued from p. 363.)

In the first part of this paper, which appeared in the last number of 'Brain,' I described a case (Case II.) of double athetosis with aphasia. It may be worthy of notice in passing, that Sir Walter Scott's description of Nick Strumpfer, the dwarf in 'The Pirate,' corresponds so accurately to the phenomena presented by my patient, so far at least as the facial contortions and disorderly movements are concerned, as to leave little room for doubt that the picture was drawn from real life. The Udaller, with his two daughters, pays a visit to their kinswoman, Norna of the Fitful Head. After a vigorous knocking, "the door opened, and displayed, to the alarm of Brenda, and the surprise of Minna herself, a square-made dwarf, about four feet five inches high, with a head of most portentous size, and features correspondent—namely, a high mouth, a tremendous nose, with large black nostrils, which seemed to have slit upwards, blubber lips of an unconscionable size, and huge wall-eyes, with which he leered, sneered, grinned, and goggled on the Udaller as an old acquaintance, without uttering a single word." The Udaller facetiously assured his daughters that the dwarf could "keep his mistress's counsel, and never told one of her secrets in his life." "The ugly dwarf grinned ten times wider than before, and showed the meaning of the Udaller's jest by opening his immense jaws, and throwing back his head, so as to discover that in the immense cavity of his mouth there only remained the small shrivelled remnant of a tongue, capable perhaps of assisting him in swallowing his food, but unequal to the formation of articulate sounds.

Whether this organ had been curtailed by cruelty, or injured by disease, it was impossible to guess; but that *the unfortunate being had not been originally dumb, was evident from his retaining the sense of hearing.*" "Having made this horrible exhibition, he repaid the Udaller's mirth with a loud, horrid, discordant laugh, which had something in it more hideous, that his mirth seemed to be excited by his own misery." It may safely be assumed that the description which even such an acute observer as Scott gives of the small and shrivelled tongue, was biassed by the preconceived theory that the inability to articulate must have been occasioned by a local defect of the tongue. The last case which I reported in the first part of the paper was an example of congenital hemiplegia, with a defect in the parietal bone on the opposite side. The following case, kindly sent to me for examination by my friend Dr. Cullingworth, is an example of congenital hemiplegia, without any other defect being present.

Case VI.—Margaret H. K., aged 8 years, came under my observation on July 19, 1881. An aunt of the child, under whose care she is at present, stated that the patient never suffered from any illness, and is particularly positive that she never had convulsions. The parents are convinced that the left hand was paralysed at birth, and it was noticed that she never moved it in early infancy.

Present condition.—The subject is a little girl, 3 feet 11 inches in height, well-proportioned, healthy in appearance, of fair complexion, and with a bright and intelligent countenance. She goes to school, and has attained the second standard, and is supposed to be a very good scholar for her years. The left arm and leg are partially paralysed. The muscles about the left shoulder-joint are not implicated in the paralysis, but the patient cannot fully extend the fore-arm upon the arm, or supinate the fore-arm, and considerable resistance is offered to passive supination of it. All the movements of the affected hand and fingers can be performed, but they are not fully co-ordinated, and the patient experiences considerable difficulty in picking up a pin, or grasping small objects with this hand. The tendon reflexes are not decidedly increased. The various segments of the left lower extremity are extended upon one

another, the foot is kept in a state of extreme talipes equino-varus, and considerable muscular tension is excited by attempts at passive movements of the limbs. The patellar-tendon reflex is only feeble in the left, and absent in the right leg. Ankle-clonus cannot be elicited. The following measurements were taken:—

		Right.		Left.	
Clavicles	Length	4	inches.	3⅞	inches.
Upper arm	"	8¾	"	8¼	"
"	Circumference	7	"	6¾	"
Fore-arm	Length	6½	"	6	"
"	Circumference	6¾	"	6¼	"
Hand	Circumference	5½	"	5⅛	"
Thigh—From ant. sup. spine of ilium to external condyle		13½	"	12	"
"	Circumference, thickest	14	"	13	"
Legs, calves	Circumference	9¾	"	9	"
" Length from head of fibula to external malleolus		10¼	"	9½	"
Foot	Length	7½	"	7	"
"	Circumference of instep	7½	"	7½	"

The face is quite symmetrical, and there is not the slightest evidence that the facial muscles have been implicated. The faradic contractility of the affected muscles is normal, and there are no sensory disturbances. The skin over the left leg and foot is blue and mottled, and becomes much colder than that of the right leg after a short exposure.

The case which has just been described demands no lengthened comments. A porencephalous defect is probably present in the right hemisphere of the brain. This case is remarkable for the complete absence of idiocy or any mental defect. It will be interesting to know whether epilepsy will become established at puberty, as occurs in cases of unilateral atrophy of the brain.

My thanks are due to Dr. Cowan, of Newchurch, for sending me the following case, and for obtaining a post-mortem examination after the child had died from an attack of croup.

Elizabeth B., aged 2 years and 5 months, came under my observation as an out-patient at the Royal Infirmary, some time in November last year. The following notes of her case were taken on Dec. 19, 1881. The parents are healthy, and have several other children, all of whom are healthy. The mother has not had any miscarriages. The child appeared to have

been well-formed and healthy at birth, and it was only at three months of age that the parents observed that she could not hold her head up, and that her hands were stiff. The infant had not at any time suffered from convulsions, and has been singularly free from any infantile disease.

Present condition.—The child is small for her age, but fairly nourished. The lower extremities are slightly flexed at the hip and knee-joints, the feet are maintained in positions of talipes equinus; the muscles are rigid, and the spasm is much increased on any attempt being made to alter the position of the limbs by passive motion, the spasm being particularly manifest in the adductors of the thighs. The spasmodic rigidity of the muscles of the lower extremities also becomes much increased when an attempt is made to place the child erect on the floor. The muscles of the calves then contract powerfully, and the heels become elevated, so that only the points of the feet touch the ground. The adductors also contract strongly, so that the limbs are closely applied to one another when they are in the same plane, and become crossed when one is in front of the other. When the patellar tendon is struck, a spasm of all the muscles of the lower extremity supervenes, so that a true knee-jerk cannot be elicited. Ankle-clonus cannot be provoked in either foot. The muscles of the upper extremities are also in a state of spasmodic rigidity, and the various segments of the arms are held in more or less fixed positions. The upper extremities are held in symmetrical positions. The arms are abducted, so that each elbow is about four inches removed from the body; the fore-arms are semi-flexed on the arms, and pronated; the hands are slightly flexed in the fore-arms, and a little inclined to the ulnar borders; the thumbs are flexed into the palms; and the fingers are semi-flexed over the thumbs. It is impossible to produce complete extension at the elbow-joint by passive motion, and any attempt at altering the position of the limbs increases the spasmodic rigidity of the muscles. The patient can voluntarily grasp an object with either hand, but the movements made in order to reach the object are irregular and uncertain, and it is impossible for her to pick up small objects.

The head is habitually kept bent forwards, so that the chin rests on the sternum. The sterno-cleido-mastoid muscles are tense and prominent, the left muscle being more prominent than the right, and consequently the head has a lateral inclination towards the left. The child can raise her head by a voluntary effort, in order, for instance, to look at an object placed above the level of the axes of vision, but the necessary effort cannot be long sustained, and the head soon assumes its bent and inclined position. The child does not speak yet, but the parents state that she makes an effort to utter a few simple monosyllables, but she could not be persuaded to do so in my presence. I could gather from the description of the parents that such attempts are accompanied by contortions of the mouth and tongue, caused, doubtless, by spasmodic contractions of the muscles.

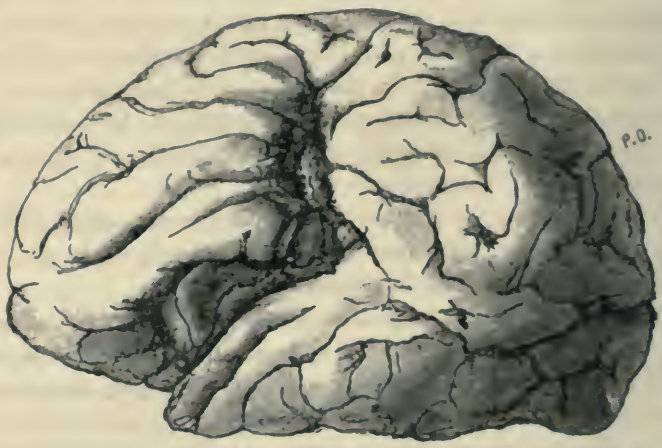
A few weeks after this report was made the child suffered from measles, and subsequently from an attack of croup, from which she died on Jan. 22, 1882.

The post-mortem examination was conducted on the evening of the 24th of January, by Dr. Cowan, Dr. Coutts, and myself.

The skull was symmetrical, smaller, if anything, than corresponds with the age, the transverse axis from one parietal bone to the other being unusually short. On the calvarium being removed, the dura mater appears in every respect healthy; but when it is reflected back, a deep sulcus is observed on each side, corresponding to the positions usually occupied by the fissures of Rolando. The external opening of these sulci is bridged over by the visceral layer of the arachnoid. Each sulcus extends from the point of bifurcation of the Sylvian fissure upwards to near the longitudinal fissure, a distance of 1½ inch. The lateral walls of each sulcus are formed by an infolding of the grey matter of the cortex, and these walls are so closely applied to each other that there is no great cavity left between them. In depth each sulcus extends the whole thickness of the centrum semi-ovale, and opens into the roof of the corresponding lateral ventricle. The opening into the ventricle on each side is only large enough to admit the little finger, and each opening is surrounded by a ring of grey matter, having all the naked-eye appearances of

the cortex. The cavities communicate freely with the ventricles without being covered by the ependyma. The ventricles are of normal size, or only slightly dilated, and only about one ounce of serous fluid has escaped during the removal of the brain. With regard to the fissures and convolutions, those in the neighbourhood of the defect are radially directed towards the centre of the cavity (Fig. 1). The ascending parietal and

Fig. 1.



ascending frontal convolutions appear to be absent on both sides. The posterior limb of the Sylvian fissure is very short, and runs almost vertically instead of horizontally as usual. The first and second temporo-sphenoidal convolutions have also a vertical inclination. The fissure of Rolando is replaced by the sulcus already described; the anterior wall of the sulcus is formed by the posterior extremities of the first and second frontal convolutions, and the posterior wall by the angular and supra-marginal gyri and, at the upper extremity, by the parietal lobule. On the internal surface of the hemispheres the calloso-marginal fissure does not extend backwards as far as usual, and the paracentral lobules are only imperfectly represented. The crura cerebri, pons, medulla oblongata and spinal cord appeared quite normal to the naked eye.

A microscopical examination of the grey matter forming the walls of the cavity show a structure more or less similar to

that of the healthy cortex. The giant cells of the inner division of the third layer of the cortex are, however, absent. This division contains large, round, nucleated cells, but they are entirely destitute of processes; and indeed absence of processes is a marked feature of all the cells observed, the cells being in this respect like those met with in the cortex of the brain in the embryo. The spinal cord on microscopical examination appeared in every respect normal, except that the lateral columns were somewhat smaller than the corresponding parts in a healthy cord. The anterior pyramids of the medulla oblongata are not much more than half the size of the pyramids of the medulla from a healthy child of the same age. The accompanying woodcut (Fig. 1) is from a sketch of the right hemisphere of the brain, kindly taken by Mr. A. H. Young.

Remarks.—In this case it would appear that the porencephalous defects were due to a simple arrest of development, and no cicatricial tissue was found in the walls of the cavities to indicate that they were caused by a destructive process. When the case came for the first time under my observation I was somewhat puzzled as to the nature of the lesion. As the mother was attended during her confinement by a midwife, it occurred to me that probably undue traction had been made on the head, thus injuring the upper cervical vertebræ, and giving rise to a pachymeningitis and subsequently to a transverse myelitis. But although such a course of events might account for the paralysis and contractures, it would not account for the affection of speech present. With regard to the affection of speech, it was difficult to determine whether it was of the nature of aphasia or of dysarthria. I felt inclined to believe that the disturbance of speech was of the latter character, inasmuch as the symptoms were almost in every respect similar to those observed in the case of James W. (Case IV.) when he first came under my care. It thus occurred to me that a single lesion in the pons might account for all the symptoms, for the same reasons which induced me to believe that such a lesion would account for the symptoms in the case of James W.

Against this view were to be set the facts that the child had a

very small head, and the expression of a microcephalic idiot. The condition which was actually found requires no further comment.

Spastic Paraplegia.—Let us now pass on to the consideration of the affection which may be termed *Spastic paraplegia of infancy.* The following four cases may be taken as good examples of this affection.

Case VII.—Wilfred R. L., aged 8 years, was kindly sent to me by Mr. Southam in April 1882. The parents of the child are living and healthy. The mother has had four children; one died in infancy from scarlet fever, and the two living, besides the present boy, are strong and healthy. No tendency to any kind of nervous disease can be discovered in the family. The mother thinks that the head of the child was born first, but she states that it was a "cross-birth," and that one foot was presenting at first. She does not know whether the operation of "turning" was undertaken by the medical man in attendance. The mother observed that the right ankle was slightly deformed when the child was a few days old, but nothing seriously wrong was discovered until it was time for it to walk. The patient never had convulsions.

Present condition.—The boy is well developed, with a ruddy complexion and healthy appearance. Height 41 inches. The right foot is in a permanent condition of talipes equino-varus; the toes are spread out, and there is a slight hyper-extension at the torso-phalangeal, and flexion at the phalangeal joints. The left foot is similarly deformed, but to a much less extent than the right. The boy can separate his thighs well, but to a less extent than is usual in health, and during the action the adductors can be felt in a state of spasm. The leg cannot be fully extended on the thighs, or the thighs on the trunk. The patient habitually stands on the sole of the left and the toe of the right foot, the latter being considerably in advance of the former. In this position the vertebral column is slightly curved to the right in the lumbar region, while there is a slight compensating curve to the left in the dorsal and lower cervical regions, and another to the right in the upper cervical region. The buttocks are somewhat projecting, and the trunk is slightly inclined forwards, so that a plumb line let fall from

the most prominent of the spinous processes of the vertebræ falls well within the sacrum. The dorsal curve which is so prominent a feature of pseudo-hypertrophic paralysis is absent. When the boy is made to stand with both feet in the same antero-posterior plane, he experiences great difficulty in bringing his left heel to the ground, and the muscles of the calf are then very tense and stretched, but he is wholly unable to bring his right heel lower than to within one inch from the ground.

Whenever he attempts to walk, both heels become immediately elevated, the left heel being one inch removed from the floor when the leg of the same side is active, and the right heel two and a half inches removed from it under similar circumstances. The boy consequently walks on the balls of his toes, and both feet are in positions of talipes equinus or equino-varus, the deformity on the right side during locomotion being so great that the foot assumes almost a vertical position. When the right leg is being moved forwards, the various segments of the extremity become nearly extended upon one another, with the exception of the slight degree of permanent flexion at the knee-joint. The distance between the trochanter and the toes is increased, and consequently the toes cannot clear the ground, and the leg cannot swing forwards by its own weight as in health. In order to effect the forward movement the boy leans his body well over to the left, thus elevating the right side of the pelvis along with the trochanter, and allowing the right leg to be projected forwards in one piece. But notwithstanding the elevation of the pelvis on the right side, the toes are only partially cleared off the ground, and every forward movement of the foot is attended by a scraping noise, and the toe of the boot on the right side is much worn. The forward movement of the leg is a compound one. The leg is moved forwards in one piece by the flexors of the thigh upon the body, but at the outset of the movement an outward inclination is given to the foot by a voluntary contraction of the abductors of the thigh, in order to raise the toes off the ground more effectually, while at the close of the movement the strong involuntary contractions of the adductors predominate, and the foot is dragged over to the left, and planted in front of the left foot. The foot, therefore,

instead of being moved forwards in a straight line, describes a semicircle, with its convexity to the right. When the right foot is brought to the ground and the right leg becomes active, the left leg is moved forwards in a similar manner to that described as occurring on the right side. But inasmuch as the toes are much less depressed on the left than the right side, the pelvis requires to be less elevated, and the toes do not drag on the ground with the same scraping noise. The alternate elevations of the pelvis on each side during the forward movements of the legs give to the gait a waddling appearance, not unlike that observed in pseudo-hypertrophic paralysis, but readily distinguished from it by the absence of the dorsal curve.

The patellar-tendon reflexes are very lively, probably exaggerated, but ankle-clonus cannot be elicited on either side. There is complete absence of all sensory disturbances, the electrical reactions of the nerves and muscles are normal, the nutrition of the muscles is preserved, and there are no disorders of the functions of the bladder or rectum.

The head is unsymmetrical, the parietal protuberance being well marked on the right side and deficient on the left. The circumference of the head is 20½ inches. The forehead is narrow, but otherwise well formed. The boy manifests a slight hesitation of speech, but there is no distinct stammering. The mother states that the boy's memory is very good, and that he is particularly quick at learning his lessons at school.

The following are brief notes of a similar case which came under my observation.

Case VIII.—T. M., aged 4 years, was brought to me by his parents in July 1881. The parents are healthy, and there is no evidence of a neurotic tendency, or predisposition to any constitutional disease in the families on either side. The mother had an easy confinement, and the head presented. The boy never suffered from convulsions in infancy, and he always enjoyed good health. Soon after birth it was noticed that the feet were deformed and stiff, but not much attention was paid to this condition until the child attempted to walk.

Present condition.—The patient is small for his age, but well developed, and of healthy and intelligent appearance.

On standing, he cannot bring both heels at once to the ground. Whenever he walks, a spasm of the muscles of the calf of the active leg supervenes, which elevates the heel to a distance of 1 to 1½ inch from the ground, so that the patient walks on his toes. The toes of the boots are worn, and he is very liable to catch the toe of the advancing foot against any slight inequality on the ground, and is consequently very liable to fall; but there is no scraping of the toes on a level floor. The gait is awkward and waddling, but there is no dorsal curve; the sacrum projects slightly behind the most prominent of the spinous processes of the vertebræ. The patellar-tendon reflexes are exaggerated, and a considerable degree of muscular tension is provoked by passive movement, but ankle-clonus cannot be elicited on either side. The electrical reactions of the nerves and muscles are normal, and there is no muscular atrophy. There are no sensory disturbances, and no disorder of the functions of the bladder and rectum.

The head is symmetrical, measures 19 inches in circumference, and no squinting or other disorder of the cranial nerves has been observed. The patient can speak fluently, and is believed by his parents to be as intelligent as other children of the same age.

Case IX.—James B., aged 4 years, was brought to the Royal Infirmary as an out-patient on August 16, 1882. The father and mother are both healthy, and no hereditary disease can be traced on either side. The parents have been married five years, and this boy is their only living child. Another boy died when six months of age, from convulsions. The mother had a tedious labour when confined of this child, but instruments were not used to facilitate delivery. When the child was born, his head was noticed to be distorted, the one side being much more prominent than the other. Immediately after birth the infant was seized with convulsions, and these continued to recur for the first five days after birth. He had no difficulty in swallowing after the convulsions ceased, and he does not appear to have dribbled more than usual. At six months of age he had two slight fits, but they appear to have passed off without leaving any trace of disease. He also suffered from some abscesses during childhood, but the slight

marks left by these show that they were only subcutaneous. With these exceptions the child has enjoyed good health, and it was not until he was two years of age that it was observed that anything was wrong with his legs.

Present condition.—The child is small for his years. He measures 33 inches in length, and 17 inches round his waist. His head is well formed, but small, measuring only 18 inches in circumference. The face is also small, and the boy has a sharp, restless, but fairly intelligent expression. When the child is sitting there is no perceptible deformity of the lower extremities; but when his feet are placed on the ground, the heels become immediately elevated, so that he stands on his toes. The thighs are slightly flexed on the body, and the legs on the thighs; the toes are somewhat inverted, and

Fig. 2.

the knees are strongly drawn together by a spasm of the adductors of the thighs. The boy is unable to stand or walk unsupported, and when he walks by the aid of a support, the knees rub against each other. When the patient

stands or walks, the muscles of the lower extremities, especially the gastrocnemii and the adductors of the thigh, become hard, prominent and tense (Fig. 2); the patellar-tendon reflexes are exaggerated, but ankle-clonus cannot be elicited. The child is able to speak without any stammering, but his speech is drawling, and more like that of a child of two than of a boy of four years of age. There are no sensory disturbances, and the bladder and rectum are unaffected. The accompanying woodcut is from a photograph, kindly taken for me by my friend Dr. Larmuth.

Case X.—Henry F., aged 8 years, entered the Manchester Royal Infirmary on August 1, 1882. His parents are quite healthy. This boy is one of a family of four children, all of whom are healthy, the other three being free from any defect whatever. The mother of the boy has had six miscarriages, some of them occurring at the third, and some at the sixth month. No other evidence of a syphilitic history can be ascertained. The boy was quite healthy as an infant, but at six months of age he began to have fits, and had as many as two or three in the course of a day, and the fingers and toes remained strongly flexed in the interval between the convulsions. It was observed that his head was large as an infant, and he was then supposed to have water in the head.

Present condition.—The boy is well-nourished and healthy-looking. His head is large, measuring 22 inches in circumference, 9 inches in the longitudinal and 6 inches in the transverse diameter; the forehead is projecting; the fontanelles are closed, but depressed; and the eyes are normal. When the boy is sitting, there is no manifest deformity of the lower extremities; but when he attempts to stand or walk, the heels become elevated, and he has to balance himself on his toes. The legs cannot be fully extended on the knees, and the knees are drawn together by a strong contraction of the adductors of the thighs. The thighs are also during station slightly flexed on the trunk, and the toes are inverted. The knees rub against each other when the patient walks. The patellar-tendon reflexes are exaggerated, and a slight ankle-clonus can be provoked on the left side, but not on the right. There are no sensory disturbances and no bladder troubles.

Although the hands were implicated in the spasm when the patient suffered from fits, they are now normal. The feet become dark red on exposure, and are very liable to suffer from cold. The faradic contractility of the muscles affected by the spasm is normal.

