A TEXT-BOOK

OF THE

PRINCIPLES AND PRACTICE

OF

VETERINARY MEDICINE

BY

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PREFACE TO THE SECOND EDITION.

An endeavor has been made in this edition to incorporate in the text the most important contributions to veterinary medicine made during the past three years. To this end some of the chapters were rewritten, and all of them revised and brought up to date. The book has been enlarged to the extent of fifty pages.

The reception of the first edition was very gratifying. Only a few months after its appearance all copies of the book had been sold. Most of the veterinary and many of the agricultural schools have adopted it as a text-book.

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D. S. W.

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PART I.

DISEASES OF THE RESPIRATORY ORGANS.

CHAPTER I.

DISEASES OF THE NOSE AND ADJACENT SINUSES.

RHINITIS. NASAL CATARRH.

Forms.—Depending upon the degree of inflammation the following clinical forms of rhinitis may be distinguished:

Catarrhal (acute and chronic).
Croupous.
Diphtheritic.
Follicular.

Acute Nasal Catarrh (*Rhinitis, Acute Coryza*).—**Definition**—An acute catarrh of the air passages of the head. It may occur as a primary affection or may be secondary to another disease.

Etiology.—*Primary.*—It is rarely due to the inhalation of foreign matter such as dust, chaff, grains, seeds, etc. Sponges placed in the nostrils to prevent discharge or to reduce the sound in roaring is an infrequent cause. Irritants, fumes, gases or smoke occasionally produce it. The common predisposing cause is cold. Rhinitis is most frequent during the changeable weather of the late fall and early spring. The exciting cause is undoubtedly infection.

Secondary.—Nearly all of the diseases of the respiratory tract are accompanied by rhinitis, as are specific infectious diseases, such as strangles, influenza, hog-cholera, glanders.

Symptoms.—The general condition of the patient is usually somewhat disturbed; it seems stupid, languid, and shows slight fever. The mucous membrane of the nose is swollen and reddened and at first drier than normal; later an irritant, watery discharge appears, which in a day or two becomes turbid and more profuse. In the earlier stages the patient sneezes frequently and rubs its nose against objects. The nasal discharge dries and forms crusts at the openings of the nostrils. In exceptional cases small, round, superficial erosions are noted on the mucous membrane, which usually heal in a few days. In severe cases there is conjunctivitis present. If the larynx is involved there is cough; if the pharynx, difficulty in swallowing. The submaxillary lymph glands in the horse are slightly swollen.

Course.—The course is usually rapid and the termination favorable. The condition in ordinary cases disappears in seven to ten days.

Treatment.—The acute catarrh seldom requires treatment. Protecting the patient from draughts and dust is all that is necessary in the average case. When, however, the discharge is profuse or the patient shows fever, injections into the nostrils of 1 to 2 per cent. creolin solution are recommendable. Inhalations of volatile substances are of value. Turpentine or benzoin (1 to 5 per cent. in form of steam) may be tried. The crust on the nostrils may be greased with vaselin. In man the "snuffing" of the fluid extract of Hamamelis every two or three hours is much employed.

Chronic Nasal Catarrh (*Chronic Rhinitis, Ozena*).—Chronic rhinitis rarely develops from the acute. This form is nearly always secondary to some other disease involving the respiratory tract.

Etiology.—Bad teeth with filling of the maxillary or frontal sinuses with pus. It may also accompany glanders, tumors (polypi) in the nostrils, animal parasites (estrus larva in sheep), chronic bronchitis, verminous bronchitis. In surgical conditions of the head such as necrosis of the turbinated bone, empyema of the sinuses, etc., nasal catarrh is noted.

Symptoms.—The principal symptom is nasal discharge, which varies greatly in quantity and character. The

discharge may be quite copious, or, on the other hand, very limited. As a rule, even in a given patient, the discharge is greater at times (after a long drive and when the horse is unreined). In character the discharge may be mucous and viscid, or purulent or even blood streaked (glanders, polypi). It is often fetid (teeth, necrosis of turbinated bones, polypi). The discharge if limited may dry at the nostril openings, forming green to brownish crusts (glanders), or it may flow over the upper lip depigmenting the skin over which it passes.

The mucous membrane assumes usually a leaden hue, although it may appear brown. It is at times swollen, nodular and pitted with round erosions or superficial ulcers. The swelling may interfere with breathing causing nasal dyspnea with sound, heard particularly at inspiration. From a filling and distention of the turbinal bones with exudate, the nasal septum may become atrophic and distorted through pressure leading to partial occlusion of the nasal passages. The submaxillary lymph glands become indurated and enlarged, but usually do not adhere to the jaw.

Diagnosis.—While the diagnosis of chronic catarrhal rhinitis is not difficult, to determine whether it is primary or secondary often requires careful judgment. A thorough inspection of the teeth should be made in all cases, especially where the discharge is fetid and unilateral. Tumors may often be felt with the aid of a urinary horse catheter or nasal tube. A veterinary rhinolaryngoscope (Polonsky-Schindelka) is useful in diagnosis. If the tumor is of considerable size, the air current from the partially occluded nostril will be less in volume. A sudden increase in the discharge when the head is lowered after a drive speaks for sinus empyema or more rarely filling of the guttural pouches. In filling of the head sinuses an exploratory puncture with a gimlet or drill will reveal the presence of exudate in these cavities. In pulmonary abscess the expirium is fetid and nasal discharge occurs. The discharge is bilateral, usually not copious, is accompanied by cough, and the submaxillary lymph glands are generally not involved. Percussion and auscultation usually determine the existence of lung lesions.

Where glanders is suspected the complement-fixation, mallein, agglutination, precipitin or guinea pig tests may be applied. (See Glanders.)

Treatment.—The treatment of chronic nasal catarrh is largely surgical (trephining sinuses, extraction of diseased teeth, removal of tumor, etc.). At any rate it is governed by the primary disease which the discharge accompanies. The local treatment of the nose by irrigation or inhalation is of little value except in purely primary cases and then it must be persisted in for weeks before permanent healing is affected. The following combination may be tried:

℞—Tannoformi	ʒvj
Mentholi . .	ʒss
Alcohol (50 per cent.)	Oj
Aqua dest.	Oss

M. D. S.—Apply as a nasal douche.

Croupous Rhinitis.—Definition.—An inflammation of the mucous membrane of the nasal chamber with the formation of pseudomembrane.

Occurrence.—Seen in the horse and ox. Enzoötics have been noted in the horse.

Etiology.—It is attributed to the inhaling of irritant gases and smoke. It is undoubtedly due in many cases to microorganisms. It may follow suppurative mastitis or metritis in the mare and cow, and may be secondary to malignant head catarrh, purpura hemorrhagica, and strangles.

Symptoms.—An intense inflammation of the mucous membrane which is deep red and greatly swollen. Patches of gray or yellow red fibrinous membranes form. The false membrane sloughs off in a few days leaving behind raw bleeding surfaces which soon heal without scar. There is a free discharge, which may be blood-streaked and containing shreds of croupous membrane. The submaxillary lymph glands and vessels are swollen and sensitive. There may be nasal dyspnea. Temperature is elevated.

Course.—Usually ends in healing in about one week.

Treatment.—It is recommended to touch the affected parts with hydrogen dioxid, full strength, to each 2 ounces

of which has been added a dram of sodium bicarbonate. Do not forcibly remove the membranes. Affected animals should be isolated.

Diphtheritic Rhinitis.—Definition.—A necrotic inflammation of the mucous membrane of the nasal cavities.

Occurrence.—Seen in horses and cattle. It is usually secondary to such infectious diseases as acute glanders, purpura hemorrhagica, malignant head catarrh.

Etiology.—A primary diphtheritic rhinitis may result from the action of any mechanical, thermic, chemical or infectious irritant which is sufficiently intensive to destroy the mucous membrane. In all probability the necrosis bacillus is a factor. As noted, diphtheritic rhinitis may be secondary to certain infectious diseases.

Symptoms.—The condition is characterized by the appearance of yellowish-gray patches in the mucosa which when detached or sloughed leave behind deep, dark red or grayish-red ulcers. The borders of the ulcers are thickened and surrounded by a zone of reactive inflammation. The submaxillary lymph glands are swollen.

Diagnosis.—Glanders should be thought of and excluded before a diagnosis of "primary diphtheritic rhinitis" is made. In purpura hemorrhagica the differential diagnosis is less difficult (petechiæ, typical cutaneous swellings). The eye affection absent in primary nasal diphtheritis is characteristic of malignant head catarrh of cattle.

Treatment.—See Croupous Rhinitis.

Follicular Rhinitis (*Pustulous Coryza*).—**Definition.**—An inflammation of the nasal mucous membrane involving particularly the mucous glands forming pustules and later ulcers. The sebaceous glands of the skin of the nostrils may also become affected.

Occurrence.—Peculiar to the horse. It may occur enzootically.

Etiology.—Undoubtedly microörganisms. The Streptococcus equi has been accused.

Symptoms.—The symptoms are those of a severe nasal catarrh with the presence of numerous small nodules of the size of flea bite which appear on the nasal septum. By con-

fluence large nodules may form. The nodules soon undergo puriform softening, become yellow and form ulcers which heal in a few days without leaving a scar. A similar eruption may occur on the skin of the nostrils and lips. The lymph vessels of the cheek are sometimes greatly distended, abscesses forming rapidly along their course. The submaxillary lymph glands become enlarged and sensitive. A follicular (granular) conjunctivitis may be present.

Diagnosis.—The disease might be confused with glanders. The benign course and rapid, clean healing of the ulcers make the differentiation not difficult.

Treatment.—Treat as in catarrhal rhinitis. Ulcers may be dressed with any strew-powder (iodoform, compound alum powder, etc.). The swollen lymph vessels may be smeared with gray mercurial ointment.

EPISTAXIS.

Definition.—Bleeding from the nose.

Etiology.—(*a*) *Heredity.*—In some families of horses it is an inherited predisposition. In such cases (hemophilia?) epistaxis occurs without apparent cause. It may follow overexertion in race horses (predisposition?).

(*b*) *Traumatism.*—Epistaxis commonly results from direct or indirect traumatism. Passing objects up into the nostrils (sponges, straw whisps, nasal tubes, etc.) is often followed by bleeding. In forcibly ejecting dust and foreign matter from the nose horses sometimes induce hemorrhage. Fractures of the jaw (maxillary process) and of the base of the cranium may be followed by nose bleed.

(*c*) *Heart and Lung Diseases.*—In animals suffering from chronic heart and lung diseases this diathesis is present.

(*d*) *Pressure on Jugulars.*—Continued pressure on the jugulars may be a cause.

(*e*) *Infectious Diseases.*—In many of the acute infectious diseases it is a symptom (anthrax, purpura hemorrhagica, glanders, septicemia), and in such blood diseases as leukemia, pseudoleukemia, anemia, etc.

(*f*) *Tumors.*—Tumors in the nasal chambers (angiomas, polypi, sarcomas) induce bleeding.

Symptoms.—The blood either drops from the nostril or flows in a thin stream. It is not foamy and not attended by dyspnea. There are no general symptoms unless the hemorrhage is copious or recurs frequently where it causes general anemia (pale mucous membranes, small rapid pulse, anxious expression, dyspnea). Repeated hemorrhages at frequent intervals may become fatal in six to nine days.

Diagnosis.—It is usually not difficult to determine the origin of the hemorrhage but to find the cause may require a most careful examination of the patient. Pulmonary hemorrhages are characterized by a bright red, foamy, bilateral, nasal discharge accompanied by cough, dyspnea, and weak pulse. On auscultation of the chest, rales are heard. However, if from a nasal hemorrhage some of the blood is aspirated into the lungs symptoms of pulmonary hemorrhage may be simulated, complicating the diagnosis. In gastric hemorrhage the blood is more or less clotted, brown in color, of acid reaction, and mixed with food particles. In the horse and ox it may be ejected through the nostrils, but in the hog and in carnivora it is vomited through the mouth.

Treatment.—The patient should be kept in a cool, quiet place. If the hemorrhage is severe enough to warrant it, inject into the nostrils adrenalin in salt solution (1 to 5000). Tincture of ferric chlorid is serviceable. If injections do not suffice tampon the nostril or nostrils with gauze soaked in adrenalin. In the horse and ox tracheotomy should be performed before plugging both nostrils. Intravenous injection of a 2 per cent. gelatin solution in salt solution has been effective. In slight hemorrhages turpentine may be given internally. When bleeding comes from an angioma in the septum touching with lunar caustic may stop it.

INFECTIOUS RHINITIS OF SWINE. SNUFFLES.

Definition.—An infectious disease of the nose, ethmoid and brain of young pigs due to the Pseudomonas pyocyaneus, the microörganism of blue pus.

Natural Infection.—Takes place through the nose from the pigs rooting in infected straw, manure, etc.

Symptoms.—The prodromal symptoms are those of fever (loss of appetite, languor). Nasal symptoms soon appear. The patient passes air rapidly in and out of the nostrils, producing a snorting or sniffling sound; the snout is frequently rubbed against objects. There is bloody nasal discharge mixed with pus. The snout becomes edematous and swollen. In severe cases in about the third day of the attack, symptoms of cerebral excitement occur, the pig showing rabiform symptoms and convulsions.

Course.—The usual course of the disease is from two to seven days. Sometimes it assumes a chronic form. Recovery is rare.

Diagnosis.—The high fever, rhinitis with pronounced brain symptoms and without deformity (bulging) of the facial bones are characteristic. It is distinguished from rachitis by the absence of fever, facial deformity and dropping of the hard palate in this disease.

A sporadic nasal catarrh occurs in swine. The symptoms of nasal discharge (often blood-tinged), snuffling, etc., resemble infectious rhinitis. The disorder, however, is benign, produces no general symptoms except in neglected cases, and once the predisposing causes (damp, draughty stys, cold cement floors, lack of adequate bedding; inhalation of dust, irritant vapors, etc.) are removed healing is usually prompt.

Actinomycosis and tuberculosis may cause thickening of the snout and nasal discharge in swine. The course, however, is chronic and there is no nasal hemorrhage. In doubtful cases a microscopic and bacteriological examination of the discharge may be made. It might be confused with hyperacute cases of hog-cholera, in which at times epistaxis and brain symptoms occur. The epizoötic character of this disease, the bowel lesions on postmortem and the absence of rhinitis make the differentiation usually easy.

Treatment.—Of little avail. In valuable pigs irrigating the nostrils with bichlorid solution (1 to 1000) may be tried.

It is usually better to slaughter the affected animals and thoroughly disinfect the pens. As the disease is spread by the infected nasal discharge, a separation of the healthy from the sick is indicated.

ANIMAL PARASITES IN THE NOSE AND SINUSES OF THE HEAD.

Grub in the Head of Sheep. Œstrus Ovis.

Definition.—A catarrh of the nasal chambers and sinuses of the head due to the larvæ of the bot fly, Œstrus ovis.

Occurrence.—Found in all countries where the sheep bot exists. Australia is said to be exempt. Few American sheep are free from grub.

Etiology.—The sheep are usually attacked by the parent bot fly during the hot season. It is said that the fly may even be active in winter in warm, sunny sheepfolds.

Life History.—The larvæ are deposited by the swift-flying female bot on the margin of the sheep's nostrils, from whence they crawl up into the nasal chambers, cavities of the turbinal bones, ethmoid cells and even horn cores. It is possible that the brain is invaded in some cases. They attach themselves to the mucous membrane to feed and develop. They usually remain in the sheep about ten months, or until the following spring when they leave *via* nasal passages and reach the ground. In the soil in one or two days they pass into the chrysalis form out of which emerges in six to eight weeks the mature bot fly. The impregnated female bot seeks sheep herds. She usually hides in cracks and crevices in the sheep barn or on shrubbery or underbrush near the sheep pasture. Sheep try to avoid the attacks of the fly by running away from it with their noses held close to the ground or by grouping themselves in a great mass, their heads toward the center. If the fly touches the nostril, the sheep will snort, stamp its feet and rub its nose on the forelegs or ground. They often seek dusty places in the pasture to avoid the fly. Once the larvæ have gotten into the head,

however, the sheep become quiet and remain so until the emigration of the parasite begins.

Symptoms.—If only one or two grubs are present, beyond a slight nasal discharge, the sheep may show no symptoms. When large numbers are in the head, however, the patients show profuse mucopurulent or even bloody nasal discharge, sneezing, snorting, sniffling, shaking the head and rubbing the nose against objects. In severe cases symptoms of vertigo occur, the patient staggering, reeling, and falling to the ground. Sometimes fatal convulsions are noted. Conjunctivitis may be present. As a rule, however, in about ten days after the first signs appear, and the larvæ are expelled, the symptoms subside.

Diagnosis.—Grub-in-the-head may be confused with sturdy or gid (Cœnurus cerebralis). However, in sturdy the older sheep are attacked, there are no nasal symptoms and the forced movements of the patient are more pronounced. Gid is furthermore as yet an uncommon disease in the United States. In doubtful cases a postmortem or the microscopic examination of the discharge must decide.

From verminous bronchitis it may be distinguished by the absence of the cough and rales on auscultation so characteristic of bronchitis.

Treatment.—Very unsatisfactory. Trephining the sinuses and removing the larvæ with forceps is only practicable in isolated cases and among valuable sheep. Nasal douches do not reach the larvæ in the sinuses. In severe cases slaughter is the most economical disposition.

Prevention.—It is recommended to place in the pasture field a log in which a number of 2-inch augur holes have been bored. Salt is put in the bottom of each hole and around the margin tar. When the sheep attempt to get the salt the nostril becomes tar-smeared, which partially, at least, protects against the bot fly. Allowing the sheep constant access to lime is of some value. Plowing a few furrows in the pasture serves to furnish dust in which the sheep may burrow their noses to avoid the fly.

CATARRH OF THE MAXILLARY AND FRONTAL SINUSES.

Definition.—A collection of mucopurulent exudate in the sinuses of the head.

Occurrence.—Commonest in the horse, but is seen in the ox. In cattle the frontal sinuses are usually involved.

Etiology.—Most frequently due to diseases of the teeth (caries dentium; alveolar periostitis). In cattle it may follow dehorning. Certain infectious diseases such as glanders, malignant head catarrh, etc., may induce it.

Symptoms.—Nasal discharge which is often unilateral. It may be intermittent, copious or limited, and sometimes fetid. In some cases the bone covering the sinuses is atrophied and bulged outward, distorting the face. There may be conjunctivitis and partial occlusion of the nasolacrimal duct. Swelling of submaxillary lymph gland of the affected side is usually noted.

Diagnosis.—With a drill or gimlet bore into the suspected sinus.

Treatment.—Surgical.

CATARRH OF THE GUTTURAL POUCHES.

Definition.—An accumulation of exudate in the guttural pouches. In some cases the pouch is filled with solid, cheesy bodies, each of about the size of a bean (dried pus). Distention of the pouch with gas may result from a decomposition of the exudate.

Catarrh of the guttural pouches is rare. It is mainly of surgical interest.

Etiology.—Rarely foreign bodies; spread of inflammation from neighboring organs; infection (glanders).

Symptoms.—Similar to those of chronic nasal catarrh. Swallowing and breathing may be interfered with. The discharge is usually unilateral, mucopurulent, fetid, and increases when the head is lowered. In typical cases the parotid region is swollen and if pressed upon the discharge is temporarily increased, dyspnea induced and a roaring sound

emitted. Abscess formation in the parapharyngeal lymph glands (strangles) might be confused with filling of the guttural pouches. In doubtful cases a surgical operation (opening the abscess from in front of the wing of the atlas) will assist in differentiation. When glanders is suspected mallein should be used for diagnosis.

Treatment.—Surgical.

TYMPANY OF GUTTURAL POUCH.

Definition.—A distention of the pouch with air or gas.

Occurrence.—Colts born with it. In older horses may also occur secondary to catarrh of pouches.

Etiology.—A congenital deformity of the tube or its valve. Secondary to catarrh of the pouches.

Symptoms.—An elastic, pneumatic swelling in the parotid region. May induce dyspnea, roaring, and dysphagia. Nasal discharge may fail.

Treatment.—Surgical. Rarely successful.

CHAPTER II.

DISEASES OF THE LARYNX.

LARYNGITIS.

CLINICALLY laryngitis may be classified into:
Catarrhal (acute and chronic).
Croupous.
Edematous.

Catarrhal Laryngitis.—Definition.—A superficial inflammation of the mucosa of the larynx. Usually the upper part of the trachea and often the pharynx are also involved (laryngotracheitis, laryngopharyngitis).

Occurrence.—Catarrhal laryngitis is one of the commonest diseases of horses and cattle. It frequently assumes an enzoötic form, being very prevalent during the changeable weather of spring and fall. The disease may be primary or secondary.

Etiology.—The causes of primary laryngitis are: Refrigeration, inhalation of irritant dust, gases, ingestion of fermenting foods (brewer's grains, distillery slops, potato residue), continued bellowing of cattle, throat latch of bridle too tight, and primary infections.

Secondary laryngitis accompanies many of the infectious diseases, especially influenza and strangles of the horse, tuberculosis of the ox, cholera of swine, and verminous bronchitis of sheep. A spread of inflammation from neighboring organs (pharynx, trachea) may induce laryngitis. The causes of chronic catarrhal laryngitis are the same as those of the acute form, the irritant acting mildly but repeatedly or persistently.

Symptoms.—A dominant symptom of laryngitis is a dry, harsh cough which the patient seeks to suppress. It is especially noticeable when the animal is brought out into

cold air or given a cold drink of water. Excitement also induces cough. The larynx is sensitive to pressure which may bring about a paroxysm of coughing. In some patients hoarseness is evident. On auscultation over the larynx stenotic, rough, sometimes whistling tones are heard which tend to diminish in intensity toward the chest. Nasal discharge is usually present. If the pharynx is also involved (laryngopharyngitis) there will be dysphagia. The lymph glands of the submaxillary region are swollen and sensitive. Except in secondary laryngitis the pulse and temperature remain about normal. Dyspnea is only present when there is marked swelling of the mucosa.

Diagnosis.—The diagnosis depends upon the presence of cough, sensitiveness of the larynx, mild fever, and the negative evidence adduced from a thorough examination of the lungs. Secondary laryngitis may be distinguished from primary forms by the bigh temperature, general depression of the patient and symptoms of the primary disease.

Course.—In acute catarrhal laryngitis the course is usually six to ten days, ending in complete recovery. Neglected cases may become chronic and last for months, causing persistent, obstinate cough but usually no further symptoms.

Treatment.—The patient should be allowed fresh air (not too cold) free from drafts, dust, and stable gases. If the weather permits, exercise in the open should be allowed. In mild attacks horses may be employed at light work provided they are protected against high wind or drenching rains. The food should be laxative (roots, grass) and free from dust.

Priessnitz applications to the throat are valuable. During the early stages inhalations of steam (camphor, turpentine, creolin) are of service. On the other hand in the later stages when the mucosa is covered with tough mucus, such solvent agents as common salt solution or bicarbonate of sodium (1 to 3 per cent.) are effectual. If the exudate is abundant and fluid, astringents (alum 1 to 25 per cent., nitrate of silver 0.5 to 1 per cent.) are indicated. In horses these remedies may be injected through a small catheter inserted in the nose. Internally heroin in the form of

glycoheroin (1 to 2-oz. doses three times daily) is of service. Very popular is "Equine Cough Syrup" (Parke Davis). The following prescription is effectual in horse practice:

℞—Heroini hydrochloridi	gr. viij
Potassii acetatis . .	ʒiij
Tinct. aconiti . .	ʒiv
Spiritus ætheris nitrosi .	ʒiv
Syrupi . . . . q. s.	Oj

M. Sig.—An ounce every two hours for cough and fever in horse.

For expectorant powders which may be used see Bronchitis.

Chronic laryngitis in large animals may be treated by intralaryngeal injections through the cricotracheal ligament, using a special curved hollow needle. Usually 1 oz. of the following solutions is used: 0.5 per cent. acetate of lead, 0.1 per cent. nitrate of silver, 0.5 per cent. alum. The bowels should be kept open by administering Carlsbad salts.

Obviously in secondary laryngitis accompanying contagious diseases the separation of the sick from the healthy, and a thorough disinfection of the premises should be enforced.

Croupous Laryngitis (*Membranous Laryngitis*).— **Definition.**—A form of laryngitis characterized by the development of a fibrinous pseudomembrane on the laryngeal mucosa. Usually the pharynx and trachea are also involved.

Occurrence.—This is a rather rare disease occurring primarily in sheep and cattle and more rarely in horses and swine.

Etiology.—Croupous laryngitis may be either primary or secondary. Primary cases result from the inhalation of irritant gases, smoke, heated air, etc. Occasionally following a stable fire or the use of strong irritant disinfectants cases occur. Sheep driven through deep dust to increase the weight of the wool are sometimes attacked. The exciting cause, however, is evidently infection with probably the necrosis bacillus or streptococci.

Secondarily, croupous laryngitis is a symptom of malignant head catarrh, necrotic stomatitis of calves, purpura, glanders (acute), rinderpest, etc.

Symptoms.—The disease begins as an acute catarrh of the larynx that develops rapidly, producing severe dyspnea, which reaches a high degree in one or two days. The temperature is high, chills occur, and the patient is prostrated. There is loss of appetite. A prominent clinical symptom is dyspnea associated with loud laryngeal tones, swelling, and sensitiveness in the region of the throat. Slight pressure produces spasmodic cough which may lead to apnea. During the act of coughing the patient ejects at first quantities of mucus and pus through the nostril. Later croupous masses may be coughed up which usually temporarily or permanently improves the condition. The lymph glands of the submaxillary region are swollen and tender. In some instances the feces are covered with flakes of mucus or fibrin.

Course.—Very acute. In fatal cases death usually results in three days from asphyxia, or the patient may die in ten days.

The prognosis is fair. About 50 per cent. of the cases recover.

Diagnosis.—The high fever, peculiar hoarse cough, stenotic (roaring) inspiratory tone and the coughing up of fibrinous masses are the most important diagnostic points. One should be on the lookout for foreign bodies, edema of the glottis, and tumors in or near the larynx from the standpoint of differential diagnosis.

Treatment.—The inhalation of alkalies such as lime water repeated every two hours and infrictions over the throat are recommended. The principal indication, however, is an early tracheotomy. Alcohol per rectum may also be used.

Edematous Laryngitis (*Edema of the Glottis*).—**Definition.**—By edema of the glottis we understand a serous infiltration of the submucous connective tissue of the vestibule of the larynx (ventral surface of the epiglottis, epiglottic-arytenoid folds and saccules of the larynx). The edematous swelling produces marked stenosis of the larynx with resulting severe dyspnea. According to cause we may distinguish:

(*a*) An inflammatory edema.

(*b*) A non-inflammatory edema.

The inflammatory edema may be primary due to the causes of inflammation, or secondary and associated with purpura, glanders, pyemia or pneumonia.

The non-inflammatory edema is the result of the venous congestion of the larynx due to chronic heart diseases, traumatic pericarditis of the ox, and compression of the jugulars in horses from ill-fitting collars.

Symptoms.—The symptoms in both of these conditions are much the same except that the non-inflammatory form usually develops more slowly. Clinically, edema of the glottis is manifested by a severe inspiratory dyspnea of sudden development. The patient shows marked "air hunger" by standing with head held extended, anxious expression, general outbreak of sweat, forefeet apart, and rarely saliva and food dropping from the nose and mouth. The dyspnea is attended by a loud roaring sound occurring at each inspiration. The mucous membranes become bluish and the pulse and respiration increased. Sometimes paroxysms of cough are present. Unless relieved the patient may die in a few hours. Some cases recover spontaneously however.

Prognosis.—The prognosis is not favorable unless treatment is administered early.

Treatment.—The treatment consists in performing tracheotomy, and in cases due to heart diseases, etc., blood letting may be practised. In milder cases Lloyd's lobelia (20 c.c. subcutaneously) has given good results.

PARALYSIS OF THE RECURRENT NERVE.

Roaring.

Definition.—Roaring may be defined as an unsoundness characterized by dyspnea (inspiratory), and cough due to paralysis of the left recurrent nerve. The condition is always chronic and can be relieved in about 80 per cent. of the cases by operation.

Etiology.—From a practical standpoint recurrent paralysis may be classified as primary or secondary.

The causes of the primary paralysis are not understood.

It seems as if heredity played a part in that stallions and mares which are roarers transmit the tendency to their progeny. The condition usually does not develop until about the fourth to sixth year. As a rule, only the left side is affected, although exceptions are noted.

Secondarily, recurrent paralysis may be a sequel to influenza, strangles, and dourine, or it may follow an attack of forage poisoning or poisoning with lead or, more rarely, goitre or direct injury to the nerve itself.

Symptoms.—Usually in primary cases the disease comes on gradually. It is at first but slightly developed, the patient only emitting a noisy sound when exercising. As a rule, if the horse be at rest no signs of the disorder are noticed. If the upper rings of the trachea be pinched a prolonged, hoarse throat cough is heard. In many instances, however, cough is absent. It is sometimes possible to cause the patient to emit a peculiar grunt if it is struck a sudden, unexpected blow with the hand on the side of the chest. The principal symptom of roaring, however, is the audible laryngeal sound emitted during and increased by exercise. The quality of the sound suffers many modifications from a whistle to a pronounced roar, which in some animals can be heard a distance of several yards. Pressing the left or right arytenoid cartilage with the index finger increases the sound. In well-developed cases sufficient exercise can produce apnea. By compressing the nostrils to one-half their normal dilatation the sound is temporarily diminished. Generally the roaring sound ceases after five to ten minutes' rest but returns again during exercise. In mild cases it stops as soon as the animal is "pulled up" after a hard gallop. In bad cases the dyspnea is both expiratory and inspiratory.

Diagnosis.—The examination of the patient should be made under motion. The horse may be ridden, galloped on a lunging line or led behind a buggy. In some mild cases the animal may suppress the sound by extending the head. To avoid this the head should be drawn in, the neck held well arched. The use of the laryngoscope which shows the asymmetry of action of the arytenoids is of great value in diagnosis.

Course.—The course of primary roaring is chronic. Due to the increasing atrophy of the cricoarytenoid muscles on the left side the condition grows worse with time. Many roaring horses may be used for light slow work, while others are practically worthless for service. This depends largely upon how deep into the lumen the arytenoid cartilage sinks and whether or not the hypertrophic fellow muscles of the opposite side can act as compensators for the diseased ones of the left.

Some cases of secondary roaring (forage poisoning, laryngitis, strangles) recover spontaneously in four to six months.

Prognosis.—About 80 per cent. can be either relieved or cured by surgical interference.

Treatment.—The most successful treatment consists in the removal of the laryngeal saccule of the affected side.

TUMORS IN THE LARYNX.

Occurrence.—In domestic animals tumors in the larynx are not common. Occasionally we run across them in horses and cattle. The commonest ones are cysts which develop on the anterior surface of the epiglottis or exceptionally, below the cricoid cartilage. They attain the size of a hen's egg and are filled with a slimy fluid. Fibromas, lipomas, melanomas, and carcinomas are rare. Chondromas sometimes occur in horses involving the arytenoid and cricoid cartilages and produce symptoms of roaring. Polypoid tumors occur associated with chronic laryngitis. In cattle tubercular and actinomycotic growths are not uncommon.

Symptoms.—Tumors of the larynx produce dyspnea and dysphagia. The dyspnea resembles that noted in roaring. It is, however, not infrequently intermittent and occasionally tends to decrease rather than increase on exercise. There is sometimes blood-streaked nasal discharge. A diagnosis can usually be arrived at by palpation through the mouth, the use of the laryngoscope or an exploratory opening of the larynx. The treatment is surgical and consists in the extirpation of the tumor. (See Surgery.)

CHAPTER III.

DISEASES OF THE BRONCHI.

CATARRHAL BRONCHITIS.

Definition.—By the term bronchitis an inflammation of the larger bronchi is understood (macrobronchitis). Bronchiolitis (microbronchitis) is used to express an inflammation of the capillary bronchi (bronchitis capillaris).

Occurrence.—Bronchitis occurs either as a primary or secondary disease. It is very common among all domesticated animals particularly during the spring and fall when it may become enzoötic among horses and cattle. It may occur alone but is usually associated with tracheitis and laryngitis (catarrh of the air passages), or on the other hand may attend pneumonia (bronchopneumonia).

Forms.—Several different forms of catarrhal bronchitis are recognized. When the exudate is fluid and abundant, moist bronchitis or blenorrhea of the bronchi is spoken of. If the exudate is rather limited and not so fluid a dry bronchitis exists. A fetid bronchitis develops from a bacterial decomposition of the exudate. From the standpoint of course, catarrhal bronchitis may be either acute or chronic, and from the causes a verminous and a mycotic bronchitis may be distinguished.

Etiology.—The causes are usually refrigeration (changeable weather), inhalation of mechanical and chemical irritants (dust, smoke, chemical fumes), aspiration of fluids such as liquid medicines unskilfully administered, blood, pus or solid matter such as food which gains access to the windpipe especially when the pharynx is paralyzed. Certain animal parasites (strongylus) and bacteria are also causes.

Secondary bronchitis occurs with most of the infectious diseases affecting the respiratory tract (influenza, strangles, tuberculosis, hog-cholera).

Symptoms.—The characteristic symptoms of acute catarrhal bronchitis are cough, which is at first short, dry and painful, but later with the accumulation of liquid exudate becomes looser and less painful. Nasal discharge is present and during the act of coughing bronchial slime is ejected through the mouth and nose. In the early stages especially the respirations are increased. Percussion is normal and on auscultation rales are heard. Rales may be absent in the early stages but will appear generally about the second or third day. The character of the rale will depend upon the size of the bronchus involved and the consistency of the exudate. In the larger bronchi, provided the exudate is rather fluid, the rale is of the character of bursting large bubbles, while in the smaller bronchi the rales are much finer and of a crepitant character. If the bronchial mucosa is much swollen, narrowing the lumen of the bronchi, whistling, piping or hissing tones may be heard. As a rule the animal shows fever in the early stages (104° to 106° F.), but usually within two or three days the temperature drops. With the continuance of the fever the pulse frequency increases.

Chronic Catarrhal Bronchitis.—Chronic catarrhal bronchitis usually develops from the acute form. It may occur, however, as a symptom of chronic heart and lung disease. It is very frequently associated with chronic pulmonary emphysema or seen to accompany such chronic infectious diseases as tuberculosis, glanders, or verminous pneumonia. Generally speaking, chronic bronchitis leads to irreparable injury not only of the walls of the bronchi but of the neighboring lung tissue (parabronchitis, bronchiectasis, atelectasis, emphysema). The symptoms of chronic bronchitis are much the same as those of the acute except that the condition is feverless and suffers many exacerbations and remissions. The general condition of the patient may not be much disturbed, and the only evidences of the disorder are chronic cough, dyspnea, and nasal discharge which is often foamy and white in appearance. Obviously if chronic bronchitis is a symptom of an infectious disease like tuberculosis or glanders, the symptoms which typify these con-

ditions will be associated with those of bronchitis. In practice chronic bronchitis is most commonly met with in horses suffering from "heaves" (pulmonary emphysema). It also occurs frequently in dairy cows in the Eastern States, especially during raw, damp weather. The principal symptoms are a persistent cough and slight nasal discharge which is usually wiped off with the tongue. There are no constitutional symptoms. The course is benign except in neglected cases.

Course.—The acute form usually terminates in two to three weeks in healing. When the smaller bronchi become involved (bronchiolitis) the course is more prolonged and is apt to lead to bronchopneumonia and death. Death may also result from pulmonary edema.

Chronic bronchitis may last for months or years depending upon the cause. Generally in time the patient becomes anemic, cachectic, and finally death results from inanition.

Diagnosis.—The diagnosis of bronchial catarrh is, as a rule, not difficult. The presence of the characteristic rales or rhonchi are evidence enough especially when taken into consideration with the other symptoms and course of the disease. It is sometimes impossible to determine whether the bronchitis is primary or secondary. Generally, however, when bronchitis is secondary to some acute infectious disease the high temperature which the patient shows is indicative. When associated with a chronic infection a thorough clinical examination of the patient will usually reveal the presence of a primary disease (tuberculosis, tuberculin test; glanders, various tests).

Prognosis.—A primary catarrh of the larger bronchi is usually more benign than that of the bronchioli. In very young or very old animals on account of the prevalence of bronchiolitis the prognosis is not as good as in animals in the prime of life. Bronchitis, the result of inhalation of smoke or fire, is usually diffuse and dangerous. If pneumonia develops, which is not common, obviously the prognosis is not so good.

Bronchites which are secondary are governed, from a prognostic standpoint, by the course of the primary disease.

Treatment.—The patient should be kept in a light, clean, well ventilated place and every attention given the hygiene of the skin. The horse should be covered with a light blanket and the legs, if cold, rubbed and wrapped in soft bandages. If the dyspnea is marked an oil of mustard infriction (1 to 12) should be applied to the chest. The food should be laxative (bran, oats, grass, carrots). The bowels should be kept open by using Carlsbad salts. If the cough is dry and painful, inhalants (camphor, turpentine, or creolin) may be administered. Of value in dry bronchitis is temperate, moist air which may be created with a spray or placing water in buckets in the stable. Tartar emetic combined with heroin is of some use as an expectorant and to allay distressing cough. The following prescription is suggested.

℞—Heroini		gr. viij
F. E. hyoscyami or belladonnæ		ʒj
Ant. et potassi tart.		ʒiv
Syrupi	q. s.	Oj

Sig.—One ounce every two hours.

If the cough is troublesome treat as indicated in laryngitis. Intratracheal injections are of little value as the fluid does not reach beyond the larger bronchi. If the accumulation of liquid exduate is excessive a hypodermic of atropin (gr. $\frac{1}{4}$) will temporarily arrest the discharge and dyspnea.

Chronic bronchitis is usually incurable. Expectorants, diuretics and sedatives often used combined may temporarily alleviate the symptoms. (For details see Heaves.)

VERMINOUS BRONCHITIS.

Lung Worm Plague.

Definition.—A form of bronchitis due to the presence of palisade worms in the air tubes. There is usually associated with it bronchopneumonia.

Occurrence.—The disease is very common in all countries and assumes the form of an enzoötic causing great losses among sheep, cattle and more rarely swine. Horses and

asses are rarely affected. The lung worm plague is most apt to follow after wet summers and among animals kept in swampy pastures or lands subject to overflow. Outbreaks have occurred, however, among stabled sheep.

Etiology.—From a clinical standpoint the following varieties of the nematode worm are important.

1. Strongylus filaria of sheep. (Dictyocaulus filaria.)
2. Strongylus micrurus of cattle. (Dictyocaulus viviparus.)
3. Strongylus paradoxus of swine. (Metastrongylus apri.)

The life history is not entirely known. These parasites, which are long, slender, filiform worms, in the adult stage inhabit the bronchi and trachea. Their eggs and embryos are eliminated from the body by coughing and with the feces. Outside of the body they probably undergo changes. Sheep, cattle and swine take them up with food and water of infested pastures, feed boxes, stable floors, etc. The parasites pass first to the stomach; probably during rumination they reach the pharynx, trachea, and bronchi. It is also probable that the embryos are carried to the lungs by the blood, as nodules containing them are not infrequently found in the lungs. In about two months after ingestion the strongylus becomes sexually ripe. The worm brood is usually taken into the body during the spring and the clinical symptoms of the disorder produced develop two to three months later. Infestation, however, is possible during the summer or fall. Young animals (lambs and calves) are more susceptible than adults. Suckling lambs and calves may be infected from the udders of their dams.

Necropsy.—On necropsy the lungs of affected sheep show chronic bronchitis with bronchiectasis and usually catarrhal pneumonia and nodular parabronchitis. The cadaver in the later stages of the disease is anemic and hydremic, transudates occurring in the body cavities and the connective tissue. There is obviously evidence of bronchitis, the bronchial tubes being partially filled with mucopurulent exudate. Numbers of sexually mature worms are present and under the microscope ova and embryos can be seen.

Symptoms.—In practice outbreaks affecting herds of lambs and calves usually occur in the summer and fall. The early symptoms are those of a chronic bronchial catarrh, the patient coughing, showing nasal discharge and on auscultation rales are heard. Later the affected animals become emaciated, anemic, hydremic (cold edemas under the throat and brisket), dyspneic and cachectic. The symptoms in calves are much the same as those in lambs.

Diagnosis.—A positive diagnosis can be made only on necropsy or by microscopical examination of the feces or bronchial exudate coughed up by the patients.

Prognosis.—The prognosis depends upon the severity of the symptoms and the age and condition of the patient. The disease is much more serious in lambs and calves than in pigs. Adult animals frequently recover spontaneously. In lambs the mortality will vary from 10 to 70 per cent. in different outbreaks; in calves the prognosis is more favorable.

Treatment.—The most effective treatment consists in intratracheal injections of antiparasitic drugs. The use of a spray or the inhalation of sulphur fumes, smoke from burning feathers, etc., affords usually only temporary relief. The following mixtures may be used intratracheally:

℞—Ol. terebinthinæ,
Ol. olivæ . āā ʒiij
Creolini . . . . . ʒiiss
M. Sig.—Inject into the trachea 5 c.c. for lambs, 20 c.c. for calves; repeat twice at four-day intervals.

℞—Creosoti . . . . . ʒv
Ol. olivæ . . . . . ʒiij
M. Sig.—Intratracheal injection 5 c.c. for lambs; 15 to 20 c.c. for calves; repeat in four days.

Potassium picronitricum 1 per mille has given good results (20 to 60 c.c. according to age).

Prophylaxis.—The sheep and lambs should be removed from infested pastures and fed highly nutritious food. Water should be supplied from a well and dry pavement kept about the drinking troughs. The sheep pens, lots, mangers, floor, etc., should be thoroughly disinfected. If the

floor is of earth it should be removed to the depth of six inches and filled in with fresh uninfested clay or cemented. Infested fields should be thoroughly tile-drained and cultivated. Many sheep owners recommend that tobacco leaves mixed with salt be made accessible to the sheep at all times. Sulphate of iron and salt may be fed once weekly with the grain.

CHAPTER IV.

DISEASES OF THE LUNGS.

CIRCULATORY DISTURBANCES.

Congestion of the Lungs.—Definition.—An engorgement of the pulmonary capillaries with blood, due either to a heart which is overexerting itself (active hyperemia), or to some obstacle which prevents the prompt return of the pulmonary blood to the heart (passive hyperemia).

Occurrence and Etiology.—Active congestion is quite common in horses especially during hot weather in unconditioned, untrained animals which are put to violent exertion such as racing, hunting, hard pulling. In all probability the primary lesion is cardiac rather than pulmonary and represents an acute dilatation of the heart. (See this.) Fat cattle and swine are often attacked when being driven to market on a hot day.

An active congestion of the lungs may attend heat stroke. It can arise from the inhalation of irritant fumes (stable fires). The first stage of pneumonia is congestion.

Passive Congestion.—Two forms of passive congestion are recognized: (*a*) Mechanical, and (*b*) hypostatic.

(*a*) The former occurs whenever there is some condition of the heart present which prevents the free return of blood to it. It, therefore, may attend mitral insufficiency, valvular stenosis, cloudy swelling, or dilatation of the heart. It can occur during the course of infectious diseases which affect the heart. Passive congestion may follow filling of the pericardium (pericarditis), and develops secondary to gastric or intestinal bloat.

(*b*) Hypostatic congestion occurs chiefly in large animals which are forced to lie in one posture for a long time (cast in stall; azoturia; fracture of leg bones), the blood gravitating

to the lower lung. The attending heart weakness undoubtedly assists.

Pulmonary Edema.—Definition.—A transuding of serum into the alveoli and bronchioli. It is the next step in congestion, which it accompanies; it also occurs in pneumonia.

Etiology.—Edema usually follows congestion, the advanced stages of which it represents (serous pneumonia). The causes are, therefore, those of congestion and pneumonia, *i. e.*, inhalation of irritant gases, acute infectious diseases (malignant edema, anthrax); may attend severe acute nephritis; any heart weakness; bronchial parasites (Str. paradoxus in swine).

Symptoms.—As pulmonary congestion and edema are always more or less combined, their symptoms may be treated together. They are: Dyspnea which develops rapidly. Sometimes the patient becomes apneic. The respirations may reach 80 to 100. If the edema is well developed, there is a foamy, blood-tinged nasal discharge. The mucous membranes may be cyanotic. The heart beat is often palpitating, the pulse, at first full and rapid, later becomes weak. Percussion is normal. Auscultation: Exaggerated vesicular breathing, crepitant rales, moist rales.

Course.—May be very rapid, especially if it follow active congestion. In six to twelve hours the symptoms may abate or lead to death. Following passive congestion, the course may be four to six days with lethal termination. Apoplectic death may also occur. Rarely pneumonia develops.

Diagnosis.—The dyspnea, sudden onset and usually rapid course are characteristic. From acute bronchitis it is distinguished by the absence of fever and distressing cough and the presence of crepitant rales and foamy nasal discharge. Heat stroke resembles it but the high temperature (may reach 112° F.) and nervous prostration of this condition are sufficient for differentiation.

Prognosis.—Usually good in robust patients. A weak pulse and fever are bad signs. Pulmonary congestion and edema are often fatal.

Treatment.—Bleeding is indicated when the dyspnea is very great. In large animals remove 4 to 6 quarts of blood from the jugular. Its affect is often life-saving. The application of oil of mustard to the chest is helpful. While the heart is weak give excitants (alcohol, ether, caffein). Sulphate of atropin (gr. $\frac{1}{4}$ to $\frac{1}{2}$ subcutaneously) is good in cases where edema predominates (foamy nasal discharge). Digitalis and strychnin are also recommended. In milder cases an aloes ball affords relief. Nitroglycerin (gr. $\frac{1}{2}$ to j) is sometimes used.

Bronchopulmonary Hemorrhage (*Bronchorrhagia, Pneumorrhagia, Bleeding from the Lungs, Hemoptysis*).—**Definition.**—Bleeding from the lower air passages and lung tissue.

Etiology.—Bleeding from the bronchial mucous membrane may be due to overexertion, as fast driving, racing. It is seen in valvular heart disease, congestion of the lungs, aneurysms (aorta, pulmonary artery), lung infarctions, thrombosis, embolism. In infectious diseases it is at times a symptom (fibrinous pneumonia, purpura hemorrhagica, anthrax, glanders, tuberculosis). Where caverns of the lung remain after pneumonia, hemorrhage occurs. Frequently no cause can be found on postmortem to explain the hemorrhage.

Symptoms.—Bleeding from the nose is the principal symptom. If the blood come from the larger bronchi, it will flow from the nostrils in drops or in a thin stream and is usually not frothy. From the finer bronchi and lung the blood is frothy and of light red color. The patient is dyspneic and coughs. In severe cases the patient is restless, the mucous membranes pale, the body covered with sweat and the legs and ears cold. The pulse becomes rapid, very weak and thready. In profuse hemorrhage the bronchi and trachea may fill with blood leading to suffocation. On auscultation rales are heard. Percussion normal.

Diagnosis.—When the discharge is bilateral, light colored, foamy and attended by cough, the diagnosis is not difficult. When the blood comes from the nostrils, pharynx, mouth or gullet it is usually dark colored, not frothy or contains only a few coarse air bubbles. Blood vomited through the

nostrils is of dark color, acid reaction, partially coagulated and admixed with food particles.

Prognosis.—The prognosis depends upon the quantity of blood lost. A gallon will not seriously affect a full sized horse. If, however, the hemorrhage continues it induces a general anemia which may lead to cachexia and death. Very profuse hemorrhage can terminate fatally in a few minutes.

Treatment.—The patient should be kept as quiet as possible. Ice packs (or cold water) may be applied to sides, vulva or scrotum (reflex affect). Internal medication does little good. Ergot in the form of fluid extract is recommended by most practitioners; others condemn it. Acetate of lead (℥j) given three times daily is employed in obstinate cases. Sulphuric acid (dilute ℥vj) is sometimes beneficial. Theoretically aconite (Fleming's tincture ♏x) is good as it lowers blood-pressure. Lung hemorrhages which are due merely to congestion of the bronchial mucous membrane or lung usually stop spontaneously when the blood-pressure becomes low. On the other hand those hemorrhages due to a ruptured vessel in the lung (aneurysm) are generally copious enough to produce death.

PULMONARY GANGRENE.

Definition.—A decomposition of the dead lung tissue due to the microörganisms of putrefaction.

Occurrence.—Most often in horse, swine, and sheep.

Etiology.—May result from fibrinous pneumonia, diseases of the pharynx which make swallowing difficult (pharyngitis, paralysis), general diseases affecting the pharynx (tetanus, parturient paresis, forage poisoning) causing the food swallowed to "go the wrong way"; the aspiration of foreign matter (dust, saw-dust, blood, pus, grains, hair, plant fibers, etc), drenches unskilfully administered (pneumonia medicantaria) especially in horses, sheep, and swine; by metastasis emboli develop from ulcerative processes in the bowel, hoof matrix (gangrenous pododermatitis), and bone (caries). In these instances the necrosis bacillus is active. Traumatic injuries rarely cause pulmonary gangrene.

Symptoms.—The most characteristic symptom is fetid expirium.[1] There is nasal discharge of a muddy reddish-brown or greenish color. The discharge, which is more copious after coughing, has a fetid odor. It contains bits of dead lung tissue, fat crystals, pigment, and innumerable microörganisms. Under the microscope elastic fibers are seen. The respirations are dyspneic especially in the latter stages (toxemia). Fever is nearly always present. It is usually about 104° to 105° F., and assumes an intermittent type. In gangrene due to aspirated foreign matter, the temperature may not be over 102° F. for several days. The pulse is frequent, arhythmic, and thready. Chills are frequent. The patient loses flesh rapidly, is weak, languid, and stupid. The appetite is capricious; toward the end there is diarrhea. Pregnant animals often abort. On percussion the sound is flat over the ventral and middle portions of the lung. Sometimes over caverns the "cracked-pot" tone is emitted. On auscultation rales and tubular breathing are most often noted.

Diagnosis.—The finding of elastic fibers in the nasal discharge is pathognomonic. The fetid breath and nasal discharge are indicative. In putrid bronchitis there are usually no general symptoms such as fever and the condition of the patient is, as a rule, good.

Prognosis.—Bad. Only when local foci are present is encapsulation or the ejection of the dead mass by coughing probable.

Treatment.—Unsatisfactory. The inhalation or intratracheal administration of antiseptics may be tried. Pneumotomy is employed in man. Give good food and care and treat the heart, bowel (diarrhea) and fever as the indications warrant.

ABSCESS OF THE LUNG.

Definition.—Collection of pus in cavities in the lung.

Etiology.—Embolic, metastatic abscesses following general pyemic diseases (strangles, purulent metritis, bacillosis of

[1] If the gangrenous mass does not communicate with a bronchus the expirium may not be fetid. Diagnosis in these cases is very difficult or impossible.

sucklings), or secondary to primary abscess (abscess within hoof, abscess following castration). It is rarely due to fibrinous pneumonia, but may follow catarrhal pneumonia due to foreign matter entering the bronchi, especially medicines.

Symptoms.—Pulmonary abscess usually follows an attack of pneumonia with delayed resolution. The temperature again rises, the pulse becomes rapid and weak, and the patient shows a purulent nasal discharge. On percussion a flat area may be determined; on auscultation rales and tubular breathing. If complicated with gangrene the expirium is often fetid. The course of the fever is very atypical showing marked variations from day to day. At times the temperature may drop to almost normal, to rise again next day.

Course.—Usually death in seven to ten days. Isolated abscesses may become encapsulated or break into a bronchus and be discharged. Diagnosis in these instances is difficult. Death may result from pyemia or more commonly from serofibrinous or purulent pleuritis.

Diagnosis.—A copious nasal discharge which is largely made up of pus is the chief clinical feature which distinguishes it from pulmonary gangrene.

Treatment.—Usually of little avail. In man well defined, superficial abscesses are surgically treated by opening and draining. The use of antiseptics as inhalations (bichlorid, 1 per mille, as a spray) or intratracheal injections are suggested.

CHRONIC ALVEOLAR EMPHYSEMA.

Definition.—Chronic alveolar emphysema is a permanent overdistention of the alveoli with an increased amount of air present in the lung. The walls of the alveoli become anemic and atrophic.

Occurrence.—Most common in horses, especially old, hard-worked individuals. It is a common cause of so-called "heaves" or "broken wind."

Etiology.—(*a*) Continued severe exercise (hard pulling, fast driving, high jumping). This causes repeated physiological dyspnea inducing overdistention at inspiration and

causes the expiration to become forced. These two factors in time lead to weakening and finally atrophy of the alveolar walls. The alveoli can be ten times their normal diameter, the walls very thin and anemic. Later rupture of the interalveolar septa occurs permitting direct communication between the distended air cells. The lung thus loses its power of contraction at expiration, which causes this act to become forced so that the air from the emphysematous part of the lung is expelled. (*b*) Severe dyspnea due to diseases of the larynx or bronchi is a rare cause. (*c*) Severe coughing, especially in chronic bronchitis. (*d*) Occasionally chronic alveolar emphysema results from the acute form. (*e*) The feeding of bulky forage, particularly clover or dusty timothy hay is no doubt an important predisposing cause of pulmonary emphysema, the dilatation of the stomach and bowels which such foods produce interfering with respirations. (*f*) As some strains of horses seem more subject to emphysema than others, an inherited predisposition (lacking resistance in the septa) has been assumed. Of this, however, there is no substantial proof. Use and feeding methods may explain the assumption.

Cases of "heaves" have occurred among horses which were never exercised vigorously or overexerted. In fat, old, well kept carriage horses and pet ponies it is relatively more common than among highly bred race or hunting horses. Horses fed bulky foods, especially hay made of tame grasses, seem most subject to the disorder. It has been stated that before tame grasses were introduced into the far Western States "heaves" was unknown in that part of the country. While "heaves" does occur occasionally among very young horses, it is essentially a disease of older age increasing in frequency with age. A great many cases of "heaves" are combined with chronic bronchitis.

Symptoms.—Dyspnea which increases on exercise. It is always most pronounced at expiration and is often accompanied by a double movement of the flanks and the interrupted protrusion of the anus. The first part of the expiratory act is passive, but after a very brief pause, the movement becomes active, the abdominal muscles contracting with

vigor in an effort to compensate for the lost elasticity of the lung and to expel the air. A marked groove appears along the costal arches ("heave line"). The inspiratory act is shorter than the expiratory. The ribs are seen to roll forward beneath the skin, the intercostal spaces deepen and the ventral portion of the thorax and the anterior aperture of the chest sink inwardly. In advanced cases the ribs are kept rolled forward, the thorax appearing barrel-shaped. If bronchitis is present, there is cough which is usually short, weak, and dull. The cough is often attended by the discharge of flatus through the anus ("breaking wind"). The heart sounds are at times feebler than normal. The diastolic sound may be accentuated. Percussion gives a sound which is too full and drumlike (hyperresonance). The heart dulness may be obliterated. Posteriorly, the area of the field of percussion is enlarged, the posteroventral limits extending through the 18th, 17th and 14th ribs at the heights on the thorax of the external angle of the ilium, tuberosities of the ischium and shoulder-joint respectively. (With normal lungs the figures would read 17, 15, and 11.) Auscultation: If the bronchitis is present, dry or moist rales are heard, otherwise the vesicular murmur is weakened. When the bronchi are involved there is a bilateral, white nasal discharge. The general condition suffers in old cases. The patient loses flesh, becomes anemic with a tendency for edema to form under the chest and belly and in the limbs.

Course.—The course is prolonged, the condition lasting for months and years. Once affected the patient never fully recovers. The symptoms improve as the attending bronchitis improves, the patient's work lightened and the quantity of roughage fed, especially tame hay, reduced. On the other hand exposure, hard work and the feeding of bulky, dusty food (hay), and allowing the thirsty patient to drink at one time all the water it will, greatly increases the dyspnea and cough.

Diagnosis.—Only advanced cases can be diagnosed. The characteristic dyspnea, which increases on exercise, the dilated nostrils, the anal protrustion, cough, and absence

of fever are characteristic. Its chronicity (absence of fever) and physical signs (auscultation and percussion) differentiate emphysema from acute febrile diseases of the respiratory system (pleuritis). According to King mild cases of "heaves" may be exaggerated for diagnostic purposes by intravenous injections of arecolin. He injects 30 drops in ʒss of normal salt solution into the jugular. While in "non-heavy" horses the injection produces a passing dyspnea, in "heavy" ones a pronounced double-pumping of the flanks is induced which lasts several minutes.

Prognosis.—As far as the life of the patient is concerned the prognosis is good, but from the standpoint of healing there is no hope of a permanent cure. Diffuse bronchial catarrh and weak heart are bad complications.

Treatment.—No treatment, hygienic, dietetic or medicinal will cure chronic pulmonary emphysema. By using the patient only for light work, feeding good nutritious food (clean oats, bran), allowing only small quantities of dustless (moistened) hay, and watering frequently but in small quantities at a time, the symptoms may be overcome so long as the above dietetics are persisted in.

Drugs.—There are several drugs such as belladonna, datura stramonium, hyoscyamus, which contain atropin, that will mask the symptoms (dyspnea) in a marked degree. By using such drugs horsetraders often deceive prospective buyers into believing the horse to have "good wind." However, the abatement of the symptoms is only temporary, lasting usually but one day. The abnormal dilatation of the pupil, dryness of the mucous membranes and rapid pulse, which usually follow the use of a "dope," should put the veterinarian on his guard. Gypsies not only administer drugs but drench "heavy" horses with shot, in this form giving as high as two pounds of lead at a single dose just before the animal is to be sold. In addition very little water is allowed. No satisfactory explanation of the therapeutic effect of this agent has been given. Its action is probably mechanical.

Arsenic is useful, usually given in the form of Fowler's solution (ʒss) three times daily in the drinking water.

Subcutaneous injections of atropin temporarily allay the symptoms. Treating the attending bronchial catarrh is helpful (see this). In "heavy" horses avoid using arecalin, eserin, or barium chlorid.

ACUTE INTERSTITIAL PULMONARY EMPHYSEMA.

Definition.—Rupture of the alveoli with the entrance of air into the interstitial tissue of the lung, bubbles appearing beneath the pleura.

Occurrence.—Has been noted in horse and ox.

Etiology.—Anything which greatly increases air pressure in the alveoli. Violent coughing fits to dislodge foreign bodies, medicine, etc., which have gotten into the bronchi. It rarely accompanies acute catarrhal and croupous bronchitis. Violent contractions of the abdominal muscles (hard pulling, retching, continued bellowing in cattle). Violent struggles to get free from hobbles, or if a horse is cast in the stall and makes vigorous efforts to free itself.

Symptoms.—Sudden dyspnea which may rapidly lead to suffocation. Emphysema of the skin (in ox) of the aperture of the chest, shoulders, and side of thorax. It may involve the whole trunk. Percussion is practically normal. On auscultation crackling sounds, rales.

Course.—Usually fatal in twenty-four to thirty-six hours. Healing only in less acute cases.

Diagnosis.—Unless subcutaneous emphysema develop, may be impossible. Can easily be confused with pulmonary congestion and edema. The history is helpful.

Treatment.—Allay cough. Scarify skin.

INFLAMMATION OF THE LUNGS.

Pneumonia. Pneumonitis.

The following clinical forms of pneumonia may be distinguished:

Fibrinous.
Catarrhal.
Foreign body.
Metastatic.
Interstitial.

Fibrinous Pneumonia (*Lung Fever, Croupous Pneumonia*). —**Definition.**—An inflammation of the lung characterized by its typical course, and the formation of fibrinous coagulæ in the alveoli of the invaded area. It affects the lobe rather than the lobule.

Etiology.—The existence of fibrinous pneumonia as a primary disease in animals is open to question. At any rate it has not been proved.

In the horse it is usually expressive of infectious fibrinous pneumonia although it may accompany strangles or purpura hemorrhagica.

In the ox it most commonly is noted as a symptom of hemorrhagic septicemia due to the Bacterium bovisepticum.[1] It also occurs from foreign bodies entering the lung from the reticulum, the aspiration of ingesta in choke or when the pharynx is paralyzed.

In swine it is a symptom of hemorrhagic septicemia (swine plague), and anthrax. A mixed fibrinous and catarrhal pneumonia may occur in swine due to the aspiration of medicine unskilfully given as a drench (melted lard).

In sheep fibrinous pneumonia is seen in hemorrhagic septicemia (Bacterium ovisepticum).

Cold, the inhalation of irritant gases, smoke, steam, etc., great exhaustion from overwork, casting, tying the head of the horse too high, etc., are merely predisposing factors in the etiology of fibrinous pneumonia.

Symptoms.—The onset of the disease is usually sudden. Without warning the patient is seized with fever, which in a few hours, in the horse, may reach 104° to 106° F. The patient is stupid, languid, and loses appetite. In some cases a pronounced chill ushers in the disease symptoms. The fever is of the continuous type remaining up for seven to nine days when it drops rapidly to normal (by crisis), or on the third or fourth day may begin to gradually decline reaching normal in four to eight days following (by lysis). Cough is short, painful, and frequently restrained. At first it is dry, later moist in character. Nasal discharge

[1] In these cases the pneumonia is a mixture of catarrhal and fibrinous and the course of the disorder is atypical.

is not always present, especially in continuously stabled horses. In some cases, during the stage of red hepatization, a rusty brown ("prune juice") discharge occurs which may last only twenty-four to forty-eight hours. In the stage of resolution a yellow-colored discharge may appear. The pulse at first is not much affected, but as the disease progresses, due to cloudy swelling of the heart, its frequency is increased to 60 to 80 or higher. Quite often the pulse remains high after the fever has gone down. The respirations are accelerated early and the patient breathes with distended nostrils. The conjunctiva in severe cases often assumes a spotted mahogany color. The percussion varies with the stage of the disease. In the earliest stage (congestion) there is little appreciable change (somewhat tympanitic);[1] in the second stage (hepatization) a flat sound is emitted. The sound begins about the second day and is retained three to five days. During the third stage (resolution) the sound becomes tympanitic again. The area of dulness is usually confined to the ventral portion of one lung, its dorsal limits often describing an upward curved line. Auscultation: In the first stage crepitant rales at inspiration —fine crackling sounds like rubbing hair between the fingers. These sounds are usually present for the first twenty-four to forty-eight hours, then pass away. In the second stage the vesicular murmur is gone and there is either no respiratory sound audible or tubular breathing (bronchial) is heard. In the third stage moist rales are heard (the return rale). General condition: Varies greatly with the case. In mild attacks the appetite may be retained and the mind little perturbed. In severe cases there is no appetite while the fever is on, and the animal is very stupid and languid. Horses usually do not lie down until the fever drops. Small animals and even ponies lie down most of the time during the disease, and if only one lung is affected, on the diseased side. The urine is scanty and high colored until the fall of the fever when its specific gravity drops and the quantity, voided frequently, greatly increases.

[1] The flat percussion sound may not be obtained, if the pneumonia involves the central rather than the peripheral portion of the lungs (pneumonia centralis).

Diagnosis.—Acute catarrhal pneumonia may be confused with it. The principal differential features are: (See table). From pleuritis it may be distinguished by auscultation and percussion. In cases complicated with pleuritis (pleuro-pneumonia) differentiation may be impossible. However, pleuritis is usually bilateral, the upper margin of the zone of dulness on percussion is horizontal and the resistance under the hammer pronounced. In pleuritis there is further a tendency for edema to form in pendent parts of the body. Cough is usually absent in pleuritis; present in pneumonia. A test puncture of the thorax may be made in doubtful cases.

Complications.—(*a*) Heart weakness due to cloudy swelling. The beat is fast, arhythmic, and palpitating. The pulse may be weak (thready) and runs about 76. The patient is weak, may be cyanotic, superficial veins distended.

(*b*) Pleuritis: A common complication, leading to effusion in the chest, displacement of the heart and characteristic dyspnea. (See Pleuritis.)

(*c*) Gangrene of the lung: May develop during convalescence. The temperature again rises, the patient continues to lose flesh and the expirium assumes a sweetish, fetid odor.

(*d*) Further but less common complications are: Nephritis (albumin in urine), jaundice (catarrh of duodenum), tendovaginitis (leg swelling and lameness), founder (laminitis), cerebral and meningeal symptoms. Purpura hemorrhagica may occur during convalescence.

Course.—The usual course is typical, ending in recovery in two weeks. In some cases, especially in old horses, cattle and swine the course may be much shorter (larval or abortive type). Death may occur suddenly during convalescence from heart failure. If pleuritis complicates the pneumonia, the course is much prolonged. It may lead to death, or adhesions (lung to thoracic wall) may cause permanent dyspnea ("heaves").

Chronic induration of the lungs is a common termination following certain outbreaks. It is characterized by the continuation of the fever and dyspnea after the usual period of convalescence has passed. The patient is generally left short-winded. Roaring may sometimes follow an attack of fibrinous pneumonia. Pericarditis is a rarer complication.

Prognosis.—The prognosis is good in typical and uncomplicated cases. Of importance is the behavior of the heart during the attack. A continued high pulse is dangerous to the patient. The extent of the area involved has much to do with the outcome of the case. If confined only to the ventral portion of one lung, the danger is not so great as when the dorsal part of the lung is also involved, or if both lungs are diseased. When pleuritis complicates the case the prognosis is naturally less favorable.

Treatment.—The patient should be placed in a light, clean and well-ventilated place. If feasible, keep the case out-of-doors as much as possible, guarding it, of course, against wind and rain. Use only light covering (in horses). The legs may be bandaged (use Derby bandages with cotton underneath). Removing the bandages once daily and rubbing the legs well before re-applying is helpful. The horse-patient should be groomed well each day. Feed any easily digested food which the patient can be coaxed to eat. Good clean oats over which a little sugar has been sprinkled is often tempting to the appetite. Give only small quantities at a time. Before feeding syringe out the mouth with clean water. If obtainable fresh grass is very palatable and nutritious. A few handfuls over which is strewn a little salt is often eaten with avidity. The hay should be bright and free from dust. Feed about 6 pounds daily, divided into three feeds. Roots (carrots, beets) and bran mashes are recommended (some horses do not like bran). Eggs and milk may be given if appetite is entirely gone. Keep pure water constantly before the patient, and where it can be gotten at without undue exertion. Rectal and artificial feeding may be resorted to in patients unable to swallow or without any appetite.

Surgical.—Puncturing the thorax with a sterile trocar, permitting the instrument to penetrate the inflamed lung, was practised extensively in the army during the World War. The operation was performed irrespective of the existence of exudate in the chest cavity. The results seem to have been satisfactory enough to warrant further experiment. It appeared most beneficial in cases of delayed resolution.

Drugs.—In typical cases of fibrinous pneumonia drugs are often not only superfluous but may do actual harm.

It is very important to watch carefully the heart. Minor irregularities may be overcome by small doses of brandy (℥ij) mixed with ether (℥ij) in a pint of water, or alcohol (℥ij) in a pail of drinking water may be kept before the patient, especially during the night. It may be repeated every three hours. Digitalis in the form of Squibb's fluid-extract (℥iv–vj), giving one dose only, has often a toning effect upon the heart (avoid repeated small doses of this drug). When the pulse reaches 80 or more and becomes weak, subcutaneous doses of the oil of camphor (℥j) are good. Caffein (℥j–℥ij subcutaneously) is useful. For great depression (general loss of arterial tone—toxemia) an intravenous infusion of normal salt solution (2 to 4 quarts in horse) may be tried. (If heart is weak, look out for pulmonary edema). Subcutaneous doses of ether and alcohol in ℥ss doses are valuable in this condition.

Unless the fever be unreasonably high (106° to 108° F.) or threatens the heart's force, it should be let alone. In robust patients cold water infusions into the rectum, cold compresses over the chest are useful in reducing the temperature a degree or two. Acetanilid (℥iv to ℥j) combined with caffein (℥j) is recommended. (Affect depressing.)

To favor resorption of the exudate diuretics may be employed. Acetate of sodium (℥j to ℥j) is serviceable. Spirits of nitrous ether (℥j) is recommended. Iodid of sodium (℥iv) is useful. In delayed resolution the resorption of the exudate is said to be stimulated by puncturing the infiltrated lung as in paracentesis thoracis. Local applications: In severe dyspnea the application of mustard (oil of mustard in alcohol 1–12–16) is advisable. (Apply in airy room and use light blanket over patient after application.) An ice-bag over heart or cold compresses changed every fifteen minutes yield good results.

During convalescence keep the animal as quiet as possible if heart be weak and assist the appetite and digestion by giving artificial Carlsbad salts to each pound of which 2 ounces of nux vomica have been added. For treatment of complications, see these.

Catarrhal Pneumonia (*Bronchopneumonia*).—**Definition.**—An inflammation of the lungs affecting isolated lobules or groups of lobules, the exudate and desquamated cells in the alveoli seldom undergoing fibrinous coagulation.

Occurrence.—Most common in very young or aged animals. Less frequent in the horse than in the ox, sheep, and swine.

Etiology.—Catarrhal pneumonia occurs either as a primary or as a secondary affection. As catarrhal pneumonia is clinically a collective term it includes a group of pneumonias, the causes of which are varied. It may be due to: (*a*) The spreading of bronchitis to the lung parenchyma; (*b*) food entering the windpipe in patients suffering from dysphagia (tetanus, milk fever, feeding too soon after chloroform narcosis); (*c*) foreign matter (dust, sand, sawdust) which may be drawn into the lungs in recumbent patients; (*d*) the aspiration of pus, blood, saliva or mucus (head operations, patient recumbent); (*e*) unskilled administration of medicines, especially drenches; (*f*) result of hypostasis of the lungs (recumbent position, long stable confinement in old horses); (*g*) infection, especially with the Bacillus bipolaris septicus (in the ox) and the Bacillus pyogenes.

Cold, bad sanitation and exhaustive railway and ship transportations are predisposing factors.

Catarrhal pneumonia is secondary to several acute infectious diseases as malignant head catarrh, hog-cholera, hemorrhagic septicemia; it often accompanies tuberculosis, glanders, and occasionally actinomycosis.

Symptoms.—The prodromal symptoms are those of bronchitis which it usually follows. As the areas of solidification in the lung may be small and scattered, they are difficult to detect clinically. The cardinal symptoms are: Cough which is short, dull, and often painful, the patient trying to suppress it. Nasal discharge which is at times copious and white in color. Fever which may run about 104° F. The fever does not take a typical course as in fibrinous pneumonia, but is intermittent in character, continuing until the termination of the disease. In aged horses fever is often absent. Dyspnea, the respirations are accelerated, and labored. Percussion is often painful and

induces coughing. Areas of dulness may be determined, provided they are of the size of a clenched fist and superficially located in the lung. Auscultation: Rales of a fine subcrepitant and whistling character. If large areas of the lung are involved, bronchial (tubular) breathing is heard. If the bronchi and bronchioli in the affected area are plugged with exudate, no sounds will be emitted. In the neighboring lung tissue, however, the vesicular murmur is harsher than normal. Usually the physical signs of the disease are noted in both lungs. The appetite is at times impaired or absent. Some patients, however, eat well. The general condition of the patient varies with the extent of the lesions. In some cases the dyspnea, distressing cough and fever greatly debilitate the patient, while in others, especially in horses, the general symptoms are not marked. It can happen that the catarrhal pneumonia is overlooked by the owner and the patient presented to the veterinarian only after pulmonary gangrene or some other termination has set in.

Course.—Atypical. Depends much upon cause. Acute cases may terminate in healing in two to three weeks. Exacerbations, however, are common (formation of new foci). As a rule, catarrhal pneumonia takes a prolonged course lasting weeks or even months. Terminations: (*a*) Healing in two to three weeks; (*b*) death from asphyxia, heart weakness, exhaustion; (*c*) death from pulmonary gangrene or septicemia (diarrhea); (*d*) induration of the lungs causing chronic dyspnea ("heaves").

Diagnosis.—If larger areas of the lung are involved, in the early stages it is difficult to distinguish catarrhal from fibrinous pneumonia. The following table may be of value on this point:

Fibrinous Pneumonia.	Catarrhal Pneumonia.
Onset sudden.	Onset gradual.
Course and fever typical.	Course and fever atypical.
Begins as pneumonia.	Begins with bronchitis.
Percussion: Diffuse dulness over one lung.	Percussion: Normal or isolated areas of dulness over both lungs.
Auscultation: Bronchial breathing.	Auscultation: Rales.
A benign disease.	A malignant disease.

From glanders (horse) and tuberculosis (ox), non-specific catarrhal pneumonias are now best differentiated by the use of such scientific aids as the mallein (eye), agglutination, complement fixation, tuberculin tests, etc.

Treatment.—Place the patient in a light, clean, and well-ventilated place. Apply suitable covering if the weather is cold. Give good easily digested food (clean oats, bran mash, bright hay, grass, milk, eggs, etc.). As there is no specific, the treatment is symptomatic and follows that outlined for fibrinous pneumonia. Intratracheal irrigations are of little value in catarrhal pneumonia, unless applied very early and before the lung proper is attacked (washing out the bronchi) following aspiration of blood and exudate after operations ("roaring," head sinuses).

Foreign body Pneumonia (*Inhalation Pneumonia*).—**Definition.**—Foreign body pneumonia is a clinical term used to include all forms of inflammation of the lung due to the entrance of coarse foreign matter.

Etiology.—Foreign body pneumonia is due to the aspiration of dust, gases, food, liquids, blood, pus, etc. While it occurs in all animals the horse is the most common victim due to improper drenching and the frequency in this animal of pharyngitis, strangles, tetanus, encephalitis and purpura in which diseases dysphagia is a common symptom. The dysphagia following chloroform narcosis and attending forage poisoning may also lead to it. In the ox the pharyngeal paralysis occurring in parturient paresis is the most common etiological factor, paunch contents being regurgitated and liquid medicines unskilfully given reaching the windpipe. Foreign material (sharp objects) may also penetrate the lung from the reticulum.

The foreign material *per se* is not the cause of the pneumonia, but various microörganisms for which it paves the way. Bacteriologically staphylococci, streptococci, the necrosis bacillus, and various other septic organisms occur in the inflamed lung tissue, depending on the individual case.

Necropsy.—On postmortem it will be noted that the foreign material has produced bronchitis and areas of bronchopneumonia. Due to the entrance of pus bacteria

and germs of putrefaction there result purulent infiltration, necrosis and decomposition of the lung tissue. Therefore gangrene, abscess and putrefaction are found combined, changing the lung into a miscolored, fetid, odorous, smeary mass. By contact the pleura also becomes involved so that a purulent or putrid pleuritis is present. More rarely there may be pneumothorax.

Symptoms.—The onset in foreign body pneumonia is usually insidious and may be entirely overlooked especially by the owner or attendant. The disease begins as a bronchitis and bronchopneumonia (cough, rales). When gangrene sets in the expirium has a sweetish odor which later becomes fetid. Soon nasal discharge appears which is discolored and contains an admixture of lung tissue elements. On percussion, depending upon the character and extent of the lesions, there may be flatness, tympany, or even a "cracked-pot" tone emitted. The patient shows a septic fever and rapid, weak, pulse (80 to 120). Symptoms of pleuritis (empyema) are not infrequent. In many cases the appetite remains fairly good until the end and the temperature may be little above normal.

Diagnosis.—The diagnosis depends upon the physical signs of pneumonia with fetid expirium and the discolored nasal discharge containing bits of dead lung tissue. A microscopic examination will show elastic fibers from the parenchyma of the lung. In all pneumonias following unskilful drenching or where dysphagia exists foreign body pneumonia should be suspected. From the standpoint of differential diagnosis, diseases of the teeth and sinuses of the head causing fetid breath must be considered. An examination of these parts and of the lungs should suffice for differentiation. Fetid bronchitis, which is most common in dogs, does not affect the general condition of the patient and is rarely fatal.

Course and Prognosis.—Once the disease is recognized the course is usually about one week. The prognosis is bad especially in horses, the disease leading to sapremia and death. Occasionally in cattle the gangrenous mass remains local in the lung and becomes encapsuled by connective

tissue forming a sequester which protects the rest of the organism. Such cases will heal.

Treatment.—The treatment is unsatisfactory. Intratracheal injections of antiseptics are of no value except in the early stages. The treatment suggested for catarrhal pneumonia is usually followed.

Metastatic Pneumonia.—Definition.—A secondary pneumonia the result of embolism.

Etiology.—Metastatic pneumonia is the result of a spread of infection *via* embolism from a primary focus containing pus bacteria, septic bacteria or necrosis bacilli which reach the lung through the blood. The primary focus is usually an abscess occurring in the course of strangles, purulent arthritis, gangrenous pododermatitis, phlegmon of the hind limbs, etc. It may also result from navel infection (thrombophlebitis), of which it is a common sequel, or it may originate from inflammation of the jugular or saphenous veins. It is a frequent sequel of acute gastro-intestinal disorders in calves.

Necropsy.—Metastatic or embolic pneumonia is characterized by multiple abscess or necrotic centers which occur throughout the lung tissue. At the same time there are symptoms of septicemia and pyemia.

Symptoms.—The symptoms of metastatic pneumonia are often quite vague as the embolic center cannot always be determined by percussion and auscultation. Where a primary abscess exists, and the patient suddenly shows septic fever, dyspnea and cough a metastatic pneumonia should be thought of. In the later stages symptoms of abscess of the lung (purulent nasal discharge, etc.) are significant.

Treatment.—Treatment is unavailing.

Interstitial Pneumonia (*Chronic Pneumonia*).—**Definition.**—Interstitial pneumonia is an inflammation of the connective tissue of the lung which proliferates causing induration or sclerosis.

Etiology.—It is a secondary disease and may follow any form of pneumonia especially the catarrhal. It most frequently occurs in the course of chronic pulmonary tuberculosis,

glanders, contagious pleuropneumonia of cattle, verminous pneumonia or any form of inflammation of the lungs which is chronic.

Symptoms.—The symptoms depend upon the extent of the connective tissue proliferation and the amount of infection. Briefly they consist in dyspnea, dulness on percussion, absence of the vesicular murmur and the gradual emaciation of the patient. There is usually no increase in temperature.

Diagnosis.—The diagnosis of chronic interstitial pneumonia is extremely difficult unless it follow an acute attack of croupous or catarrhal pneumonia or pleuritis. In cattle if due to tuberculosis it may be diagnosed by the tuberculin test and in the horse the presence of glanders may be determined by the various tests for this disease.

Course and Prognosis.—The course is usually chronic, the disease lasting for weeks or even months. The prognosis is bad, for although death may not always ensue, the patient is left with a chronic incurable dyspnea.

Treatment.—Treatment is of no avail. Life may be prolonged by treating as in catarrhal pneumonia. Edible animals should be slaughtered. In horses fibrolysin (grs. xv) given subcutaneously every other day is recommended.

TUMORS IN THE LUNG.

While tumors in the lung are not uncommon they rarely attain clinical importance as the diagnosis is so difficult They sometimes produce symptoms of dyspnea, pulmonary hemorrhage, flatness on percussion and emaciation. Fever is not present and the course is chronic. Many of them originate by metastasis. The most common tumors are sarcomas, melanomas, adenomas, fibromas, and in aged animals particularly carcinomas.

CHAPTER V.

DISEASES OF THE PLEURA.

PLEURITIS.

Definition.—An inflammation of the pleura. Pleuritis is nearly always a secondary condition in animals.

From a pathological standpoint pleuritis may be classified into: Serofibrinous, fibrinous, purulent (empyema of thorax), ichoric (putrid) and hemorrhagic. Clinically these differentiations are difficult to make. Fibrinous pleuritis ("dry pleurisy") is rarely diagnosed *intra vitam*. By puncturing the thorax with a trocar the character of the exudate may be determined, *i. e.*, whether serous fluid with flocculæ admixed, pus, ichor, etc. The most common form of pleuritis is the serofibrinous here described.

Occurrence.—Affects all animals but principally the horse. In the horse pleuritis is usually a symptom of infectious fibrinous pneumonia; in the ox of tuberculosis, contagious pleuropneumonia and hemorrhagic septicemia, and in swine most frequently of so-called swine plague. Pleuritis, however, may occur unattended by pneumonia as is frequently observed in horses.

Etiology.—Pleuritis in animals is always due to infection. Cold, which was believed to be the most potent etiological factor, is now considered merely predisposing (pleuritis in sheep following shearing; exposure of horses to cold wind and rain). The microörganisms which produce pleuritis are many. Rarely is pleuritis a primary disease—it is most commonly seen in practice accompanying diseases of the lungs (pleuropneumonia). The microörganisms causing pleuritis may enter as follows: (*a*) Through penetrating thoracic wounds; (*b*) through deep contusions on the chest wall, especially if rib fractures be present (kicks, blows,

falls); (*c*) from disease foci in the lung, which are in contact with the pleura; (*d*) *via* blood and lymph microörganisms of certain specific diseases, notably those which affect principally the respiratory tract, may also invade the pleura and cause inflammation thereof (influenza, fibrinous pneumonia, swine plague, hemorrhagic septicemia, acute rheumatism). It may happen that the dominant lesions are in the pleura, in which case primary pleuritis is spoken of (pleurisy of the horse without pneumonia).

As predisposing factors may be mentioned refrigeration (cold), overexertion, long railway transports, and acute diseases of the respiratory tract (laryngitis). Subacute and chronic pleuritis may accompany tuberculosis, glanders, contagious pleuropneumonia of the ox, tumors (spread of sarcoma or carcinoma *via* contiguity of tissue, or metastasis, and animal parasites (echinococcus of ox, Cysticercus tenuicollis of sheep, sclerostomes in colts).

Symptoms.—Depending upon whether it is acute or chronic, primary or secondary the symptoms of pleuritis will vary greatly. In mild, circumscribed and in chronic pleuritis the symptoms are so vague that the condition is rarely recognized clinically. In the acute form, which is at times primary, they are as follows:

(*a*) First stage (congestion): The onset is sudden. The patient stops eating, seems stupid, and may show pains simulating mild colic. There is often a marked chill during which the temperature rises rapidly to 104° to 106° F. The muscles of the thorax (intercostals) tremble. The pulse is frequent (70 to 80), small and hard ("serous membrane pulse"). The respirations are accelerated (25 to 40) and of the abdominal type. If the pain is great, and the diaphragm not involved, the ribs may be rolled forward and held, breathing being performed by the flanks. The patient may not show cough or nasal discharge.

Percussing the thorax in this stage pains the patient and causes coughing. Sometimes on palpating the intercostal spaces sensitiveness is shown, especially in the region of the elbow. If the examiner's hand be laid against the thorax a marked fremitus may be felt. Unless the lung is

involved in this stage there is no change in the percussion sound.

The respirations seem shorter than normal and of an interrupted, catching character. On auscultation a rubbing, grating, frictional sound is heard synchronous with the respirations. In rare instances the grating sound may be heard a distance from the patient. The animal is usually stiff and when turned "moves as one piece" in a rigid wooden fashion.

(*b*) Second stage (effusion): In this stage the clinical picture is a good deal modified. The patient becomes more dyspneic, and the character of the breathing changed depending upon the quantity of exudate in the chest. If a considerable amount of fluid forms rather rapidly in the thorax (25 to 40 liters), at inspiration the ribs are rolled forward *ad maximum* and at expiration, which is accomplished by a double-pumping movement of the flanks, the lumbar region is elevated and the anus protruded, the manner of performing the respirations much resembling that noted in pulmonary emphysema. Along the costal cartilages at each expiration a groove is formed. The nostrils are dilated and often flapping. Percussion: As high up as the fluid in the chest extends, a marked flatness with resistance under the hammer is noted. The flat area extends across the ribs in a straight horizontal line. Above this line subdued resonance is heard. Changing the position of the body will shift the horizontal line. (Only feasible in small animals.) Auscultation: When effusion occurs the frictional sound disappears (in some instances it may still be heard above the area of flatness), and, as a rule, no respiratory sounds can be determined below the horizontal line. Above it the vesicular murmur is harsh; tubular breathing is frequently present.

The heart beat is weakened in this stage and may often be heard more distinctly on the right than on the left side of the chest. The pulse is rapid and softer than in the first stage. The temperature is very irregular. In serofibrinous pleuritis, when effusion takes place, it usually drops to nearly normal, but may rise again later. Its

character is decidedly intermittent or even remittent. Very high fever speaks for purulent pleuritis. General condition: In acute pleuritis the patient often remains standing during the entire attack (horse). If the patient lies down in the first stage, due to the pain, it rests on the well side, or if the condition is bilateral, on the sternum. In the stage of effusion, the patient lies on the diseased side. In pleuritis there is a tendency to edema on pendent portions of the body (under chest, etc.). A total lack of appetite persists.

Course.—Mild cases make a very rapid recovery, and are often not recognized during life. The effusion forms rapidly, in three to four days the thorax may be half filled; the resorption of the exudate, however, takes place gradually and may require two to three weeks or even several months, during which time the life of the patient is in jeopardy. The more serous the effusion, the more likely and rapid the resorption. With much fibrinous exudate present, adhesions between lung and thoracic wall are frequent. These adhesions usually persist and cause the patient to be ever afterward short-winded. Chronic pleurites are incurable. Death in acute cases may follow from asphyxia or exhaustion in two to three weeks.

Diagnosis.—The pathognomonic symptom of pleuritis is the frictional (rubbing) sound on auscultation. A sensitiveness of the intercostal spaces occurring in a disease (pneumonia) which pleuritis is apt to follow, is significant. In the second stage the horizontal line, limiting dorsally the extremely flat percussion sound, is characteristic. In pleuritis the onset is usually different from fibrinous pneumonia. In the latter the pulse is full, the conjunctiva congested (often mahogany colored), there is a rusty brown nasal discharge and the area of dulness on percussion is not so flat and resistant under the hammer. In pleuritis marked dyspnea is an early symptom, the pulse is hard and small (wiry) and on palpation muscular tremors over the region of the thorax are felt. Pneumonia is usually unilateral, pleuritis bilateral. Cough is much more easily induced in pneumonia than in pleuritis. The temperature is high

usually only in the beginning of pleuritis; in pneumonia the fever is of the continuous type and lasts five to nine days, to fall by crisis. In cases complicated with pneumonia the recognition of the pleuritis may be difficult. Weakening of the heart sounds and edema of the ventral part of the thorax are significant. In doubtful cases the use of the exploring needle to determine whether effusion is present or not is advisable. By drawing off some of the fluid and subjecting it to chemical (albumin), microscopic (pus cells, specific bacteria), and bacteriological examination (inoculation of animals) the form of pleuritis may be determined.

Prognosis.—Should be guarded. In fibrinous forms complicating pneumonia, the outlook is usually good. With great effusion affecting seriously the pulse, respirations and appetite, the prognosis is bad. If pus infection occur, death may be looked for. In pleuritis relapses are common. In cases which do recover from the prolonged acute attack, "heaves" (adhesions) is a common sequel.

Treatment.—The hygienic and dietetic treatment is the same as in pneumonia. Local applications to the chest, especially cold water in the early stage (first two to three days, when friction sound is heard) are good. When effusion is developed, hot applications (blankets wrung out in hot water) are better. In protracted cases or in chronic pleuritis, employ sharp blisters (spirits of mustard).

Drugs.—If there is acute pain (sensitiveness of intercostal spaces, marked stiffness on turning the patient), or in distressing cough, morphin (grs. v) or tincture of opium (℥iij) may be given. When effusion forms, diuretics and physics assist in the elimination of the fluid. Calomel (℥j) and aloes (℥vj) are given. Small repeated doses of the fluidextract of digitalis (℥j) so often recommended, should be administered with caution, watching its effect on the appetite and heart. Acetate of potash (℥j), pilocarpin (grs. iv), arecalin (gr. j), and eserin (gr. j) should be used only when the heart is not too weak.

If the quantity of effusion warrant (dyspnea) puncture of the thorax should be practised at once. If thoracentesis is properly performed it is not dangerous. The operation

is simple: In the seventh intercostal space, close to the anterior margin of the rib, and about 1 inch above the union of the cartilage and rib, shave, disinfect, and puncture the chest with a small sterile trocar. It is recommendable to first cut through the skin with a bistoury and draw the incision to one side that the skin and muscle wounds do not cover each other when the puncturing instrument is withdrawn. Care should be taken to prevent air entering the thorax during the operation. The fluid should be removed slowly and if the dyspnea become worse, coughing induced or the pulse become weak, the cannula should be instantly withdrawn and the opening covered with tar or collodion. Thoracentesis should be performed early and repeatedly to be of curative value.

HYDROTHORAX.

Definition.—The presence of an effused fluid in the pleural cavity. It is never of inflammatory origin and therefore occurs quite independent of pleuritis.

Etiology.—In a general way hydrothorax is due to a congestion in the venæ cavæ or its tributaries. It is most commonly noted in chronic heart, lung and kidney diseases and is usually associated with ascites, anasarca, and hydropericardium.

It may also accompany general anemia, hydremia and prolonged cachectic conditions following parasitism, carcinomatosis, etc.

Symptoms.—Same as the effusion stage in serofibrinous pleuritis.

Diagnosis.—History, finding organ primarily attacked (heart, lung, kidneys) and the prolonged, feverless course generally suffice to secure a diagnosis. In doubtful cases, the thorax may be tapped and fluid withdrawn and examined. It is usually much clearer, less flocculent and more watery than pleural exudate. It is straw-yellow in color and has a specific gravity of 1016, the albumin content below 2 per cent. Leukocytes are only sparingly represented.

Treatment.—As the primary condition is usually incurable, little can be done in hydrothorax. In great dyspnea, thoracentesis will afford relief.

PNEUMOTHORAX.

Definition.—The entrance of air into the pleural sacs. It is rarely due to other gases.

Etiology.—(*a*) Penetrating wounds through the outer wall of the chest or through the diaphragm (from the reticulum). (*b*) Rupture of the esophagus from the rough use of the probang. (*c*) Abscesses or gangrenous foci in the lungs which rupture opening bronchi into communication with the pleural sacs. (*d*) In rare instances rupture of the lung may be a cause.

Symptoms.—Severe dyspnea, which develops usually rapidly and may lead to death in twenty-four hours (pulmonary collapse), or from the microörganisms carried in by the air a purulent pleuritis (empyema) develops. The sound on percussion has a peculiar metallic ring which is usually heard over the whole chest. Pleuritis develops in most cases which modifies the percussion sound. On auscultation often no sounds are audible. If fluid is present metallic gurgling sounds are heard.

Diagnosis.—Usually not difficult in veterinary patients, as most cases are due to penetrating chest wounds.

Course and Prognosis.—Usually leads to pleuritis and death. Cases not due to wound infection, such as may follow rupture of the lung, can recover.

Treatment.—In human practice the air is pumped out of the pleural sacs by a special apparatus. Seldom useful in veterinary practice.

PART II.

DISEASES OF THE CIRCULATORY ORGANS.

CHAPTER I.

DISEASES OF THE HEART SAC.

PERICARDITIS.

Definition.—An inflammation of the heart sac.

Occurrence.—In animals pericarditis is usually secondary and associated with pleuritis. It, therefore, often accompanies pleuropneumonia. In the horse it may complicate infectious pleuropneumonia and in the ox and swine the pleuropneumonia of hemorrhagic septicemia. It may also be a symptom of influenza, strangles, glanders, or tuberculosis. In fact any extensive inflammation of the pleura may involve by continuity and contiguity of tissue the pericardium. From a clinical standpoint the symptoms of pericarditis are so often masked by those of the primary disease that it is overlooked.

In the ox pericarditis usually results from direct injury by foreign bodies which pass from the reticulum or rumen (traumatic pericarditis); or it may be due to tuberculosis.

Forms.—From a pathological standpoint pericarditis may be classified, like pleuritis, into fibrinous, serofibrinous, purulent, ichoric or hemorrhagic. This classification is of limited value clinically.

Etiology.—*Infection.*—Most of the pathogenic microorganisms affecting animals can produce pericarditis. In practice therefore it occurs concomitant with many infectious diseases.

Symptoms.—The serofibrinous form is most common in the horse. The patient presents symptoms like those in the first stage of pleurisy. It moves about very little, seems stiff and may show lameness in a forelimb. When effusion occurs the distention of the pericardium interferes with the heart action. The respirations become dyspneic and the pulse small, irregular and weak. The temperature is usually not high unless the primary disease is associated with high fever. The jugular veins become distended and undulate. Later dropsical swelling will appear below the sternum.

Course.—The course in serofibrinous pericarditis varies. Where the infection has been mild and little effusion occurs healing is prompt. However, in most cases the condition either leads to death within a week or ten days or becomes chronic causing symptoms of general heart weakness, distention of the jugular veins and dropsical swellings on the ventral portion of the thorax.

Diagnosis.—The diagnosis rests upon the physical signs determined by palpation, percussion and auscultation of the heart. On palpation in the early stages the animal may evince pain. In fibrinous pericarditis fremitus is felt by placing the hand over the cardiac region. When effusion is marked the heart beat becomes weak to imperceptible. On percussion the area of cardiac dulness is greatly increased extending as high as the shoulder line and as far back as the seventh rib provided the effusion is considerable. On auscultation friction sounds are heard resembling those of pleuritis but occurring synchronously with the heart beat and not with respirations. Obviously the friction sound will disappear with the development of effusion. It may return, however, when the fluid subsides. The splashing, cooing, tinkling or gurgling sounds described are rarely heard except in traumatic pericarditis of the ox.

Differential Diagnosis.—From pleuritis pericarditis can be differentiated only when the symptoms are well developed. On auscultation the friction is confined to the cardiac region and occurs synchronously with the heart beat. In pleuritis, unless the heart is dislocated and forced toward the right side where its beat is audible, the cardiac impulse is not much

affected. From diseases of the heart pericarditis can be distinguished only by the symptoms of local sensitiveness on palpation, increase in the area of cardiac dulness on percussion and the absence of cardiac bruits. Where the friction sound can be heard the diagnosis is greatly facilitated.

Prognosis.—The prognosis in pericarditis should be made with caution. While many wild cases recover, the severer usually lead either to death or chronic disability.

Treatment.—Beyond rest, good care and food, little can be done in pericarditis. Recommended are ice packs over the heart, but these are difficult to apply and their value problematical. During the stage of effusion digitalis (ʒss), caffein (ʒj), oil of camphor (ʒss), strychnin sulphate (gr. ¼) are recommended. The administration of normal saline solution subcutaneously is valuable.

Surgical.—Where the effusion causes great dyspnea and threatens cardiac arrest puncture of the pericardium should be performed. The trocar is inserted on the left side in the fifth or sixth intercostal space directing the instrument upwardly to avoid injuring the heart. Some practitioners follow with the injection of sterile Lugol's solution. The operation should be performed aseptically. It is rarely economical to treat chronic cases.

Traumatic Pericarditis of the Ox.—Definition.—An inflammation of the heart sac caused by foreign bodies.

Occurrence.—Traumatic pericarditis is one of the most common sporadic diseases of the ox. With the possible exception of tuberculosis of the heart sac it is the most common disease of the heart. It occurs not only among dairy cattle but also beef cattle, especially on farms where hay baling is practised, pieces of baling wire being picked up and swallowed.

Etiology.—The frequency with which foreign bodies (needles, wire, etc.) are found in the reticulum, to which attention is drawn in dealing with the diseases of the digestive tract, the close proximity of the reticulum to the pericardium and the marked contractions of this compartment of the stomach, are the most important factors in the etiology of this common condition.

Symptoms.—In traumatic pericarditis of the ox, the heart symptoms are usually preceded by those of traumatic indigestion (see this). Inquiry, therefore, should always be made into the past history of the patient in this regard. The cardinal symptoms are as follows: (*a*) In the early stages stiffness and disinclination to move. The patient remains down most of the time. (*b*) Dyspnea when the patient is forced to exercise, the abdominal type of respiration predominating. (*c*) The pulse is rapid and irregular. (*d*) A pronounced undulation in the jugulars (venous pulse) is seen. (*e*) Later edematous swellings appear under the throat, neck, brisket, and chest. (*f*) Percussion is usually painful, the animal wincing and grunting when the chest is struck over the heart region. An increased area of cardiac dulness may be determined in cattle if not too fat. (*g*) On auscultation, provided no effusion has taken place, a friction tone like that heard in pleuritis but synchronous with the heart beat is heard. If the heart sac is filled with fluid and gas, metallic tinkling tones modify the normal heart sounds which are muffled and distant. (*h*) The patient usually shows rise in temperature, but the fever is generally mild and atypical. Not infrequently the clinical symptoms of traumatic pericarditis are entirely overlooked, the first intimation of any trouble appearing when the patient drops over dead. The general condition of the patient due to the loss of appetite and attending infection or intoxication (septicemia, sapremia) grows bad. The patient emaciates, becomes anemic, weak, and may suffer from diarrhea.

Course and Prognosis.—The course in traumatic pericarditis is usually a prolonged one, the condition lasting often several weeks or even months. Exacerbations and remissions are very common. As a general rule, however, there is a slow but steady decline. Metastases are not uncommon, the disease assuming the form of a pyemia, leading to enlargement of the joints, lameness, etc. Pneumonia and pleurisy, and gastro-intestinal catarrh are frequent complications. Death may occur at any time during the disease from the foreign body penetrating the heart muscle or from injury to the coronary bloodvessels causing fatal

hemorrhage. The patient may also die from the attending sapremia. Occasionally cases occur in which great improvement in the condition is noted, the patient gaining in flesh, appetite, and strength. Usually, however, the improvement is only temporary. Rarer still are those instances where a spontaneous recovery follows the escape of the foreign body through an abscess to the outside world.

Diagnosis.—While in typical and advanced cases the diagnosis is easy, traumatic pericarditis in the earlier stage may be exceedingly difficult to recognize. Eber recommends, where the condition is suspected and fever exists, to give acetanilid (ʒiij) daily. This reduces the temperature but not the pulse, which remains high (100 to 120) if pericarditis is present. In doubtful cases an explorative puncture of the pericardium will determine the presence of fluid.

Treatment.—As nearly all cases are fatal, the immediate slaughter of the animal is recommended. In very valuable pregnant animals an effort to prolong life may be made by the use of such drugs as digitalis (ʒss); caffein (ʒj), or oil camphor (ʒj) subcutaneously. Stimulants (alcohol and ether) are also in order. In Europe puncturing the pericardium with a trocar has been employed.

HYDROPERICARDIUM.

Definition.—A filling of the heart sac with transudate not due to a pericarditis.

Etiology.—Usually is associated with hydrothorax, ascites, and anasarca. It may be secondary to chronic heart, lung, liver, and kidney diseases, or diseases of the blood, as hydremia, anemia, the cachexia of chronic parasitism, and the last stages of chronic infectious diseases (glanders, tuberculosis).

Symptoms.—First, those of the primary disease followed by general dropsy which involves the heart sac. The area of cardiac dulness is enlarged, heart tones weak, pulse weak, edema of the skin, dyspnea, and albuminuria.

Diagnosis.—Similar to acute pericarditis except that acute inflammatory symptoms (fever, pain, etc.) fail.

Treatment.—Generally of little value as the primary disease cannot be eradicated. Diuretics and diaphoretics (pilocarpin) are indicated. Tapping the pericardium may be tried.

PNEUMOPERICARDIUM.

Definition.—Gas in the heart sac.

Occurrence.—Rarely met with except in traumatic pericarditis of the ox.

Etiology.—Due to gas forming in the putrid exudate contained in the pericardium, the result of sharp-pointed foreign bodies, which come from the reticulum, penetrating the organ. Rarely occurs from penetrating thoracic wounds involving the heart sac.

Symptoms.—Like those noted under traumatic pericarditis: Dyspnea; on percussion increased area of cardiac dulness. Auscultation: Metallic tinkling sounds—heart beat muffled and distant. In some cases splashing sounds resembling those of a waterwheel may be heard a distance from the patient.

Prognosis.—Bad. Healing rare.

Treatment.—See Traumatic Pericarditis.

HEMOPERICARDIUM.

Definition.—Collection of blood in the heart sac. The blood comes from either the heart cavities, coronary blood-vessels, aortic or pulmonary trunk.

Etiology.—Spontaneous rupture or injury of the heart, coronary vessels, aortic or pulmonary trunks.

Symptoms.—In most instances leads to death in a few minutes. Only in those cases where the hemorrhage developed slowly would the patient survive long enough for clinical symptoms to appear. In such cases the symptoms are those of filling of the pericardium, pale mucous membranes, profuse sweating, dyspnea, rapid, irregular, feeble pulse, uneasiness and finally death.

CHAPTER II.

DISEASES OF THE HEART.

NERVOUS PALPITATION OF THE HEART.

HYPERKINESIS CORDIS.

Definition.—A sudden, tumultuous beating of the heart of purely nervous origin, and independent of any lesions in the organ.[1]

Occurrence.—Not common in animals, but may occur in highly nervous horses, especially colts handled too roughly when being broken, etc.

Etiology.—Nervous palpitation may result from over-exertion, great fear (stable fires), or anything which produces undue excitement. It may be secondary to mild gastric indigestion and occur in anemic conditions, or may follow influenza.

Symptoms.—The characteristic symptom of this disorder is a thumping movement of the thorax which may be seen, felt or heard. The patient is usually anxious, sweating copiously and somewhat dyspneic. On auscultation the heart beat is loud and fast, often at each impulse jarring the whole body. The pulse may be quite weak.

Course.—Acute, lasts in most cases only a few hours to one day.

Diagnosis.—Absence of organic heart disease and short duration of the condition are indicative.

Treatment.—Patient should be kept quiet and in a cool place. Moderate walking exercise is helpful. Internally chloral hydrate (℥j), morphin (grs. v), or bromides (℥ss) may be given.

[1] Most cases described under palpitation of the heart are undoubtedly due to (*a*) spasm of the diaphragm; (*b*) organic heart disease.

SLOW HEART BEAT. BRADYCARDIA.

Definition.—The frequency of the heart beat is less than normal.

Etiology.—From a physiological standpoint it is due to a stimulation of the vagus nerve from diseases of the brain affecting course of vagus, or reflexly from gastro-intestinal disorders. Disease of the heart (degeneration, atrophy, myocarditis) may also induce it. Often the cause cannot be determined.

Symptoms.—The pulse is too slow. In the horse it may be in extreme cases only 9–12–20 per minute. The force of the beat is normal and the condition of the patient good.

Diagnosis.—To determine whether bradycardia is due to an irritated vagus or to some lesion of the heart itself, sulphate of atropin (gr. $\frac{1}{4}$) may be injected subcutaneously. If from the vagus it will temporarily disappear after giving the atropin.

Treatment.—Excitants (alcohol, ether, atropin) may be tried if the condition produces symptoms of heart weakness, languor, stupor, or nervous symptoms (convulsions).

INTERMITTENT HEART BEAT.

Arhythmia Cordis.

Definition.—A condition in which one or more heart beats are omitted.

Etiology.—An intermittency of the heart is quite common in horses. It may exist for some weeks and disappear spontaneously. On exercise the symptom may temporarily disappear. Common causes are: (*a*) Brain diseases affecting the vagus (hydrocephalus, tumor, cerebritis); (*b*) digestive disorders (constipation, catarrh); (*c*) diseases of the heart itself (myocarditis, endocarditis).

Symptoms.—One or more heart beats are dropped. It may be every fourth, sixth or eighth beat and occur with great regularity. Usually the beat following the pause is louder than the others. It may happen that two beats

together are dropped. Occasionally, two beats occur in rapid succession followed by a long pause. In some cases exercise emphasizes the condition, in others it temporarily relieves it.

Course.—If due to some acute disease which it accompanies, it will disappear with the healing of the disease. In some cases it remains during the life of the patient, but never causes disorder.

Treatment.—If secondary the disease which it accompanies must first be eradicated (gastric disorders). Usually no treatment is demanded in idiopathic cases.

HYPERTROPHY AND DILATATION OF THE HEART.

Definition.—Hypertrophy is an enlargement of the heart due to a thickening of its musculature. Dilatation is an enlargement of the heart due to an increase in the size of its cavities. The two conditions usually coexist.

Etiology.—A pathological hypertrophy of the heart may be due to anything which interferes with the heart action from without: As an adhesion of the pericardium to the heart; chronic lung, liver and kidney diseases, in that they increase the heart's work by resisting the free flow of blood; or from within, as a valvular lesion. Generally the hypertrophy is confined to one chamber, although all may be involved. When the heart has increased sufficiently in size and strength to overcome the obstacle, the free circulation is restored and the hypertrophy is spoken of as compensatory. In case the hypertrophy cannot overcome the obstacle, dilatation will result.

Symptoms.—A compensatory hypertrophy may not cause any symptoms. In hypertrophy with dilatation the symptoms are as follows: The owner is attracted by the dyspnea which develops during work. In some instances the patient may have occasional attacks of vertigo or palpitations ("thumps") when exercised. Undulation of the jugulars is sometimes noted. In the latter stages edema appears under the chest. The pulse[1] is weak and arhythmic. Per-

[1] In hypertrophy without dilatation, the pulse is full and quite strong.

cussion: The area of cardiac dulness is increased extending back as far as the 7th rib; it may extend upwardly to the height of the shoulder joint. Auscultation: The heart beat is stronger and louder than normal. In some cases it may shake the whole body (palpitation). In the last stages general dropsy usually sets in.

Course.—Acute cases of dilatation often recover if properly treated. As a rule, however, the condition, coexisting with hypertrophy, becomes chronic leading to general dropsy and death. In advanced cases any unusual exertion (hard pulling, racing) may result in the patient falling dead in harness.

Diagnosis.—Increase in extent of cardiac dulness, abnormally loud systolic tone, weak arhythmic pulse and tendency to dropsical swellings are indicative of dilatation. A hard, full pulse with increased area of cardiac dulness on percussion, speaks for hypertrophy. From pericarditis the condition may be differentiated by the weakness of the heart tone and the frictional bruit in this disease. From hydropericardium by the absence of the gurgling or metallic tinkling sounds on auscultation.

Treatment.—Healing only possible in acute cases of dilatation. Allowing the patient absolute rest and giving cardiac tonics, especially digitalis fluidextract (℥ss) to which strychnin nitrate (gr. j) may be added are helpful. The patient should be well cared for and fed highly nutritious food.

MYOCARDITIS.

Definition.—An inflammation of the heart muscle.

Etiology.—Myocarditis in animals is usually of infectious origin. In practice it is seen to accompany influenza, septicemia and foot-and-mouth disease. Cases occasionally occur in azoturia or may follow overexertion or heatstroke. In cattle foreign bodies penetrating the heart muscle form a cause. Myocarditis may further be secondary to endocarditis and pericarditis. In man myocarditis commonly results from arteriosclerosis which in animals is extremely rare.

From a pathological standpoint three forms of myocar-

ditis may be distinguished: (*a*) Acute parenchymatous, (*b*) chronic interstitial, and (*c*) purulent (heart abscess).

(*a*) The acute parenchymatous form involves the muscle fibers of the heart. Macroscopically the heart appears often striped or spotted (tiger heart), is of friable consistency and lighter color than normal.

(*b*) The chronic interstitial myocarditis involves the intermuscular connective tissue which proliferates, leading in many cases to a thinning from atrophy of the heart muscle with occasional local distention of the cavities (heart aneurysm.)

(*c*) Abscess of the heart is usually seen in traumatic myocarditis of cattle and in pyemia. The musculature of the heart will contain a number of pea- to walnut-sized abscesses, or in some cases small, multiple, miliary pus centers.

Symptoms.—Acute myocarditis begins suddenly with symptoms of heart weakness (palpitation, very rapid pulse, dyspnea). Death may be apoplectic due to heart paralysis. Clinically it is extremely difficult to distinguish between myocarditis and acute dilatation of the heart, or the parenchymatous degenerations so common in febrile disorders. During life myocarditis is usually not diagnosable in animals. From pericarditis and endocarditis it may be distinguished by the absence of the characteristic bruits of these conditions.

Treatment.—Heart stimulants especially injections of oil of camphor (℥iij), or caffein (℥j), or veratrin are recommended.

ENDOCARDITIS.

Definition.—An inflammation of the endocardium which may be (*a*) acute or (*b*) chronic.

Acute Endocarditis.—Acute endocarditis is usually of infectious or hematogenous origin. It may therefore be due to several different microörganisms. In animals it is usually caused by the bacteria of septicemia and pyemia. The streptococci, staphylococci, and varieties of the colon bacillus, which have entered the blood in puerperal septicemia and pyemia of the ox or wound infection in the horse, are common causes.

Secondarily acute endocarditis may occur as a symptom of various infectious diseases (erysipelas of swine, influenza of the horse, articular rheumatism of cattle). Endocarditis the result of refrigeration, traumatism or from a spreading of the inflammation of pericarditis or myocarditis is rare.

Pathologically two forms of acute endocarditis are distinguished: (*a*) Verrucous, (*b*) ulcerous. These forms, however, are frequently combined.

The verrucous form leads to organized exudate forming on the margins of the valves, so-called vegetation, which leads to thickening, distortion and adhesions, rendering the valve inefficient. The ulcerous endocarditis is a necrotic inflammation of the endocardium with the development of pea- to hickory-nut-sized ulcers. These are usually the result of metastasis (lungs, kidneys).

Symptoms.—The symptoms are heart palpitation and very rapid, weak, often imperceptible, irregular, intermittent pulse (horses 80 to 160). In rare instances the heart beat is twice as fast as the pulse. There is dyspnea and high fever (104° to 105° F.) Characteristic of the condition are the systolic or diastolic bruits which occur, depending upon which valves or openings are involved. When of metastatic origin there may be brain (apoplexy), lung (asphyxia), kidney (hematuria), or limb (lameness) symptoms.

The course is either peracute causing death in a few hours, or acute ending fatally in a few weeks; or more rarely the course is chronic, leading to chronic valvular disease of the heart.

Differential Diagnosis.—The condition may be confused with a number of acute disorders such as pulmonary edema, pneumonia (dyspnea), septicemia and puerperal fever. If the characteristic bruits are absent a diagnosis may be impossible.

Treatment.—Consists in quiet and cold applications to the chest. Ulcerous endocarditis is usually incurable. The verrucous form, however, is more benign. Recommendable is digitalis (℥iv) followed by strophanthus (℥j). Heart weakness may best be combated by subcutaneous

injections of oil of camphor. Where fever is present, acetanilid (℥j) or salicylate of soda (℥j to ij) may be tried.

Chronic Endocarditis.—Etiology.—Occurs in the dog, horse, swine and ox in frequency in the order named. It usually follows acute endocarditis (septicemia, articular rheumatism of the ox, influenza of the horse, erysipelas of swine and hog-cholera). Otherwise the disorder may develop gradually following the abuse of the heart (overexertion, refrigeration, psychic influence). In animals it is rarely due to arteriosclerosis. In rare instances valvular troubles may be congenital (defects in the valves, oval foramen, or septum). Tumors leading to stenosis of the ostia are rare causes.

Pathologically valvular troubles lead either to insufficiency or stenosis of the valve or ostium concerned. Commonly these are combined.

General Symptoms.—The clinical features of valvular disease may be divided into two stages: (*a*) The stage of compensation, and (*b*) the stage of disturbance in compensation.

(*a*) From a resulting compensatory hypertrophy the trouble with the valve may be for a long time overcome. Clinically no symptoms exist other than either a systolic or diastolic murmur with hypertrophy of the left or right heart. The general condition and efficiency of the patient are not much disturbed.

(*b*) In the stage of disturbance in compensation the compensatory hypertrophy has been partially or totally overcome. Dilatation displaces hypertrophy. This induces symptoms of heart weakness. The pulse becomes fast, weak, inequal and irregular. The patient is dyspneic, especially after exercise, from a resulting congestion of the lungs. The mucous membranes and skin are cyanotic, and undulation of the jugulars is noted. The patient may show vertigo, especially when the head is elevated as in drenching, and drop to the ground. In the later stages symptoms of general dropsy appear (anasarca, ascites, hydrothorax, hydropericardium). The animal loses flesh, is weak and anemic. The urine may contain albumin (albuminuria from congestion of the kidneys).

Course.—The course is chronic. While an animal with chronic endocarditis may live a long time and give efficient service, especially in the stage of compensatory hypertrophy, the disease is progressive and sooner or later leads to disability and death. Once the compensation is overcome the patient develops the symptoms cited above and usually rapidly declines.

Differential Diagnosis.—In the absence of bruits a diagnosis may not be possible. On the other hand if the bruit is well developed, from its appearance at systole or diastole, the part of the cardiac region over which it can be best heard, the dyspnea on exercise, the increased area of cardiac dulness, the venous pulse, tendency to attacks of vertigo, absence of fever, etc., will furnish the clinician evidence on which to rest a secure diagnosis. Suspected cases should be exercised before examination.

An exact location of the valvular lesion in chronic endocarditis is much more difficult and not as important as in man. The following suggests the principal symptomatological differences noted in the individual valvular and ostial defects which result from this disease:

Individual Valvular and Ostial Defects.—(*a*) Mitral Insufficiency: Most common in the horse, dog and swine (left heart). Systolic bruits very loud; accentuated second heart tone. Pulse normal to weaker. Dyspnea, substernal edema.

(*b*) Mitral Stenosis: Diastolic bruits, weak pulse.

(*c*) Tricuspid Insufficiency: Most common valvular trouble of cattle (right heart). Systolic bruits, venous pulse, cyanosis.

(*d*) Tricuspid Stenosis: Diastolic bruits, venous pulse in the ox.

(*e*) Aortic Insufficiency: Diastolic bruits. Pulsation at base of neck; peculiar swishing sound on auscultation. Best heard near anterior border of fourth rib, approximately eight inches from bottom of sternum. Sometimes fremitus may be felt. Pulse strong, rapid, jerking ("water-hammer" pulse). (P. celer.)

(*f*) Aortal Stenosis: Systolic bruits with a very small, slow pulse in horse and dog. Vertigo from brain anemia.

(*g*) Pulmonary Insufficiency: Diastolic bruits (very rare).

(*h*) Pulmonary Stenosis: Systolic bruits (very rare).

Prognosis.—Unfavorable. The usual case of chronic endocarditis does not reach the veterinarian until so advanced that treatment is no longer economical.

Treatment.—Treatment of chronic endocarditis and valvular failure is indicated only in the stage of disturbance in compensation. Here the most valuable agent is digitalis, given in the form of Squibb's fluidextract (℥i). Associated with this, strychnin is often used (gr. $\frac{1}{4}$ to $\frac{1}{2}$ subcutaneously). When dropsy sets in such drugs as caffein, diuretin and strophanthus are indicated.

RUPTURE OF THE HEART.

Rupture of the heart, when not due to traumatism, is the result of pathological changes in the myocardium (fatty degeneration, aneurysms, myomalacia, echinococcus). The predisposing causes are conditions which increase blood pressure, such as excitement (operations, coitus, etc.); tympanitis or severe concussion of the body due to falls, blows, etc. Heart rupture leads to apoplectic death under symptoms of internal hemorrhage.

ANEURYSM OF THE AORTA.

This is a rare condition in animals due to arteriosclerosis. In horses it occurs most commonly at the root of the aorta near the bifurcation. Aneurysm also occurs in the anterior mesenteric and the external iliac arteries in the horse. Usually they produce no symptoms during life. In a few cases there may develop symptoms of heart hypertrophy with disturbed compensation and on auscultation over the region of the spinal column a peculiar buzzing sound is heard. The patient may also show epileptiform attacks. Usually death results suddenly and without warning from internal hemorrhage.

TUMORS OF THE HEART.

During life they are difficult to diagnose. Sometimes they produce symptoms of heart weakness or heart paralysis but generally are symptomless. The most common tumors are sarcomas, fibrosarcomas, and the less, lipomas, myxomas, fibromas, and osteosarcomas.

PART III.

DISEASES OF THE DIGESTIVE ORGANS.

CHAPTER I.

DISEASES OF THE MOUTH.

STOMATITIS.

Forms.—Depending on the cause, anatomical character, course and species of animal various kinds of stomatitis are recognized. The same cause may produce different varieties of the disease.

Catarrhal Stomatitis.—Character.—A catarrhal inflammation of the mucous membrane of the mouth.

Occurrence.—A very common disease of all domestic animals which when primary is due to irritants of various sorts which are taken into the mouth voluntarily with the food and water or involuntary as medicines in the form of drenches, electuaries and boli. Catarrhal stomatitis is secondary to a number of infectious diseases (foot-and-mouth disease, influenza, Rinderpest, etc.), diseases of the stomach and bowels, diseases which affect nutrition (anemia, rachitis) and poisoning with aconite, mercury, and lead.

Etiology.—The causes of catarrhal stomatitis are varied. In considering the etiology one should distinguish between the primary and secondary forms.

Primary Form.—(*a*) Direct injuries (sharp bits, rough forage, foreign bodies [corn cobs, bits of bone or wood], rough manipulations during dental operations, licked-off body hairs, etc.). (*b*) Chemical irritants (plants: aconite,

hellebore, euphorbium, tobacco, digitalis; minerals: chloral hydrate, bichlorid of mercury, chlorid of zinc, various blistering agents licked off the skin). (*c*) Thermic causes (hot drenches, frozen food). (*d*) Fungi (molds, rusts, smuts and yeasts). (*e*) Insects (caterpillars on leaves in fall, leaf-lice). (*f*) Bacteria.

Secondary Form.—(*a*) Symptomatic of diseased teeth (alveolar peritonitis, caries dentium and many surgical conditions of the teeth). (*b*) Shedding of decidious teeth (causing gingivitis or "lampas"). (*c*) Some of the infectious diseases (foot-and-mouth disease, Rinderpest, contagious pustulous stomatitis, etc.). (*d*) Spread of pharyngitis to mouth cavity. (*e*) Most of the diseases of the stomach and bowels. (*f*) Constitutional diseases seriously affecting nutrition and resistance (anemia, rachitis).

Symptoms.—In acute cases the patients resist attempts to examine the mouth ("mouth shy"), will eat slowly, especially roughage, show frequent thirst and salivation. In the early stage (congestion) the mucous membrane of the lips, cheeks and tongue is red, dry and swollen. Later the tongue is coated with a sticky, grayish (greenish in grass-fed horses) often foamy exudate. The hard palate is swollen ("lampas") and sometimes the tongue. There is salivation ("slobbering") a viscid, ropy saliva drooling from the commissures of the lips, especially marked when the mouth is opened. Sometimes the saliva is foamy. It may be retained in the mouth to be ejected at intervals. The saliva has a peculiar sweetish odor due to its retention and decomposition.

Usually there are no marked lesions present. Occasionally, however, small, gray papules appear on the teeth, surfaces of the lips and under the tongue from which later shallow, quick-healing ulcers develop. Constitutional disturbance is rarely noted.

Course.—In primary stomatitis the course is benign, ending in recovery in fourteen days. The course in the secondary form varies with the primary disease with which it is associated.

Diagnosis.—The recognition of stomatitis *per se* is not difficult. To determine, however, whether it is primary or

secondary is often not easy especially early in its development. From the history, the temperature, pulse and other symptoms of constitutional disturbance which occur in those diseases where stomatitis is a symptom, the diagnosis "secondary stomatitis" usually can be made.

Treatment.—Once the cause is removed the symptoms rapidly subside. The principal indications are to change the food, look after the teeth, remove any foreign bodies from the mouth and allow the patient constant access to good drinking water. Various "mouth washes" are recommended. The following are examples: Alum water (1 per cent.), creolin (1 to 2 per cent.), boric acid (2 per cent.), permanganate of potash (1 to 200). Vinegar one-half pint, common salt one tablespoonful mixed together in a quart of water is useful. In chronic cases nitrate of silver (1 per cent.) is employed.

Simple Vesicular Stomatitis.—*Sporadic Aphtha.*—**Definition.**—A sporadic inflammation of the mouth characterized by the formation of superficial vesicles in the mucosa.

Occurrence.—The disease is seen in horses and cattle. In certain years it may appear as an enzoötic, large numbers of animals becoming affected.

Etiology.—The cause is not definitely known. Animals pastured on fungi-infested clovers are most commonly attacked. It is probable that the fungi Uromyces occultus and Polydesmus exitiosus are factors. In some outbreaks infection seems to play a role. A simple vesicular stomatitis can be due to horses licking blistering ointments or the administration of irritant drenches not properly diluted. It can also occur from eating plants with sharp awns (barley), spicules, or even sharp stubbles. The disorder is not transmissible by inoculation.

Symptoms.—In the horse, following prodromal symptoms which resemble those of the initial stage (congestion) of catarrhal stomatitis, a vesicular eruption appears in the mouth, particularly under the tongue, mucous surface of the lips, at the commissures of the mouth and sometimes on the tongue. The blisters vary in size from a grain of wheat to a small bean, are sometimes umbilicated and filled with a clear, serous fluid. In three or four days they erupt

leaving behind erosions which usually heal in about a week. In some cases the eruption may extend to the skin about the mouth and nose. The patient is usually "mouth shy," slobbers, shows impaired appetite or is unable to nurse.

In the ox blisters from the size of a dime to a half dollar piece occur on the dental pad. These erupt leaving behind dark red, painful erosions which in two or three days are covered with new epithelium. The patient shows salivation and smacking of the lips.

Course.—The course is rapid and benign.

Diagnosis.—In the horse vesicular stomatitis might be confused with contagious pustular stomatitis. In the latter, however, pustules appear the eruption of which occurs simultaneously and frequently involves the external skin of the nostrils and lips. In the ox the disease is distinguished from foot-and-mouth disease by its non-infectiveness (animal inoculation), and the absence of fever and foot lesions.

Treatment.—The same as in catarrhal stomatitis.

Aphthous Stomatitis. — **Definition.** — In human medicine aphtha is a term used to express an inflammation of the mouth characterized by the formation of small, superficial ulcers on the mucous membrane. In veterinary medicine it is often employed to designate vesicles in the buccal mucosa. Here the term aphtha expresses the presence of a fibrinous false membrane due to infection, resting upon a comparatively intact mucous membrane. The following two diseases are considered aphthous:

Aphthous Stomatitis of Sucklings.—**Definition.**—An infectious disease characterized by the development of fibrinous pseudomembranes on the inflamed mucosa of the mouth.

Occurrence.—The disease occurs among calves, kids, lambs and foals. It is much less frequent after weaning. It usually appears enzoötically.

Etiology.—While undoubtedly due to infection the specific germ has not yet been isolated. Short streptococci and Gram-positive cocci have been isolated from the pseudomembrane. Healthy lambs inoculated with pure cultures developed stomatitis. The disease is most common in neglected sucklings kept in unsanitary quarters.

Symptoms.—Following symptoms of congestion of the buccal mucous membrane, in one or two days roundish aphthous areas appear in the mouth. These vary in size from a pinhead to a dime. They occur most commonly on the back and borders of the tongue, gums and at the commissures of the mouth. The areas are at first white but as they thicken become gray to yellow. Surrounding each area is a red zone. The pseudomembrane adheres rather firmly to the underlying mucosa and when removed it leaves behind a very red, bleeding erosion. Sometimes small vesicles occur along the borders of the lips, which erupt, forming brownish-yellow crusts. When the pseudomembranes slough the erosions left behind are soon covered with epithelium. In severe cases the number of areas constantly increases. Many become confluent, forming thick, brownish-yellow deposits upon the mucous membrane, which may become ulcerous. The process may extend to the gums and teeth which, through suppurative alveolar periostitis, often become loosened.

The young animal does not nurse well, shows salivation and fetid breath. Due to lack of nourishment and general infection the little patient becomes anemic, emaciated, may suffer from diarrhea or pneumonia, eventually dying of inanition.

Course.—In very young animals the disease frequently leads to death. The mortality varies from 5 to 25 per cent. depending on the outbreak.

Diagnosis.—In well-developed cases the white or yellow spots associated with acute stomatitis and the vesicles occurring on the borders of the lips are very characteristic. It may be differentiated from vesicular stomatitis by the presence of blisters in the latter and from so-called diphtheria of calves by the presence of the well marked, thick, necrotic areas and deep ulcers seen in this disease.

Treatment.—As the disease is communicable a separation of the healthy from the sick and the constant inspection for and sorting out of new cases should be practised. Patients which cannot nurse should be hand-fed. The medicinal treatment consists in syringing out the mouth with disinfect-

ants, such as boric acid (4 per cent.), copper sulphate (2 per cent.), or potassium chlorid (3 per cent.).

Papulous Stomatitis.—Definition.—A benign, contagious disease of the mouth of cattle, due to an ultramicroscopic virus and characterized by an eruption in the mucosa and skin around the mouth of yellowish-gray, flattened papules. Probably does not occur in the United States.

Etiology.—An ultramicroscopic virus. The disease is readily transmitted to healthy animals by inoculation into the mucous membrane of the mouth, subcutaneously and intravenously. The mode of natural transmission is not yet known.

Symptoms.—The period of incubation is one to two weeks. The nodular eruption may involve the muzzle, lips (outer and inner surface), palate, tongue, cheeks, and gums. The nodules are from the size of a wheat grain to a small pea, are at first red and later grayish-yellow in color. Around each nodule is an area of congestion. In the latter stages the centers undergo softening, irregular-shaped pits forming. The bases of the pits are granular, at first red or black and later yellow in color. By coalescence large areas (dollar-sized) appear. The condition may persist for several weeks. There is usually no general disturbance, although in isolated cases fever and fetid breath have been observed.

Diagnosis.—The peculiar, flattened nodules which appear not only in the mucous membrane, but also on the external skin (muzzle) are significant. Vesicles do not occur and the feet are not involved.

Prognosis.—The disease always ends in healing.

Treatment.—The same as in catarrhal stomatitis. As the disease is contagious separation of the affected from the healthy is indicated.

Mycotic Stomatitis of the Ox.—Definition.—This is a non-infective inflammation of the mouth, muzzle, skin of the region of the coronets and sometimes of the udder and teats which occurs in cattle on pasture. It is characterized by the formation of minute vesicles and later ulcers which usually readily heal.

Occurrence.—The disease is quite common in the United States among cattle running at pasture, and most often breaks out in the fall, especially when a rainy season follows a period of drouth.

Etiology.—The cause of mycotic stomatitis is evidently certain fungi which infest grasses. Clover pasture seems to be the most dangerous in this regard. The disease is often enzoötic, affecting a number of animals subjected to like condition in the community.

Symptoms.—The initial symptoms are those of impaired appetite, painful mastication and slobbering. In severe outbreaks lameness may be the first symptom noticed by the owner. On examination of the mucous membrane of the mouth minute vesicles are noted. Later small ulcers appear particularly on the mucous surfaces of the lips, under the tongue and on the dental pad. Sometimes gray-colored fibrinous deposits are present. Erosions, scabs and crusts form on the muzzle and external surface of the lips. When the animal is lame the skin and subcutis of the coronet become edematous, hot and tender. In some instances the swelling is cracked and creviced and may show evidence of secondary pus infection. When the udder is involved scabs and fissures appear on the teats and skin of the udder, causing cows to resist the operation of milking. Milk secretion may be partially suspended. The general condition of the animal is involved only in severe attacks. They usually move about stiffly, frequently shaking their feet, or when standing assume the attitude of a horse with founder. The temperature may be slightly elevated (mild fever). There may be emaciation from inability to eat and in rare instances individual animals may show diarrhea.

Diagnosis.—The diagnosis is not difficult, the disease occurs among pastured cattle, usually attacks only a percentage of the herd, is generally benign in its course, and once the patients are removed from the infested pasture the symptoms rapidly subside. From foot-and-mouth disease it is distinguished by the fact that it is not transmissible by inoculation, does not affect sheep and swine, fails to develop the characteristic large vesicles and is less apt to

involve the feet. Foot-rot and ergotism are not attended by stomatitis. Necrotic stomatitis of calves is seen only in very young animals, does not involve the external skin and the lesions are characterized by a necrosis of the mucous membrane of the mouth. The feet are not attacked.

Prognosis.—The prognosis is good; only in aggravated cases are losses recorded from secondary infection.

Treatment.—The principal indication in treating this disorder is to remove the cattle from the infested pastures, best placing them in a barnyard and feeding soft feeds. The animals should be given constant access to fresh water. It is recommended to place in the water borax (1 oz. to 1 gal. of water). In range cattle four ounces of crude carbolic acid may be mixed with twelve quarts of barrel salt, the cattle being permitted to lick this at will. The foot lesions are treated according to the general principles of surgery. As a rule the animals rapidly recover when they are removed from the infested pasture.

Phlegmonous Stomatitis.—Definition.—A purulent infection of the mucous membrane and submucous connective tissue of the mouth.

Etiology.—Phlegmonous stomatitis is the result of infection with pus-producing bacteria or fungi. It may be either primary or secondary. The primary form results from traumatism. The ingestion of food containing caterpillars, aphis, irritant insects or molds may also produce it. Irritant drugs or chemicals which gain access to the mouth, caustics, acids, strong carbolic acid, croton oil, chloral hydrate crystals, etc., are causes. Secondary phlegmonous stomatitis may be a symptom of strangles, anthrax, purpura hemorrhagica, and malignant head catarrh of the ox.

Symptoms.—The symptoms are those of a severe inflammation of the buccal mucous membrane involving principally the tongue and lips. In some instances the lips are so swollen that the mouth cannot be closed. The tongue may be swollen so that imprints of the incisor teeth are left upon it. There is salivation and the patient is unable to eat. Fever may be present. The process may involve the pharynx leading to dysphagia and nasal discharge. The sublingual and para-

pharyngeal lymph glands become acutely enlarged and abscess formation in them often results.

Course.—Primary cases usually heal after the abscess is evacuated. In exceptional cases the larynx, trachea or even the bronchi may become involved leading to death. In secondary cases the course is contingent upon the disease which the phlegmonous stomatitis accompanies.

Treatment.—The treatment is the same as for other forms of stomatitis the only exception being that abscesses in the neighboring lymph glands should be opened and drained.

CHAPTER II.

DISEASES OF THE PHARYNX.

PHARYNGITIS.

SORE-THROAT. ANGINA SIMPLEX.

Definition.—An inflammation of the pharyngeal structures which usually involves the soft palate and tonsils. Pharyngitis is frequently associated with laryngitis, and may appear as a primary or a secondary disease.

Occurrence.—Horses and swine are the principal victims. Cattle and sheep are seldom attacked. When due to infection pharyngitis often occurs as an enzoötic. The disorder is most common in the spring and fall when weather changes are sudden and frequent.

Etiology.—Primary pharyngitis. As in stomatitis, pharyngitis may be due to direct injury to the pharynx from foreign bodies or chemical substances (strong medicines, poisonous plants, chloroform, etc.), or the giving of hot drenches. Refrigeration is a common predisposing cause. Infection with streptococci or the necrosis bacillus and other bacteria commonly produce it. In rare instances it may be due to parasites (gastrus larvæ).

Secondary pharyngitis may be due to a spread of stomatitis, rhinitis or laryngitis to the pharyngeal mucosa or it may be a symptom of many of the specific infectious diseases such as strangles, influenza, purpura hemorrhagica, hemorrhagic septicemia, hog-cholera, anthrax, etc. In the horse suppurative pharyngitis with peri- or parapharyngeal abscess formation is usually a symptom of strangles.

Forms.—From a pathological standpoint the following forms of pharyngitis are distinguished: (*a*) The catarrhal, which is the mildest form. (*b*) The suppurative, which

usually leads to abscess formation about the pharynx. (*c*) Croupous, a pseudomembrane appearing over the mucous membrane. (*d*) Diphtheritic, a necrosis of the mucous membrane associated with which is phlegmon and swelling of the lymph glands. Diphtheritic pharyngitis commonly is associated with foreign body pneumonia and general septicemia.

Symptoms.—The most conspicuous symptom of pharyngitis is difficulty in swallowing (dysphagia). In the horse this is expressed by regurgitation through the nostrils of fluids (drinking water) and food. In swine the patient holds the head and neck stiffly, is restless and often squeals when it attempts to swallow. In acute pharyngitis the solid food may be ejected from the mouth after being partially chewed. As saliva is swallowed only in part, slobbering is a common symptom. The patients usually hold the head extended, nose poked out and are disinclined to flex the head upon the neck. Palpation over the region of the pharynx shows the parts to be hot and tender. There is usually bilateral nasal discharge mixed with saliva and food particles. The patient usually coughs especially when the upper trachea is pinched (larynx involved). In severe cases (phlegmon, abscess, diphtheritis) there may be pronounced dyspnea (edema of glottis), rattling sounds in the throat and marked swelling of the parotid region. If embolic or foreign body pneumonia is present, the expirium becomes fetid, there is dulness on percussion over the thorax and rales and bronchial tones on auscultation. Fever is present in most cases, especially in those arising from infection, the temperature reaching 104° F. Pharyngitis due to traumatism or chemical action is only associated with fever when secondary infection takes place. If the appetite is impaired, the patient loses flesh during the attack.

Diagnosis.—The diagnosis of pharyngitis is usually not difficult especially in animals where an ocular examination of the throat is possible. In horses, however, where this is not permissible it is more difficult. To determine whether the condition is primary or secondary one must pay especial attention to the other symptoms present, such as would

occur in strangles, influenza, etc. Obviously the examiner should be on the alert for foreign bodies and tumors in the pharynx which produce symptoms of dysphagia.