The four cases which have just been described, although differing widely from one another in many respects, are good examples of the spastic paraplegia of infancy. The occurrence of spastic paraplegia in infancy is, if I may judge from my own experience, by no means rare. Such cases have been long known to orthopædic surgeons. Even as far back as 1840,[1] Heine described three cases of spinal paralysis of infancy characterised by "a spastic peculiarity of the contracted muscles;" and the accompanying figures, as well as the description of these cases, leaves no room for doubt that they were examples of the disease under consideration at present. The clinical features of spastic paraplegia have also been faithfully described by Dr. Little in 1853,[2] and again in 1862,[3] and by Mr. Adams in 1866.[4]

Stromeyer also in 1864 directed attention to Dr. Little's observations, and described this affection under the name of "permanent tetanus of the extremities in children."[5] In 1877[6] Erb described three cases of this affection; a few months later[7] he reported two other cases; and in 1877, and again in 1880, Dr. Gee communicated several cases of the disease.[8] When Erb assimilated these cases clinically to the spastic paraplegia of adults, he suggested to the mind that the former

[1] 'Beobachtung über Lähmungszustände der untern Extremitäten,' von J. Heine, 1843, p. 61 et seq.

[2] 'Deformities of the Human Frame,' by W. J. Little, M.D. Lond. 1853.

[3] "On the influence of Abnormal Parturition, Difficult Labours, Premature Birth, and Asphyxia Nonatorum, on the mental and physical condition of the Child, especially in relation to Deformities," by W. J. Little, M.D. 'Obstetrical Transactions,' vol. iii. 1862, p. 293.

[4] 'On Club Foot,' by W. Adams. Lond. 1866. P. 46.

[5] Stromeyer's 'Handbuch der Chirurgie.' 2 Bd. 1864. Pp. 17 and 174.

[6] "Ueber die Spastische Spinalparalysie" (Tabes dorsalis spasmodique, Charcot). Virchow's 'Archiv,' Bd. lxx. 1877, pp. 241 and 293.

[7] 'Ueber das Vorkommen der "Spastischen Spinallähmung" bei Kleinen Kindern, Memorabilien aus der Praxis.' Heilbronn, 1877, 12 Heft.

[8] 'St. Bartholomew's Hospital Reports,' vol. xiii. 1877, p. 71, and vol. xvi. 1880, p. 27.

disease is, like the latter, due to a primary sclerosis of the lateral columns of the cord. He is, however, careful not to commit himself to this view, and he has even suggested that the infantile disease may be due to an arrest of development of some portions of the spinal cord, or at least of the nervous system.

But it becomes a question whether the disease is of spinal or of cerebral origin. The paraplegic distribution of the spasm, and the frequent absence of any distinctively cerebral symptom, would seem to indicate that the affection is, sometimes at least, caused by a spinal lesion. But on the supposition that this disease is a spinal one, the questions arise, whether it is due to a congenital absence of some of the fibres of the pyramidal tracts or to sclerosis of them? and, whether the paralysis becomes established before, at, or after, birth? Satisfactory answers to these questions can only be obtained by careful clinical reports with subsequent dissections. It is rather remarkable that in several cases which came under my observation, but of which I have not kept notes, the children were born with the feet first. It has occurred to me that injury might have been done in these cases to the vertebral column and spinal cord by undue traction during delivery. Such an injury might produce the disease in several ways. Injury to one of the vertebræ, for instance, might set up a limited pachymeningitis, with subsequent transverse myelitis, and the latter would cause a descending sclerosis of the pyramidal tracts. Compression of the spinal cord by the formation of a blood-clot between the injured vertebra and the dura mater might lead to a similar result. Transverse myelitis would affect the sensory as well as the motor mechanism of the cord, but it is well known that in the adult, sensory disturbances disappear much more readily than motor disorders; and this would be much more likely to take place in the infant, inasmuch as the pyramidal tracts are only imperfectly developed at birth. That the vertebral column may suffer injury during the extraction of the child at birth has been proved by dissections. A portion of a vertebral column from a child extracted by the feet was exhibited in 1876 to the Obstetrical Society of Leipzig,

by Ahlfeld,[1] showing that the bodies of two vertebræ were divided. The child lived nine days, and the existence of the lesion was not suspected before death. Out of 64 cases of extraction by the feet observed by Ruge, rupture of the vertebral column occurred 8 times. The rupture occurred in the body of a vertebra in the upper dorsal or cervical region, and in the line of the epiphyses; the anterior common ligament was torn, and the spinal cord was always found surrounded by an extravasation of blood at the seat of the injury. It is very probable that no child will long survive injuries so extensive as were found in the cases reported by Ahlfeld and Ruge, but it is quite probable that a slighter degree of injury may be quite compatible with life, or may indeed give rise to no very manifest symptoms.

But it is probable that hæmorrhage of the spinal membranes might give rise to a similar result in the absence of any rupture or other injury of the vertebræ. Out of 161 post-mortem inspections by Weber, of newly-born children from the 'Clinic' of Litzmann,[2] the vertebral canal was opened 81 times, and of these, extravasation of blood was observed 33 times, the hæmorrhage being very extensive in 19 cases. Of the 33 cases in which extravasation had occurred, the blood was found on the external surface of the dura mater alone, 23 times; both on the external surface of the dura mater and the arachnoidal sac, 4 times; in the arachnoidal sac and sub-arachnoid space, once; in the arachnoidal sac alone, 4 times; and in the sub-arachnoidal space once. Extravasation of blood of meningeal origin was also found within the cranium in these cases in addition to that in the spinal canal. A wound of the vertebral canal was not observed in any of these cases; it is therefore manifest that spinal meningeal hæmorrhage is by no means an uncommon occurrence at birth, and it is not to be supposed that all the children must necessarily die of a lesion which does not appear to be a very fatal one in the adult. It is therefore not improbable that some cases of spastic paraplegia may owe

[1] See Litzmann, 'Archiv für Gynäkologie.' Bd. xvi. 1880, p. 97.

[2] "Ein Beitrag zur Kenntniss der Spinaler Lähmung bei Neugeborenen," von C. C. M. Litzmann. 'Archiv für Gynäkologie,' Bd. vi. Berlin, 1880. S. 99.

their origin to a spinal meningeal hæmorrhage. Hæmorrhage into the substance of the spinal cord itself would injure the grey matter, and lead to atrophic paralysis of some groups of muscles, but no atrophy is ever present in the cases under consideration at present.

But it is possible that the spinal cord itself may suffer direct injury during efforts at extraction of the child. Parrot[1] records a case in which a midwife heard, while strongly pulling the feet of a child during birth, a crack which appeared to come from the upper part of the child's body. Death occurred on the sixth day after birth, and at the autopsy the spinal cord was found completely torn on a level with the sixth and seventh cervical vertebræ, a rent being also observed in the membranes on the same level. A clot of blood, which filled the whole breadth of the spinal canal, lay between the divided ends of the cord. It is not very clear whether the vertebral column was injured or not, but it would be difficult to imagine that complete rupture of the cord could occur without simultaneous rupture of one of the vertebræ.

But if it be possible for complete rupture of the spinal cord to take place under any circumstances, surely it is possible for partial rupture to take place. A slight degree of traction upon the spinal cord might injure some of the fibres of the pyramidal tracts, a considerable proportion of these being non-medullated at birth, while leaving the earlier developed fibres of the cord, as well as the grey substance, intact. The peripheral portion of the injured fibres would subsequently undergo descending sclerosis, and thus give rise to the spastic paralysis.

But although some cases of spastic paralysis may be accounted for by injury done to the vertebral column, spinal membranes, or spinal cord itself, during the act of birth, yet all cases cannot be explained in this manner. In many cases of spastic paraplegia the mothers have had a perfectly natural labour, and there can be little room for doubting that the spasmodic condition is fairly established at birth. In these cases we must assume that the lesion is a congenital one, and we

[1] 'Union Médicale,' 1870, Jan. 27, quoted by Leyden, 'Klinik der Rückenmarkskrankheiten.' Bd. ii. 1875. p. 88.

turn for an explanation of such cases to Erb's supposition that the spasmodic condition is due to an arrest of development of certain portions of the nervous system. Now the only portions of the spinal cord, arrested development of which would cause spastic paraplegia, are the pyramidal tracts, and arrest of development of these tracts is well known to occur sometimes. These tracts have been found absent by Flechsig in the spinal cords of anencephalous children; they were imperfectly developed in a case of porencephalus reported by Kundrat already alluded to, and also in the case of porencephalus just described by myself. It would appear, however, that arrest of development of the pyramidal tract is associated in the cases hitherto examined with a congenital lesion in the motor area of the brain, and it will probably be found that these conditions always co-exist. If that be the case, we may expect that at least a considerable proportion of cases of spastic paraplegia are really cases of bilateral hemiplegia, caused by a porencephalous defect of the cortical motor centres of the affected limbs, and arrest of development of the fibres of the pyramidal tract, which would have sprung from these centres. Even a clinical consideration of cases of spastic paraplegia suggests that the brain is implicated in the disease.

Speaking of the more aggravated cases, in which the upper as well as the lower extremities are implicated, Mr. Adams says: "Frequently strabismus, and occasionally wry-neck, coexist with these deformities."[1] "The head," he continues, "frequently exhibits a close approximation in form to that commonly existing in idiots; and imperfect development of the intellectual faculties sometimes accompanies this sad condition. These children have not infrequently a vacant or half-silly expression of countenance, and yet are more intelligent than might be supposed. Their speech is often difficult and imperfect, and in consequence of this, the intellectual faculties are often supposed to be more impaired than they really are."

And even in the minor grades of the affection, when the spasm is restricted to the lower extremities, evidence is not

[1] 'Club-Foot: its Causes, Pathology, and Treatment,' by William Adams, F.R.C.S. 2nd Edition. Lond. 1873, p. 65.

wanting to show that the primary lesion is situated in the brain. In the case of James B. (Case IX.), for instance, the patient may be regarded as a microcephalic idiot; while Henry N. (Case X.), although not now suffering from any cerebral symptoms, had "fits" at six months of age; the fingers as well as the toes were contracted in the intervals between the seizures; and from the size and form of his head he was supposed to be affected with hydrocephalus. Even Wilfred R. L. (Case VIII.), who is a very bright intelligent boy, has an impediment in his speech, and his head is unsymmetrical; while T. M. (Case IX.) has a small head, although it is symmetrical.

The opinion that a large number, if not all cases of the spastic paraplegia of infancy are caused by a porencephalous defect of the cortical motor centres, along with an arrest of development of the corresponding parts of the pyramidal tracts, is here advanced with due reserve. The obtainable evidence only warrants us in regarding such an opinion as more or less conjectural, and we must be content to wait until careful dissections shall enable us to place the pathology of this affection upon a secure basis.

THE PHENOMENA OF ANGINA PECTORIS, AND THEIR BEARING UPON THE THEORY OF COUNTER-IRRITATION.

BY W. ALLEN STURGE, M.D.

In the following paper I shall endeavour to make an analysis of certain of the phenomena met with in cases of angina pectoris, with a view not only to throw some light if possible upon the explanation of angina, but also to carry the process somewhat further, and through it to seek for a solution to certain other problems in nerve physiology.

I will take as my text the description of his attacks given me by a patient who suffered from very typical attacks of angina. This patient, who had no perceptible organic heart-disease, described his attacks as consisting of a sudden severe gnawing pain at the apex of the heart, shooting through to the bladebone and down the left arm to the tips of the fingers, where he felt a strong tingling sensation. The pains were accompanied by palpitations of the heart, and towards the end of the attack by the belching of a quantity of wind. After the attack, there was soreness and tenderness of the skin of the left side corresponding to the seat of the pain. The attacks lasted from five to fifteen minutes. Let us endeavour to ascertain the meaning of this series of phenomena.

In the first place, what in physiological language is the explanation of a sudden severe attack of pain? It means that the person is conscious of an abnormal commotion in the grey matter of the sensory region of the brain, corresponding to the region of the body where the pain is felt. This commotion may have arisen spontaneously in this part of the brain, as is probably the case in most cases of epilepsy associated with sensory phenomena; or it may be due to the transmission

upwards of the results of a commotion in the grey matter of one of the subordinate parts of the nervous system, as for instance in the sensory grey matter of the spinal cord, or, lower still, in the grey matter of the sympathetic ganglia. With these, again, it is also possible that the commotion began spontaneously; or, on the other hand, they in their turn may only be passing onwards a commotion which has begun still further away from the centre, viz. (1) in the trunks of the sensory nerves, or (2), far more frequently at their peripheral extremities, in both of which situations the commotion is rarely spontaneous, but is almost invariably due to something outside the nerve or its extremity, which has brought the nerve into action.

Thus, then, an attack of pain localising itself in the heart may be due in theory to any one of the following causes:—

(1.) Some structural change in the heart substance, or some change in the heart contents, acting upon the peripheral terminations of the cardiac nerves.

(2.) A commotion arising spontaneously in the grey nervous ganglia of the cardiac plexuses of the sympathetic, the commotion being transmitted upwards to the spinal cord and brain.

(3.) A commotion arising spontaneously in the cervical ganglia of the sympathetic which give off branches to the cardiac plexus; or in the ganglia of the pneumogastric.

(4.) A spontaneous commotion in those portions of the grey matter of the spinal cord which are connected with these ganglia by the bands of communication passing from the cord to the sympathetic.

(5.) A spontaneous commotion in that part of the grey matter of the brain which in any of the previous cases would be the recipient of the nervous impulses passed up from below.

These, then, are the various conditions upon which the pain may in theory depend It would be interesting to inquire whether all of them are in practice met with. This, however, would require a long and difficult examination of the evidence derived from a large number of carefully observed cases. I am not sure whether in the present state of our knowledge

such an inquiry is likely to lead to satisfactory results. At any rate, for my present purpose it will be enough to say that in most cases of angina associated with structural disease of the heart or large vessels the primary irritation probably takes place at the peripheral extremities of the cardiac nerves; but that there is some evidence to show that there are varieties of angina beginning spontaneously in the nerve-centres. I shall say little, moreover, as to the *nature* of the commotion set up in the grey matter, beyond stating that it is probably closely allied to that met with in cases of epilepsy, and still more so to that present in the so-called epileptiform neuralgia. The relationship between epilepsy and angina pectoris was long ago pointed out and insisted upon by Trousseau.

Let us pass on to consider another feature of the attack: the pain having begun suddenly in the neighbourhood of the apex of the heart, shot through to the left bladebone, to the left shoulder, and down the left arm to the tips of the fingers.

The radiation of the pain from the limited district in which it began until it extended over a wide area is an evidence of an extension of commotion from one small patch of grey nerve-substance to other parts of grey matter more or less intimately associated with it. An extension of nerve commotion of this kind is a very familiar phenomenon to those who have carefully studied the phenomena of epilepsy. When an epileptic fit instead of destroying consciousness at once begins slowly with the production of an elaborate warning, it is no rare thing to find that a nerve commotion giving rise (say) to a tingling in one finger, gradually spreads until the tingling passes to the other fingers, and thence up the arm to the trunk; thence it may pass up the neck to the tongue, face and head, or downwards to the thigh, leg and foot; bright sparks may perhaps next be seen, or a noise heard in the ears. Then a few muscles may begin to twitch in the hand; the arm may next be drawn up, and at this point perhaps the patient loses consciousness, the commotion extending rapidly until the whole body is convulsed. These various sensations and phenomena following in regular succession do but mark

the extension of the original commotion over a wider and ever more widely extending area of grey nerve substance. This extension depends upon the intimate association of one nerve-cell with others, either in its immediate neighbourhood, or closely connected with it in function, an association established by means of commissural interpolar fibres passing from cell to cell. When one of these cells becomes abnormally excited, as in the case of a slowly beginning fit, the commotion makes itself rapidly felt in the other cells connected with it. These will tend in consequence to become similarly excited; and they, in their turn, will excite others still further removed from the first cell, and so on in endless progression.

So it is in angina pectoris. Whatever may have been the original cause of the commotion, the excitement having once been started in some one of the situations above mentioned, will tend to overflow from the original focus into neighbouring parts. But now comes the question, Why does the excitement adopt the peculiar mode of extension seen in the case of angina pectoris I have quoted, and which, as all will allow who have seen much of the disease, is a very common form of attack? Why, in other words, does the pain extend from the heart region down the arms, and down the left arm rather than the right? What possible nervous communication can there be between the heart and the arms, since the heart is supplied by the pneumogastric and sympathetic nerves, and the arms by the lower cervical spinal nerves?

It is at once evident that no intercommunication between these two regions can take place through the cardiac ganglia, nor through the ganglia of the sympathetic giving origin to the nerves which pass to the cardiac ganglia. Neither do the pneumogastric nerves or their nuclei of origin offer a means of intercommunication. Does the spinal cord offer a more probable solution to the problem? The cardiac nerves of the sympathetic come from the three cervical ganglia on both sides. Of these, the largest nerves are the two nerves which come from the middle cervical ganglia. The strands passing from these ganglia to the spinal cord pass in the trunk of the fifth and sixth cervical nerves; those passing from the inferior cervical ganglia, in the trunks of the seventh and eighth

cervical nerves. It is these four nerves which, in conjunction with the first dorsal, form the brachial plexus. We thus see that the region of the spinal cord which gives origin to the brachial plexus gives origin also to the greater part of the fibres which eventually find their way to the heart. Wherever, then, the original commotion may have taken place in an attack of angina pectoris; whether in the cardiac ganglia, or in the cervical ganglia of the sympathetic, or in the spinal cord; and whether the commotion be due to some peripheral irritation from disease of the heart's substance, or be a spontaneous outburst on the part of the nerve-cells implicated, it is evident that it is only when the commotion has begun in the cord, or has passed up to the grey matter of the spinal cord from the sympathetic, that any great extension in its area can take place such as that of which I am speaking. The next question is: Since the cardiac nerves come from the sympathetic on both sides of the body, and must consequently be connected with both sides of the spinal cord in an equal or nearly equal degree, how is it that in the vast majority of cases of angina pectoris, as in the case mentioned above, it is the left arm alone that is affected, and that affection of the right arm alone is so exceedingly rare, although in some few cases both arms may be involved? The answer to this question is of very great interest, for on it, I believe, depends the explanation of the theory of counter-irritation.

It is well known that in the grey matter of the spinal cord, in addition to the fibres passing from the nerve-cells to form the roots of the spinal nerves, and to those which pass upwards to the brain, there is an extensive system of commissural fibres connecting the two sides of the cord, and connecting also cells and groups of cells together. It is, indeed, upon this intercommunication of groups of cells that reflex action depends. It is no less certain that cells endowed with a similar function, as, for instance, that of motion, but connected with different regions of the body, are more or less intimately associated together. It might at first sight be thought that this grouping of cells of similar function would coincide strictly with the grouping of the fibres emanating from these cells to form nerve trunks, e.g. that the cells whose emerging fibres go to

form the motor part of the musculo-spiral nerve in the arm would form one group; and that those whose fibres go to form the motor part of the musculo-cutaneous nerve would form another. Similarly it might be expected that the cells governing the sensation of those parts of the skin of the arm supplied by the musculo-spiral nerve would be associated together; and that the same would be true of those governing the sensation in the skin supplied by the musculo-cutaneous nerve.

Now it has been shown by Remak, Erb, Ferrier, and others that this is not the case with the *motor* nerves, but, on the contrary, that the grouping of motor cells in the spinal cord depends on the physiological association of muscles in the production of certain movements, and not on the mere anatomical fact of their being supplied by the same nerve-trunk. In a paper I read before the Medical Society of London,[1] I narrated two cases proving the truth of this proposition most conclusively. The patients were suffering from progressive muscular atrophy, involving the arms. In one of them the only muscles involved were the biceps, the brachialis anticus, and the supinator longus—the three muscles, in fact, which are associated together in bending the elbow. In the other case, all the muscles of the arm and hand were completely atrophied *except* these three muscles. Now two of these muscles are supplied by the musculo-cutaneous nerve, and the other by the musculo-spiral.

If this is so with the *motor* functions of the cord, we might expect to find somewhat the same variety of association in connection with the *sensory* functions. In this case we should expect that the cells connected with the sensation of the upper arm, taken as a whole, would be associated in a group together, in spite of the variety of nerves distributed to the skin of this part; whilst, on the contrary, the cells governing the sensation of a scattered district supplied by one nerve, such, for instance, as that supplied by the musculo-spiral nerve, would belong to several groups, rather than be collected into one group. In other words, cells governing the sensation of neighbouring parts of skin would be more or less closely

[1] 'Proceedings of Medical Society,' vol. v.

connected with one another; whilst those supplying patches of skin at a distance from one another would be correspondingly separated.

But if this be true of neighbouring parts on the *surface*, we may expect that it will be equally true of parts which, like the skin and the structures lying *beneath* it, are contiguous to one another; thus we should expect to find that the centres controlling the sensation of any particular portion of skin would be intimately associated with those governing the sensation of the subcutaneous fat, of the muscles, and even possibly of the bones, lying beneath that area of skin. Similarly, that the sensory centres for the skin over any particular organ would be associated more or less closely with those controlling the sensory functions of the subjacent organ.

This theory of association of cells in the spinal cord, according to physiological requirement and correlation, has, as I have already said, been proved to be true in the case of the motor functions, and I believe that the distribution of the pain in angina pectoris proves its truth in connection with the sensory functions.

Let us for the moment grant that it *is* true, and let us inquire what method of distribution of pain we might expect to find in connection with an attack starting in the nervous apparatus of the heart or great vessels, and spreading.

It would first attack the part, superficial to the heart, or great vessels; that is to say, the sternum and ribs and their muscles, and the skin covering them. Having reached the skin, it would tend to spread from one part to another, but this spreading would be more likely to follow certain directions than certain others; for instance, as we have followed the majority of the sympathetic cardiac nerves into the cervical enlargement of the spinal cord, we may expect to find that the pain will be confined to the upper extremities, rather than that it would pass from the skin of the thorax to that of the abdomen. Again, the skin of the left side of the thorax would be much more affected than the right side, because of the situation of the heart to the left, and therefore as the extension of the pain goes on, it will be the left arm that will be chiefly involved. At the same time we should expect that in

severe cases the radiations would cross to the right side, and that the right arm might be affected, though rarely, if ever, to such an extent as the left. We should also expect that it would be exceedingly rare to find that the pain passed to the right arm without affecting the left.