Course.—The course is very varied. A simple catarrhal pharyngitis in horses usually heals in three to four days. In swine, however, the termination is often fatal. Suppurative pharyngitis leading to secondary abscess and ulcers are often quite obstinate and may continue until surgical interference provides drainage for the pus. Pharyngeal paralysis and roaring are not uncommon sequels. Death may occur from asphyxia, septic infection or intoxication or from pulmonary gangrene.

Treatment.—The patient should, if possible, be placed in a warm, well ventilated stable free from dust and irritant gases. Only soft foods (gruels, bran mashes, grass) should be fed. In swine milk may be given. In horses where dysphagia is complete on account of the danger of even fluids entering the lungs the patient should be made to fast for two or three days or fed and watered through a stomach-tube or through the rectum. Chlorate of potash (1 oz. to 2 gal. of water) is popularly used. For threatening dyspnea tracheotomy should be employed. Subparotid abscesses should be opened and drained. Local applications, hot water (Priessnitz cataplasm) are helpful. Infrictions of gray mercurial ointment are recommendable. Strong blistering liniments, however, should be avoided. Local applications are too dangerous in the larger animals. Drenching should be prohibited. When the patient is able to swallow, expectorants such as tartar emetic and ammonium chlorid combined with powdered licorice root may be given as an electuary. Symptoms of septicemia (high temperature, rapid pulse, muddy mucous membranes, etc.), are best combated with large doses of oil of camphor administered subcutaneously. In swine when suffocation threatens, an emetic should be given (white hellebore or ipecac, grains xxv).

PARALYSIS OF THE PHARYNX.

Definition.—Any condition of the pharynx which interferes with swallowing.

Etiology.—Paralysis of the pharynx is usually secondary to: (*a*) An acute pharyngitis which accompanies an attack of strangles (parapharyngeal abscess). (*b*) Forage poisoning of which it is often a prominent symptom. (*c*) Bulbar paralysis in diseases of the central nervous system (meningitis, cerebrospinal meningitis). (*d*) Tumors in the pharynx (cysts, papillomas, polypi, carcinomas) and more rarely along the course of the pneumogastric nerve. (*e*) In certain infectious diseases (rabies, acute infectious bulbar paralysis of cats). (*f*) In certain intoxication diseases (parturient paresis of cows).

Symptoms.—The principal symptom is dysphagia. In horses and cattle food and water mixed with saliva are regurgitated through the nose. If no food is taken drooling from the mouth occurs. Attempts at swallowing produce loud, gurgling sounds. Palpation of the pharynx through the mouth fails to produce contraction of the pharyngeal muscles.

Course.—The course depends upon the cause. When due to inflammation healing may follow in a few weeks (rupture of abscess). The successful removal of tumors will immediately arrest the symptoms. When due to forage poisoning or acute diseases of the central nervous system (rabies, bulbar paralysis) the course is rapid and fatal. In parturient paresis most cases recover under modern treatment (air inflation of the udder). As a general proposition prolonged paralysis of the pharynx is serious as it prevents the proper nutrition of the patient and from food and saliva entering the windpipe and lungs frequently is followed by fatal foreign body pneumonia.

Diagnosis.—A careful palpation and inspection, when possible, of the pharynx should be made in all cases to exclude foreign bodies (corn cobs, pieces of wood, etc.) or to determine whether tumors or parapharyngeal abscesses are present.

Treatment.—As noted, in cases of paralysis due to acute inflammation (abscess) a spontaneous recovery may occur. Surgical intervention is often effective (see surgery). Blisters and the electric battery applied to the external throat rarely

do much good. Subcutaneous injections of nerve tonics (strychnin nitrate gr. $\frac{1}{4}$-$\frac{1}{2}$ once daily) are recommended. While the patient is unable to swallow it should be fed through a stomach-tube. Obviously animals suffering from rabies should be destroyed.

PARASITES IN THE PHARYNX.

Larvæ of the bot flies (Gastrophilus equi and G. hæmorrhoidalis) sometimes are found attached to the upper wall of the pharynx. In rare instances they have been known to induce severe pharyngitis, or by entering the larynx, suffocation.

Horse leeches (Hæmopis sangui suga) affect horses and mules in Southern countries. They attach themselves to the wall of the pharynx and suck blood. Their presence is suspected from nasal and buccal hemorrhage, which they occasionally induce. Large numbers may cause fatal loss of blood or serious anemia. The treatment usually advised is to irrigate the throat with salt and vinegar or creolin (2 per cent.). Inhalations of turpentine or ammonia fumes are also useful. Prevention consists in filtering the drinking water.

Hungarian flies (Simulia columbacœusis) attack Hungarian cattle and sometimes reach the pharynx through the mouth and nose. Occasionally they produce serious pharyngo-laryngitis leading to suffocation.

CHAPTER III.

DISEASES OF THE STOMACH AND BOWELS.

VOMITING. VOMITION.

Definition.—The forcible ejection of the contents of the stomach through the esophagus and mouth or nose.

Occurrence.—Vomiting is a symptom and not a disease. It occurs frequently in carnivorous animals but is rare in herbivorous. Horses seldom vomit and when they do it commonly indicates rupture of the stomach. True vomiting, the ejection of the contents of the abomasum, in the adult ox and sheep is so rare as to be denied by some authorities; the regurgitation of paunch contents, however, quite common.

Vomiting may have either a central or a peripheral origin. The former is due to some disturbance acting directly upon the vomiting center in the medulla oblongata (central vomition), and the latter to an irritation of peripheral nerves, usually of the pharynx or stomach, which reflexly stimulates the vomiting center (reflex vomition). In animals vomiting is usually reflex. It is one of Nature's efforts at self-healing, not only removing indigestible or deleterious material from the stomach, greatly to the relief of the patient, but at the same time, being a complex phenomenon, exerts a beneficial effect upon many other organs, provided, of course, the vomition is not continued too long.

Etiology.—Reflex vomition most commonly is caused by: (*a*) Overloading of the stomach; (*b*) irritation of the stomach by irritant foods or certain drugs (emetics); (*c*) gastric catarrh; (*d*) gastric parasites; (*e*) foreign bodies in the stomach; (*f*) occlusion of the pylorus or in ruminants of the openings between compartments of the stomach (tumors, foreign bodies); (*g*) stoppage of the intestines (dislocation of bowel, tumors, foreign bodies); (*h*) acute peritonitis, either

parietal or visceral; (*i*) irritation of the pharynx (foreign bodies, tumors, catarrh); (*j*) diseases of the esophagus (rare), or enlarged tubercular mediastinal lymph glands in the ox compressing the esophagus.

Central vomition, which is rare in animals, is due to: (*a*) Injuries and diseases of the brain (medulla); (*b*) uremia and cholemia; (*c*) certain poisons (apomorphin, arecalin, chloroform, veratrin); (*d*) sea-sickness.

Symptoms.—Vomiting is often ushered in by symptoms of restlessness, retching, salivation and dyspnea. While in cattle a regurgitation of paunch contents is often not associated with these prodromal symptoms, the stomach contents being ejected without suffering, in some cases the act of vomiting is distressing. The ox is restless, trips from side to side and switches its tail, may get up and down, arch the back, draw its feet up under the body, violently contract the muscles of the abdomen, and through the open mouth and nose discharge in a stream one or two gallons of stomach contents. The horse seems anxious, restless, covered with sweat, draws its legs up under the abdomen, contracts the muscles of the neck, extends the head, draws up the muscles of the abdomen and ejects through the nostrils, and in part through the mouth, the stomach contents.

The act of vomiting may occur only once or be repeated. Repeated vomition speaks for gastritis, peritonitis or occlusion of the bowels. In rare instances it may be due to disturbance in the central nervous system. After the act the patient is more or less distressed and if the vomiting is continuous quite exhausted.

In the ox the ejected mass consists mainly of paunch contents recognizable by its characteristic odor. In the horse it is made up of partially digested, fluid stomach contents sometimes mixed with blood. The reaction is decidedly acid.

Diagnosis.—Obviously it is not difficult to recognize that vomiting exists. It is not always an easy matter, however, to determine its cause. Where the act is the result of a mere overloading of the stomach it is usually not repeated and the animal permanently relieved. On the other hand its continuance speaks for some pathological change in the stomach

or the presence therein of a toxic substance (arsenic, carbolic acid). Blood in the ejected mass speaks for gastritis, ulcers, tumors or wounds of the stomach. If bowel contents are ejected it indicates an occlusion of the bowel. In some cases of secondary dilatation in the stomach of the horse the ejected mass will resemble the contents of the colon. The significance of vomition depends upon its cause and the disease underlying it. In the horse it is considered more serious than in other animals as it so often expresses rupture of the stomach. Further, some of the vomited mass may become aspirated and produce foreign body pneumonia, or more rarely suffocation from blocking the nostrils. It is, therefore, recommendable to make a careful examination of the animal after vomiting to determine whether or not the stomach is intact. As rupture of the stomach leads inevitably to peritoneal sepsis or peritonitis the symptoms of these conditions will soon become evident. The pulse, temperature, visible mucous membranes and peristalsis should be carefully noted for any change which may be significant of this fact.

Treatment.—Theoretically if vomiting is an effort to remove from the stomach a surplus of contents or irritant matter it should not be interfered with. Emetics (tartar emetic, apomorphin, white hellebore, veratrin, ipecacuanha) may even be given to stimulate further vomiting. However, in animal practice, except with carnivora, this is rarely necessary. In the horse it is recommended to use the stomach-tube as directed in acute dilatation of the stomach. In the ox we are sometimes called upon to arrest vomition. Narcotic drugs (opium, chloral hydrate, bromides) may be given per rectum or morphin subcutaneously. Camphor (℥ij) mixed with egg yolks is recommended.

GASTRO-INTESTINAL CATARRH OF THE HORSE.

Catarrhal Gastro-enteritis.

Definition.—A catarrhal inflammation of the mucous membrane of the stomach and bowels. While it occurs as a primary disease, it is often a secondary condition. It may be acute or chronic.

Occurrence.—Gastro-intestinal catarrh is a very common disease of horses.

Etiology.—Primary gastro-intestinal catarrh is due to: (*a*) Bad food (moldy forage, smutty oats, rotten straw), forage containing irritant weeds or sharp objects. Food which is too hot or on the other hand frozen and food containing foreign material as sand.

(*b*) Good food injudiciously fed. (Too rapid eating with incomplete mastication when very hungry, not enough saliva being mixed with food hastily swallowed; overloading stomach. Sudden change from accustomed to unaccustomed foods as oats to corn, corn to barley or wheat, etc.).

(*c*) Water. Large quantities of cold water when hot and fatigued. Water from stagnant pools.

(*d*) Disturbance in mastication (bad teeth).

(*e*) Psychic influences. (Extreme nervousness in race horses, casting, tying head too high, pain following operations or wounds.)

(*f*) Animal parasites (ascarides).

(*g*) Irritant drugs and poisons (arsenic, calomel, acids, alkalies).

Chronic gastro-intestinal catarrh is due to much the same causes as the acute but acting less intensively. They are:

(*a*) Bad food (sanded food, frozen food).

(*b*) Improper feeding.

(*c*) Bad teeth (sharp teeth, split teeth, alveolar periostitis, caries, etc.).

(*d*) Vices (wind sucking, cribbing).

(*e*) Chronic diseases of liver, lungs, heart (induce congestion of portal system).

(*f*) Parasitism.

(*g*) Senility (most decrepit "anatomy skates" suffer from chronic gastro-intestinal catarrh).

Acute gastro-intestinal catarrh is secondary to acute general infectious diseases (influenza, strangles), blood diseases (anemia, leukemia, pseudoleukemia, etc.). It may also be embolic in origin from Strongylus armatus in anterior mesenteric artery.

Symptoms.—*Gastric Symptoms.*—Impaired, lost or capricious appetite. In some cases the appetite is vitiated (eat unnatural things). The patients drink little water. Tendency to yawn and vomiting is rare. The mucous membranes are "muddy," discolored, those of the mouth often coated with soapsuds-like foam. The expirium is sweetish, nauseating. The pulse, respirations and temperature are usually little affected in primary cases. The patient is languid, lazy, sweats and tires easily when at work.

Intestinal Symptoms.—If the stomach is not involved appetite may be normal. If diarrhea exist there is great thirst. The peristalsis is lively and the borborygmus may be audible quite a distance from the patient. Colicky pains especially after eating or drinking. Dung passed at first in small, hard, mucus covered pellets, later softer (cow dung consistency) and finally diarrhea, the discharges very fluid and fetid. Anal flatus is frequent, loud and fetid. Some patients are sensitive to palpation over region of small bowels. Icterus appears if duodenum is involved. Urine is acid; indican increased. In chronic cases on account of the irreparable connective tissue thickening of the bowel mucous membrane and the atrophy of the glands the nutrition of the organism suffers. The patient loses flesh, the abdomen becomes "tucked up," the hair coat dull, long and erect, the skin "scurfy," harsh, inelastic and leather-like ("hide bound"). Anemia, emaciation, cachexia appear toward the end. Vertigo and symptoms of immobility appear in some cases.

Course and Prognosis.—Acute gastro-intestinal catarrh usually heals in three to seven days if the case is properly handled. It rarely becomes chronic. Chronic cases, however, with frequent exacerbations and remissions, last for months and finally lead to death from inanition. The prognosis in acute cases in very young or very old patients is less favorable. The mortality is about 1 per cent. Chronic catarrhs are much more serious and especially in old horses with bad teeth and where a prolonged treatment with regulation of the diet is not feasible, usually end in death.

Treatment.—A hygienic and dietetic treatment is all-important. The surroundings of the patient should be light, clean and well ventilated. Good grooming should be insisted upon. Examine and if necessary "dress" the teeth. In acute catarrh it is advisable to withhold food for two or three days or permit only small quantities of easily digested food (fresh grass, fine Timothy hay, linseed meal, bran mashes if palatable to patient). The following mixture is suggested: Oats, 2 parts; bran, 1 part; malted barley, 1 part. Scald or steam and let stand twenty-four hours, then feed. A teacupful of linseed which has been boiled to a jelly in a gallon of water and poured over a bran mash is useful. Allow the patient plenty of salt and free access to water. The medicinal treatment is largely symptomatic. In overloading of the stomach use:

1

℞—Hydrargyri chlorid. . . . ℥iv
Pulv. sacchari. . . . . ℥ij
M. f. pulv. nr. iv.
Sig.—One daily.

2

℞—Arecalin . . gr. ss
Aqu. dest. . . . . ℥ijss
M. D. S.—One dose subcutaneously.

3

To arrest fermentation:
℞—Acid hydrochlor . . ℥ss
In bucket of drinking water.

Or:

4

℞—Creolini Pearsonii . . . . ℥ss
Pulv. rad. glycyrrhiza . . . . . ℥j
Pulv. althæ et aqu. q. s. f. boli nr. iii.
Sig.—Daily one bolus.

5

℞—Sodii sulph. . . . ℥vss
Potassii sulph. . gr. xv
Sodii bicarb. . . . . ℥ivss
Sodii chlorid. . . . . ℥ijss
M. D. S.—Tablespoonful in each feed.

To the above gentian, rhubarb or calamus may be added. Diarrhea is combated by employing first a laxative followed

with antiseptics (creolin ℥j; or opium ʒj; or styptics such as tannin ʒj, acetate of lead ʒj, silver nitrate grs. x–xv, dissolved in rain water). Starch water, alum water per rectum or a solution of silver nitrate (1 to 500), alum or tannin (2 per cent. solution) are very useful in persistent diarrheas.

In threatened collapse oil of camphor (ʒij) subcutaneously.

SO-CALLED COLICS OF THE HORSE.

The term colic is a collective one and applies to all conditions which cause abdominal pain. Colic is, therefore, a symptom and not a disease. While most abdominal pains come from the stomach and bowel they may also emanate from a number of other organs. Acute diseases of the peritoneum, liver, kidneys, urinary bladder, uterus, ovaries, esophagus and pleura (rarely) may too be accompanied by symptoms usually termed "colic." To consider all conditions in the horse which produce more or less violent abdominal pain a specific disease, and to treat them all more or less alike, is unscientific and a menace to the patient.

In the older literature abdominal pains due to stomach and bowel disorders were called "true colics," while abdominal pains originating in other organs were known as "false colics."

Were it possible in all cases to make an accurate diagnosis the clinical term "colic" would disappear from veterinary as it has from human medicine.

The principal conditions which produce severe gastro-intestinal pain named in order of frequency are: (*a*) Impactions of masses of feces in the small and large intestines. (*b*) Impactions of the small or large intestines complicated with displacement of the bowel. (*c*) Distention of the stomach with food masses or gas, and (*d*) a primary inflammation of the walls of the stomach and bowels.

Simple impactions, impactions with displacement and distention of the stomach may become complicated by rupture of the wall leading to peritonitis or peritoneal sepsis. Following displacements enteritis and peritonitis usually occur and from the absorption of toxins and bacteria

contained in the stationary fecal mass an intoxication or infection of the patient may result. As a general proposition gastro-enteric pain appears suddenly, lasts for several hours and ends in the recovery or death of the patient. Occasionally, however, due to some organic lesion in the bowel wall (stenosis, tumors, ulcers or abscesses, diverticula, dilatation of the cecum) or hernias or intestinal stones or parasites, the symptoms of pain may last for several days or weeks. They are, however, usually intermittent and not continuous.

Etiology.—In general those symptoms of pain in the horse which were formerly designated "true colics" are due to causes which may be classified under two groups: (1) Predisposing, which may be either anatomical or pathological, and (2) exciting or immediate causes.

1. Predisposing Causes. — *Anatomical.* — To the anatomical causes may be ascribed the peculiar anatomical arrangement of the stomach and bowel in the horse. The small stomach and peculiar implantation of the gullet which make vomiting difficult, long mesentery, narrow ileo-cecal opening, the pouch-like dilatation and funnel-like termination of the right upper colon, the pelvic flexure, and the large cecum with both of its openings at the upper end, are the principal anatomical factors which interfere with the normal progress of the ingesta.

Pathological.—Diseases of the digestive organs: For instance, diseases and irregularities of the teeth, catarrh of the mucosa of the stomach and bowels, internal abscesses, paralysis with dilatation of the cecum or rectum, stenosis of the ileum, tumors, hernias, enteroliths and animal parasites in the bowel and bloodvessels.

2. Exciting Causes.—The exciting causes of gastro-enteric pains are found chiefly in the food. Good food if taken in too large quantities, food which is unfit (wet straw) sudden changes from one kind of food to another, food difficult to digest (rye, barley), food which is fermenting (new hay, new oats, new corn) food infested with fungi, or toxic plants, and feeding at irregular intervals are the principal exciting causes. Indirectly the weather is of im-

portance. Very hot or cold, damp weather which no doubt influences metabolism is an etiological factor to be reckoned with. Finally overexertion especially in hot weather and after a heavy feed, or on the other hand lack of exercise are causes. In rare instances the vice known as "wind sucking" may induce gastric distention and pain.

Statistics.—*Morbidity.*—About 10 per cent. of all the diseases of horses and about 50 per cent. of all of the internal diseases are attended by gastro-enteric pain. The mortality is about 10 per cent., divided as follows:

Displacement of colon	1.5 per cent.
Volvulus of small intestine	1.5 "
Rupture of stomach	1.5 "
Simple obstipation	1.5 "
Rupture of cecum	1.0 "
Rupture of colon	1.0 "
Embolism, tumors, enteroliths, hernias, and animal parasites	1.0 "

Forms.—From a practical standpoint so-called colics may be classified as follows:

(*a*) Acute dilatation of the stomach.

(*b*) Simple impaction of the intestines (small or large intestines).

(*c*) Impaction complicated with displacement of bowel.

(*d*) Embolic colic.

(*e*) Spasmodic colic.

(*f*) Worm colic.

Acute Dilatation of the Stomach (*Gastrectasis*).—**Definition.**—By acute dilatation of the stomach we understand a sudden gaseous distention of the organ due to an unusual fermentation of its contents. A primary and a secondary dilatation are distinguished.

Occurrence.—This condition is not uncommon in horses, forming about 10 per cent. of the cases of colic.

Primary dilatation is due to overfeeding or more commonly to irrational feeding, especially where large quantities of corn, barley, bran or chop are fed. Horses which are fed irregularly, such as cab horses, express wagon horses, etc., which often eat their feed out of a nose bag and are placed at hard work too soon thereafter are the most frequent sufferers. On

the other hand horses which are regularly fed or on pasture are only occasionally attacked. There can be no doubt that extremes in atmospheric temperature may predispose an animal to an attack. It is commonly observed, therefore, during very hot weather especially when humid, or on the other hand during very cold weather particularly when damp.

Secondary dilatation is due to stasis of the gastric contents resulting from impaction of the bowel (either simple or complicated). Secondary dilatation is more common than primary.

Diagnosis.—As a general proposition gastric dilatation may be diagnosed if a clear history of the kind of work, food and method of feeding is obtainable, and a careful examination of the patient made. In most cases the attack of gastric pain comes on just after feeding or in some cases during feeding. However, there are exceptions to this and attacks are not infrequent as long as seven or eight hours after the consumption of a meal. The patient is usually dyspneic which, depending upon the degree of the dilatation, will vary. It is usually quite marked, however, and due to the hindrance offered the diaphragm by the distended stomach. The dyspnea increases when the animal lies down. The conjunctiva in the early stages is slightly congested, in severe cases cyanotic and "muddy." Depending upon the duration and the severity of the attack the pulse varies from normal frequency and strength to weak, often imperceptible, the number going as high as 80 to 100. The temperature varies between 100.4° to 101.9° F., although where the condition is protracted it often reaches 104.5° F. Symptoms of pain are usually not very marked except in the beginning. The intestinal peristalsis in nearly every case is partially or entirely suppressed due to the associated involvement of the bowel. In mild attacks there is usually little or no sweating, but in severe cases the sweat outbreak may be profuse. A symptom of great diagnostic importance but unfortunately not always present is esophageal eructation (in 48 out of 142 cases, Behrens). Vomiting is an occasional symptom which by no means speaks for rupture of the stomach. In not over 20 per cent. of the cases of vomiting

does rupture precede or follow the act. Rupture of the stomach not infrequently is the result of dilatation and is often not attended by vomiting. A very valuable aid to diagnosis is the use of the stomach-tube, through which, when introduced, is discharged a large quantity (2 to 5 gal.) of fluid, gaseous, acid, partially digested food. Unless the gastric dilatation is complicated with intestinal disorder a rapid disappearance of the symptoms of colic follow the use of the tube. According to some authorities, displacement of the spleen is a tangible symptom of gastric dilatation. This organ may be felt through the rectum, where it has become displaced posteriorly lying in the region of the left flank. Inasmuch as such a displacement has been noted in apparently healthy and even fasting horses this symptom is not pathognomonic. While the spleen may be reached per rectum, it is sometimes difficult to feel it through the wall of the bowel overlying the hand.

Course.—In mild cases the symptoms may subside in a few hours, but very frequently a catarrh of the stomach remains behind which persists for two or three days. In some instances gastritis sets in, leading to death. Foreign-body pneumonia is an occasional complication due to aspiration after belching or vomiting. Some patients die of suffocation, but more commonly the condition leads to rupture of the stomach.

Treatment.—The only safe method of treating this disorder is to use the stomach-tube, which permits the imprisoned gas to escape bringing with it large quantities of the gastric contents. The stomach may be then washed out (lavage) by repeated injections of lukewarm water to which creolin has been added, siphoning out as much as possible after each injection. If applied early this method will yield to healing in nearly 100 per cent. in cases of primary dilatation and 50 to 75 per cent. of secondary dilatation. The use of mild laxatives such as salts, aloes, etc., are rarely indicated and are effective only in mild cases. Barium chlorid, arecalin and eserin are dangerous in that they may cause rupture of the stomach.

Simple Impaction of the Intestines.—Definition.—Simple impaction of the bowel (obstipation) consists in an accumulation of feces which obstructs the lumen of the bowel. After a time the bowel surrounding the impacted mass loses its tonicity, dilates and becomes paralyzed.

Occurrence.—Simple impactions are very frequent in horses. According to the records of the Berlin Clinics 75 per cent. of the cases of colic are due to this cause. The frequency of this disorder, however, varies with the kind of food and manner of feeding, so that these figures do not apply to all parts of the world. For instance, in Budapest the statistics show a much lower prevalency (6 to 20 per cent.).

Etiology.—The exciting causes of this form of colic are due to feeding food rich in cellulose and wood fiber, the character of this type of food requiring that large quantities be ingested to supply nutrition. Straw, chaff, corn fodder, clover or alfalfa which is not young and tender are, therefore, causes. Overfeeding any sort of food may have a like effect. Foods which contain a large amount of mineral matter or earth, sand, etc., often lead to impaction (bran, barley, swamp hay).

Horses suffering from chronic gastro-intestinal catarrh, and those with diseased teeth preventing proper mastication are commonly victims. Old and very fat horses which are not exercised sufficiently are predisposed.

Impaction may also be secondary to pathological conditions of the bowels (dilatation of the cecum, stenosis of the ileum, paralysis of the rectum, enteroliths, worm parasites and embolism).

Forms.—From the standpoint of diagnosis two forms of simple impaction are distinguished:

Impaction of the small bowel.

Impaction of the large bowel.

Impaction of the Small Bowel. — *Diagnosis.* — The principal symptoms of this form of impaction are the suddenness of the attack of colic which appears usually a few hours after feeding. The pain is usually quite marked, the patient often assuming the attitude of the male horse when urinating. The peristalsis of the left side is suppressed and

defecation ceases. Rectal examination usually gives negative results. In small horses, however, an examiner with a long arm may palpate the ileum at its union with the cecum, as a smooth, cylindrical, firm mass about the size of an arm located to the right of the spinal column and extending from above obliquely downward and backward toward the cecum. The size of the bowel and the absence of bands indicate that it is small and not large intestine. If the duodenum is impacted, it may be felt where it crosses the abdominal cavity from right to left just in front of the anterior root of the mesentery. It is attached to the roof of the cavity by a short mesentery.

The pulse, temperature and conjunctiva are usually normal in the early stages (contrary to volvulus).

Course.—Fresh cases if properly treated usually recover in three to six hours. Attacks lasting longer should be looked upon less favorably. The colic in these instances may continue for several days the patient showing intermittent pain which occurs following feeding. The pulse becomes very rapid, the temperature feverish and the conjunctiva cyanotic. Death may also result from ensuing volvulus which is fatal in eight to twelve hours, intestinal sepsis or enteritis.

Prognosis.—The prognosis in impaction of the small intestines is generally good. Not over 5 per cent. of the cases die.

Treatment.—In the early stages a subcutaneous injection of arecalin ($\frac{1}{2}$ gr.) which may be followed, if the results are not satisfactory, by a hypodermic of eserin ($\frac{1}{2}$ gr.). Usually the administration of these drugs is followed in from thirty minutes to one hour by defecation and recovery in three to six hours. If the condition has been neglected or the action of arecalin and eserin unsatisfactory, aloes (℥j) may be administered. Peristalsis may be further stimulated by infusions of water into the rectum, massage of the impacted bowel through the rectum, and moderate exercise. In prolonged attacks lasting several days the patient should be muzzled to prevent feeding.

If secondary distention of the stomach occur, treat as

in gastric dilatation. It may be necessary to use the tube repeatedly to prevent rupture.

IMPACTION OF THE LARGE BOWEL.—Usually the impaction occurs in the cecum, pelvic flexure of the colon or at the termination of the right upper colon. Occasionally the rectum is impacted (pregnant mares).

Impaction of the Cecum.—The causes of cecal impaction are practically the same as those of the small bowels. Usually when the cecum becomes impacted it is due to an organic change in the wall of the bowel which undergoes gradual dilatation with thickening. Eventually, the walls of the cecum lose their normal tonicity and paralysis develops predisposing to impactions. As a result intermittent attacks of chronic colic occur which generally lead to obstinate constipation and eventually to toxemia, enteritis, rupture and death.

Diagnosis.—An accurate diagnosis of cecal impaction can be made only by rectal examination. In the right upper flank region one can determine a swelling of about the size of a human head. The enlargement is round, surface smooth and is not sensitive. Sometimes the bands of the cecum may be felt. The consistency will vary from quite soft, doughy (retaining finger imprints) to firm or hard. The fixed position of the enlargement, its size and location in the upper right region of the flank make the diagnosis not difficult. Error would be possible only in case there was impaction with displacement of the left lower colon, the bowel extending from the left to the right side of the abdominal cavity. The impacted left lower colon, however, usually occupies the right lower region of the flank and hugs rather closely the median line. The shape of the impacted mass is furthermore elongated and, finally, the base of the cecum can be felt *in situ.*

Prognosis.—In early cases proper treatment usually produces healing. However, cecal impaction in the nature of things tends to become chronic the patient suffering from time to time with periodical attacks of pain the condition finally leading to rupture and death.

Treatment.—Arecalin (½ gr.) combined with aloes (℥j) is most effective. In prolonged attacks this treatment may be repeated. As an auxiliary the use of rectal infusions, massage (*via* rectum) and light exercise are helpful. In case the cecum bloats the trocar may be used.

IMPACTIONS OF THE COLON.—Impactions of the colon usually occur either (*a*) in the left layers including the pelvic flexure or (*b*) in the terminal portion of the right upper colon.

(*a*) Impaction of the left layer of the colon. When the left lower layer is impacted, on rectal examination may be felt a cylindrical mass usually just in front of the pelvic inlet or more rarely protruding into the pelvic cavity. The left lower layer is distinguished by its bands and pockets, the left upper layer by the absence of bands, its caliber, and direction in practically a straight line forward. The greater diameter and straight direction of the left upper colon differentiate it from the smaller, tortuous loops of small bowel.

(*b*) Impaction of the right upper colon. The impacted mass is imprisoned in the "stomach-like" dilatation of this bowel, beginning at the funnel-shaped termination and extending forward and including the widest diameter of the bowel. Except in small horses the results of rectal examinations are almost negative. When the impaction may be felt it is distinguished by its location anterior to the cecum, slightly to the right of the median line. The mass is somewhat round, firm, often covered by the root of the mesentery, and tends to move synchronous with respirations.

In case the result of the rectal examination is negative, it would be impossible to distinguish between impaction of the right upper colon and that of the small bowel. In the last stages, if quantities of gas accumulate in the left layers of the colon the condition may be assumed; if on the other hand the gaseous distention is confined to the small bowels an impaction of these is probable. The general condition of the patient in impaction of the colon remains for quite a time good. The pulse and conjunctiva usually are about normal. Obviously when enteritis, septic intoxication or rupture occur the general condition becomes bad.

Impaction Complicated with Abnormal Displacement.—Forms.—The following abnormal displacements of the bowel have been noted: Torsion of the large bowels (colon and cecum), volvulus of the small bowels, intussusception of the small bowels, incarceration of the bowel (inguinal canal, epiploic foramen, rents in the diaphragm, omentum, mesentery, etc.), and strangulation of the intestines from tumors.

All of these pathological displacements lead to a sudden occlusion of the bowel and fatal colic. They constitute about 5 per cent. of all colic cases. Of great practical importance are:

(*a*) Displacement of large bowels.

(*b*) Displacement of small bowels.

Displacement of Large Bowels.—Most commonly this consists in a rotation (torsion) of the left colons around their long axes. On account of their free position in the abdominal cavity, contrary to the colons on the right side, a certain predisposition to abnormal displacement is present.

Etiology.—The exciting causes are usually primary impactions behind the point of torsion. Therefore, an impaction of the upper may lead to torsion of the lower colons, or an impaction of the lower to torsion of the upper colons. Impaction may be determined in at least 80 per cent. of all cases of displacement of the large bowels. Impaction induces in the bowel which is in front of it a violent antiperistaltic movement whereby secondarily a torsion follows. In other instances paralysis of the bowels due to embolism is the cause. Very rarely rolling in horses suffering from colic may lead to displacement.

Diagnosis.—A correct diagnosis can be arrived at only through rectal examination. Very important in this regard is the course of the bands of the left lower colon. In place of their normal straight course the bands will be found bent spirally to the right or to the left. The twist is always opposite to the direction of the bands. For instance, the common twist of the left layers of the colon is to the right; the bands are twisted spirally toward the left in this condition. Besides the result obtained from rectal examination it will be noted that the general condition of the patient is

rapidly becoming serious which is usually indicated in one to two hours by the change in the pulse, peristalsis, temperature and conjunctiva. There is, further, profuse sweating, great prostration, collapse, etc.

Prognosis.—If the torsion is not soon removed death will result in six to twelve hours.

Displacement of Small Bowels. *Volvulus.—Etiology.*—Usually a primary impaction of a part of the bowel behind the volvulus is the cause of change in position. The impaction may be either in the small or large bowel. Volvulus of the jejunum is commonly produced by impaction of the ileum (stenosis). As in the case of the large bowels a violent antiperistaltic movement of the bowel lying in front of the impaction favors torsion.

Diagnosis.—Contrary to torsion of the large bowel volvulus can rarely be determined per rectum. A diagnosis is usually only possible by way of exclusion and is as such a probable one. If on rectal exploration no change can be found in the layers of the large intestine and the condition of the patient is rapidly growing bad, the probability of a volvulus is great. In some cases the bloating of the small intestines, which may be determined per rectum, points to volvulus.

Prognosis.—The prognosis is bad. Death usually results in eight to twelve hours.

Treatment.—Treatment as a rule is without avail. Attempts to relieve the animal by a laparotomy have not proved to be feasible.

Embolic Colic.—Definition.—By the term embolic or thrombo-embolic colic we understand intestinal pain from a disorder of the intestines due to the presence of a worm aneurysm in the anterior mesenteric artery. The cause of the worm aneurysm is the larva of the Strongylus armatus, the armed palisade worm. The adult worm lives in the large bowel and the eggs are discharged with the feces. In the soil or stable floor the larvæ are hatched and are taken up by healthy horses (colts) with the bedding, or grass of the pasture, and with the drinking water. From the bowel the larvæ pass into the veins of the mucous membrane of the

intestines, reach the right heart and, after passing through the lungs, the arterial circulation. According to some authorities they wander direct from the intestines between the leaves of the mesentery to the anterior mesenteric trunk. They are found principally in this trunk or its principal branch, the ileocecocolic artery. By irritating the inner wall of the artery they produce a chronic endarteritis. The results of the inflammation of the artery are: Thrombosis, dilatation and calcification of the arterial wall (aneurysm).

Notwithstanding that nearly all horses (about 90 per cent.) suffer from this aneurysm, embolic colic occurs in only about 5 per cent.

Pathogeneses.—The worm aneurysm of the anterior mesenteric artery produces disorder of the bowel in three different ways: (*a*) Detached fragments (embolic) of the thrombus may reach the peripheral intestinal arteries. (*b*) The thrombus itself may become prolongated into branches of the artery. (*c*) The thrombus may in rare instances completely obstruct the lumen of the mesenteric trunk. In all three of these cases, depending upon whether or not the collateral circulation suffices, there results anemia, hemorrhagic infarction, and ultimately a necrosis of the mucosa of the bowel. On necropsy, therefore, we find principally the symptoms of a hemorrhagic inflammation of the bowels with necrosis and at the same time occlusion or thrombosis of the afferent and peripheral arterial branches.

Symptoms.—The attack of colic usually begins suddenly, mostly during work. In mild cases the attack resembles somewhat spasmodic colic in that the pain is intermittent. On rectal examination everything seems intact provided there is no displacement secondary to the thrombosis; or we may be able to feel fremitus over the region of the anterior mesenteric artery. In many instances the thrombus can be palpated per rectum. In the severe type bloating is an ordinary symptom. This form of colic tends to hang on with periods of remission for one or two weeks although it usually lasts but a few hours. It is frequently attended by fever and sometimes the stools are blood-stained. It is very apt to lead to enteritis, rupture of the stomach or bowel,

septic intoxication or peritonitis. Embolic colic is a frequent cause of impaction especially impaction complicated with displacement.

Diagnosis.—In the living horse the diagnosis of embolic colic is always a probable one. It may be suspected when without apparent cause repeated attacks of colic occur and more especially if the feces contain blood.

Treatment.—The treatment is the same as in simple impaction. Atoxyl (ʒiij) of a 3 per cent. solution given intravenously are said to eradicate the strongylus.

Spasmodic Colic.—Definition.—By spasmodic colic we understand a rather severe attack of abdominal pain due to spasmodic contractions of the bowel probably superinduced by intestinal catarrh. It is characterized clinically, in contradistinction to impaction colic, by diarrhea, rapid course, intermittent pain and favorable termination.

Treatment.—This form of colic should be treated with drugs which allay pain such as morphin (grs. iij-vj), chloral hydrate (ʒj). Arecalin, and especially eserin and barium chlorid, are contra-indicated. Warm applications to the belly are valuable.

Worm Colic.—Intestinal Parasites.—The intestinal parasites of the horse (spool worms, tapeworms, palisade worms) notwithstanding their frequency rarely produce colic. However, if present in large numbers they may (1) obstruct the bowel producing simple impaction, (2) mechanically irritate the mucous membrane inducing enteritis, or (3) by emigrating into the abdominal cavity lead to peritonitis. In this connection the following parasites are important.

Ascaris megalocephala, the spool worm of the horse, produces obstruction, perforation of the bowel at the attachment of the mesentery, worm cysts and peritonitis.

Tænia plicata, T. perfoliata and T. mamillana, the tapeworms of the horse, produce obstruction and perforation.

Strongylus armatus and Str. tetracanthus, the adult palisade worms of the horse, produce hemorrhagic enteritis.

Oxyuris curvula, the pin worm of the horse, produces proctitis.

The larvæ of Gastrophilus equi and G. pecorum rarely pro-

duce colic. In exceptional cases in colts they may induce traumatic gastritis or, by perforating the stomach wall, peritonitis.

Treatment.—Through the use of purges alone intestinal worms cannot always be removed. The most valuable agent for their removal in horses is tartar emetic (ʒij to ʒss) daily, given in drinking water two or three times. Besides arecanut (ʒiij), arsenic (grs. xv–xxx in form of boli), santonin (ʒiiss) and turpentine oil (ʒiss–ʒiij) may be used. For gastrus larvæ in the stomach bisulphid of carbon (ʒiiss) given at night in capsules, four capsules given one hour apart. Follow with linseed oil (Oj).

Flatulent Colic.—In some cases of simple impaction or in impaction complicated with displacement an abnormal fermentation of the bowel contents results. The gas (CO_2, CH_4, H, air) formed leads to a distention of the abdominal wall. It is more rarely due to "wind sucking."

Symptoms.—The symptoms are those of simple or complicated impaction with great distention of the abdominal wall, and from compression of the diaphragm, severe dyspnea.

Treatment.—The treatment consists in using the trocar either through the side (cecum) or more rarely through the rectum. If the stomach is bloated the stomach-tube should be used.

GASTRO-ENTERITIS.

Inflammation of the Stomach and Bowels.

Definition.—Gastro-enteritis is a symptom rather than a disease. It is a collective term covering all conditions which directly or indirectly induce serious inflammations of the walls of the gastro-intestinal tract. From catarrh pathologically it varies only in degree; clinically it differs in the intensity of the symptoms and in the usual fatal termination.

Occurrence.—Gastro-enteritis is common in all animals, occurring as either a primary or a secondary disorder.

Forms.—From a pathological standpoint, croupous, diphtheritic, hemorrhagic, purulent, and phlegmonous forms are distinguished; from a topographic, duodenitis, ileitis, typhlitis, colitis, and proctitis, depending upon the part of the bowel

tract involved. From a clinical standpoint the following forms occur:

(*a*) Simple, (*b*) croupous, (*c*) mycotic, (*d*) toxic.

Simple Gastro-enteritis.—Etiology.—The causes of simple gastro-enteritis are in a general way the same as those of gastro-intestinal catarrh, but acting more intensively (see these). Food which is infected with bacteria or fungi or damaged by improper harvesting; frost, or containing poisonous, irritant weeds; or good food injudiciously fed may, therefore, be predisposing factors. Overexertion, especially in very hot weather (horses at hard work, animals shipped long distances by rail, etc.), predisposes by greatly lowering resistance. Copious draughts of cold water, the body being hot, act in much the same way.

The exciting causes of gastro-enteritis are evidently bacterial. Probably no one species, however, is a constant cause. For some as yet unknown reason microörganisms which are regularly found in the digestive tract assume pathogenic activity once the resistance of the patient is reduced by dietary errors, overexertion, extreme heat, etc. It may thus be caused by some of the colon bacillus group. The Bacillus enteritidis, the necrosis bacillus and pus bacteria seem to have been active in some cases.

Animal parasites (Strongylus tetracanthus and Spiroptera megastoma in horses, and Spiroptera strongylina in swine) are justly accused.

In the ox a traumatic gastritis results from foreign bodies penetrating the walls of the stomach (see Traumatic Indigestion of Ox).

Certain irritant drugs (aloes, cantharides) can cause toxic gastro-enteritis.

Symptoms.—The symptoms of simple gastro-enteritis are much more intensive than those of catarrh. While at times they develop gradually, (follow catarrh) as a rule the onset is sudden (six to ten hours). The principal symptom is abdominal pain, which is continuous and generally severe. Appetite for food is entirely absent, although water may be taken at frequent intervals. In the early stages the bowels are constipated and peristalsis suppressed. Later diarrhea

sets in; profuse quantities of miscolored, liquid feces are voided. Depending upon the form of inflammation, the feces may be admixed with blood (hemorrhagic enteritis), pus (abscess, purulent enteritis), fibrinous masses (fibrinous enteritis), necrotic tissue (diphtheritic enteritis), and mucus (proctitis). The pulse reaches 70 to 90 (in horse) and becomes weak, small and hard in character. The mucous membranes (eyelid) grow cyanotic (toxemia). The temperature is elevated (104° to 106° F.) and the type of fever intermittent in prolonged cases. Toward the end the temperature is subnormal.

The general condition of the patient is that of great prostration. The body may be wet with sweat, the countenance relaxed, eyes staring, legs and ears cold, and gait staggering. The pulse becomes very weak, finally imperceptible, the mucous membranes grow cyanotic, and toward the end the patients are down in a soporous condition and may show convulsive movements of the legs.

Diagnosis.—Simple gastro-enteritis is characterized by its sudden onset, rapid, fatal course, and the gravity of its symptoms of abdominal pain (colic), tender, "tucked-up" abdomen, obstinate diarrhea, weak, wiry, frequent pulse, fever, and in the last stages the marked mental depression (sometimes excitement) and exhaustion. It might be confused with certain poisonings (toxic gastro-enteritis). The history of the case, number of animals affected, and the special symptoms which accompany each form of poisoning generally suffice for differentiation. In some cases, however, only the necropsy and chemical analysis of the ingesta will finally determine.

Gastro-enteritis may be secondary to many disorders of the stomach and bowels, such as colic, helminthiasis (Strongylus armatus inducing thrombi and emboli), latent bowel ulcers following an attack of influenza or strangles, and more rarely may be due to enteroliths. In the above cited instances usually the grave symptoms of severe inflammation are preceded by milder symptoms of digestive disorder.

Course.—The course is usually rapid, death occurring in one to three days. Some patients die in a few hours after the

symptoms appear. In isolated cases the disease may take a subacute course, and end in recovery in one to six weeks. In the ox an ordinary gastro-intestinal catarrh may after one to two weeks suddenly assume the form of a gastro-enteritis terminating fatally in forty-eight hours. Swine offer more resistance than do horses or cattle.

Prognosis.—Unfavorable to bad. Fully 90 per cent. of the patients attacked die.

Treatment.—No food should be given during the attack. In subacute cases gruels (flaxseed tea) may be allowed. The patient should be kept dry and warm by frequent skin rubs and warm, dry blankets. Careful nursing is essential.

The medicinal treatment is symptomatic and of secondary importance. Mild laxatives may be used in the early stages (calomel in horses ℥j; and hogs grs. x; Glauber salts in cattle, castor oil). Strong purges should be avoided (arecalin eserin, aloes). Slimy, mucilaginous agents (linseed tea, gum arabic) are indicated. They are usually used as vehicles for opium (powdered opium ℥ij, tinct. opii ℥ss to j) or belladonna (fluidextract ℥j). Opium (℥ij) combined with calomel (℥ij), and powdered althea (℥iij) in the form of an electuary is useful. Morphin (grs. iij to v) may be employed subcutaneously to lessen pain.

Gastro-intestinal disinfectants, creolin (℥j), therapogen (℥ij to iv), sodium salicylate (℥ij to iij) or "sulphocarbolates," *i. e.*, sodii phenolsulphonas (℥ij to ℥j), zinci phenolsulphonas (℥j to iv) are often used, but are of little value except when the bowel is still intact. They may be even harmful in enteritis.

In weakness and collapse, oil of camphor (℥j) subcutaneously, alcohol (℥ij), ether (℥ij), caffein (℥ij) are indicated.

Subcutaneous, intravenous, or rectal injections of sterile physiological salt solution to which 2 to 3 per cent. of grape sugar has been added are reliable. The dose is H. & C. Oviij to x. Calves and colts Oiv, swine Oij.

Croupous Enteritis. *Membranous Enteritis.*—**Definition.**—A subacute enteritis, usually of cattle, characterized pathologically by the formation of a fibrinous pseudomembrane over the mucosa of the intestines.

Occurrence.—The disorder is not common, although isolated cases are noted in cattle, especially young, fat bulls and pregnant cows. Croupous enteritis has also been observed in horses and sheep. Cattle which are turned out to grass very early in the spring are most often attacked.

Etiology.—The exciting cause, which is probably bacterial, is not known. Predisposing causes are refrigeration (cold, damp weather), irritant foods (mustard grass), and the ingestion of such drugs as strong camphor and cantharides.

The seat of the lesions is generally in the small bowels, which on necropsy are lined by a grayish-yellow, rather friable, and easily removable mass under which the mucosa is catarrhally inflamed.

Symptoms.—The early symptoms are those of gastro-intestinal catarrh (lost or impaired appetite, suppressed rumination, constipation, etc.). Some patients show colic attacks (switching of tail, kicking bind legs against abdomen, or more rarely rolling). The symptoms of colic temporarily recede in twelve to fourteen hours. The patients show marked constipation, which usually lasts one to two weeks, at the end of which time the symptoms of abdominal pain return and diarrhea sets in. The liquid feces are brown in color, quite fetid, and eventually admixed with yellowish-gray croupous masses in the form of shreds, flakes, or sometimes cylinders several feet in length. These tubular masses appear to the novice as portions of the intestines from which they are differentiated by their homogeneous structure, absence of mesentery, and bloodvessels. The cylinders often contain feces.

In some mild cases the passage of croupous masses is not preceded by symptoms of ill health beyond those of a mild indigestion. In other cases the prodromal symptoms are those of a severe gastro-enteritis, with great prostration, lost appetite, high fever, bowel hemorrhage, etc.

Diagnosis.—A diagnosis is only possible when croupous membranes are found mixed with the feces. Shreds of the pseudomembranes may be mistaken for worms, prolapsed bowel, and portions of tendons accidentally swallowed.

Course.—The course is usually one to two weeks, ending in recovery. Death occurs only in those cases in which symptoms of severe gastro-enteritis appear or more rarely may be due to a complete obstruction of the bowel through the accumulation of fibrinous masses.

Prognosis.—Usually good. Most cases recover.

Treatment.—A large dose of salts (lbs. j to ij) is very useful. It may be followed by the administration of oils (raw linseed or castor oil Oj to ij). The after-treatment is the same as recommended in gastro-intestinal catarrh (diet, hygiene, stimulants, demulcents).

Mycotic Gastro-enteritis. *Silage Poisoning. Forage Poisoning. Mold Poisoning. Cryptogamic Poisoning. Falsely called "Cerebrospinal Meningitis." Leukoencephalitis.*—**Definition.**—A form of gastro-enteritis leading to intoxication of the central nervous system, affecting herbivorous animals and due to the ingestion of food infested with certain bacteria or fungi. The disorder is probably not a clinical entity.

Occurrence.—Forage poisoning is very prevalent in the United States, appearing particularly among horses which have eaten corn silage, shredded fodder, corn stalks or corn cobs. The disorder may, however, occur in horses on pasture and which have not been fed corn. During hot, showery seasons the rank growth of grass, which mats together, forms an ideal medium for the development of various molds which are pathogenic. In all probability water drunk from stagnant pools or shallow wells may also be a factor. Lands in low, flat sections which are periodically flooded by streams flowing through them are especially dangerous in this regard. The disease is, therefore, quite prevalent along the river valleys of the United States. Cattle and sheep are also affected, but less frequently than are horses. Cattle are sometimes infected while on orchard pastures, the ground strewn with "wind-fall" apples, which they eat. (Acidosis?)

Etiology.—Molds (mucor, aspergillus, penicillium), "blights" or smuts (Tilletia caries, ustilago), rusts (puccinia, uromyces), and yeasts (Polydesmus exitiosus), which infest forage, grain, and water at times, are pathogenic, and through their toxins produce in the animal body symptoms which fall,

generally speaking, under two groups, *viz.*, gastro-intestinal and nervous. In some outbreaks the nervous symptoms predominate, in others the gastro-intestinal, depending probably on the kind of fungus taken into the body, the quantity of toxins produced, and the resistance of the individual.

Symptoms.—The symptoms of forage poisoning, as noted, may be grouped under two heads: (1) Nervous, and (2) gastro-intestinal. Either may dominate or both may be combined in individual outbreaks.

Nervous Group.—The most conspicuous symptoms are dysphagia from paralysis of the pharynx (inability to swallow, slobbering), paralysis of tongue, roaring, incoördination of body movements, staggering, shambling gait, weakness of hind parts; strikes forefeet in stepping over door sill, paralysis of tail, spasms of certain groups of muscles (twitching of face, lips, neck, shoulder); mental excitement, due to active cerebral congestion, causing rabiform symptoms (tendency to climb over any obstacles, biting and striking at attendants); mental depression (stupor, pushing head against wall); forced movements (travelling in a circle to right or left, individual patients always in one direction); amaurotic blindness (running against objects), opisthotonos (head drawn backwardly), and finally profuse diaphoresis (heavy sweats along neck, shoulders, sometimes on one side only, may lead to loss of hair from maceration).

Gastro-intestinal Symptoms.—Colic (pawing, restlessness), constipation, often obstinate at first; or diarrhea, the feces liquid, sometimes blood-stained and fetid. There is occasionally slight bloating. The peristalsis is suppressed in constipation, lively in diarrhea. Tenesmus is occasionally observed.

General.—The conjunctiva shows petechia and icterus, the temperature in the early stages is up to 105° F., but soon drops to normal or subnormal, where it continues until death. The pulse is usually normal until the last stages, when it becomes rapid (occasionally slower), weak, and irregular. Dyspnea is generally present, but varies greatly in degree in different cases. Occasionally the respirations are subnormal. Polyuria is noted in some outbreaks, although retention of

urine (paralysis of bladder) is more commonly observed. In the ox hematuria is often a symptom. Stomatitis and eczemas of skin (especially of head) have been noted in some outbreaks.

Diagnosis.—Usually the diagnosis is not difficult. The number of animals affected, the history of food eaten (silage, shredded fodder), the nervous symptoms, rapid course, and the lack of apparent contagiousness are important factors. The disease might be confused with rabies, hemorrhagic septicemia, anthrax, and poisonings with drugs. In most outbreaks a combination of nervous and gastro-intestinal symptoms is suggestive. Differentiation in sporadic cases may be impossible without the aid of a necropsy and bacteriological examination (rabies, anthrax, hemorrhagic septicemia).

Course.—The course is usually rapid. Individual patients die apoplectic, others in a few hours (seven to twelve), while many live one to two weeks and succumb. Patients which recover usually do so gradually, and such sequels as lumbar weakness (wobbling gait), hemiplegia (paralysis with atrophy of the muscles of one side of the body), blindness, epileptiform seizures, etc., follow and persist for weeks or months. In occasional cases relapses occur during convalescence.

Prognosis.—The prognosis is doubtful to bad. The mortality varies from 25 to 90 per cent.

Treatment.—Feeding infested forage or water should be immediately stopped. Pastured animals should be placed in stables and fed only uncontaminated foods. While the separation of the healthy from the sick does not seem necessary, as an extra precaution it is recommended at least until our knowledge of the etiology is more definite.

Medicinal.—The patients should be purged as soon as possible (aloin ℥j to ij; salts lbs. j to ij; arecalin grs. j to ij, combined with strychnin gr. ¼). Large doses are usually required to move the bowels. Calomel (℥j to ij) is useful in horses. Owing to pharyngeal paralysis drenching is contraindicated. Medicine should be given *per orem* in form of boli or electuaries.

The purgative may be followed by a disinfectant, such as

Pearson's creolin (℥j to ij), therapogen (℥ij to iv), etc. In weakness and collapse give alcohol (℥ij), ether (℥ij), caffein (℥ij), oil of camphor (℥j) subcutaneously. For remainder of treatment see Simple Gastro-enteritis and Pharyngitis.

When patient begins to improve and eat it should be placed on a light, laxative diet. If able to stand in them, support with slings.

Toxic Gastro-enteritis.—Toxic gastro-enteritis is an inflammation of the stomach and bowels due to the ingestion of poisons, such as arsenic, mercury or any irritant drug or chemical. The symptoms and treatment of this form of gastro-enteritis are best given in books on toxicology which deal with poisons, their effects and antidotes.

DISEASES OF THE STOMACH OF THE OX.

General Remarks.—The first three compartments of the stomach are in a sense dilatations of the esophagus and serve to store up and further prepare food for its reception in the abomasum where true gastric digestion takes place. The rumen is a receptacle for food which the animal has rather hastily prehended, only partially masticated, mixed somewhat with saliva, and swallowed. The function of the reticulum is not clearly understood. Normally it contains little solid food and is commonly a receptacle for foreign bodies. The omasum is a "chewing stomach" where solid matter is still further crushed and pulverized before it reaches the abomasum. Rumination consists in the contents of the paunch becoming regurgitated through the esophagus into the mouth where they are thoroughly masticated, mixed with saliva and reswallowed. Obviously all solid matter is not necessarily ruminated; a portion of it, at least, once it reaches the rumen, does not return to the mouth. Ruminating animals fed liquid foods do not chew the cud. Cud chewing is therefore only employed when necessary, *i. e.*, when the food is not adequately ground, pulverized and thoroughly mixed with saliva in order to be more effectively acted upon by gastric and intestinal digestion.

The first three compartments of the stomach are lined with a thick coating of epithelium and are not supplied with digestive glands. They, therefore, present a remarkable resistance to inflammation. Most of the diseases of these compartments are functional. When for any reason the musculature of the rumen fails to function adequately, the constant mixing of the gaseous, liquid and solid contents is interfered with. The result is that the liquid, having the greater specific gravity, tends to collect at the bottom of the organ, the solids to occupy the center, and the gas the top. For this reason where gastric peristalsis has been for a time arrested the solid paunch contents become dry, firm and hard, and the hollow of the left flank distended. Obviously the degree of intensity with which these things may occur will depend upon the character of the food.

In the present state of knowledge any attempt to classify dogmatically the diseases of the stomach of the ox is impractical. If any one compartment ceased to function it will affect all of the other compartments, so that in practice it is not always possible to make the same fine distinctions made in print.

IMPACTION OF THE RUMEN.

Overloaded Paunch. Mawbound.

Definition.—An inordinate accumulation of food in the paunch without undue gas formation.

Occurrence.—Impaction of the rumen is usually the result of dietary errors and therefore a common disease among cattle, especially stable-fed animals subject to artificial feeding conditions. It is unusual among pastured cattle except under extraordinary circumstances, such as follow extreme drought, etc.

Etiology.—(*a*) Overfeeding with good food. It sometimes happens that an individual animal is fed beyond its digestive capacity or, what is more common, the animal gains access to the corn field or grain crib and overeats. (*b*) Bad food. Improperly harvested, over-ripe hay, clover

hay, alfalfa, dried out, brittle corn fodder, wheat straw, rye straw, etc. Food which is bulky containing an unproportionate amount of woody material, as swamp grasses, tree tops. Food which is spoiled (moldy meal or bran, bad ensilage). Residuary foods, such as brewer's grains, distillery slop, sugar beet pulp, kitchen offal. (*c*) Lack of water. During drought or through negligence the cattle are not given sufficient pure drinking water.

Predisposing causes would include anything which prevents the free movement of the rumen interfering with its peristalsis. Lack of exercise, ingestion of large quantities of very cold water, sudden change from green food to dry; more rarely general weakness, peritoneal adhesions, diseases of the wall of the rumen (actinomycosis, tumors or catarrh).

Symptoms.—The symptoms usually noted by the owner are the patient does not eat well, fails to chew its cud, stands back from the manger with its hind feet in the gutter, or, if with the herd in the field, is off by itself. The patient usually stands listless, with feet drawn up under the body or on the other hand spread apart, back arched, facial expression anxious. In some instances colicky pains appear causing the patient to switch its tail, kick its hind feet against the abdomen, turn its head toward the sides, show restlessness, and alternately lie down and get up. As a rule, cattle suffering from impaction of the rumen do not lie down much and if they do usually on the right side. The abdomen appears more rotund than usual and there is a slight filling of the hollow of the left flank. The paunch movements are either absent or very feeble. On palpation over the flank or through the rectum the contents of the paunch feel doughy to hard and may receive finger imprints much as unset cement. The temperature and pulse are normal at least in the earlier stages. The bowels are usually constipated although in some cases, depending upon the character of the food or an existing bowel catarrh, diarrhea may be present. Milk secretion falls off. In individual cases the patient will show weakness of the hind parts, staggering gait, may be unable to rise and show symptoms resembling to a marked degree those of parturient paresis.

Course.—The course of impaction of the rumen is usually from three to ten days. It is contingent upon the character of the paunch contents. If they be very undigestible the condition may last for one or two weeks leading to death from gastro-enteritis (fever; rapid, weak pulse; great mental depression). On the other hand death may ensue from tympany within twenty-four hours. The symptoms of improvement are the return of rumination, paunch movements and copious bowel evacuations.

Diagnosis.—The diagnosis depends upon the history, the enlargement of the abdomen, especially on the left side, and the doughy or hard consistency of the paunch contents. Fever and accelerated pulse are usually absent.

Prognosis.—The prognosis is usually good. When the character of the contents of the rumen is such as to be highly undigestible, causing the symptoms to continue for a week or more, the prognosis is bad. Inflammation of the stomach, or peritonitis can result, leading to death.

Treatment.—The treatment is directed to promoting the movements of the paunch and thus stimulating rumination, arresting the fermentation of the stagnant food masses, and relieving any resulting impaction of the omasum or constipation of the bowels.

Hygienic.—The patient should be made to fast from one to two days. Liquids, especially water, may be allowed liberally. Kneading the walls of the abdomen over the rumen five to ten minutes every three hours or three times daily is helpful. If food is given at all it should be easily digestible and laxative (fresh grass, bran gruels, fine hay, root crops).

Medicinal.—To stimulate paunch movements and to relieve constipation laxative drugs are indicated. The rule should be to employ the milder drugs of this sort first. At any rate drastic purges should be avoided. As physics, oil and salts do little good in obstinate cases. In mild attacks Glauber salts (℥xxiv), raw linseed oil (Oiss) are effective. More active is castor oil (℥xxiv) in equal volume of warm water. Ether (℥ij) may be added to the mixture. Ammonium carbonate and nux vomica are much employed

in Great Britain (℞—Pulv. ammon. carb., ℥iv; pulv. nucis vom., ℥iss; pulv. gentian, ℥ij, M.D.S.; one powder every four hours in Oj of cold water). The tartrate of eseridrin is recommended (℞—Eseridrin,[1] grs. iij; acid. tartar, grs. iss; aqu. dest., ℥vj, M.D.S. subcutaneously). The sulphate of veratrin (1 to 50 in water—dose ℥ij *per orem*), or barium chlorid (℥ij *per orem*). Common salt has proved useful in obstinate cases, given in moderate and repeated doses and alternated with the ammonium carbonate and the nux vomica. The first dose of salt (℥vj) is made into an electuary with ginger and molasses and smeared on the back of the tongue. This is said to induce thirst and the animal to take the further doses dissolved in the drinking water. It should be pushed until the bowel movements are satisfactory. Barium chlorid is recommended. It is given *per orem* in doses of ℥ij to ℥iv in a pint of salt water three times daily until the bowels become loose.

Impaction of the paunch may be relieved by direct infusions into the organ through a trocar which has been inserted in the hollow of the flank. The water should be tepid and may contain creolin. Several gallons may be allowed to flow in at a time and the treatment repeated daily. By placing a rail, held at each end by a person, under the abdominal wall, and working it up and down, the contents of the rumen may be readily mixed with the water infusion. In case this does not afford relief, rumenotomy should be performed.

To arrest gastric fermentation and act as a tonic hydrochloric acid may be given (℥v diluted in water 1 to 250) to which pepsin (℥iiss) may be added. Creolin (℥j) may be used to arrest fermentation.

The appetite may be stimulated, once the patient begins to eat, by bitter, aromatic herbs (℞—Calamus, gentian, peppermint, caraway āā ℥j M.D.S.; tablespoonful three times daily).

The animal should be brought to full feed gradually. Usually one should wait until rumination reappears and

[1] Very expensive! Arecalin is a good substitute.

begin with one-fourth to one-half rations. The drinking water should not be too cold.

Prophylaxis.—To prevent impaction sudden changes of food, irregular feeding and the feeding of bulky roughage in large quantities should be avoided. Cattle should be allowed plenty of pure drinking water. When intensive feeding is practised as during "milk tests," at the first sign of trouble three or four feeds should be withheld for each one refused, at the same time allowing plenty of water and proper medicinal correctives.

BLOATING IN THE OX.

Tympanites.

Definition.—A rapid distention of the abdomen of cattle due to gas formed from the fermenting contents of the rumen and reticulum.

Occurrence.—A very common disorder especially among cattle on pasture during hot, damp weather. Clover and alfalfa pastures are most dangerous in this regard.

Two clinical forms of bloating are recognized:

1. Acute tympany.
2. Chronic tympany.

Acute Tympany—Etiology.—(*a*) Pasturing or feeding green grasses especially legumes, such as clover, red clover, alfalfa, vetches, peas or such foods as buckwheat, swamp-grasses, etc. Such grasses are most dangerous just before they bloom, particularly when wet with rain or dew. Young grass growing in stubble fields is a common cause. (*b*) Feeding foods which readily ferment, such as potatoes, beets (residue of sugar beets), malt, withered, heated grass, etc. (*c*) Certain toxic plants (spotted hemlock, water hemlock, colchicum, tobacco). (*d*) Occlusion of the esophagus (choke) will induce bloating if the stomach contents are of a kind which easily ferment.

Symptoms.—There is a rapid distention of the abdomen especially of the left side. The abdomen assumes the shape of an apple and is of the consistency of a partially inflated

pneumatic tire. On percussion a hyperresonant tone is emitted. On auscultation no peristalsis is audible. The patient is usually quite dyspneic (mechanical compression of lungs, CO_2 intoxication) breathing with open mouth and tongue protruding. The mucous membranes become cyanotic, there is restlessness, loss of appetite, suppressed rumination and eructation of gas through the esophagus. Regurgitation of food masses occurs occasionally.

Diagnosis.—Usually easy. The rapid distention of the abdomen, characteristic indications of gas on percussion, dyspnea and anxiety suffice for a diagnosis. Chronic bloat is much slower in development and does not lead to serious distention. The bloating which attends choke has a different history, belching is absent and by using the probang the obstruction is encountered.

Course.—The course is rapid in some cases the accumulation of gas, which occupies only an hour or so, leads to death from asphyxia (CO_2 intoxication) in a few hours. Other cases develop slower, the excess gas being belched out from time to time, eventually leading to spontaneous recovery.

Prognosis.—Acute bloating is always serious, especially in sheep. If, however, treatment is prompt and properly administered recovery soon follows.

Treatment.—Various methods of relieving bloating are recommended. Generally speaking they may be placed in one of two groups: *1.* Palliative; *2.* radical.

1. Among palliative measures are: (*a*) Kneading the abdomen. The abdomen is gently but firmly massaged by the knee of the operator, the foreparts being elevated by standing the patient on a steep incline. Sheep may be made to stand on their hind legs. Driving the patient up a steep hill yields good results in milder cases. (*b*) Cold water irrigation to the flanks. Bloated sheep may be driven through a stream of water. (*c*) Inducing belching by irritating the throat with a blunt instrument inserted through the mouth, the tongue being drawn forward. Less efficient is the use of a bit made of twisted straw and smeared with tar. (*d*) Passing a hollow probang is rarely

of value, as it soon becomes blocked with food masses. In severe dyspnea it is contra-indicated. That the above cited methods may be combined is obvious. (*e*) So-called "absorbent" drugs are sometimes employed (burnt magnesia, 10 per cent.; lime-water, spirits of ammonia, 2 per cent., and soap 2 per cent.). Turpentine in oil (℥ij to Oj of oil), or formalin (℥ss to water Oij), is commonly employed. Veratrin, creolin and alcohol are sometimes given. The use of drugs in the treatment of acute bloating plays a very subordinate part. The effect is largely due to the belching which the act of drenching induces.

2. Radical measures: When the life of the patient is threatened by severe dyspnea or the application of the palliative measures are not advisable or have proved insufficient, puncturing the rumen with a trocar is often life saving. The instrument, which should be sterile, is plunged into the center of the triangle forming the hollow of the left flank, or, in case the landmarks are obliterated by the bloating, where the distention is greatest. If time is available, shaving and disinfecting the skin at the point of operation are recommended. In withdrawing the cannula the trocar should be first inserted and care taken that the skin is not pulled loose from the underlying connective tissue as this permits air to enter and may lead to infection. In case the operation must be repeated it is advisable to make a fresh wound rather than use the old puncture. In thick-skinned animals an incision through the skin only may be made with a sharp bistoury which facilitates the insertion of the trocar. After withdrawing the trocar the wound may be dressed with tar or any antiseptic. After the bloating is relieved the patient may be given a physic (Glauber salts, 1 lb., linseed oil Oj), and placed on restricted diet.

Prophylaxis.—It is recommendable to feed cattle about to be placed on clover or alfalfa pastures a quantity of dry hay before being turned out. Cattle grazing in fields covered with rich pasture or sheep on stubble fields should be carefully watched by attendants. Farmers should keep trocars handy, as in acute primary bloat a veterinarian cannot always be called early enough to prevent death.

Chronic Tympany.—Definition.—A tendency to bloat noted in some cattle due to conditions affecting the esophagus, stomach or bowels, which interfere with belching. It is therefore secondary.

Etiology.—The causes are: (*a*) Esophageal. Compression of the esophagus from tubercular mediastinal lymph glands; stenosis of the esophagus. (*b*) Gastric. Atony of the forestomachs. (*c*) Adhesions, the result of traumatic gastritis. (*d*) Hair balls in cattle and wool balls in sheep (infrequent). (*e*) Intestinal. Stenosis or impaction of the intestines (rare).