But the original commotion in the cardiac centres will not only affect those parts of the nerve-centres governing the sensations of the front part of the heart or its large vessels, but all parts will partake in it. Consequently the radiation to contiguous districts will take place not only towards the front, but in other directions also. Hence we may expect to find that the patient will complain of pain in the left scapular region, whence also the way is not far to the left arm. The radiation downwards would tend to pass to the stomach, and we might thus look for some affection of that organ. We should be prepared to find also that there would be more or less affection of the lungs.

Let me narrate very briefly the account given of their attacks by a number of different patients. It will be remembered that the patient who has served as the text for this paper experienced a severe gnawing pain near the heart's apex, which extended over the whole cardiac region, shot through to the left bladebone, and down the left arm to the fingers. At the end of the attack he belched a quantity of wind. The attack left a soreness and tenderness in the skin of the left side of the chest. Another patient of mine, suffering from severe aortic valvular disease, had attacks in which he felt a sudden pain beginning over the heart, going back to the left shoulder-blade, and down the left arm to the finger tips, where he had a strong sense of tingling. Sometimes the pain extended also to the right shoulder and down the right arm, but here it never extended farther than the elbow. At the end of the attack, he belched a quantity of wind for a few minutes. Occasionally he suffered from shortness of breath during the attacks.

Another of my patients suffering from aortic valvular disease had attacks which began with a sudden choking sensation at the top of the sternum, with intense dyspnœa; this was followed by intensely sharp pain over the apex of the

heart, which extended to the left shoulder and down the left arm, which felt numb and heavy. After a few minutes he began to belch a large quantity of wind, the eructation continuing for some time.

A fourth patient of mine, a woman without evident organic heart-disease, had attacks, almost always at night, in which she had sudden severe pain in the cardiac region of a shooting or dragging kind, which went down both arms, but down the left more than the right. The hands tingled, and she felt as though the nails were being dragged out. Towards the end of the attacks she belched a quantity of wind. After the attacks the arms felt numb and weak.

Trousseau describes the following cases :[1]—

(1.) A woman with aneurism of the aorta had attacks of sharp sudden pain in the precordial region, radiating to the base of the chest, where it produced an intense sensation of constriction ; thence to the loins, up to the neck, down the left arm to the extremities of the fingers, leaving the arm numb and heavy.

(2.) Another woman, without organic heart-disease, had pain behind the sternum, passing to the left shoulder and down the left arm, causing numbness.

(3.) A man without organic disease had pain behind the lower part of the sternum, passing to the base of the neck and down both arms equally, giving rise to painful numbness.

(4.) A man with aneurism of the aorta had attacks beginning with a sense of suffocation, followed by severe pains behind the sternum, passing to the left shoulder and down the left arm.

(5.) A man without organic disease had attacks of pain behind the sternum, accompanied by intense dyspnœa. The pain passed to both arms, but chiefly to the left, giving rise to numbness. The attack ended with a strong desire to pass water, and with a sense of congestion in the nasal mucous membrane.

In the majority of these cases, then, we see that the attack, beginning in the cardiac region, passed to the left arm alone; in three it passed to both arms; but in only one was the right arm affected equally with the left. In no case was the right

[1] 'Clinique Médicale,' 4ème édition, vol. ii. p. 527 *et seq.*

arm alone affected. Trousseau mentions a remarkable case in which the right arm was affected and not the left; but in this case the pain was entirely confined to the *right* side of the chest, and hence can scarcely be looked upon as one of ordinary angina pectoris, the case being probably one of epileptiform neuralgia of certain intercostal nerves of that side.

What evidence is there to show that the pain in the cardiac region was situated in the skin, covering that region as well as in the heart or its dependencies? An answer to this question is of importance, if my theory be correct that the pain passes from the deep organ to the superficial structures over it, and thence spreads along the skin to the arm, &c.

In the first place, I would quote the statement of my first-mentioned patient, who said that the skin over the heart was sore and tender to the touch after the attacks. Does not this recall at once the tenderness over a neuralgic district; and, on the other hand, if the intercostal nerves had been unaffected, why should the skin be tender? Again, Trousseau describes the case of a man with an aortic aneurism subject to attacks of angina pectoris, who after his attacks had *numbness of those parts of the chest where he had experienced the pain.*

In the next place, I would quote the description so often given by patients, and upon which Trousseau and other authors lay so much stress, viz. that even where there is no actual dyspnœa, the patient ceases at the height of the attack to draw breath, or draws it as lightly as possible, on account of the pain that it gives him to do so. Does this description not resemble exactly the condition of a patient with severe intercostal neuralgia—severe pleurodynia?

It will be seen that in several cases the patients made special mention of pain in the bladebone following on that in the precordial region. Walshe speaks of "pain shooting to the mid-dorsal spine" as not infrequent. This would appear, therefore, to be a radiation independent of that over the præcordium, and if this be so we should be justified in referring it to an extension from that portion of the cardiac centres of the spinal cord governing sensation in the back of the heart.

In all my cases gaseous eructation from the stomach

was a marked feature, and most authors who have treated of angina mention it as a frequent symptom. This is the most usual form of implication of the stomach, and it is one of very great interest, as proving conclusively the influence of the nerves of the stomach over the secretion of gas in that organ. Just as the connection between the heart and its superficial neighbours is evidently established through the sympathetic nerves passing to the cervical spinal cord, so here the connection between the heart and its deep neighbour the stomach is probably made through the pneumogastric nerves. If therefore there be a form of angina having its origin in the cardiac ganglia, and another form having its origin in the centre in the spinal cord controlling the heart, as is by no means improbable, we should expect that the former variety would be accompanied by stomach disturbance, and that the latter would not. The materials at my disposal are not sufficient to enable me to treat further of this point; but it will be an interesting question to ascertain whether the anomalous cases of angina not connected with organic cardiac disease, and not following the more usual methods of distribution (some of which cases may be supposed to arise in the spinal centres), present the symptom of eructation as frequently as the more typical cases of angina, or those definitely connected with heart-disease. This symptom of gas secretion under purely nervous influence is a most curious one, and worthy of further examination. I do not propose, however, to go further into this branch of the subject on the present occasion, beyond calling attention to the probable connection between the gaseous eructation of angina pectoris and the remarkable eructations of gas from the stomach in certain hysterical patients; the well-known phenomena of phantom tumour; the eructations which follow the fit in some cases of epilepsy; and probably also certain forms of flatulent dyspepsia in neurotic subjects.

As regards radiation of the nerve-commotion from the cardiac nerve-centres to those of the lungs, this is less frequently seen. In one of my cases dyspnœa was a prominent feature, and it was so, moreover, in two of the cases narrated by Trousseau. Walshe says that real dyspnœa is exceedingly

rare in angina pectoris; but in my own case above mentioned, there could be no doubt of the presence of real and most urgent dyspnœa.

The infrequency of lung symptoms may be taken, I think, as an indication that the affection of the stomach in angina pectoris is due to the close connection of the cerebro-spinal centres controlling the heart with those controlling the stomach, and that this connection depends upon the close proximity of the organs in the body, rather than upon the fact that both organs are supplied by the same nerves. Were it true that the almost constant association of symptoms in the heart and stomach in angina pectoris is due to the latter cause, it would be difficult to explain why the lungs, which also derive a large part of their nerve supply from the same nerves, are so very much less frequently affected. If, however, the explanation I have offered be the true one, then we should expect that, as by far the larger part of the lungs is situated at a distance from the heart, the communication between the heart and lung centres in the cerebro-spinal axis would not be particularly close, and that the lungs would only be affected in special cases.

In some of the more severe cases the radiation is much more extensive than to the back, left arm, stomach or lungs. We have already seen that it may go to the right arm; but it may also go up to the head down the trunk, and even to the lower limbs. In one of Trousseau's cases above described it affected the bladder and the nasal mucous membrane.

One of the most instructive phenomena of radiation to my mind is the sense of intense constriction, which is occasionally complained of. One of Trousseau's patients described it as being like a bar of iron violently tightened round the chest. Does this not at once recall to our minds the *douleur-en-ceinture* of spinal-cord disease which we have every reason to believe to be due to the involvement of the sensory centres of the intercostal or lumbo-cutaneous nerves? Its presence in angina pectoris is a fresh proof, if such be required, that wherever in angina pectoris the original irritation may have been, the seat of the commotion of which the patient is conscious is in the spinal cord. It shows, moreover, the inti-

mate connection between the spinal centres of deep organs, and those for the superficial structures covering them; for there is much less evident nervous connection between the cardiac ganglia and the intercostal nerves, than between these ganglia and the arms; yet we see that a commotion in the spinal cardiac centres may propagate itself rapidly to the centres from which emanate the sensory fibres of the intercostal nerves.

And now for the bearing of these remarks upon the theory of counter-irritation. When we use the actual cautery to the skin over the spines of the vertebræ in a case of disease of the spinal cord, what are we doing? We are producing a profound impression upon the peripheral endings of the nerves of the skin over the diseased part. We produce, moreover, a moderate degree of inflammation in the skin and parts immediately subjacent to it; but considering the depth at which the diseased part lies below the surface, we cannot imagine that the direct effect of the burn reaches so far inwards. Neither is it possible to suppose that the small amount of blood drawn to the skin by this slight degree of circumscribed inflammation can modify in any perceptible degree the amount of blood in the deeply subjacent organ. Can we not from our knowledge of what takes place in angina pectoris deduce an explanation of the effect of counter-irritation? An analysis of the phenomena of angina pectoris showed the intimate relationship which exists between the nerve-centres governing adjacent structures. We saw there how an irritation at the periphery of the nerves governing the deeply-seated organ, an irritation arising from heart-disease, aneurism of the aorta, &c., caused a commotion in the spinal centres with which those nerves are connected; that this commotion rapidly passed to centres from which emanate the nerves supplying the parts superficial to that organ, and that in these centres also a violent storm arose.

Can we not easily imagine the reverse; that an irritation of the peripheral endings of the nerves supplying the superficial structure, should set up a commotion in the spinal centres to which these nerves pass; that this commotion should extend to the closely-related centres governing the

subjacent organ or structure, and should profoundly affect this structure?

As a matter of strict fact this reverse process does sometimes take place in connection with the heart. Let us refer once more to Trousseau's masterly lecture on angina pectoris. One of his patients without organic heart-disease, but with a gouty history, had attacks *beginning in the left arm* and passing upwards to the chest, where the pain was chiefly confined to the cardiac region; here the pain was so intense that it seemed to the patient himself as though he must die. The attacks lasted three minutes, and in all points resembled attacks of angina pectoris, with the exception that the radiation of the pain took place in an inverse order. Another patient, without organic heart-disease, but who was also gouty, had attacks beginning in the muscles of the left arm, and thence passing to the cardiac region. In a third case the pain began in the two shoulders, and rapidly passed to the neck, to the tongue, to the arm, and to the chest. Here also there was gout, but no organic heart-disease.

In a fourth case the pain began in one of the dorsal vertebræ. It passed thence to the left arm, and finally to the cardiac region. No organic heart-disease was discovered in this case.

These cases, it is true, were not traceable to any morbid condition of the peripheral ends of the nerves of the arm, the primary commotion probably taking place in the spinal centres governing the sensation of the arm; but they serve to show how readily a commotion in these centres passes to the centres governing the cardiac region; and if a commotion beginning spontaneously in the spinal centres for the arm can thus so readily be transmitted, a similar commotion of these centres, induced by peripheral irritation, would do so with equal ease.

But if I am correct in believing that the evidence derived from an analysis of the phenomena of angina pectoris and allied conditions points to an intimate communion in the spinal cord between the sensory centres for superficial parts, and those for subjacent organs or other structures, ought we

not to find corroborative evidence of this fact in other physiological or pathological conditions?

It is less easy than might at first sight appear to get such evidence; since severe superficial localised lesions, not in themselves affecting deeper structures, are not very common. The following conditions must not, however, be passed over, as at any rate suggestive of an action of this nature.

(1.) The occurrence of deep-seated inflammations from surface chills. In most instances of this kind it is quite impossible to believe that the organs themselves are affected by the chill, as for instance when the kidneys, wrapped up as they are in their thick coatings of fat, &c., take on acute inflammatory action after exposure to a cold wind. In these cases the inflammation is probably produced through the mediation of the nervous system; and there can be little doubt that the peripheral sensory nerves take an important part in the process. As, however, it is very exceptional for the initial chill to be local in its action, it is difficult to be sure that the deeply-seated inflammation has been induced by the action of the chill on the overlying parts. We, nevertheless, tacitly by our action, admit the probability of this connection; for the man with a weak chest is warned to take especial precaution in wrapping up his chest; the man with delicate kidneys is made to wear a flannel-band round the abdomen; "cholera-belts" are served out to troops in warm climates, and so on.

There is one instance of a deep-seated inflammation occurring from a local chill which is, I believe, quite confirmatory of the views above stated. I refer to the inflammation of the facial nerve in the Aquæductus Fallopii resulting from exposure of the cheek to a draught. It is difficult to believe that the nerve lying deeply in and beneath the parotid gland can itself be sufficiently affected by the chill to induce inflammation. The inflammation is probably strictly analogous to that met with in other deeply-seated structures as the result of chill; and in this case the relation between the seat of the chill and the inflammatory effect is a very definite one. I am aware that I am entering upon debatable

ground, as to the respective parts taken by the central nervous system and sympathetic and vaso-motor systems in the production of inflammation. Nevertheless, I would offer these as suggestions, in the hope that those more competent than myself to deal with these difficult questions may be able to throw further light upon the subject.

There is one curious fact which I would mention in this connection, viz. the occurrence of duodenal ulcers in cases of surface burns. Having no good medical library within reach, I am unable to ascertain in what class of burns these ulcers have been chiefly met with. If, as I believe it has been stated, they occur in connection especially with superficial burns of the abdomen, may we not look upon their occurrence as another instance of the effect upon deeply-seated organs of superficial lesions over the organ?

(2.) I now pass to another point. If the radiation of the pain in angina pectoris, and the effects of counter-irritation over deeply-seated morbid structures, be both of them due to sensory co-ordination in the spinal cord, can we, by a study of the former, ascertain any facts which may help us in understanding the modus operandi of the latter?

It is exceedingly common to find in patients subject to attacks of angina pectoris extending to the left arm, that the arm remains in an abnormal condition for a longer or shorter time after the attack is apparently over. The most usual description is that the arm is numb—the sensation to touch is dulled. Frequently also there is a subjective "numb-feeling," i.e. not only is the sensation to touch dulled, but the patient has the positive sense of numbness—as one of my patients described it, a "buzzing" in the limb, because it seemed to him so allied to buzzing in the ears. Then again there may be the opposite condition to loss of sensation, viz. pain; either spontaneous, or developed by pressure—soreness. Thus, one of Trousseau's patients suffered from "painful numbness" in the left arm after the attacks; and on the other hand the patient who served as my text, had soreness and tenderness in the left side after his attacks.

(3.) A third variety of after-effect is the sense of weakness or "heaviness" that some patients experience in the arm. And

lastly, in one of Trousseau's patients the left arm became pallid, and subsequently of a bluish tinge.

Let us translate these various descriptions into their physiological equivalents. Loss of sensation in such a case means that the sensory nerve-centre implicated (in all probability the spinal centre) is exhausted by the nerve storm through which it has passed to such an extent, that it is no longer capable of acting fully under ordinary stimuli; and observe, that in not a few cases it is the centres to which the storm has passed from the primary source of commotion which give evidence of this great exhaustion.

The positive sensation of numbness, the "buzzing" in the limb, must mean that the commotion does not at once die down, but goes on for a time in a quiet manner; sufficient to attract the patient's attention, but not enough in most cases to amount to actual pain. The positive sensation is generally accompanied by more or less exhaustion of the centre, as is shown by the presence simultaneously of dulness of sensation to touch.

Soreness or tenderness mean that the centre is not exhausted, but is, on the contrary, left by the attack in an irritable condition, so that ordinary stimuli produce an over-action in the centre. The sense of weakness or heaviness in the limb shows that in those cases in which it is present, the whole nervous apparatus of the limb is for the time disorganised, the motor functions suffering as well as the sensory. It would be interesting to enquire into the mechanism by which this modification in the motor functions is produced, but I do not propose to do so in the present paper.

In order to apply the above rough accounts of what probably takes place, so as to give a full explanation of the action of counter-irritants, it would be necessary to have a clear knowledge of the influence of the central nervous system upon the production of inflammation. My knowledge of this branch of the pathology of inflammation is insufficient to enable me to do this, but I would make the following suggestions:—

(1.) Whatever may be the influence of the nervous system over inflammation, it is probably an *over-active* or *irritative* influence rather than the contrary.

(2.) Two methods suggest themselves to combat this over-activity. In the first place, we endeavour to soothe it; but failing in this, we endeavour to tire out the centre more rapidly than it would tire of itself.

(3.) Since in the case of deeply-seated inflammations we cannot act directly on the centre by stimuli, we act upon those centres with which it is intimately associated; stimulating these powerfully, in the expectation that they will pass on the commotion induced in them to the centre upon which we wish to act.

(4.) The centres most intimately associated with that governing the inflamed part are those controlling the tissues in the neighbourhood of the part, and hence we stimulate the surface with a view to acting on the organ lying beneath it.

(5.) The centre controlling the inflamed part being thus goaded to further action, rapidly becomes exhausted, and thus its irritative action ceases.

(6.) We have seen that even after so severe a commotion as that present in an attack of angina pectoris the centres are not always exhausted to the point of ceasing to be excitable, but on the contrary are occasionally left in a state of irritation. I believe that the analogue of this is now and then met with in counter-irritation. A patient was under my care suffering from long-standing spastic paraplegia, associated with spinal pachymeningitis. The actual cautery was freely applied on several occasions with great ultimate benefit, but the first effect of the cautery was to increase markedly the spastic symptoms (i.e. the symptoms of irritation), which after a few days diminished, until they were considerably less than before the application.

(7.) I have hitherto said nothing of the vaso-motor nerves, which play so important a part in inflammation. Without pretending to have much knowledge of the mechanism by which the vaso-motor nervous system acts in inflammation, I believe I shall be correct in saying that the influence of the central nervous system on this process is largely exerted through the mediation of the vaso-motor nerves, and the action of the latter has to some extent been presupposed in speaking

of that of the former. The profoundly intimate connection between the central nervous system and the vaso-motor system is a matter of every-day observation. In this connection the observation of Trousseau already mentioned is of great interest, viz. that in one case where the pain of angina pectoris radiated to the left arm, the arm became first pallid and then bluish. This indicates most clearly the close connection that subsists between the spinal sensory centres for a region, and the vaso-motor centres for the same region.

In the above account I am aware that I have touched upon much debatable ground; where, moreover, a far greater knowledge than I possess would be necessary to do full justice to the subject. It is quite certain that much has yet to be learnt in connection with many of the points I have mentioned, and my object in writing this paper has been to offer a few hints as to the directions in which investigation is likely to lead to successful results.

SOME STATISTICS OF CHOREA.

BY ANGEL MONEY, M.D.

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Dr. Gowers suggested to me that in the records of University College Hospital there would be found a number of cases of Chorea Sancti Viti which had up to the present never been worked up. The readers of this article will not fail to perceive that the method so completely followed by Dr. Gowers in the tabulation of his large number of cases of epilepsy has been applied to the statistics here presented; with one or two exceptions, the shape the following tables have taken was drawn up by Dr. Gowers himself. The bulk of the cases here used come from the case-books of the various physicians, past and present, to University College; the remainder have been culled from the practice at the Hospital for Sick Children. For permission to make free with the notes, I sincerely thank the present physicians of both hospitals. I have been led by my study to form no peculiar views concerning the malady. Dr. Sturges has advanced the notion that Chorea may be thought of as a sample of *Dissolution* in the sense in which Dr. Hughlings-Jackson has used that term, and which was borrowed from Herbert Spencer.

If Dr. Sturges think that the restlessness of infancy be comparable to chorea, he must be prepared to explain away certain objections which, nevertheless, I do not say really invalidate his theory. With the roll of the tide of infant restlessness the babe gathers strength; such is not the case with sufferers from chorea. I have not the least bias against the notion that the disease is a *functional* one; but there must be an anatomical basis for chorea, just as there is a proper structure corresponding to healthy movements of all sorts. This, anatomy

has up to the present defied to observation, but it certainly exists, even if it be ultra-microscopical.

Total number of Cases.—Sex.—Age.—Number of Attacks.

The total number of cases collected was 236; of these 214 were available as regards sex, 52 were males, 162 females. The age was available in 186 cases:—1 was 4 years old, 3 were 5, 6 were 6, 15 were 7, 13 were 8, 20 were 9, 21 were 10, 20 were 11, 15 were 12, 17 were 13, 10 were 14, 13 were 15, 9 were 16, 6 were 17, 5 were 18, 3 were 19, 6 were 20, 1 was 21, 6 were 22, and there were 14 above this age. In 197 available cases there were 131 who had 1 attack, 46 who had 2, 15 who had 3, 1 who had 4, 1 who had 5, and 3 who had more relapses than this.

Month of the Year.

With regard to the month of the year when the case first came under observation, there were 30 in January, 16 in February, 23 in March, 27 in April, 20 in May, 25 in June, 11 in July, 11 in August, 11 in September, 17 in October, 16 in November, 17 in December; these, re-arranged according to quarters, give 69 for the first quarter, 68 for the second, 33 for the third, 50 for the fourth. It is interesting to remark the great falling off during the warmer months of the year; what the explanation of this is, however, is not to be too hastily concluded.

Rheumatism.

Of 214 available, there was a history of genuine rheumatic fever in 33, of *rheumatism* in 23, and of very doubtful rheumatic history, 9; so there was an undeniable rheumatic history in about 16 per cent., a possible rheumatic history in about 29 per cent., and the utmost that can be made of rheumatism is 32 per cent.

Heart-disease.—So-called functional murmurs.