Symptoms.—The symptoms consist in a gradually developing usually moderate distention of the left flank. The condition is usually an intermittent one and an attack follows the ingestion of food which easily ferments. In a few individuals the distention may be permanent. There is usually not much disturbance of the general condition. The animal may eat, ruminate and seem in normal health except for the distention of the abdomen. In some cases the patient may show symptoms of indigestion.

Diagnosis.—The diagnosis of chronic tympany is usually not difficult, although to determine the exact cause of it during the life of the patient may be impossible. It is advisable in all cases to test the animal with tuberculin, and at the same time carefully sound the esophagus with a probang to see whether tuberculosis exists or not.

Prognosis.—While chronic tympany usually does not lead to immediate death the prognosis is more serious than in acute primary bloating as the causes cannot always be removed.

Treatment.—The treatment is practically the same as that suggested for acute tympany to relieve bloating. This may be followed by the treatment advised for indigestion in cattle. Where there is reason to believe that the intermittent bloating is due to some foreign body in the stomach, rumenotomy may be performed for relief. This is especially indicated in periodic tympany of calves.

ATONY OF THE FORESTOMACHS.

CHRONIC INDIGESTION OF RUMINANTS. CHRONIC GASTRIC CATARRH.

Definition.—An indigestion, which usually develops insidiously, due to a paresis of the musculature of the forestomachs. The contents of the rumen, reticulum and omasum are, as a result, not kept properly mixed and their discharge from these organs is retarded.

Occurrence.—Atony is most common in cattle but is noted in sheep and goats. It usually is the result of improper feeding and care and therefore most commonly seen in cows belonging to poor dairymen in the neighborhood of large cities, especially if kitchen offal, large quantities of brewer's grains and food of poor quality are fed. Atony may attend advanced pregnancy. It also occurs in calves just after weaning and too suddenly changed from a milk to a solid food diet.

Etiology.—Atony may be primary or secondary. In primary cases foods and feeding are the most potent causes. Foods which are too woody (chaff; dried, coarse grasses; shrubs; underbrush; tree tops), improperly harvested forage (cut too green, fermenting clover, over-ripe hay, etc.), spoiled food (moldy meal, decayed vegetables), otherwise damaged food (frozen grass or silage, frosted beets), and feeding kitchen offal in the form of swill, etc.

The injudicious feeding of good food can produce atony as, for instance, overfeeding or the sudden change from a well-balanced to a very narrow ration. Atony may follow impaction of the rumen, provided it has existed long enough to produce a paresis of the muscles of the stomach walls. Withholding roughage is also a cause. Allowing a full drink of cold water on a full stomach may also produce it.

Psychic influences (removing young calf, strange environment, severe pain) in nervous cattle predispose to atony.

Atony of the forestomachs may be secondary to bad teeth, which prevents adequate mastication, chronic disease of the tongue (actinomycosis), traumatic gastritis, and

inflammation of the forestomachs or abomasum. It may be associated with pregnancy especially in those cases which cause an undue uterine enlargement (placental dropsy). Chronic peritonitis which leads to adhesions between the compartments of the stomach or the stomach and the parietal wall is also a cause. Atony may be associated with gastric tumor formations, liver diseases (echinococcus, tuberculosis, pseudoleukemic infiltration, gall-stones). It may also be secondary to chronic heart and lung diseases.

Symptoms.—In acute cases the symptoms are those of a simple indigestion (dyspepsia). The appetite and thirst are diminished, rumination is impaired, the animal chewing its cud at rare intervals and with little vigor. The movements of the paunch are feeble but may occur with normal frequency. If no corrective measures are taken the condition becomes chronic.

The chronic form may develop from the acute or, what is usual, gradually of itself. The appetite is capricious. At times it may be practically normal. Roughage may be preferred to concentrates. The patient usually drinks little water although some cases show increased thirst. Rumination becomes suppressed. Eructations of fetid gas from the gullet are common, and a regurgitation of rumen contents through the mouth is occasionally noted. The ejected mass has usually a fetid odor. The hollow of the left flank is distended with gas, the paunch movements either cease or are very feeble and on palpation (over flank or through rectum) the food in the rumen feels firm and retains finger imprints longer than in health. The bloat is often not continuous but intermittent and increasing following the ingestion of food, a rather characteristic symptom.

Occasionally colicky pains are manifested by the animal switching its tail and kicking against the abdomen with its hind limbs. Rolling, as in the horse, is rarely observed. The bowels are generally constipated, the feces black, hard and dry, sometimes covered with blood-stained mucus and passed in the form of fist-sized balls. If the patient has been fed succulent food, or if the constipation has existed

for a time, diarrhea develops, the feces having a sour, fetid odor and occasionally admixed with undigested food and mucus. A fetid diarrhea may alternate with constipation in protracted cases. Milk secretion is diminished from the beginning.

General.—In mild, acute cases there is little general disturbance beyond a certain degree of languor or restlessness. When the atony persists for several days the patient may become distressed, stand with all four feet drawn together, its head forced into a corner or against the stanchion, the teeth are gnashed frequently, the back is arched, the muzzle is dry and the eyes retracted. The animals lie down a good deal and in the later stages, especially if weakened by lack of food, are brought to their feet with difficulty, presenting symptoms resembling antepartum paresis or parturient paresis. The temperature is rarely high (104.5° F.). In the latter stages the pulse becomes rapid and weak. If bloating or great impaction of the rumen is present, dyspnea exists. In protracted cases the general condition grows continuously worse, the patient becoming anemic, emaciated, even cachectic and eventually dies of inanition.

Diagnosis.—The disease should be distinguished from acute bloat (marked distention, dyspnea, sudden appearance, short duration); chronic bloat (usually no general disturbance, appetite and rumination good); impaction of the rumen (sudden development, no movement of rumen discernible, results of flank and rectal palpation); traumatic gastritis (pain over region of diaphragm, evidence of peritonitis, stiffness of movement, heart symptoms). In acute gastritis the fever, absence of bloat or impaction of the rumen, and the severity of the attack are sufficient for differentiation.

While in many cases it is difficult to determine whether the atony is primary or secondary, usually the history of the case, the insidious development and the negative findings on examination of the other organs, coupled with the effect of the treatment, suffice for differential diagnosis. However, in not every case of secondary atony is it possible to determine accurately the primary disease. Johne's

disease, tuberculosis, tumors, adhesions, chronic metritis, pyelonephritis or even rabies may each have associated with it atony of the forestomachs.

Course.—While mild attacks usually yield to treatment in a few days, chronic cases often become protracted into weeks during the course of which exacerbations and remissions are noted. Signs of improvement are a copious discharge of feces, the disappearance of bloat and impaction from the rumen and the return of paunch movements and rumination. In pregnant cows quite commonly the symptoms subside a few days after parturition. Unfavorable symptoms are complete loss of appetite, continuous bloat, obstinate constipation or, on the other hand, exhaustive diarrhea, fever or subnormal temperature, pain on pressure over the rumen, paralytic symptoms (inability to stand), emaciation. The duration of atony, while usually about one month, may become protracted into several months.

Prognosis.—The prognosis depends upon whether or not the atony is primary or secondary. Primary cases usually yield to corrective treatment provided they have not lasted too long. Obviously success in secondary cases depends upon the primary disease. Healing is slow, usually requiring two to three weeks. Atony attending pregnancy usually disappears after the birth of the calf.

Treatment.—*Hygienic.*—Food should be withheld or restricted (muzzle) for a few days in acute cases. Kneading the walls of the abdomen five to ten minutes every three hours or three times daily is helpful. Salt and plenty of water should be made accessible. If food is allowed it should be easily digested and laxative (fresh grass, bran gruels, fine hay, root crops). Turning the animal out to grass is good.

Medicinal.—Where necessary to relieve constipation laxative drugs are indicated as in impaction of the rumen (see this). The same applies to agents which stimulate paunch movements. Alcohol in small doses (℥iss) well diluted in water given every two hours is serviceable. Hydrochloric acid (℥j in water Oj) not only stimulates digestion but tends to arrest fermentation in the stomach. Creolin

(℥i) three times daily is recommended. Ammonium carbonate (℥vj) in a quart of cold water every four hours is valuable. Once the bowels are open nerve tonics and bitter stomachics with alkalies should be prescribed as in impaction of the rumen.

In obstinate cases the administration of large quantities of water through the stomach-tube or a trocar inserted into the flank is recommendable. To the water may be added an antiseptic or hydrochloric acid. Occasionally obstinate cases are relieved by rumenotomy, a partial removal of the contents and the irrigation of the rumen and abomasum with normal salt solution.

TRAUMATIC INDIGESTION OF OX.

Traumatic Gastritis.

Definition.—An inflammation of the stomach (rumen or reticulum) due to foreign bodies ingested with the food.

Occurrence.—The disorder is common in cattle, especially dairy cows the property of the poorer, shiftless dairymen who permit nails, wire, needles, bolts, etc., to accumulate in the feed troughs or where the cattle feed. Cows with woman attendants frequently swallow darning needles, hair-pins, open safety-pins, etc., which come from the clothing and hair. On arms where the hay is baled for market, pieces of baling wire are commonly picked up by the cattle. Where the herd is grazing on flooded pastures or must drink from shallow sandy springs large quantities of silt and sand will be ingested.

Cattle which have developed the habit of licking stable walls, partitions, etc., which may develop into the vice known as "pica," take into the stomach considerable deleterious foreign matter.

Sometimes masses of hair (wool in sheep) are licked off other animals, or usually due to some itching skin lesion, from the patient itself. These accumulations form hair (or wool) balls in the stomach.

Etiology.—The fact that an animal ruminates does away with the necessity of thorough mastication following prehension. The food is rolled in the mouth by the tongue, which is not very sensitive, mixed with saliva and swallowed without being chewed. Any foreign bodies in it, therefore, reach the stomach. While blunt foreign bodies usually do no harm unless large quantities of them accumulate, or if they block the natural openings of the stomach, sharp-pointed objects (needles, wire, nails) are frequently forced through the walls of the reticulum by the peristaltic movements, causing a traumatic gastritis. Usually the sharp-pointed object penetrates the diaphragm in the direction of the heart sac. Sometimes another route is taken, the wire, needle, etc., entering the lung, liver, spleen, uterus, or even aorta, where it sets up a suppurative inflammation leading to abscess formation or fatal hemorrhage. In some cases the abscesses may become encapsuled and the condition latent, or by way of metastasis, pyemia with multiple abscess formation in parenchymatous organs (lungs, liver) results.

Symptoms.—In general the symptoms are those of an indigestion not traceable to errors in diet, which is periodical (remissions and exacerbations), and often associated with rheumatic-like stiffness of the patient. Blunt foreign bodies produce symptoms of indigestion, which without a clear history of the case are extremely difficult to differentiate from atony of the forestomachs. Accumulations of sand or silt in the rumen sometimes induce symptoms of loss of appetite, suppressed rumination, slobbering, stiffness of movement and frequent groaning. In some cases the rumen feels abnormally hard on palpation and the feces will be found to contain particles of sand. Occasionally the cattle will show toxic symptoms resembling those of parturient paresis.

If the blunt foreign bodies block the natural openings of the stomach sudden illness is produced. This is most commonly seen in calves and lambs where hair, wool, or food balls are the offending objects. The symptoms are those of bloating, colic, restlessness, retching, dyspnea and occasionally epileptiform attacks.

Sharp-pointed foreign bodies produce symptoms of subacute gastro-enteritis, which is intermittent in course. The patient shows usually symptoms of indigestion, bloating, stiffness and falls off in flesh. Pinching the patient in the center of the back, percussion over the region of the attachment of the diaphragm, and palpation over the region of the reticulum produce pain. The gait of the animal is stiff, dyspnea appears on exercise, the temperature is somewhat elevated, and the patient usually considerably prostrated. Parturition, railway journeys, and placing the hind end of the animal higher than the front cause the symptoms to become worse. The usual treatment for indigestion is ineffective. As a rule, in time symptoms of traumatic pericarditis develop (see this).

Diagnosis.—The diagnosis depends largely upon the history of the case (no dietetic errors), the intermittent and variable character of the symptoms, the stiffness of the patient, and the result of percussion and palpation over the region of the diaphragm and reticulum. It has been suggested that drugs which stimulate gastric movements be used to aid in diagnosis. For instance, 1- to 2-grain doses of arecalin or 1-grain doses of sulphate of veratrin given subcutaneously will cause in cases of traumatic indigestion contractions of the stomach with which are associated great restlessness, groaning, and a general intensification of the symptoms, the condition of the animal becoming worse. In ordinary indigestion, on the other hand, the administration of such drugs will tend to improve the condition of the patient. There are, however, a great many exceptions to this rule. In some cases a diagnosis can only be made on necropsy.

Traumatic indigestion may be confused with atony of the forestomachs, bloating, tuberculosis of the mediastinal lymph glands, pneumonia or pleuritis. The differentiation is aided by the history of the case, the specific symptoms of traumatic indigestion (stiffness, intermittency), and in tuberculosis through the tuberculin test.

Course.—The course of the disorder produced by foreign bodies is generally chronic, lasting for weeks or months. Occasionally, due to the fact that a blunt foreign body

obstructs a natural opening or a sharp one begins to wander in the body, symptoms such as described are suddenly precipitated, which result in death in a short time; thus accumulations of sand in the rumen may cause death in two to three weeks, obstructing hair or wool balls in one to two days. In some instances the condition may heal spontaneously if the sharp foreign body works back into the stomach or in case it perforates the wall of the abdomen, producing an abscess and subsequently a fistula, through which it makes its escape into the outside world. These terminations, however, are comparatively rare. In practice most of the patients which develop clinical symptoms either die of traumatic heart disease or pyemia.

Prognosis.—The prognosis is usually bad. Most cases die with pyemia or heart disease.

Treatment.—Medicinal treatment is of little value, although until a diagnosis is established it is recommended to treat as in atony of the forestomachs. In a few instances surgical interference has been successful. Rumenotomy may be performed and the foreign body removed, provided, of course, it has not completely penetrated the wall of the reticulum. Another method is to cast and place the patient on its back. The operator stands with one foot on a chair and the other on the body of the patient over the region of the ensiform cartilage. By pressing the abdominal wall in this region downward with his foot seven to ten times it is claimed the foreign body will be made to slip back into the reticulum. However, it would be just as liable to penetrate in some other direction, injuring new organs. Its use is, therefore, not unattended with danger.

As a general proposition the immediate slaughter of the patient is recommended. From a prophylactic standpoint, removing foreign bodies from the mangers, keeping cattle away from where hay has been baled, etc., are important.

GASTRO-INTESTINAL CATARRH OF SUCKLINGS.

SCOURS OF SUCKLINGS. SPORADIC DYSENTERY.

Definition.—A catarrh of the mucosa of the stomach and bowels of calves, lambs, or foals. Clinically it is a diarrhea

usually occurring in the first few days (two to ten) after birth.

Occurrence.—A very common disease among sucklings, especially those which are "raised by hand," or where the young animal is permitted to suck its dam only at long intervals.

Etiology.—The cause of gastro-intestinal catarrh is undoubtedly bacterial. Probably the Bacillus coli communis is an important factor. While this germ is regularly present in the bowels of sucklings, and usually does no harm, once the resistance of its host is lowered by error of diet, exposure, etc., it can become pathogenic. Other intestinal bacteria may assume a similar role. In a few outbreaks the abortion bacillus has been found. This has led some observers to believe that the diarrhea of calves is identical with the infectious dysentery of the newborn, the disease assuming a milder type due to the greater age of the calf attacked. In the light of present knowledge no one germ can be accused of producing all cases; probably a great many different bacteria produce gastro-intestinal catarrh of sucklings.

Predisposing causes are: (*a*) Weaning too early; (*b*) feeding boiled milk (destroys ferments in milk which aid digestion), or substitutes (flour, linseed, cotton seed) for milk; (*c*) feeding spoiled, contaminated (sour, putrid) milk out of filthy vessels; (*d*) overfeeding, allowing the hungry suckling to gorge itself with milk after too long a period of fasting, as in the case with foals of working mares; (*e*) sucking the diseased udder (various forms of mastitis); (*f*) the eating of solid foods by the newborn animal, which it is unable to digest, such as hay, straw, etc.; (*g*) preventing the suckling from obtaining the colostrum, which removes the meconium from the bowel; (*h*) refrigeration (cold, damp stables).

Symptoms.—The first symptom is usually refusal to suck or, if weaned, to drink the milk offered. The little patient is languid, depressed. Colicky symptoms are not rare. There is often bloating. The most marked symptom is diarrhea. The feces are thin, yellowish or dirty white in

color, often foamy, of pungent, sour odor and voided with tenesmus and considerable force. Sometimes they are flocculent (like buttermilk). They are usually sticky and adherent to the anus, tail, buttocks, etc., which regions they excoriate. As the disease progresses the patient becomes weak and anemic, and remains lying most of the time. The eyeballs retract, the skin feels cool, moist, and sticky, and a most disagreeable odor is emitted from the body. In fatal cases toward the end the periphery of the body grows cold, the anal sphincter relaxed, and incontinence of feces follows. Death may occur under convulsions.

Diagnosis.—Gastro-intestinal catarrh may be confused with infectious dysentery of newborn animals. This latter disease, however, usually appears earlier (may be born with it), runs a much more rapid course, and is very fatal (80 per cent. mortality).

Course and Prognosis.—The course is generally favorable. Often, even after several days' illness, the diarrhea suddenly ceases, the appetite returns, and recovery is rapid. In grave cases the diarrhea may persist for weeks and the disease finally lead to death through catarrhal pneumonia. The younger the animal at the time of attack the less favorable the prognosis.

Treatment.—The diet should be restricted. The surroundings, drinking vessels, etc., of the patient should be made clean and sterile. Hand-fed calves may be given pasteurized or formalin milk (1 to 25,000). Lime-water added to the milk (℥j to the quart) is good. Boiled oatmeal is a valuable addition to prevent bloating. The teats of the mother should be cleansed before the young are allowed to suck. Castor oil (℥j) is a valuable drug in the earlier stages, as it removes from the bowel the irritant contents. This may be followed by an intestinal disinfectant, such as salicylic acid (℥ss) sometimes combined with tannin (℥ss). Calomel (grs. xx) is a valuable drug for foals. To check the diarrhea, opium (℥j) combined with tannin (℥j) and whisky (℥ij) given in a pint of warm water is efficient. A non-officinal preparation to check diarrhea is favorably spoken of. It is called "Mistura contra Diarrhœum," and has the following

formula: ℞—Tinct. opii, tinct. capsica, tinct. rhei, tinct. camphor, tinct. menthi āā℥j, M.D.S. Dose, ʒj to v. Hertwig's mixture is time honored. It is as follows: ℞—Opii, gr. v; mag. carb., gr. xv; pulv. rhei, ʒj, M.D.S. Give in dilute alcohol ℥iss. Dose, tablespoonful twice daily. In calves bismuth subnitrate (ʒj) daily is good.

On collapse subcutaneous doses of slightly alkaline salt solution (sodium chlorid 0.8 per cent., carbonate of sodium 0.25 per cent.) are beneficial. Two quarts of the solution are injected under the skin of the neck. May be repeated in two to five hours. Rectal injections (same dose) are also recommendable. Oil of camphor subcutaneously is an excellent stimulant.

CHAPTER IV.

ANIMAL PARASITES IN THE STOMACH. HELMINTHIASIS.

GASTROPHILUS. BOTS.

Varieties.—The larvæ of botflies occur in the stomach and bowel of the horse. The following varieties have been described: (*a*) Gastrophilus equi, which is usually found in the esophageal portion of the stomach. (*b*) Gastrophilus pecorum, found in the stomach, duodenum and rectum (Hungarian and Russian horses). (*c*) Gastrophilus hemorrhoidalis, found in the pyloric portion of the stomach, duodenum and rectum. (*d*) Gastrophilus nasalis, found in the pyloric portion of the stomach and duodenum.

Life History.—The female botfly, which is common in summer, flying over the pasture fields and roads, deposits her eggs on the skin of the horse, especially on the hairs of the front legs, mane, neck and about the nose and mouth. In from three to five days there issue from the eggs the larvæ, which are licked off by the horse, reach the stomach, where they attach themselves to the mucous membrane, to remain until the following spring. In the months of May to July, and sometimes during the later summer, the bots leave the stomach, pass into the intestines and are voided with the feces. Either on the ground or in the manure they change into the chrysalis stage. In about one month the mature fly emerges, and after copulation the females deposit their eggs as described.

The gastrus larvæ are found in the stomach of all horses on pasture, in small towns or where the botfly appears. Generally speaking, the larvæ are harmless. Only in very exceptional cases do they cause illness through traumatic gastritis. In still rarer instances they produce perforation

of the stomach and peritonitis. Once in a while bots induce inflammation of the rectum or even prolapsus recti, and still more rarely they may enter the brain, larynx and other organs.

Symptoms.—Bots affect principally colts and young horses which have been running on pasture during the summer. When stabled for the winter and placed on dry feed the symptoms become manifest. The animals are unthrifty, thin, show impaired appetite, pale mucous membranes and intermittent attacks of colic. Due to the resulting anemia the pulse becomes weak and palpitation of the heart appears especially on exercise. A cold edematous swelling commonly appears between the jaws. Where the invasion has been great the patient will eventually become emaciated, cachectic and die in three or four months. When the Gastrophilus hemorrhoidalis is present in the rectum the irritation induced leads to restlessness, the colt rubbing its tail, frequent defecation, the formation of whitish, dried crusts of mucus about the anus, and sometimes tenesmus. In rare instances prolapsus recti may result.

Diagnosis.—The diagnosis depends upon the history of the case (occurring after a long run at pasture) and the presence of the bot larvæ in the feces or rectum. Round worms may be excluded only by a microscopical examination of the feces for their eggs. Differentiation from sclerostomiasis (larvæ of the Sclerostomum edentatum) is often very difficult. Not infrequently both parasites occur concomitantly. In important cases a necropsy may be necessary for diagnosis. Infectious anemia affects horses of all ages, is characterized by periodical attacks of fever and rarely affects the appetite seriously.

Treatment.—The only agent of any value to eliminate the bot larvæ is the disulphid of carbon. It should be administered on an empty stomach in capsules in doses of ℥ijss, four doses of which are given, with an hour between each. After twelve to twenty hours administer a good purgative. The larvæ may be removed from the rectum by injections of soap suds or oil. It is also recommended to smear the rectal mucosa with borated vaselin.

Prophylaxis.—Prevention consists in destroying the botflies when observed about horses and by removing the eggs from the hairs once a week with a sharp knife. Cleaning out of the underbrush in woods, pastures, along the roadways, and in the fields is contributory to this elimination.

NEMATODES IN THE STOMACH OF THE HORSE.

The Spiroptera megastoma form tumors in the pyloric portion of the stomach varying in size from a hickory-nut to a hen's egg. When the tumor is incised the thin thread-like worm appears. They sometimes induce gastritis and colic.

Diagnosis.—The diagnosis is only possible by finding the eggs in the feces which is extremely difficult as the embryos (0.6 to 0.7 mm. long) leave the egg *in utero*.

The Spiroptera microstoma are found free in the stomach contents although some of them may be attached to the mucous membrane of the cardiac region. They produce ulceration and induration of the mucous membranes and occasionally mild colic symptoms occurring during feeding, weakness and emaciation.

Treatment.—According to some authorities spiroptera may be expelled by disulphid of carbon or large doses of lysol.

ROUND WORMS IN THE STOMACH OF THE OX.

Occurrence.—Several varieties of round worms (genus Strongylus) inhabit the abomasum of cattle. They usually do harm but occasionally in run-down young cattle on pasture they produce formidable symptoms in a number of animals in a herd or community.

Varieties.—Several varieties of these parasites have been described. The following are the most important:

Ascaris vitulorum, male 15 to 24 cm. long; female up to 30 cm.; found in the abomasum of calves.

Hæmonchus contortus, male 10 to 20 mm. long; female 18 to 30 mm.

Ostertagia ostertagia, male 6.5 to 7.5 mm. long; female

8 to 9 mm. In some parts of Europe 90 per cent. of cattle slaughtered are found infested.

Cooperia oncophora, male 5 to 9 mm. long; female 6 to 8 mm.

Trichostrongylus axei, male 3.5 to 4.5 mm. long; female 4.5 to 5 mm.

For the development of these parasites appropriate books on zoölogy should be consulted.

Necropsy.—In advanced cases the cadaver is emaciated and may show subcutaneous dropsical swellings. The mucous membrane of the abomasum is swollen, edematous, shows punctiform hemorrhages and is sometimes strewn with whitish-gray, pin-head sized elevations, each surmounted by a clear vesicle as big as a poppy seed. Croupous patches may also develop on the mucous membrane. In the contents of the abomasum occur a large number of living parasites.

Symptoms.—The clinical symptoms develop in the late summer or fall and are very similar to those observed in sheep. In the feces a few parasites may be found.

Diagnosis.—The diagnosis depends upon a microscopic examination of the feces for the characteristic eggs. Due to the development of a croupous membrane a differentiation from Johne's disease should be considered.

Treatment.—Treatment is not very satisfactory. Among the simpler remedies recommended is tobacco. Leaf tobacco, coarsely pulverized, is mixed with salt and the cattle given access to it. A decoction of tobacco leaf may be given as a drench. It should contain not more than ʒiij of tobacco. Cold-tar creosote (ʒiv to Oj, depending upon size of patient) is recommended. Copper sulphate is useful. It may be given in rain water in doses of ʒiss to ʒiij.

Building up the animal with abundant food and good care is most important. To prevent spread it is recommendable to feed in a bare barnyard so that the infested droppings may be destroyed and the feed troughs kept disinfected. Cattle should be kept off infested pastures for one year. Such pastures should be put in crops especially corn to destroy worm brood.

ROUND WORMS IN THE STOMACH OF THE SHEEP.

While several varieties of round worms affect the sheep the principal one is the Strongylus contortus or twisted palisade worm. This parasite is a brownish colored thread worm; female 18 to 30 mm. long, male 10 to 20 mm.

Life Cycle.—The life cycle of this parasite is not understood. Probably the embryos are taken up on pastures or perhaps in the sheep fold. From heavy invasions with this parasite lambs become seriously diseased especially in the spring and during wet summers. It is probable that the lambs become infested mainly around shade trees where they gather during the heat of the day. On the open range, in sandy or volcanic ash soil, sheep are rarely affected.

Symptoms.—The symptoms in general are those of a chronic gastro-intestinal catarrh leading to emaciation, anemia, hydremia and death. The infested lambs begin showing symptoms in July and August. They are noticed hanging back from the rest of the flock, are weak, thin and walk with a staggering gait. A cold, edematous swelling develops beneath the lower jaw and the mucous membranes and skin become pale, the latter parchment-like. As quite commonly the lambs are also infested with lung worms the symptoms of lung worm plague (see this) may also be present. Death usually occurs from inanition in two weeks to two months. On postmortem the abomasum of the infested sheep will contain a large number of the small, filiform, reddish parasites.

Treatment.—Treatment is fairly successful. Cull out all suspicious cases and place them in a separate enclosure. The remainder of the flock should be placed on a well-drained pasture in which there are no pools or stagnant water. The lambs to be treated should be starved for twenty-four hours but allowed water. Each lamb should be given gasoline (℥ss) in raw linseed oil (℥ss) mixed with cow's milk (℥iv). The treatment should be repeated for three evenings in succession allowing food several hours after giving the medicine. It may be repeated in ten days. Copper sulphate

is a useful remedy. (Dissolve cupri sulphas 1 pound in water Ox. Dose: 3 months old lamb, ʒvi; 6 months, ʒxii; 1 year, ʒxviii; 2 years, ʒxxv.) Fast twenty-four hours before giving. In the meantime the sheep should be fed ample grain, sheltered from the wet, tagged and given the best possible care. Access to coarsely powdered leaf tobacco mixed with salt is also helpful.

Prophylaxis.—Keeping the sheep in small flocks, constantly changing the pasture, dipping to prevent ticks and feeding ample grain (corn and oats mixed) will accomplish a great deal in the way of prevention.

PARASITES IN THE STOMACH OF SWINE.

The following parasites have been noted in the stomach of swine:

(*a*) **Strongylus Rubidus**: This parasite occurs commonly in the United States. In the abattoir at Washington, D. C., 25 to 75 per cent. of all swine killed were found infested. It has been known to produce a chronic diphtheroid inflammation of the mucosa with anemia and emaciation leading to death. In one instance a number of brood sows kept in a filthy, unfloored pen, became infested. The feces contain a large number of eggs.

(*b*) **Spiroptera Strongylina**: Male 10 to 13 mm. long, female 12 to 20 mm. It is a slender white worm which produces in the submucosa small tumors. It occasionally bores its way into the mucous membrane producing a severe ulcerative gastritis. In marked infestations this parasite has produced a violent inflammation of the stomach ending in death in from three to four days. It is most commonly met with in wild boars and peccary.

(*c*) **Gnathostoma Histidum**: Male 15 to 25 cm. long, female 2 to 3 cm. This is a long tapering worm provided with spiculæ. It is found principally in the mucous membrane of the base of the stomach, its head attached. It is known to produce severe gastritis, a thickening of the gastric wall, dilatation of the stomach, digestive disturbances and eventually cachexia.

(*d*) **Simondsia Paradoxa:** The female, 45 mm. long, is found in cysts which it forms in the wall of the stomach. The male occurs free in the stomach contents.

ANIMAL PARASITES IN THE INTESTINES.

Animal parasites in the intestines may injure their hosts in several different ways. They may produce lesions in the mucous membrane to which they attach themselves, especially worms provided with hooks with which they may perforate the deeper layers or the entire wall of the intestine. Perforative peritonitis may thus result. Where large numbers of parasites are accumulated in balls or bundles they may block the lumen of the bowel. Quite commonly a parasite strays into a duct such as the hepatic duct or may even enter the stomach, esophagus or pharynx. Parasites abstract from the host nutritive material, which leads to an impoverishment of the blood, anemia and emaciation. Probably the greatest injury to the host is wrought through toxic substances eliminated by the parasite. Toxins have been isolated from several worm parasites. When introduced into the body they produce destruction of the red blood corpuscles, loss of hemoglobin, anemia, poikilocytosis and eosinophils.

In some instances a secondary infection with bacteria or an intoxication with the toxins of the parasites may occur. Therefore helminthiasis may have associated with it pronounced nervous symptoms such as hyperesthesia, spasms, or on the other hand, paralysis.

As a general proposition intestinal parasitism becomes serious only when the host has become run down through neglect of proper care and feeding. In well-fed, well-cared-for animals parasites only do harm in case the number is very great or from the fact that the parasite is armed with hooks which may lesion the mucous membrane. Furthermore certain individuals seem to suffer more from the parasites they contain than do others.

Tapeworms (*Cestodes*).—**Life Cycle.**—In its development the tapeworm passes through two stages, *viz.*: The cyst or

larval form and the adult tapeworm. Usually the cyst is found in one animal host and the tapeworm in another. The hosts are usually not even of the same species. As an example, the tapeworm Tænia cœnurus has for its host the dog, while the larval stage, the Cœnurus cerebralis, has for its host usually the sheep. The Tænia cucumerina, the common tapeworm of the dog in its cyst form, is harbored by the common dog flea (Pulex serraticeps).

The development of the tapeworm is as follows: The eggs with the segments of the adult worm which inhabits the intestines are voided with the feces and thus reach the outside world. There they are taken up by a second host, in the stomach of which the embryo, often provided with hooks, is hatched. These embryos penetrate the bowel wall and enter the bloodvessels, and are carried to distant organs, such as the muscles, brain, lungs, etc., developing in these organs cysts. If organs containing such cysts are ingested by animals which would form the proper host there develops in the intestine a tapeworm. The tapeworm is provided with a head or scolex to which is attached a number of segments or colonies that, when ripe, contain the fertile egg of the tapeworm.

Varieties.—Each of the domesticated animals has species of tapeworms peculiar to itself.

Horse: 1. Tænia perfoliata.
2. Tænia plicata.
3. Tænia mamillana.

Ox: 1. Tænia expansa.
2. Tænia denticulata.
3. Tænia alba.

Sheep: 1. Tænia expansa.
2. Tænia ovilla.
3. Tænia alba.
4. Tænia fimbriata.

Symptoms.—Tapeworms, unless present in large numbers, rarely produce symptoms. For instance, over 50 per cent. of the dogs harbor tapeworms, particularly the Tænia cucumerina. On the other hand, tapeworms sometimes cause chronic intestinal catarrh (diarrhea alternating with con-

stipation, icterus and eventually anemia). In horses colic may be caused by them. In sheep they induce chronic bowel catarrh, anemia, and often fatal cachexia. In rare instances a perforative peritonitis may be due to tapeworms.

Diagnosis.—Teniasis can be diagnosed positively only by an examination of the feces in which will be found the segments or eggs of the tapeworm. In sheep a diagnosis may be made by a necropsy.

Treatment.—In horses the most valuable tapeworm agent is tartar emetic (℥ss) daily for three or four days. Turpentine (℥j to ij) is a good teniafuge. It should be given in milk or oil to prevent blistering the mouth or throat.

In lambs picrate of potash (grs. ij to vi), kousso (grs. ij to iij) or male fern (℥j) are recommended. Teniafuges should be given on an empty stomach and followed in twelve to twenty hours by a good purge. Treatment is successful only when the head of the tapeworm is also removed.

Prevention.—In dealing with an enzoötic of teniasis it is advisable to give the animals plenty of good, nutritive food and excellent care. Sometimes these alone are all that is necessary. Obviously, cysts and tapeworms should be destroyed as a preventive measure.

Round Worms (*Ascarides*).—The ascarides or round worms resemble earth worms somewhat in form. The life-cycle of these parasites has not yet been fully determined. Unlike the tapeworms they do not seem to require an intermediate host. The transmission to susceptible animals occurs through the ingestion of fecal matter containing the eggs and not through drinking water. Milk, however, can become contaminated and therefore very young animals are infested. The development of the embryo from the egg is very rapid. For instance, the egg of the Ascaris megalocephala of the horse will develop a complete embryo in three days. The following are the varieties of ascarides:

Horse: Ascaris megalocephala.

Ox and Swine: Ascaris lumbricoides.

Symptoms.—In most cases round worms produce no symptoms. Occasionally, however, if large numbers of them are present in the intestines, they will produce symp-

toms of chronic intestinal catarrh, obstruction of the bowel or more rarely hemorrhagic enteritis. A few exceptional cases of bowel perforation with fatal peritonitis have been due to round worms. At times they may block up the bile ducts and produce icterus. Besides acting in a mechanical way it is very possible that round worms eliminate a toxic substance which acts upon the nervous system, producing symptoms of epilepsy, tetanus or paralysis (anaphylaxis). In calves an enzoötic ascariasis has been observed leading to considerable losses from symptoms of chronic intestinal catarrh (diarrhea). Peculiar to these cases is a penetrating ether-like odor of the breath of the sick patient. The meat has also a similar smell.

Treatment.—In the horse tartar emetic (ʒss) may be given or Fowler's solution of arsenic (ʒj), or arsenic (grs. xv to xxx). Turpentine (ʒij) in oil (Oj), and disulphid of carbon (ʒij in oil Oj) are also recommended.

Calves may be given tartar emetic (grs. xiv) dissolved in water (ʒv), one tablespoonful every three hours in milk. For swine arecanut (ʒj–iv) with flour as an electuary is good.

Prophylaxis.—To prevent reinfestation it is recommended to burn the manure of the affected and thoroughly disinfect the stable.

Palisade Worms in the Intestine.—Following are important worms of this group:

Horse: Strongylus armatus and Str. tetracanthus.
Sheep: Strongylus contortus.
Ox: Strongylus convolutus.

Strongylus Armatus (*Sclerostomum Equinum*).—Strongylus armatus is the most common of the palisade worms of the horse. Its life cycle is not entirely understood. Three varieties have been described: (*a*) Sclerostomum bidentatum, (*b*) Sclerostomum edentatum, and (*c*) Sclerostomum quadridentatum.

Sclerostomum Bidentatum.—Sclerostomum bidentatum is the cause of the common worm aneurysm found in 90 per cent. of all horses in the trunk of the anterior mesenteric artery. The mature worm lives in the large intestines of the horse. The eggs are voided with the feces where

they hatch producing the larval form. The larvæ are taken up with contaminated food and bedding by healthy horses and reach the intestines. From the lumen of the intestines they pass probably into the veins of the mucous membrane and reach the liver through the portal circulation; or they may pass into the lungs through the right heart and from the lungs enter the general arterial circulation. In the trunk of the anterior mesenteric artery they form the worm aneurysm which is one of the causes of colic (embolic) in the horse. The emigration of the larvæ from the aneurysm back to the intestine occurs in that they are carried by the arterial blood to the peripheral intestinal arteries. They then form embolic worm nodules in the bowel walls. Later the larvæ perforate the mucous membrane, re-enter the bowel and attain sexual maturity.

Sclerostomum Edentatum.—This parasite is found in the large intestines in about one-half of the horses. The eggs are passed out with the feces. The embryos are taken up with the food and water and enter the digestive tract of healthy horses. They pass into the bowel walls and reach the subserosa of the parietal peritoneum, where they produce hemorrhages in the peritoneum. Occasionally in colts they cause anemia. Their presence frequently induces an adhesive peritonitis. Rarely the parasite is found free in the abdominal cavity or in the scrotum. From the subserosa the larvæ wander between the leaves of the mesentery back to the large intestines, in the wall of which they become encysted. They later enter the lumen of the bowel and attain sexual maturity.

Sclerostomum Quadridentatum.—Sclerostomum quadridentatum is very rare and does not seem to have a pathogenic action.

Strongylus Tetracanthus.—This palisade worm lives in the mature state in the colon where often large numbers appear in colts. They produce a hemorrhage and even necrotic enteritis with colic and bloody diarrhea. In the feces large numbers of very small strongyli are found. Sometimes death results from bowel hemorrhage. The eggs pass out with the feces and from them rod-shaped

embryos form which are ingested by horses with the food. In the intestines they bore into the mucous membrane of the colon and cecum and become encysted. They are frequently found as submucous nodules in the middle of which are the larvæ surrounded by pus. Finally the larvæ penetrate the capsule into the lumen of the bowel where they reach sexual maturity.

Treatment.—The treatment consists in the use of vermifuges as recommended for round worms. Of late atoxyl (grs. v) given in repeated doses once daily has been recommended for colts.

Prevention.—As a preventive measure filtering the drinking water is advisable.

Strongylus Contortus.—(See Round Worms in the Stomach of Sheep.)

Strongylus Convolutus.—This parasite is found in young cattle and calves, also in sheep. It occurs in the abomasum, where, under the epithelial layer of the mucous membrane, it forms gray, pin-head to pea-sized nodules which contain the parasites. If present in large numbers they produce symptoms of gastric disturbance leading to diarrhea, emaciation and cachectic hydremia.

Œsophagostoma in the Intestines (*Nodule Disease*).—The larvæ of the œsophagostomum frequently produce disease, especially among cattle and sheep. Inasmuch as the most characteristic lesion on necropsy is the nodule produced by the larvæ, the name "nodule disease" has been given to this disorder. The following œsophagostomas occur in domesticated animals:

(*a*) Œsophagostomum columbianum of the intestines (American sheep).

(*b*) Œsophagostomum radiatum in the intestines.

(*c*) Œsophagostomum venulosum of European sheep and goats.

(*d*) Œsophagostomum dentatum of the intestines of swine.

Life History.—The life history of the œsophagostomas, as far as is known, is as follows: The ova which are voided with the feces hatch outside of the body. When ingested by an animal during the late summer they pass from the lumen of the intestine into the walls, where they become

encysted. They remain in the walls six or seven months undergoing in this time three successive changes. In the early spring the larvæ pass back into the bowel lumen and attain full development. In midsummer the females lay their eggs. This applies to all œsophagostoma except the Œsophagostomum dentatum of swine, which is said to mature within the worm nodule in the wall of the intestine.

Necropsy.—In sheep and cattle there appear along the course of the small intestine, and often of the cecum, nodules which vary in size from a pin-head to a shoe-button or even larger. The nodules vary in color, the smaller being black or gray and the larger grayish-white. On incision they are found to consist of a thick capsule of connective tissue surrounding a greenish colored, cheesy, or even purulent mass. The small nodules will be found to contain the larvæ.

Symptoms.—Sheep and cattle when feeding upon low-lying pastures, especially during the months of August and September, ingest the larvæ. It is also possible that they may become infested in stables. As a rule if only a few nodules are present no symptoms of disease are produced. On the other hand if large numbers occur (as many as 6000 have been noted) symptoms of chronic intestinal catarrh, obstinate diarrhea, anemia, emaciation and cachexia are noted. Nodule disease seems most fatal in pregnant ewes fed dry feed during the early spring months. As half of the flock can die in two or three months the disorder assumes economic importance.

Diagnosis.—A positive diagnosis can be obtained only from a necropsy. An examination of the feces is of no value, since the ova have not yet been found.

Treatment.—Medicinal treatment is not successful. However, feeding highly nutritious food, keeping the sheep in small flocks and less crowding during the winter season, especially among pregnant ewes, are the principal indications to be followed.

Echinorhynchus Gigas (*Thorn-headed Worm*).—Echinorhynchus gigas is a long, round, white worm varying, depending on sex, from 6 to 35 cm. in length, which inhabits the intestines of swine. The head of this parasite is armed with several rows of strong hooks.

Life History.—This parasite lives in its larval form encysted in the abdominal cavity of the grub of the May bug. Even adult May bugs may contain them. Hogs which run on woodland pastures, old manure piles, or clover fields, where the May bugs abound, feed on the grub or the adult bugs. In the bowels of the hog the larvæ attain maturity and fasten themselves by means of their hooks to the mucosa of the intestinal wall. In the small intestines the adult worm can produce local inflammation, sometimes even perforation with peritonitis. The patients show symptoms of colic by grunting, restlessness, snapping toward the abdomen, or even other animals. Occasionally in young pigs epileptiform convulsions are produced, which are generally fatal.

Diagnosis.—A diagnosis can be made only by an examination of the feces for the eggs of the parasite. When a number of pigs are sick with symptoms of colic, convulsions, etc., a necropsy will reveal the existence of the worm.

Treatment.—Treatment is not very successful, as the worms are hard to dislodge. One of the best agents is oil of turpentine (℥ij).

Prevention.—Prevention consists in keeping swine out of woods pastures, manure piles, etc., and the destruction of the May bug and its grub.

Oxyuris Curvula (*Pin Worm*).—This parasite is found living in the large intestines of the horse. Frequently, however, it is found in the rectum, where it causes irritation and proctitis, inducing the patient to rub the root of the tail and buttocks. Quite often, as a result of the catarrhal inflammation of the rectum, a layer of white to yellowish dried exudate is found about the anus and perineum.

Treatment.—The usual treatment consists in giving clysters of raw linseed oil (Oj) or vinegar. Tobacco decoction (℥j to Oj of boiling water), oil of turpentine (℥ij in oil or gruel Oj), and quassia infusion are recommended. Clysters, however, reach only the worms in the rectum. Those in the colon may be eliminated by the use of santonin (℥j), tartar emetic (℥ij) or other anthelmintics given *per orem*.

Uncinariasis (*Dochmiasis*).—Uncinaria rarely affect cattle, although cases are reported from the states of Florida and

Texas, where the disorder produced by them is known as "salt sick."

Life History.—The ova which are found in the feces of infested animals develop into embryos within twenty-four hours under favorable conditions. In water and damp earth they undergo several changes, so that after about one month they are capable of further development in the bowel of a susceptible host. The adult worm varies from 3 to 8 cm. in length and its anterior end is bent in the form of a hook.

Natural Infection.—The infection takes place among cattle by drinking water from stagnant pools containing the embryos or from infested low-lying pastures. Obviously, drought, lack of proper food and care, digestive disturbances, etc., which reduce the resistance of an animal, are contributing causes.

Symptoms.—In general the uncinaria produce symptoms of a gastro-intestinal catarrh leading to anemia, hydremia, and cachexia. One of the first symptoms noted is a morbid desire on the part of the patients to lick objects. The cattle become thin, show diarrhea, often alternating with constipation and bloating. Symptoms of colic are not infrequent. Later hydremia develops with edema of the region of the throat, hydrothorax and ascites. The patients usually die of inanition.

Diagnosis.—Diagnosis can be made by a microscopic examination of the feces for the ova. Johne's disease is distinguished from it by the character of the diarrhea, which is more watery than in uncinariasis and the presence of acid-fast bacilli in scrapings from the anterior portion of the rectum.

Treatment.—Treatment is only fairly satisfactory. Intensive feeding and better care keep up the strength and health of the patient, greatly assisting the cure. Medicinal treatment is the same as for round worms.

Prevention.—Keep cattle away from infested pastures, water only from running streams, wells, or good springs, sprinkle the infested manure with lime water, and thoroughly clean and disinfect the stable.

CHAPTER V.

DISEASES OF THE LIVER.

JAUNDICE. ICTERUS.

Definition.—Jaundice, or icterus, is due to the deposit of bile pigment in the organs of the body, especially in the skin and mucous membranes. Jaundice is a symptom and not a disease, and may be due to a variety of conditions. From a pathological standpoint three forms of jaundice are distinguished: (*a*) Catarrhal jaundice, (*b*) hepatogenous jaundice, and (*c*) toxic jaundice.

From a clinical standpoint three types of jaundice are differentiable: (*a*) Obstructive jaundice, (*b*) malignant jaundice, and (*c*) jaundice of the newborn.

Obstructive Jaundice.—Definition.—Obstructive jaundice, sometimes called catarrhal icterus, is the commonest form in animals, especially dogs, and is usually associated with a gastro-intestinal catarrh which involves the duodenum, leading to a tumefaction of the mucosa of the bile ducts. As a result the bile cannot be properly eliminated, therefore, some of it is resorbed by the blood. Obstruction by foreign bodies within the ducts (parasites, rarely gall-stones) may be a cause. Obviously, tumors, enlarged lymph glands, or even fecal accumulation, provided they press upon the ducts, can have the same effect. The icterus seen in influenza of the horse is probably the result of catarrh of the bile ducts, although it may be in part toxemic.

Symptoms.—Obstructive jaundice usually begins with symptoms of digestive disturbances (gastro-intestinal catarrh). Later there develops a tinting of the mucous membranes, skin and urine. The color ranges from a lemon yellow (catarrhal icterus) to a deep olive green in permanent obstruction (malignant icterus). In the urine the presence of bile pigment may be determined chemically

(Gmelin's test). As no bile reaches the intestinal tract the feces are pale drab or slate colored, and usually very fetid. The general symptoms are those of languor, dulness, slow pulse and subnormal temperature. In man the respirations may fall below normal and xanthopsia, or yellow vision may occur.

Prognosis.—The prognosis in this form of icterus is usually favorable.

Treatment.—Treatment consists in regulating the diet (gastro-intestinal catarrh) and the administration of anti-catarrhal agents, such as Carlsbad salts. To relieve constipation, purgatives (aloes, calomel) are recommended. Pilocarpin (grs. iij), followed by potassium tartrate, is given.

Malignant Jaundice (*Icterus Gravis*). — **Definition.** — This form of jaundice is marked by its malignancy, and characterized anatomically by a destruction of the liver cells, with reduction in volume of the organ.

Occurrence.—Malignant jaundice is a rare disease in animals. Occasionally cases are seen in the horse and sheep.

Etiology.—The causes are not well understood. It has been known to follow phosphorus poisoning, feeding lupine, sour potato peelings, vetch straw, and hay from flooded meadows. In some cases it is probably due to a toxin eliminated during septicemia and gastro-enteritis. In man there seems to be a close association between the disease and pregnancy.

Symptoms.—The principal symptom is a profound icterus, associated with which are weakness, coma, sinking of the temperature, hemorrhage of the skin and mucous membranes, and often a rapid, fatal course.

Prognosis.—The prognosis is bad. Death usually occurs in about one week.

Treatment.—Treatment is unsatisfactory. Recommended are disinfectants, purgatives and diuretics.

Jaundice of the Newborn (*Icterus Neonatorum*).—**Definition.**—This form of icterus, as the name indicates, occurs in animals just after birth.

Occurrence.—It is more common in calves, although occasionally is met with in foals during the first few days after birth.

Etiology.—It seems to have several different causes: Constipation, which prevents the discharge of the meconium; stenosis of the gall-ducts; increased secretion of bile in the first few days of life; reduced blood pressure, and probably diseases of the umbilical veins.

Symptoms.—Symptoms are those of a catarrhal icterus which in some instances is preceded by gastro-intestinal disturbances.

Prognosis.—The prognosis is bad, as the majority of cases soon die. Those animals which do recover usually do so when the digestive disturbance is relieved.

Treatment.—Same as for catarrhal icterus.

INFLAMMATION OF THE LIVER. HEPATITIS.

Definition.—By the term hepatitis we understand an inflammation of the liver. Depending upon the seat of the inflammation, *i. e.*, whether in the liver cells or interstitial tissue, the course and the anatomical character of the inflammation, three clinical forms may be distinguished: (*a*) Parenchymatous hepatitis, (*b*) chronic interstitial hepatitis, and (*c*) purulent hepatitis (abscess of the liver).

Acute Parenchymatous.—Definition.—This consists in an inflammation of the liver cells with cloudy swelling and fatty infiltration of the same.

Etiology.—The disorder is almost always a secondary condition. Primary cases may be induced by numerous toxic substances, such as spoiled food, poisonous plants, phosphorus or bacteria which are carried to the liver through the portal system, or occasionally through the umbilicus. In the latter case the hepatitis may be enzoötic, as occurs in pigs and lambs.

Hepatitis is secondary to many infectious diseases (septicemia, influenza). It may also be secondary to poisoning with phosphorus or arsenic. In some instances the cause is

parasitic (distoma, cysticercus, sclerostoma), which wander into the liver substance.

Necropsy.—The liver is swollen (borders rounded), congested, spotted with dark red hemorrhages. The consistency is softer and more friable than normal.

Symptoms.—The symptoms of acute parenchymatous hepatitis are usually very vague and masked by those of the primary disease, to which it is secondary.

Diagnosis.—A diagnosis can rarely be made during life, except in the dog, where the symptoms are those of weakness, icterus, and sensitiveness over the region of the liver.

Treatment.—Treatment consists in the administration of salts, which mildly purge, and the use of intestinal disinfectants. The diet should be regulated by excluding food rich in fats.

Chronic Interstitial Hepatitis (*Cirrhosis of the Liver*).—**Definition.**—Cirrhosis of the liver consists of an inflammatory proliferation of connective tissue with atrophy of the liver cells. Two forms of cirrhosis may be distinguished, the hypertrophic and the atrophic. In the first form, due to cellular infiltration and the increase in connective tissue, the liver is increased in size; in the latter form, due to a shrinkage of the connective tissue, a marked decrease in the size of the organ occurs. The consistency of the liver is very hard and firm and its surface very irregular (hobnail liver), or the surface may appear granular or lobed or there may be diffuse induration.

Occurrence.—Chronic interstitial hepatitis, while usually sporadic, in some instances may assume an enzoötic distribution (Schweinburg disease, North Dakota bottom disease), causing considerable loss among cattle, sheep and swine.

Etiology.—The cause of interstitial hepatitis in animals is unknown. In all probability it may be toxic and due to plants of the Senecio group (rag wort), or it may be infectious. In animals it is more often the result of animal parasitism (sclerostomiasis). Congestion of bile is a rare cause. Cirrhosis of the liver is a symptom of distomatosis.

Symptoms.—The symptoms of the disease do not usually attract attention until the latter stages, and even then they are rather vague in animals. In general they consist in symptoms of chronic gastro-intestinal catarrh, with a tendency to icterus. Impaired or capricious appetite, periodical attacks of colic, especially after feeding roughage, symptoms of immobility (stupor, forced movements, vertigo), icteric or pale mucous membranes, loss of condition, hide-boundness, and eventually emaciation are the symptoms usually noted. In small animals, and in large ones if sufficiently emaciated, it is possible to percuss out an enlarged area of hepatic dulness (see larger works). In small animals ascites and anasarca appear toward the end.

Course.—The course is chronic and extends over months and years.

Treatment.—The treatment is usually of little value, although some observers report temporary improvement after using iodin preparations.

Purulent Hepatitis (*Abscess of the Liver*).—**Etiology.**—Liver abscesses are not uncommon in calves and adult cattle, but are rare in horses. They may originate in the following ways: (*a*) Enterogenic infection due to pus organisms or the necrosis bacillus from an infection of the portal system from the bowel. (*b*) Embolic or pyemic abscesses, which are formed *via* metastasis in strangles or pyemia. (*c*) Infection through the umbilicus of newborn colts and calves. (*d*) Traumatic abscesses due to foreign bodies which penetrate the liver usually from the reticulum and more rarely from without. (*e*) Parasitic abscesses due to echinococci, distomes and other animal parasites which have invaded the organ.

Symptoms.—The symptoms are very indefinite, and a diagnosis is rarely made during the life of the patient. Briefly they consist in fever, symptoms of indigestion, pain on palpation, and the presence of bile pigment in the urine.

Course and Prognosis.—The course is chronic and the prognosis bad.

Treatment.—Treatment in animals is of no avail. In rare instances opening the abscesses has been attempted, but with indifferent success.

THE LIVER FLUKE DISEASE. DISTOMATOSIS.

Definition.—An inflammation of the liver and bile ducts due to the presence of trematode parasites of the genus fasciola.

Occurrence.—The disease produced by these flukes is commonly known as "liver rot" and sometimes assumes an epizoötic distribution, particularly among sheep, where it causes considerable loss. The disease is sometimes noted in cattle, but rarely attains in these animals economic importance. There are two forms of liver fluke in animals: (1) Distoma hepaticum (Fasciola hepatica), and (2) Distoma lanceolatum (Fasciola lanceolata).

Natural History.—The eggs of Distoma hepaticum reach the outer world with the feces. On damp pastures and under the influence of summer heat the embryo is hatched in about one month. They then enter snails, in which in about four weeks they become converted into sporocysts, out of which there develop rediæ. From the rediæ pass out the cercariæ. The cercariæ swim about in water and attach themselves to blades of grass, on which they become enclosed by a sticky substance. The infestation of the animal occurs from eating the grass or forage or from the drinking water containing these encysted cercariæ. It is possible for infestation to take place in the sheepfold either from green food and water or probably from dry food. After ingestion the parasites pass through the bile ducts, in which they become sexually mature in from five to six weeks. From the gall ducts, in which they have produced chronic inflammation and dilatation, they pass into the liver substance. Here they induce hemorrhagic foci, multiple abscesses and ultimately cirrhosis of the liver. In rare instances they may perforate the capsule of the liver or even the portal vascular system, causing phlebitis and thrombosis. Sometimes they enter the lungs, inducing hemorrhagic foci and encysted worm nodules may be found in the bronchi. From the lungs they may enter the arterial circulation and pass through it to all parts of the body. A passage from the mother to the fetus through the placental

circulation occasionally happens, which accounts for their appearance in newborn calves.

Symptoms.—In cattle, as a rule, notwithstanding the great inroads which the parasites make on the liver, clinical symptoms are absent. In sheep, however, they produce symptoms of a severe general disease—anemia, hydremia and ultimately cachexia. After a latent period of one to two months, and usually in the autumn, the infested sheep begin showing symptoms of general anemia ("paper skin"). In the early winter the sheep grow thin, show chronic indigestion, weakness, edema of the eyelids, throat region, and ventral portion of the abdomen (anasarca), and also ascites. As these symptoms may also occur in other worm plagues of the sheep a diagnosis can be made only by a necropsy or by examining the feces for eggs.

Course.—The course is very varied. In severe cases after a period of three months death ensues. In other instances the sheep may live through the winter and die in the spring, or recover after the emigration of the flukes, which takes place in the following autumn.

Treatment.—A medicinal treatment is of no value. Experience with arsenic and the various vermifuges has given no results. The principal indication is to strengthen the constitution of the sheep by intensive feeding and proper care.

Prophylaxis.—The sheep should be kept off infested pastures, especially land which lies low, be given a wide range and kept in small flocks. Short pasture is dangerous because the cercariæ are found close to the grass roots. The egg-infested manure should be spread out on land which is to be tilled and the livers of the dead sheep rendered harmless.

ECHINOCOCCUS DISEASE OF THE LIVER.

Definition.—An inflammation of the liver due to an invasion of the echinococcus parasite.

Occurrence.—The disease is quite common in cattle and swine but rare in other animals.

Natural History.—The echinococcus cyst is the larva of the Tænia echinococcus, a tapeworm of the dog. The segments of the tapeworm are voided with the feces of the dog, polluting stables and pastures where they are taken up by herbivorous animals. The embryos which hatch from the eggs in the stomach and intestines of the new host wander into the bile ducts and portal circulation of the liver in which organ they form cysts some of which contain scolices. The cysts vary in size from a pea to a human head. In turn if a liver containing the cysts is eaten by a dog there develops in the intestines of the dog the parent tapeworm, the Tænia echinococcus.

Two varieties of Tænia echinococcus have been determined: (*a*) The Echinococcus unilocularis which is the most common form and usually found in sheep. This cyst forms a simple cyst or there may be adherent to a principal cyst secondary or tertiary cysts. (*b*) The Echinococcus multilocularis. This cyst is comparatively rare and is found in cattle. It has no capsule but forms in the liver a proliferating tumor containing many lacunæ and macroscopically resembles somewhat a carcinoma.

Besides the liver the echinococcus may invade the lung, heart, muscle, brain and peritoneum, or even occasionally bone or cartilage (the sternum).

Symptoms.—The symptoms produced by the presence of this parasite in the liver are usually very vague. In cattle notwithstanding a great development of cysts in the liver the animals usually appear perfectly healthy. In general the symptoms are those of a chronic digestive disturbance and emaciation. Sometimes the animals show sensitiveness over the region of the liver and on percussion an increase in the area of hepatic dulness. On rectal examination sometimes the enlarged nodular liver may be felt. In individual cases chronic bloating may result as in mediastinal tuberculosis causing compression of the gullet. In swine ascites is an occasional symptom. In very rare instances the rupture of superficial cysts causes fatal peritonitis. Recent application of the complement-fixation test, using the fluid contained in the cysts as the antigen, has demonstrated that it is valuable in diagnosis.

Treatment.—No satisfactory treatment for the disease is known. Of great importance is the prevention which consists in the destruction of all echinococcus cysts found on postmortem and the use of teniafuges to drive out the adult parasite in dogs.

RUPTURE OF THE LIVER.

Definition.—Rupture of the liver with hemorrhage into the abdominal cavity.

Etiology.—The causes are generally traumatic (falls, kicks, jumping, overexertion, etc.) or more rarely rupture may occur spontaneously from fatty liver, carcinoma of the liver, or in primary or secondary (anthrax, purpura) hepatitis. It can result from amyloid liver and embolism. Phosphorus poisoning may also be a cause.

Symptoms.—The symptoms of rupture of the liver attended by marked hemorrhage are those of internal hemorrhage, such as pale mucous membranes, imperceptible pulse, general sweating and staggering gait. Death usually ensues in about one to ten hours. Small hemorrhages may heal under cicatrization.

Treatment.—Treatment is rarely successful unless the hemorrhage is relatively slight. Recommended are subcutaneous doses of ergot (℥ij), hydrastis or adrenalin (1 to 5 c.c. of a 1 to 1000 solution for each 250 pounds body weight). To strengthen the heart oil of camphor may be given.

NECROSIS OF THE LIVER.

Definition.—A multiple necrosis of the liver is a process characterized on necropsy by pea- to walnut-sized, dry, yellow, circumscribed centers in the livers of cattle, swine and sheep. It is due to the necrosis bacillus. Fibrinous peritonitis frequently attends the condition.

Symptoms.—The symptoms are very vague and consist in an enlargement of the liver, sensitiveness over the region of the liver, high fever, languor, disturbance in appetite, alternating constipation and diarrhea.

Treatment.—The disorder, which can be rarely diagnosed, does not yield to treatment.

AMYLOID LIVER.

Character.—Amyloid degeneration of the liver occurs in animals and generally in connection with amyloid kidney. It is usually the result of chronic suppuration (strangles, pleuritis, abscess of the liver, etc.). The amyloid liver is characterized by its large size, grayish or yellow-gray color, and soft, cheesy, friable consistency. In the presence of iodin it gives a characteristic rich mahogany-brown color.

Symptoms.—The clinical symptoms are very indefinite. They are those of anemia, cachexia, intermittent colic, icterus, rupture of the liver, and internal hemorrhage.

CARCINOMA OF THE LIVER.

Mostly secondary from metastasis. Except in older dogs is rare in animals.

Symptoms.—The symptoms are very vague and consist in disturbance in digestion, emaciation, anemia, icterus, colic symptoms, peritonitis, rupture of the liver and fatal hemorrhage.

GALL-STONES. CHOLELITHIASIS.

Very rare in animals. They occasionally are met with in cattle and dogs.

Symptoms.—The principal symptoms they induce are colic (horses and cattle), icterus and digestive disturbances.

Diagnosis.—A diagnosis is rarely made during life.

Treatment.—The treatment consists in the use of artificial Carlsbad salts.

PARASITES IN THE LIVER.

Parasites other than those mentioned in the liver of large animals are Cysticercus tenuicollis, the cyst of the tapeworm Tænia marginata of the dog. These cysts are usually found in sheep, swine and calves. The Sclerostomum bidentatum is occasionally found in the liver of the horse.

CHAPTER VI.

DISEASES OF THE PERITONEUM.

PERITONITIS.

Definition.—An inflammation of the peritoneum. The following forms may be distinguished: From the standpoint of course, acute and chronic; extent, circumscribed and diffuse. According to the exudate a dry (sicca), and an exudative, the latter being serous, serofibrinous, purulent, ichoric or hemorrhagic. Specific types are the tubercular and glanders peritonitis.

Occurrence.—Peritonitis is usually secondary in animals. The horse is most commonly subject to it the result of colics and laparotomies (cryptorchid castration). It is also seen in cattle due to puerperal infection and traumatic gastritis.

Etiology.—The causes of peritonitis are varied and from an etiological standpoint the following forms may be distinguished:

(*a*) Traumatic peritonitis due to penetrating abdominal wounds from without or from rupture and perforation of internal organs (stomach, bowel, uterus, bladder, spleen, or liver), or the bursting of a mesenteric abscess from within. Most active in traumatic peritonitis are streptococci and the colon bacillus. In peritonitis following stomach or bowel rupture death may occur before peritonitis actually develops, due to peritoneal sepsis.

(*b*) Peritonitis is secondary to inflammation of organs contiguous to the peritoneum, the inflammation spreading from the stomach, bowel, uterus, bladder, or liver. Peritonitis may be secondary to pleuritis.

(*c*) *Via* metastasis peritonitis may develop in the course of pyemia, septicemia, tuberculosis or glanders. Carcinoma and sarcoma may involve the peritoneum. Peritonitis may also accompany hemorrhagic septicemia in cattle.

Symptoms.—As peritonitis is nearly always secondary its symptoms are preceded by those of the primary disease (colics, metritis, septicemia).

In acute, diffuse peritonitis the following symptoms are present: Abdominal pain (colic) is prominent especially toward the end. The patient usually does not roll or even lie down as in colic. The gait is stiff, the animal moving as one piece, avoiding short turns, and in peritonitis following castration the hind legs are advanced in abduction, the animal walking in a straddled fashion. The abdominal wall is tense and in many instances bloating is present. Sensitiveness is not so marked in horses but in some cases pain is evinced on manual pressure.

The pulse is rapid (80), irregular and hard ("serous membrane" pulse), often "wiry." The respirations are increased, shallow and of the costal type. With increasing bloating or accumulation of abdominal exudate, the dyspnea becomes more marked. The temperature is usually high (108° to 109° F.) but of no particular type.[1] The conjunctiva is highly reddened in the early stages but later becomes "muddy" or even cyanotic. The peristalsis is suppressed and usually there is obstinate constipation with tenesmus. The urine is voided at frequent intervals under symptoms of strangury.

The general condition of the patient is that of great prostration. The facial expression is anxious, and the body often covered with a cold sweat. Forced movements are not uncommon (walking in a circle, etc.).

On rectal examination at times the surface of the peritoneum may be felt roughened.

Diagnosis.—The sudden development, the fever, serous, membrane pulse, bloating, stiff gait, obstinate constipation, and colicky pains form characteristic symptoms of peritonitis, especially if they follow a primary disease or operation (colic, castration) to which peritonitis can be a sequel. Most valuable is the finding of the rough and sensitive peritoneal surface on rectal examination.

Confusion with "colics" due to acute dilatation of the

[1] In rare instances in perforative peritonitis (sepsis!) it is subnormal.

stomach, tympanites, displacements of bowel or gastro-enteritis is very probable especially if the pulse becomes frequent and irregular early. However, the character of the pulse in peritonitis is harder and in gastro-enteritis diarrhea with loud peristalsis is present. In ordinary "colics" due to fecal stasis, etc., there is no fever present and the pulse is softer in quality.

Chronic or circumscribed peritonites can rarely be diagnosed.

Course.—In acute, diffuse peritonitis, especially when a sequel to gastric or intestinal rupture or the bursting of a mesenteric abscess (strangles), the course is obviously rapid and fatal causing death within twenty-four hours. In less violent cases the disease may last several days (four to fourteen) and lead to death; or more rarely become chronic, lasting for months, leading to adhesions (adhesive peritonitis) and ascites, causing the patient to suffer from periodical attacks of colic. There is usually edema of the ventral part of the abdomen.

Prognosis.—Acute, diffuse peritonitis is a very fatal disease usually leading to death in a few hours. In mild infections, not associated with the perforation of the stomach or bowel or the escape of pus in the abdominal cavity, death may not ensue for several days. Circumscribed peritonitis rarely leads to death. Chronic peritonitis may persist for months or even years and cause adhesions which may in some cases affect digestion. Peritonitis due to penetrating abdominal wounds, if treated according to surgical principles before infection has become extensive, may be kept under control, its spread prevented and healing produced.

Treatment.—The treatment of acute, diffuse peritonitis consists in the use of hot applications or of sharp counterirritants, such as turpentine or the oil of mustard in alcohol (1 to 12). To prevent the spread of the inflammation by the movements of the intestines, opium (℥j to ij) is given. Later mild purgatives such as calomel (℥j) may be employed. Attempts, however, should be made to allay constipation by using lukewarm clysters rather than through the use of drugs. Later to assist in the absorption of ascitic fluids diaphoretics

and diuretics may be tried. Tapping may be employed and repeated as often as the indications warrant. If the heart becomes weak oil of camphor may be used. This drug is also recommended as an intraperitoneal injection (℥v) which decreases the absorbing power of the peritoneum. In small animals a laparotomy can be performed and the abdomen flushed out with antiseptics.