There was real heart-disease in 31 cases, or about 13 per cent.; 29 were cases of mitral disease, 2 of aortic regurgitation; no distinction is drawn betwixt mitral obstructive and mitral regurgitant, purposely. There was a systolic murmur

in 64 cases, or about 23 per cent.; this was apical in all the cases except 11.

Cause assigned.

The cause assigned (214 cases available) was fright in 60 cases; accident in 13 (a distinction of doubtful value has been made betwixt psychical and physical shock); hard school-work in 9 (none of these cases come from the University case-books); anxiety and worry, 6; imitation, only 2; want of food, 2; sore thumb, 1; exposure, 1; no assignable cause, 120.

Heredity.

With regard to Heredity (214 available), there was a rheumatic history in 28: 9 times the father was rheumatic; 9 times the mother; 3 times the father and mother; once the mother, brother, and sister; once mother and brother; once father, brother, and sister; once mother and mother's brother; once the grandmother; twice the brother. There was a history of chorea in 14: 3 times the father; thrice the brother; thrice the sister; once the sister and cousin; once the niece; once the mother, father, and brother; once the mother's sister; once the mother's brother. There was a history of fits in 6: twice in the mother, once in the father, once in the brother, once in the sister, once in the father's mother. There was a history of rheumatic gout in 5; 4 times it was the father, once the father and father's mother. There was a history of megrim twice; once in the mother, once in the father and others. Gout was recorded twice; once with the father, once with the aunt. Epilepsy was noted twice; once in the father, once in the uncle. Convulsions were registered once in the brother; once the brother was an imbecile, once the brothers and sisters had suffered from "head affection," once the father had delirium tremens, and once the mother was a drunkard. Several of these collateral diseases were thus associated: once the father had rheumatic gout and rheumatic fever; once the father had rheumatic gout, and the mother and brother had rheumatic fever; once the father had rheumatic gout and epilepsy; twice the father had had rheumatic fever and chorea; once the father and mother had had rheumatic fever and the brother chorea; once the father had rheumatic fever and the niece chorea; once

the mother had rheumatic fever, and mother, father, and brother had chorea (this and the two next are highly pertinent); once the mother had rheumatic fever and her sister chorea; once the father had had chorea and the brother was insane.

Starting-place.

The chorea was said to have begun 6 times in the right hand, 6 times in the right arm (=12 times in right upper limb); 6 times in the left hand, 6 times in the left arm (= 12 times in left upper limb); once in the right leg, twice in the left leg, twice in the left face, 3 times in the right arm and leg, once in the left arm and leg, thrice in the head, twice in the speech, once in the legs, once in the arms, once in the speech and hands. These are the only cases out of 214 in which the site of origin of the disturbance was given.

The chorea started 33 times in the right side, 30 times in the left; it was worse 55 times in the right, 49 times in the left. There was hemichorea 3 times in the right, and 4 times in the left.

The march of the Chorea.

In 24 cases a description of the progress of the chorea from part to part was given. In one case the movements began in the right arm, then went to leg, then to left leg and arm; in another it commenced in the left arm, then turned to leg, then to right hand, and on to right leg and face; in another, it first struck the left leg, then affected speech, and lastly, both hands; in another, the left face was first affected, then the hand and leg, and afterwards the right side and speech; in one case the head first moved, then the left hand, left leg and right arm in succession; again, it was the left arm and leg before the right; in another, the right arm and leg first suffered, then the speech; in one, the right-hand disturbance was followed by that of the arm, leg on the same side, and then the opposite arm and leg; in the ninth instance, the left hand was the first troubled, then the leg, and then the face; in the next, it was the left arm, and afterwards the leg; in the next, the right hand shook first, then the right leg, then the opposite limbs; in another, the left arm was jerked, and later, the face, and latest, the leg; yet again, it was the legs which

first shuffled, then the hands, and then the arms; in another instance, the right arm was first fidgety, then the right leg; in the fifteenth case, the left hand first fumbled, then the arm, and then the legs were affected; in the next, the right hand first dodged about, then the leg, and then the left side; in another, it was the head and then the arms; in another, the left face twitched, and afterwards the left arm and leg; again, it was the right brachial which preceded the right crural disturbance; in yet another, the arms were first awkward, then the legs, and finally the head; the right leg, arm, face, and left side, was the order of succession in another; the head, hands, feet, in another; the left limbs before the right in the last but one; and in the last, the passage was from left leg to left arm, to left face. These are the only cases which describe the course of the chorea from part to part. I may as well state, once for all, that the material here used is not to be implicitly relied on; but, I think, it may be used with a certain amount of trust and reserve. One thing seems to come out of this small number of cases, viz. that when the disturbance of motion starts unilaterally in a particular region, it will travel over the whole of that one side ere it pass to the opposite. Generally speaking, my notes show that the side of the body first affected was the worse; but this was not always the case.

Duration.

In 19 cases the affection lasted 1 month; in 66, two months; in 33, three months; in 18, four months; in 7, five months; in 9, six months; in 1, eight months; in 1, nine months; in 1, eleven months; in 5, one year; in 4, two years; in 1, three years; in 5, *many years*. The above estimate is probably in many instances too short, as many patients left the hospital still showing some movements.

Duration qua *Age.*

The patient being under 7 years of age, 4 cases lasted two months; and 4, three months: the patient being 7 years old, 1 case lasted one month; 7 cases, two months; 4 cases, three months; 2 cases, four months. These statements will serve to explain the following table:—

Duration.	Age in Years.												
	Und r 7	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	—20.	20 +
1 Month ..	..	1	1	1	3	1	2	2	..	2	1	4	..
2 Months ..	4	7	3	6	4	6	5	5	7	5	4	10	2
3 „ ..	4	4	5	7	5	4	3	2	2	1	2	3	2
4 „ ..	..	2	1	3	3	2	2	1	2	3	1	..	..
5 „ ..	..	..	..	1	..	..	2	2	..	2	..	..	..
6 „ ..	..	..	1	..	..	3	..	1	..	1	..	..	..
Over 6 Months	..	..	..	..	..	..	1	1	..	..	..	..	..
Over 1 year ..	1	..	1	1	3	3	2	1	1	..	..	1	..

Duration qua *Fright.—Rheumatism and Heart-disease.*

In 8 there was a history of fright, rheumatism, and the patient had heart-disease, and the disease lasted under three months; in 1, with a similar history, the disease lasted under six months; in 1, with the same history, it lasted over six months, but under a year.

In 6 there was a history of rheumatism, and the patient had heart-disease, but no fright, and the malady was over in less than three months; in 5, with same history, it endured under six months; in 1, under same history, it continued over six months.

In 10 cases there was a history of rheumatism and fright, but no heart-disease, and the affection lasted under three months; in 4 it continued for less than six months.

In 23 cases with rheumatic history alone, the disturbance lasted under three months; in 5, less than six months; and in 1, longer than six months.

In 7 instances where heart-disease only existed, the chorea was over in less than three months, and once only lasted less than six months. In 2 cases where there was a history of fright with heart disease, the chorea finished up in less than three months. In 21 cases in which only fright existed in the history, the disease lasted less than three months; in 8, it went on for less than six months; and in 4, for over six months.

In 51 cases in which there were neither heart-disease, nor rheumatism, nor fright, the malady ran its course in less than three months; in 15, in less than six months; and in 1 in less than a year.

Intervals between Relapses.

Five times the interval between the first and second relapse was under half a year; 5 times it was half a year; 13 times, one year; 15 times, two years; 3 times, three years; 5 times, four years; twice, five years; once, six years; twice, eight years = 51 cases in which relapse occurred once. Three times there was an interval between the second and third attacks of less than half a year; 3 times half a year, 6 times of one year, 4 times of two years, once of five years, once of six years = 18 cases of a second relapse. Once there was an interval of half a year between the third and fourth attack; once of a year, twice of two years = 4 cases of a third relapse. Once there was an interval of less than half a year between the fourth and fifth attack; once of half a year, once of a year = 3 cases of a fourth relapse.

Intervals after Fright.

The St. Vitus's dance began immediately after the fright in 9 females and 1 male = 10. Two of these began in the right side, and 1 in the left (the rest not available in this regard). The chorea began one day after the fright in 3 female cases; it began two days after in 1 female case; it began one week after in 1 female and 1 male case; two weeks after in 2 females, three weeks after in 1 female, one month after in 2 females.

TABLE showing the relation of Male and Female Cases with any combination of Rheumatism, Fright, and Heart-disease. R = Rheumatism; F = Fright; H = Heart-disease; O means nothing; M = Male; F = Female.

Age.	R. H. F.		R. H. O.		R. O. F.		R. O. O.		O. H. O.		O. H. F.		O. O. F.		O. O. O.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Under 7	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	5
„ 7	..	1	..	..	..	..	..	1	..	2	..	..	..	1	..	7
„ 8	..	1	..	1	..	..	..	1	..	..	..	..	..	2	1	6
„ 9	1	..	..	1	..	..	..	1	..	1	..	..	..	1	3	5
„ 10	..	..	..	1	..	2	2	2	..	1	..	..	..	3	1	3
„ 11	..	1	1	..	1	2	1	2	1	..	..	..	..	3	1	4
„ 12	1	..	..	1	..	2	1	2	..	1	..	..	..	1	2	7[1]
„ 13	1	..	..	1	..	..	1	2	..	1	..	1	1	4	1	3
„ 14	1	..	..	..	..	..	..	..	..	..	..	..	..	1	1	5
„ 15	..	..	..	..	..	..	1	3	..	..	..	..	..	3	..	7
„ 16	1	..	..	..	..	1	1	2	..	..	..	..	..	3	..	..
„ —20	..	..	..	..	..	3	1	3	..	1	..	1	..	2	..	7
„ 20+	..	..	..	..	..	..	..	2	..	..	..	..	..	6	4	6

[1] As an example, it will be understood that 7 females of the age of 12 had no heart-disease, no history of fright or rheumatism.

TABLE showing the Month at which the Disease came under Observation, and the Presence or Absence of Heart-disease and Rheumatism. The letters mean as in the previous Table.

R. H.		R. O.		H. O.		O. O.		
M.	F.	M.	F.	M.	F.	M.	F.	
[1]2	1	1	4	..	2	1	17	January.
1	..	..	1	..	..	3	7	February.
..	2	..	7	..	..	1	13	March.
..	2	4	4	..	1	5	10	April.
..	2	..	3	..	..	2	7	May.
1	..	2	2	..	..	1	10	June.
..	1	..	..	..	3	1	6	July.
..	..	..	1	..	..	4	5	August.
..	..	3	1	..	..	2	3	September.
2	2	..	3	..	..	1	7	October.
..	..	1	2	..	..	4	7	November.
1	1	..	2	..	..	3	10	December.

Re-arranged thus—

[2]6	13	2	42	1st quarter.
5	15	1	35	2nd „
1	5	3	21	3rd „
6	8	..	32	4th „

[1] *E.g.* 3 cases (2 males, 1 female) who had heart-disease and had had rheumatism were affected in January.

[2] There were 6 cases with heart-disease and rheumatic history affected with chorea in the 1st quarter of the year.

TABLE showing Number of Cases with or without History of Rheumatism and Fright, qua Age and Sex.

	R. F.		R. O.		F. O.		O. O.	
	M.	F.	M.	F.	M.	F.	M.	F.
Under 7 Years ..	..	..	..	..	..	2	1	5
„ 7 „ ..	..	..	..	1	..	1	..	9
„ 8 „ ..	..	1	..	1	..	2	1	6
„ 9 „ ..	..	1	1	1	3	1	4	6
„ 10 „ ..	..	2	2	3	3	3	1	3
„ 11 „ ..	2[1]	2	1	3	1	3	1	4
„ 12 „ ..	1	2	2	2	1	1	2	7
„ 13 „ ..	..	1	2	2	..	5	1	4
„ 14 „ ..	..	..	1	..	1	1	1	6
„ 15 „ ..	..	..	1	3	..	3	..	8
„ 16 „ ..	..	1	2	2	..	3	..	1
„ —20 „ ..	..	6	1	3	1	2	..	4
„ 20+ „ ..	..	..	..	1	..	5	..	2

[1] *E. g.* there were 4 cases (2 males, 2 females), aged 11 years, who had a history of both rheumatism and fright.

Heart-disease: Time; Relation to Chorea.

The heart-disease preceded the chorea certainly in 7 cases (6 mitral regurgitation, 1 aortic and mitral regurgitation); in 18 cases it was doubtful; it was probable in 5 cases (mitral regurgitation, 4; double aortic, 1); the heart-disease appeared after the chorea in one case.

Qua *side.*

In 8 cases the right side was affected when the patient had heart-disease; in 7 cases the left.

When the heart-disease certainly, or probably, preceded the chorea, the right side was affected thrice, the left twice.

(Only 5 out of the 12 cases show which side was most affected.)

At different Ages.

From five to ten years of age there was heart-disease in 12 cases, no heart-disease in 56.

From eleven to fifteen years of age there was heart-disease in 12 cases, none in 67 cases.

Over fifteen years of age the heart was diseased in 7 instances, not diseased in 45. The heart was affected therefore 31 times out of 168 available cases.

Qua *Relapse.*

There was a relapse of the chorea without the presence of heart-disease in 48 cases; there was no relapse with the presence of the heart-disease in 17 cases; there was relapse and heart-disease, 14 times.

Fallacies here are great—e.g. one cannot say whether a relapse might not occur.

Fright qua *Relapse.*

There was a relapse of the chorea and a history of fright in 25 cases; there was a history of fright and no relapse in 42 cases; there was a relapse and no history of fright in 41 cases.

Rheumatism qua *Relapse.*

There was a relapse and a history of rheumatism in 19 cases; there was a history of rheumatism and no relapse in 45 cases; there was a relapse and no history of rheumatism in 45 cases.

In thus presenting these statistics to the medical profession, I am well aware that they may be thought to be very uninteresting, that is because I have not been able to draw from them any very notable general principles; but they are of value, if only in a negative way, and this must be my reason for publishing them.

HEMIPLEGIC MUSCULAR ATROPHY OF PERIPHERAL ORIGIN.

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Apart from mere functional disturbances, either of exaltation or depression, to which many convulsive and paralytic symptoms may be ascribed, resulting from peripheral lesions of nerves, it is established that lesions of nerves may also induce anatomical changes in the nerve-centres. And the experimental induction of such changes promises to be one of the most reliable methods of determining the position of the various sensory and motor centres and tracts in the brain and spinal cord. It has been shown by the investigations of Dickenson, Vulpian, and others, that as the result of section of nerves, and in cases of long-standing amputation or congenital absence of a limb, certain centres and tracts in the spinal cord and brain undergo atrophic degeneration. In these cases there seems to be mere simple atrophy, without indications of inflammatory action. In the spinal cord the wasting is visible both in the white matter, particularly the posterior columns, and also in the grey substance corresponding to the attachment of the nerves in question. In the cerebral cortex, also, atrophy has been observed in the motor regions, corresponding to those movements which have been long lost.

The effects on the nerve-centres of irritation or inflammation of peripheral nerves are, however, of much greater pathological importance. Under this head come the so-called reflex paralyses, the pathology of which has been the subject of much discussion. It has been found experimentally, and also in man, that in connection with severe injuries of viscera and

traumatic lesions of nerves, paralysis may occur in regions not directly related to the nerves actually injured. These paralyses come on at once, and subside mostly with the irritation which has induced them. Many such facts have been recorded by Weir-Mitchell, and others. It is obvious in such cases that the paralyses are of a purely functional nature, as they occur before any organic changes could possibly be induced in the nerve-centres. What is the exact nature of the functional disturbance is a matter more of speculation than actual demonstration, and the theory of vascular spasm of Brown-Séquard, or the theory of exhaustion of Jaccoud, may be regarded as more or less satisfactory.

It is otherwise, however, when the symptoms of morbid affection of the nerve-centres do not show themselves at once, but at some considerable interval after the infliction of the injury. The cases of reflex paraplegia which have been so often seen in connection with affections of the bladder and prostate in man, and the uterus in women, seem to be of a different type from the above; and the views that the paralytic affections in these cases are not merely functional, but due to actual organic changes in the spinal cord, propagated along the nerves of the diseased viscera, have received much support from experimental and pathological investigation of recent years.

Inflammation of peripheral nerves induced in the lower animals by irritants, mechanical lesions and the like, has by some observers been found to be altogether negative as regards propagation to the spinal cord. But others (Hayem, Klemm, Niedieck, &c.) have described both meningeal and myelitic inflammation as the result of such lesions. The resulting neuritis, which, according to Hayem, is both interstitial and parenchymatous, ascends or descends from the primary lesion not in a continuous manner, but interruptedly—*neuritis disseminata vel migrans*—portions of apparently healthy nerve alternating with inflamed portions. In this manner the neuritis reaches the cord, and induces either meningeal inflammation from the direct continuity of the spinal membranes with the neural envelopes and interstitial tissue, or a poliomyelitis with atrophy of the multipolar cells, of greater or less

extent and acuteness, probably through the medium of the posterior roots.

These experimental data render it probable, if it has not been actually demonstrated, that similar processes underlie certain symptoms which are occasionally seen in man in connection with peripheral nerve lesions. It has been found that at more or less distant intervals of time after traumatic lesions of nerves, articular inflammations, and amputation of limbs, atrophic paralysis or muscular atrophy has occurred in regions not directly related to the nerves primarily injured. Thus, after an injury to the ulnar nerve, as in a case reported by Brown-Séquard, muscular atrophy showed itself in the region of distribution of other nerves of the brachial plexus, in a manner which could not be accounted for by mere extension by contiguity. Many other instances are on record of atrophy of a limb from injury of one of the branches of the plexus supplying it.

Not unfrequently, also, atrophic degeneration occurs in the opposite limb, presumably from a transverse extension of the myelitic process; and there are instances, also, which would seem to show that the myelitis may be propagated longitudinally in an ascending or descending manner, so that atrophy occurs in both the limbs on the side of the original lesion.

Cases of this kind have been recorded by Vulpian, Charcot, Leyden, Terrier, and others.[1]

In addition to those already recorded, I may mention the following case, which has just come under my observation.

A young man, aged 27, who had left hip-joint disease in childhood, and had recovered with a stiff and atrophied limb, with which, however, he was able to support himself and walk, a few months ago rapidly lost power in this leg, so that now he is unable to support himself with it. A few weeks afterwards he began to experience numbness and weakness in his left hand, and lately he has experienced numbness in the ulnar region of the right hand also.

On examination, the interossei of the left hand were found

[1] See an able summary and review of facts bearing on this question by Ch. Talamon, 'Revue et Mensuelle de Méd. et Chir.,' July 1879, *et seqq.*

to be considerably wasted, the basal phalanges hyper-extended, and the use of the fingers characteristically impaired. The wasted muscles showed well-marked reaction of degeneration.

Though the right lower limb was apparently unaffected, yet there was greatly increased knee-jerk, and tendency to ankle-clonus. The power of retention of urine had also become considerably impaired since the symptoms first developed themselves.

In this case there seems to be both an ascending and transverse myelitis of a subacute form, the starting-point of which is probably the articular inflammation of the left hip, and the atrophic changes in the lumbar region of the spinal cord, which have long remained quiescent. The patient dated his symptoms from the use of a new boot which he had himself constructed, and the use of which in walking seemed to cause unusual fatigue.

A truly hemiplegic form of muscular atrophy has very rarely been observed. Leyden, in his work on 'Diseases of the Spinal Cord,' reports a case of a gunshot injury of the left thigh which was followed by severe pains in the leg, and afterwards in the left arm. Nearly three years after the receipt of the wound the patient became paralysed or paretic on the left side, leg, arm, and face. The acuteness of vision also of the left eye was impaired. Examined by Leyden in Jan. 1874 (the wound having been inflicted in Nov. 1870), the patient was weak on the left side, and complained of a feeling of heaviness in it. The muscles generally on the left side were flaccid and less voluminous than those of the right, and the electric contractility was somewhat diminished, and abnormal.

The following case which I have had under my care shows by its history and progress the recurrence of a truly hemiplegic muscular wasting, with paræsthesia, in connection with neuritis of the stump of an amputation of the left hand at the wrist.

The patient, ætat. 28, is a member of the medical profession; and he thus described his case. While shooting in India one day in June 1877, his gun burst, inflicting a scalp wound, also rupturing the left membrana tympani and

seriously lacerating his left hand. The hand was at once amputated just above the wrist, and in a little more than three weeks he was able to return to duty. The stump had almost entirely healed, and the scalp wound and laceration of the tympanum had quite healed up.

For two years subsequently to the amputation he experienced almost daily pain in the stump. The pain varied in character. He described it as like that caused by tension of the flaps over the face of the stump; as a burning sensation; as if something were contracting and subjecting the ends of the nerves to pressure; as a throbbing sensation.

Later on, this condition of the arm appeared to be greatly influenced by changes in the weather. A hot and dry climate was most agreeable, whereas a sultry and moist atmosphere was always the cause of pain; as also cold and frosty weather.

Soon after the operation he observed wasting of the left arm.

Towards the end of 1878 he began to feel as if a piece of parchment were drawn tightly over the left side of his face, which was so unpleasant that he frequently tried to relieve it by rubbing.

He also felt "unbalanced" or "lop-sided," which was particularly disagreeable. This feeling of "lop-sidedness" grew worse about August 1879, and at this time he observed some wasting of his left leg. Later, he noticed a similar wasting of the left side of his thorax and abdomen.

All these symptoms the patient noticed before they could be recognised by others.

During the attacks of pain in the stump the face always felt more drawn than at other times. One night after his return to England, at the end of 1881, during a severe attack of pain, he felt as if there were a muscular thrill through the left side of his chest. For the last year there have been sensations of pins and needles in the left foot, and fibrillar twitchings in the left leg and thigh. Similar twitchings have also been felt in the left side of the face, thorax and abdomen. He now—January 1882—feels as if there had been a layer cut off the whole of the left side of the body, and complains much of his "unbalanced," "lop-sided" sensation.