ABDOMINAL HYDROPSY. ASCITES.

Definition.—Strictly speaking ascites is an accumulation of transudate in the abdominal cavity. In a broader sense it would include the fluid exudate the result of chronic peritonitis.

Occurrence.—Ascites, while common in dogs, is rare in the horse and ox. An exception is noted in cattle fed large quantities of turnips or the residue of beet-sugar factories. In the horse ascites is generally a symptom of chronic peritonitis or interstitial hepatitis.

Etiology.—From an etiological standpoint three forms are recognized, *viz.*, ascites due to (*a*) passive congestion, (*b*) hydremia, and (*c*) peritonitis.

In ascites due to passive congestion there is a mechanical disturbance to the blood circulation in the course of chronic heart (valvular disease), lung, liver (cirrhosis), or kidney disease, leading to blood stasis in the venæ cavæ and portal veins. A portal congestion can also result from enlarged lymph glands (mesenteric!). A hydremic ascites is most common in sheep and cattle due to animal parasitism (liver fluke disease) and the hydremia which attends cachectic conditions. The peritonitic ascites accompanies simple chronic peritonitis and is sometimes a symptom of tuberculosis (ox), carcinomatosis or sarcomatosis of the peritoneum.

Symptoms.—The principal symptom is the distention of the abdomen, which becomes pear-shaped, due to the fluid contained therein. Thirty to forty gallons may collect in the horse or ox. Fluctuation may be noted on palpation. On percussion of the lower abdomen a flat tone is emitted with a horizontal upper line which shifts as the position of the

patient (small animals) is changed. When tapped a clear, yellow fluid may be drawn off. The specific gravity of the fluid is about 1012 and the albumin content about 2 to 4 per cent. There is usually no fever. If a large quantity of fluid is present it may press the diaphragm forward and interfere with the action of the lungs, inducing dyspnea. Usually there are also symptoms of the primary disease present (heart bruits, albuminuria, examination of blood). Often associated with ascites are hydrothorax, hydropericardium, and anasarca (speaks for heart lesions). If the portal system alone is involved (cirrhosis of the liver) only ascites may be present.

Diagnosis.—In large animals, unless the ascites is marked (abdominal distention), due to the tenseness of the abdominal walls, it may be overlooked. Obviously any condition which enlarges the abdomen might be mistaken for it. Therefore, pregnancy, dropsy of the fetal membranes in cows, distention of the bladder, urine accumulation (rupture bladder in ox), and the rare cystic ovary or tumors (dogs) should be thought of. In large animals rectal exploration (pregnancy, distended bladder) is helpful in diagnosis. An explorative puncture can be employed in cases of doubt. It not only reveals the presence but the character of the fluid (transudate, exudate, urine, etc.). The determination of the primary disease is often difficult, especially when a lung or liver disease, as the symptoms are vague.

Prognosis.—Unless the result of feeds too rich in water, or due to hydremia the prognosis in ascites is bad. The primary disease, of which it is merely a symptom, is usually incurable.

Treatment.—Treatment is generally unsatisfactory. In case it is due to food too rich in water or too high an altitude (brisket disease of Colorado cattle), placing the patients on dry food or bringing them to lower levels is curative. In large animals medicinal treatment rarely pays. Diuretics, such as digitalis (fld. ext. ʒj) or theobromia (ʒij), may be tried. Arecalin (gr. j) and pilocarpin (grs. v) are recommended if the heart will stand them. A good purge of aloes (ʒvj) or Glauber salts (lb. j) is useful.

Tapping the abdomen is advisable when dyspnea is distressing the patient. The operation may be repeated several

times provided the patient eats and is allowed ample nourishment to compensate for the loss of albumin. Omentopexy is employed in human practice. This is the operation of suturing the omentum to the abdominal wall, securing anastomotic communication between the portal system and that of the vena cava, thus producing a collateral circulation between the portal and general circulation.

TUMORS OF THE PERITONEUM.

Carcinoma and occasionally melanotic sarcomas occur in the peritoneum. On the visceral peritoneum fibromas, lipomas, and myxomas are occasionally seen.

Symptoms.—The symptoms of tumors of the peritoneum are usually too vague for diagnosis. When generalized carcinomatosis is present the patient becomes cachectic and shows ascites, which would lead to the suspicion that the peritoneum was involved. In horses and cattle the tumor may sometimes be palpated through the rectum.

Treatment.—Treatment is rarely possible, although benign tumors might be removed surgically.

ANIMAL PARASITES IN THE PERITONEUM.

Horse.—The Filaria papillosa is commonly found in the peritoneum of horses. The larvæ of sclerostomes also occur. They are commonly encapsuled, or more rarely adult specimens are found free in the abdominal cavity.

Sheep.—In sheep the Cysticercus tenuicollis is very frequent and may lead to acute peritonitis. It appears as large, hickory-nut to walnut-sized cysts surrounded by peritoneum. The neck of the parasite is inverted into the cyst. Liver flukes are also occasionally found in the peritoneum of sheep.

Swine.—In swine the Stephenurus dentatus, a thread-like parasite, is not uncommon. More rarely echinococci and the Cysticercus tenuicollis occur.

PART IV.

DISEASES OF THE REPRODUCTIVE ORGANS.

General Remarks.—The comprehensive consideration of the diseases of the reproductive organs is not within the scope of a work of this kind. There are now available several works which include these diseases and their treatment. The diseases of the reproductive organs are usually studied in courses on obstetrics and most books on this subject include them. On account of the importance of sterility in breeding animals, especially cattle, many veterinarians in districts where breeding is carried on extensively make it a specialty. Obviously persons so engaged should consult special works.

PUERPERAL SEPTICEMIA.

Definition.—A septicemia which comes from infected wounds in the birth passages.

Occurrence.—This form of septicemia, which follows parturition, is most common in cows, although it occurs occasionally in mares due to an infected wound (tears) in the vulva, vagina or uterus.

Etiology.—The causes are usually streptococci or the colon bacillus. The infection is carried into the vagina or uterus with the hands, instruments, ropes, etc., used by the obstetrician in attempting delivery. The result of the infection is usually an ulcerous or croupo-diphtheritic inflammation of the uterus and vagina, with which is usually associated phlegmon. A metritis and perimetritis may at times develop and also a peritonitis. Besides the local conditions noted we have the general changes due to septicemia and pyemia, such

as swelling of the parenchymatous organs, hemorrhages, and metastatic abscesses in internal organs.

Symptoms.—The symptoms of puerperal septicemia develop within one to four days after parturition. The animal may show abdominal pain, straining, and from the vagina there will flow at first a blood-stained discharge which later becomes putrid and odorous. The lips of the vulva are edematous, cold, and discolored. The temperature is high, reaching in the cow 107.6° F.; the pulse rapid, irregular, weak; there is no appetite; rumination is suppressed and usually the patient soon lapses into a state of paralysis of the hind parts and unconsciousness. Decubitus develops very rapidly.

Diagnosis.—Puerperal septicemia might be confused with parturient paresis. However, it may be distinguished from the latter by the local swelling of the genital organs, vaginal discharge, high fever, and absence of pronounced muscular paralysis. Puerperal septicemia clinically very closely resembles sapremia due to retained placenta. In some cases a differentiation cannot be made until after the uterus has been cleaned of its putrid contents and disinfected. In sapremia the patient rapidly recovers once the cause is removed.

Course.—Puerperal septicemia is very often fatal, death occurring within three or four days or in some instances within a single day. Recovery may occur in one or two weeks. In some cases the patient is left in a state of chronic pyemia which leads to emaciation, intermittent fever, chronic vaginal discharge, and purulent endometritis (pyometra). If secondary abscesses develop in organs, such as the lungs, kidneys, udder, joints, etc., the course is prolonged. In horses purpura may be a sequel.

Prognosis.—Prognosis is generally unfavorable, 70 per cent. of the patients dying in the acute attack or from resulting complications.

Treatment.—Treatment consists in a thorough disinfection of the uterus and vagina with a solution of lysol or creolin (2 per cent.). In the mare bichlorid of mercury (1 to 1000) may be employed. The general symptoms, such as fever

and weakness, may be combated symptomatically with alcohol and veratrin (grs. ij).

Prevention.—Prevention consists in having all obstetrical instruments, ropes, and the hands of the operator disinfected before attempting to assist in delivery.

PARTURIENT PARESIS. MILK FEVER.

Definition.—A non-febrile disease of cattle, swine, and goats occurring at or following parturition and characterized by general paralysis and usually unconsciousness.

Occurrence.—The disorder is common among cows, especially valuable dairy cows, which are heavy feeders and deep milkers. It usually occurs at the acme of lactation and in cows that are well bred and in prime condition. Thin cows or very fat cows do not seem predisposed. When delivery has been difficult, parturient paresis is less apt to occur than when the birth has been easy and the expulsion of the afterbirth prompt.

Primipara are very rarely attacked. Usually it occurs in cows from the third to the fifth calving.

Etiology.—The causes of parturient paresis are unknown. The following theories have been suggested: (*a*) Auto-intoxication from the uterus or udder resembling ptomaine poisoning; (*b*) it may be an anaphylactic phenomenon, or (*c*) an anemia of the brain, the result of the sudden blood flow to the udder or in consequence of a vasomotor collapse.

Symptoms.—The symptoms usually begin twelve to forty-eight hours after delivery. A few cases are recorded where the attack came on during or even before the birth. The principal symptoms are suddenly developing general motor and sensory paralysis, with loss of consciousness. After showing some symptoms of languor, weakness, and staggering gait the cow lies down. She may regain her feet but arises with difficulty. Finally she becomes completely paralyzed and unconscious. Often the patient is found lying on her sternum with her head thrown around against the flank, the muzzle resting close to the udder. In other cases she lies flat on her side. The respirations are slow and deep, the temperature

normal to subnormal. From the nostrils a lymph-like fluid is discharged. Besides these general symptoms those of specific paralyses of the cranial nerves occur, especially the oculomotor, trigeminal, glossopharyngeal, vagus, hypoglossus and sometimes the opticus.

The oculomotor paralysis is expressed by drooping of the upper eyelids (ptosis) and dilatation of the pupil; the trigeminal paralysis leads to sinking of the lower jaw; the glossopharyngeal paralysis leads to dysphagia; the vagus paralysis produces inactivity of the muscles of the larynx, leading to stenotic, noisy respirations. It also increases the frequency of the pulse and induces a paralysis of the gullet and stomach (tympanites); the paralysis of the hypoglossus causes prolapse of the tongue and the paralysis of the optic nerve produces amaurosis. As the sympathetic nerve is also involved, paralysis of the brain and urinary bladder occurs.

Course.—The course is very acute. Untreated animals may die in twelve to forty-eight hours. In a few cases the disease may take a subacute course, with relapses. Cases which recover from the parturient paresis may die in about one week from foreign body pneumonia, due to the dysphagia, which allows medicines, saliva, and paunch contents to enter the windpipe and lungs. Once in a while a case is left with a chronic paraplegia which may last for two or three weeks, and end in recovery or through decubital gangrene lead to death. Mastitis and necrosis of the deep muscles of the femur are rare complications.

Prognosis.—While formerly the mortality was 50 per cent., since the use of the new method of treatment it has been reduced to 10 per cent.

Treatment.—The best treatment for parturient paresis is that first suggested by Schmidt of Kolding, Denmark, who in the year 1897 recommended the injection of a solution of iodid of potash into the udder. Later oxygen gas was employed, and still later sterile air. This treatment is simple: By means of a pump air is forced through surgeon's cotton into the teat canals until the udder is well inflated. In most instances a remarkably prompt reaction on the part of the patient is obtained. Obviously the instrument, especially

the teat tube, should be sterile, the ends of the teats thoroughly disinfected and the hands of the operator clean. The instrument should be sterilized by boiling rather than the use of antiseptics. In fact it is not advisable to permit antiseptics to enter the udder as mastitis is apt to result. If the sphincters of the teats are too weak to retain the air, bandages may be placed around the teats to reinforce them. Otherwise ligation should not be employed. If after three to six hours no results come from the first inflation of the udder a second may follow. If done under aseptic precautions several inflations will be tolerated by the mammary gland without danger of infection.

Whether or not medicinal treatment in addition to the use of air is of value is debatable. In the average run of cases it is certainly unnecessary, except perhaps for the effect it may have on the owner. The use of excitants such as the subcutaneous injections of caffein (℥j), strychnin (gr. ss) or arecalin (gr. j) is recommended as an auxiliary treatment. Giving medicine *via* mouth should be avoided.

ABNORMALITIES IN SEXUAL DESIRE.

In animals the sexual desire may deviate from the normal in two ways, *viz*: It may be abnormally increased producing a condition known as nymphomania in the female and satyriasis in the male. On the other hand the sexual desire may be diminished in either the male or the female. Of particular importance is the nymphomania of cows, mares and sows and the diminished sexual desire of the male. Occasionally cows do not come in estrum which greatly lessens their economic value.

Nymphomania.—Etiology.—The causes of nymphomania are not always determinable. In cows a common cause is cystic ovaries or more rarely tuberculosis or tumors (sarcoma, carcinoma) in the ovaries. Diseases of the uterus such as endometritis, cervicitis and very rarely occlusion of the os may lead to a cow failing to conceive and therefore a repeated return of the period of estrum. In other instances the causes seem to lie outside of the reproductive organs and probably

emanate from the spinal cord. Obviously anything which prevents conception and renders the female animal sterile will produce a return of the period of heat which is not a true nymphomania.

Symptoms.—In cows the symptoms are those of an exaggerated estrum. Repeated copulation fails to produce conception. The period of heat is also prolonged. The patients are nervous, excited, keep up a continued bellowing and running around the pasture or enclosure, mounting other animals and even human beings. Milch cows fall off in their milk and the milk itself is of poor quality. Later the patient emaciates, the croup sinks on each side of the root of the tail, as in pregnant animals, and the neck thickens resembling that of the male.

In mares there is a frequent and almost continuous estrum followed by failure to conceive or abortion if bred. The patient is ticklish, nervous, frequently switches her tail, blinks the vulva, ejecting small quantities of urine, and often becomes vicious, kicking in harness or biting at other animals or persons who come near her. Some mares on the contrary show symptoms of cerebral depression as in hydrocephalus (act like dummies). Very rarely they show maniacal symptoms or convulsions. If the condition persists for any length of time the patient loses flesh and shows a capricious appetite.

In sows much the same symptoms occur as in the horse and ox. The sow is continuously in heat, does not conceive when bred, eats poorly and consequently loses flesh and becomes unprofitable. She will mount continuously other swine and may become vicious, attacking other animals or man.

Treatment.—The best treatment for confirmed cases of nymphomania is ovariectomy which is especially valuable in mares and cows. In some instances crushing the ovarian cysts or expressing yellow bodies through the rectum and vagina has given favorable results. The amputation of the clitoris which for a time was much practised in mares is only occasionally successful. Placing a leaden ball in the uterus is rarely of benefit. The use of narcotic drugs such as bromid

of potash, morphin and chloral hydrate has but a temporary effect. Laxatives are also employed but do no permanent good. In America large doses of salix nigra have been recommended. The preparation of this drug made by Lloyd Brothers of Cincinnati is highly spoken of. Obviously it will have no permanent effect upon cases of nymphomania due to gross lesions in the reproductive organs.

Diminished Sexual Desire. — Etiology.—A great many causes may diminish the sexual appetite. The patient may be over-fat, of phlegmatic temperament or may be debilitated from recent illness. Special causes are congenital defects and acquired diseases of the reproductive organs. As examples may be mentioned chronic endometritis, atrophy, degeneration and aplasia of the testicles or ovaries. Excessive sexual use, especially when the animals are young, and long continued masturbation are causes. Often the cause is purely psychic. For instance many jacks refuse to cover mares although jennets are served with promptness. Zebra stallions will rarely copulate in the presence of persons, although if left alone with the female the coital act is promptly committed.

Treatment.—Diminished sexual desire should not be confused with impotency or sterility although it may be a cause of these. The food and care of the animal should be looked after and any apparent discrepancies in these factors removed. If the patient is overfat curtailing the food ration, allowing greater quantities of protein and plenty of exercise may overcome the condition. Some male animals which have been kept isolated or away from females for a long time seem to lose the sexual habit. Very young stallions which have never covered a mare must sometimes be encouraged and stimulated to perform the coital act. Jacks which refuse to serve mares can often be stimulated sexually by allowing them to smell the urine from a jennet in heat or sometimes even by holding a jennet where they can see her while approaching or teasing the mare to be served. Certain drugs (aphrodisiacs) will stimulate sexual desire. Cantharides (cows ʒj, mares ʒss; or the tincture, cattle ʒv and mares ʒiiss) has long been employed as an agent to promote sexual desire.

Of late Yohimbin, used subcutaneously, is recommended. Its cost makes it almost prohibitive for veterinary use.

STERILITY.

Definition.—By the term sterility we understand that the female animal is barren or unable to produce young.

Occurrence.—Sterility is very common especially among well bred cows and obviously attains great economic importance.

Etiology.—Sterility is a symptom and not a disease. It can have therefore a great many causes. To go into these in detail is beyond the scope of this work. The most common causes, however, are: Disease of the uterus, often a chronic catarrh the result of retained placenta or abortion. In some instances purulent endometritis or chronic catarrh of the cervix or vagina may be the cause. In other cases sterility may be due to the ovaries which harbor retained corpora lutea, or cysts, or are fibrously degenerated. Tumors, or an adhesive peritonitis with displacement of the ovary are rare causes. Furthermore the uterine tubes may be stenotic or occluded. Occasionally tuberculosis of the uterus or ovaries is a cause.

Symptoms.—The symptoms are failure to conceive although the animal may be bred repeatedly. Occasionally she may never come in heat. In either case she remains barren.

Prognosis.—Obviously the prognosis depends on the cause. If due merely to a catarrh of the vagina or uterus, which yields to treatment, to retained corpora lutea or ovarian cysts which can be crushed manually, recovery may be expected. On the other hand if due to displacement, tumor formation, tuberculosis, atrophy or aplasia of the ovary, or occluded uterine tubes, the case is hopeless. The same is true of hermaphrodism. Occasionally twins are sterile though not always.

Treatment.—The treatment consists in removing the cause. In cases of chronic catarrhal or suppurative endometritis or vaginitis a thorough disinfection of the genital tract often produces results. If due to occlusion of the os (a rare cause) dilating the opening may suffice. Quite often reducing the

acidity of the catarrhally inflamed vagina, by flushing it out with a one-half of one per cent. solution of bicarbonate of sodium, is useful. Yeast has also been recommended. Obviously on the other hand where there is an organic disease of the organs of reproduction all of these treatments will fail. Therefore many cases of sterility are incurable.

IMPOTENCY.

Definition.—By impotency is meant the inability of the male to impregnate the female. There are two forms of impotency, *viz*: (*a*) The animal may be incapable of performing the coital act (coital impotency), and (*b*) while the coital act may be performed, living, virile spermatazoa are not discharged (azoöspermia).

Etiology.—The principal causes of that form of impotency which prevent coition are diseases and injuries of the penis, such as paralysis, tumors and fractures, or of the prepuce such as phimosis and inflammation (posthitis). In other instances the impotency may be due to diseases of the brain and spinal cord or to general weakness of the body as the result of chronic disease. In stallions painful lameness such as spavin, gonitis or sacral lameness may be causes.

The causes of the second form of impotency are usually due to diseases or degeneration of the testicles as orchitis, aplasia, atrophy, tumors or cryptorchidism leading to aspermia and azoöspermia.

Treatment.—Treatment consists in removing the cause wherever this is possible. Obviously if organic diseases or injuries which cannot be remedied involve the testicles or penis, treatment is out of question. On the other hand if due to inflammation of the sheath the use of disinfectants and cleanliness will remove this cause. If bodily weakness and general debility are the causes, rest, good food and care are all that are necessary. Young males should not be allowed to serve too many females within a short period of time. Painful conditions of the limbs are sometimes removable by the application of surgical treatment.

PART V.

DISEASES OF THE BLOOD AND BLOOD-PRODUCING ORGANS.

ANEMIA.

Definition.—Anemia is a condition in which the blood is deficient in quality or in quantity. The deficiency in quality may be a diminution of the amount of hemoglobin (oligochromemia) or in the number of red corpuscles (oligocythemia). Anemia may be local, due to the fact that the blood supply to a part is diminished, or it may be general. A primary and a secondary anemia are also distinguished.

Etiology.—Local anemia is due to a mechanical interference with the blood supply to a part. The interfering factor may be an embolism, tumor, spasm, etc., which impedes the onflow of the blood. A primary anemia is due to diseases of the blood-making organs and is usually an independent disease such as pernicious anemia, or leukemia. A secondary anemia develops from faulty nutrition (poor food), severe hemorrhage or may accompany bacterial, animal parasitic or protozoan diseases such as tuberculosis, distomatosis, piroplasmosis, etc.

Symptoms.—The symptoms which characterize anemia are paleness of the mucous membranes and skin, loss of energy, dyspnea, emaciation and hydremia with edematous swellings. The patient will also show rapid pulse, palpitation of the heart, systolic heart murmurs and often fever. An examination of the blood shows a diminution of the red corpuscles from one quarter to one-half, so that in place of eight million there may be only four or even two million per c.mm. The

number of leukocytes and the form of the erythrocytes remain, however, practically normal.

Course.—While anemia produced by a sudden loss of blood may be fatal within a few minutes, the course in most anemias is chronic, lasting for months or years.

Prognosis.—The prognosis depends upon the cause of the anemia. If the causes are benign and removable, such as moderate hemorrhage, poor food, etc., proper care and treatment will rapidly replace the lost blood. On the other hand if the cause is malignant and cannot be removed, such as advanced tuberculosis, chronic protozoan diseases or generalized carcinomatosis, the prognosis is bad.

Treatment.—The treatment must be governed by the causes of the anemia. Where due to hemorrhage, poor food and the like obviously these conditions must be rectified. When this is done a rapid recovery follows. In secondary anemias the patients are best treated by allowing plenty of good food and such medicinal blood plastics as iron, arsenic, and phosphate of lime. In man the transfusion of blood from a healthy individual to the anemic one is advisable. Sometimes good results are obtained by the infusion of physiological salt solutions, which may be combined with adrenalin, into the veins or rectum. The patient should be allowed plenty of drinking water provided there is no hemorrhage present. Where the anemia is secondary to a malignant disease like tuberculosis or cancer the treatment has only a temporary effect and is not curative.

LEUKEMIA.

Definition.—Leukemia is an anemia with a marked increase in the number of leukocytes in the blood. Contrary to a simple leukocytosis the course is chronic.

Etiology.—Leukemia is evidently a specific disease of the organs which form the leukocytes such as the bone marrow, lymph tissue and spleen. The causes are not understood.

Forms.—Two forms of leukemia are now distinguished in animals, *viz*: (*a*) Lymphatic leukemia (lymphemia), and (*b*) myeloid leukemia (myelemia).

(*a*) Lymphatic leukemia is characterized by enlargement of the lymph glands and the presence of small, uninuclear lymphoctyes in the blood.

(*b*) In myeloid leukemia there is an enormous enlargement of the spleen, the follicles of which are hyperplastic (lineal leukemia). In the blood large numbers of neutrophil polynuclear leukocytes occur even as many as from one to five hundred thousand in place of eight thousand per c.mm. The erythrocytes are diminished in number and their form is changed. In the myelogenic leukemia the red bone marrow is hyperplastic, infiltrated and often resembles pus. In the blood there is an increase of the large uninuclear myelocytes. In both forms of leukemia anemia is present.

Symptoms.—The development of leukemia is insidious. Generally the patient shows lack of energy, sweats easily, shows capricious appetite, heart palpitation and rapid, small pulse. The mucous membranes become pale even to pure white in color. The patient grows thin, eventually emaciates and from time to time edematous swellings appear on different portions of the body. In the lymphatic form, which is commoner in animals than in man, swelling of the lymph glands appears symmetrically on both sides of the body. The swellings are firm, round and non-sensitive. The lymph glands of the maxillary space, the prepectoral and precrural lymph glands are commonly involved. In some cases the enlargement of lymph glands is so great as to interfere with the function of organs. Therefore dyspnea, roaring, lameness, etc., result. Occasionally an enlargement of internal lymph glands (mediastinal, sublumbar) leads to severe dyspnea, fecal retention and the like.

In the lineal form the spleen is enormously enlarged. In the horse it may be palpated through the rectum and in small animals occasionally a distention of the left side of the abdomen designates the enlarged spleen. The liver is also greatly enlarged, in one case in the horse weighing nearly fifty pounds. The blood is pale, stains less intensively and coagulates very slowly. Under the microscope the number of leukocytes is greatly increased. In some instances there may be as many white as red corpuscles in the microscopic field.

The coagulated blood is separated into two layers, a lower of red corpuscles and of violet color and an upper of milky appearance, grayish-white, made up of white corpuscles and some fibrin. The temperature is usually little changed but toward the end often becomes subnormal. In the last stages hemorrhages occur in the conjunctiva, gums, bowels, etc.

Diagnosis.—The diagnosis can be made with accuracy only by examining the blood microscopically. In pseudoleukemia the symptoms described also occur but the blood shows no change in the number of white corpuscles. With the aid of the microscope the form of leukemia may also be determined by proper stains.

In tuberculosis, glanders and in malignant tumors enlargement of lymph glands also appears, but the distribution of the enlarged glands is rarely so symmetrical and the accompanying anemia not so pronounced as in leukemia. Furthermore, by a microscopical examination of the blood, and the application of the proper tests, a differentiation from glanders or tuberculosis can be made.

Prognosis.—The prognosis in leukemia is bad. No case of the disease correctly diagnosed has ever recovered.

Treatment.—Treatment in animals should not be attempted as it will not lead to success. In man arsenic, iron and the *x*-ray have been used to prolong life. In human patients leukemia has been known to last for twelve years before causing death.

PSEUDOLEUKEMIA. HODGKIN'S DISEASE.

Definition.—Pseudoleukemia is a chronic disease of the blood-forming organs almost identical with leukemia except that there is no increase in the number of white blood corpuscles.

Occurrence.—Pseudoleukemia occurs in horses and cattle and is much more frequent than leukemia. By some authorities pseudoleukemia is supposed to represent a form of leukemia without increase in the number of leukocytes. The true cause of pseudoleukemia is not known.

Symptoms.—The symptoms are identical with those of leukemia with the exception that the relation of the red to the white corpuscles is not conspicuously changed.

Prognosis.—The prognosis is bad.

Treatment.—In man iron, arsenic, iodin, and the *x*-ray are used to prolong life.

HYDREMIA.

Definition.—Hydremia is a condition in which the proportion of the serum to the corpuscles of the blood is excessive. In cattle and sheep a severe anemia occurs with which is associated a general hydremia leading to edema of the skin (anasarca), accumulation of transudate in the abdominal cavity (ascites), thoracic cavity (hydrothorax) and heart sac (hydropericardium).

Etiology.—The causes are chronic diseases of organs, which are usually due to parasites (liver flukes or lung and stomach worms), or from the feeding of foods containing too much water such as slop, the offal of distilleries, beet sugar factories, etc. A hydremia due to high altitude occurs among cattle in the mountainous west (Colorado). The principal symptoms of this disorder are heart palpitation, weak pulse, loss of flesh, languor, and edematous swellings particularly under the sternum. Locally the condition is known as "brisket disease." Removing the cattle to lower altitudes usually brings about a speedy recovery.

HEMOPHILIA.

Definition.—Hemophilia is a marked and abnormal tendency in some individuals to bleeding or hemorrhage. Sometimes the slightest wound will bleed so profusely as to become serious. The condition is usually hereditary.

Etiology.—The causes are not known. The condition is rare in animals.

SCURVY. SCORBUTUS.

Definition.—Scurvy is a disease resembling purpura, rare in animals although occasionally seen in dogs. It is characterized by a tendency to bleed from the gums and to hemorrhage in various organs of the body. In animals the disease is probably infectious. In man scurvy is most often

seen among sailors and persons who live upon salted meats and canned food but get no vegetables. Drinking lime juice and eating fresh vegetables usually bring about a rapid cure.

INFECTIOUS ANEMIA OF THE HORSE.

SWAMP FEVER. RIVERBOTTOM DISEASE. LOIN DISTEMPER.

Definition.—An infectious disease of horses which is characterized by being a specific septicemia accompanied by intermittent or remittent fever, albuminuria and ultimately progressive anemia. It is probably due to a filtrable virus.

Occurrence.—Infectious anemia while confined to infected areas in a country is widely distributed. It occurs on the Continent of Europe and in North America where it has been reported from the province of Manitoba and the states of Minnesota, Nebraska, Kansas, Missouri, Arkansas, Wyoming, Colorado, Washington, Mississippi and Texas. It also occurs in the Panama Canal Zone. While probably most prevalent in, it is by no means confined to swampy districts but appears on high, well drained lands. Naturally the disease is confined to the horse although other equidæ may be infected artificially. Most of the cases occur during the summer and early fall. While it may appear at other times of the year, cold weather seems to cause its abatement. As the disease affects a large number of horses on infected farms rendering them incapable of performing work and causing many deaths, its economic importance locally is very great. In a herd of 28 horses used for railroad construction in North Dakota in the year of 1908, the loss from infectious anemia was 17. In another reported instance in the same state, of 242 head the loss sustained in a single year was 98 (40.5 per cent.). While it is very probable that the laity in swamp fever districts is apt to accredit to this disease losses in horse flesh from other causes, nevertheless it forms a serious menace to the horse industry and should it become more widespread the financial loss resulting would be very great.

The disease will remain on a given farm for a number of

years (10–15) where annually it causes losses among the horses.

Etiology.—Infectious anemia is due to a filtrable virus which cannot be demonstrated by staining methods nor by cultivation. The virus is contained in the blood, urine, and feces of both clinical cases of the disease and apparently healthy horses. According to some authorities the feces, however, will not transmit the disease. It is very probable that the feces are infectious only when mixed with infected urine.

The disease may be transmitted by virulent blood or urine given intravenously, subcutaneously or orally. Other domesticated animals and guinea pigs do not seem susceptible.

Natural Infection.—The disease seems to be taken up through the digestive tract in food, water, stable litter, etc., which have become contaminated with the urine and feces of infected animals. On poorly drained pasture fields pools of water may easily become polluted with the discharges of infected horses, especially with urine, thus serving as sources of infection. The disease does not seem to be directly communicable. Cases are recorded where healthy horses have been confined in stables and mingled freely for months with sick ones without evidence of any transmission of the disease. It is possible that patients are not eliminating the virus continuously and during all stages of the disease.

Suckling colts are not infected through nursing diseased mothers nor has an intra-uterine infection been observed.

The disease is usually introduced into a community by the purchase of either a clinical case or an apparently healthy horse ("missed case").

Necropsy.—In general the postmortem findings are those of an acute or chronic septicemia. Depending upon the duration of the disease they offer great variations. There are no postmortem changes which can be considered pathognomonic. The principal changes noted are: Petechiæ and ecchymoses occurring under the serous membranes especially of the epicardium and endocardium, spleen and bowels; swelling of the lymph glands which are usually blood-shot; changes in the color and structure of the bone marrow

especially of the long bones. A longitudinal section of the femur or humerus, for instance, will show in the bone marrow dark red areas which are sharply demarcated from the yellow portion. In acute cases there may be marked swelling of the spleen, a symptom not so manifest in chronic cases. The blood may show no macroscopic changes; in chronic cases it appears of lighter color, watery. While the condition of the cadaver may be good in acute cases, in chronic ones it is usually emaciated and shows edematous swellings of the skin of pendent portions (sheath). Icteric discoloration of the tissues is not rare.

Symptoms.—The period of incubation after artificial inoculation varies from five to nine days. From natural infection it is usually two to three weeks. Two clinical types of the disease may be recognized: (*a*) *Acute Type.*—The acute form which begins suddenly with symptoms of languor and muscular weakness. The horse tires easily at work, may fall to the ground in harness and must be assisted to its feet. Quite often this weakness is most pronounced in the hind parts ("loin distemper").

Fever is a constant symptom the temperature reaching its acme in two to three days, ranging from 104–107° F. The fever is of a remittent or intermittent type continuing until the death of the patient, periods of increased temperature alternating with feverless periods lasting one or two days. The pulse usually ranges from 60–90, is soft and weak. The heart beat is often tumultuous, a symptom increased by exercise.

The conjunctiva appears puffy, swollen, slightly tinged with yellow and occasionally spotted with petechiæ which are principally on the nictating membrane. There is usually slight lacrimosis. The nasal mucosa is congested and shows petechiæ. There may be a slight nasal discharge of reddish color. The patient often shows diarrhea, the feces blood-stained. A constant symptom is albuminuria, the amount of albumin varying from a mere trace to 1.5 per cent. The albuminuria is, however, not continuous but usually appears synchronous with a rise in temperature. The appetite in acute cases, especially during a fever attack, is impaired; in

chronic cases a good appetite may be retained. The general condition of the patient rapidly becomes bad. Accompanying the febrile attacks there is usually great loss of flesh, the patient finally becoming extremely emaciated.

There is a tendency for edemas to appear on the limbs, under the thorax or ventral portion of the abdomen. Quite often, however, these swellings are absent.

The blood shows little tendency to coagulate and the blood serum is discolored yellow or somewhat greenish, its color varying with each change in angle of observation. There is a great diminution in the number of red corpuscles which in a period of two weeks can be reduced to two million or less.

As the disease progresses the animal grows weak, emaciated, cachectic, paralyzed behind, covered with decubital sores and dies of inanition. Pregnant mares often abort. The acute form usually lasts from one to four weeks. In young colts it may end fatally in from one to two days.

(*b*) *Chronic Type.*—The chronic type of infectious anemia is characterized by periodical attacks of fever with feverless periods of varied duration between. Sometimes the feverless interval is only a few days, at others it may be a few weeks or even months. The fever attack may last two to four days or longer. The rise and fall of the temperature take place gradually. The pulse is usually increased when the temperature is high but generally only slightly. The mucous membranes in the early stages are normal but later become anemic. Petechiæ are rarely seen. During the fever periods the anemia may temporarily disappear. Albuminuria is a constant symptom occurring concomitant with a fever period. In some instances diarrhea is noted and occasionally colic. The general condition of the patient becomes bad, it grows thin, the hair loses its luster, stands on end, the skin becomes dry. The patient is extremely weak, the tail hangs motionless, the sphincter ani relaxes and incontinence of urine and feces appear. Quite often the appetite is preserved until the end. In some cases the condition of the animal is retained to a remarkable degree; beyond an intermittent or remittent fever the patient may show no symptoms for many weeks or

months. During the course of the disease in individual animals many exacerbations and remissions are noted.

The chronic form may last several months; in a few cases even years.

Diagnosis.—In some instances the diagnosis is not difficult; in others it is impossible unless blood inoculations are made. Where a district is known to be infected and the symptoms of anemia, emaciation, edematous swellings, anemic pulse, extreme weakness and usually good appetite are present, a correct diagnosis can generally be arrived at without much danger of error. On the other hand where the patient shows no anemia, and the condition remains good, the diagnosis can be extremely difficult. Only by taking the temperature daily for a long period of time or by blood inoculations can the disease be determined. The complement-fixation test has shown itself to be of no value.

Infectious anemia might be confused with influenza, forage poisoning, anthrax and sclerostomiasis. However, a careful study of the clinical phenomena coupled with a good history (infectious anemia having existed on the premises) will usually make the differentiation attainable.

Course and Prognosis.—The acute form usually leads to death as such. It is exceptional to find acute cases going over into the chronic type. Recovery is very exceptional and death may occur quite unexpectedly. The mortality is over 90 per cent. Apparent recoveries are often noted.

Treatment.—A medicinal treatment is without avail. Quinin, trypan blue, atoxyl, collargol, and arsenic preparations were found of no permanent benefit. Absolute rest and very intensive feeding will prolong life and cause improvement in many cases. Rest in the stable, keeping the temperature down with cold baths and enemata, and arsenic administered internally have been recommended. This treatment should be continued for about four to six weeks. Generally speaking, however, the treatment of infectious anemia is very unsatisfactory.

Prophylaxis.—The prophylaxis consists in destroying all animals diseased, segregating the suspects and protecting the food and water from contamination with urine. The

stables should be disinfected and wet pasture lands drained. In the purchase of new horses, especially those coming from known infected districts, all anemic animals (pale mucous membranes, early fatigue at work, increased heart frequency after slight exercise, etc.) and those showing albuminuria should be rejected or at least placed in quarantine for a period of three months. Experiments to produce artificial immunity have so far proved unsuccessful.

AZOTURIA. PARALYTIC HEMOGLOBINURIA.

Definition.—An acute auto-intoxication of the horse characterized by degeneration of certain muscles and the presence of hemoglobin in the urine.

Occurrence.—Most frequent in young, well fed horses accustomed to regular work. The disease usually follows a transient rest of a day or so and appears when the horse is again put to work.

Etiology.—The predisposing causes are heavy feeding during a short (two to three day) rest, the horse being used to regular work. Exceptions, however, are noted: Horses are sometimes befallen in the stable, and those which have not been rested, may be attacked in harness. More rarely are poorly fed, thin horses affected. Occasionally azoturia may follow unusually severe exercise as struggling in the hobbles, becoming cast in the stall, etc. The disease is more common in winter than during the hot months but may occur any time of the year. The disease occurs in hot, winterless climates. Badly ventilated, dark, damp stables are said to be predisposing causes.

The exciting cause of azoturia is not known. In all probability it will be found to be due to the formation of certain toxins which develop either in the muscle or in the digestive tract during rest. These toxins degenerate the muscle parenchyma and induce a dissolution of the red blood corpuscles, setting their coloring matter free. Cold may assist in that it stimulates metabolism.

Symptoms.—The attack usually occurs without warning and within ten to fifteen minutes after leaving the stable and

while being driven on the street. In rarer instances several hours may elapse before symptoms appear. The patient, which has been playful and lively perhaps, suddenly becomes stiff behind or may knuckle in a hind fetlock. If the horse be forced ahead it may fall to the ground, where it generally makes vigorous but ineffectual efforts to regain its feet. Some cases do not fall, however, but retain their feet; the gait is stiff and the hind parts not fully under control. As a rule the patient cannot walk and is therefore transported to the stable or hospital in an ambulance. When down during the first stages of the disease, the patient is restless and may struggle desperately to rise. Quite often the head, especially around the eye is contused, the recumbent patient recklessly throwing its head against the street or the stall partition. While the mind is clear the face shows great anxiety, no doubt due to pain. The respirations are increased and the whole body dripping with sweat. The muscles of the hind parts, especially the crural muscles, the quadriceps femoris and the adductors are harder than normal and swollen. In some instances the muscles of the anterior limb are involved, especially the anconeus group. The affected muscles are not particularly sensitive and in a day or two become softer and relax. The temperature in mild cases is not much influenced after the restlessness subsides. In severe cases, however, due to such complications as decubital gangrene, great blood dissolution, nephritis, pneumonia, etc., the patient may show high fever. The pulse is high during the excitement early in the attack, but later becomes slower unless complications involving the heart muscle set in. It is often 50–60 but may reach 80–100.

The urine is often retained, distending the bladder. Where drawn it is found turbid and dark, the color varying from that of coffee mixed with a little milk to an almost inky blackness. It is rich in sediment and if strained becomes clear but does not lose the dark coloration. The specific gravity is high, the reaction usually alkaline. Albumin is usually present, phosphate, urates and some hippuric acid are generally found.

The appetite is usually retained until serious complications

appear. If nephritis complicates the case, large quantities of albumin appear in the urine and uremic spasms and loss of consciousness follow.

The blood coagulates readily but the clot is not firm and the serum limited. The serum is usually red stained. The quantity of hemoglobin present varies greatly. After the sweating has subsided and the patient has begun to drink freely it will be found slightly below normal. The specific gravity of the blood is normal and the number of red corpuscles somewhat reduced.

Complications.—(*a*) Decubital gangrene which may cause a general infection; (*b*) hypostatic congestion or even inflammation of the lungs; (*c*) uremia following nephritis; (*d*) fracture especially of the pelvis and limb from falling during the disease.

Course.—In mild cases where the patient retains its feet, the duration may be very short, lasting but one or two hours. Severe cases usually continue for two to three days when the patient begins rapidly to improve or becomes worse and dies. While death may occur on the first to third day, the patient usually lives a week. General infection is the commonest cause of death. In some cases a paralysis of the quadriceps femoris, adductors or crurals are sequels which may delay complete recovery for several months. A given patient may suffer repeated attacks of azoturia within a few weeks or months.

Diagnosis.—The cardinal symptoms of azoturia are: 1. The muscular paralysis, and 2. the dark-colored urine. The history of the patient should also be taken into consideration. The following diseases and conditions resemble it: (*a*) Colic. Confusion with colic would be excusable only in the early stages of the attack. There is neither paralysis nor hemoglobinuria in colic.

(*b*) Injury to the spinal cord. Here the paralysis is complete behind the lesion; the tail, sphincters, bladder and rectum are also paralyzed. The skin posterior to the injury is not sensitive to pin-pricks. There is no hemoglobinuria.

(*c*) Fractures of the pelvis and posterior limb bones may sometimes simulate azoturia. This is also true of rupture of

the Achilles tendon. Only a careful examination of the patient will determine these surgical conditions. The history and absence of hemoglobinuria are indicative.

(*d*) Thrombosis of the posterior aorta and its branches may resemble azoturia in that the patient falls upon the roadway, sweats, etc. However, the attack is of short duration (fifteen minutes) when the horse gets up again. By driving it further an attack can be brought on as before.

Prognosis.—Mild cases which do not "go down" as a rule recover. In the lighter horses the prognosis seems more favorable than in the heavier breeds. The greater the severity of the attack and the darker the urine, generally speaking, the graver the prognosis. When the patient has not been properly nursed (frequently turned and deeply bedded) decubital sores develop and lead to fatal septicemia. Nephritis may cause death in a few cases. The mortality is about 80 per cent. There are, however, great variations in this regard. Some years the disease seems more severe than others. Not a few cases recover from the azoturia but are left lame in one or both hind limbs, due to a quadriceps paralysis commonly called "azoturia drop." A rapid atrophy of the affected muscles takes place from which the patient usually recovers in three to twelve months. The crural muscles or adductors may be similarly affected.

Treatment.—There is no specific treatment. The use of drugs is secondary to proper dietetics and hygiene. The patient should be placed in a well ventilated, clean, light stall and be given plenty of bedding. It is advisable to bolster the horse with straw bundles so that it lie on its sternum. Every three to four hours, if the patient lie on the side, it should be turned over. Where feasible use slings to raise the horse up. Even if it cannot rest in the slings more than a few minutes at a time, relief is afforded in that a better circulation of the blood is induced and a change of the bedding made possible. In the early stage when the patient is thrashing about a good deal, an opiate such as morphin (grs. v subcut.), chloral hydrate (℥j per os.) or cannabis indica (℥j intravenously) is indicated. The bowels should be moved by rectal infusions of water or by the use of arecalin (grs. j

subcut.) or pilocarpin (grs. iij subcut.). Aloes and salts are also employed. The urine should be drawn only if necessary, best by pressing the bladder with the hand, or, in case this does not suffice, by the use of the catheter. The body should be rubbed frequently. Hot applications over the loins and croup are recommended. Blisters, however, are not indicated. Bleeding is of no therapeutic value, but can do no harm. The patient should be given plenty of water in which sulphuric acid (℥vj to a pailful) has been placed. The decubitus should be fought off by frequently shifting the patient and by the use of astringent strew powders, such as compound alum powder, liberally applied. Heart weakness is combated by using excitants such as oil of camphor (℥ss subcut.) or caffein (℥j subcut.). Alcohol and black coffee are also indicated. For threatening paralyses, which may become sequels, use strychnin nitrate (gr. ¼ subcut.).

Digalen, a proprietary preparation of digitalis, has been recommended. The dose is 15 c.c. twice daily. In some cases a single dose sufficed. A subcutaneous injection of morphin is recommended to be administered 20 minutes before digalen is given. Pantopon, a preparation containing the alkaloids of opium in soluble form, may be substituted for the morphin. This treatment has not given very general success although some practitioners still adhere to it.

The food should consist of bran mashes, green food and small quantities of hay.

Prophylaxis.—Horses accustomed to regular work should be exercised when off duty and the food reduced.

PART VI.

DISEASES OF METABOLISM.

CHAPTER I.

DISEASES OF METABOLISM.

DIABETES.

Definition.—Diabetes is a condition marked by habitual discharge of an excessive quantity of urine. Two forms of diabetes are recognized in veterinary medicine, *viz*: (*a*) Diabetes insipidus, and (*b*) diabetes mellitus.

Diabetes Insipidus.—Diabetes insipidus is a chronic disease marked by great thirst and the passage of large quantities of urine with no excess of sugar. The urine is of low specific gravity. Diabetes insipidus should not be confused with polyuria which is transitory and a symptom of some irritation to the kidneys produced by spoiled food (moldy oats), and irritant grasses. During the stage of climax in diseases associated with high fever the exudates and debris of the disease pass out through and irritate the kidneys. In man two forms of diabetes insipidus are recognized from an etiological standpoint, *viz*: Primary or idiopathic which appears to have no organic basis, and secondary or symptomatic in which there is evidence of disease of the brain or some other organ. Whether or not true diabetes insipidus exists in animals is questionable. Polyuria (diuresis), however, does occur and, as noted, is a symptom of irritation of the kidneys due to irritant foods. It may also attend chronic nephritis or appear as the critical polyuria of such infectious diseases as pneumonia, pleuritis, etc.

Diabetes Mellitus.—Definition.—Diabetes mellitus is a disease marked by the passage of an excessive amount of urine containing an excess of grape sugar.

Occurrence.—Except in dogs, where it is not common, the disease is extremely rare in animals.

Etiology.—The causes are not well understood. Heredity, direct injury, severe mental shock, nervous strain and worry are accused in man.

Symptoms.—The principal symptom is a copious secretion of urine with increased thirst. The patients are languid, grow thin but retain a splendid appetite. A chemical analysis of the urine will show it to contain 4–8 per cent. grape sugar. In the advanced stages cataract (c. diabetica) and ulceration of the cornea have been observed.

Course.—The course of the disease is chronic lasting for months but ending fatally. Death may occur suddenly under coma probably due to an intoxication with β-oxybutyric acid which accumulates in the tissues and blood in large quantities and is eliminated in the urine.

Treatment.—The treatment of diabetes mellitus is largely dietary and symptomatic.

GOUT. PODAGRA.

Definition.—Gout is a painful constitutional or diathetic disease with arthritis and an increase of uric acid in the blood. There is a gradual deposition of chalky material (sodium biurate) in and about the joints. The disorder may be acute or chronic. In animals gout occurs only in birds.

OBESITY.

Definition.—Obesity or corpulence is an excessive development of bodily fat occurring principally in the subcutis. It may be hereditary. In animals it is usually due to high feeding and lack of exercise. For edible animals a certain corpulence is desirable. It is also of value in horses as it enhances their appearance and makes them more salable.

Obesity is one of the causes of sterility in the female and

impotency in the male. In these cases it assumes economic importance and requires treatment.

Treatment.—Treatment consists in reducing the quantity and character of the food and allowing the animal plenty of exercise. Foods containing large amounts of carbohydrates and fats should be fed sparingly while those consisting largely of protein may be allowed. Thyroid gland preparations are recommended as iodothyrin (grs. x–xx).

LICKING DISEASE. PICA. ALLOTRIOPHAGY.

Definition.—Licking disease or pica is a perversion of the appetite manifested by a craving for unnatural food. The afflicted animals will lick, gnaw and even eat objects which they would not touch in health. Associated with the symptoms of perverted appetite are nervous nutritive disturbances.

Occurrence.—Licking disease is usually confined to localities or even certain premises. It may sometimes occur enzoötically. It is most commonly seen among cattle which are kept stabled continuously and appears especially during the winter months. It is therefore more common in Europe than in America. Pica is sometimes a forerunner of osteomalacia.

Etiology.—The causes of pica are not known. In some outbreaks it has been shown to be due to spoiled forage and therefore a form of forage poisoning. As pica is so frequently associated with osteomalacia in all probability it may have the same causes and may be the prodromal stage of the same disease.

Symptoms.—The first symptoms are those of indigestion. The appetite is impaired, rumination suppressed and the patient is constipated. The symptoms of perverted appetite begin gradually, the cattle licking the walls, partitions and even the floor of the stable almost continuously. In some cases they gnaw or swallow objects of a various nature, including indigestible and often loathsome things. Soon the patients begin to lose weight, grow thin and eventually emaciate. Finally they become cachectic and usually die in a few months.

Course and Prognosis.—The course is chronic with exacerbations and remissions extending over several months. If the

hygienic and dietary conditions are not changed the result is fatal.

Treatment.—If taken early and before pronounced symptoms of emaciation appear, a change in the food and stable arrangement usually suffices to cure. The cattle should be turned out on pasture if the season permits. If not, the light, ventilation and dampness of the stable should be corrected and a good, well balanced ration fed. Much recommended is apomorphin (grs. ij–iij) three times daily for three days in succession but in some outbreaks it has not given results.

Prevention.—Keeping the cattle out of doors especially on well drained pastures and preventing their eating food which is spoiled will avert the disease.

WOOL EATING.

Definition.—Wool eating is a perversion of the appetite of sheep which causes them to eat each other's wool.

Occurrence.—Wool eating is observed among the finer breeds of sheep when in winter quarters. As it causes loss of wool and a formation of wool balls in the stomach, which may lead to death, it attains economic importance.

Etiology.—Wool eating is chiefly due to foods deficient in nutritive material. Idleness and habit are no doubt predisposing factors.

Symptoms.—In a flock of sheep one or two of the lambs begin nibbling the wool of their mothers, preferably wool which is stained with manure and urine. Soon other lambs and finally the adult sheep take up the habit. Usually one sheep of the flock is chosen to furnish the wool until the supply from this source is exhausted when a new sheep is selected. Ultimately nearly all of the members of the flock become wool eaters. Ordinarily the sheep remain healthy although once in a while lambs die from an occlusion of the stomach openings or intestine.

Diagnosis.—The disease is easily recognized and differentiated from itchy skin diseases and the "trotter disease" by the fact that the sheep do not gnaw their own fleeces, and the absence of skin lesions.

CHAPTER II.

DISEASES OF METABOLISM AFFECTING PRINCIPALLY THE BONES.

RACHITIS. RICKETS.

Definition.—Rachitis or rickets is a disease of young animals characterized by faulty calcification of the growing bones and impaired nutrition.

Occurrence.—Rachitis exists in all parts of the world but is much more common in Europe than in America. It is chiefly seen among young swine and dogs.

Etiology.—The real cause of rachitis is unknown. Want of sunlight, impure air, confinement and lack of exercise are no doubt important predisposing factors. Several theories have been advanced to explain the etiology of the disorder. Briefly stated they are the following:

(*a*) Infection producing a parenchymatous osteitis. The sometimes enzoötic occurrence, postmortem changes and a similarity to the osteitis of phosphorus poisoning lend to this theory some support.

(*b*) Inanition due to feeding foods wanting in lime such as potatoes, sour milk, bran, etc. Rachitis has been experimentally produced in young animals by feeding them foods poor in lime.

(*c*) A disturbance in metabolism which induces an increase in the elimination of lime from the body and a lessening of the amount of lime to the bones.

Necropsy.—Postmortem changes in rachitis are confined largely to the epiphyses. They consist in a chronic hyperemia and inflammation of the bone with abnormal proliferation of the cartilages of the epiphyses. The quantity of lime contained in the bone is deficient. As a result of the proliferation the epiphyses become overdeveloped, the cartilage formed being relatively too great in proportion to the bone. Consequently the epiphyses are enlarged, distorted and the

shafts of the bone bent. The periosteum also proliferates forming periosteal enlargements.

Symptoms.—The symptoms of rachitis are those of deformity of the bone such as enlargement in the region of the joints and bending of the shafts particularly of the bones of the extremities. The patient therefore appears coarse-jointed, bow-legged, or on the other hand cowhocked or knock-kneed. The deformity may also involve the back, causing lordosis (sway back) or kyphosis (roach-back) or scoliosis (bent sideward). The pelvis may also be deformed and the bones of the face thickened and distorted. As usually a chronic rhinitis attends the facial deformity there is nasal discharge with wheezy respirations. This form of rachitis is spoken of as the "sniffle disease" or "the snuffles." Along the course of the ribs at their cartilaginous unions appears a row of nodules. These are spoken of as the "rosary." Occasionally rickets affects the phalangeal articulations causing ring-bone-like enlargements. The patients usually remain stunted, grow pot-bellied and are unthrifty. They are quite often stiff and lame. The shedding of the milk-teeth is postponed, teeth diseases are common and eczemas of the skin frequent. While some of the young animals retain their flesh as a rule they grow thin and emaciated.

Prognosis.—The disease is rarely fatal but often leaves the patient unthrifty and more or less deformed.

Treatment.—One of the commonest treatments for rachitis in animals is phosphorus. It should be given in the form of the oil of phosphorus in small doses (H. & C. gr. $\frac{1}{6}$–$\frac{3}{4}$ and S. gr. $\frac{1}{32}$). For small animals pills (gr. $\frac{1}{100}$) may be substituted. Powdered carbonate of lime fed with milk is also recommended. Usually unless the disease has advanced too far, turning the animal out to pasture and allowing plenty of good nutritious food suffice not only to check but cure it.

OSTEOPOROSIS. OSTEOMALACIA.

Definition.—Osteoporosis is a disease marked by increased softness of the bones so that they become frangible and brittle in consequence of a resorption of the lime content.

Occurrence.—The disease is not uncommon among horses in certain regions of the United States. Along the river valleys of the Middle West numbers of cases occur. On the higher ground and especially in the limestone districts it is of rare occurrence. It is probably more frequently seen among city than country horses. Cattle are also affected especially dairy cows. In Europe the disease seems most common among cattle. It is occasionally seen in sheep and swine. Osteoporosis is a disease of adults resembling rachitis in the young animals.

Etiology.—The causes of osteoporosis are not well understood. It is probably due to infection as the anatomical changes present in the affected bones are of the character of infectious inflammation. Predisposing causes are pregnancy, excessive lactation in cows, lack of lime in the food and soil, darkness, dampness and poor ventilation in the stables.

Necropsy.—The postmortem changes consist in an inflammatory hyperemia with decalcification and softening of the bone and marked dilatation of the Haversian canals. The bones affected are brittle and fracture spontaneously. They are also enlarged, extremely light in weight, the cortical substance abnormally thin and the medullary cavity greatly increased. The medulla appears as a reddish, gelatinous mass. The bones chiefly involved are the pelvis, femur, facial bones and mandible. However, it may affect any bone of the skeleton. Healed fractures are not uncommonly noted.

Symptoms.—While in some cases the affection of the bones is preceded by digestive disturbances and symptoms of vitiated appetite ("licking disease"), most commonly the first symptoms are disturbance in locomotion, the patient becoming lame or stiff and experiencing difficulty in getting up or lying down. If the maxillæ are involved there may be difficulty in mastication. Quite commonly the horse is thought to be suffering from rheumatism which usually affects the stifle or fetlock joints. After rest the symptom of lameness may disappear to recur again, but in a different joint, when the animal is returned to work. In other cases the owner complains of the horse eating slowly and losing flesh and requests that its teeth be dressed. In advanced cases a

prominent symptom is enlargement of one or both rami of the lower jaw or a bulging of the bones of the face ("big head"). Later the patient becomes more and more emaciated, the gait stiff, the flank tucked and finally remains recumbent and unable to regain its feet without assistance. Spontaneous pelvic and limb bone fractures are common in cattle and goats and are not rare in horses, especially if cast for an operation (castration) when fracture of the femur or spinal column often results. More rarely the Achilles tendon tears loose from its attachment to the os calcis.

Diagnosis.—Until enlargements of the bone or spontaneous fractures occur the diagnosis is difficult. In the horse in all cases of obscure lameness resembling rheumatism osteoporosis should be thought of. In cattle pica or licking disease is a common forerunner. Later symptoms of painful mastication, emaciation, difficulty in rising from a recumbent position, stiffness of gait, tucked up abdomen and enlargement of the maxillæ appear, which are indicative of the disorder. In districts where the disease is enzoötic obviously the diagnosis is not so difficult as when a sporadic case is met in a section where osteoporosis is rare.

Prognosis.—Advanced cases are hopeless and should be destroyed. If able to walk they are often rested, which ameliorates the condition, and sold, but usually when returned to work they again grow worse. Sometimes transplanting the case to a district where the disease is not enzoötic is followed by good results. The better the care and food and the lighter the work, as a rule, the longer the patients last.

Course.—The course is chronic and extends from three months to two years from the time the diagnosis is made.

Treatment.—The principal thing in the treatment is to change the environment and food of the patient. Where it is not possible to remove the animal to another district, feeding alfalfa, alfalfa feed and molasses and alfalfa hay have been of great benefit. Medicinal treatment is of little value. Phosphate of lime in the form of bone meal is useful. Or phosphorus (gr. $\frac{1}{6}$–$\frac{3}{4}$) in oil is recommended. For symptoms of "licking disease" in cattle the hydrochlorid of apomorphin (grs. ij–iij subcutaneously) is advised.

PART VII.

DISEASES OF THE ORGANS OF LOCOMOTION.

MUSCULAR RHEUMATISM.

Definition.—Muscular rheumatism is a form of myositis.

Occurrence.—It is not common in animals. In large animals it is most frequent in horses and cattle.

Etiology.—The causes of muscular rheumatism are unknown. Cold and dampness combined are important but probably only predisposing factors. In all probability the immediate cause is either an infection or an intoxication although the absence of fever and localization in individual muscles rather speak against this theory.

Necropsy.—On necropsy the muscles attacked are hyperemic, hemorrhagic, and show serous exudate and cellular infiltration. In some cases there is cloudy swelling and fatty degeneration of the muscle fibers. In chronic cases a proliferation of connective tissue occurs in the muscles attacked.

Symptoms.—Characteristic of rheumatism is a suddenly appearing, painful condition of certain muscles which usually follows exposure to cold. There is a tendency for the pain to shift from one muscular group to another. The inflammation disappears often as suddenly and mysteriously as it came but there is always a tendency to relapses. On palpation the affected muscles feel tense and are very sensitive. Depending upon the location of the inflammation different forms of rheumatism are distinguished clinically:

(*a*) Shoulder rheumatism (omalgia, which affects the biceps, brachiocephalicus and the supra- and infraspinati muscles causing in horses shoulder lameness of the swinging-leg type.

(*b*) Lumbago. Rheumatism of the muscles of the loins particularly of the psoas group producing paraplegia in the horse, characterized by stiffness of the back and weakness or paralysis of the hind parts.

(*c*) Torticollis. A rheumatic torticollis (myalgia cervicalis) produces a stiffness and bending of the neck. The splenius, trapezius and brachiocephalicus muscles are principally involved.

(*d*) Intercostal rheumatism which produces pleurodynia. Pleurodynia in horses frequently follows long railway journeys. It is characterized by dyspnea and sensitiveness of the intercostal muscles.

Among other locations for rheumatism may be mentioned the region of the hip (hip lameness), masseter muscles (disturbance in mastication) and abdominal muscles (constipation). Sometimes muscular rheumatism is generalized affecting practically all of the muscles of the body (polymyositis). If the case is severe and attended by fever, death may ensue. Frequently as the result of chronic rheumatism the animal is left permanently lame in the shoulder or back.

Treatment.—Rheumatism is usually treated by local applications, such as massaging the affected muscles with soap liniment or by applying heat. To relieve intense pain narcotics are useful. Preparations of salicylic acid or salicylate of soda (℥ij) are of some value. For chronic, rheumatic, shoulder lameness the following prescription is recommended:

℞—Veratrin gr. vij
Alcohol. diluti ℥ij
M. f. sol.
Sig.—Inject 5 c.c. every twelve hours subcut. over region of shoulder.

ARTICULAR RHEUMATISM.

Definition.—Articular rheumatism is undoubtedly an infectious disease. It affects the joints producing in them a serous or serofibrinous inflammation.

Etiology.—While the causes are not known very probably staphylococci and streptococci are the chief offenders. Cold is never more than a predisposing cause. The germs enter

the blood either through the throat (in man the tonsils) or in cattle through the puerperal uterine mucosa. The post-mortem lesions are those of a serous and serofibrinous arthritis which in chronic cases develops into an arthritis chronica deformans.

Symptoms.—Articular rheumatism is rare in animals as compared with man. Cattle are frequently attacked, horses rarely. The most important symptoms are found in the joints, tendon sheaths, and the heart. The joint symptoms usually begin suddenly with a painful, hot swelling of one or more joints accompanied by severe lameness or inability to stand. The joints most commonly affected are the stifle, fetlock, hock and front knee. One peculiarity of the arthritis is the tendency to shift suddenly from one joint to another and the occurrence of relapses. The tendon sheaths in the neighborhood of the affected joints are frequently involved particularly the sheaths of the perforans and perforatus, a painful, hot, fluctuating swelling appearing along the course of the tendons. Endocarditis is a common complication and is therefore very characteristic of this form of rheumatism. Through an ulcerous endocarditis death may occur suddenly. Usually, however, it appears as a verrucous endocarditis leading to valvular disease of the heart. The temperature is elevated, appetite and rumination suppressed, which, together with the fever and pain, lead to rapid emaciation. Rare complications are metastatic pleuritis, peritonitis and pericarditis.

Diagnosis.—Articular rheumatism may be confused with any other arthritis. Most commonly it is mistaken for traumatic arthritis. However, in these cases, unless suppurative, there is no fever and no general disturbance. Tubercular arthritis, which is more common, affects only one joint and is usually associated with tuberculosis of other organs. In adults osteomalacia and in young animals rachitis should be thought of. Here, however, other symptoms of these diseases are also present, the process is less acute, does not shift and usually several animals are similarly attacked in a herd.

Course.—While some cases heal in two or three weeks there is always a tendency to relapse. The course in articular

rheumatism in animals is usually chronic and the prognosis unfavorable. The disease generally lasts several months with exacerbations and remissions. Many of the patients unable to stand die of decubitus. Others which recover from the acute attacks are left with an incurable deforming arthritis with contracture and anchylosis of the joint and great muscular atrophy.

Treatment.—As a specific treatment for articular rheumatism salicylate of soda has been highly recommended. It should be given in very large doses. In the horse a daily dose may be as high as three ounces usually given in one ounce doses, three times daily. Other remedies are acetanilid (ʒj) and salol (ʒj). Local applications of heat to joints relieve pain and assist somewhat in resorption. Antiphlogistin is useful in this regard where it can be applied.

TRICHINOSIS.

Definition.—A disease of swine and more rarely of other animals due to the presence of the larvæ of the Trichina spiralis. This parasite is harbored by swine in both the adult and larval forms. The adult worms live in the bowels, the larvæ in the muscles.

Occurrence.—Trichina are very common in swine. Probably 5 per cent. of American hogs harbor the parasite. Occasionally outbreaks of trichinosis occur in man from eating the flesh of the hog which has not been thoroughly cooked.

Mode of Infestation.—Swine are generally infested by eating rats, which very commonly harbor trichina, or the carcasses or offal of infested swine. In a few isolated instances horses have been infested through rats. Mice can also be the host of trichina. In the life cycle of this parasite four stages of development are recognized:

(*a*) The larvæ which develop in the intestine into sexually mature males and females. One week after infestation the females bear living embryos.

(*b*) The embryos wander into the muscles passing through the chyle vessels into the thoracic duct and from there through the bloodvessels to the muscles.

(*c*) The embryos in about one month become encapsuled which process lasts about three months.

(*d*) A calcification of the encapsuled trichina begins in from three to six months and lasts about eighteen months. Encapsuled trichina can live for years in the muscle. They produce infestation when ingested; unencapsuled trichina are killed in the stomach. The favorite seats of trichina are the muscular portion of the diaphragm, the larynx and tongue, abdominal and intercostal muscles.

Symptoms.—Symptoms of trichinosis in swine from natural infestation have not been observed. From artificial infestation the symptoms resulting are diarrhea and colic at the end of the first week, and stiffness, paralysis of the limbs, pruritis, difficult mastication, dysphagia, dyspnea, hoarseness, and edematous swellings in the second to third week. Usually, however, these cases fully recover in from four to six weeks.

Treatment.—No treatment is of any avail once the worms have reached the muscles. As a prevention the hog yards should be kept free from rats and mice.

HOG MEASLES. CYSTICERCUS CELLULOSÆ.

Definition.—Cysticercus cellulosæ is the juvenile form of the tapeworm Tænia solium of man. Young swine are infected by eating the proglottides gathered from human feces. The eggs are digested in the stomach and the six hooked embryos are set free after which they pass through the bowel wall and *via* the blood reach the muscles where after three months they form cysts. The favorite seats of the cysts are in the abdominal muscles, diaphragm and tongue. The cysts are pea- to bean-sized, dull white in color each having an inverted head provided with four suckers and a double row of hooks. Measles is a rare disease among American swine.

Symptoms.—During life symptoms are rarely observed although in isolated instances severe brain disturbance, blindness, paralysis of the tongue, pleuritis and peritonitis have been noted. If the tongue is involved the cysts may be seen on its ventral surface.

CYSTICERCUS INERMIS.

Definition.—Cysticercus inermis is the juvenile form of the Tænia saginata of man. Cattle become infested by ingesting the proglottides of the tapeworm found in human feces. The favorite seat of the cyst is in the masseter muscles. These parasites cause no symptoms in cattle.

MIESCHER'S TUBULES.

Definition.—These are sarcosporidia (protozoa) that appear in colonies surrounded by a sack-like membrane. They occur in the striated muscles forming elongated sacs which contain a number of kidney- or bean-shaped bodies (sporozoites). The favorite seats are the mouth, throat and esophagus. They produce no clinical symptoms. In rare instances the sac surrounding them may rupture and lead to an invasion of the muscles producing a myositis. This occurs usually in the tongue; or they may induce neoformations in the lumbar muscles.

PART VIII.

DISEASES OF THE KIDNEYS.

INFLAMMATION OF THE KIDNEYS. NEPHRITIS.

WHILE from a pathological standpoint a great many different varieties of nephritis may be distinguished, from the clinical side four forms are recognized in animals:

(*a*) Acute parenchymatous.
(*b*) Chronic interstitial.
(*c*) Purulent.
(*d*) Pyelonephritis.

Acute Parenchymatous Nephritis.—Definition.—An inflammation of the functional tissue of the kidneys.

Occurrence.—This form of nephritis is the most common in animals. It may be primary and due to the action of toxic substances or perhaps cold on the kidneys, or secondary to infectious diseases.

Etiology.—Irritants, such as cantharides, mustard, colchicum, carbolic acid, gasoline, mercury, cotton-seed meal, fungi, free hemoglobin, etc., are the commonest causes of primary nephritis. Cold is probably also a cause or at least a refrigeration of the body can predispose an animal to nephritis. Most cases of nephritis in animals, however, are secondary to such infectious diseases as influenza, hog-cholera, glanders or tuberculosis. It frequently complicates azoturia, probably due to the action of the free hemoglobin on the kidney. Nephritis may further be due to an inflammation of the renal pelvis or urinary bladder (pyelitis, cystitis). In rare instances nephritis may be caused by traumatism or still more rarely attends rheumatism. Diffuse skin diseases

such as eczema and mange may cause nephritis. Obstructive icterus may also be a cause.

Symptoms.—The principal symptom of acute parenchymatous nephritis is albuminuria. The albumin content may reach several per cent. Usually the quantity of urine voided is greatly diminished (oliguria), the specific gravity high, color dark, and, as a rule, it is rich in sediment. An examination of the urine under the microscope shows it to contain tube casts, renal epithelium, white or red corpuscles and in some cases blood (hematuria). Later symptoms of dropsy appear, such as edema of the ventral portion of the abdomen, scrotum, and legs. In some instances the patient will appear stiff, stand with its legs spread apart, or, on the other hand, drawn up under the body and the back is held arched. Palpation of the kidneys through the rectum (small horses) causes the animal to evince pain. Pressure over the kidneys from the outside rarely produces symptoms. The general condition of the patient is disturbed in that it seems stupid, weak, has no appetite and shows increase in temperature. In some cases the urine is voided frequently in small quantities, often only a few drops dribbling away with considerable straining (stranguria). These symptoms are most commonly seen in diffuse nephritis the result of poisoning with gasoline, turpentine, or cantharides. In stallions there may be a partial erection of the penis (priapism) from the urethral irritation. In some instances there is a total suppression of urine for as long as a week (anuria). In fatal cases toward the end symptoms of uremia appear which are recognized by convulsions, coma, and usually subnormal temperature.

Diagnosis.—A diagnosis can be made positively only by a chemical and microscopic examination of the urine for albumin, the presence of tube casts, renal epithelium, red and white corpuscles, and even blood.

Course.—Cases of nephritis secondary to infectious diseases usually disappear with the recovery of the infectious disease, healing occurring in about fourteen days. Cases which end fatally, as noted, terminate under symptoms of uremia.

Prognosis.—The prognosis is always doubtful. While apparently severe cases do recover, milder ones often grow

worse and lead to death. When oliguria or complete anuria persists and the chemical and microscopic findings continue to show evidence of further kidney destruction the prognosis is bad.

Treatment.—Care should be given to regulate the diet, avoiding feeding acrid and irritant substances. Herbivorous animals should be allowed grass, hay, and roots rather than intensive foods like grain. When available, milk forms a good article of food. Plenty of water should be supplied the patient, as it tends to flush out the obstructed tubules. Linseed tea is recommended for its soothing effect. The indications are to relieve the kidneys as much as possible by utilizing the skin and bowels to rid the body of waste products. Among the drugs diuretics are indicated, such as acetate of potash (℥ij to iv) or potassium nitrate (℥ij to iv). Diaphoretics also assist, such as pilocarpin (grs. iij to vj). Keeping the patient in a warm place, hot baths, or blankets assist in producing sweat. To further relieve the kidneys laxatives should be given. When the patient shows symptoms of uremia narcotics, such as morphin (grs. iij to v) or bromides, are useful.

Chronic Interstitial Nephritis.—**Definition.**—A form of inflammation of the kidneys which attacks principally the interstitial connective tissue. The chronic fibrous inflammation leads to a contraction and hardening of the kidney, the surface of which becomes rough, uneven, covered with numerous projections and depressions leading to what is known as granular kidney. Within the kidney are numerous centers of connective tissue. The cortex is contracted causing the tubular portions to be drawn nearer the surface of the organ. This is called sclerotic kidney (nephritis fibrosa multiple). When the sclerosis is diffuse the organ is greatly increased in size, is of semicartilaginous consistency and of white color (nephritis fibrosa diffusa).

Occurrence.—Chronic interstitial nephritis is less common in animals than in man. It does occur, however, in horses and occasionally in cattle. In swine, it is rare, as these animals are usually killed when young.

Etiology.—This form of nephritis usually develops from the acute parenchymatous, the causes of which have been given. In the horse it may have an embolic origin emanating from the worm aneurysm in the anterior mesenteric trunk. In cattle it sometimes results from pyelitis. In man, sclerosis of the arteries is a common cause.

Symptoms.—The principal symptom of chronic interstitial nephritis, contrary to the acute and chronic parenchymatous nephritis, is an increase in the quantity of urine voided by the patient (polyuria). The urine is of low specific gravity (1001 to 1010) and contains little albumin, few tube casts and renal epithelium. Generally associated with the polyuria are symptoms of hypertrophy of the heart (see this), which is later followed by dilatation of the organ, leading to symptoms of dropsy, such as anasarca, hydrothorax, hydropericardium and ascites. In some cases uremia may follow with convulsions and coma. In rare instances blindness results from albuminuria retinitis.

Prognosis.—The prognosis is bad as the condition is generally incurable. As the efficiency of the patient (horses) falls below the cost of keeping, many of them are destroyed or traded off.

Treatment.—While in man a palliative treatment which will prolong life is indicated (nitroglycerin, digitalis, strychnin) in animals this does not pay. Otherwise the treatment is the same as for acute parenchymatous nephritis.

Purulent Nephritis.—Definition.—A suppurative inflammation of the kidneys leading to diffuse pus infiltration or to abscess formation in the organ.

Occurrence.—This form of nephritis is usually secondary to such diseases as strangles, pyemia, wound infections, etc.

Etiology.—The causes are pus-producing bacteria which are carried to the kidney either through the blood or come from the bladder or pelvis of the kidney through the urine. Specifically staphylococci and streptococci are the principal causes. These bacteria produce in the organ either a disseminated, purulent nephritis in the form of large numbers of small abscesses, found principally in the cortex (nephritis punctata), or a few isolated large abscesses (pyonephrosis).

In cattle this form of nephritis is most commonly a sequel to puerperal septicemia and in the horse to strangles, pyemia and more rarely purpura.

Symptoms.—The symptoms of diffuse, purulent nephritis are usually so vague that a diagnosis cannot be made during life. Where symptoms occur that are at all characteristic, both kidneys are involved. The symptoms are very like those of acute parenchymatous nephritis (in the horse colic attacks, arching and rigidity of the back, shortening of the stride in one or both hind limbs, etc.). Cases are recorded where the enlarged kidney produced swelling in the lumbar region. On microscopic examination pus cells may be found. Albuminuria is also present.

Prognosis.—The prognosis is grave as sooner or later, if both kidneys are involved, death ensues (uremia).

Treatment.—Medicinal treatment in animals is of no value as far as producing healing is concerned. In man the removal of the kidney (nephrectomy) is practised.

Pyelonephritis.—Definition.—Pyelitis is an inflammation of the pelvis of the kidney. Pyelonephritis is a combination of pyelitis and nephritis.

Occurrence.—In Europe pyelonephritis is common in cattle (cows). A few cases have been recorded in swine. Statistics for this country in regard to the prevalency of the disease are wanting.

Etiology.—Pyelonephritis may develop in one of two ways: (*a*) As a bacterial disease which is the most common form in cattle, or (*b*) it may be caused by stones or concrements which form in the pelvis of the kidney. A rare cause of pyelonephritis is the giant palisade worm, Eustrongylus gigas.

Bacterial Pyelonephritis of Cattle.—As noted this is the most common inflammation of the kidney in the ox. It is probably due to more than one microörganism (streptococci, colon bacilli, staphylococci), but the corynebacillus renalis is the chief offender. The microörganisms enter the kidney either by the blood or the urine. The bacteria which produce pyelonephritis cause a variety of pathological changes in the kidney. As a rule there are present in combination dilatation of the pelvis of the kidney, which is usually found filled

with pus and its walls ulcerated, necrosis of the papilla, diffuse, purulent nephritis with abscesses in the cortex and chronic interstitial nephritis.

Symptoms.—Most cases of pyelonephritis occur in cows following parturition. This is especially true when injuries have occurred in the uterus or vagina or where retention of the afterbirth has followed. It may, however, originate independent of parturition. In sucklings infection through the navel can occur and in male animals it has resulted from primary abscesses. There is also a probability that the infection may be introduced *via* the digestive tract. The clinical symptoms are usually rather indefinite. In a cow which has calved with difficulty or suffered from retention of the placenta the condition of the animal grows bad, the appetite and rumination suppressed, the patient losing flesh, showing fever, colicky pains, irregular gait, frequent urination and sometimes strangury. Pressure over the sacrum causes pain and the tail and buttocks are soiled with pus which flows from the vagina. The urine in rare instances remains clear, but is usually cloudy. On rectal examination the bladder is found partially filled, the ureters dilated and sometimes the kidneys enlarged, sensitive and fluctuating. The chemical analysis of the urine shows albumin and free ammonia. Under the microscope pus cells, blood, crystals of triple phosphates, and renal epithelial cells are found. With the Gram stain large numbers of bacteria—the corynebacilli, are found generally arranged in clumps. As cystitis is a common complication, bladder epithelium will also be found in the urine.

Diagnosis.—Diagnosis depends upon the examination of the urine and the determination of the corynebacillus which is usually present. The rectal findings, the fact that the urine contains pus, and the failing of the patient after parturition, point to pyelonephritis in cows.

Prognosis.—Prognosis is bad, therefore it is advisable to make the diagnosis as early as possible so that the animal may be slaughtered.

Calculous Pyelonephritis (*Kidney Stones*).—*Definition.*—This is an inflammation of the pelvis of the kidney due to

the presence of renal stones (nephrolithiasis). The stones consist of carbonate of lime, oxalate of lime, silicates and phosphate salts. Renal stones probably develop from the deposit of salts around a nucleus, such as mucous epithelium or even bacteria. They induce in the kidneys various changes such as pyelitis or pyelonephritis and the so-called hydronephrosis.

Occurrence.—While kidney stones are rarer in animals than in man, they are seen occasionally in horses and cattle. They also occur among sheep and swine. In sheep they commonly result from the feeding of root crops, such as potatoes, sugar beets, beets, etc. In some instances among sheep, kidney stones appear enzoötically.

Symptoms.—The symptoms are rather vague. Very often they produce no symptoms whatever. In other cases the patient suffers from renal colic, bloody urine, partial or total suppression of urine, and even uremia. The urine passed is sometimes bloody or may be cloudy from pus. Sometimes sand or grit is passed, which adheres to the hairs around the sheath opening. On rectal examination the symptoms of pyelonephritis may be determined, and occasionally a slight crepitation can be felt, due to the movement of the stones against one another.

Treatment.—The treatment in animals is not successful, and operative procedure, except in dogs, is hardly advisable on account of the risk.

UREMIA.

Definition.—A poisoning of the blood with the constituents of the urine.

Occurrence.—Uremia can occur in any condition in which the normal discharge of urine is impeded.

Etiology.—Uremia may result from nephritis, kidney tumors, kidney stones, obstruction of the ureters, bladder or urethra, or paralysis of the bladder.

Symptoms.—The symptoms usually follow kidney disease where there has been complete retention of urine. The patients show spasms, weakness, coma and subnormal temperature. The respirations are generally retarded, and the sweat may have a urinous odor, especially after rupture

of the bladder. The symptoms, as a rule, occur periodically and very often lead to death, which may follow the first convulsive attack or after repeated attacks.

Treatment.—Unless the cause of the retention can be removed, treatment is useless. The remedies advised in acute nephritis (diuretics, diaphoretics, laxatives) may be employed.

CONGESTION OF THE KIDNEYS. HYPEREMIA.

Definition.—Hyperemia of the kidneys may be either arterial or venous. Arterial hyperemia is the first stage of nephritis (see this). It is caused by such irritant drugs as cantharides, turpentine, or gasoline. Acrid plants, moldy food, sea-water, etc., can also produce it. Venous congestion is usually due to organic heart disease and more rarely to pulmonary emphysema.

Symptoms.—The principal symptom of arterial congestion is polyuria, with its attending increased thirst. Sometimes the patient shows a stiff gait and sensitiveness over the kidneys. In venous congestion, due to the fact that less arterial blood flows through the kidneys, oliguria occurs. Albuminuria may also be a symptom, due to the fact that the nutrition of the renal epithelial cells suffers.

Diagnosis.—Arterial congestion can be differentiated from diabetes insipidus only by the fact that it is temporary while the latter is chronic. Diabetes mellitus, on account of its extreme rarity in animals, need hardly be taken into account. Venous hyperemia is differentiated from nephritis by the urine, which contains very little albumin and no cell elements. The patient is at the same time suffering from some chronic heart or lung disease.

Treatment.—Arterial congestion disappears as soon as the causes are removed. Otherwise the treatment is similar to that of acute nephritis. Venous hyperemia can rarely be removed, as the basic disease is usually incurable.

RENAL HEMORRHAGE.

Etiology.—Hemorrhage from the kidneys may result from traumatism or the worm aneurysm, or it may be embolic in

origin. It is quite often fatal in horses, especially when traumatic.

Symptoms.—The symptoms are hematuria, blood-tube casts in the urine and general anemia.

AMYLOID KIDNEY.

Amyloid kidney is usually associated with chronic suppurative conditions such as are seen in strangles, liver abscesses, etc. It is of no clinical importance in veterinary medicine.

Symptoms.—The symptoms are very vague (anemia, cachexia, albuminuria without tube casts).

KIDNEY TUMORS.

The kidneys are the seat of several kinds of tumors, such as sarcoma, carcinoma, adenoma, melanoma, etc. They can rarely be diagnosed clinically. Occasionally they may be palpated through the rectum. If kidney tumors produce symptoms they are hematuria, uremia, emaciation and intermittent lameness from compression and thrombosis of the posterior aorta.

Treatment.—Treatment is of no use except in dogs, where occasionally nephrectomy is practised.

HYDRONEPHROSIS. CYSTIC KIDNEY.

Etiology.—This condition is found frequently in edible animals. Cystic kidney is usually due to kidney stones, especially in sheep, where 80 per cent. of the cases are due to this cause. In the hog it is said to result from a congenital defect in the opening of the ureters, which are placed too low at their point of entrance into the bladder, periodically preventing the urine from escaping.

Symptoms.—Cystic kidney rarely produces symptoms during life, although very rarely a kidney may become so enlarged as to distend the abdomen in swine. Occasionally in horses and cattle the condition may be palpated per rectum.

Treatment.—Treatment is unavailing.

HEMATURIA.

Definition.—By hematuria is meant blood in the urine. This is a symptom of several diseases of the kidney, bladder and urethra. It is also associated with infectious diseases (purpura, anthrax), poisoning (turpentine, gasoline) and blood diseases (leukemia).

HEMOGLOBINURIA.

Definition.—The presence of hemoglobin without blood in the urine. It is a symptom of several different diseases (azoturia, Texas fever, etc.).

PARASITES IN THE KIDNEY.

Eustrongylus Gigas.—This parasite is rare in horses and cattle. It is a large worm about the diameter of a lead-pencil, varying in length, depending upon the sex, from 13 cm. to 1 m., the male being the smaller. It is not known how the worm enters the pelvis of the kidney. It produces a suppurative pyelonephritis, and eventually total destruction of the kidney, which is transformed into a thick-walled sac containing pus and the coiled worm. In large animals the symptoms are very vague, as usually but one kidney is involved.

A diagnosis can be made only by finding the brown-colored oval eggs, showing on the surface numerous round depressions. The worm may occasionally pass through the ureters to the bladder, where it produces cystitis. Treatment is not satisfactory in large animals.

Other Parasites in the Kidneys.—The larvæ of the sclerostomum are found in the kidneys of the horse, where they give rise to hemorrhage from the renal arteries. In swine the Stephanurus dentatum is not uncommon in the fat (leaf lard) surrounding the kidney, and more rarely in the kidney itself. It is usually found on slaughter. Cysticercus cellulosæ has been found in the kidney of pigs; Echinococcus polymorphus in the kidney of sheep.

PART IX.

DISEASES OF THE NERVOUS SYSTEM.

CHAPTER I.

DISEASES OF THE BRAIN.

Two groups of symptoms characterize brain and meningeal lesions, *viz.*: A. General, and B. focal or topical symptoms. The general symptoms result from a diffuse disorder of the brain cortex with increased intracranial pressure, or they may arise from increased intracranial pressure alone. The focal symptoms result from lesions which involve well-defined fields, centers, or tracts of the brain, the functions of which are interfered with. Both groups of symptoms can occur simultaneously or each independently of the other.

A. General Brain Symptoms. These consist in: (1) Disturbed consciousness. This is manifested by stupor, dulness, sleepiness, sopor, or even coma; vertigo and syncope may occur. On the other hand there may be excitement, restlessness, maniacal or rabiform manifestations. (2) Disturbance in respirations. The respirations are affected in frequency and rhythm, producing in some instances a change in the mode of breathing (Cheyne-Stokes, Biot respirations, etc.). (3) Disturbance in the manner of prehension and mastication of food. The appetite may be impaired, lost or vitiated, and food and water are taken in an unphysiological manner. (4) Abnormal muscular movements. These may be expressed in general

tonoclonic spasms (epileptoid), forced movements, or there may be paralysis (general, hemiplegia, etc.). (5) Changes in the pulse. Variations in the frequency and rhythm of the pulse are observed. Quite often the pulse is slow, due to the effect of increased intracranial pressure upon the vagus. If excitement, fever or heart weakness be present, however, the pulse will be fast and irregular. Marked variations in the frequency and rhythm of the pulse are noted in acute meningitis. (6) Eye symptoms. These are manifested by contraction or, on the other hand, marked dilatation of the pupils. One pupil may be dilated, its fellow contracted. Rolling of the eyeballs (nystagmus) and amaurosis are not unrarely observed. (7) Tendon, skin and pupil reflexes. The tendon reflexes may be increased, especially in chronic brain diseases, if the inhibitory action of the cortex is out of function. The reflexes may be reduced in some acute brain diseases where the inhibitory function of the cortex becomes on the contrary increased. When coma is present both the skin reflex and pupil reactions become nil and the tendon reflex (patellar, Achilles) reduced to a minimum.

B. Focal or Topical Symptoms. The knowledge of topical symptoms in animals is very limited. Localized nervous diseases in animals are, however, much rarer than in man. Topical symptoms produce, generally speaking, sensory or motor disturbances.

The sensory disturbance consists in (*a*) decreased sensibility or hypesthesia; (*b*) absence of sensibility or anesthesia; (*c*) increased sensibility or hyperesthesia.

The motor disturbance induces (*a*) spasms which may be clonic or tonic; (*b*) involuntary (forced or unphysiological) movements; (*c*) disturbance of the muscular sense, causing loss of coördination, unphysiological attitudes, and irregularities in gait, or paralysis, which may be complete or partial (paresis), unilateral (hemiplegia), bilateral (paraplegia), or in a single organ or part (monoplegia). Hemiplegia has its origin in the brain, paraplegia in the spinal cord, and monoplegia may have a central (brain) origin, or may be due to disorder of peripheral nerves.

ANEMIA OF THE BRAIN AND ITS MEMBRANES.

Etiology.—Where the body has suffered a sudden loss of a quantity of blood, or where a rush of blood from the brain to other organs occurs, an acute anemia of the brain results. Examples of the latter instance are furnished when large quantities of exudate or transudate are removed too rapidly from a body cavity (hydrothorax, ascites) or large amounts of gas from the bowels. Acute heart weakness or a general dilatation of the bloodvessels, as is sometimes associated with severe infections and many poisons, will also produce acute anemia.

A chronic anemia of the brain accompanies general anemia, specific diseases of the blood (leukemia), increased intracranial pressure, and very rarely results from compression or thrombosis of the carotids.

Symptoms.—Acute anemia of the brain produces disturbance in consciousness. The animal walks with a staggering gait, finally falling to the ground as if lifeless. Vomiting animals may vomit. The mucous membranes are very pale, the pupils dilated, the pulse rapid and threadlike, the respirations either slow and deep or accelerated and superficial. In severe cases the syncope may be interrupted by convulsions and the death of the patient. In milder cases the animal returns gradually to consciousness, remains for a time stupid and languid, and with a tendency to relapses.

Chronic brain anemia usually produces no symptoms, as the condition develops gradually, the brain adapting itself to it.

Prognosis.—The prognosis varies with the cause. In acute cases as long as the pupil reacts to light recovery is probable. On the other hand, if there is no reaction, and especially if the patient shows symptoms of convulsions, the prognosis is bad.

Treatment.—Stimulating agents are recommended, such as rubbing the surface of the body; allowing the patient to inhale fumes of ammonia; subcutaneous injections of ether, caffein, or oil of camphor; internally alcohol or black coffee.

In small animals the electric battery may be useful. In chronic cases only a successful treatment of the basic disease will heal the brain anemia.

CONGESTION OF THE BRAIN AND ITS MEMBRANES.

Brain congestion may be active, due to an engorgement of the brain with arterial blood, or it may be passive, caused by a stoppage of the outflow of venous blood.

Etiology.—Passive congestion rarely produces symptoms in animals. An active hyperemia of the brain can be caused by an increased heart activity and the loss of tonus in the cerebral arteries due to overwork, rough treatment of young animals (breaking colts; the excitement of railway or ship transportation); estrum; fright; hypertrophy of the heart; acute alcohol poisoning; sunstroke and heatstroke. A collateral congestion may result from compression of the large bloodvessels of the abdomen in severe bloating of the stomach or bowels. An active hyperemia of the brain is the first stage of inflammation.

A passive hyperemia occasionally occurs from compression of the jugulars, due to ill-fitting collars, too tight throat latches ("choking down" of horses), tumors, inflammatory swellings or enlarged thyroids which press upon the jugulars. It may also be a symptom of heart weakness, chronic diseases of the lung, or compression of the lung from gas accumulation in the stomach or bowels.

Symptoms.—The principal symptom of active hyperemia is excitement, which usually is soon followed by a stage of depression. The pupils are dilated, the mucous membrane of the head congested, the pulse and respirations are increased in frequency, and the poll feels warm. Very probably, however, these symptoms represent a transient inflammation of the brain often the result of a chronic hydrocephalus, which occasionally "flares up" in this form.

A severe passive hyperemia causes the animal to show stupor, sopor, the mucous membranes cyanotic, the pulse small and rapid, and the patient dyspneic.

Course.—In primary active hyperemia the symptoms disappear suddenly in a few hours, or they may subside gradually. In the passive form the symptoms disappear as soon as the causes are removed. Where this is impossible the patient will be subject to repeated attacks, and eventually the condition may lead to the animal becoming a "dummy."

Diagnosis.—The diagnosis largely depends upon the short duration of the symptoms. Obviously we should take into consideration the species, age and condition of the patient. There are many conditions which simulate and are probably accompanied by cerebral congestion (nymphomania, infectious diseases, gastro-intestinal troubles, especially forage poisoning).

Treatment.—The patient should be kept in a cool, quiet place and fed easily digested food. Bleeding from the jugular and cold applications to the poll are recommended. Internally a good purge should be given. Passive hyperemia due to ill-fitting collars demands a change in the harness. Cases of heart weakness should be treated.

SUNSTROKE AND HEATSTROKE.

Sunstroke.—Sunstroke is a disorder of the brain and spinal cord produced by exposure to the direct rays of a hot sun acting upon the head. From a pathological standpoint it may be, depending on the degree, a congestion, hemorrhage, inflammation, or paralysis of the brain. Therefore the patient will show varied symptoms, such as excitement, raging, spasms, or death may occur suddenly from apoplexy or respiratory arrest.

Treatment.—Treatment consists in the application of cold to the head and the use of caffein, camphor or veratrin. To relieve the bowels arecalin or eserin may be used.

Heatstroke.—Heatstroke is produced by prolonged exposure to high temperatures, especially during exercise. It is seen most commonly in swine that are being driven or horses overworked in hot weather.

Symptoms.—The symptoms are those of heart weakness (palpitation, rapid, weak pulse, dyspnea and cyanosis). The

patient is usually very languid, the gait staggering, and there is a profuse outbreak of sweat. Temperature may reach 110° F. or over.

Prognosis.—The prognosis is bad, the animal usually dying of asphyxia.

Treatment.—Heatstroke is treated much the same as sunstroke. Atropin, caffein and camphor are used with cold applications (hosing) to the body. To relieve the dyspnea, due to edema of the lungs, bleeding may be resorted to. The temperature may be reduced by cold enemas.

TRAUMATIC INJURY AND CONCUSSION OF THE BRAIN.

Definition.—A bruising of the brain the result of direct injury to the cranium or other part of the head.

Etiology.—In horses contusion and concussion of the brain may result from kicks, blows, collisions during runaways, falling upon the head, etc. In cattle it may be due to the animal falling heavily or being horned by another. The result of the injury is very varied. In some instances a fracture of the skull with hemorrhage results; in others there is a bruising of the brain substance without even the skin showing lesion. Undoubtedly more or less hemorrhage occurs in the brain substance and intracranial tension is increased. The patient also suffers from shock.

Symptoms.—Following violent blows on the head the symptoms usually appear at once; in some cases, however, an hour or two may elapse before they make themselves manifest. The symptoms are generally the result of accumulations of blood in the brain, due to the traumatism. Where the injury has not been great the animal appears stunned, falls to the ground, where it may remain for ten to twenty minutes, to finally regain complete consciousness. Sometimes permanent symptoms may be left behind. When the injury has been severe the animal is found lying unconscious, pupils dilated and reflexes dormant. The respirations are slow and irregular, the pulse usually rapid, but occasionally slow and often arhythmic. Incontinence of feces and urine may occur. Usually after lying uncon-

scious for a few hours the reflexes react to stimuli; twitching of muscles and rolling of the eyeballs appear. The animal finally rises to its feet, and provided no injury to the motor tracts has resulted, it appears normal. In the latter case it may be left paralyzed. In severe cases the patient does not regain consciousness and dies under convulsions.

With the return of consciousness sometimes the animal is left paralyzed; quite commonly the paralysis assumes the form of a hemiplegia. However, depending upon what motor tracts are involved, the form of paralysis will vary.

Diagnosis.—When the history of injury is clear the diagnosis is not difficult. On the other hand if there is no such history and no lesions about the head can be noted, a contusion might easily be confused with cerebral hemorrhage (apoplexy) or inflammation of the brain. The sudden appearance of severe brain symptoms without fever is significant in this regard.

Prognosis.—Contusion of the brain in most cases leads directly or indirectly to the death of the patient. Obviously mild cases, when the animal has been only stunned, recover. Many patients which do not die are left permanently crippled through resulting paralysis.

Treatment.—The head of the patient should be elevated and cold applied to the poll. Excitants, such as ether or camphor, may be used internally. After return to consciousness the use of deep bedding or slings may be recommendable, depending upon the individual case.

LIGHTNING STROKE. ELECTRIC STROKE.

By lightning or electric stroke we understand an injury to nerve tissue produced by powerful currents of electricity. In some instances no pathological change is found in the tissue, the condition being functional.

Occurrence.—Lightning stroke commonly occurs in the open country, animals on pasture being victims. In the city horses occasionally come in contact with high-tension wires which have fallen upon the street. During the sum-

mer season thousands of animals are killed or injured annually by lightning on the farms of this country.

Symptoms.—Usually a lightning stroke kills the animal either instantly or in a few minutes. In other cases the animal appears stunned from the stroke, but soon regains consciousness and normal condition. In a few instances, however, the patient is left for a time with irregular gait, weakness of the hind parts, may show forced movements, and appear stupid and dull. Usually they recover after a few days or weeks. In rare cases topical symptoms are retained, such as monoplegia, paraplegia, paralysis of individual nerves, from which the animal usually recovers in one or two months. Horses are sometimes left permanently blind.

Occasionally lightning stroke produces peculiar markings or figures on the hair or skin. These figures are often branched and forked, and may involve pigmented as well as unpigmented skin. On the unpigmented skin they are dark-colored and sometimes resemble a tree or forked shrub.

Treatment.—If the patients remain down good bedding should be provided. To assist the return to consciousness excitants (alcohol, ether, camphor, caffein, skin rubbing) may be tried. Usually treatment is unnecessary.

HEMORRHAGE IN THE BRAIN AND ITS MEMBRANES. APOPLEXY.

Definition.—By apoplexy in a narrow sense is understood a hemorrhage of the brain or its membranes which is due neither to traumatism nor inflammation.

Etiology.—The causes are very varied. In many of the acute infectious diseases (anthrax, purpura hemorrhagica, hemorrhagic septicemia), blood diseases (anemia, leukemia), and in chronic inflammation of the liver and kidneys the walls of the bloodvessels in the brain become weakened, a condition conducive to hemorrhage. In rarer instances parasites (larvæ of sclerostomes) form a cause. Arteriosclerosis, a common cause of apoplexy in man, probably does not occur in animals.

Symptoms.—If the hemorrhage is severe enough, general brain symptoms appear with which are associated topical symptoms. The gait becomes irregular, the patient may show forced movements, and, eventually, will fall to the ground unconscious and die in convulsions. In other cases the animal later regains consciousness but for an indefinite period shows topical symptoms which may later lead to death.

Diagnosis.—The symptoms are obviously very similar to those noted in contusion of the brain. The diagnosis depends largely upon the sudden appearance of severe disturbance in consciousness without history of injury.

Treatment.—During the stage of unconsciousness the treatment is the same as for contusions and concussion of the brain. The topical symptoms (local paralyses) which remain behind may be treated by passive movements of the paralyzed extremities and the use of the electric battery. Iodid of potash is also recommended.

MENINGO-ENCEPHALITIS.

Definition.—By meningo-encephalitis is meant an inflammation of the pia mater and brain. Throughout the brain substance occur numerous small centers of cell infiltration.

Occurrence.—In a primary form the disease is most common in horses. It is rare in other animals. In the horse it is most apt to occur during the warm season.

Etiology.—The disorder may be primary or secondary. The primary cases are usually the result of infection. Several organisms (micrococci, diplococci) have been accused. Other infectious agents, such as the necrosis bacillus and the Micrococcus ascoformans, have been determined in the meningeal exudate.

In cattle the disease is quite commonly associated with parturition occurring as a puerperal meningitis manifesting itself up to two days before calving. Certain predisposing causes which reduce the resistance of the patient are undoubtedly factors. Therefore, unfavorable weather, working the horse in the hot sunshine, intensive feeding, hot,

illy ventilated stables, overexertion, tying the head too high after operations, etc.

A secondary meningo-encephalitis may follow strangles in the horse or tuberculosis in the ox. Obviously, meningo-encephalitis may also result from traumatism, inflammation of the brain and meninges following an injury. Abscesses in the neighborhood of the cranium (eye socket), necrosis of the atlas, diseases of the middle ear, etc., may lead to an infection of the brain.

In rare cases parasites (sclerostomes, Gastrophilus equi, cœnurus and cysticerci) may be causes.

Symptoms.—The symptoms of brain disturbance usually develop rapidly. The patient appears stupid, languid, the facial expression staring, and the attitude unphysiological. Horses often stand with their forefeet and hind feet drawn together, the head pendent, and the eyelids partially closed. The patient pays little or no attention to its surroundings, does not eat and fails to obey commands. The gait is awkward, stumbling and sometimes the forefeet are lifted as if the horse were wading in water. There are often marked symptoms of cerebral excitement, the patient running about in an aimless fashion not infrequently colliding with the fence, building, or whatever may come in its way. Forced movements are also observed, the animal walking in a circle.

Cattle are restless, look wild, bellow, tear up the earth with their horns and may even attack persons. They finally drop to the ground and are seized with convulsions. In tubercular meningitis symptoms of excitement are usually absent.

Following the stage of excitement which usually lasts not over half an hour the patient goes over into a stage of stupor, seems oblivious of its surroundings, stands with the eyelids half closed, head sunken, chin resting upon the edge of the manger, or quite commonly the head is forced into a corner. The gait is often irregular, awkward, the patient stumbling and falling as it progresses.

While the respirations are accelerated in the stage of excitement, in the second stage they are usually slower

than normal and deeper. Sometimes Cheyne-Stokes respirations have been noted. The pulse may be too rapid or too slow.

During the stage of excitement the sensibility of the patient is increased; later greatly reduced. The poll of the head may feel warm; striking it lightly with a percussion hammer causes the patient to wince. The papilla of the eye is intensely congested.

Topical symptoms in meningo-encephalitis are rare in animals. They consist in spasms of the eye muscles (nystagmus) or a deviation of one of the eyes from its proper direction (strabismus); the pupil may be fixed or react slowly to light; quite frequently the pupils are of unequal size; spasms of the masseter muscles causing gnashing of the teeth or even trismus; the muscles of the lips, nose, ears and neck may also show spasmodic contractions. Paralysis of peripheral parts, such as the pharynx, tongue and the lids, occasionally occurs. Hemiplegia is a rarer consequence.

Fever is usually noted in the beginning of the disease. If the temperature remains high during the latter stages, it is probably due to septic infection or pneumonia which may complicate the disease.

The appetite is impaired or suppressed and the prehension of food unphysiological the animal eating and drinking much as does a "dummy."

Course.—The disease develops in two to three days. It may develop suddenly with symptoms of excitement and violence followed by those of mental depression and stupor the animal dying in less than a day. In other cases the development is much slower the animal showing no very pronounced symptoms but seems mentally perturbed, shows impaired appetite, expressionless countenance, labored locomotion, etc., symptoms which in two to three weeks attain a higher degree. Tubercular meningitis in cattle usually assumes a subacute course and develops slowly. In some cases improvement is followed by a relapse. Meningo-encephalitis is not infrequently complicated by pneumonia (hypostatic or foreign body), septicemia or pyemia.

Diagnosis.—The diagnosis depends upon the history of some infection, the symptoms of rapidly increasing disturbance in consciousness, the eye symptoms (pupils of unequal size, strabismus, nystagmus, congestion of the papilla), trismus, and sensitiveness of the poll. Where these symptoms are vague the diagnosis is extremely difficult.

From the standpoint of differential diagnosis the following conditions must be taken into consideration:

(*a*) Functional disturbances of the brain such as accompany acute, feverish, infectious diseases. These are accompanied by mental depression, disturbance in consciousness, etc. The diagnosis here would depend upon the evidence of the existence of the primary disease, and the fact that the brain symptoms are not as well developed as in meningo-encephalitis.

Meningo-encephalitis might be confused with rabies. In rabies, however, consciousness is not disturbed in the beginning, the clinical symptoms develop progressively and characteristically (melancholia, frenzy, paralysis), and forced movements and spasms fail. Acute encephalitis could only be excluded in cases where topical symptoms appear early (hemiplegia, ataxia, monoplegia, etc.). A tubercular meningitis could only be diagnosed by discovering a tubercular iritis (rare), the evidence of tuberculosis in other organs and the tuberculin test.

(*b*) Poisonings (lead, mercury, brine, santonin, tobacco, poppy leaves, opium, etc.) also produce functional brain disturbances which resemble the symptoms of meningo-encephalitis. Usually the history coupled with the fact that the patients show at the same time gastro-intestinal symptoms suffice for the diagnosis. Brain disturbance is also noted in some cases of helminthiasis and in forage poisoning.

Prognosis.—Except in the puerperal form in cows, which often reacts favorably to proper treatment applied early, meningo-encephalitis is a very fatal disease. The mortality is over 75 per cent. In the horse those cases which do recover are left "dummies" from consequent hydrocephalus.

Furthermore amaurosis, deafness, muscular paralysis frequently follow in the wake of the disease.

Treatment.—The treatment consists in placing the animal in a cool, darkened, well ventilated stall, best in a box stall where it may run free, using short straw for bedding so that its feet will not be entangled. The food should be easily digestible; if the animal cannot eat, rectal feeding may be resorted to. To the poll cold applications may be applied. Internally, provided the animal can swallow, cooling laxatives such as salts should be given. Pilocarpin (3 to 6 grs. subcutaneously) is recommendable. Arecalin (1 to 2 grs. subcutaneously) can also be employed. In the early stages bleeding has been tried with apparently good results.

When the animal is very restless and excited clysters of chloral hydrate may be used. Convalescence is usually protracted.

ENCEPHALITIS. INFLAMMATION OF THE BRAIN.

Definition.—Encephalitis is an inflammation of the brain which is usually circumscribed and confined to certain well defined areas. It nearly always results from infection and appears either as a suppurative or a non-suppurative process.

Non-suppurative Encephalitis.—Definition.—Non-suppurative encephalitis is an inflammation of the brain tissue occurring usually in the form of multiple foci which sometimes are hemorrhagic. It is not an uncommon sequel to acute infectious diseases although it may occur independent of these.

Occurrence.—While any of the domestic animals are subject to it, it is most frequent in the horse and dog.

Etiology.—Non-suppurative encephalitis is the result of infection or bacterial intoxication. In the first instance it may be secondary to specific infectious diseases (infectious pneumonia of the horse, strangles), the viruses of these diseases circulating through the brain, or it may be secondary to some local bacterial infection, the toxins of which reach the brain.

Cases of encephalitis may occur concomitant with or

follow infectious pneumonia or strangles in the horse, which in some outbreaks of these diseases occurs more commonly than in others. In rabies encephalitis, often hemorrhagic, is occasionally well developed.

Encephalitis may also result from sunstroke. Feeding heavily on certain foodstuffs (legumes, rye) may predispose the animal.

Symptoms.—If encephalitis is secondary to some specific infectious disease the symptoms of it may be masked by the basic disorder. As a rule, the cerebral symptoms which begin either gradually or quite rapidly (hemorrhage) manifest themselves as disturbance in consciousness. The patient appears languid, stupid, more or less oblivious of its surroundings, and assumes unphysiological postures. The gait is staggering or the patient may be down in a soporous or even comatose condition. Sometimes in the horse the patient will show symptoms of cerebral excitement or even rabiform symptoms. These are usually followed, however, within a short time by stupor. The patient may show forced movements.

The topical symptoms are usually not determinable if there is much mental depression. However, some of them may be notable, such as paralysis of the pharynx, tongue, larynx, eyelids, dilated pupils, etc. If the inflammation of the brain is diffuse a general paresis may result, the patient being unable to regain its feet when down or walk without support when up. If the respiratory center becomes involved fatal dyspnea may result. The temperature is usually elevated (105° F.), but the fever is mild and may be entirely absent in protracted cases. The pulse is generally in harmony with the temperature. Both are increased during the stage of excitement. In the earliest stages the appetite is good, provided the basic disease present has not already interfered. If the mental depression is marked, however, the patient may refuse to eat.

Course.—The usual run of acute encephalitis is two to five days. Subacute cases may last for weeks and chronic ones for years, producing the so-called "dummy."

When the development is rapid, disturbance in conscious-

ness soon appears. With the development of the mental symptoms the topical symptoms usually keep pace. Recovery occurs exceptionally. In influenza the course is more favorable. Cases which do not die usually lead to the patient becoming a "dummy" which not infrequently suffers from a temporary return of the encephalitic symptoms. If topical symptoms are left behind obviously they may interfere with the animals' efficiency.

Diagnosis.—The diagnosis depends upon the symptoms of a severe brain disturbance with which are associated well-defined topical symptoms, such as hemiplegia, monoplegia, ataxia, facial paralysis, etc. If these symptoms occur with or follow an infectious disease with which a non-suppurative encephalitis is apt to occur a diagnosis is possible. On the other hand, primary encephalitis is quite difficult to diagnose unless both the general and topical symptoms are well developed. From purulent encephalitis the non-suppurative form can usually be distinguished by the absence of injury to the cranium or the absence of a primary abscess in some removed organ or in the cranial wall. It may be impossible to distinguish between encephalitis and meningo-encephalitis in those cases of encephalitis in which the topical symptoms fail. Furthermore, in some cases of encephalitis the meninges may be also involved. Encephalitis is distinguished from chronic hydrocephalus by its more sudden development, the severity of the brain symptoms, and the presence of topical symptoms. Encephalitis usually follows some infectious disease. From forage poisoning encephalitis is distinguished by the severity of the brain symptoms, the sporadic appearance of the disease, the absence of intestinal symptoms, and no history of the animals having eaten food which was moldy or otherwise spoiled.

Treatment.—The treatment is the same as for meningo-encephalitis, and is usually of little aid to recovery.

Suppurative Encephalitis.—(*Abscess of the Brain.*)—**Occurrence.**—Brain abscesses are most apt to occur in young horses. In the other domesticated animals abscess of the brain is extremely rare.

Etiology.—The most common cause of abscess of the brain is strangles, which assumes the irregular form and leads to internal metastatic abscesses. It may occasionally result from other infectious diseases, such as puerperal septicemia, purulent pneumonia, infectious pneumonia (with secondary pus infection) and pyemia. Occasionally an abscess of the brain may result from direct injury to the cranium or from abscesses which occur in the neighborhood of the brain. In rarer instances parasites (estrus, cœnurus, echinococcus in sheep; Estrus bovis in cattle, and gastrophilus in the horse) are causes.

Symptoms.—The symptoms of brain abscess may develop either very rapidly or gradually. In the former case the symptoms are acute; in the latter subacute. When the symptoms develop rapidly the patient shows fever and not infrequently mental excitement, even amounting to rabiform symptoms. These may be followed by mental depression or may persist until the death of the animal. The muscles may twitch or undergo clonic spasms; forced movements are not infrequently observed. The patient usually dies in a few days or in less than two weeks. In other instances, as noted, the symptoms develop gradually, the patient showing disturbance in consciousness, forced movements, usually walking in a circle. There may be occasional manifestations of cerebral excitement; epileptiform attacks with intervals between during which the patient appears normal. The temperature may not be increased, although usually it is intermittent or remittent in type (pus temperature). Topical symptoms, such as sudden blindness in one or both eyes; the pupils may react unsymmetrically. Hemiplegia has also been observed. The head of the patient is often held to one side and attempts to straighten it cause symptoms of excitement. Some patients show vertigo, irregular gait, and a tendency to fall while in motion. Sometimes pressure on the poll produces epileptiform convulsions.

Diagnosis.—The diagnosis depends very largely upon the history of the case, *i. e.*, whether or not the patient has suffered from a disease of suppurative character (strangles,

puerperal septicemia). The acute form of abscess of the brain cannot be distinguished from many cases of acute encephalitis or meningo-encephalitis. On the other hand brain abscess which assumes a subacute course may be distinguished by the periodicity of the brain attacks between which the animal may seem in good health, a remittent fever (take temperature for several days), the absence of sensitiveness of the poll, and the peculiar topical symptoms shown.

Treatment.—In animals little can be done to relieve the patient. The treatment is therefore the same as for meningo-encephalitis. In rare instances the abscess may be opened, after trephining the cranium, and its contents evacuated. Obviously the abscess must first be located, a difficult matter in animals, and it must lie superficially if results are to be expected.

INFECTIOUS MENINGO-ENCEPHALOMYELITIS.

Borna Disease. Enzoötic Cerebrospinal Meningitis of Horses.

Definition. — Enzoötic meningo-encephalomyelitis is an acute infectious disease of the brain and spinal cord of the horse which is characterized clinically by symptoms of cerebral excitement followed by depression, paralysis of peripheral nerves and general paralysis. It is usually fatal.

Occurrence.—The disease was first described in Württemberg, Germany, in 1813. It is common in the state of Saxony, where, in 1894, it attacked a number of horses in and near the city of Borna, from which place the disease derived its name. Whether or not this disease has ever existed or does exist in the United States is in dispute. Some authorities claim to have recognized it; this is denied by others, who believe that outbreaks of so-called forage poisoning among horses were mistaken for it. Until a complete scientific study is made of forage poisoning, which is probably not a clinical entity, this question will not be definitely settled.

In Europe, Borna disease is confined to limited districts,

in which the disease breaks out at different periods, sometimes annually, sometimes with several years between outbreaks. It is most apt to occur following wet seasons. The disease is usually confined to certain farms, but during some years becomes a more widely distributed enzoötic.

Etiology.—The cause of the disease has not yet been determined. The Borna coccus (Diplococcus intracellularis equi) has not been proved to be the cause.

Natural Infection.—Horses are probably infected through contaminated food and water. Some authorities believe that the infection is acquired through the respiratory organs. However, Borna disease is more common in the winter and early spring months than in summer, the dusty period of the year. It is possible that the virus is voided with the urine. The disease is not communicable. It is much more common among farm than city horses.

Necropsy.—Macroscopically the brain appears normal, but under the microscope a marked cellular infiltration is noted in the meninges, brain and spinal cord. Especially typical changes have been noted in the ganglionic cells in the olfactory lobes and horns of Ammon which contain peculiar, intensively stained bodies within the cell nucleus ("nuclear inclusions").

Symptoms.—The symptoms are varied, but in general are: Early fatigue, icterus of the mucous membranes and digestive disturbances, sometimes amounting to colic attacks. These are followed by symptoms of cerebral excitement; twitching of the muscles of the face; spasms of the muscles of the neck, sometimes producing torticollis; occasionally trismus, nystagmus, unequal dilatation of the pupils, skin hyperesthesia, exaggerated reflexes, occasionally increased sexual desire and psychic phenomena. In some instances the patients are vicious, aggressive; in others they show epileptiform spasms. Later the patients appear depressed, stupid, even soporous, and may show forced movements. Motor paralysis is recognized by a weak, staggering gait, paralysis of the pharynx and general paralysis. The pulse, respirations and temperatures are usually little affected. In a few cases an eczema of the skin appears.

Course and Prognosis.—The course is usually from eight to fourteen days. The mortality is over 90 per cent. Those which recover are often left infirm through blindness, epilepsy, permanent loin lameness or they remain "dummies."

Treatment.—Treatment is of no avail. As a preventive it is recommended to change the food and drinking water and to keep the animals from infested pastures. Water from wells and cisterns which are contaminated with stable seepage should be especially avoided.

CHRONIC HYDROCEPHALUS.

Definition.—Chronic hydrocephalus is a brain disorder common in horses, but rare in other animals caused by the collection of serous fluid in the lateral ventricles of the brain. It leads through pressure to dilatation of the lateral ventricles, an increase in the size of the brain and an elevation of the intracranial pressure. The condition is rarely congenital, more often acquired.

Etiology.—Two types of hydrocephalus may be distinguished from the standpoint of etiology: (*a*) Inflammatory hydrocephalus, the result of acute inflammation of the brain of which it is a sequel developing in about one month. In this condition the fluid is an exudate. (*b*) A primary or idiopathic hydrocephalus is probably of mechanical origin and the fluid a transudate. It may be due to a congenital constriction or closing of the Sylvian aqueduct. An inherited predisposition to this form of brain hydropsy is probable.

Symptoms.—Chronic hydrocephalus in the horse is the commonest cause of the so-called "dummy." There is usually more or less disturbance in consciousness which the animal shows by a number of clinical symptoms. The following are the most characteristic all of which are made more prominent by vigorous exercise: The attitude of the patient is unphysiological, the head is held low, the limbs are frequently misplaced, the legs being crossed, and the patient is apt to stand diagonally in the stall. The patient seems indifferent to its surroundings, is sleepy, the eyelids partially closed, little attention is paid to commands, and an effort to

back the horse is futile. The heart action is slow, the pulse in some cases dropping to 20 or 30, although it retains its normal softness. The symptoms of depression, appearing from time to time, are due to a rise of intracranial pressure. Periods of excitement may occur which cause the animal to show symptoms as in the stage of excitement in acute inflammation of the brain. The appetite of the "dummy" is often impaired and the prehension of food unphysiological. Eating is quite frequently interrupted, the animal apparently forgetting for the moment that it is at a meal. In drinking the head is often projected up to the eyes in water; in some instances the animal tries to "eat" rather than drink the water. As noted these symptoms are usually emphasized by exercise until the animal is in a profuse sweat. Occasionally symptoms of vertigo and syncope occur. The skin reflexes (snapping the forehead, poking a finger in the ear, treading upon the coronet) are either diminished or may in some cases be exaggerated. In driving a "dummy" sometimes the animal tends to go to the left or right of the road notwithstanding the effort of the driver to prevent it. The gait is often abnormal, the animal walking as if in water, with a high wading movement of the forelimbs, or, on the other hand, it may frequently stumble, setting its feet down in an uncertain fashion.

Course.—The course is chronic and accompanied by many exacerbations and remissions. The animal may live for many months or even years showing improvement in cold and becoming worse in warm weather. In exceptional cases it may even refuse to eat and die of starvation. It is remarkable how often the condition as to flesh is retained notwithstanding a very variable appetite and the relatively small quantity of food consumed.

Prognosis.—The prognosis is bad as far as producing healing is concerned. However, many "dummies" can render service at slow work and especially during cool weather for months or even years. In time, however, through gradual mental and locomotor disturbances, their usefulness ceases, and eventually they are destroyed.

Treatment.—Treatment is of no avail. The efficiency of the animal may, however, be prolonged by feeding only light, laxative food and giving the patient good care. To relieve constipation, salts should be given, and during an exacerbation, hypodermic injections of pilocarpin (grs. iv to vj) or arecalin (grs. j to ij) afford relief.

BRAIN TUMORS.

In animals brain tumors are comparatively rare. In the horse the cholesteatoma has been noted quite frequently on necropsy. During life it rarely produces symptoms. The tumor varies in size from a pea to a hen's egg. Other tumors occurring in the brain substance are gliomas, gliosarcomas, melanosarcomas, and very rarely carcinomas. Some of the chronic infectious diseases may produce growths in the brain, such as tuberculosis, actinomycosis and botryomycosis.

In the meninges, fibromas, lipomas, angiomas, sarcomas, epitheliomas, papillomas, and dermoid cysts have been noted.

Symptoms.—Due to the slow growth of brain tumors they rarely produce any symptoms during life. A tumor the size of a hen's egg (melanoma) has been found in the brain of a horse without the animal seeming in any way disturbed by it.

In rare instances brain tumors may produce periodical increases in intracranial tension, especially after vigorous exercise, causing symptoms of transient cerebral excitement, followed by depression or the patient may show symptoms of chronic hydrocephalus. More rarely the symptoms closely simulate acute meningo-encephalitis or encephalitis, the animal dying in a short time. Sometimes brain tumors produce epileptiform seizures, cerebral ataxia, forced movements, hemiplegia, blindness, and an abnormal carriage of the head. Congestion of the papilla is thought by some observers significant of brain tumor, and is a probable cause of the blindness (amaurosis).

Diagnosis.—The diagnosis is obviously extremely difficult. The gradual development of the symptoms, both general and topical, the congestion of the papilla, and the absence of

fever point to the condition. In rare cases in which the tumor leads to a deformity of the cranium the diagnosis is easier. The clinical symptoms of chronic hydrocephalus, chronic meningo-encephalitis, abscess, and parasites of the brain so closely resemble those of tumor in many cases that an accurate diagnosis becomes impossible.

Treatment.—Treatment for brain tumor in animals is rarely of avail. In man they are occasionally removed surgically.

GID. CŒNUROSIS.

Definition.—Gid is a chronic parasitic disease of sheep and cattle, due to the presence of the Cœnurus cerebralis in the brain and very rarely in the spinal cord.

Natural History.—Cœnurus cerebralis is the cyst form of the tapeworm Tænia cœnurus. The adult worm is harbored by dogs, principally shepherd and butchers' dogs. Occasionally wolves and foxes are hosts. The infestation of sheep and cattle takes place by their ingesting the eggs or proglottides which are voided with the feces. The shell of the egg is dissolved in the abomasum allowing the six-hooked embryo to escape. The embryos perforate the bowel wall and probably through the bloodvessels reach the brain and spinal cord where they produce a hemorrhagic leptomeningitis and in some instances a purulent infection. They ultimately develop into cysts from the size of a pigeon's to a hen's egg. The cyst produces atrophy of the brain tissue surrounding it and also of the overlying skull.

Occurrence.—While gid is common in some districts abroad it is a comparatively rare disease in the United States, although isolated outbreaks have occurred in various parts of this country. Sheep are much more commonly infested than cattle, in which it is a very rare disease. Young sheep are more susceptible than aged ones.

Symptoms in Sheep.—From a clinical standpoint, and due to the development of the parasite in the brain, three stages of the disorder may be determined: (*a*) The stage of acute cerebral inflammation, (*b*) the stage of latency due to the gradual growth of the cyst, and (*c*) the stage of gid, the cyst being completely developed.

The stage of brain irritation sets in from one to two weeks after infestation, and, as a rule, lasts about one week. Usually this stage is overlooked by the shepherd, as most commonly the symptoms are not marked. Otherwise the sheep present symptoms of excitement, fright, forced movements, and even convulsions; or, on the other hand, may show languor, stupor, irregular gait, and more rarely maniacal symptoms.

In the stage of latency, which lasts from three to six months, the patient appears normal.

The gid stage usually develops in winter or early spring, and lasts about one month. In this stage the patient shows mental or motor disturbance and often topical symptoms. Quite commonly the first symptoms noted are those of mental disturbance. The sheep appear stupid, and in some instances act like a horse with chronic hydrocephalus. Later characteristic forced movements appear, the sheep running around in circles, trotting across the field with head up and high knee action or they may roll over the long axis of the body using a limb or the head as a pivot. Sometimes the sheep falls suddenly on its side or may fall over backward, the head being held high, with spasms of the muscles of the neck. Epileptiform convulsions, nystagmus, strabismus, and blindness are occasional symptoms. In rare instances a soft, fluctuating area appears at the top of the skull which, if punctured, discharges a clear fluid. The disease usually leads to death through brain paralysis and inanition.

Treatment.—The most important is the prophylaxis which consists in driving out the tapeworm from the dog and preventing dogs from obtaining the brains of sheep containing the cyst. An operative treatment consists in trephining and trocaring the cranium over the seat of the cyst and removing its contents.

INFECTIOUS BULBAR PARALYSIS.

Infectious Itching Disease. Mad Itch. Pseudorabies.

Definition.—A peculiar infectious disease which manifests itself mainly by marked pruritus of the skin, nervous irri-

tability and sometimes paralysis of the throat and general paralysis.

Occurrence.—Bulbar paralysis was first definitely recognized in Hungary in 1902. The disease has been noted in the United States, especially in the Southern States (Alabama), where it is much confused with rabies.

Etiology.—The cause of the disease is unknown. It may be readily transmitted by inoculating brain tissue from animals which have died of it into healthy cattle, sheep, and goats. Horses and asses do not seem to be as susceptible to artificial inoculations as other animals. Dogs, cats, rabbits, guinea pigs, rats, and mice also acquire the disease when injected with virulent material. The virus seems most potent in the tissue at the point of inoculation, next in the blood, and then in the central nervous organs. Bile, saliva and urine do not seem to be infective. The virus does not pass through fine porcelain filters. Infection through the digestive tract has been produced.

Symptoms.—In horses and mules the first symptom is usually an itching of the skin, especially about the head, which causes the patient to rub the part often so violently that it may be denuded of hair, excoriated, or even lacerated. The patients are further excitable, irritable, show dysphagia, salivation, gritting of the teeth, and finally paralysis. The temperature usually does not rise much above normal.

In cattle the infection generally appears about the head, lips and nose which parts the animal rubs violently, producing hemorrhage and inflammatory swellings which extend over the head, throat, and sometimes the neck. The patient is restless, moves its legs convulsively, keeps rubbing the head against objects, or scratches it with its hind feet. Salivation and inability to swallow are often noted. In some cases the digestion is impaired, the patient showing flatulency. The animals usually die in one to two days after the first symptoms appear.

Course and Prognosis.—The course is rapid, the patients dying within twenty-four to thirty-six hours. The prognosis is bad; nearly every case dies.

Diagnosis.—The diagnosis during the life of the patient is not so easy on account of the similarity of the disease to rabies. However, the patient with bulbar paralysis does not show the aggressive and destructive tendencies of the rabid animal and the symptom of pruritus is much more marked; on postmortem Negri bodies are absent; animals inoculated usually die more rapidly (rabbits in convulsions in one to three days), and the saliva is not infective.

Treatment.—Treatment is rarely of avail. It is recommended to apply tincture of iodin to the skin lesions, and if possible to so tie the animal that it cannot bite and rub itself. Edematous swellings may be scarified and iodin injected.

Believing the Rhus toxicodendron (three-leaved poison ivy) or the shrub Rhus vernix might be etiological factors, Cary, of Alabama, recommends that these plants be eradicated from pasture fields or that animals be kept from pastures containing them. He also suggests the use externally of permanganate of potash solution (1 per cent.) two or three times daily. Internally, Epsom salts or raw linseed oil is recommended.

CHAPTER II.

DISEASES OF THE SPINAL CORD.

TRAUMATIC INJURY OF THE SPINAL CORD.

Contusion of the Cord. "Broken Neck." "Broken Back."

Definition.—A bruising or laceration of the cord due to direct or indirect injury and usually the result of fracture of vertebræ.

Occurrence.—Traumatic injury to the cord is not uncommon in horses. Occasionally it occurs among cattle and more rarely in swine and sheep.

Etiology.—It is usually due to falls, blows, and in horses from struggling in the hopples especially if the animal is permitted to arch the back and neck upward, the head not being held properly. Either fracture or dislocation of vertebræ results leading to sudden pressure upon the cord with bruising and sometimes laceration. In some instances osteoporosis of the vertebræ predisposes to fracture; very rarely fissures of the vertebræ exist. Obviously a hemorrhage into the cord occurs. In rare instances the injury to the cord is due to the blood clot alone, neither dislocation nor fracture of the vertebræ having taken place.

Symptoms.—The symptoms will depend upon what part of the cord is injured and the degree of injury. If the cervical portion between the medulla oblongata and the origin of the fifth and sixth cervical nerves is involved and the cord completely crushed, the patient will die almost immediately from respiratory arrest. If only a part of the cord is crushed, however, it is possible for the patient to live several hours or even weeks after the accident. It will show symptoms of paralysis behind the seat of injury, dysphagia, dyspnea, and slow pulse.

If the cord is crushed just behind the origin of the phrenic

nerve, a paralysis and anesthesia of the parts behind will result. Breathing will be performed by the diaphragm, the ribs remaining stationary. The patient will also show paralysis of the bowels, bladder, and tail which becomes as limp as a dish-rag ("dish-rag tail"). The pupils may be unequally dilated but react to light.

If the cord is crushed in its thoracic portion the symptoms are the same except that the foreparts of the animal are not paralyzed and the ribs are employed in respirations.

In the lumbar portion of the cord the symptoms are similar except that the paralysis is confined to the hind limbs, tail, rectum and bladder.

For a time after the injury to the cord the patient may show profuse sweating, marked dyspnea and spasmodic contractions of the muscles in the neighborhood of the injury. In large animals sensitiveness along the fracture is rarely noted and crepitation cannot usually be determined. Obviously anesthesia exists behind the point of lesion.

Course and Prognosis.—Nearly all cases of fracture or dislocation of vertebræ are fatal. In horses and cattle death usually ensues within forty-eight hours. Some cases of apparent recovery suffer relapse and death from subsequent dislocation of broken fragments or the formation of masses of callus which encroach upon the cord.

Diagnosis.—As a rule the diagnosis is not difficult especially where there has been a history of direct or indirect injury. The bilateral paralysis and anesthesia occurring immediately behind the affected area, the limp tail and the rapid development of the symptoms are significant. From the standpoint of differential diagnosis fracture of the pelvis (no anesthesia, tail, rectum, or bladder paralysis) and azoturia (history, no tail paralysis, dark urine) should be thought of.

Treatment.—Treatment is of no avail. Cases which recover are usually the result of hemorrhage only into the cord. With valuable animals it is sometimes advisable to wait one or two days before dispatching the patient, to determine whether or not the symptoms arise from irreparable crushing of the cord or a blood clot. In the latter case approaching recovery is manifested by a rather rapid disappearance of the symptoms.

INFLAMMATION OF THE COVERINGS OF THE CORD.

Acute Spinal Meningitis. Meningomyelitis.

From a pathological standpoint may be distinguished: (*a*) Spinal meningitis, an inflammation of the meningeal coverings of the cord. If the hard spinal membrane is involved, a spinal pachymeningitis is spoken of; if the soft, a spinal leptomeningitis. (*b*) Myelitis, an inflammation of the substance of the cord. In practice the inflammation so commonly involves both the cord and its coverings that the term meningomyelitis is usually most applicable to the condition.

Occurrence.—Spinal meningitis is a rather rare disease in horses except when it occasionally assumes an enzoötic distribution.

Etiology.—The principal cause of spinal meningitis is infection. The condition is rarely secondary to acute infectious diseases, such as infectious pneumonia of the horse, strangles, pyemia and septicemia. It may also result from inflammation which exists in the neighborhood of the cord, such as caries of the vertebræ, abscesses which erupt into the vertebral canal and it is possible that infection may be carried along the nerve trunks to the cord and its coverings. A primary spinal meningitis may be occasionally the result of traumatism, such as a blow over the back, or rarely where a horse's tail is docked too closely. In very rare instances sharp pointed foreign bodies which have been swallowed by cattle have wandered into and injured the cord. The form of inflammation may be either serofibrinous or suppurative.

Symptoms.—The gait of the animal affected is usually stiff, straddling and labored. In the first stages the skin over certain areas, especially in the region of the back, is extremely sensitive. Stroking the animal in a direction contrary to the lay of the hair causes severe pain, the patient arching the back, becoming restless and making every effort to evade the examiner. Striking the tops of the spines of the vertebræ with the handle of a percussion hammer may cause the animal to evince pain.

Groups of muscles show twitching or more marked spasm-

like contractions which are usually initiated whenever the skin is touched. In some instances the patient is so sensitive that it may rear into the air. The muscles of the back and neck appear firm and extremely tender. If the abdominal muscles are involved the respirations are rapid and superficial. Urination and defecation are painful and difficult. In some cases spasm of the sphincters of the bladder and anus produces retention of urine and feces. In rare instances in male animals priapism occurs.

As the cord substance usually becomes involved later, paralyzing the roots of the motor nerves, paralysis of muscle groups, diminished reflexes, and decreased sensibility occur. In the latter stages the animal becomes paralyzed, the paralysis involving all parts of the body behind the cord lesion. The temperature of the patient is usually increased.

Course.—As a rule, death ensues within a week. When the inflammation is confined to circumscribed areas of the cord the patient may live for months.

Diagnosis.—If the disease develops typically and each stage can be observed a diagnosis usually can be made. The gradual diminution of the symptoms of extreme skin sensitiveness and muscle spasms in the region of the spinal nerves, the recognizable spread of the inflammation along the course of the cord and the presence of a primary disease in the neighborhood of the spinal canal are significant. The peculiar course differentiates spinal meningitis from contusion of the cord. From the standpoint of differential diagnosis muscular rheumatism, laminitis and tetanus should be thought of. From acute muscular rheumatism the marked sensitiveness of the skin seen in spinal meningitis is significant; in laminitis the presence of foot symptoms, and in tetanus the absence of sensory disturbances and the prolapse of the nictating membrane when the head is elevated, serve for differentiation. A distinction between spinal meningitis and myelitis is not always possible. However, myelitis is characterized by sensory and motor paralysis with which is usually associated paralysis of the bladder and rectum and does not present symptoms of hyperalgesia and muscular spasms.

Treatment.—The patient should be made as comfortable as possible. Horses should be given deep bedding and the paralyzed parts kept scrupulously clean and protected to avoid decubitus. If the conditions permit, a sling may be used. Internally salicylate of sodium (℥ss) or calomel (℥j) may be used. When the pain is very great and the spasms of the muscles marked, narcotics such as chloral hydrate or morphin may be administered.

INFLAMMATION OF THE SUBSTANCE OF THE CORD.

Spinal Myelitis.

Definition.—Spinal myelitis is an inflammation of the substance of the cord usually due to infection or intoxication.

Occurrence.—Spinal myelitis is a rare disease in horses and cattle.

Etiology.—It is usually secondary to influenza, rabies, rarely to tuberculosis in the ox, and still more rarely to strangles. In so-called forage poisoning of horses occasionally myelitis has been noted. In many instances no cause can be determined. Refrigeration, overexertion, and abuse of the sexual organs are probably only predisposing factors.

Symptoms.—Three forms of myelitis are described from a clinical standpoint, *viz*: (*a*) Transverse, (*b*) disseminated, and (*c*) diffuse. In animals a differentiation among these, however, is not always possible. The symptoms of myelitis are dependent upon the site and extent of the spinal inflammation and vary accordingly.

Transverse Myelitis.—A focal lesion affecting more or less completely the whole transverse area of the cord. Depending upon whether the cervical, dorsal, or lumbosacral regions are involved, the symptoms will vary.

The sensory and motor disturbances usually develop gradually. For a time the animal may show only early fatigue when at work, may lie down frequently and rise to its feet with difficulty. Later the gait becomes irregular behind, the animal seems "weak in the back," and frequently knuckles

in the hind fetlocks. Later it may become completely paralyzed behind the point of lesion. Horses may therefore assume a sitting posture; cattle and small animals may drag the hind parts. The skin and tendon reflexes may be exaggerated, especially if the myelitis has developed gradually and the paralysis be of spastic type; or, on the other hand, they may be greatly diminished, the muscles involved lose tone, become flabby, and no longer contract. The bladder, rectum, and tail eventually become paralyzed, leading to incontinence of urine and feces and the development of a limp tail. Provided the animal live long enough there usually later develops atrophy of groups of muscles. Edema of the skin and diffuse sweating have also been noted.

Disseminated Myelitis.—This should be regarded more as a multiple of the transverse type than as a separate disease. It is very rare in animals. Depending upon the seat, size, and number of inflammatory foci the symptoms vary. In some instances they are identical with transverse myelitis. A diagnosis is only possible when the patient manifests a circumscribed motor and sensory paralysis which may be confined to one hind limb or to certain groups of muscles. Not infrequently the muscles involved may show rhythmic twitchings or contractions. In the horses a string-halt-like movement of a limb has been noted. In the dog a desire to gnaw at a part until it became mutilated has been observed.

Diffuse Myelitis.—This is sometimes spoken of as ascending or descending myelitis. It is characterized by progressive paralysis, motor and sensory, usually beginning in the hind limbs, croup and tail and gradually involving the whole of the body as the paralysis progresses anteriorly. Conversely it may begin anteriorly and spread toward the tail.

Course and Prognosis.—The course depends upon the seat and the rapidity of extension of the inflammation. Transverse and diffuse myelitis usually end fatally in a short time while disseminated myelitis may last for months. Death usually results from septicemia (decubitus), inflammation of the paralyzed bladder and bowel, or in some cases from respiratory arrest. Recovery is very rare.