The patient was a well-developed, vigorous and healthy-looking man. All the organic viscera and functions were normal. It was plainly evident on examination of the two sides of the body that there was a want of symmetry, but the respective measurements were not determined at this time. The muscles of the left side reacted well to the faradic current, and no very evident difference could be discovered between those of the right and left side.

Sensibility was normal.

The question of re-amputation was discussed, and the patient further consulted Mr. Lister, who advised this at once. The stump was re-amputated by him on January 28, 1882, and the healing of the wound proceeded satisfactorily. A few days after the operation, pain recurred in the stump, accompanied by twitchings of the arm, and tenderness, with hyperæsthesia to cold over the whole of the left side.

After that, the patient ceased to complain of attacks of pain, and the paræsthesia of the left side of the body somewhat diminished. But the feeling of "lop-sidedness" still continued.

On February 22 I made comparative measurements of the two sides of the body.

By stretching a tape-measure between fixed points on the two sides of the face in various diameters, there appeared a difference of one-eighth of an inch in favour of the right side—

		In.		In.
Widest part of thigh . .	Right	= 21⅜	.. Left	= 20⅜
Four inches above patella .	Right	= 15½	.. Left	= 14½
Calf of leg	Right	= 13⅝	.. Left	= 13⅛
Abdomen—two inches above umbilicus	Right side	= 16⅜	.. Left	= 16½

The arms were not strictly comparable for obvious reasons, but the circumference round the acromion and axilla was one inch less on the left side than the right.

There was thus an appreciable difference between the right and left side of the face, trunk and limbs, varying from one-eighth of an inch on the face, to one inch in the limbs.

The patient shortly after this put himself at my advice under the care of Dr. de Watteville for electrical treatment.

This was carried out by him for about six weeks, by his method of galvano-faradisation, which he has recently described in the 'Neurologisches Centralblatt' for June 15, 1882.

Under this treatment the patient gradually improved in all respects, and on my again taking the same measurements on May 23, I found that there was now no appreciable difference between the two sides of the face. The widest part of the right thigh now exceeded that of the left by only one-eighth of an inch; the right calf measured only one-eighth more than the left; and similarly as regards the two sides of the trunk.

The patient ultimately returned to his duties in India, to all appearance perfectly recovered.

The above particulars show an evident causal relationship between the neuralgic stump of the left hand, and the occurrence of the peculiar "lop-sided" paræsthesia of the left side, and actually demonstrable wasting of the face, trunk and limbs on that side, which speedily ceased after re-amputation of the stump, and properly directed electrical treatment.

The condition of the nerves of the stump was examined by Mr. Watson Cheyne, who has furnished me with the following account of his examination.

"Examination of the stump immediately after amputation showed the presence of a firm neuroma, about the size of a small hazel-nut, affecting the divided end of the radial nerve. The nerve itself was thickened, and had a distinctly rosy hue, one or two vessels being also evident in its sheath. The lower end of the ulnar nerve was somewhat expanded, but this expansion did not present the distinct form of the radial neuroma, nor was it so firm. The nerve was of the normal white appearance. The median nerve was apparently quite healthy.

"*Microscopic characters.*—Longitudinal and transverse sections were made both of the neuromata and of the trunks of the radial and ulnar nerves. The neuroma at the end of the radial nerve was found to consist of a large amount of fibrous tissue, enclosing in small alveoli bundles of degenerating

nerve-fibres, in some places indeed the arrangement of the fibrous tissue showed that nerve-bundles had already disappeared. Here and there the fibrous tissue was still almost embryonic. In the trunk of the nerve there was great increase of the fibrous tissue between the bundles of the nerve-fibres which were undergoing degeneration. There was a considerable number of small blood-vessels, some of them surrounded by embryonic cells. This appearance was especially marked towards one side of the nerve, where there was a distinct line of granulation tissue. In the ulnar nerve there was thickening of the fibrous septa to some extent, but not nearly so marked as in the radial; there was no appearance of granulation tissue either in the trunk of the nerve or in the enlargement at its end.

"The appearances indicate chronic inflammation of the radial nerve, both at the divided end and along the trunk. This process was still going on at the time of the operation, as evidenced by the granulation tissue found along one side of the nerve trunk. There was no evidence of continuance of inflammation in the ulnar nerve."

Whether in this case there was any morbid change in the anterior grey column of the spinal cord of the left side capable of histological demonstration, or whether it was merely a so-called functional depression due to the long-standing peripheral irritation, can only be a matter of speculation. The latter view might be considered as the more probable from the fact of recovery on cessation of the irritation, though this would not necessarily follow.

The point of importance is the possibility of a morbid condition of the whole of the grey column of one side of the spinal cord being developed in connection with long-standing peripheral irritation, ascending and descending from the point of primary attachment of the inflamed nerve.

Critical Digests and Notices of Books.

Leçons Cliniques sur des Maladies Mentales et sur les Maladies Nerveuses, professées à la Salpêtrière. Par le Docteur Auguste Voisin, Médecin de la Salpêtrière. Paris, Baillière et fils. 1883. 8vo.

Lectures are perhaps not strictly clinical when they are delivered in the theatre of an hospital, and are not illustrated even by reference to cases actually under treatment. Yet these lectures of M. Voisin, replete as they are with the records of illustrative cases, strike one almost with the vividness of bedside teaching. Moreover, we learn from some casual remarks, that on one day of each week a limited number of the students are admitted within the wards of the great asylum, to observe for themselves the phenomena of mental disease. The authorities of the Salpêtrière take a middle course between the free admission of medical observers, which some people think injurious to patients within an institution, and the practical exclusion of unofficial medical men, which more certainly, by perpetuating professional ignorance of insanity, is, and must be, detrimental to that larger body of the insane who have not been placed under such treatment as that of the hospital of the Salpêtrière.

The difficulty of providing real clinical instruction in mental disease, that is to say, instruction illustrated by actual cases, for the vast number of recruits which the great profession of medicine demands, is perhaps so great that it may never be overcome, and so the public will in time become aware that the bulk of medical men may know as little from observation about lunatics as living priests know about the angels whom they have never seen. In this country the promising commencement of such teaching has all but withered in the bud. In France, however, as we see in this publication, it is still

attempted by means of lectures, more or less illustrated by examples, and such lectures, if they fail to attain the highest value of teaching by demonstration, are of great worth, especially to those fortunate practitioners of the medical art who are not compelled to draw entirely upon the resources of their imagination for some kind of picture of the lunatics they hear about. The resources of photography are but a feeble aid, except to the initiated. M. Voisin gives several pages of excellent photographs; but to any one who has never learned to read the lunatic feature and figure, we venture to think that they will convey little meaning, except of strong variations of temper and of good or bad looks. To the thousands of medical students who have never seen the play of a lunatic's features, or listened to the accents of his voice, even such instruction as that contained in our author's pages must, to a great extent, remain incapable of being appreciated; and until some plan of oral teaching, duly combined with actual demonstration, be devised and diligently carried into effect, it is likely that the early stages of insanity will remain practically unobserved or lamentably misunderstood, that the safety of the public from the insane who are at large will continue to be unguarded, and that the insane who are not permitted to remain at large will continue to be deprived of their liberty on judgments which are erroneously supposed to be the results of medical science.

We raise this lament, not over M. Voisin's important work, imperfect as his clinical demonstration appears to be, but because his work so far excels anything which we are likely to have in this country, where the neglect into which the teaching of this great department of medicine has fallen is so great that no one could be found in a position corresponding to that of this eminent Frenchman, with his materials for teaching, and especially with his audiences to be taught. M. Voisin has done well; no doubt he might do better if he could bring his students to more intimate observation of his patients. In this country the teaching of mental disease is, with one or two exceptions, either neglected or pretended. The medical men to whom the custody of the insane in ever-increasing agglomeration of numbers is entrusted, are losing the character of

physicians in that of administrators, and medical treatment for the cure of mental disease in the very institutions supposed to be devoted to the purpose seems to be passing out of use.

The first Lecture on Classification will serve as well as any subsequent one to elicit the criticism that these lectures are wanting in method, that their matter is less regular in arrangement, or, to use a homely phrase, less cut-and-dried than is usual in medical writings. We are inclined to accept this quality of the author's instructions as not altogether a disadvantage. The mass of observations on such a subject as insanity are so heterogeneous and vast, that it is impossible to reduce them to strict order, except at the expense of truth. There will be a time for method later on, when the true generalisations have been seized, upon which alone the facts can be rightly marshalled. But there is also in the development of any complicated science as it struggles out of ignorance, a time when facts and thoughts about them are grouped, and best grouped, in much detachment; and thus it is that we are far from finding fault with the somewhat rambling manner in which M. Voisin treats the various subjects in which he instructs us, leaving us not uncertain as to the main direction of his line of thought, but at liberty to feel ourselves by no means bound either to agree or to disagree with him absolutely. In a difficult country we follow a leader with all the more confidence, when we perceive that he is willing to wander from a straight line by practicable deviations.

With regard to classification, we quite agree with the author in the belief that the classification of *délires* is insufficient.

We also entirely concur in the remarks made upon the new classification attempted by the late M. Morel, and which has been imitated and exaggerated by certain writers on lunacy on this side. He says:—

"Morel, of Rouen, had much felt the defect of the actual classification, since he attempted to found one upon etiology; but for want of a sufficient number of pathogenic and anatomo-pathological facts and observations his new classification was deficient in solid foundation; it sinned also on an important side, by tending to confound forms altogether different, when it admitted hereditary insanity, for there are few mental aberrations in which one does

not find morbid heredity. By classifying in the same category all cases of alcoholic insanity one conjoins cerebral and meningeal hæmorrhages, fatty degeneration and chronic meningitis. The same difficulty presents itself in the hypochondriasis which one meets with in simple insanity and in general paralysis. I think, therefore, that it is necessary to reject all classification founded upon etiology alone. As in all branches of medicine, so progress in mental pathology is only possible by the participation of anatomical pathology, and every classification aiming at being rational, must be established upon etiology, pathogeny, clinical and anatomical pathology."—Pp. 15 and 16.

How different are these ideas from those which have been so earnestly pressed upon the profession by the Scotch school of lunacy, which is in so many other respects so practical and progressive, but which in this respect has developed the indefensible project of M. Morel into a fantastic system of etiological classification, which must, indeed, have brought down the animadversions of our author had he known of it! It would seem that M. Voisin has escaped the task of demolition in this instance by his ignorance of English. Apparently he is not acquainted with German either. Probably there is enough nonsense written in his own language for him to refute, but the literary knowledge which he possesses of his subject is very far from being cosmopolitan, and perhaps it would not be unjust to call it narrow.

As a partial classification, he divides *délire* into partial, general, and complex. We have not attempted to translate the word *délire*, which he uses, we believe, as almost tantamount to insanity; a sense commonly given to it by his countrymen, but which certainly does not correspond with our own use in medical literature of the word *delirium*. An English physician hearing that a person was delirious would be apt to conclude that he was rambling in thought in an unconscious or semi-conscious state of febrile or cerebral disease, and he certainly would not conclude that he was insane; but our professional brethren in France apparently apply the term to all insane deviations from reason in thought, and especially to delusion. The curious derivation of the word *delirium* from *de lira*, out of the furrow, seems to justify its French use as expressive of any morbid deviation from the straight and

right line of sound-minded reason. Might we not with advantage adopt this word from the French into our medico-psychological vocabulary, replacing by it the word "delusion," excepting where the latter is really appropriate, which it is not twice out of three times where it has come to be employed?

M. Voisin has not been proof against the temptation of formulating a classification of his own. He says that—

"Every rational classification must be founded on the *ensemble* of notions given to us by the symptoms, the pathogenesis, the pathological anatomy, and the etiology.

It is thus that I shall divide insanity into six classes.

1st. *Folie acquise*, that is to say, which comes on in the course of life and has been preceded by a state of reason and intelligence.

2nd. *Folie native*, a form in which the intellectual troubles manifest themselves at the earliest age, especially under the influence of heredity.

3rd. *Folie par intoxication et par virus*, the nature of which is clearly indicated by the name, or by alteration of the blood, or by diathesis.

4th. *Le Crétinisme, l'Idiotie, l'Imbécillité.*

5th. *La Paralysie générale.*

6th. *La Démence sénile.*"

The scheme is far from being unobjectionable, for is not the third class included in the first, and the fourth in the second, and why should senile dementia, so variable in its antecedents, form a class apart? Are the cases of the first, second, and third classes distinguishable either by their symptoms or by their pathological anatomy? No doubt the distinction of having been acquired from what the author calls native insanity is important, being, it would seem, the distinction which we have ourselves insisted upon between what we have called developmental insanity and accidental insanity, but it seems to us scarcely a distinction which it is wise to select as the principal basis of a system of classification.

The interesting lecture, being the thirteenth of the series, on Insanity caused by the siege of Paris and the Commune, throws light upon what the author means by acquired, as distinguished from native insanity. It had been remarked, and, if we remember rightly, an article had been written to

show, that there was no increase but even a diminution of recent cases of insanity in Paris during the fearful ordeals to which the population had been subjected from July 1870, to June 1871. M. Voisin, however, thinks that the allegation having been made upon statistics drawn from establishments which only receive patients from the rich and easy classes of society is untrustworthy, and that the poorer and labouring classes did suffer mentally, and in great numbers. However, he does not attempt to prove his case by means of statistics drawn from establishments which receive only the insane of the suffering classes, and the cases of individual suffering which he adduces, however interesting, are liable to be misleading, for it is all but impossible in any individual case to prove that there was no predisposition. It is not by heredity alone that the predisposition to insanity is formed, and when one person out of many succumbs to influences which act with equal force upon all, the inference must be that there was a predisposition to be so affected. It would seem that most men are so constituted that all the horrors of the 'Pilgrim's Progress,' or of Dante's 'Inferno,' could not drive them mad. Others there are who become insane upon severe exciting causes acting upon the mind, or upon slight ones, or upon none at all which can be discovered. While, on the other hand, it may safely be affirmed that there can be no man born of a woman who is not liable to become a madman from accidental causes of a physical nature, traumatic or toxic. Blows on the head and starvation must have caused a certain number of insanities in persons not predisposed; but it is by no means certain that the psychical horrors of war must have had any such effect. Moreover, it would seem more likely that men of leisure, culture, and opulence would be more affected by national disgrace and defeat, by social anarchy and the destruction of property, than the labouring, struggling, and suffering classes of society. M. Voisin therefore delivered an eloquent and interesting lecture, but he did not prove his case, nor upset the statistics of Charenton. The lecture also seems to indicate that the author is by no means clear as to what he really means by the somewhat taking terms of his classification, *Folie acquise* and *Folie native*. That which we have to

repeat in the matter is that there is a type of man, often enough of a sane family, who will become insane, and nothing can prevent him. His disease we call developmental insanity. Also there is another type of man whom nothing can make insane short of a physical injury affecting the brain in a certain manner, as by a syphilitic neuroma, or by poisoned blood, or some such physical interference with the organ of mind. Insanity in such a man we call accidental insanity. The two types must be recognised, but between them there is an infinite variety of susceptibility and of insusceptibility, and therefore we venture to think that this leading notion of our author's lectures ought only to be accepted with the reservation that it shall not be allowed to explain more than it is able to make really clear. The idea upon which M. Voisin founds his classification is a true one, but it is too diffuse to be of such use. Like flood-water, it will not turn a mill.

Our space limits will not permit even an appreciative reference to all the contents of the author's substantial volume of well-nigh eight hundred pages. Suffice it to say that, while some lectures are devoted to forms of insanity whose real existence we are fain to dispute, a larger and more valuable part of the volume discusses very fully, and with an ample supply of illustrative cases, such important topics as epilepsy, idiocy, general paralysis. The lectures on this last subject occupy 164 pages, and, in themselves a treatise, are too valuable and important to be dealt with in a brief critique. We may remark, in passing, that the author is inclined to call forms of disease, especially consecutive forms, by the name of general paralysis, where identity with the disease to which we in this country restrict the term is by no means certain. That the disease does vary more than our writers of even a few years back were aware of, may be admitted, but the variations described in these pages are wider and greater than we have as yet recognised.

The treatment of the subject of alcoholic insanity is well worthy of the large space devoted to it. The author shows none of that sentimental tenderness for the drunkard which has been rife among ourselves. He declares that alcoholic intoxication almost always has its origin in brutal appetites,

and that it is one of the passions, one of the vices of greatest tenacity.

If we may judge from some of the author's remarks, and still more from some of his cases, drunkards are incarcerated as lunatics at the Bicêtre and the Salpêtrière who would scarcely be detained in our asylums. The author says:—

"The most common type (of alcoholic insanity), and certainly the most interesting to study, is that of the individuals whose mental state only betrays itself very rarely by an excitement too violent or an incoherence too manifest, and who either maintain themselves in society, or form in asylums a category relatively superior; at least if one takes the reason for the term of comparison, for it is no longer so if one places oneself at the point of view of the moral faculties."

And he comments without reserve upon the calmness, amiability, and obsequiousness with which these drunkards will deceive the medical man, while in reality their conduct is but a little comedy which conceals the most outrageous lying, rascality and immorality. Evidently the little comedies and beautifully constructed but utterly false autobiographies of the habitual drunkard will never succeed in deceiving this astute observer. He draws, however, a most important distinction between two classes of persons who become insane by alcoholic poisoning. In the first, he places those persons who, being of fairly sober habit, and never having had a previous attack of *delirium tremens*, in consequence of great excess committed within a few days, or even after sudden deprivation of alcoholic drink, necessitated by indisposition or penury, have an attack of mental aberration, consecutive or not to an attack of *delirium tremens*.

In the second class he places drunkards by profession, who have had one or more attacks of *delirium tremens*, and who may be regarded as affected by chronic alcoholism. Not one example of this class on leaving hospital after cure of acute delirium but retains, as symptomatic of his chronic state, enfeeblement of memory and intelligence and of moral energy, diminished aptitude for his habitual employment and loss of muscular force. In some of them the delirium ceases after a month or more, and, notwithstanding enfeeblement of mind,

they may be allowed to leave the hospital; while in others the delirium does not cease, and gradually becomes complicated with confirmed dementia (p. 221).

The number of cases of the first class is small compared with those of the second; during the year only four of the former having been admitted into the hospital, against twenty-eight of the latter. It is rare that after a residence of two months in hospital even the chronic cases cannot be set at liberty. They are not really cured, indeed, but with diminished muscular force and moral energy, and very generally with enfeebled memory, they are yet in a state in which it is impossible to detain them longer. So they leave the asylum, carrying with them the tendency to become again insane with great facility and from slight causes, as, for instance, some excess, or some moral trouble, or even after more or less absolute abstinence from food and alcoholic drink.

The remarks of the author on the delirium of insane drunkards are valuable. He does not agree with some other writers in believing that it is always marked by sadness or depression. It is not a systematic, and, so to say, co-ordinated delirium, like that of monomania. It is essentially illogical, superficial, and impresses its mark rather upon the words and facial expression of the patient than upon his conduct. The expressions of unreason are transitory, and the patient makes light of them if they are disputed. In these respects it resembles the delirium of general paralysis, and differs from that of monomania. The patients, however, do not present the self-control and personal satisfaction which prevails among general paralytics, and this constitutes a diagnostic mark of the two kinds of delirium. Some insane drunkards have amnesia and trouble of speech. Their pronunciation is hesitating, stuttering, confused. Sometimes, perhaps, the hesitation of speech depends upon imperfection of memory, an indication of which is afforded by the fact that the written language is just as imperfect as that which is spoken. The conscience is affected, or that which the author calls conscience. It is impossible to make the sufferer comprehend that it is due to his own fault that no one has been willing to employ him because he was a drunkard, or that his wife has left him

for the same reason. Towards the medical attendant these patients are kindly, submissive, and hypocritical, but towards their wives and children they are far from being so amiable, and are generally irritable and often violent. Without appreciating the cause, they become unable, from loss of muscular strength and freedom of action, to earn their former wages, and this is one reason why they fall into the state of wretchedness which is so common to them; a reason which they do not seem to appreciate.

From the manner in which the author discusses and refrains from deciding whether lunatics from drink can properly make a will, and whether they are responsible for offences against the law (p. 253), it would seem that he does not draw any definite line between drunkards who are not really lunatics, and lunatics who have become such from drink. No doubt there are cases in which the transition from one state into the other is not marked by any well-defined boundary. But the two states are essentially distinct, and the author has failed to define them. Here, as elsewhere, when he trusts himself to any argument or observation outside his rôle as physician, the author lays himself unusually open to criticism. For instance, with regard to responsibility of these people, he says:

"De pareils hommes sont assez le malheur de leurs familles pour que la loi soit en droit de prévenir, par la rigueur de la répression, de semblables habitudes chez d'autres; qu'adviendrait-il, au contraire, s'il était notoire que l'alcoolisme exempte de toute pénalité?"

One would have thought that the continuous misery which a drunkard inflicts upon his family is no reason why he should be exempt from the rigour of the law on account of the occasional misery which he may inflict upon others.

We have no space left for further remarks upon this suggestive volume, except for some brief ones upon the most important subject of treatment. In a few paragraphs the author justifies the use of mechanical restraint, but as a counterpoise he displays the old-fashioned, and we fear the out-of-fashioned, faith of a physician in the efficacy of medicinal remedies. The 28th and 29th lectures are a valuable disquisition on the employment of bromide of potassium in nervous diseases, and

the 30th is a still more important one on the treatment of insanity by the muriate of morphia, endermic injection being the mode of administration mostly used and expressly recommended. Highly as we ourselves value opium and its salts in the treatment of mental diseases, we are not a little surprised at the variety of forms of insanity which have been successfully treated by the author with very large doses of morphia. When he first commenced to treat maniacal, melancholic-persecuted and hallucinated cases with the endermic injection of muriate of morphia, he found it often caused intense vomiting, but, under the advice of Roller of Illenau, he disregarded this inconvenience, and increased the doses with the remarkable success of which his pages are the astonishing record. The contra-indication of the morphine treatment is cerebral congestion, and this he attempts to remove beforehand by blisters to the shaven occiput and nape of the neck. Considering the large doses of morphia which he gave and the long time during which they were sometimes administered, he became apprehensive lest the red blood globules should be diminished by the drug, and he made observations which proved to him that this fear was not well founded. Chronic insanity may itself lead to a diminution of the red globules, but the heroic administration of morphia would seem from the author's observations to resist rather than increase this tendency (p. 703).