Diagnosis.—The diagnosis of spinal myelitis depends upon the presence of cord symptoms without history of injury. The absence of extreme skin sensitiveness and muscular spasm differentiates it from traumatic injury. From muscular weakness attending general diseases which do not involve the cord, the condition may be differentiated by the absence in these of any sensory disturbance, bladder, rectum or tail paralyses.

Treatment.—The treatment is rarely satisfactory. The patient should be provided with a clean, deep bed. Slings may be used when feasible. The bladder and rectum may be emptied manually. Every effort should be made to avoid decubital gangrene. Drugs, such as iodid of potash, strychnin or arsenic, do little or no good. Constipation may be relieved by subcutaneous injections of arecalin (gr. i) and rectal infusions. Electricity is much employed but little is to be expected from it. As a rule, it pays to dispatch the patient.

COMPRESSION OF THE SPINAL CORD.

Definition.—A condition whereby the cord is pressed upon by a growth, tumor, abscess or parasite which invades the vertebral canal.

Occurrence.—Compression of the cord is comparatively rare in large animals. Occasional cases are recorded in horses, cattle and swine.

Etiology.—The following pathological conditions may lead to compression of the cord:

(*a*) Ossification of the intervertebral discs: Occasionally in old horses a senile ossification occurs and if the ossified disc happens to protrude into the lumen of the vertebral canal, compression of the cord results.

(*b*) Inflammatory growths: In swine tuberculosis and in cattle tuberculosis and actinomycosis of the vertebræ may involve the vertebral canal and encroach upon the cord. Occasionally the tuberculosis may develop upon the meninges of the cord with similar results. In very rare instances tuberculosis in the horse may involve the cord. More commonly glanders of the vertebral column may affect the cord.

(*c*) Tumors: Rarely do tumors cause compression of the cord. Generally the tumor develops in the neighborhood of the spinal column (sarcoma), proliferates through the intervertebral openings or through the substance of the vertebræ enters the canal and invades the cord. In gray horses these tumors are usually melanotic (melanosarcomas).

(*d*) Abscesses: Abscesses which develop in the neighborhood of the vertebral column may invade the canal, producing compression. As a rule, however, this is not the case, although the pus may enter the canal and infect the meninges.

(*e*) Parasites: In cattle and sheep the Cœnurus cerebralis; in swine cysticerci, and in cattle echinococci invade the spinal cord. They rarely produce symptoms during life and therefore have only a pathological and sanitary importance.

Symptoms.—The symptoms in large animals are usually those of rigidity of the spine which may make it difficult for the animal to eat off the ground or to rise from a recumbent posture. The gait is also stiff and labored. As a rule the patient gradually becomes paralyzed behind the seat of the lesion so that paralysis of the tail, rectum and bladder appears.

Course.—The course is chronic. The paralysis, which is gradual in its development, eventually leads to permanent recumbency and death from decubital gangrene (septicemia). In other cases hypostatic pneumonia or cystitis may be the cause of death.

Diagnosis.—The diagnosis is not easy. The gradual progressive paralysis, anesthesia, stiffness of gait, and atrophy of muscles are noted in other diseases of the spinal cord. Obviously in those cases in which swelling and pain in the neighborhood of the vertebral column occur the diagnosis is easier.

Prognosis.—The prognosis is unfavorable. In rare cases a temporary improvement has been noted. However, usually this is followed by an exacerbation.

Treatment.—After waiting until the patient can be observed long enough to determine that there is no hope of recovery it should be dispatched. Obviously in edible animals the destruction of the patient should be undertaken earlier than in horses.

INFECTIOUS SPINAL PARALYSIS OF THE HORSE.

Enzoötic Paraplegia.

Definition.—An enzoötic spinal paralysis of horses, which usually takes an acute course and is characterized pathologically by numerous small hemorrhages in the different organs, but particularly in the spinal cord. The genital organs and bladder not uncommonly show gelatinous infiltration.

Occurrence.—The disease is found in different parts of Europe, occurring mostly among cavalry horses and in studs. No outbreaks have been reported in the United States.

Etiology.—The cause of the disease is believed to be a streptococcus (Streptococcus melanogenes) which is found in the blood, parenchymatous organs, medullary substance of the bone, spinal cord, and in the gelatinous infiltration of the genital organs and bladder.

Natural Infection.—Horses are infected probably through the digestive tract with contaminated food and water. It is possible that the streptococcus assumes a saprophytic life outside of the body. Horses of low resistance may be first attacked, and from them later others are infected. The urine and feces are probably infective.

Symptoms.—A preliminary stage is characterized by weakness behind, rapid fatigue, and emaciation. In some cases spasms of the muscles of the loins, croup, and abdomen have been noted. In some outbreaks mild edema of the prepuce or vulva has been observed. The patients urinate frequently.

In the later stage of the disease the patient may collapse during work or more rarely even when at rest. Paralysis of the hind limbs develops, making it impossible for the animal to rise from a recumbent position or stand without assistance. The digestive tract remains practically intact. In some outbreaks the temperature may reach as high as 107.6° F., and the pulse become very rapid. The patient may also show strangury; the urine is stained red and contains albumin. Sensibility is not much impaired, and paralysis of the sphincters is little developed. Occasionally there may be marked

inflammation of the external genitals. The penis is swollen, edematous, and protrudes from the prepuce. In mares the external genitals may be swollen and edematous.

Course.—The course is very varied. The duration of the disease may be from a few days to three months. Convalescence is slow. The mortality varies from 50 to 100 per cent.

Diagnosis.—The paralytic symptoms with little impairment of sensibility, a good appetite, the swelling of the external genitals, and the enzoötic occurrence of the disease are significant. In sporadic cases only the determination of the streptococci in the blood would furnish tangible evidence of the existence of the disease. From the standpoint of differential diagnosis, infectious spinal paralysis might be confused with azoturia, infectious anemia, sclerostomiasis, and forage poisoning.

Treatment.—Medicinal treatment is of little or no value. It is purely symptomatic.

Prophylaxis.—The food and water should be looked after to see that they are good and pure. The administration of antistreptococcic sera to healthy but exposed horses is thought to have a preventive action.

CHAPTER III.

FUNCTIONAL NERVOUS DISEASES.

VERTIGO.

Definition.—Vertigo is a symptom and not a disease. It is characterized by dizziness, a disorder of the equilibrating sense, causing a feeling of instability and apparent rotary movement of the body or other objects. Vertigo is very possibly due to a disturbance in the equilibrating center in the cerebellum.

Occurrence.—Vertigo is not common in animals, but is occasionally seen in horses and dogs. As a rule, the heavier breeds of horses are affected.

Etiology.—In animals vertigo is usually secondary to brain diseases, such as acute and chronic encephalitis and epilepsy. It may also attend chronic diseases of the lung and heart, which cause venous congestion in the brain. Compression of the jugulars from the collar of the harness is a common cause. Vertigo is a symptom of anemia of the brain; it can also come from eating poisonous plants. An ocular vertigo is occasionally seen in horses due to light effects, such as result from shiny blinders or bright light shining in the face or occasionally from driving the horse through an alley of trees which cast their shadows across the roadway.

Symptoms.—The horse is usually attacked at work. The patient begins throwing and shaking its head, running backward a few steps, swaying in the shafts, and after staggering about falls to the ground, where it lies unconscious and quietly for from one to five minutes. After coming out of the attack the animal regains its feet and seems to be normal again.

Diagnosis.—The diagnosis of vertigo is usually readily made, but to determine its cause is often extremely difficult. It may be distinguished from epilepsy by the absence of convulsions while the animal is down.

Treatment.—During an attack the patient should be unharnessed and made comfortable. Some recommend throwing a blanket over the head. Ice or cold water to the poll is often of advantage. If the cause of vertigo can be determined and removed, permanent healing is possible. Usually the best results are obtained by changing the harness to avoid constriction of the jugular veins or interference with vision.

EPILEPSY. FALLING SICKNESS.

Definition.—Epilepsy is a chronic disorder of the nervous system, characterized by attacks of unconsciousness and spasms, which occur periodically. Between the attacks the patient appears in normal health.

Occurrence.—Epilepsy is rare in horses and cattle, but is relatively common in dogs.

Etiology.—The causes of epilepsy are not known. In true epilepsy there are no lesions which are characteristic. Heredity has been accused.

Symptoms.—Two forms of epilepsy may be distinguished: The severe type characterized by a complete epileptic convulsion (grand mal), and the milder type characterized by incomplete or partial attacks (petit mal).

Grand Mal.—In animals the epileptic seizure comes about suddenly and usually without prodromal symptoms (aura epileptica). The patient falls to the ground after showing symptoms of dizziness, and is rapidly overcome with severe tonic spasms of the head, neck, body, and limbs. The jaws may be locked, the neck and back bent backwardly, and the limbs extended. There are clonic spasms of the lips and the lower jaw, inducing movements of the jaw and foamy salivation. The eyes are rolled in their sockets and the limbs move convulsively. At the same time the patient is unconscious, sensitiveness is lost, and the pupil dilated. The duration of the attack is only for a few minutes. After it is over the animal regains its feet, seems for a time languid, but is soon normal again. Between such attacks the patient appears in perfect health. Attacks follow at very irregular intervals. Epilepsy may exist for years or even during the whole life of the animal.

Petit Mal.—The mild type manifests itself by spasms of certain groups of muscles, usually of the head (lips, facial muscles, eye muscles), neck, and front limbs. This is accompanied by a partial and temporary loss of consciousness. In some cases there may be no convulsions, only loss of consciousness, as in vertigo.

Treatment.—No successful treatment is known for epilepsy. In animals bromid of potash (ʒj) is helpful.

ECLAMPSIA.

Definition.—A convulsive seizure like that of epilepsy, but which assumes an acute character and terminates either in permanent recovery or may end in death shortly following the attack. The term eclampsia may be used in a broad and in a narrow sense: Eclampsia in a broad sense would include brain convulsions or tonoclonic spasms, with loss of consciousness occurring usually in the course of acute encephalitis, influenza, lead poisoning, or uremia.

Eclampsia in a narrow sense would be the peculiar, acute, epileptiform spasms in suckling animals (eclampsia infantum), and in mothers which have just given birth to young (eclampsia puerperalis).

The most important type of eclampsia in animals is puerperal eclampsia, which is extremely rare in cows and sows, but relatively frequent in bitches. (See other works.)

CATALEPSY.

Definition.—Catalepsy is a peculiar nervous disorder, characterized by loss of consciousness combined with cramp-like contractions of the musculature of the body, which becomes rigidly fixed. The animals remain immovable in the position placed and the joints may be readily bent passively. At the same time there is loss of sensitiveness of the skin. The cataleptic state has been noted in dogs. It is extremely rare in animals.

CHOREA. SAINT VITUS' DANCE.

Definition.—Chorea, or Saint Vitus' dance, is an involuntary, rhythmic twitching of certain muscles producing irregular jerking movements usually in the head, eyelids, and facial muscles, and occasionally of the limbs.

Etiology.—The cause in animals seems to be a brain neurosis. Chorea-like twitchings sometimes result from distemper in dogs. This, however, is a sequel of encephalitis or meningo-encephalitis and is not a true chorea.

SPASMS OF THE DIAPHRAGM.

Definition.—A rhythmic, spasmodic contraction of the muscles of the diaphragm, with which are usually associated clonic spasms of the abdominal muscles.

Occurrence.—Spasms of the diaphragm are most frequently noted in horses. Exceptionally, cases have been described in cattle.

Etiology.—Digestive disturbances of an acute character affecting the stomach (acute catarrh, bloating) and bowels (catarrh or constipation). Very probably toxic substances absorbed from the gastro-intestinal contents reflexly stimulate the nerves of the diaphragm producing the symptoms. Overexertion, especially when accompanied by mental excitement (runaways, overdriving, pulling an object of which the horse is afraid), may also induce it. Acute inflammatory diseases of the thoracic organs and pleura may be occasional causes.

Symptoms.—The symptoms are somewhat similar to those of palpitation of the heart, except that the spasms do not occur synchronously with the pulse. They consist in rhythmic, electric-stroke-like shocks, which can be seen and felt especially along the ribs, the loins, hollow of the flank, and over the chest. If the hand is placed upon the patient, throbs may be felt which are most intensive over the diaphragmatic attachments. While coincidently the number of beats may be equal to those of the heart, they are generally less in number (ten to fifteen per minute) and do not corre-

spond with the heart beat. In some cases synchronous with each spasm a forced, noisy expiration is noted at the nostrils. The patient usually does not eat and is restless.

In cattle traumatic indigestion causing injury of the diaphragm may be attended by diaphragmatic spasms.

Course and Prognosis.—The course will vary from a few minutes to several days; most cases recover, however, in about two days. Generally speaking the prognosis, which depends upon the cause, is favorable.

Diagnosis.—The rhythmic throbs which produce synchronously a momentary protrusion of the hollow of the flank and epigastrium, with a simultaneous sinking of the intercostal spaces, are significant. In doubtful cases a rectal exploration, the hand coming in contact with the attachment of the diaphragm, is assuring.

If the spasms are confined to the abdominal muscles a marked twitching of these muscles may be seen and felt; synchronous with them the epigastrium sinks in and the intercostal spaces are protruded.

Treatment.—The patient should be placed in a quiet place and a subcutaneous injection of morphin given (grs. iij to vj). Bromid of potash and chloral hydrate (℥j) may also be used. Attending digestive disturbances should be treated.

PART X.

DISEASES OF THE SKIN.

ECZEMA.

Definition.—Eczema is a dermatitis accompanied by exudation and itching involving the superficial layers of the corium.

Course.—In the course of the dermatitis there develop erythema, papules, vesicles, and pustules, followed by desquamation.

Forms.—From a clinical standpoint, and depending largely upon the pathological character of the dermatitis, the following forms of eczema are distinguished:

(*a*) Erythematous eczema, an inflammatory congestion of the skin.

(*b*) Papulous eczema, characterized by nodules due to a cellular infiltration and swelling of the papilla.

(*c*) Vesicular eczema, consisting in the development of circumscribed areas of serous exudation, or vesicles, beneath the external layer of the skin.

(*d*) Eczema madidans, red or weeping eczema, due to the rupture of the vesicles from the patient biting and scratching them.

(*e*) Pustulous eczema, characterized by the appearance of vesicles containing pus, or pustules.

(*f*) Impetiginous eczema, originating from the rupture of the pustules causing the surface of the skin affected to be covered with moist or dry pus.

(*g*) Crustated eczema, crusts and scabs forming on the skin from the drying of the exudate.

(*h*) Squamate eczema, where the epidermis is covered with masses of scales.

(*i*) Seborrhoic eczema, where the crusts are infiltrated with masses of fat from the sebaceous glands, forming white or yellowish, greasy scales.

(*j*) Sycosiform eczema, which is an inflammation of the hair follicles, forming papules or pustules that are perforated by hairs.

Etiology.—The causes of eczema are usually external, and consist in mechanical, chemical, thermic, and infectious irritants. The most common of these is neglect of skin cleanliness. Eczema, therefore, is most often seen on parts of the skin of the horse where the least grooming is done. On the other hand too much water coming in contact with the skin can produce eczema. Examples are the eczema of sheep from exposure to excessive rainfall, effect of dewy pastures, muddy roadways, etc., upon the skin of the legs of horses or the too frequent bathing of dogs. Eczema can also originate from the discharge in diarrhea, incontinence of urine, and profuse sweating. The mechanical insults which produce eczema are insect bites (fleas, lice), biting, rubbing, and scratching on the part of the patient and the friction of the harness or saddle. Thermic influences are cold, the sun's rays (solar eczema), or fire. Many chemicals can produce eczema, such as mercury, mustard, tobacco, etc., when applied to the skin. Mercury or iodin given internally for a long period of time can have the same effect.

Internally eczema, or more properly exanthema, can be due to disturbances in the digestive tract where an auto-intoxication is produced. Exanthema also accompanies many infectious diseases and appears associated with conditions leading to cachexia (lung worm plague of sheep). Finally, mange mites produce eczema. In man certain individuals seem predisposed to eczema.

Symptoms.—The various pathological changes, such as the papule, vesicle, pustule, etc., in eczema can be noted on the living animal. The process passes through its varied stages rather rapidly, one phase developing out of the other. By lifting the crusts the moist areas over the papillary layer of

the skin are exposed, forming very characteristic lesions. It is, further, not uncommon to see all or most of the different stages on the different parts of the skin appearing at the same time. Where the skin has been rubbed, scratched, or bitten secondary changes appear which modify the character of the lesions.

Pruritus is a constant symptom of eczema. In acute cases it is often quite pronounced. If the eczema is acute and diffuse, fever may be present. From the inconvenience and suffering which diffuse eczema produces, the fever and loss of albumin to the body, in time the patient becomes anemic, emaciated, and cachectic.

Prognosis.—The prognosis is usually good, healing occurring in about three weeks. A seborrhoic eczema occasionally is seen in cattle. It is usually diffuse and eventually leads to complete loss of hair. Dampness is a common cause of eczema in sheep which have been exposed to continuous rainfall which keeps the wool soaked. The eczema is usually found along the back and croup. This form of eczema is sometimes called "rain rot." The skin becomes swollen and creviced, and is quite itchy. The areas involved are covered with crusts which, when lifted, expose moist reddened surfaces. The wool becomes tufted and falls out. Provided the sheep are not removed to shelter they become anemic and emaciated. When the weather gets dry, usually the eczema subsides.

Horse.—In the horse the favorite seats of eczema are the skin of the body, the flexion surfaces of the hock, fetlocks, and the mane and tail. In the late spring and summer a rather diffuse, papulovesicular eczema occurring in the form of nodules and scabs appears on the neck, back, sides of the shoulders, and croup. This is thought by the ignorant to be due to some "disorder of the blood," and is commonly known as "summer surfeit." In saddle horses in the saddle rest an eczema occurs, largely induced by sweating under the saddle blanket. A seborrhoic eczema of the mane and tail is more common than suspected. In these cases the proximal ends of the hairs are embedded in and matted together by a mass of fat, fetid masses of exudate and sebum, over which

large, fish-scale-like, enlarged epithelial cells are found. The distal end of the tail is most frequently involved. This form of eczema may lead to the loss of the tail hairs and more rarely to those of the mane. In old, neglected horses out of condition (bad teeth, gastro-intestinal catarrh) a generalized, squamous eczema is common. Eczemas of the limbs to which are given such special names as scratches, mallenders, etc., are included in works on surgery.

Ox.—Eczema is not so common in cattle as in the horse, but is seen to occur where malt or potato residue is fed. It affects principally the hind limbs, and is largely due to the liquid feces which come in contact with the skin of the legs. It may, however, involve the forelimbs, the body, and neck. This eczema is vesicular and crustated. It usually begins with an erythema, the skin being highly reddened, swollen, and painful, especially about the hind fetlocks. The patient is usually lame. Later there appear vesicles which soon rupture, leaving moist areas which in turn dry, forming crusts. Usually the eczema tends to spread on the limb to the height of the carpus or tarsus. The patients generally show symptoms of loss of appetite, diarrhea, fever, and emaciation.

Swine.—A non-parasitic eczema is rare in swine. In young, unthrifty pigs a squamous eczema occurs with brown or black crusts, hence the name "soot of young pigs." This form of eczema is usually seen in pigs which are kept in unsanitary quarters and are generally neglected. It appears particularly on the inner surface of the thighs, along the abdomen, sides of the chest, and inner surface of the forelimbs. More rarely it affects the head. The affected skin is erythematous and soon becomes covered with vesicles filled with a clear, sticky fluid. Pustules soon form, break, dry, and form crusts which from the admixture of dirt have a black appearance. Associated with hog-cholera, eczema or more properly an exanthema occurs.

Treatment.—The treatment of eczema is mainly external. In all cases the cause should be removed. This in itself often suffices. Before treatment is applied the affected skin should be prepared by removing the hair and the accumulations of exudate. For the latter, bathing the skin in tepid soft water

and a non-irritant soap followed by thorough rinsing and drying is important. If there are thick scabs or crusts, these may be softened with an ointment such as creolin ointment.

In choosing the drugs to apply it must be borne in mind that a distinction must be made between acute and chronic eczema.

Acute Eczema.—Water, soap, and if possible air should be kept from the affected skin. Crusts, scabs, and secretions should be removed with Burrow's solution (5 per cent.), lime water and oil (equal parts), or a salicylic acid salve (4 per cent.).

When the skin is moist drying powders should be used.

℞—Amyl. tritici . . . ℥iij
Pulv. alumini silic. ℥v
Zinci. oxid. . . . ℥j
M. f. strew powder.

or

℞—Zinci. oxid.
Bismuth subnitrici . . āā ℥j
Plumbi carbonici grs. xiv
Pulv. magnes. silic. ℥iij
M. f. strew powder.

Later ointments which have a metallic base or in some instances a plant base may be used. As examples, zinc salve, lead salve, and nitrate of silver salve (1 to 10). Unna's zinc paste:

℞—Oxidi. zinci . . 10 parts
Terra silic. . 2 "
Adeps benzoat . 28 "
M. f. ungt.

When pruritus is marked and is not ameliorated by ointments a silver nitrate solution (10 per cent.) or an ichthyol salve is good.

℞—Ichthyoli
Zinci oxid.
Amyl. tritici . . . . . āā 1 part
Vaselin . . . . . 2 "
M. f. ungt.

In very moist eczemas, powders are more valuable than salves, which do not adhere to the skin. Powders should be

applied bountifully two or three times daily, the old powder removed before applying the new, using cotton and oil. In obstinate cases nitrate of silver (2 to 6 per cent.) or picric acid (1.5 per cent.) followed by powders, and, when the exudate is dry, by salves.

Chronic Eczema.—Chronic eczemas are treated, contrary to acute, by irritant agents. The old crusts and scales should be removed with castile soap and soft water or vaselin. If water is used the skin afterward should be rubbed perfectly dry. If the skin is moist from exudate, apply powders, as in acute eczema. Later, coal-tar products are good, such as tar ointment (1 to 10) or tar liniment:

℞—Pix liquida and kaolin	āā 1 part
Alcohol	5 to 10 "

Creolin preparations are also indicated. The following prescription has been found useful:

℞—Creolini .	ʒj
Phenoli .	ʒss
Flores sulph.	ʒj
Spts. terebinth. . . .	ʒiv
Liquor ammonii fortes . .	ʒiij
Kerosene . . .	ʒxl

M. f. emulsio, apply with brush.

Naphthol or naphthalin salve (5 to 15 per cent.) or iodin-glycerin is of value.

℞—Tinct. iodini	1 part
Glycerini . .	4 "

Salicylic acid ointment (5 to 15 per cent.) and pyrogallic acid (5 to 15 per cent.) are also employed. Pyrogallic acid ointment is especially useful if the skin is much thickened. All of these agents tend to loosen the crusts, reduce the pruritus, and assist in the resorption of the exudate.

Internally mild cathartics and intestinal disinfectants (calomel) assist. Fowler's solution of arsenic in horses is useful. The patient should be given good care and food and prevented from biting or scratching the parts.

URTICARIA. NETTLE RASH.

Definition.—Urticaria is an eruption of the skin appearing in the form of rounded, flattened elevations, which appear and disappear suddenly, due to an infiltration of the papillary bodies and the Malpighian layer of the skin.

Etiology.—Urticaria is common in horses, cattle and swine. The causes are in part external and in part internal. Urticaria is often secondary to infectious diseases.

External Causes.—The external causes are irritant substances, such as insect stings, chemical agents (turpentine), nettles, and rubbing and scratching the skin. When the body is covered with sweat and suddenly cooled as by a shower of rain urticaria often develops in horses.

Internal Causes.—Urticaria is very often a symptom of digestive disturbances, gastro-intestinal catarrh, constipation, and so-called indigestion, or of general infectious diseases. There are probably formed in these cases in the fermenting ingesta toxins which are absorbed and irritate the peripheral bloodvessels. Certain foods like rye, potato tops, and wheat are causes. The toxins of specific microörganisms also are causative, *viz.*, dourine, swine erysipelas ("diamond disease"), purpura, influenza, and strangles. Urticaria also occurs after injections with tuberculin or mallein. Cows which have gone long unmilked sometimes suffer from it. In certain individuals there is an idiosyncrasy existing favoring urticaria when certain foods are eaten. In some instances no cause can be determined.

Symptoms.—Usually without any prodromal symptoms there suddenly appear upon the skin of the neck, shoulder, sides of the chest, and croup, swellings or nodes flattened on top and varying in size from a pea to a clenched fist or by confluence even larger. The hairs over the swellings are dry and stand erect; the skin between is normal. In some instances vesicles appear on the swellings which erupt, causing them to be moist or covered with dry exudate. In other instances the centers of the nodes sink, leaving a wall-like periphery (U. annularis). By confluence grotesque figures may appear.

Similar nodes may form on the mucous membranes of the nose and larynx. This complication may lead to cough, dyspnea, and edema of the glottis. In the vagina and rectum they may induce swelling and occasionally prolapsus recti. Urticaria may also involve the mouth and conjunctiva. Concomitant with the eruption there may be symptoms of general disturbance, such as fever and loss of appetite.

Course.—Urticaria usually disappears in from one to two days. In swine it may take from four to six days. In rare cases so much serum has accumulated between the corium and Malpighian layer that vesicles appear and the healing, which takes place under scab formation, is prolonged. Chronic cases have been noted, occurring with frequent relapses and causing the condition to last several weeks or months. Very rarely death results from edema of the glottis.

Treatment.—As urticaria usually heals spontaneously, treatment is rarely necessary. The animal should be placed on short rations and a good laxative given. Local applications to the skin are rarely indicated.

ALOPECIA.

Definition.—By alopecia is meant a falling out of the hairs without apparent skin lesion, and not due to parasites. Clinically two forms of alopecia are distinguished: (*a*) General alopecia (A. symptomatica), and (*b*) local alopecia (A. areata).

Etiology.—(*a*) General alopecia, which consists in the hair falling out over the whole body, seems to be due to some disturbance in the nutrition of the skin with atrophy of the hair roots. It is most frequently seen to follow infectious diseases, such as influenza, purpura, or tetanus. Poisoning with mercury and lead and with certain foodstuffs (potatoes) in horses is a cause. Obesity is also accused.

(*b*) Local alopecia is probably due to a trophic neurosis.

Symptoms.—General alopecia usually affects the whole body, the mane and tail being the last to fall out. The haircoat gradually grows thinner and thinner until it disappears except in a few places, where it is usually retained. The

loss of hair is often only temporary, the denuded skin soon again becoming covered with fine downy hair which grows out to full length. Local alopecia appears in the form of small, round, bald areas which gradually increase in size and by coalescence form large, bald spots. The skin is intact, although, as a rule, it is darker in color than normal.

Diagnosis.—Alopecia might be confused with herpes tonsurans or with mange. However, the absence of scales and broken hair shafts differentiate it from the former; the eczema and presence of the mange mite suffice to distinguish mange from it.

Treatment.—While general alopecia usually leaves of itself in a few weeks, provided the patient and skin are given good care, the local form is often quite obstinate and frequently incurable. Fluids containing alcohol and some irritant are generally recommended. Tincture of cantharides (1 to 5 alcohol), tincture of iodin (1 to 1 to 5 alcohol), and creolin (1 to 10 to 20 alcohol) are examples.

HAIRLESS PIGS.

Definition.—A generalized alopecia occurring in newborn pigs of sows afflicted with goitre.

Occurrence.—In some parts of the country large numbers of hairless pigs are born each spring. It has been estimated that nearly one million pigs are lost annually in the state of Montana alone from this cause.

Etiology.—As far as is known the cause is goitre which affects both sows and young pigs. The pigs born of gilts are more often affected than from older sows.

Symptoms.—Hairless pigs are of normal weight and size and are usually carried the full period of gestation or often a week beyond it. The degree of hairlessness is very varied. Some are born absolutely hairless with a smooth, shiny, bald skin, except for a few hairs around the eyes and nose. Others have a little hair. The hoofs of hairless pigs are thin-walled and undeveloped. It is not uncommon that full hair-coated and hairless pigs occur in the same litter. The hairless pig, if born alive, usually lives only a few hours.

Treatment.—While the recognition of goitre in the sow is extremely difficult it is recommended that, whenever hairless litters of pigs are born on the farm, the pregnant sows be fed iodin in the form of iodid of potash (10 grams to each 100 pounds feed). The iodid of potash should be fed during the entire period of gestation or at least during the last seventy-five days of it. It is also recommended as a prevention to feed plenty of roughage, alfalfa and clover hay. Intensive grain feeding with too little roughage tends to predispose to the disorder.

ERYTHEMA.

Definition.—Erythema is an inflammatory congestion of the skin which may be primary and result from various mechanical, chemical, and thermic causes; it may be a symptom of eczema, or it occurs secondary to infections and intoxications. In hog-cholera, erythema is occasionally seen; in swine erysipelas it is a constant symptom. White swine and sheep are affected by it if they eat growing buckwheat in the bright sunlight.

PRURITUS.

Definition.—By pruritus an itching of the skin without visible lesion is meant. It may be local or general.

Etiology.—The causes are not well understood. In some of the infectious diseases, such as rabies, dourine, and in disorders due to worms like trichinosis and grub-in-the-head of sheep, it has been observed. Severe, general diseases are sometimes accompanied by pruritus, such as chronic nephritis, icterus, and diabetes mellitis.

Treatment.—Treatment is usually of little avail, and consists in the application of local anesthetics or irritants.

TRICHORRHEXIS NODOSA.

Definition.—This is a condition of the hair in which nodules occur along the shaft, causing the hair to bend and break off.

It may affect the hairs of the body or the mane and tail of the horse. As it sometimes occurs enzoötically it is probably

due to an infection of the hair. It may be transmitted from one animal to another or to man.

Prognosis.—The prognosis is unfavorable.

Treatment.—The treatment consists in the application of antiseptics, such as bichlorid of mercury (1 per cent.), creolin (2 per cent.), pyrogallol (1 per cent.), or pyoctanin (1 per cent.).

ACNE.

Definition.—By acne is meant a purulent inflammation of the hair follicles. There form on the skin, papules and pustules. It is due to an infection with pus cocci and the resulting decomposition of the sebum. A parasitic form which is rare in large animals is due to the mite Demodex folliculorum.

In horses acne usually occurs in the saddle or harness rests where firm, painful nodules or nodes appear out of which may be expressed in the form of yellow, waxy cylinders an exudate which is a mixture of sebum and pus.

Treatment.—The treatment is surgical, and consists in opening, expressing the exudate, and curetting out the cavity, which is later cauterized.

PEMPHIGUS.

Definition.—By pemphigus is understood a skin eruption characterized by isolated vesicles, which may attain the size of a hen's egg or even larger, between which the skin appears intact.

Etiology.—The causes are not known.

Prognosis.—In animals the prognosis is good, most cases healing in about two weeks.

SCABIES. MANGE. SCAB.

General Remarks.—Mange is a communicable skin disease due to minute, insect-like parasites which irritate the skin in various ways producing an itchy dermatitis. The mites, which belong to the family Sarcoptidæ, are very small in size only the larger varieties being visible to the naked eye. Mange mites are widely distributed, therefore mange occurs

in every country. When large numbers of animals are congregated as are sheep on the western ranges, or horses and mules in time of war, the disease often assumes the character of an enzoötic. During the Great War, and until proper remedial agents were employed to combat it, fully 40 per cent. of the animals of the army became infested, unhorsing many military units. While mange in its incipient stages is a local disease, confined to restricted areas of the skin, under favorable circumstances it spreads over the entire body and leads to dermatitis, itchiness, loss of hair, emaciation, anemia, cachexia and sometimes death. While some varieties spread over the surface of the skin more rapidly than do others, each type of mange mite can, under favorable conditions, invade the whole skin.

Mange depends largely for its spread upon the fact that in its beginning stage it is a local disease affecting only a small area of the skin. The host may appear perfectly healthy, perform its usual functions normally and at the same time harbor a circumscribed area of mange which fails to attract attention. Such an animal if allowed to commingle with others will spread the disease. Under adverse circumstances, especially during the winter season when animals are subjected to rather unfavorable conditions as to feeding and care, the mange mites multiply and the skin lesions increase until marked symptoms of dermatitis develop. Ordinarily, therefore, methods of control and eradication are not employed until a number of animals are affected and general symptoms have developed.

Definition.—Mange is a dermatitis produced by minute, insect-like parasites, and attended by intense itching.

Occurrence.—As mange is communicable it assumes at times an enzoötic or even epizoötic distribution. This is particularly true of the scabies of sheep, which in a single state may affect thousands of animals, or, as noted, of horses and mules in time of war.

Etiology.—Mange is caused by a small mite which rarely can be seen with the naked eye. They are from 0.2 to 0.8 mm. in size, provided with four pairs of legs and an undivided trunk. The males are smaller than the females. In most

cases the mites are oviparous but some are ovoviviparous. The females lay eggs which in about eight days hatch six-legged larvæ. The larvæ moult forming in about three to four days nymphs. Three to four days later the nymphs develop to adult males and sexually mature females. The females live for three to five weeks, the males about six weeks. Under favorable circumstances mites may live as long as eight weeks, and under contrary conditions as short a time as two weeks. Infestation may be from direct contact or through intermediary agents, such as harness, grooming utensils, blankets, bedding, etc.

Varieties.—Four varieties are distinguished: (*a*) Sarcoptes, (*b*) Psoroptes, (*c*) Chorioptes (Symbiotes), (*d*) Cnemidocoptes, found only in birds.

Sarcoptes Mite.—This is a burrowing parasite which lives in the Malpighian layer of the skin where it forms tunnels under the epidermis. It is the smallest species (0.2 to 0.5 mm.), shaped like the turtle, with a head like a horseshoe and very short legs, the anterior two parts of which are marginal and the posterior two subabdominal. The males have two copulatory suckers. Three varieties of sarcoptes occur in animals: (*a*) Sarcoptes scabiei of the horse, (*b*) Sarcoptes squamiferus of the dog, and (*c*) Sarcoptes minor of the cat.

Psoroptic or Sucking Mite.—This parasite lives on the surface of the skin and is nourished by the blood and lymph. It is the largest of the mites (0.5 to 0.8 mm.), and under favorable circumstances (on black paper, for instance) may be seen with the naked eye. Its head is pointed, legs very long, and feet provided with tulip-shaped suckers. Two varieties of this mite occur in animals: (*a*) Psoroptes communis of the sheep and horse, and more rarely in cattle and buffalo. (*b*) Psoroptes cuniculi of rabbits.

Chorioptes (Symbiotes) or Scale Eating Mite.—This mite lives on the surface of the skin and on scales of epithelium. In size it stands between the Sarcoptes and the Psoroptes (0.3 to 0.5 mm.). The head is short, blunt, almost round, and the legs long, provided with suckers. Two varieties belong to this species: (*a*) Chorioptes (symbiotes) equi, bovis and ovis; (*b*) Chorioptes (symbiotes) felis, canis and cuniculi.

Mange of the Horse.—Forms.—On the skin of the horse three forms of mange mites appear.

Sarcoptic Mange.—*Definition.*—This is the most important form of mange in the horse because of its frequent occurrence and the fact that it is more difficult to cure than the other forms. It attacks the skin of the body, neck and head and tends to become general.

Symptoms.—The skin lesions usually occur first about the head, sides of the neck and shoulders and on the external surface of the legs. If the case is neglected it can spread over the whole body. The first symptom is severe itching which causes the animal to rub, bite and scratch itself especially during warm nights. An examination of the affected parts shows loss of hair, formation of papules, vesicles and crusts under which the skin appears moist. The lesions tend to increase peripherally, merging into larger, bald areas covered with crusts, scabs or layers of desquamated epithelial cells. In protracted cases the skin becomes thickened and ridged. From the rubbing, biting and scratching secondary lesions commonly occur. In about six weeks practically the whole skin may become involved. Characteristic of mange is the tendency to spread over the skin and the transmissibility to other horses and even to man.

Diagnosis.—The diagnosis depends upon the discovery of the sarcoptic mange mite under the microscope. Otherwise it is difficult to distinguish from the psoroptic or chorioptic forms which in some cases may also involve the skin of the entire body. As the mites are in tunnels under the epidermis scrapings should be made deep enough to reach them and as the number of mites found is generally limited several slides should be examined before a negative opinion is given.

Prognosis.—The prognosis is doubtful for unless circumscribed it may take several weeks of persistent treatment to eradicate the mites and heal the skin lesions.

Treatment.—Success in treating sarcoptic mange is largely personal and lies not so much in the agent used as in the thoroughness and the persistency of its application. The following treatment is recommended: The affected animals should be clipped and the manes roached. The removed

hair, which will contain many mites, should be burned. A dressing of sulphur (1 part) and lard or fish oil (4 parts) should then be thoroughly applied to the whole skin. During each of the following four days the patient shoud be exercised to cause the skin to act, or, if available, may be placed in a steam heated chamber until it sweats profusely. On the sixth day apply an emulsion of kerosene (Oss-i), soap solution (1 gallon). On the seventh day the animal should be thoroughly bathed in water to remove the dressings and the stall thoroughly disinfected. This course of treatment is repeated the second week. In obstinate cases a third week's treatment may be necessary. Thoroughness in application is all-important. As a rule, the more thorough the first applications the less necessary become later ones. Creolin (3 per cent.), creolin salve (1–25), creolin liniment, which consists of:

℞—Creolini et sapon viridi	āā	℥iv
Alcohol		Oj

Tar liniment:

℞—Pix's liquida et flores sulph.	āā	℥xvj
Sapon viridi and alcohol	āā	Oij

As further agents may be mentioned tobacco decoction (5 per cent.) and sulphur salve:

℞—Flores sulph.	℥iij
Potassii carbon.	℥iiss
Adipis suilli.	℥iij

Besides the treatment of the patient blankets, clothes and hands of attendants, grooming utensils, stable utensils, harness, etc., which might carry the mites, should be thoroughly disinfected.

Animals which have gone through a mange treatment should be isolated for a time to observe the results. During this period no dressings of any kind should be applied. If lesions reappear treatment should be employed as in a fresh case. During treatment the animals should be well-fed and cared for. Animals which have been in contact with those suffering from mange should be treated with kerosene oil

emulsion. This should be washed off with warm water the following day to be repeated a week later.

Psoroptic Mange.—This form of mange usually begins in the most protected parts of the body, such as the mane, tail, sheath, medial surface of the hind limbs, region of the throat, etc. It may, however, spread over the whole body.

Symptoms.—At the point of origin small, circumscribed areas appear partially bald and covered with a scab. The affected parts are itchy causing the animal to bite and scratch them to such an extent that the skin surface becomes raw. The symptoms in general, however, resemble closely those of sarcoptic mange.

Diagnosis.—An accurate diagnosis is only possible by finding the psoroptic mite under the microscope. As these mites are larger and live on the surface of the body they are more readily collected than the sarcoptic mite. Occasionally both forms of mange coexist and lousiness may also be present.

Prognosis.—The prognosis is more favorable in this than in the sarcoptic form, as the mites are on the surface. However, especially in cold weather, which interferes with the application of the treatment, the case may prove obstinate.

Treatment.—Treatment is the same as for sarcoptic mange.

Chorioptic (Symbiotic) Mange.—This form occurs about the fetlocks especially under the flexion of the joint. In neglected cases it may extend over the whole limb and even to the body where it produces symptoms resembling psoroptic mange.

Symptoms.—The symptoms are similar to those of scratches (squamous and crustated eczema). The affected parts are itchy causing the animal to gnaw them and stamp its feet. Such horses are restless, often difficult to shoe and do not thrive well. Heavy draft horses seem most predisposed and the disease is much more severe in winter than during the summer season.

Diagnosis.—The location and itchiness of the lesions are suggestive. The mites are easily found under the microscope.

Prognosis.—The prognosis is favorable. Under proper treatment a cure is usually attained in about three weeks.

Treatment.—The affected parts should be clipped, rubbed well with a stiff brush and washed with soap and water to remove the scabs. A creolin, coal-tar or sulphur salve suffices to produce a cure.

Mange of Sheep.—*Sheep Scab.*—The most common form of mange in sheep, so-called sheep scab, is psoroptic. However, sarcoptic and chorioptic mites also attack sheep.

Psoroptic Mange.—*Occurrence.*—This form of mange or scab is widely prevalent in the United States. Through the efforts of the Bureau of Animal Industry, with the coöperation of the great sheep-grazing states (Colorado, Montana, Wyoming) sheep scab has been greatly reduced in the past few years.

Symptoms.—Psoroptic mange affects the parts of the skin which are covered with wool. It therefore usually appears in the shoulder region, along the back, croup and along the sides of the chest and neck. It does not involve the ventral chest or abdominal regions.

The first symptoms noted are usually those of itchiness. The sheep are uneasy, bite and rub the affected skin. The wool soon becomes loose and tufted. The itchiness is most noticeable in hot weather, after exercise, in warm stables and at night. If the wool is separated and the skin examined red or yellowish papules the size of a hemp seed are noted; eventually vesicles or pustules, as well as thin, yellowish, fatty crusts and scabs, appear. By confluence large, irregular areas are formed, covered with thick, yellow crusts. The crusts or scales adhere to the wool and by its growth are elevated from the underlying skin. The wool soon becomes tufted and the surface of the fleece uneven; or it falls out, leaving bald areas especially along the back and sides. In shorn sheep or on the bald areas the scales are dry and brown in color. The disease develops rapidly under the fleece, especially in stables, so that in six to eight weeks the whole body may be denuded of wool. When the whole trunk is involved on account of the loss of albumin, unrest, and interference with feeding, the weaker sheep become anemic, emaciated, cachectic, and often die. This is especially true in winter, when the sheep are stabled. After shearing and turning out on grass, temporary improvement is noted.

Diagnosis.—The diagnosis depends upon finding the mites by scraping freshly infected areas where the scabs are soft, placing the scraping on a piece of black paper in the sun and with a hand lens noting the minute mites which are seen crawling toward the edge of the paper. Obviously a better view may be obtained through the microscope. In very old cases (scabs hard and dry) or if dips have been used the mites are not numerous.

Prognosis.—The prognosis in sheep scab is generally good provided prompt and effective remedial measures are taken.

Treatment.—The treatment consists in dipping the whole flock in some solution which will destroy the parasites. Several solutions are recommended for this purpose:

(*a*) Lime-sulphur dip, made by mixing eight pounds of fresh quicklime and twenty-four pounds of sulphur and boiling with thirty gallons of water for not less than two hours. All sediment should be allowed to subside before the liquid is placed in the dipping vat. Before using this mixture it should be diluted with water to make 100 gallons.

(*b*) Tobacco-sulphur dip which is made with the extract of tobacco or nicotin. The strength of the mixture should be no less than 0.05 per cent. nicotin and 2 per cent. flowers of sulphur. For the first dipping of infected sheep the mixture should contain no less than .07 per cent. nicotin.

(*c*) Coal-tar preparation to 50 or 75 parts water. Dipping solutions should be warm (110° F.).

Sheep should be immersed at least once and should remain in the solution for at least two minutes. It is best to dip after shearing. Ten days later the entire flock should be dipped a second time. After dipping, the flock should be placed where no scabby sheep have been for at least four or five weeks.

Sarcoptic Mange.—As this form of mange does not usually invade the wool-covered portions of the skin it is found mostly on the head about the lips, nose, face and ears. It is therefore known as "head scab." More rarely it involves the flexion surfaces of the carpi and tarsi.

Symptoms.—The symptoms are similar to those noted in the horse. Small papules, vesicles, and pustules appear which

rupture and dry to form thin and later thick, gray scabs; from rubbing the skin becomes swollen and creviced.

Prognosis.—The prognosis is good if treated early. In some cases the sheep is unable to eat.

Treatment.—Treatment is the same as for sarcoptic mange in the horse.

Chorioptic (Symbiotic) Mange. — Chorioptic (symbiotic) mange is very rare in sheep and appears around the fetlocks and coronets, usually of the hind feet. Besides an eczematous eruption there is pruritus, causing restlessness and stamping. In rare instances the scrotum or udder may be attacked. Healing usually occurs spontaneously when the sheep are turned out on grass.

Cattle Mange.—In the ox the principal mange is the psoroptic. The favorite seats are the lateral surfaces and crest of the neck, base of the horns, withers and root of the tail. It rarely appears over the whole body.

Symptoms.—The symptoms are those of pruritus and the appearance on the skin of brownish-gray crusts and scabs, the skin becoming thickened and welted. The hair falls out over the infested regions. Excoriations of the skin may be caused by the patient rubbing the part. If generalized the patient becomes anemic, emaciated, cachectic and often dies of inanition. The disease is worse in winter when the cattle are stabled but improves in the spring when they are turned out to graze.

Treatment.—Treatment is the same as for horse mange, avoiding, however, applications which contain mercury.

Chorioptic (Symbiotic) Mange.—This form occurs at the tail root, on each side of the anus, and also on the legs. In rare instances it may appear over the croup, loins, back, side of the thigh and udder. It is characterized by pruritus, scabs, squamæ and loss of hair. It usually does not affect the animal seriously but is worse during winter than in summer.

Treatment.—Treatment is the same as for psoroptic mange.

Mange of Swine.—In swine the only mange is the sarcoptic. This is not a common skin disease in America. The favorite seats are about the head (eyes, cheeks, ears), although it may extend from these parts to the neck, trunk, and limbs.

Symptoms.—The symptoms are those of severe itching, with which is associated a squamous eczema, the skin being covered with bran-like scales which accumulate to form thick crusts. If diffuse, the skin surface assumes a peculiar silvered appearance "as if strewn with guano." The underlying skin is thickened and creviced. If diffuse and of long duration emaciation follows. It is difficult to find the mites under the microscope, as they are limited in numbers.

Treatment.—The skin should be thoroughly scrubbed with green soap and water to soften the crusts. This may be followed by the use of sulphur ointment, creolin liniment, or a tar salve.

Follicular Mange. — Occurrence. — This form of mange which is common in dogs, where it assumes two forms, the squamous and the pustular, is rare in other animals, with the possible exception of the pig.

Etiology.—The hair follicle mites, Demodex folliculorum, live in the sebaceous glands and hair follicles. Following their invasion pus cocci enter, leading to abscess formation. In swine the Demodex folliculorum (var. suis) is the offender. The region of the snout, neck, ventral portion of the chest, the abdomen and inner parts of the thighs are the favorite seats. In these regions pox-like pustules to hazel-nut-sized boils or abscesses as large as walnuts appear. In the center of an abscess hundreds of the mites may be found.

Diagnosis.—Diagnosis depends upon finding the mites under the microscope.

Prognosis.—The prognosis in swine is not good, as the disease is very obstinate. An operative treatment which consists in removing the affected skin is recommended.

HERPES. RINGWORM.

Definition.—A contagious skin disease due to a fungus and characterized by the formation of round, sharply defined areas covered with scales and short, uneven hair stumps.

Occurrence.—Ringworm is very common in cattle, especially during the winter season, when they are stabled. It is sometimes, therefore, known as "barn itch." While more or

less troublesome it is a benign disease. Horses are also affected with it, as are sheep and pigs.

Etiology.—Ringworm is caused by the fungus Trichophyton tonsurans. This fungus is made up of threads (hyphæ) which are interwoven to form mycelia and large, round, light-refracting spores which predominate. The fungus lives in the hair sac, where it produces an inflammation of the hair follicle, causing the hair to fall out, and also within the hair shaft, which it makes brittle, causing it to break off. Infection is spread by direct contact or through intermediary agents, such as posts, stall partitions, etc., against which affected animals have rubbed themselves. Harness, saddles, and grooming utensils are also carriers.

Forms.—Four forms of herpes tonsurans are distinguished clinically, depending upon the species of animals attacked and the location and age of the lesion: (*a*) Herpes tonsurans which is characterized by round, bald spots without inflammation of the skin. (*b*) Herpes circinatus the center healing but the periphery remaining diseased thus forming a hoop-shaped, circular lesion—hence the name "ringworm." (*c*) Herpes maculosis occurring on the skin of old animals and leading to alopecia and a superficial chronic dermatitis with desquamation of the epidermis which forms slate-gray colored, asbestos-like scales. (*d*) Herpes vesiculosus which attacks the thin skin of young animals (calves, lambs). This is a dermatitis with vesicle formation. The vesicles soon rupture forming thick, yellow, scaly scabs or crusts in the neighborhood of the mouth, face and eyes.

Cattle.—In adult cattle herpes usually attacks first the head and neck, where it forms small, well-defined, round, bald spots which by confluence may reach the size of an open hand. These areas are covered with grayish-white, asbestos-like crusts. On black skin the crusts are thicker than on white. The skin is sometimes thickened and creviced. Healing usually takes place under the crusts in two or three months. When there is much pruritus, causing rubbing and scratching, secondary lesions form, prolonging the course of the disease several months.

Diagnosis.—A positive diagnosis can be made only with the microscope. In sucking calves herpes attacks the skin around the mouth. There appear round spots covered with yellowish scabs made up of bran-like scales.

Horses.—The favorite seats of herpes in the horse are the head, back, shoulders, and croup. As a rule it assumes the form of round, well-defined, bald spots the size of a 25-cent piece. Usually there is no pruritus. In rare instances a herpes vesiculosus occurs on the sheath and inner surface of the hind limbs, with the formation of scabs and crusts.

Treatment.—The treatment depends somewhat on the extent and form of the eruption. When feasible it is advisable to shear off the hair and soften up the crusts with green soap. This may be followed by painting the areas with tincture of iodin or salicylic acid in alcohol (1 to 10). In horses mercuric ointment is useful. In obstinate cases nitric acid may be tried. However, most cases yield to treatment with any disinfectant. One attack of herpes produces a certain immunity against subsequent ones.

FAVUS.

Favus is a contagious skin disease common in man but very rare in animals, caused by a fungus similar to the Trichophyton tonsurans and called the Achorion Schoenleinii. It forms round, thick scabs on the skin, which, being depressed in their centers, are saucer- or shield-shaped. The scabs have a sulphur-yellow color, especially toward the center. They appear upon the head, abdomen, and feet of carnivora. In fowls the comb and wattles are the favorite seats. The treatment is the same as for herpes.

CONTAGIOUS PUSTULOUS DERMATITIS. CONTAGIOUS ACNE.

Definition.—Contagious pustulous dermatitis is a benign pustular exanthema of horses which occurs in the regions of the harness and saddle rests.

Etiology.—The disease is not very common, but occurs occasionally enzoötically. The pustules are caused by the acne bacillus. Infection takes place usually through intermediary agents, such as harness and saddles.

Symptoms.—In the regions noted round or oval swellings the size of a 25-cent piece appear, which in a day or so become covered with small, hempseed-sized vesicles, which rapidly form pustules. The pustules usually break in twenty-four to thirty-six hours, forming thick, yellow, sticky crusts which heal in about two weeks. There is usually little or no pruritus. In severe cases boils or even abscesses may form as in simple acne. The abscesses may rupture, forming ulcers which heal under the scab. In rare instances an inflammation of the lymph vessels and glands complicate the case. Healing in these cases requires one to two months. This form of contagious pustulous dermatitis may resemble skin glanders. However, even in the most severe cases there are no general symptoms.

Diagnosis.—The location, absence of itching and contagious character differentiate contagious pustulous dermatitis from acne or other suppurative conditions of the skin due to traumatism.

Treatment.—The patient should not be worked, the sick isolated from the healthy, and the pustules opened and treated with antiseptics. The harness, saddle, and stable should be disinfected.

HYPODERMA LINEATUM. WARBLE FLIES.

The cattle bot, or warble fly, one variety of which appears in the United States, produces serious discomfort to cattle and damage to hides, due to the perforations which they cause in the skin of the shoulder, back, and breast.

Life History.—The female gadfly deposits her eggs in summer, while the cattle are on pasture, in the region of the heel where they are licked off by the animal reaching the mouth and throat where they hatch. The larvæ perforate the gullet and, following the course of the bloodvessels in the mediastinal tissue, reach the vertebral foramina, through

which they pass into the vertebral canal, eventually emigrating to the subcutaneous tissue of the back. In the subcutus they become encapsuled in a sac of connective tissue, forming pigeon-egg-sized enlargements which are called warbles. When fully developed the following spring they perforate the skin, pass out through the openings, drop to the ground, into which they burrow and pupate, finally emerging as adult flies. As many as 50 to 100 of these larvæ may perforate the skin, causing the hide, which is called "grubby," to be docked one-third in value on the market. Occasionally they may produce emaciation.

Treatment.—The treatment consists in applying turpentine to the opening over the warble. Those not killed by this method may be pressed out by hand and destroyed.

Prevention.—Applying various disinfectants to the backs of cattle is of no value from a prophylactic standpoint.

LICE.

Definition.—Lice are blood-sucking parasites (hæmatopinus). Each one of the domesticated animals harbors its own species. They are frequently found on cattle (Hæmatopinus eurysternus), and horses (Hæmatopinus equi). However, occasionally lice will pass from one host to another of a different species. Chicken lice, for instance, will attack horses and human beings. Lice are very prolific. It is said that a single pair may produce in three months 125,000 individuals. Lice produce itching, loss of hair, and give the animal an unthrifty appearance. While they may be found on any part of the body they first appear about the neck. As they are large enough to be seen by the naked eye, if the light is good, the diagnosis is usually easy. Even the eggs or nits are visible as small, ovoid bodies attached to the hairs.

Treatment.—Several remedies will kill lice. For horses one of the most valuable is gray mercurial ointment. Creolin (2 per cent.) is also good. An infusion of tobacco made by boiling tobacco (℥iij) in water (Oij) is recommended. A great favorite is kerosene emulsion made by dissolving soft soap (Oij) in boiling water (Oij) and adding kerosene (Oj). This mixture should be churned or stirred violently and 3

quarts of water added to it before using. Pure kerosene is too strong and causes loss of hair. Stavesacre seeds (℥ij) boiled in water (Oij) is an efficient agent to destroy lice.

TICKS. IXODOIDEA.

Definition.—Ticks are skin parasites which, however, do not remain parasitic throughout life. The females especially attach themselves to the skin into which they bore. The most important tick is the Texas cattle tick (Margaropus annulatus) which transmits the germ of Texas fever. While common on cattle in the Southern States it is only an occasional visitor in the North, where it is unable to withstand the cold winter. Ixodes reduvius, which is parasitic on sheep and cattle, is not uncommon. The same treatment recommended for lice will destroy ticks. The best treatment for ticks in sheep is the arsenical dip. (For eradication of the Texas fever tick see Texas Fever.)

SPINOSE EAR TICK.

A troublesome tick found in the semiarid west (Oklahoma, New Mexico, Arizona, California, and occasionally as far north as Oregon) is the spinose ear tick (Ornithodoros megnini) which infests the outer ear of cattle, horses, dogs, and sheep. In badly infested areas even wild animals, especially jack rabbits, are attacked.

This tick does serious harm to live stock causing filling of the ear with masses of debris, ear wax and ticks, and not infrequently leads to ulceration, external otitis, a condition known locally as "ear canker." In badly infested animals emaciation, impaired milk secretion in cows, nervous and digestive disorders have been noted. Old range cows and run-down animals have been known to succumb during the late winter or early spring.

Life History.—The spinose ear ticks enter the ears of animals when small, six-legged larvæ invisible to the naked eye. They usually attach themselves to the tender skin inside the ear, just below the hair line. In one to two weeks they have grown to several times their original size and

acquired a yellowish-white or pink color. The larvæ moult and emerge as nymphs provided with eight legs, the body covered with small spines (hence the name). The nymphs remain in the ear from one to seven months. They then drop off to the ground, seek cracks and crevices in buildings, fences, trees, etc., in which they moult again and develop into adult ticks, copulate, and the females lay their eggs. The eggs hatch under favorable conditions in about ten days. The larvæ ("seed tick") seek animals soon after hatching. Adult females which have never mated may live a year; seed ticks which have found no host three months. The adult tick has no spine.

Symptoms.—While mild infestations may give rise to no symptoms and thus be overlooked, severe ones produce rather marked symptoms. The infested animal will shake its head, repeatedly turn it from side to side, inverting first one ear and then the other. The patients rub or scratch the ears repeatedly. The badly infested animal appears unthrifty, is losing weight and may show symptoms of a gastro-intestinal catarrh.

Diagnosis.—If the ticks are very numerous, the ear packed full of them, they are easy to detect. In milder infestations, however, they often escape detection, as they are apt to hide under folds of skin or crawl into the depths of the ear. As their excretions accumulate, mixing with the ear wax, masses of this mixture may entirely occlude the ear canal.

Where ear tick infestation is suspected, and no ticks are visible, the ears should be thoroughly probed with a 6-inch piece of baled-hay wire, one end of which is bent into a $\frac{1}{2}$ inch loop.

Treatment.—The best treatment is to syringe out the affected ears with a pine-tar-cottonseed-oil mixture (pine tar 2 parts; cottonseed oil 1 part; mix when warm). About one-half ounce should be injected. Before treatment all undue wax accumulation should be scraped out with the wire probe in order that the mixture come in contact with the ticks, otherwise it will do no good. One treatment usually suffices to produce a cure. Reinfestation, however, occurs as more seed ticks are picked up. It is recommended to treat late in the fall or in early winter to protect against late winter and early spring losses.

Prevention.—As the ticks occur upon hosts of many different species, some of them wild animals, and are thus widely disseminated, complete eradication offers almost insurmountable difficulties. Dips which kill ordinary ticks, or the Texas fever tick, are worthless for the spinose ear tick.

THE SCREW FLY.

The screw fly (Compsomyia macellaria) is a small, bluish-green fly with a brown head and three black longitudinal stripes on the thorax. The female deposits her eggs in wounds, where they hatch in a few hours (larvæ, maggots), the maggots burrowing into the tissue, where they remain for a week. They then escape from the wound, reach the ground, pupate, and change to adult flies. In some regions of the South they are very troublesome, infesting the smallest wounds and causing serious losses.

Treatment.—Pure creolin should be injected into the wound. Fresh wounds can be protected by a tar covering.

CHICKEN LICE.

The red chicken louse (Dermanyssus avium) has as its host chickens and other feathered animals. It will, however, attack horses and cattle, producing eczema. In cattle it has been known to enter the ears, causing an external otitis. In rare instances lice penetrate the middle ear, causing symptoms of cerebral excitement.

SKIN FILARIA.

Thread-worms, which are parasitic, invade the skin and subcutaneous tissue of animals. The following varieties are the most common:

Filaria Irritans.—This is a silver-white larva 2 to 3 mm. long, the adult form of which is unknown. It lives in the subcutaneous connective tissue of the horse and is the cause of a skin disease commonly known as "summer sores," or granular dermatitis. The lesion manifests itself first by little

nodules which appear in the harness rests, legs, or other parts of the body. The nodules ultimately erupt, leaving behind ulcers which are covered with reddish-brown granulations. On careful examination small, hempseed-sized, yellow, cheesy, or calcified foci may be noted. In these centers the filarial larvæ are found. The ulcers show a tendency to spread and are very obstinate to treat. This is due to biting and rubbing by the patient and the irritation caused by flies. The ulcers usually heal after the fly season, but recur the following summer.

Treatment.—Treatment consists in preventing the animal from rubbing or biting the part and flies from reaching it. It is recommended to paint the ulcer with a thin layer of sulphid of arsenic, which forms a dry scab, under which healing occurs. Picric acid (1 per cent.) and glycerin are also good. Other remedies are chloroform, ether, or iodoform applied daily. Sometimes the hot iron is effective, as is curetting or even the total extirpation of the affected area.

Filaria Hemorrhagica.—This is a fine, thread-like worm 28–70 mm. long which is parasitic and lives in the subcutaneous and intermuscular connective tissue of Oriental horses. It causes during the summer months skin hemorrhages. The blood raises the epidermis or skin in the form of a hazelnut-sized or small hematoma which ruptures in one to two hours, its contents flowing over the skin. Usually a number of these nodules occur close together so that the invaded area is covered with blood. The condition disappears in winter to recur the following summer. Healing usually occurs spontaneously. The disorder is not serious, although objectionable.

Treatment.—The treatment consists in washing the skin with an antiseptic and protecting the bloody area from the harness.

LIP-AND-LEG ULCERATION OF SHEEP.

Necrobacillosis.

Definition.—Necrobacillosis is an inflammatory process which terminates in necrosis of the skin and mucous mem-

branes attacking the mouth, lips, legs, feet and external genital organs of sheep.

Occurrence.—The disease is closely allied to necrotic stomatitis of calves and pigs. It attacks sheep on the Western ranges, particularly in Montana and Wyoming, where it sometimes assumes a malignant form, causing considerable losses through deaths, lost service of bucks, and abortion in ewes. While during favorable weather and with abundant feed the disorder is mild, where opposite conditions prevail it is often malignant, leading to the loss of from 10 to 20 per cent. of the sheep herd. The disease has been introduced into the Eastern States by the importation of western sheep. It also exists in England, on the continent of Europe, and has been reported from New Zealand.

Etiology.—The cause of the disease is the Bacillus necrophorus, which enters skin lesions often made by cactus or other sharp pointed particles of plants. The disease is most frequent during cold seasons and among sheep which are pasturing among thistles, cacti and briers. It is transmissible both by natural and artificial inoculation.

Symptoms.—From a clinical standpoint four forms are described: (*a*) The lip-and-leg, (*b*) the venereal, (*c*) the foot-rot, (*d*) the sore mouth of lambs.

(*a*) *Lip-and-leg Form.*—The first stages are those of acute inflammation of the skin, usually of the lips. Pustules develop which undergo puriform softening, erupt and lead to ulcers. The ulcers are usually covered with exudate which dries to form dark-gray-colored crusts. By confluence a large, irregular shaped scab forms along the lip margins and extending upwardly in front to the nasal openings. The lips are swollen and present a scabby or warty appearance. The appetite usually remains good though the prehension of food is difficult owing to the sensitiveness of the lips. In some cases the cheeks may also be involved or even the eyelids. Occasionally the mucous membrane of the nose and mouth is involved.

Quite commonly lesions occur on the legs. The favorite seats are about the coronet, under the fetlock or in the neighborhood of the fetlock joint. The ulcers upon the legs are

similar to those found upon the lips, they are covered by a thick, dry crust which when removed exposes a granulating surface covered with pus.

(*b*) *Venereal Form.*—In this form the external genital organs of both males and females are involved. It may be associated with the lip-and-leg form, but not infrequently occurs independently. In males the skin of the sheath especially at its orifice is inflamed. The first symptom is the appearance of one or more light-yellow-colored centers, necrotic areas, which appear at the junction of the skin and mucous membrane of the prepuce. Soon there develops an ulcer which usually extends outwardly over the skin rather than inwardly over the mucous membrane. By coalescence the whole swollen sheath may be covered with ulcer. Occasionally the pendent portion of the prepuce may slough. In wethers the penis is rarely if ever attacked. In bucks, however, the necrosis attacks the penis, which swells ("big pizzle"), becomes ulcerous on its surface, and occasionally gangrenous, the distal portion of the organ not unrarely sloughing off.

In ewes the ulcerations occur in the skin and mucous membrane of the vulva, the ventral surface of the tail, and the perineal region. Occasionally the vagina may be involved, leading to a discharge.

(*c*) *Foot-rot Form.*—The foot-rot form involves at first the interdigital spaces, the erosions usually making their first appearance at the heel. The inflammation invades the hoof matrix leading to the discharge of a thin ichor which has a very characteristic, pungent, penetrating odor. The foot-rot form may attack any sheep in the herd irrespective of age or sex.

(*d*) *Sore Mouth Form.*—This form is seen in lambs during the fall of the year. The symptoms are similar to those of the lip form in older sheep. The lips of the lambs swell, prehension of food becomes difficult, and the patient falls off in flesh and remains stunted in growth. At the junction of the mucous membrane and skin of the lips, nodules or necrotic areas appear. By confluence large, diffuse, fissured scabs form covering in some cases the whole muzzle. If

the scabs are removed raw, bleeding surfaces or pus-covered, ulcerous areas are exposed. In some cases the inflammation spreads to the mucous membrane of the mouth forming ulcers or soft, red, fungoid proliferations. The expirium is very offensive, resembling the odor of Limburg cheese.

Treatment.—Once the disease has broken out in a herd or band of sheep, treatment should be begun early and applied energetically. In milder cases of the lip-and-leg form the scabs and shreds of tissue from the diseased areas should be scraped off and antiseptics applied to the raw surface three or four times weekly. A mixture of creolin (5 parts), sublimed sulphur (10 parts), mutton tallow, vaselin, or lard (100 parts) mixed to form an ointment has been found very serviceable. In aggravated cases the scab should be removed and all diseased tissue from the ulcer scraped away. The raw surface may then be touched with zinc chlorid (10 per cent.) or nitric acid (15 per cent.). These caustics should be applied very carefully. The unskilful and indiscriminate use of them will do more harm than good. As it will require a month or more to produce a cure in severe and chronic cases the expense of the treatment may exceed the value of the sheep. Such cases had best be dispatched.

Where a large number of range sheep are affected with the leg or foot-rot form, hand-treatment is not always feasible. In these cases the sheep may be waded through a trough containing creolin or sheep-dip solution (5 per cent.) three times a week. Bad and obstinate cases which do not yield to these foot baths may be hand-treated. In the foot-rot form it is often necessary to use the knife skilfully to remove all diseased or loosened horn.

The venereal form of the disease requires careful treatment. In bucks the diseased penis should be forced out of the sheath and the necrotic areas on its surface carefully cauterized with zinc chlorid or nitric acid. This may be followed by daily injections of permanganate of potash (1 to 500) or a sheep-dip solution (1 per cent.). In females the same treatment may be applied to the vulva or vagina. Abscesses containing inspissated pus should be opened, their contents evacuated and antiseptics applied. Ulcers of the mucosa of the mouth

of lambs may be treated with creolin (1 per cent.) or boric acid solution (3 per cent.). Advanced, aggravated cases which obstinately resist treatment should be killed.

After-treatment consists in giving the sheep access to medicated salt (crude carbolic acid ℥iv, poured over ordinary barrel salt, 12 quarts, and thoroughly mixed). It is also recommended to dip all recovered animals before turning them out on uninfected pastures or premises. A complete disinfection of all pens, corrals, walls, partitions, racks, and troughs should be made. The manure and surface soil of the corral should be removed and the ground sprinkled with disinfectants.

Prophylaxis.—Healthy sheep should not be permitted to pasture on infected ranges until a winter has passed, after which the pasture is safe. Obviously with range sheep this precaution cannot always be employed. No new sheep should be introduced into the herd without being subjected first to a two weeks' quarantine. If no cases of lip-and-leg ulceration develop in this time it will be safe to place them with the original flock. The sheep herd should be carefully supervised and frequently inspected to find any chance cases of this disease. All sick sheep should be immediately removed and if possible given proper hand-treatment.