The list of the forms of insanity for which he uses the morphia treatment seems to include all varieties of mental disease except those which he considers to be dependent upon a congestive state of the brain. In these it may be injurious, and he well remarks, that this distinction of the forms of insanity, which may and of those which may not be properly treated by morphia, depending upon the state of the organs, as regards congestion, shows that "the diagnosis of the anatomical nature of insanity is of the most serious importance."

The author cites his experience to prove that insanity of many years' standing, five or six years even, may be treated successfully by the morphine method, and that forms of insanity hitherto thought incurable, as *folie à double forme et circulaire*, can be cured by it. In this form the dose of muriate of

morphia needs to be from six to eight grains a day. In lypemania with groaning, the high dose of from seven to ten grains a day is, he says, requisite.

It would be presumptuous to criticise the remedy or the posology of an author who gives such abundant and circumstantial evidence of his success; but it will scarcely fail to strike the readers of Dr. Voisin's volume that muriate of morphia has become with him more of a panacea than will be thought consistent with the variability and many-handedness of scientific medicine. We have no doubt that opium is the most useful of all drugs in insanity; and yet we cannot but express some astonishment at the unexampled success of our French confrère. We can, however, strongly recommend his volume to the study of our psychological compatriots. They will perhaps not accept with implicit faith everything which they find in its pages, but they will find it replete with opinions and experiences gathered in a field of observation new to them, and formed in a widely different spirit to the jejune officialism into which English psychiatry is passing. The volume is illustrated by some excellent engravings and by some interesting photographs. It, however, has no Index, and a very inadequate Table of Matters. Why any one should take the trouble to write a valuable book of nearly eight hundred pages, and not take the trouble, or pay some one else a trifle to take the trouble for him, of constructing an Index, is one of those mysteries of authorship for which we have never yet discovered any reasonable explanation.

JOHN CHARLES BUCKNILL.

Les Hystériques; état physique et état mental. Par le Dr. LEGRAND DU SAULLE, Médecin de la Salpêtrière, &c. &c. Paris, J. B. Baillière et fils, 1883.

FROM France we receive another great work on Hysteria. Although this disease is so widely spread, so universally distributed throughout all countries, climates, and conditions of life, it is a significant fact that the most important literature on the subject is practically confined to certain countries. In England, if we except the writings of Brodie, Todd, Conolly,

Laycock, Skey, and a few others, there are comparatively few special treatises devoted to this question. In France, on the contrary, not only do its medical publications teem with original investigations and observations, but nearly all the great and elaborate memoirs on hysteria emanate from that country, as evidenced by the classical works of Tissot, Louyer-Villermay, Georget, Dubois, Brachet, Landouzy, Briquet, Charcot, and a host of others. Reflection on this point alone would seem to suggest that, although this disease is met with in every part of the globe, in certain special countries it attaches to itself peculiar attention and interest on account of its unusual prevalence or severity. That such is the case is evident from the study of the hysteria at home as compared with the same abroad. Just as the affection seems to attack certain groups of individuals and special constitutions, so do we find it more universal and severe in particular races of mankind, invading those particular temperaments and nervous organisations which would seem to be more specially prone to neurotic excitement and instability.

A consideration of the works of French writers on hysteria indicates not only by their number, but by the nature of their contents, that this disease is much more universal in that country than in England; that phenomena of frequent occurrence there, are extremely rare here; and that the malady, attended with comparatively little danger on this side of the Channel, is surrounded on the other with symptoms of the most serious import. This difference in the two cases is due to the peculiar habits, customs, and mode of education of the respective peoples; and it may be that the fundamental distinction of race is the predisposing cause, in which the nervous organisation of the Latin nation is more prone to this form of derangement than that of the more phlegmatic Saxon. However it may be accounted for, the fact remains that in France, hysteria is a more universal and serious disease than it is in England, where, although common, its type is of a milder and more innocuous form. This, of necessity, attracts public attention; the supply of literature on the subject is in proportion to the demand, and hence the number and importance of works on hysteria in the former country as compared with our own.

The most recent book on the subject lies before us, a volume of 625 pages. To give a complete idea of the contents of so voluminous a work would occupy more space than is permitted us; suffice it to say that the result is eminently worthy of the author, of the hospital he represents, and of the great country from which emanates such valuable neurological researches. It essentially consists of an able *résumé* of all the most modern observations and investigations on hysteria, evidently compiled by a writer of large personal experience, and who has deeply studied the subject, as he tells us, for a period of thirty years. It cannot be said that any startling novelties are advanced, and M. du Saulle throughout largely quotes from Briquet, and on the whole endorses the views of that elaborate and statistical investigator. At the same time the tone of the volume is original, in so far as its conclusions are supported by the personal observations and extensive experience of the author. Above all, the literary style of the book is admirable, and leaves little to be desired; it reads like a romance, and reminds us of the late Sir Thomas Watson's poetical narrations, giving even the dry details of unattractive phenomena a charm which can be perused with pleasure as well as profit.

Without attempting to review this work in detail, we shall direct special notice to one or two points of general interest. In discussing the etiology of hysteria, M. du Saulle proves himself to be one of the modern school who believes that the influence of the uterus in the production of that disease has been, and is, greatly exaggerated. The arguments he employs in favour of this view are precisely similar to those which we ventured to advance in a former number of this Journal (April 1879) in referring to the Hystero-neuroses, and which need not again be repeated. It is there maintained that the genital organs have no necessary connection with the causation of hysteria, although, when disordered, they may, in common with diseases of other organs of the body, be the origin of the nervous phenomena. In short, as the author states, hysterical women have been calumniated when it is insisted that the disorder is due to perverted sexual desires or to functional derangements of the genital organs. On the contrary, it is a neurosis due to instability of the general nervous system as a

result of hereditary transmission, or acquired by unfortunate circumstances, or a faulty education. Such a predisposed constitution is subject, as a result of various exciting causes, to develop those phenomena to which we apply the term hysterical, and among others those of the uterus may claim a share, but not a necessary or unique one. It is known that by far the most common exciting causes are those of a mental or emotional character, and that those of a physical nature are comparatively less frequent. Of these, pain and distress in any form may induce or originate the first manifestations of the disorder, and it is not surprising, considering the prevalence of uterine troubles amongst women, that these should be amongst the number. Gynecologists who see much of hysteria combined with sexual disturbance, and little of it under other circumstances, naturally associate the two together, and many believe the one to be a constant factor of the other; but the fact that such is common in special practice is no proof that hysteria, as a disease, is always or even generally the result of uterine derangement; on the contrary, the experience of the general physician indicates that in the majority of cases there is no evidence of disease of the genital organs, and that when this is present the functional derangement is as often the result as the cause of the general nervous disorder.

One chapter of this work is devoted to an interesting *résumé* of the most recent observations on Hypnotism as occurring in hysterical persons. The author more particularly refers to the researches of his colleague, M. Charcot, at the Salpêtrière. This condition, he explains, may at least assume three different forms in the same subject—the lethargic, the somnambulistic, and the cataleptic—each of which may be either spontaneously or artificially produced, and each of which may be readily transformed into the other. In the first, the patient is in a profound sleep, the senses are blunted, the reflexes are increased, and the muscles and nerves are in a high state of hyper-excitability. In the second, the intellect and senses are acutely sensitive, the reflexes are normal, there is no special irritability of the neuro-muscular system, and the patient readily obeys suggestions from without. In the third, the patient's limbs remain in any position in which they are

placed, the condition otherwise being as in sleep. These, and numerous other complicated and interesting phenomena, are described as occurring in cases of grave hysteria during the hypnotic condition; to appreciate the details of which, the reader should consult the work under consideration, as well as the researches, more especially, of M. Charcot. Such instances of hypnotism, if they exist, must be very rare in this country. Braid and others have recorded cases of somewhat a similar nature, but from all accounts they must be much more frequent amongst our neighbours on the other side of the Channel. The present writer, when in Paris some years ago, saw at the same time in the Salpêtrière a large number of severe cases of hystero-epilepsy, some of whom exhibited the most astounding phenomena under the hypnotic condition. Although he has had ample opportunity in connection with metropolitan hospital practice, and has been on the look-out for such cases for years, he has never met with a single instance of this remarkable form of the disease, and therefore has had no opportunity of testing the effects of hypnotism.

The most novel and perhaps the most important section of this work are the chapters devoted to the medico-legal aspects of hysteria. The author points out that in Paris alone there are no less than fifty thousand persons suffering from the disease who are intimately mixed up in society and in all domestic concerns. That amongst these should arise infractions against the law is not surprising, and, as a matter of fact, the hysterical subject is frequently involved in judicial proceedings. Are such persons responsible agents in the commission of misdemeanour or crime? Such a problem is obviously of the highest difficulty and importance. The author enters fully into the subject, indicating that while many hysterics in offences against the law may be perfectly responsible for their acts, there are others whom it would be a miscarriage of justice to look upon as criminals, as in a certain number of cases the mental faculties are as profoundly disturbed as in serious forms of insanity—of which, indeed, this aberration is only one of the forms. The various states of mind and abnormalities of intellect are detailed, and the different kinds of misconduct and crime fully described, and a

series of typical and interesting cases are given in illustration of the writer's views. M. du Saulle thus studies a question of great practical utility, and one which has been much neglected. He not only investigates the disease itself, and those whom it attacks, but he follows them into society, in its varied relations of civil and domestic life, and into the multiple and complicated conditions demanded by social existence. He looks upon the affection not as a mixture of fanatical caprice or wilful misconduct, but as a serious material complaint, which should be studied on the basis of other corporal diseases, not by appealing to the imagination, but on the dry measurement of facts.

There are many questions considered in M. du Saulle's book to which the space at our disposal does not even permit us to refer. It is sufficient to say that we can strongly recommend the work to the careful consideration of our readers. If it is not profound, elaborately statistical, and bristling with original facts, it is none the less agreeable and profitable reading, and, by its fresh and graphic literary style, pleasantly initiates the English student into the details, from a French point of view, of a disease of which he sees comparatively little in his own country.

A. Hughes Bennett.

La Pellagra nella Provincia dell' Umbria. Del Dott. Roberto Adriani. Perugia, 1880.

In a pamphlet of 78 pages, Dr. Adriani gives us the result of his studies upon pellagra and pellagrous insanity in the Province of Umbria. This disease, so interesting to the pathologist, is never met with in the British Isles, hence we should have liked if instead of enumerating the bare symptoms the author had given a complete sketch of the course and sequence of the malady. The picture actually presented by the sufferers is, he tells us, very distinctive, though difficult to describe. Pellagra is characterised by muscular weakness, accompanied with pain, anæmia, emaciation, burning in the stomach, colliquative diarrhœa, a livid redness in the face and

hands, a staring and fearful look in the eye, a tottering gait, and a general air of squalor and misery. The loss of strength is gradual, and the nervous symptoms seem to come on at a later period of the disease, consisting in spasms, mental anxiety and discouragement, passing into apathy and stupor. If the loss of strength be not too profound, the patients very often recover during the winter; but the disease is apt to return in the spring. According to Dr. Adriani, about 5·5 per cent. of those affected with pellagra become insane. Some physicians state the proportion as high as 11 per cent. The mental derangement generally takes the form of melancholia, often with great anxiety about religion, and fears of damnation. Not unfrequently there is a tendency to suicide. Sometimes they are troubled with hallucinations; the memory is impaired, the understanding confused, and the speech extravagant. In some cases the appetite is voracious; in others they neither eat, speak, nor sleep. If well cared for and properly fed, they are sent out of the hospital or the asylum with their health restored; but if they return to their old diet, they become worse than ever, till at last they fall into a condition of hopeless dementia.

Pellagra has been in Italy for above a century. It is most frequent in Lombardy and Venetia. On the west it has invaded Piedmont; and on the south it has entered Emilia, Tuscany, Umbria, the Marches, and the Romagna. Towards the south of these provinces it becomes rarer, till it disappears at the confines of the Abruzzi. Pellagra was first seen in 1840; the earliest cases appeared about the Thrasimene lake. Year by year it becomes more common, It was at first confounded with paludal malaria. From 1847 to 1879 about a thousand cases have been observed in Umbria. In some cases the mental symptoms are slight and transitory. About a third of those who become insane are treated in their own homes. From 1854 to 1880 there have been 306 admitted into the Asylum of Perugia from pellagrous insanity.

Dr. Adriani gives the population of Umbria at 549,203 inhabitants, of which 273,292 are dwellers in the country, and 245,767 are engaged in agriculture. He calculates that there are 2·8 pellagrous for every thousand of the general popula-

tion; 5·7 for every thousand of the rural population; and 6·3 for the agricultural population. One reason why the disease does not excite more attention is that its ravages fall principally upon the poorest of the country people, who suffer in silence.

Dr. Adriani feels himself constrained to combat the views of superficial theorists, who attribute pellagra to an insufficient diet, to unhealthy surroundings, overwork, want of flesh-meat, or insolation. Were the disease owing to any of these causes, severally or jointly, assuredly its distribution would be much more extensive. Like goitre, cretinism, or malarious fever, it must be owing to a specific cause. The arguments adduced by Dr. Adriani, that pellagra is the result of an altered or degenerate condition of the maize which forms the staple food of the peasantry, seems to be of irresistible force.

Pellagra is observed only where maize is an article of food, and it appears in localities where it was previously unknown after maize comes into use, quite independently of the condition of the air, of the nature of the soil, of the quantity and quality of the water, and of the healthiness of the houses. The disease, moreover, becomes aggravated and diffused in proportion as the consumption of Indian corn is increased and extended. In those years in which the corn and rye crop was scanty, and maize entered more largely into the dietary of the peasantry, pellagra became more frequent; whereas it disappeared where the maize crop failed, as in the province of Brescia in 1816 and 1817. It has been asserted by Henry Gintrac, in the article "Pellagra," in the 'Nouveau Dictionnaire de Médecine et de Chirurgie Pratique,' published in 1878, that the disease has appeared in cases where maize was never used for food. Dr. Adriani has examined some of these instances, and finds them incorrectly reported. In one case a woman had given up the use of Indian corn by the advice of her medical attendant after she had fallen ill. Calling in another physician, she had told him that she was not using any maize, on which he had assumed that the disease had been produced without the use of that grain.

In another case which came under the direct observation of Dr. Adriani, the mother of the family had herself lived upon

maize, which she had obtained at a low price from the miller, and had given up her share of better food to her children. The doctor, not knowing of the self-denial of the poor woman, published the case as one of sporadic pellagra coming on without the use of maize.

Small properties seem to be common in Umbria. Where the proprietors do not cultivate their own land, they give it to metayers, who divide the produce with the proprietor, generally in equal halves, the proprietor furnishing the seed. This answers better than where the land is given to small farmers, who get into debt and exhaust the soil.

The labourers who work for the metayers are very poor. Maize forms a proportion varying from two-thirds to four-fifths of the dietary of the agricultural class. Besides this, they eat beans with rice, potatoes, and cheese in small quantities; oil and salt are the condiments used. They generally drink light wine, but seldom eat flesh. The poorest classes live principally upon maize, half cooked, with a little salt. The kind of Indian corn mostly seen is the Zea mais vulgaris æstiva; more rarely the Vulgaris autumna. The culture of maize is always extending, hence they are not so careful in choosing situations where it may be expected to ripen perfectly. Italy consumes more maize than it produces, so there is a considerable importation from Albania and the Danubian Provinces. In a village in Poland pellagra once appeared, from the inhabitants using maize brought from the Danubian Provinces. It would appear that often there is not heat enough in the summer of Northern Italy fully to ripen the grain. The immature and damp corn is liable to degenerate. This tendency is often increased by the manner in which it is stored. In Corfu an epidemic of pellagra followed a cold and wet summer. Balardini found that he could produce symptoms similar to those of pellagra by giving spoiled maize to animals as well as to human beings. Lombroso, by administering for a long time small doses of a tincture of degenerated maize, containing a red oil of a bitter taste, produced some of the symptoms of the disease—such as voracious appetite, with nausea and burning of the stomach, heat and pruritus of the limbs, diarrhœa, emaciation, and weakness. Through the

researches of Lombroso, which were at a later time associated with those of Dupré, and of Pesaro and Ebra of Milan, an oily resin and an azotized alkaloid, called pellagrozeina, were discovered. This substance resembles strychnine in some of its chemical properties. Pellagrozeina is said to be of use in some diseases of the skin. Given to chickens, it produced diarrhœa, paralysis of the limbs, tonic convulsions, and death. With grown-up cocks it produced immobility, diarrhœa, and paresis of the lungs. Given to dogs, it was found to cause somnolence, diarrhœa, thirst, refusal of food, and diminution of weight. In a man who had swallowed some pellagrozeina there followed confusion in the head, nausea, diarrhœa, and disgust of food; in another man there was a sense of weight in the lower abdominal region, burning in the throat, loss of appetite, pruritus, and melancholy. The experiments with the drug were pushed so far as to produce death in animals. It was found that the effects varied when the pellagrozeina was obtained from maize. The spinal cords examined showed indications of diffused granular myelitis, with a characteristic softening of the white substance, and in some cases of the grey matter. This softening sometimes occupied the whole cord; in other cases it only implicated the lower portion. There was also fatty degeneration of the sympathetic nerves. Dr. Adriani points out that pellagra has already had a serious effect upon the general health of the rural population of Umbria. It has brought about a notable diminution in their physical force and intelligence, has increased the number of the insane and suicidal, and raised the annual mortality. He discusses in a comprehensive manner the question how to prevent and remedy the disease; but for these and other interesting passages we must refer to the pamphlet itself.

W. W. Ireland.

Clinical Cases.

NOTES ON A CASE OF CEREBRAL TUMOUR.

BY A. HUGHES BENNETT, M.D.

Physician to the Hospital for Epilepsy and Paralysis, Regent's Park, and Assistant Physician to the Westminster Hospital.

T. K., ætat. 49, Railway Guard.—*Antecedent History.*—The patient says that he has always been a perfectly healthy man, but his wife states that for some time past he has been irritable and depressed, and had deficient memory. There is no history of syphilis, and with the above exceptions he has been considered in robust health, till about two months ago, when for the first time he observed slight weakness of his left leg. This has gradually increased till the present time. Two weeks ago, being in this condition, in the course of a day his left arm became very feeble, which has slowly progressed since, but he has managed to continue his work till three days ago. There has been no pain in the head, nor has he ever suffered from fits, or from cerebral or other abnormal symptoms.

Present condition.—The patient has all the appearance of a man in robust general health, and all his functions, except those to be described, are normal. There is no pain in the head or elsewhere; the special senses and the actions of all the cranial nerves are normal, except that the tongue, when protruded, is pushed very slightly to the left side. There is no optic neuritis. The face is perfectly straight, and the speech is unaffected. The movements of the left upper extremity are greatly impaired, those of the shoulder-joint are very weak, those of the elbow more feeble, and those of the wrist and fingers abolished, so that there is complete wrist-drop and complete immobility of the whole hand. The left arm is colder and bluer than the right. The sensibility is everywhere normal, and there is no wasting of the muscles. The patient can walk, but is very lame with the left leg, which is feeble, and has a tendency to sink under the weight of the body, but there is no rigidity or special gait. When sitting

on a chair the movements of the lower extremity can be performed, but slowly and weakly, and its sensibility is normal. The cutaneous reflexes are as in health, the knee-jerk phenomenon is most marked on the affected side, where there is also evidence of slight ankle-clonus. There is no wasting of the muscles. The electrical reactions are the same on both sides, and normal.

Progress of the Case.—The patient was under observation three weeks in the hospital, under the care of Dr. Sturges, before he died. The entire left side gradually became weaker, and was occasionally attacked with paroxysmal twitchings, which included the side of the face. Slight hesitation of speech was noticed, with a tendency to the repetition of words, but this was very indefinite. Soon after supervened dulness of intellect, depression of spirits, a feeling of numbness of the left leg, and slight twisting of the face towards the right side. These symptoms continued to increase, so that a few days before death he was dull, partly comatose, and very difficult to rouse; the speech was much embarrassed, and he had visual hallucinations, fancying he saw all sorts of things and people in the ward. The paralysis of the left side became complete, he passed all his motions in bed, vomiting ensued, the pupils were strongly contracted, deep coma, and death ended the scene.

Post-mortem examination, (18 hours after death).—Permission was only given to examine the head. The following notes are mainly abstracted from the report of Dr. Hebb. The body is well nourished. The membranes of the brain are healthy, and there are no evidences of meningitis. The blood-vessels are everywhere normal. The convolutions of the brain are flattened on both sides. There is no excess of subarachnoid fluid. On removing the membranes, just anterior to the fissure of Rolando is seen a circular patch the size of a crown, of mottled appearance, and consisting of softened or broken-down grey matter. This occupies nearly the whole breadth of the ascending frontal convolution at its middle, but not extending quite as far back as the fissure of Rolando. It also involves the whole of the posterior end of the middle frontal convolution, and a small portion of the bases of the superior and inferior frontal gyri. On making an incision through this softened area in the line of the ascending frontal convolution from the surface down to the ventricle, it is found that a mass of spheroidal shape, and about the size of a small orange, occupies the centrum ovale of the right hemisphere, its apex constituting the floor of the cortical softening, and its base resting on the roof of the lateral ventricle, into

the cavity of which it projects, without, however, apparently pressing upon the structures in its interior. The tumour is a gelatinous-looking mass, consisting apparently of broken-down nervous structure, and traversed by dilated blood-vessels, and was subsequently ascertained to be a glioma. It is sharply defined from the neighbouring nervous tissues, which appear healthy. The remainder of the brain is normal.

Commentary.—On this case little comment is necessary, the facts being simple and apparent. Beyond the gradual and progressive appearance of the paralysis, there were no symptoms during the life of the patient to indicate tumour of the brain. There was no pain in the head, no marked implication of the cranial nerves, no convulsions or sickness, and no optic neuritis. It would seem as if the lesion originated in the centrum ovale, where, by slow growth, and gradually pushing aside the tissues without destroying them, it attained considerable size without causing symptoms of any kind. Finally, some weeks before death, by extending towards the cortex and involving its structures at the motor centres, or by pressing upon their conducting fibres, paralysis was induced.

On the question of cerebral localisation this case does not throw any positive light, as the tumour was not confined to the grey matter of the cortex alone, but extended largely into the medullary substance below; and although the basal ganglia and structures of the brain generally, elsewhere appeared healthy, it is always difficult to estimate how far in such a case the effects are due to disease of the convolutions, or to pressure on or destruction of the conducting media. The area of degenerated cortical substance did not involve those portions of the convolutions in which are supposed to originate movements of the lower extremity, and only to a very slight extent those presiding over the motion of the arm. During life, however, there was marked paralysis of both these limbs. This, of course, proves nothing against the observations advanced in favour of cerebral localisation, as the tumour extended downwards in the conducting matter, and may have thus interfered with its function in transmitting influences from the parietal cortex to the periphery, without the former being itself directly implicated.

The lesion, however, suggests certain negative conditions which may be noted. The patch of softened grey matter was definite and complete, occupying the area already described. Within this space is what is supposed to be the oculo-motor centre, which on this side must have been in great part destroyed. During life it was certain that the patient displayed no abnormalities in connection with the movements of

the eyeballs, the pupils, or the eyelids. It may be, as Ferrier suggests, that the disease proceeding slowly, and one side being healthy, the bilateral association of the oculo-motor nuclei may compensate for disease on the other.

The area of softening also involved what is stated to be the centre for facial movements. It was, however, particularly noted that until a few days before death the face of the patient was in no way affected. It is true that near the end a deviation was observed, but this was very slight.

These points are here noted as matters of fact, and not with the view of throwing doubt on the question of cerebral localisation, for, as already stated, the nature and distribution of the morbid lesion utterly precludes any accurate conclusions on this subject. In this case we are even uncertain of the exact limits of the destructive process in the grey matter, and of its duration in point of time, not to speak of the complications arising from the presence of a large tumour in the encephalon.

The diseased mass must have been in close proximity to the right island of Reil, and being on that side probably accounts for the absence of any defect of speech. Towards the close, slight difficulty of articulation was noticed, but there was no true aphasia.

Finally, it may be noted that in this case of cortical lesion there were no convulsive attacks. A few days before death, occasional twitchings of the left arm, leg, and side of the face were observed, but these were very slight.

LEFT HEMIPLEGIA AND LEFT-SIDED DEAFNESS AFTER WOUND OF BRAIN THROUGH RIGHT ORBIT. PROBABLE LESION OF INTERNAL CAPSULE AND FERRIER'S AUDITORY CENTRE.

BY H. DONKIN.

THE following case of accidental "vivisection" in the human subject is placed on record for its clinical and physiological interest.

R. Putt, a boy of twelve years old, was carried into the East London Hospital for Children at Shadwell, at 5 P.M. on Feb. 16th, 1882, in a sensible, but very drowsy state, with the history of having had the steel rib of an umbrella "run into his eye." It appeared that in playing with his schoolfellows he had been shut into a room alone, and upon this, applied his right eye to the keyhole to see what was going on outside. One of the boys, not knowing that the eye was on the other side, thrust forcibly through the keyhole a detached steel umbrella-rib, which, according to R. Putt's positive statement after his recovery, entered the outer corner of his eye. He felt giddy, fell down, and then, while on the floor, pulled out the spike. He cannot say how far it went in. Nothing more was remembered by him till he was brought to the hospital about twenty minutes or half an hour after the accident.

I saw the boy immediately after his being taken to the Casualty Room. He answered when spoken to, but had considerable difficulty in articulation. His left arm seemed quite powerless, falling heavily down when lifted, and he could scarcely move his left leg. The mouth was markedly drawn up towards the right side, and the tongue, protruded with some difficulty, pointed to the left. The right eye was invisible, the tissues around being much swollen and discoloured by extravasation.

Half an hour after admission to the ward, the boy's condition was noted by Mr. J. Scott Battams, the Resident Medical Officer, as follows:—

"Boy sensible but drowsy: has just vomited. Speaks in a

drunken spluttering fashion: says he felt giddy after the accident and could not stand. Has pain in the occipital region. Considerable loss of power in left leg: resists flexion and extension very feebly: can just raise the leg from the bed. Left arm weaker than leg: cannot squeeze with the left hand at all. No pain or affection of sensation in paralysed limbs. Sole and patella reflexes present: cremaster reflex not elicited on left side.

"Right eyelids much swollen, and opened with difficulty only enough to expose a small part of the eyeball, which is tense and seems pushed forward. No distinct wound to be made out.

"Cannot hear watch in left ear: hearing perfect on the right side. Left side of face paralysed: cannot wrinkle forehead on left side: right angle of mouth drawn up. Sensation normal. No affection of left eye. Tongue, protruded, points to left. No rigidity or spasm of limbs. Pulse 100, irregular in force and frequency. T. 98."

At 10 P.M. (same night) the boy was noted to be lying on his left side: he would not lie on right side or back. He had vomited twice. He was very drowsy, but quite conscious. He was said by the sister to have had some convulsive movements of the *right* arm. When the right hand is held there are some convulsive movements of the fingers.

The next morning (17th) the following note was taken:—"Slept well, not sick since 2 A.M. Slept with hands at back of head. Cheeks flushed, more on left side. Is quite sensible and speaks plainly. Still deaf in left ear. Tongue still points to left. Now only complains of pain in left occipital region. Can now raise the left arm from the bed: and can resist flexion and extension of the leg better. Right side of face still drawn up. No loss of sensation on left side, but the left hand and fingers are said to feel as if wrinkled."

18*th*.—"Decidedly better. No pain. Talks more plainly. Power in limbs increased: coarse and fine movements of left hand and fingers well performed. Still facial and lingual paralysis, though less marked. Less swelling of right eyelid: can see indistinctly with right eye. Vision in left eye normal. Pulse 80, regular. Last night the pulse was intermittent. *Cannot hear on the left side a watch placed close to the ear, or on the skull.*"

21*st*.—"Boy has gone on improving. No headache, tongue protruded straight. Facial palsy much less marked. There is considerable power in the left limbs. No twitchings."

23*rd*.—"All paralytic symptoms have disappeared from the left side, including the face and tongue. The right eyeball is

now, owing to subsidence of the swelling, completely uncovered. It can be moved only in the upward direction, and but slightly. In all other directions it remains motionless as the left moves. He can see with the right eye, though indistinctly."

After this date no fresh symptom showed itself: the boy appeared perfectly well. Gradually the movements of the ocular muscles of the right side returned, and on March 20 the paralysis was apparently confined to the external rectus muscle; all other movements being well performed. The right pupil is slightly larger than the left, and acts more to light.

On admission, the boy's temperature was 98. For the first three days after the accident it varied between 99 and 100, and was afterwards normal.

On May 8, after being at the convalescent home, the boy was seen again, presenting no abnormal symptom other than a slight squint, owing to a small degree of loss of power in turning the right eyeball outwards. He made no complaint at this time of seeing double, but was found to do so on June 6, when he was re-admitted for a short time, owing to an alleged slight weakness of his left leg. The fundus of the right eye showed nothing abnormal. He complained of not being able to read for any length of time. Sees double, unless he closes the right eye, and complains of pain in it when he tries to read. The false image is to the right, and a little above the true one. Cannot hear so well with the left as with the right ear, but there is no complaint of deafness. *Periosteal hearing on both sides perfect.* There is no discoverable mark of any wound in the structures of the right eye, nor was such at any time made out. There is no evidence of any weakness of the left leg.

Remarks.—This case of injury to the brain, producing motor and probably auditory paralysis of the opposite side, is of great physiological interest, owing to the apparently strict limitation of the injury. Such limitation is shown by the definite nature of the functional disturbance produced, and the recovery of the patient without marked constitutional symptoms. It is, moreover, rendered probable by the character of the instrument which penetrated the orbit, an ordinary umbrella-rib, having at the end, which in the complete umbrella is connected with the central part of the frame, a tolerably sharp chisel-shaped edge.

The symptoms to be accounted for are mainly these:—

1. Left hemiplegia of the ordinary type, with rapid recovery.
2. Probable nerve-deafness on left side, with recovery.

3. The paralysis of the extrinsic muscles of the right eyeball.

1. The hemiplegia being of the ordinary pattern implies, with the greatest probability, not a cortical lesion, but some injury to the motor tract in the internal capsule of the right side.

2. The deafness (of which there was no history or suspicion previously to the accident) being an *isolated* sensory symptom, points probably either to a right-sided cortical or left-sided nerve lesion. On the justifiable assumption of the impossibility of a multiple lesion, not to mention the serious symptoms which must have resulted from the passage of the instrument to the base of the brain on the left side, the theory of injury to the auditory nerve may be dismissed, and a cortical lesion inferred.

With these inferences the facts of the case remarkably coincide. The symptoms are explicable by the supposition of an injury to the internal capsule and to the superior temporo-sphenoidal convolution (or Ferrier's auditory centre) on the right side.

The instrument above described, under the conditions alluded to, might easily penetrate the thin bone of the orbit, and having once entered the brain would have no obstacle to its further progress. The probable course of the instrument was demonstrated to me by Dr. Ferrier, who kindly studied at my request the notes of the case, and showed me by means of a skull, with surface markings corresponding to the convolutions beneath, that the spike having pierced the orbit on the outer side, and passing nearly straight backwards in a horizontal plane, might easily have traversed the region of the island of Reil, injuring the internal capsule, and then found its way into the superior temporo-sphenoidal convolution. Thus, then, the two salient symptoms of *hemiplegia* and *deafness* are satisfactorily accounted for. It will be observed that the temporary nature of the symptoms is in accord with the probable character of the wound caused by the instrument in question. Such a wound would be in itself slight; the symptoms being probably caused by the pressure of effused blood in the track of the instrument.

3. The paralysis of the extrinsic muscles of the right eyeball, which gradually disappeared with the exception of that of the external rectus, is best explained by a local injury done by the passage of the spike through the orbit, setting up a considerable degree of extravasation, and probably some inflammation of the structures in that region. The boy very clearly stated

that he was sure the spike ran into the outer corner of his eye —a fact which falls in with the more permanent injury done to the nerve of the external rectus.

P.S.—I have lately (Nov. 20, 1882) seen the boy again. I find that he has now a discharge from his left ear, which interferes with his hearing by the meatus. But his hearing is equally acute on both sides when tested by a watch placed on the skull. This discharge from his ear has taken place from time to time for several years, according to his mother's statement. While in the hospital the boy had no discharge, and the history taken at that time did not reveal any previous deafness, neither the mother nor the boy having noticed it, as appeared on inquiry.

This fact does not negative the probability of the deafness observed after the accident being caused by an injury to the auditory centre. For there was at that time a complete inability to hear a watch placed on the skull on the left side; while the present condition shows no impairment of the function of the auditory nerve. Mr. Clutton, of St. Thomas's Hospital, has kindly examined the case for me, and reports normality of the nervous apparatus on both sides, with tympanic inflammation on left and Eustachean obstruction on right side. He considers this condition to be of long standing.

COMPOUND FRACTURE OF SKULL AND ABSCESS OF FRONTAL LOBES.

BY J. M'CARTHY, F.R.C.S., ETC.

T. W., a boy aged 6 years, was admitted into the London Hospital on March 18th, 1882, with a compound fracture of the skull, caused by his having been run over by a hansom cab. There was a lacerated wound on the left temple, a little above the zygoma, communicating with the fracture, which could be traced under the skin transversely across the forehead, a little above the supraciliary ridge, forming a prominent line with a considerable depression above. Both upper eyelids were swollen with extravasated blood, and there was a small but rapidly increasing hæmorrhage beneath the left ocular conjunctiva. The pupils were active, and of equal size. The nasal bones were displaced forwards, the right in advance of the left. The child was dull and fretful, but when roused, answered intelligently, and there was no loss of motion or sensation. He had bled copiously from the nose, and vomited coffee-ground fluid. I cut down upon the apparent depression with the intention of elevating it, but found that the deformity was produced by the lower part of the frontal bone with the nasal bones having been forced forwards in advance of the rest of the skull. It thus became evident that the fracture had traversed not only the vertical plate of the frontal bone, but also both orbital plates and the cribriform plate of the ethmoid. Any effort to replace the bones in position led to such abundant escape of brain-substance that the attempt had to be desisted from. He was sent back to bed, the wounds having been dressed with lead lotion, and an ice-bag was applied to the vertex. The next morning he was drowsy, but when roused could answer intelligently, and recognised his mother. He took fluid nourishment fairly well.

During the next few days he continued much the same, but the temperature gradually rose. On the fifth day it was a little over 104° Fahr. and inunction of mercurial ointment was ordered. His gums speedily became spongy, and on the eighth day the temperature had fallen to 100° Fahr., and

continued about the same, with slight variations, for some time, gradually falling to normal. Both upper eyelids suppurated, and there was a profuse discharge of matter from them. He, however, improved in general condition, and his appetite was good, but when disturbed in any way uttered a peculiarly shrill and piercing cry.

About the sense of smell no satisfactory evidence could be obtained. All the wounds healed well and rapidly, and by April the 19th he had become so lively as to necessitate his being fastened in bed, to prevent his getting up and running about the ward. On May the 20th, slight apparent ptosis of the left upper eyelid was noticed, but on further examination it was evidently the result of the roof of the orbit becoming gradually depressed so as to interfere with the upward movement of the eyelid. The optic discs were examined from time to time, but were always normal. He was allowed to get up, and was now always playing about the ward with the other children. The nurse reported that he was sometimes obstinate, and got into a temper, but was of a lively disposition, and always had a good appetite.

May the 22nd there was renewed suppuration in the left eyelid, and a rise of temperature, which subsided again after the matter had discharged. Nothing worthy of record occurred until the evening of June the 7th, when he was seized with convulsions, and the right side of the face was especially affected. After this he again rallied, but at midnight, on the 10th, had another severe fit, in which the left side of his body was chiefly affected. Bromide of potassium was ordered. During the next week he was restless at night, had twitchings of muscles on both sides of the body, and complained of pain in the right hip, for which no cause could be discovered. On July 1st the left eye was noticed to be prominent, and a small swelling over the left frontal eminence appeared. At this part the fractured bones were found to be again separated by some growth from within the cranium pressing forwards. During the next ten days this increased, and the swelling was punctured, when some grumous matter escaped. The child now lay perfectly quiet, unless when disturbed, when he could be roused to partial consciousness. There was incontinence of urine and fæces. The stupor gradually increased until his death on the 22nd of July.

At the *post-mortem* examination the anterior fossa of the cranium was found to be fractured, and a disintegrated brain-substance from the under part of the left cerebral hemisphere protruded into the left orbit and the nasal cavity. The left frontal lobe was a diffluent mass. The right adhered firmly

to the membranes, and on separating it the grey matter shelled off, remaining adherent to the membranes, while the white substance gave very distinct fluctuation. On cutting into it, about three ounces of thick pus escaped, but the walls of the cavity were so rigid that they would not collapse.

The case is interesting, partly from the unusual nature of the fracture and resulting deformity, but chiefly from the severe injury which the anterior part of the brain must have sustained at the time of the accident without there being any symptoms to indicate it. With the exception of the sense of smell, his other faculties were normal until within about ten days of his death, and for about six weeks of his hospital life he was active, merry, and extremely intelligent for his years.

The temperature-charts exhibited occasional elevations, particularly in March 23, May 2, May 23, June 13 and 16, which were accounted for at the time by the occurrence of suppuration or diarrhœa.

HYSTERICAL MANIA: ATHEROMATOUS DISEASE OF LEFT INTERNAL CAROTID: THROMBOSIS OF ARTERIES AT BASE OF BRAIN, WITH CONSEQUENT SOFTENING OF CEREBRAL SUBSTANCE.

BY W. B. KESTEVEN, M.D.

The following case presents points of interest, both psychologically and pathologically.

On the 30th of September last, I was requested to undertake the care of a lady who was said to be suffering from a severe form of hysteria. The patient came to me in the afternoon of that day, was perfectly collected and cheerful, entered into conversation in the intelligent and self-possessed manner of an educated lady. She joined our family party at tea, and in the course of the evening sat down beside a whist table, to watch the game, as she herself said. In about half an hour she suddenly left the room and retired to her bedroom, where, on being followed, within a very short time, she was found almost in a state of nudity, complaining that her bowels would not act, and endeavouring to evacuate the rectum with her fingers. She talked incoherently, was incessantly moving about; so restless that it was impossible to keep her in bed by any persuasion, although at the same time declaring herself paralysed; she passed her evacuations under her. On the following morning she became quieter, kept to her bed the greater part of the day, but was still incoherent. She took no food except by strong persuasion. On the morning of the third day, while standing up to be dressed she suddenly fell to the ground, but got up again, all the while protesting that she had lost the use of her legs. She seated herself on the floor in the corner of the room, so as to support herself on either hand by the angles of the wall. She was cheerful, even loquacious, for a few hours.

It became necessary to remove her to an asylum, which was effected by carrying her, as it appeared that she would not, or could not, stand. The sequel shows that she could not. As the case, however, had previously been regarded as one of hysteria, her want of muscular power was doubted. Dr. Wright, of

Northumberland House, Finsbury Park, under whose care she was placed on Oct. 2nd, has obliged me by notes of her condition whilst under his observation. On admission, she was semi-comatose, and in a state of profuse perspiration. She did not move or speak. She would half-open her eyelids and gaze furtively around; her eyes were turned to the left. She seemed to be, to a great extent, aware of what was going on. She gave the impression that she was simulating paralysis and stertorous breathing. Her right side was "limp," but no marked difference from the state of the left side. No reflex action elicited on soles of the feet. Before she died there was a distinctly paralytic state of the left cheek and ptosis of the left eye. Her temperature rose to 103°, and her pulse to 130 during the day of her death, Oct. 4th. Dr. Wright has kindly furnished me with a copy of Dr. Goodhart's report of the macroscopic examination of the brain.

INSPECTION MADE 4 P.M., OCTOBER 5, 1882.

Cranial Bones.—Thin.

Dura Mater and Sinuses.—Healthy, except that dura mater was unduly adherent to skull.

Arachnoid and Pia Mater.—All looked quite healthy, but when stripped from the grey matter beneath on the left side the surface beneath was left ragged and soft; the colour being unduly yellow, from the intermixture of blood pigment.

The *vessels* were all healthy except one, and that the left internal carotid. This vessel was obviously plugged by firm substance, and on making a transverse section of this part and examining it closely, it became evident that the coats of the vessel were extensively diseased here; that atheromatous thickening had occurred, leading to considerable diminution of the calibre of the vessel. This had, of course, been going on for some time, probably many months. More recently, perhaps in the last two or three weeks, perhaps even more lately than this, the diminished channel had become completely closed by fresh clots being deposited upon the roughened arterial wall.

The transverse section of the artery thus showed an outer thick yellow zone, or partial zone, and an inner claret-coloured part.

Looked at from above, the left hemisphere was markedly fuller than the right, and bulged out, more particularly in the lower part of the anterior, central, or ascending frontal convolution. It was in this part that the membranes, when stripped, left a ragged surface behind. Making sections of this hemi-

sphere, it was found that the central cortex about this convolution—that is to say, it and the convolutions adjacent—was extensively swollen, so as to have lost its outline of demarcation from the white matter beneath; it was ecchymosed in many places, minutely vascular in many more, and everywhere had lost its consistence when compared with the opposite hemisphere and the sounder parts of this one.

The lenticular nucleus, the corpus striatum, had suffered in the same way—bloated-looking and pulpy.

It was particularly noticed, and this has an important bearing on the symptoms, that except in the superficial layers of the grey matter, which were beginning to disintegrate, there was as yet no evident solution of continuity of the brain-fibres, and thus there had been no marked paralysis.

The ventricles and other parts of brain all looked healthy.

To the preceding I would add the results of my microscopical examination of portions of the convolutions taken from near the seat of the softened substance. These may be summed up as disintegration of structure by softening—atrophy of nerve-cells, dilatation of the minute vessels, and extensive miliary degeneration—conditions, all of which are indicative of longer-standing disease than would appear from the history of the case. The patient had always been regarded as hysterical. In the month of June last she had a transient attack of hemiplegia. After this she experienced paroxysms of hysteria, bordering upon mania; she complained of tingling sensations in various parts of her body.

This case is worthy of note on account of the difficulty of diagnosis in its early stages, and the contrast presented between its later symptoms, compared with the extent of pathological lesions revealed after death.

Abstracts of British and Foreign Journals.

Stenger on Cerebral Affections of Sight in General Paralysis.— Stenger (*Archiv f. Psych.*, Bd. xiii. p. 218) reports five cases illustrative of a peculiar affection of sight which Fürstner, several years ago, described as occurring in the course of general paralysis of the insane. The symptoms are generally observed after the epileptiform seizures that occur in this disease. The patient can see objects and follows them with his eyes, but he has no ideas about them; they do not recall the associations they used to do, or call forth the same actions. For example, he shows no signs of fear if a burning stick is suddenly thrust before his face, perhaps he will try and catch hold of it. Though he sees an obstacle in his path, he will continue in his course till he stumbles over it. If a glass of wine is held before him, it does not seem to occur to him that it is for drinking; it is only after it has been pressed against his lips that he shows his appreciation of it, and drinks it. The patient sees, but he does not understand; just as a man cerebrally deaf hears, but does not understand. This condition lasts for a variable time. In the first case it lasted about ten days, and then rapidly disappeared, and sight was normal for three or four weeks, when another attack came on, which was in turn recovered from. After a series of such attacks and recoveries, during which the dementia and paralysis gradually increased, the patient died. In two cases there was absolute blindness for several days, which was succeeded by the condition of mental blindness described above.

It will thus be seen that there are two distinct conditions, one of absolute blindness or cerebral amaurosis, or, as it is called by Munk, cortical blindness, *rindenblindheit;* the other of partial blindness, psychical or mental blindness, the *seelenblindheit* of Munk.

The cases that Stenger has observed differ in some respects from those described by Fürstner. In Fürstner's cases, only one eye was affected; in Stenger's, with one exception, both eyes were involved. Stenger always found paralysis and dilatation of the pupil; while Fürstner states that the contractility of the pupil is retained.

Post-mortem examination showed that the symptoms were due to disease of the cortex cerebri; but the lesions were too diffuse to allow of any conclusion as to the localisation of the sense of sight.

Stenger reports several cases of general paralysis in which he has observed hemianopsia, without the peculiar symptoms of impaired vision just described. A man had an apoplectic seizure, and lost power over his left face and extremities. Three days afterwards, by which time he had regained consciousness and intelligence, there was found left hemianopsia and hemianæsthesia. The hemianopsia continued for about a fortnight, and then gradually disappeared, but returned in a few months after a fresh paralytic attack.

In another case, left hemianopsia appeared after a paralytic attack affecting the left side. Three months afterwards, right hemianopsia developed, and was followed by convulsions of the right arm and face. The patient was now quite blind, and remained so till death, which happened four weeks afterwards. During the early part of this period he was able to converse rationally, and the other special senses were normal. On two occasions he had hallucinations of sight. On post-mortem examination, in addition to other lesions, the occipital lobes were found to be much atrophied, and the pia mater injected and firmly adherent to the softened cortex.

Zacher on some peculiar forms of General Paralysis.—Zacher (*Archiv f. Psych.*, Bd. xiii. p. 155) relates a case in which in addition to the ordinary symptoms of general paralysis, there were the characteristic symptoms of spastic spinal paralysis. The only symptom, indeed, in which the case differed from a typical case of spastic spinal disease was the presence of a general hyperæsthesia. In similar cases that have been described by Claus, Schultze, and others, sclerosis of the lateral columns of the cord has been discovered. But in this case no pathological change was found, either in the lateral columns or in any other part of the cord. Zacher attributes the spastic symptoms to lesion of the cortex cerebri, more particularly that part of it from which the pyramidal strands spring. He suggests that a differential diagnosis between the cases of general paralysis in which there is sclerosis of the lateral columns of the cord, and those in which there is no sclerosis, may be founded on the absence in the former cases of decided sensory symptoms and well-marked contractures.

Zacher reports another case which presented the symptoms and the post-mortem appearances both of multiple cerebro-spinal sclerosis and general paralysis. Instances of a similar combination have been recorded by Claus and Schultze (see 'BRAIN,' Vol. II. p. 142; Vol. IV. p. 268).

Ganser on the Optic Nerve.—Ganser, as the result of experiments on cats (*Archiv f. Psych.*, Bd. xiii. p. 341), comes to the conclusion that the non-decussating fibres of the optic tract form a distinct bundle, which runs along the lateral border of the optic chiasma and the optic nerve.

In confirmation of this view, the author cites the case of an epileptic, in whose brain a thin band of fibres was seen to separate itself from the right optic tract a little in front of the corpora geniculata, pass forwards on the ventral surface of the tract to the lateral border of the chiasma and nerve, and afterwards become incorporated with the nerve. This view is in direct opposition to that of Kellermann, which denies the existence of a fasciculus lateralis, and asserts that all the fibres intermingle in the chiasma. Ganser analyses the case upon which Kellermann's theory is based, and declares it unsatisfactory and indecisive.

The non-decussating fibres are distributed to the temporal half of the retina, the decussating to the nasal half and to parts of the temporal half. The area centralis seems to be supplied by both kinds of fibres. After destruction of the decussating fibres of the optic chiasma, there is atrophy of the nerve-fibre layer, and of the ganglion-cell layer in the nasal portion of each retina.

Removal of the posterior parts of the left cerebral hemispheres of new-born cats caused homonymous right hemianopsia, and on the animals being killed nine months afterwards, the left optic tracts were found atrophied, and the atrophy was less extensive in the left optic nerve than in the right, showing that the decussating fibres are developed more largely than the non-decussating. The nerve-fibre layers were thinner on the left side of each retina; in other words, there was a homonymous left-sided atrophy of the layers.

Ganser's paper concludes with some observations on the anatomy of the corpus bigeminum anterius.

Meyer on a Case of Hæmorrhage into the Pons with Secondary Degeneration of the Fillet.—A man, aged 48 (*Archiv f. Psych.*, Bd. xiii. p. 63), after a slight apoplectic seizure

was found to have complete paralysis of the right facial and abducens nerves, with conjugate paralysis of the left rectus internus, anæsthesia of the left half of the face, trunk, and extremities (least marked in the face), hyperæsthesia of the right half of the face, and paresis of the left limbs. The paresis soon disappeared, leaving only ataxia of the left arm. Headache and vertigo were prominent symptoms in the case.

On post-mortem examination a hæmorrhagic focus was found in the tegmentum in the right half of the pons. The lesion was confined to the lower two-thirds of the tegmentum, and involved the following structures:—the common nucleus of the abducens and facial nerves with the root-fibres of the abducens, the inferior facial nucleus and the facial nerve, the superior olivary body, and a large portion of the formatio reticularis. The motor portion of the fifth was intact, and so also was the greater part of the sensory fifth nerve. Above the limit of the focus, there was secondary degeneration of the fillet (*schleife*), which ceased at the upper boundary of the pons; below the focus there was secondary degeneration of the olivary body and the fillet. The degeneration of the latter could be traced as far down as the level of the decussation of the pyramidal tracts, the area of degeneration being here situated at the periphery of the lateral columns immediately behind the anterior nerve-roots.

Meyer, in commenting on this case, draws attention to the absence of convulsions and myosis, two symptoms which are generally found in cases of hæmorrhage into the pons. The paresis of the left extremities is attributable to the pressure exerted by the clot on the pyramidal tracts (which were intact): as absorption proceeded, this pressure so far diminished that ataxia of the arm was the only symptom of interference with the tracts that remained. The paralysis of the left rectus internus was not due to lesion of the left oculo-motor nerve, it was probably due to lesion of the fibres of the posterior longitudinal fasciculus, which, as Duval and Laborde have shown, unites the nucleus of the abducens of one side with the nuclei of the trochlear and oculo-motor nerves of the other side.

Tuczek on Spinal Lesions in Ergotism.—Tuczek (*Archiv f. Psych.*, Bd. xiii. p. 99) reports 15 cases of spasmodic ergotism which came under his notice during an epidemic of the malady, all of which presented symptoms of spinal disease. The most frequent symptom was absence of the patellar tendon reflex. This was

observed in every case. Other symptoms were paræsthesiæ, ataxia, diminished sensibility to pain, &c. These symptoms showed themselves months after the acute intoxicant stage, when the patients were in a condition of marked cachexia. In four of the cases (one was only 9 years old, another 16) a post-mortem examination was made, and in all there was found sclerosis of the external division of the posterior columns. Pathologically, the cases differed from tabes solely in the rapidity of the development of the morbid process. The author finds an analogy to the appearance of tabes in ergotism in the occurrence of an acute primary disease of the posterior columns in exceptional cases of pellagra, lepra, absinthe-poisoning, scarlet fever, and diphtheria.

W. J. Dodds, M.D., D.Sc.

Lemoine and Lannois on Spinal Perimeningitis. (*Revue Mensuelle de Médecine et de Chirurgie*, Juin 1882).—A case is related in which paraplegia was rapidly developed five days after a slight fall, attended with symptoms resembling those of acute ascending paralysis; death occurring in this short period, attended with the phenomena of asphyxia. The autopsy showed the rare lesion of softening and disintegration of the spinal cord from pressure of pus formed in the perimeningeal cellular tissue.

A parallel case is quoted by the authors from the *Berliner Klinische Wochenschrift*, 1877, reported by Lewitzky.

Case of Pseudo-muscular Hypertrophy. By Dr. C. A. Pekelharing, Utrecht (Virchow's *Archiv*, Aug. 1882).—This case, in which the author had the opportunity of examining the spinal cord, was that of a boy fourteen years of age. His mother reported him to have been born without any abnormal appearance. Soon after birth the disproportion of the size of his head to the rest of his body was very evident. His growth was tardy. He only learnt to speak at three years of age. At four years his locomotion was imperfect; he limped, and often fell. The calves of his legs were thick; and when he was five years his arms also large, but powerless. His hands and feet suffered from cold. He maintained a half-sitting, half-kneeling posture, the thigh firmly bent to the trunk, and the legs flexed on the thighs. Any voluntary movement was difficult. The muscles, though they felt large, were very feeble; they responded only to a strong electric current. The body generally much emaciated; the thorax deformed. He suffered from disorders of the digestive and respi-

ratory organs. He died from an attack of bronchitis in three days. On post-mortem examination there were found traces of former disease in the lungs and pleura. The cranium was thick and hard.

Examination of the Spinal Cord.—The central canal in the cervical and dorsal regions was dilated and irregular in outline. Nuclei were accumulated, more especially where the canal appeared bent upon itself; in the lumbar region and conus medullaris the lumen was completely occluded by nuclei. On a level with the canal on both sides was a wide opening, which communicated with the anterior spinal fissure and contained a distended vessel which joined the anterior spinal vein.

Throughout the length of the cord the anterior horns were seen to be deficient in nerve-cells, and these only feebly coloured, whilst those in the lateral columns were unaltered. No change was perceptible in Clarke's posterior vesicular column. In some parts the connective tissue was thickened, but not symmetrically. The cells in one section were totally wanting. In the conus medullaris all the cells of the lateral regions of the horns were degenerated. In the grey substance the vessels were numerous and distended, and here the degeneration of the nerve-cells was most advanced.

In this instance, as in those recorded by Clarke, Gowers, and Ross, with the atrophy of the muscles was found degeneration of the cells of the anterior cornua of the cord. The question arises, whether the muscular atrophy is the cause of the degeneration of the nerve-cells, or whether the pathological condition of the nerve-centres is to be blamed for the malnutrition of the muscular fibres. It would seem that the latter is the right answer. In the first place it is not to be supposed that an inflammatory process could spread from the muscles along the spinal cord; neither the nerve-roots, nor the white columns of the cord, show any corresponding softening. The dilatation of the vessels and the abnormal state of the circulation in the grey matter may be assumed to be one cause of the atrophy of the muscles.

Perhaps in the case before us we may go a step further. The patient had been the subject of chronic hydrocephalus. The thickness of the skull left less space for the fluid in the ventricles. The wide lumen of the cord was evidence also of a degree of hydrorachis, the fluid of which had gradually decreased. Were the spinal cord a soft-walled tube, the removal of its fluid contents would cause a collapse of its walls. This not being exactly the

case, the walls of the central canal would yield towards its lumen, and the vessels of the surrounding structures would be affected by the diminished pressure. The vascular canals on both sides of the central canal were extraordinarily large. The hyperæmia hence induced would of necessity influence the nutrition of all adjacent structures—a state of œdema might take place in the grey matter, with a disintegration of the epithelial cells filling the central canal. Thus we revert to the opinion of Duchenne as to the cerebral origin of this form of paralysis. Notwithstanding that many cases of pseudo-muscular hypertrophy have been recorded since Duchenne's time, the presence or absence of hydrocephalus has not been noticed, although in some instances this must have had considerable influence in its causation.

Sarcoma of the Orbital Surface of the Brain, with wasting of the Gyri Recti. By Dr. Otto (Illenau). (Virchow's *Archiv*, Sept. 1882.)—This case, Dr. Otto observes, presents two points of interest: first, the origin and seat of the tumour, and secondly, its relation to the question of localisation. The patient was thirty-seven years of age, of healthy aspect, in December 1879. He inherited no special tendency to disease. Two of his brothers were rather feeble-minded. Himself had the ordinary mental powers. He was married, and the father of three children. Had been an intemperate man. In the summer of the same year, 1879, he had an attack of jaundice with giddiness, &c.: after this he became changed in manner, began to entertain suspicions that he was being poisoned, that his life was being plotted against, that his wife was unfaithful to him, &c. &c. Hallucinations of sight and hearing supervened, and lent force to his suspiciousness, and led to outbreaks of violence. About the middle of March these hallucinations diminished, his health generally improved; he occupied himself in garden-work, but his mind gradually became weaker. This amendment did not last. One day he complained of severe pain on the right side of the head; his conjunctivæ became congested; he declared that the drinking-water was mixed with blood. He became violent, diarrhœa supervened, and he died in about eleven hours.

Post-mortem examination.—The surface of brain was flattened, the veins turgid. On raising the brain an elliptical-shaped tumour was found between the dura mater and the under-surface of the frontal lobes; extending from the foramen cæcum to the sella Turcica. The connections of the connective tissue with the dura

mater were easily broken down with the handle of a scalpel; a turbid puriform fluid escaped from the centre. The measurements were: length, 6 cm. (= 2·36 Eng. in.); breadth, 5 cm. (= 1·96 Eng. in.); depth, 3 cm. (= 1·18 Eng. in.); the under-surface was irregular, occupied the position of the *gyri recti*, and extended backward to the border of the optic commissure. The olfactory nerves could be traced uninjured to the roots. The tumour was united to the cerebral substance posteriorly. Microscopically investigated, the mass was found to consist of round-celled sarcoma. No nerve-cells were to be found in the adjacent cerebral convolutions, but débris, fat and pigment cells.

What does this case teach with reference to the question of localisation? The great degree of pressure exerted on both gyri recti, and exclusively on them, might have been supposed to demonstrate their functions as satisfactorily as any set of experiments.

Hitherto no experiments have been directed to investigation of the under-surface of the orbital lobes, since they are almost inaccessible. The experiments of Ferrier have referred the seat of sense of smell to the *gyri uncinati*, but even this is open to doubt. Two cases are on record, one by Longet, the other by Nothnagel, in which the roots of the olfactory nerve were pressed upon, and the adjacent cortical substance had been absorbed, but in which, nevertheless, the sense of smell was preserved. It was in the present case, for the man was a great lover of flowers and their odours, and was a good judge of cigars. Thus it appears that this complete destruction of the cortex of the gyri recti affords no conclusive evidence as to other functions, but rather indicates that these portions of the brain possess some functions, latent and yet to be discovered.

W. B. Kesteven, M.D.

Sciamanna on the Excitation of the Human Brain. (*Arch. di Psichiatria*, 1882, p. 209.)—The author had the opportunity of applying both faradic and galvanic stimuli to the brain (chiefly through the dura mater) of a man who, in consequence of an accident, had lost part of the right parietal bone.

Excitation of the middle of the ascending frontal convolution produced contraction of masseters and closure of the jaws; of the lower third of the ascending parietal: raising of the left ala nasi and corner of mouth; of posterior central fissure (between ascending parietal and inferior parietal lobe): flexion of arm and raising of eyebrow; of posterior portion of plica supramarginalis (near

inferior temporal convolution): rotation of head to left, movements of orbicularis palpebrarum, of tongue, and eyebrow.

Bechterew on the Functional connection of the Olivary Bodies to the Cerebellum. (Pflüger's *Archiv*, 1882, p. 257.)—The author succeeded in destroying the olivary bodies in animals without opening the spinal canal, and observed the following disturbances of equilibration and motion:—

A. Forced movements, such as rolling on the long axis of the body towards the injured side, with nystagmus, one eye being turned down and inwards, the other up and outwards. When the lesion was of a slight nature: running movements forward, or "manège," or throwing the body backwards.

B. Forced positions: lying on the injured side, or rolled together towards the sound side.

C. Want of equilibration: reeling or impossibility of walking or standing when the lesion is bilateral; titubation and tendency to fall on the injured side, when unilateral.

The author has found that lesions in the neighbourhood of the infundibulum of the third ventricle also produce perturbations of the same nature; and concludes that these parts are in close functional relation with the cerebellum. It is probable that Goll's (sensory) columns terminate in the olives. Meynert thinks that the latter organs are connected by fibres within the medulla with the opposite lobe of the cerebellum; but this view is inconsistent with the fact of the rolling movements occurring towards the injured olive, as they do towards the injured cerebellar peduncle.

The general conclusions are, that we may conceive the semicircular canals, the infundibular region and the olives, as organs transmitting to the cerebellum centripetal influences derived from the acoustic, optic and tactile organs respectively, all of which are necessary to the elaboration of the outgoing equilibrating impulses.

Monakow on the external Acoustic Nucleus and the Restiform Body. (*Neurolog. Centrbl.* No. 21, 1882.)—The author divided the left half of the spinal cord immediately below the decussation of the pyramids in a rabbit on the day of its birth. Six months afterwards the brain was examined, and the following changes found: atrophy of left lateral columns of the medulla; partial atrophy of the left formatio reticularis; atrophy of the lateral cerebellar tract; atrophy of left funiculus cuneatus and

its nucleus; atrophy of the external acoustic nucleus; partial atrophy of the left corpus restiforme (inner side); partial atrophy of cortex of upper verniform process.

No change in the auditory roots, ascending trigeminus root, or inner part of cerebellar peduncle. Hence the author concludes that the external acoustic nucleus is in relation with spinal fibres, and not with the auditory nerve nor the cerebral peduncle; that the funiculus cuneatus passes partially through the corpus restiforme; that the lateral cerebellar columns terminate in the superior verniform process.

[In a paper recently read before the Société de Biologie, Laborde gave an account of some experiments made by M. Duval and himself on the semicircular canals and the corpus restiforme. Injury in both cases gave rise to the same phenomena (loss of equilibration). He describes certain fibres which he believes to start from the ampullæ and some of which go to the restiforme body, others to the cerebellum.—*Rep.*]

Lepine on Trismus of Cerebral Origin. (*Revue de Médecine*, 10, 1882.)—The author observed a case of left hemiplegia, with trismus, following hæmorrhage into the claustrum, external capsule, lenticular nucleus, and pressing upon the cortical layers of the insula and foot of the ascending frontal convolution. The trismus was tonic, and lasted the three days which intervened between the attack and the death. No other spasmodic symptom was present. The author thinks that the trismus depended upon irritation at the foot of the ascending frontal convolution, where Ferrier has localised the motor centre of the jaw in the monkey. It is strange, he adds, that the phenomenon is not more commonly met with, but quotes a few cases by various observers where trismus co-existed with lesion in the neighbourhood of ascending frontal.

It may be said in explanation of this fact that the masticatory movements require strong electrical stimuli for their production; mastication, like walking, is a less "cerebral" act than movements of the arm, for instance. This consideration may also explain, perhaps, the bilateral occurrence of trismus in unilateral lesion.

As a help to localising the lesion, trismus can scarcely give a certain clue when accompanied with simple hemiplegia, but can do so when associated with a monoplegia or aphasia.

Eisenlohr on Circumscribed Anterior Poliomyelitis in the Adult. (*Neurologisches Centralblatt*, No. 18, 1882.) The

author describes a case in which a number of muscles of the right arm and hand were degenerated, atrophied and paralysed. Atrophy was present in the interossei; the thenar muscles were atrophied and paralysed. Of the muscles supplied by the musculo-spiral the supinator longus was healthy; the triceps and extensors much atrophied and paralysed. The pectoralis major and serratus anticus were also deeply affected. The deltoid, pronators and flexors of forearm were somewhat wasted, and weaker than normal.

A careful examination of the cord showed it to be healthy, except in the cervical region, when, between the sixth cervical and first dorsal roots on the right side there was evidence of sclerotic changes. These were confined, at the upper part of the altered tract, to the postero lateral group of large cells. From the level of the lower fibres of the sixth root the whole anterior horn is implicated as far down as the level of the eighth root, the lateral portion being most altered. The white matter was normal, with the exception of the fibres forming the roots.

The author concludes with some remarks concerning the spinal localisation of the motor centres of the arm muscles. The escape of the flexors shows them to be innervated from above the sixth root. The implication of the triceps and pectoralis, on the other hand, proves them to derive their supply from the sixth to the eighth roots. The same remark applies to the extensors of the hand and fingers. The partial paralysis of the hand muscles agrees with the view that they are innervated by the eighth cervical and first dorsal roots. It is, however, difficult to explain the paretical condition of the long flexors and deltoid muscles. Unfortunately no examination had been made with reference to their reactions to the galvanic current.

Vierordt on Atrophic Paralysis of the Upper Extremity. (*Deutsches Arch. f. Klin. Med.* August, 1882.)—The discovery of the influence of the anterior horns upon the nutrition of muscles, gave rise to a tendency to assume poliomyelitis in almost every case where muscular atrophy was a prominent symptom. A reaction in the opposite sense has lately become manifest, and Leyden has severely criticised those views, asserting the frequency of neuritis as a cause of atrophic paralysis. Morbid anatomy is, by itself, insufficient to solve the problem before us. A careful clinical analysis of a large number of cases is required. Nineteen cases are described by the author, who insists upon the importance of four points of view from which to compare the phenomena observed in

each: the chronological relations of paralysis and atrophy; the localisation of paralysis in the various muscles; the presence or absence of concomitant sensory disturbance; the electrical reactions.

Analysis of his cases in the light of these considerations reaches the following conclusions:—

In peripheral paralysis of traumatic origin the motor disturbance is confined to muscles supplied by the injured nerve, and is followed by atrophy. Loss of sensation is present when the lesion is severe, but usually occupies only a part of the district involved. Electrical reactions vary with the depth and date of the lesion; near the injured spot swelling and tenderness of the nerve testify to secondary neuritis.

In peripheral neuritis the paralysis is usually confined to the province of one nerve. Muscles innervated by nerves given off high up by the affected trunk may escape. Paresis precedes atrophy, but not always. Sensation, when diminished, is not extensively so. Reaction of degeneration occurs when the disease is severe, but the responses of the various muscles involved differ greatly. There is frequently swelling and pain along the nerve.

Progressive muscular atrophy affects muscles either in a diffuse, untypical manner, or according to physiological groups, usually beginning in the small muscles of the hand. Atrophy and weakness progress together. Reactions of degeneration usually present, but not easy to demonstrate. No sensory symptoms. Fibrillary contractions frequent.

Anterior (chronic) poliomyelitis attacks physiological groups of muscles. Atrophy secondary to loss of power in ordinary cases, but may follow a parallel course. No evident sensory disturbance. Electrically, partial or complete reactions of degeneration.

A. DE WATTEVILLE, M.A., M.D.

END OF VOL. V.

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