BIGHEAD OF SHEEP.

Definition.—Bighead is a peculiar condition affecting western sheep and is characterized by the appearance of swellings about the head and ears.

Occurrence.—Bighead is not an uncommon disease in the Western States where sheep-raising is an important industry. Cases are reported from Idaho, Nevada, Utah, and Wyoming. It resembles the so-called buckwheat poisoning (fagopyrism) of Europe. The disease is not communicable. Neither sheep nor other animals can be infected by natural or artificial means. While some outbreaks are comparatively mild, not infrequently the affection assumes a malignant form which leads to considerable losses causing it to become a matter of importance to sheepmen.

Etiology.—The cause of bighead has not yet been determined. Outbreaks occur usually during the spring and early summer while the sheep are being driven from the winter to the summer ranges and before they are sheared. Climatic conditions evidently predispose to the disorder, which is most apt to appear following a cold, stormy night, especially if the sheep are driven the next day in the hot sunshine. The disease is most apt to affect adult sheep; it is rarely seen in young lambs. The condition of the sheep seems to have little to do with the occurrence of an outbreak; it is as apt to attack the well nourished as it is individuals poor in flesh. Buckwheat feeding has failed to produce in experimental animals symptoms of the disorder.

Symptoms.—The symptoms which precede the appearance of the swellings are jerking movements of the head; the sheep shakes its head and rubs it against objects. The eyesight seems to be impaired, the sheep often running into other sheep and objects found in its path. Symptoms of cerebral excitement are not uncommon, in some instances the sheep show maniacal phenomena. Obviously sheep so affected often leave the flock, become lost, and die of exhaustion and exposure, or become the prey of coyotes.

Later it will be noted that the ears begin to turn red and become swollen. Finally the swellings extend over the cheeks and face, closing the eyes, or in some cases, if the retrobulbar tissue becomes involved, may force the eyeballs from their sockets. The swellings may become very tense and drops of serum ooze from them. In some cases the lips, cheeks, and tongue are so swollen that it is impossible for the animal to eat or drink. Dyspnea is a common symptom. The temperature ranges from 104° to 107° F. The skin often sloughs over the swollen areas, and the wool over the entire body may be shed. Pregnant ewes frequently abort. Sheep which recover are usually left permanently unthrifty.

Treatment.—Internal treatment (belladonna, turpentine, strychnin, arsenic, digitalis) has been found useless. On the other hand, smearing the head of the affected sheep with vaselin or olive oil, especially if the sheep are permitted to rest in the shade, has been of service. Scarifying the swellings does no good, and exposes the parts to further infection.

Prophylaxis.—The disorder is prevented by not driving the sheep too far or too rapidly during the heat of the day, especially before shearing in the spring. Affected sheep should be dropped out of the herd and the entire band allowed rest in the shade if possible. Sheepmen, believing the trouble due to poisonous plants, often, when a case of bighead develops in a band, rush the sheep to get them away as rapidly as possible from the region in which the supposed poisonous plants exist. This in itself tends to produce the trouble they seek to avoid.

PART XI.

INFECTIOUS DISEASES.

CHAPTER I.

ACUTE GENERAL INFECTIOUS DISEASES.

ANTHRAX.

Definition.—Anthrax is an acute, febrile, infectious disease caused by the Bacterium anthracis affecting all domesticated animals with the probable exception of fowls. It is characterized on postmortem by splenic enlargement and serohemorrhagic infiltration of the subserous and subcutaneous connective tissues.

Occurrence.—Anthrax is a disease largely confined to permanently infected districts. Generally speaking it is rare in the United States, occurring only sporadically in the North, although in some parts of the South, especially along the rich lowlands of the lower Mississippi, it appears enzoötically, attacking numbers of horses and mules. The disease is more common in herbivora than in either omnivora or carnivora. Birds are probably never infected naturally, although the disease may be with difficulty artificially transmitted. The pigeon, however, forms an exception, being very susceptible to infection with anthrax cultures.

Etiology.—The Bacterium anthracis, which occurs in the tissue in the form of rods and in the gastro-intestinal tract and outside the body in the form of spores. The spore possesses a remarkable vitality and may remain virulent for months in soil, manure, foodstuffs, water, and in the blood, hide, wool, or hair of animals which have died of the disease.

Natural Infection.—(*a*) *Digestive Tract.*—In animals the usual mode of infection is through the digestive tract, the spores being taken in with the food and water of infected districts. This applies particularly to grazing sheep and cattle, especially if feeding on low, wet pastures subject to overflow. Certain fields, notably those in which the carcasses of anthrax animals have lain or were buried (shallow graves), or contaminated with the droppings or blood of patients suffering from the disease, are especially dangerous in this regard. It is probable that healthy animals may carry spores in the digestive contents without becoming infected themselves, their droppings, however, polluting the soil, drinking water, and forage, thus infecting other less resistant animals. The blood of the living or dead anthrax animal is also infective. Soil and water can be contaminated by the offal of tanneries, hair- or rag-sorting establishments. Flesh-eating animals can infect themselves by eating meat or blood containing anthrax spores. In this way dogs and swine take the disease. As fresh meat and blood usually contain only bacilli, which are destroyed by the digestive juices, it is not as dangerous as when stale. However, if very large quantities are eaten, infection may follow, some of the bacilli escaping the destructive influences of the gastric fluids. In swine the crypts of the tonsils may serve as points of attack for the spores taken in with the food and water. Milk does not usually contain either spores or bacilli, but at times, especially when the milk is bloody, they may be present.

(*b*) *Respiratory Tract.*—The inhalation of dried spores from contaminated dust, wool, hair, or rags is a common mode of infection in man. While this is also probable in animals, no proof is available.

(*c*) *Skin.*—An infection through the intact skin is not probable. When the skin is wounded, however, it may follow.

The enzoötics of anthrax among horses and mules in the lower Mississippi Valley are said to be induced through skin infection brought about by blood-sucking insects. Recent experiments made by Morris of the Louisiana Agricultural Experiment Station seem to prove that the horn fly (Hæmatobia irritans), the horse fly (Tabanus sp.), and

mosquitoes (Psorophora sayi; Aedes sylvestris) do transmit anthrax.

Necropsy.[1]—As man is quite susceptible to anthrax, every precaution should be taken in holding necropsies to guard against infection. The blood, flesh and digestive contents should not be permitted to pollute the forage or soil and carrion birds, dogs, insects, etc., kept aloof until the carcass and offal are made innocuous.

The cadaver is usually greatly bloated, rapidly decomposing and rigor mortis little developed. The mucous membranes are cyanotic, the rectum protruding and dark blood flows from the natural openings. If swellings in the skin and subcutis are present, they appear on cut surface hemorrhagic-gelatinous, or, if older, lardaceous, the overlying skin discolored and in part necrotic. Throughout the body the organs show petechiæ, ecchymoses and blood extravasations. Collections of blood-stained fluid are noted in the abdominal and thoracic cavities, and in the pericardium. The spleen is three to six times its normal size, the parenchyma black and soft, even fluid. Spontaneous rupture of the capsule sometimes occurs. The blood is dark, varnish-like, staining the fingers and little coagulated. The lining of the aorta is diffusely reddened. The intestinal mucosa, especially in the small bowel, is edematously swollen, jelly-like and sometimes several centimeters thick. Necrosis of the superficial layers is sometimes noted. Peyer's plaques and the solitary follicles are swollen projecting into the lumen of the intestine as dark red elevations. The lungs are edematous, congested. The mucous membrane of the larynx is swollen, congested, and ecchymosed. The lymph glands are swollen and blood-shot.

In the spleen pulp and blood, lymph glands and parenchymatous organs, anthrax bacilli are found.

In swine the necropsy lesions are confined mostly to the throat. The spleen is quite often normal in this animal.

Symptoms.—In general, anthrax is characterized clinically by its sudden appearance, stormy course leading to death in

[1] The postmortem lesions vary greatly in anthrax. In peracute cases the changes are very little marked, and the bacilli not numerous. The description given above applies to the usual acute or subacute case and may be considered typical.

one to two days, high fever, severe general disturbance, tendency for petechiæ to appear on mucous membranes, bleeding from natural openings, skin edemas, colic, dyspnea, cerebral excitement, and the presence of the Bacterium anthracis in the blood.

Period of Incubation.—The period of incubation is from one to fourteen days. The symptoms vary greatly, depending upon the severity of the attack and the resistance of the patient, which factors largely determine the course of the disease. They also vary with the point of attack, depending on whether the lungs, bowel, or skin are primarily involved. It is customary clinically to classify anthrax as:

(*a*) *Peracute Anthrax* (*Apoplectic or Explosive Anthrax, Anthrax Acutissimus*).—As a rule, this form appears in the beginning of the outbreak. The animals are usually found dead, or die in a few minutes after symptoms of asphyxia, the patient dropping to the ground in convulsions, foam coming from the nostrils and blood from the natural openings.

(*b*) *Acute and Subacute Anthrax* (*Anthrax Acutus et Subacutus*).—This form lasts usually ten hours to several days. The patient shows high fever from 105° to 107° F. The pulse is rapid, weak, and irregular, the heart beat tumultuous. The respirations are dyspneic and sometimes stertorous. The visible mucous membranes are cyanotic and dotted with petechiæ. The patient may at first show symptoms of cerebral excitement, which are soon followed by stupor and vertigo. Quite often the patient is down, unable to rise. In some cases (especially in horses) marked colic symptoms occur, but bloating is rarely noted. In the ox it may occur. In the earlier stages there is constipation, the animal straining as if to defecate, the rectum protruding; bloody diarrhea usually follows. The urine in the latter stages may be dark red and contain blood. In milk-giving animals the milk flow stops during the high fever; in rare instances the milk is bloody and bitter to the taste. In pregnant animals abortus occurs. Death usually ensues under asphyxia. While recovery is rare, toward the end of an outbreak a few cases may get well, the symptoms rapidly subsiding.

(*c*) *Cutaneous Anthrax* (*Malignant Carbuncle or Pustule*).—This form is commonest in horses and mules. Acute, edematous swellings appear on different parts of the body. The favorite seats are under the throat, on the chest, flanks, and loins. The swellings are at first hot and painful, but later become colder and less sensitive. They are usually flat and subcutaneous, and in the early stages the overlying skin and hair may appear normal. Thus they may be overlooked. When swellings occur in the throat, symptoms of edema of the glottis follow. In swine the disease usually involves the throat with swelling of the parotid and laryngeal regions, later extending to the face. The patient shows dyspnea and dysphagia. The mucous membranes are cyanotic and show petechiæ; the same symptoms may also occur in the skin. Death results from asphyxia.

Course.—The course in peracute cases, as noted, is very rapid, lasting only fifteen minutes to one hour. In acute cases it may last one to three days, and in subacute ones a week or more, exceptionally ending in recovery. In the horse the cutaneous and bowel (colic) forms seem to predominate; in the ox anthrax occurs as a general febrile disease with a tendency to bowel hemorrhages, skin swellings being rare. In the sheep the course is usually stormy, the disease rarely locating in special organs, and assumes the form of a pure septicemia.

Diagnosis.—*Intra vitam* a diagnosis of anthrax can rarely be made with any degree of certainty. As a rule a necropsy must first be held and a bacteriological examination made of the blood and parenchymatous tissues (spleen pulp). Clinically, anthrax resembles certain forms of forage poisoning, hemorrhagic septicemia, blackleg, malignant edema, and Texas fever. Peracute cases might easily be confused with edema of the glottis, cerebral congestion (sunstroke), and acute pulmonary edema. Acute poisonings might also be taken for it (temperature high here only in the latter stages). It is sometimes sufficient to draw blood from the suspected patient (ear or foot) and inoculate with it a guinea pig or white mouse, which, if the blood contains anthrax bacilli, will succumb in twenty-four to thirty-six hours. The blood and

spleen pulp will contain the capsulated rods, which may be determined by proper staining.

Prognosis.—The prognosis of anthrax is bad. The mortality is 70 to 90 per cent. In subacute cases recovery takes place occasionally, and usually quite unexpectedly, beginning on the second or third day. Nearly all peracute and acute cases die. In cutaneous anthrax an early surgical treatment of the carbuncle is occasionally life-saving.

Treatment.—A medicinal treatment is rarely of value. Internally coal-tar products (creolin, lysol ʒi) five times daily are recommended. Turpentine, chlorin water, hydrochloric acid, and calomel have been tried with small success. Surgically the carbuncles may be slit open and injected with antiseptics (bichlorid of mercury 1 to 2000 in alcohol for horses; iodin tincture in cattle).

Vaccination.—In infected districts only should protective vaccination with living anthrax bacteria or spores be practised, as the indiscriminate and unskilful use of such vaccines can readily lead to a permanent infection of the premises and the further spread of the disease. The Bureau of Animal Industry has perfected a satisfactory method of protecting against anthrax. It consists in giving healthy animals (cattle or horses) simultaneous injections of (*a*) an immunizing serum, and (*b*) a vaccine prepared from anthrax spores. The animal is thus immunized by a single "double treatment," requires to be handled but once, and the losses following the administration have been small. The vaccine, consisting of spores, has excellent keeping qualities, retaining its potency for a long time. The dose of the serum and spore vaccine of the Bureau of Animal Industry is: Serum (10 c.c.), injected subcutaneously into one side of the body, followed immediately by a subcutaneous injection of spore vaccine (1 c.c.) on the other side. Sheep which are highly susceptible to anthrax are given in similar manner serum (10 c.c.) and spore vaccine ($\frac{1}{4}$ c.c.). No animals showing high temperature should receive spore vaccine. Such an animal should be separated from its fellows and injected subcutaneously with serum alone (30 to 100 c.c.). Animals treated with serum alone should be revaccinated several

weeks later with both serum and vaccine. Older methods of protective vaccines against anthrax are the Pasteur method and the Sobernheim. The former requires two treatments several days apart which postpones the immunity until both are given or about ten days. The losses following the use of this method are not insignificant. The latter is a simultaneous serum-vaccine method and produces immediate immunity. The vaccine, however, does not keep well.

When anthrax breaks out in a herd the healthy animals should be removed from this pasture and the affected ones confined to a limited area on the pasture in which they are found. Give large doses of anthrax serum alone to all animals carrying temperature. Feverless animals may be given the serum-vaccine treatment. Those given serum alone may be given serum-vaccine after a lapse of ten days.

Control.—The principal factors in the control of anthrax are:

(*a*) The proper disposal of all anthrax carcasses. They should not be skinned nor the flesh fed to dogs or swine. Opening them should be avoided.

(*b*) The prompt treatment of herds in which anthrax occurs with serum-vaccine.

(*c*) The prevention of outbreaks in animals pastured on known infected land by a regular seasonal vaccination.

(*d*) The tile draining and cropping of infected land to eliminate wet places, stagnant pools, which may not only harbor anthrax spores but form the breeding places of insects which may be carriers of infection.

(*e*) Hair, wool, hides, infected harness, stable utensils, etc., should be disinfected by heat or a formalin solution (5 per cent.). Infected premises, barns, sheds, stables, etc., should be thoroughly disinfected.

(*f*) Healthy animals in the neighborhood of infected ones should be protected against blood-sucking insects which may be carriers of anthrax.

MALIGNANT EDEMA.

Definition.—Malignant edema is an acute, fatal, infectious disease, characterized by the formation of edematous swell-

ings, which later become emphysematous and occur in the region of a wound infected with the specific anaërobic Bacillus œdematis maligni.

Occurrence.—The disease is widely distributed. It affects mostly horses, following accidental or surgical wounds. In cattle it occurs from obstetrical operations, and in sheep may result from cuts received during shearing. In the United States it is one of the commonest wound-infection diseases extant.

Etiology.—The Bacillus œdematis maligni, which is found in both the rod and spore stages in soil, manure (droppings), dust, decomposing vegetable matter and polluted water.

Natural Infection.—Horses and sheep seem most susceptible to the infection, while cattle and swine offer more resistance. The infection is always through a wound (surgical or accidental) in the skin or mucous membrane. In practice malignant edema most frequently follows surgical operations (poll evil, castration, phlebotomy), the use of the unsterilized hypodermic syringe, rough obstetrical manipulations, skin cuts from careless shearing of sheep, etc. It may also follow accidental wounds due to wood splinters, nails, or other penetrating objects which carry the infection. Feeding and inhalation experiments with the bacilli and spores of malignant edema gave negative results. The intact skin and mucous membrane are not infectable. Infection through a necrotic area (ulcer) is probable. Normally granulating wounds are, however, very resistant to infection. Inoculations with pure cultures are not successful unless phagocytosis is overcome by mechanical (sand particles) or chemical agencies (lactic acid) or the toxins of other bacteria which may be present (staphylococci).

Symptoms.—In the neighborhood of the infected wound a rather diffuse, edematous swelling appears. The swelling is at first painful, firm, and hot, but later becomes softer, less painful, and on palpation crepitates. It spreads rapidly and in a few hours can involve the whole body and head. In a horse so afflicted the patient assumes the appearance of a hippopotamus. With the advance of the external swelling dyspnea develops with foamy nasal discharge (lung edema),

cyanotic mucous membranes, and rapid, weak, irregular pulse. The temperature is elevated early and does not sink to normal until the patient becomes moribund.

In cows infected during obstetrical manipulations, in two to four days the lips of the vulva begin to swell, the swelling rapidly involving the perineum, thighs and udder. There is an offensive, brownish discharge from the vagina. The temperature is up and bloating and diarrhea noted.

Diagnosis.—The cardinal symptoms of malignant edema are: Rapid development, emphysematous character of the swellings, high fever, and fatal course. It resembles blackleg in cattle, but may be distinguished from this disease by the following: Malignant edema usually affects the reproductive organs of cows at the time of parturition; it affects connective tissue rather than muscle, is not confined to young cattle, and appears in districts free from blackleg. Blackleg is common in the United States, and occurs enzoötically, while malignant edema is rare in cattle and is sporadic. It would hardly be confused with phlegmons resulting from pus infection. Such swellings do not develop so rapidly, do not crepitate, and are not usually fatal. Ordinary emphysemas of the skin, which accompany rib-fracture, pulmonary emphysema, or wounds (median neurectomy, trocaring, intentional inflation of the scapular region to conceal muscular atrophies), are not attended by fever and comparatively benign in course. The postmortal emphysema noted in parenchymatous organs in delayed necropsies during hot weather would hardly mislead a careful observer.

Course and Prognosis.—The course of malignant edema is usually rapid, death resulting from pulmonary edema in twenty-four to sixty hours. The prognosis is bad, 95 per cent. of the cases die. Recovery can be expected only in mild infections and when proper treatment has been prompt.

Treatment.—The treatment is surgical, and consists in slitting the swellings long and deep to allow oxygen to enter the tissue. A thorough disinfection of the incisions should follow while subcutaneous injections of iodin tincture may be made in the neighborhood of the swellings. Hot applications should be avoided.

In France, Leclainche and Vallée have practiced preventive inoculations with attenuated spores from edematous fluid and cultures. Good results are claimed. Of scientific interest is the fact that such inoculations do not immunize against blackleg.

BLACKLEG.

Definition.—Blackleg is an acute infectious disease of young cattle, due to a specific bacillus and characterized by fever and the formation of emphysematous swellings involving muscular groups in various parts of the body. It is not directly contagious.

Occurrence.—While, generally speaking, blackleg has a world-wide distribution, outbreaks occur usually only in certain so-called blackleg districts. In this respect it resembles anthrax, with which it was long confused. In the United States the western states suffer most, although sporadic outbreaks have been observed in the central and eastern states. The southern Atlantic and eastern Gulf states are said to be free. In some of the western states (Kansas, Dakotas, Nebraska) the animal losses from blackleg exceed those from all other diseases combined. The worst infected area on this continent seems to be that bounded north and east by the Missouri and Mississippi Rivers and west by the Rocky Mountains, the great cattle country.

Etiology.—The Bacillus gangrænæ emphysematosæ, an anaërobe which occurs in soil, water, and in infected districts in the droppings of cattle. Morphologically and culturally the blackleg bacillus so closely resembles the malignant edema bacillus that differentiation is very difficult.

Natural Infection.—(*a*) Through wounds in the skin and mucous membranes. Stubble, thorns, spines, barbed wire, etc., causing small punctured wounds, which on account of their character are most apt to become infected with soil, etc., containing the rods or spores.

(*b*) Through the digestive tract from the ingestion of contaminated food and water. The presence of the blackleg tumor in the throat, bowels, and other internal organs, and

the occurrence of outbreaks in stable-fed animals speak for this mode of infection. The disease is miasmic and does not spread from animal to animal. Blackleg carcasses, however, if not rendered harmless by cremation or deep burial, contribute toward the perpetuation of the infection by reinfecting the soil.

Symptoms.—Blackleg is seen almost exclusively in cattle. Probable cases in sheep and swine have been recorded. Opinions differ concerning its occurrence in horses, but no authentic data in this regard are available. Blackleg is a disease of young, full-blood, or high-grade cattle. It is rarely noted in animals under six months or over two years of age. The period of incubation is usually one to three days, occasionally longer (five days). The symptoms are both (*a*) general and (*b*) local.

General Symptoms.—Fever, the temperature reaching 107° F. The temperature rises and drops early in the disease. When the animals are down it is usually normal and just before death subnormal (95°–96° F.). There are refusal of food, suspended rumination, and great mental depression. Quite often the first symptom noticed is lameness in one limb.

Local Symptoms.—Following or sometimes preceding the general symptoms, external swellings appear in the region of the thigh, croup, loins, shoulder, breast, and throat. While at first small, the tumefactions rapidly increase in size until a large portion of the body is involved. In rare cases the swellings may be internal and located in the tongue, pharynx or bowel (colic symptoms). At first the tumors are firm, hot, and tender, but later, provided the animals live long enough, cooler, non-sensitive, the overlying skin hard and dry, and on palpation marked crepitation is noted. When the tumors are incised, a bloody, foamy fluid of a peculiar sweetish, fetid odor is discharged. The periphery and environment of the swelling are edematous. The lymph glands are swollen, and may be felt under the skin as firm nodes, at times the size of a goose egg. As the swellings enlarge the symptoms become more pronounced, the patient shows marked dyspnea, is unable to rise, muscular tremors

and even violent convulsions occur, the temperature rapidly falls and death follows.

Course.—The course is rapid and fatal. Most patients die in from twelve to forty-eight hours. Isolated cases may linger several days (mild invasion, high resistance), then die, or in rare instances recover.

Diagnosis.—Blackleg is a disease easily diagnosed, provided the typical swellings are in evidence. As a rule the owner or ranchman recognizes it without professional aid. If the characteristic emphysematous enlargements are absent, however, as is common in the first cases of some outbreaks, a diagnosis *intra vitam* is very difficult. Blackleg may be confused with malignant edema and anthrax (see these).

Necropsy.—The cadaver is greatly bloated and swollen, especially about the tumors. Hemolysis develops early in the body. Although the blood corpuscles are broken down and the hemoglobin set free, oxygen is still absorbed. This causes the parts exposed to the air to become a scarlet color. Therefore animals which have died of blackleg and have been skinned show a characteristic scarlet coloring of the surface. The affected muscle, however, does not undergo this change but remains almost black in color. When the swellings are cut into, a foamy, dark fluid flows out. The affected muscle is of a dirty brown to blackish color, very friable, and often smells like rancid butter. The blood is dark-colored but coagulates readily. In the serous cavities blood-stained fluid is found. The lymph glands corresponding to the tumors are swollen and blood-shot. The spleen is usually normal. The characteristic postmortem lesions are: Normal blood, normal spleen, and emphysematous swellings.

Prognosis.—In young cattle fully 98 per cent. die. In older individuals a few recover.

Treatment.—A medicinal treatment is useless. Surgical intervention as recommended in malignant edema is rarely advisable except in very valuable individuals. It is furthermore a menace, as the discharge from the incisions spreads the infection.

Prophylaxis.—When an outbreak occurs all cattle two years old and under should be promptly removed to a preferably

higher and drier pasture. In stable-fed victims a change to known uninfected food is imperative. The dead should be disposed of as recommended under anthrax. Exposed animals should be vaccinated, which successfully protects against serious infection. There are several anti-blackleg vaccines, aggressins, filtrates and sera on the market. The Bureau of Animal Industry and many of the agricultural experiment stations produce them.

(*a*) Originally the vaccine was made from blackleg muscle, dried, attenuated at 93.5° C., tested on guinea pigs and classified according to its ability to kill. It is used in the form of powder, pills or strings. The dose is 3 to 5 mg. While the use of this vaccine has greatly reduced the losses from blackleg, its action has always been variable, the losses reaching as high as 20 per cent. of the vaccinated animals on some farms.

(*b*) The aggressin is a vaccine produced by taking the blackleg lesion and all of the edematous tissues around it, pressing it in a hydraulic press, in order to extract all juices, after which it is preserved with phenol, clarified and filtered free of all organisms. The dose is 5 c.c. Goss, in Kansas, in three years distributed over 112,000 doses of blackleg aggressin with a total loss of less than 50 animals. While the use of attenuated vaccine necessitated that the animals be treated two or more times a year, with aggressins only one treatment in the life of an animal is necessary.

(*c*) The filtrate is made by growing the organisms on culture media for seven to ten days after which phenol is added. It is then clarified and filtered free of all organisms. The dose is 5 c.c. Properly made filtrate has given excellent results. As with the aggressins immunity is produced in four to ten days.

(*d*) Anti-blackleg serum is produced by injecting horses with blackleg cultures in doses of 50 to 150 c.c. weekly for a period of five to seven weeks. Administered in the early stages of blackleg in doses of 250 to 500 c.c. many animals suffering from the disease are cured. It is recommended to administer before the temperature drops. The serum should be followed in ten or twelve days with a dose of aggressin or filtrate.

Cattle may be vaccinated any time of the year. Obviously those already infected should not be treated with anything except serum. Heifers in advanced pregnancy should not be vaccinated. The immunity conferred by attenuated vaccines should last from twelve to eighteen months. Many cattlemen, however, vaccinate two or more times a year. With aggressins, as noted, one treatment lasts for the natural life of the animal.

BRAXY. GASTROMYCOSIS OVIS.

Definition.—An infectious disease of sheep characterized by its short, fatal course, and due to a specific anaërobic bacillus resembling the blackleg bacillus.

Occurrence.—Braxy has not yet been reported in the United States. In Norway, Denmark, Iceland, Germany and Scotland it is common, leading to great losses among yearling lambs. The disease is rarely seen in suckling lambs or in sheep over two years of age. While sporadic cases may occur during the summer months when sheep are on pasture, serious outbreaks take place during the late fall and early winter months after the sheep have been returned to the fold for the winter. Peculiarly, outbreaks seem more common following a heavy frost.

Etiology.—Braxy is due to the Bacillus gastromycosis ovis, an anaërobe which usually carries a spore near one end, produces gas and stains according to Gram. The germ is pathogenic to guinea pigs, rabbits, and pigeons. While subcutaneous inoculations generally produce the disease in susceptible sheep, feeding experiments with this bacillus have given negative results.

Natural Infection.—Probably through wounds (in upper digestive tract?) and possibly through the digestive tract with food. The disease is not thought to be spread by drinking water.

Necropsy.—In peracute cases few, if any, postmortem changes can be noted. In acute cases usually the principal lesions are found in the abomasum and duodenum, the mucous membrane of which is swollen, edematous, and spotted with

occasional bluish-red, hemorrhagic areas. Necrotic patches have been described. The blood is dark but readily coagulates, and the spleen not enlarged. The liver and kidneys show parenchymatous degeneration. In the body cavities a serohemorrhagic exudate collects and the subcutaneous connective tissue is edematous. Decomposition occurs rapidly and emphysema of the parenchymatous organs is commonly found in delayed necropsies.

In the diseased mucous membranes, the infiltrated submucosa, in the fluid of the body cavities, blood and parenchymatous organs the specific germ is found.

Symptoms.—The period of incubation is two to three days. The course of braxy is so rapid and fatal that clinical symptoms are rarely observed by the veterinarian. Even in those cases which may be examined clinically the symptoms are not particularly characteristic. It may be noted that the affected sheep are restless, alternately lying down and getting up suddenly, as if in pain. There may be slight bloating, arched back, and pendent head. When artificially infected by subcutaneous injection, the injected limb is dragged behind. The sheep champs the jaws, churning the saliva into foam. It usually leaves the rest of the herd, is very listless, and depressed. In some outbreaks diarrhea is an early symptom. The temperature is elevated (105° to 108° F.) and the respirations dyspneic. Usually after a couple of hours the patient falls over, becomes comatose, and dies.

Diagnosis.—Braxy is often confused with anthrax, especially when it occurs in anthrax districts. It might also be mistaken for blackleg or for malignant edema. By carefully weighing the history, clinical symptoms, and bacteriological findings, error in diagnosis is avoided.

Course and Prognosis.—The course is rapid; usually in from fifteen to eighteen hours the patient dies. The prognosis is bad; fully 98 per cent. succumb.

Treatment.—Medicinal treatment has not proved of value. Separation of the living, deep burial, or cremation of the dead, and a thorough disinfection of the barns, pens, yards, etc., are indicated.

Protective Inoculation.—Nielsen and Jensen have employed protective inoculation to combat braxy, the former using dried, powdered kidney substance from a fatal case, while the latter used either treated blood serum cultures or blood serum from artificially immunized horses or cultures one month old which were grown in sugar bouillon. Apparently good results have been obtained in Iceland with preventive vaccination (see Bacteriology).

SWINE ERYSIPELAS.

Definition.—Swine erysipelas is an infectious disease due to a specific microörganism. It has a varied course and appears clinically as an acute septicemia, a secondary skin exanthema, or a chronic, valvular heart disease.

Occurrence.—On the continent of Europe the disease is widespread, occurring during the hot months, and in latter years has assumed a more serious form than formerly. In Great Britain swine erysipelas is a benign disease, appearing in the skin form and, as yet, has not assumed serious proportions. To date it has not been reported in the United States. In the so-called "diamond disease," so commonly found in American abattoirs, erysipelas bacilli have not yet been determined.

Etiology.—The Bacterium erysipelatis, a fine bacillus found in the acute form of the disease in the blood, skin, and lymph glands. In the chronic form they are found in the diseased tissue, particularly in the valvular vegetations in the heart.

Natural Infection.—(*a*) *Via* the mouth with food and water contaminated with the feces and urine. The blood and flesh are also infectious. (*b*) *Via* wounds.

The disease is usually first introduced by infected hogs or by apparently healthy "germ carriers," swine which have recently recovered from erysipelas but still carry germs in the bowels and tonsils. Fields on which the bodies of dead hogs have lain or were not buried deep enough or where the feces and urine of diseased animals are deposited are most dangerous sources of infection. The disease is not directly contagious, but is disseminated largely through soil

infection. Hogs from three months to one year are most susceptible. Sucklings and old animals are usually quite resistant.

Necropsy.—In the acute form very little change is noted. The small intestines are catarrhally inflamed. Peyer's patches and the solitary follicles are swollen and small ulcerations are noted. The spleen is somewhat enlarged. Ecchymoses and petechiæ are prone to appear in the mucous and serous membranes, especially in the epi- and endocardium and in the subcutaneous connective tissue. The kidneys are swollen, and on section the cortex is grayish-red dotted with punctiform hemorrhages, the medullary substance dark. As a rule the lungs are not involved. In chronic cases the principal lesion is found in the heart, the atrioventricular valves showing lesions of verrucous or ulcerous endocarditis.

Symptoms.—The period of incubation is three to five days. The clinical symptoms vary, but the following forms are fairly well distinguished.

(*a*) *Skin Form.— Urticaria Type* (*" Diamond Disease"?*).—After a short period, during which the hog seems languid and fails to eat well, there appear in the skin on the sides of the chest, back, neck and legs, sometimes all over the body, violet-colored spots and sometimes vesicles. The spots are often rectangular or rhomboidal in form and are elevated above the surrounding skin. They are usually about the size of a 50-cent piece, larger by confluence. Sometimes vesicles occur upon them which break, the discharge drying to brownish crusts. As a rule, symptoms of fever, languor, anorexia, constipation, paraplegia, conjunctivitis, and vomiting attend the exanthema. The skin eruption usually disappears in from one to two weeks. In some cases necrosis of the skin, especially of the ears, tail, or digits takes place, leading to sloughing and healing, with pronounced scar formation.

(*b*) *Septicemic Form.*—This form very much resembles the septicemia (explosive) form of hog-cholera (see this). The skin is usually highly reddened, especially on pendent portions of the body. The red colorations have an erythematous character, finger pressure temporarily removing them. Vesicle formation and necrosis may also occur. In severe cases there

is paraplegia, cyanosis, and great dyspnea (lung edema). The patients usually die in three or four days or may live a week. In some cases the symptoms subside on about the third day, the animal recovering or the disease assuming the chronic form.

(*c*) *Chronic Form.*—In this type symptoms of chronic endocarditis (see this) and gangrene of the skin occur. It is noted in hogs which have passed through the acute stage and seem to be recovering. In about one to two months, however, they begin to droop, lose appetite, cough, become dyspneic, cyanotic, develop heart palpitation ("thumps"), and carry a mild fever. Gradually becoming weak, after several weeks they die of inanition.

Prognosis.—The skin form is benign, practically all recovering. In the septicemic form the mortality is 60 to 90 per cent. and chronic cases are generally fatal.

Diagnosis.—Swine erysipelas is easily confused with hog-cholera. Clinically the differences are not marked. As a rule, however, the lungs and bowels are less affected in erysipelas and the reddening of the skin is intra- rather than extravascular. Bronchopneumonia with pleuritis and button-like ulcers in the cecum or necrosis of the intestinal mucous membrane are lesions not seen in swine erysipelas. In doubtful cases a bacteriological examination of the blood should be made.

Treatment.—A medicinal treatment is useless.

Prevention.—As the bacillus of this disease has a saprophytic existence in soil, and is often present in the tonsils and bowels of "germ carriers," it is difficult to eradicate. However, the usual causes of infection, as noted, are affected swine, living or dead, and therefore susceptible hogs should be isolated from them. Infected pens and hog lots should be thoroughly disinfected, the droppings collected and rendered innocuous. Hog pastures contaminated with the germs should be cultivated. Carcasses should be burned or buried deep.

Protective Inoculation.—As one attack of erysipelas produces immunity, vaccination, much practised in Europe, is feasible. Two methods are employed: One known as that of Pasteur, which requires a double vaccination, the first fol-

lowed by a second in twelve days; the other is known as the simultaneous (serum and cultures together) method of Lorenz and Laclainche, which also requires that the vaccination be once repeated.

HEMORRHAGIC SEPTICEMIA. PASTEURELLOSIS.

Under the collective term "hemorrhagic septicemia" is gathered a group of diseases characterized by general septicemic infection (blood spots appearing in the skin, serous membranes, bones and joints), and in the tissues one of the varieties of a belted or cocoa bacillus known as the Bacterium bipolaris septicum or the Bacterium Pasteurella.

Under this head are usually included the following diseases: Chicken cholera, rabbit septicemia, hemorrhagic septicemia of cattle, septic pneumonia of calves, the buffalo plague, hemorrhagic septicemia of sheep, takosis of goats (?), swine plague, and enzoötic pneumonia of shoats.

At the present time, for lack of sufficient experimental data, it is difficult to decide just what relationship the bipoled bacteria bear to the diseases they are supposed to cause. The fact that the bipoled bacilli are found in the tissues and body fluids after death is not proof that they were the primary cause of the disease present. Furthermore, they are not infrequently met with in animals which showed no symptoms of disease. Prior to the discovery and use of the Dorset serum to prevent hog-cholera, a disease among swine, due to a bipoled microörganism, was recognized. It was called "swine plague." At that time the cause of hog-cholera was thought to be a bacillus which resembled somewhat the typhoid bacillus of man. For a time it was believed that inasmuch as anti-hog-cholera serum seemed also to protect against this swine plague, which frequently occurred associated with hog-cholera outbreaks, a mistake had been made in dividing hog-cholera into two diseases. In the last few years, however, field work with anti-hog-cholera serum has developed that in some outbreaks, which symptomatically and patho-anatomically resembled hog-cholera, no protection was afforded. It has, therefore, been assumed that the out-

break following the vaccination was not hog-cholera but some other disease. There is a probability that some of these outbreaks may be hemorrhagic septicemia. It is possible that swine, the resistance of which has been lowered by recent vaccination, become more susceptible to the action of the B. suisepticus. From a theoretical standpoint, if we admit the existence of hemorrhagic septicemia in other domesticated animals, its occurrence in the hog can be refuted only on well established scientific grounds. The results of exhaustive scientific research in this regard are awaited with interest. Only through it will the problem be solved.

Hemorrhagic Septicemia of Cattle (*Cornstalk Disease; Bronchopneumonia; Game and Cattle Plague*).—**Definition.**—Hemorrhagic septicemia is a fatal, infectious disease of cattle, having an acute or subacute course, which assumes a variety of forms in that it may involve the nervous system, skin, lungs, and bowels. It is probably caused by the Bacterium bovisepticum.

Occurrence.—The disease is widely spread, occurring in all parts of the world. In the United States it has been reported from all sections, but seemingly is more prevalent in the West and Northwest than elsewhere. Several outbreaks have occurred in Ohio. Besides cattle it attacks wild animals, especially deer.

Etiology.—The Bacterium bovisepticum, a variety of the cocoa bacillus, is found in soil and water and in the respiratory organs of apparently healthy cattle.

Natural Infection.—(*a*) *Via* digestive tract, the germs being taken in with the food and water. (*b*) *Via* wounds through the skin and mucous membranes. (*c*) *Via* respiratory organs (proved in rabbits). The hides and flesh of infected animals spread the disease. The blood in the later stages is infectious and probably the feces and urine.

Necropsy.—Varies somewhat with the type, but the following lesions are fairly constant: A marked congestion of the parenchymatous organs. Subcutaneous, submucous, and subserous hemorrhages generally distributed along the bowels (serous surface), over the spleen capsule, kidneys, bladder, diaphragm, peri- and epicardium, synovial membranes and

meninges. The heart, liver, and kidneys show cloudy swelling. The spleen is not enlarged. The urine is sometimes blood-tinged. In the skin (exanthematous) form the subcutis of the region of the throat and neck shows gelatinous infiltration spotted with blood patches. The mucous membrane of the digestive tract is swollen and shows petechiæ. The tongue is swollen, and submucous infiltrations are noted in the pharynx and larynx. The bowel contents are usually semiliquid and frequently chocolate colored. Blood apparently normal. In the pectoral form serofibrinous pleuritis with bronchopneumonia is found. The interlobular septa are markedly infiltrated, forming broad, yellow strands. Blood extravasations occur under the pleura.

Symptoms.—The period of incubation is short, usually one to two days. The symptoms are not particularly characteristic and quite varied, depending upon whether or not the intestinal, exanthematous, or pectoral form is present.

Intestinal Form.—The animal appears dull and shows a staggering gait and loss of sensitiveness in the skin; in some cases the neck is bent to one side (torticollis), the muscles of the neck and jaw twitching; nystagmus is also seen. Occasionally forced movements are noted, the patient rapidly wheeling in a circle, using the hind feet as a pivot (clock-hand movements). During these paroxysms, which occur intermittently, the patient may utter lowing cries. Diarrhea may or may not be present. There is drooling from the mouth and a persistent nervous champing of the jaws. The tongue is swollen and cyanotic. High fever is noted, in some cases the temperature, especially after a nervous paroxysm, reaching 108° F. and over. Some authorities, however, have found the temperature normal or even subnormal.

Exanthematous Form.—This form is less frequent in American outbreaks. Inflammatory edema of the head, neck, throat, and dewlap appears. The swellings may extend to the body and limbs. They are pronouncedly hot and painful. Conjunctivitis with profuse lacrimosis may be present. The tongue is edematously swollen, filling the whole mouth cavity, and is cyanotic. Drooling is noted. The patient is dyspneic, the mucous membranes are dark- colored and show petechiæ.

Death results from asphyxia or severe enteritis. Intestinal symptoms are commonly noted in this form.

Pectoral Form.—The symptoms of this form are those of an acute or subacute bronchopneumonia with pleuritis. The patients stand with arched back, have a painful cough and nasal discharge. Dulness is noted on percussion. Bronchial sounds, rales, and rhonchi are heard on auscultation. The patient is dyspneic, appetite is lost, and rumination suspended. The constipation is later followed by diarrhea, inanition, and death.

Differential Diagnosis.—Hemorrhagic septicemia might be confused with anthrax, Rinderpest and the subacute or chronic pectoral form with contagious pleuropneumonia of cattle. It might also resemble blackleg. The absence of splenic enlargement, the normal appearance of the blood, and the absence of the anthrax bacillus differentiate it from anthrax. Rinderpest does not occur in the United States, but is found in the Philippines. Outbreaks of Rinderpest can usually be traced to an imported source (diseased cattle), and in this disease, unlike hemorrhagic septicemia, a marked ulcerous inflammation of the mucous membrane of the eyelids, respiratory and digestive tract is present. Contagious pleuropneumonia no longer exists in this country. Macroscopically the lesions of the pectoral form of hemorrhagic septicemia and this disease may be almost identical. In such cases only the use of bacteriological methods can decide (finding the bipoled bacterium; animal inoculations). In typical cases of blackleg there should be no difficulty in differentiation, as emphysematous surface swellings are not seen in hemorrhagic septicemia. Furthermore the latter disease affects cattle of all ages while blackleg is essentially a disease of young animals (see this).

Course.—The course varies with the form the disease assumes, the severity of the infection and the resistance of the individual patient. Acute cases last only a few hours (6 to 20) while less acute ones may linger a week or even longer. Those affected with the lung form usually live longer than in the intestinal or exanthematous forms of the disease.

Prognosis.—Very bad. Fully 90 per cent. die.

Treatment.—Medicinal treatment is useless. In outbreaks, removing the sick cattle to other pastures is recommended. If the disease occurs in a stable, disinfection is in place. As a rule, after a certain number of cattle in a herd die the disease stops of itself. The carcasses should be disposed of as recommended under anthrax. The dried hides are said to be harmless.

Septic Pleuropneumonia of Calves.—Definition.—This disease is evidently a form of hemorrhagic septicemia of cattle. It is a specific pleuropneumonia of calves due to a bipoled bacillus.

Occurrence.—The disease is widely distributed and not infrequently occurs simultaneously with an outbreak of hemorrhagic septicemia of older cattle.

Etiology.—The Bacterium vitulisepticum which seems identical with the germ Bacterium bovisepticum.

Natural Infection.—Infection takes place through the digestive tract, with food and water polluted with the discharge of affected calves.

Necropsy.—The principal changes are bronchopneumonia with serofibrinous pleuritis. Catarrhal bronchitis and laryngitis are also present. Gastro-intestinal catarrh, swelling of the lymph glands, and cloudy swelling of the parenchymatous organs attend.

Symptoms.—The symptoms are those of acute or subacute pneumonia. The affected calves are listless, the hair coat staring, muzzle dry, abdomen tucked up and the general appearance unthrifty. A common symptom is a short, somewhat painful cough emitted when the animals are disturbed or driven from the barn into the outside air. The appetite may be retained. The temperature is elevated and the respirations dyspneic. Palpation over the thorax induces pain. On percussion there is dulness in the lower part of the chest, and on auscultation either the sounds of breathing are absent or bronchial tones may be heard. More rarely friction sounds are evident. The patients gradually become weaker, and in most cases either die in one or two weeks, or there develops chronic pulmonary phthisis.

Prognosis.—The prognosis is uncertain; the mortality varies but averages about 50 per cent.

Treatment.—The medicinal treatment is symptomatic as recommended in pneumonia. It is advisable to separate the affected animals from the healthy, to bury deep or cremate the dead, and thoroughly disinfect the barns and premises. As a prophylactic measure, inoculating the calves with blood serum derived from an artificially immunized horse treated with cultures of the bipoled bacillus has been practised with apparent success.

Hemorrhagic Septicemia of Sheep.—Definition.—Hemorrhagic septicemia of sheep manifests itself in acute cases as a general septicemia. In subacute and chronic cases it is characterized by nasal and eye discharge, also by pleuropneumonia. It is caused by the Bacterium ovisepticum.

Occurrence.—The disease occasions among lambs considerable loss. Older sheep are rarely attacked, and in them the disease assumes a chronic form. Hemorrhagic septicemia of sheep is most common in low, damp, marshy regions. It is rare on high, dry land. Outbreaks occur most generally at the time the lambs are weaned.

Etiology.—The Bacterium ovisepticum, a variety of the Bacterium bipolaris septicum. It is difficult to cultivate outside of the body as it is very apt to die in cultures.

Natural Infection.—Through the digestive tract, the sheep taking in infected food or water found on the pastures or in contaminated sheep folds. Young sheep at weaning time are most predisposed, and in them the disease assumes usually a very acute form. In older sheep the disease is more chronic.

The infective discharges of diseased animals obviously contribute toward the spread of the disorder. In sheep herds running in small pastures or kept crowded in folds the disease assumes a very virulent form. It is transmitted to healthy sheep folds by infected animals.

Predisposing causes are anything that will reduce the resistance of the sheep, such as cold, getting wet by rain, etc. The disease is more common in wet seasons and on low grounds.

In all probability infestation with strongyls may be a predisposing cause.

Necropsy.—In peracute cases the postmortem is largely negative, except for the symptoms noted under hemorrhagic septicemia.

In the acute form the subcutaneous connective tissue of the dewlap, neck, and throat shows gelatinous infiltration. The mucous membranes of the head, air passages, abomasum, and bowel are inflamed (reddened and swollen). The lymph glands are enlarged, diffusely reddened (bloodshot). Petechiæ and ecchymoses are noted in the serous membranes, kidneys, and lymph glands. In many cases, even in the acute form, the lungs are involved. In them are found dark, reddish-brown areas of bronchopneumonia surrounded by infiltrated interlobular connective tissue. The spleen is usually normal.

In the subacute form bronchopneumonia involving the anterior and lower parts of the lung is present. The pleura and also the pericardium are often covered with fibrinous pseudomembranes and the serous cavities partially filled with a clear, yellow or turbid fluid. Bronchitis and enteritis may also be present and in many cases fibrinous rhinitis.

In the chronic form larger areas of the lung and pleura are involved. Thickenings and adhesions are common. The involved area is hepatized, some areas which have undergone necrosis are surrounded by connective tissue capsules. The necrotic foci, when cut through, show a yellow center of viscid pus surrounded by concentric layers resembling in structure an onion. However, in some cases none of the internal organs show marked pathological changes. There are in such only the symptoms of a severe pneumonia and cachexia.

Symptoms.—In the acute form, which usually lasts two to five days, or even a shorter period, there are symptoms of a severe feverish disease. The lambs are very dull, stupid, refuse to eat, show increased thirst, dyspnea, colic symptoms, and twitchings of the muscles. The temperature is often 105.8° F.

In subacute cases the animal may live one to three weeks, during which time it shows symptoms of fever, poor appetite, and languor, with mucopurulent discharge from the eyes and nose. In some instances there are symptoms of pleuropneumonia; in others enteritis with a discharge of a yellowish-green, later darker colored, fetid feces. In some cases nodules and also ulcers are noted on the mucous membrane of the

lips and cheeks. Caries of the teeth may occur. The lips, cheeks, and tongue are swollen and cyanotic. In rare instances ulcerous keratitis has been observed. The lambs are very weak and die under symptoms of cachexia. In rare instances after a long time improvement follows. Complete recovery is, however, rare. As a rule the animals are stunted by chronic pneumonia and resulting cachexia.

The chronic form is seen usually only in adult sheep. Sometimes it develops from the acute form. The symptoms are those of a severe, chronic pneumonia. The sheep cough, show dyspnea, moderate mucopurulent nasal and eye discharge, and gradual emaciation. In some cases there develop swellings of the carpal and tarsal joints and purulent inflammation of the hoof matrix. In most cases, however, the symptoms are those of a general cachexia.

In some districts in which this infection prevails, coincidently animal parasitism is associated with the disease (strongyls, tapeworms, liver flukes).

Diagnosis.—The acute cases might be confused with anthrax. However, the normal spleen, absence of gelatinous blood extravasations, and hematuria usually suffice for differentiation. Subacute and chronic cases so much resemble the lung and stomach worm plague of lambs that differentiation would be very difficult provided parasites were found. Braxy affects yearling sheep in the fall and winter months. It is characterized by its rapid, fatal course and the hemorrhagic areas in the abomasum and duodenum found on necropsy. Icterohematuria of sheep presents on postmortem besides icterus, hemorrhagic inflammation of the abomasum, duodenum and rectum, enlargement of the spleen, the pulp of which contains large numbers of the piroplasmosæ ovis.

Treatment and Prevention.—Medicinal treatment is largely useless. A slaughter of all sheep chronically affected is recommended. The sheep should be removed from infected pastures and a thorough disinfection of the sheepfold should follow. As animal endoparasites probably facilitate the bacterial infection, the sheep should not only be kept rid of worms, but kept from worm-brood infested pastures.

Protective Inoculation.—Good results were obtained in Argentine by the use of a polyvalent vaccine of which lambs were given each $\frac{1}{4}$ c.c. injected subcutaneously. Polyvalent immunizing serum was also effective.

In several herds of infected lambs the serum of the horse which had been hyperimmunized with cultures of the Bacterium ovisepticum proved of practical value. The serum was given simultaneously with the vaccine. In these experiments, as the immunity lasted only six weeks, a second inoculation with vaccine is recommended at the end of one month. This simultaneous method proved efficacious in that it stopped the spread of the disease and in most cases healed lambs already infected.

Hemorrhagic Septicemia of Swine (*Swine Plague*).—**General Remarks.**—The existence of hemorrhagic septicemia occurring in swine as a clinical entity has been denied by some authorities. In the early eighties in America and later on the Continent of Europe, as a result of much scientific research, hemorrhagic septicemia was established as a distinct disease, separated from hog-cholera and called "swine plague." Its coöccurrence with hog-cholera was admitted, and has since been reaffirmed. It is thought, especially by the producers of anti-hog-cholera sera, to explain some of the failures in the use of serum to prevent cholera as being due to the presence of swine plague against which anti-hog-cholera sera offer no protection. Irrespective of these claims and the denials of some authorities as to the existence of hemorrhagic septicemia in swine, enough scientific evidence seems at hand to warrant placing swine plague in a chapter by itself, and considering it a disease separate and distinct from hog-cholera.

Definition.—Hemorrhagic septicemia of swine is an infectious disease due to a variety of the B. bipolaris. It appears in two forms: A general septicemia, and as a pneumonia.

Occurrence.—Hemorrhagic septicemia occurs sporadically and rarely becomes epizoötic. It is often seen accompanying an outbreak of hog-cholera, or it may attack independently individual herds in a community but showing no tendency to spread from farm to farm.

Etiology.—It is caused by the B. bipolaris suisepticum, a bipoled organism which occurs in soil, food, drinking water, and in the air passages and digestive tract of healthy swine. It is possible that this germ is facultative and can live a saprophytic existence. Swine, the resistance of which has been reduced by intestinal parasitism (Ascaris lumbricoides, Echinorhynchus gigas, Strongylus paradoxus), improper food, exhaustion from long transports, etc., may become predisposed. It is also probable that the virus of hog-cholera may lower the resistance of an animal to such an extent that the bipoled organism which enters it can assume pathogenic properties. It is possible that the first appearance of hemorrhagic septicemia in a healthy swine herd comes from soil infection plus the lowered resistance of the animal. However, once a pig is infected, through its discharges, the bipoled organism having become pathogenic, can be eliminated and infect other animals. As a rule, however, the disease does not pass rapidly through a herd and, after attacking a percentage of animals, dies out.

Necropsy.—In the peracute form the lesions are typical of hemorrhagic septicemia. Numerous small hemorrhages occur in the skin, fat, in the serous and mucous membranes, kidneys and myocardium. Larger hemorrhages are found in the perirenal connective tissue, in the mucous membrane of the renal pelvis, urinary bladder, and in the brain. The lymph glands show hemorrhagic swelling and there is a gelantinous infiltration of the subcutaneous connective tissue especially in the region of the throat and neck.

In the acute form the respiratory organs are attacked. The lungs show multiple necrotic centers with a serous or hemorrhagic infiltration of interlobular connective tissue. With this is associated a serofibrinous pleuritis. The peribronchial lymph glands are swollen and frequently traversed with hemorrhages.

The gastro-intestinal mucous membrane shows catarrhal swelling and petechiæ. The surface is often covered with a croupous membrane especially in the posterior part of the small intestine and in the large bowel. The solitary follicles and Peyer's plaques are swollen and may show ulceration. The spleen is usually intact.

Chronic Form.—The lungs show circumscribed, necrotic areas which in some instances are sequestered. In others large areas are hepatized or traversed with small, yellow-colored or gray necrotic foci. In the peribronchial and mesenteric lymph glands caseous centers occur.

Symptoms.—The symptoms so resemble those of hog-cholera that a differentiation in the field is very difficult.

Peracute Form.—In the peracute form the affected pig shows the symptoms of a general septicemia. The temperature is 108° F. and over. The patient is languid, has no appetite, remains apart from the herd and may crawl under a building or straw pile. On parts of the body, especially the ears, neck and sides, red spots appear which cannot be entirely eliminated by pressure. There may be hemorrhage from the nostrils, bowels and urinary organs. In some animals there are symptoms of a severe laryngopharyngitis. The patients usually die in twelve to twenty-four hours after the first symptoms are shown.

Acute Form.—In the acute form the symptoms are those of an acute pneumonia. The patient shows fever, a short, dry, spasmodic cough, an expiratory dyspnea, breathing with the feet spread apart and mouth open. Palpitation of the thorax causes pain. There is nasal discharge and frequent and violent fits of coughing. The mucous membranes are cyanotic; the eyes show conjunctivitis. Palpitation of the heart is a common symptom. The acute form lasts seven to fourteen days. Complete recovery is rare. Apparently recovered animals frequently harbor the chronic form of the disease.

Chronic Form.—The chronic form follows the acute. The symptoms are not very pronounced. They consist mainly in periodical fits of coughing, dyspnea, poor appetite, emaciation and chronic joint swellings. Finally the patients suffer from diarrhea and death occurs from exhaustion in one to two months. A few cases make apparent recovery but on slaughter sequestered, necrotic foci are found.

Diagnosis.—The differentiation from hog-cholera can only be made bacteriologically. The coexistence of the two diseases in the same herd and animal must be born in mind.

From lung-worm disease it may be differentiated on necropsy but the contemporary occurrence of parasitic infestation and swine plague infection is not uncommon.

Treatment.—Medicinal treatment is valueless as in hog-cholera. A prophylactic vaccine is now being employed to protect healthy herds. The vaccine should be injected intramuscularly rather than subcutaneously. In infected herds serum is employed. There are several vaccines and sera on the market to prevent and cure swine plague. Sufficient data have not yet been accumulated to form a final opinion as to the value of these agents. Swine plague has a sporadic origin, is not very communicable and therefore does not affect many herds in a community. The natural tendency for the disease to die out suddenly in a herd has undoubtedly given reputation to some of the sera.

Takosis of Angora Goats.—Definition.—Takosis is a chronic, contagious disease of Angora goats characterized by weakness, emaciation, diarrhea and pneumonia, which leads to death in from one to eight weeks.

Occurrence.—The disease is not uncommon in the United States, particularly in the northern states (Oregon, Missouri, Massachusetts, Virginia and Maryland). As the disease is fatal, and 30 to 85 per cent. of the goats in a herd are attacked during an outbreak, it attains considerable economic importance.

Etiology.—The cause seems to be a micrococcus, the Micrococcus caprinus, which is pathogenic for goats, chickens, rabbits, guinea pigs and white mice, but not for sheep, dogs, or rats.

Necropsy.—The necropsy shows evidence of general anemia. In the serous membranes petechiæ are found. In the lungs centers of pneumonia are present and in the pericardium accumulations of transudate. There is chronic catarrh of the mucous membranes, and occasional areas of necrosis have been noted.

Symptoms.—The symptoms of takosis are very similar to those following a parasitic invasion. In general there are emaciation, weakness, with symptoms of diarrhea and cough. In the early stages, except for a slight weakness, no symptoms

are observable. Later the affected goat becomes listless and languid, lags behind the flock, holds its head low, and the eyelids partially closed. There is usually some nasal discharge and occasional coughing. The pulse is slow and weak, the temperature at first elevated (104.1° F.) but later, a few hours before death, it becomes subnormal (99.7° F.). As the disease progresses the gait grows staggering, the back arched, the patient moving in a wavering, unsteady fashion. The appetite is capricious. Rumination is rarely impaired. The mucous membranes are anemic, the respirations increased. Finally the patient becomes so weak that it falls to the ground and must be assisted to its feet. Gradually losing weight it lingers from day to day, and finally under symptoms of fetid diarrhea, succumbs. Frequent, plaintive bleating is noted.

Diagnosis.—The symptoms of takosis are usually so similar to those of parasitism that from the clinical aspects alone a diagnosis would hardly be possible. The necropsy would determine the presence or absence of parasites. However, in parasitism the pneumonic symptoms are not so well developed and the tendency for submaxillary edemas to form is greater.

Course.—The course is usually chronic, the patient dying in from one to eight weeks. The mortality is 100 per cent. Pregnant does usually abort.

Treatment.—Medicinal treatment has proved unsatisfactory, as all patients naturally infected die. Some authorities recommend small doses of calomel (grs. ij twice daily for two days) followed by arsenic, iron, and quinin:

℞—Arsen. acid	gr. xx
Ferri reduct.	ʒiv
Quinin sulph. .	ʒiss

M. f. pulv. no. xx.
S.—One powder morning and evening.

Prophylaxis.—To prevent takosis it is recommended that when goats are shipped from one part of the country to another that it be done during the summer or late spring, and not in the fall or winter, thus avoiding as far as possible sudden climatic changes. It is also advisable at all times to provide the goats with a storm shelter to which they go

voluntarily during a downpour of rain. That the herd should be given proper food and careful attention is obvious.

Once the disease has made its appearance in a flock the separation of the sick and the healthy coupled with a thorough disinfection of the premises are indicated.

SEPTICEMIC DISEASES OF NEWBORN ANIMALS.

Dysentery of Sucklings (*Dysenteria Neonatorum; Calf Scours; White Scours*).—**Definition.**—This is an acute, gastro-enteritis rarely occurring in animals over one week old. It appears enzoötically and is characterized clinically by a profuse diarrhea, great exhaustion, and a rapid, fatal course.

Occurrence.—The disease occurs in calves, lambs, foals and pigs (rare in dogs and cats) and appears especially in breeding districts at the time of parturition (spring and fall), causing great losses on account of its rapid spread and fatal termination. In some outbreaks every calf or lamb born on the premises becomes infected and dies of the disease in the first few days of its extra-uterine life.

Etiology.—The etiology of dysentery has not been worked out. No one organism has been isolated which seems to explain all cases. The close relationship between this disease, pneumonia, septicemia and pyosepticemia of sucklings, has been recognized. As dysentery often occurs concomitantly with infectious abortion it has been suggested that some outbreaks of the disease might be due to the Bacillus abortus. Poels failed to recover a constant organism in calves showing symptoms of the disease. Among other organisms he isolated the Bacillus pyocyaneus. Carpenter has isolated from the stomach contents and meconium of fetal calves at different periods of gestation a number of organisms, the streptococcus predominating. What the effect, however, of these organisms might have been, were the fetus allowed to mature, could not be determined. Some outbreaks seem to have been due to the Bacillus enteriditis of Gärtner.

Natural Infection.—Once introduced into a barn the infection remains there with remarkable tenacity, causing year

after year new outbreaks of the disease. It is possible for this dysentery to develop in a stable without being imported. This is probably due to colon bacilli, which are living as saprophytes on the premises, assuming a virulent form once introduced into the bodies of calves with lowered resistance (bad sanitation, improper feeding). The infesting germs may enter the body: (*a*) *Via* navel cord, or (*b*) *via* digestive tract. Calves are often attacked before they have suckled their dams. An intra-uterine infection seems to be proved by the fact that an injection of a virulent culture of the colon bacillus into the jugular vein of a pregnant cow, was followed in eight days by the birth of a calf with the disease. Removing the pregnant dam to a non-infected place does not always protect her young from infection. Calves which have not yet sucked seem most predisposed; and resistance against infection increases as the calf becomes better nourished through its natural food. After eight days of extra-uterine life the danger of infection is passed.

Symptoms.—The symptoms appear in from a few hours to three days after birth, rarely later. The principal indication of the disease is diarrhea. The liquid feces are at first expelled with considerable straining (tenesmus), a strong stream of yellow colored, fetid discharge being shot from the rectum. Later the evacuations become whiter in color and thinner in consistency, often mixed with blood. The patient rapidly grows weak, languid, refuses to suck, the eyes become retracted and dull, the anus relaxes, causing fecal incontinence, the hair coat becomes erect, and finally under symptoms of coma death occurs. The temperature is somewhat elevated (105° F.) in the early stages, but later may become subnormal. There is also dyspnea and rapid pulse.

Diagnosis.—Usually easy. This *infectious* dysentery is differentiated from *sporadic* diarrhea of young animals in that it affects only those less than one week old, induces a fetid, exhaustive, and rapidly fatal diarrhea and assumes an enzoötic form. In acute gastro-intestinal catarrh ("scours") due to dietetic irregularities older sucklings are attacked, the general symptoms are much less pronounced, and the feces are thicker, yellower, and less fetid.

Course.—The course is usually very rapid, leading to death in from twelve to forty-eight hours. The sooner after birth the symptoms appear the more fatal and rapid the course. Occasional cases may linger as long as a week.

Prognosis.—Bad. Mortality 90 per cent. Recovery follows a protracted convalescence and results in an unthrifty calf (chronic pneumonic lesions).

Treatment.—Medicinal treatment is usually worse than useless, as it rarely prevents death, and encourages the further spread of the disease. Ordinarily it pays to kill the infected suckling and render its carcass harmless by cremation or deep burial. When an outbreak occurs every effort should be made to eradicate the infected sources by a thorough disinfection of the barn, hind parts and genitals of the dam before and after parturition, and the navel stump of the newborn animals, if it still be healthy. All fecal discharges should be removed and sterilized with a 2 per cent. formaldehyd solution. The stable partitions, mangers,[1] floors, should be thoroughly disinfected and, if possible, given a coat of whitewash. The after-birth, dead sucklings, and postpartum vaginal discharges should be completely destroyed. The vagina of the dam should be douched with a warm solution of 2 per cent. carbolic acid, the external genitals, buttocks, tail, and udder being also washed with the antiseptic. The navel of the newborn animal should not be ligated, but before it has had an opportunity to become infected, covered with some astringent, antiseptic powder which will aid its desiccation. The meconium is best removed by the colostral milk of the dam, but before sucking is permitted, the udder and teats should be disinfected with a nonpoisonous antiseptic (creolin). Hand-fed sucklings should be allowed the colostral milk for the first two days. To prevent outbreaks in contaminated premises it is recommended to remove the pregnant dams to uninfected places at least six weeks before parturition. As the infection seems to be retained in the genital passages for some time a shorter period does not suffice.

[1] If the floor is earth it should be removed to the depth of at least one foot and then filled in with fresh clay.

Prophylaxis.—As the preceding sanitary measures recommended have never proved satisfactory an effort has been made to cope with the disease from another angle. The experiments of Williams seem to indicate the importance of a clean uterus before conception. He, therefore, recommends douching the uterus to remove pathogenic organisms before the animal is bred. Obviously this must be done in a thorough manner in order that no infection remain to infect the fetus.

A serum, produced from the supposed causal germs of the disease, is now being employed to increase the resistance of the newborn calf. It may be administered subcutaneously, intramuscularly or intravenously. The dose for a newborn calf is 50 mils. Very much larger doses may be given without injury. The objection to the larger dose is purely economical, as the cost of the serum is high.

Bacterins injected subcutaneously in small doses (1 mil per day for ten days) have also been tried. Autogenic bacterins prepared from cultures taken from a calf, which has died of the infection and belonging to the infected herd, have given the best results. Like most bacterins, however, much greater success is obtained by their use in chronic than in acute cases.

The diet of the calf should be carefully attended to. Underfeeding is preferable to overfeeding. Feed only clean, whole milk during the first day or two of the calf's life. Afterwards the milk is boiled to destroy pathogenic organisms. The calves should be kept in separate pens with tight partitions and absolute cleanliness insisted upon.

Experiments to produce active immunity of the fetus *in utero* by inoculating subcutaneously pregnant cows with sterile extracts of colon bacilli, have been made with as yet conflicting results.

Pyosepticemia of Sucklings (*Pasteurellosis Neonatorum; Pyemic Arthritis; Joint Ill; Omphalophlebitis; Navel Ill*).—**Definition.**—This is an acute contagio-infectious disease of animals less than one month old, due to navel infection, and characterized by joint lesions usually of a purulent character. It is accompanied by septicopyemia. Peracute cases take the form of a general septicemia.

Occurrence.—The disease is widespread and occurs in practically all breeding districts. Foals and calves are most commonly affected. Like the dysentery of sucklings, with which it is closely related, it often appears as an enzoötic, attacking large numbers of young animals, and, as it is generally fatal, seriously interferes with breeding operations.

Etiology.—In calves the Bacterium septicum and the colon bacillus, acting jointly, are accused. In colts various pus cocci (staphylococci and streptococci) have been found in the organs, blood and joints. It is very probable that several pathogenic microörganisms are related etiologically to the disease.

Natural Infection.—*Extra-uterine.*—*Via* the navel cord which has not yet become dried and shrivelled and still contains the Whartonian gelatin. The danger of infection is probably greatly enhanced by ligation as is usually practised in animals. Naturally, filthy bedding or floors or ground covered with manure, urine or the discharges from the infected navel of a developed case of the disease increase the danger. It happens, therefore, that a sporadic case can spread the infection until every colt or calf born on the premises becomes a victim.

Intra-uterine Infection.—As some animals are born with the disease (symptoms at birth; pathological lesions present too old to have developed since birth), an infection *in utero* from the pregnant dam, in whose blood pathogenic bacteria have appeared, is assumed.

A further possibility would be the permanent infection of the uterus of the dam and the passage of the bacteria from it to the placenta and fetus. This would explain those cases where a given mare bears year after year colts which are born with the disease. In cases of antepartum infection the navel is usually intact.

Necropsy.—The postmortem lesions vary, depending on whether the case was peracute, acute, or chronic.

(*a*) *Peracute.*—There are no marked lesions except those of a general septic infection, such as cloudy swelling of the parenchymatous organs, petechiæ and ecchymoses in the serous and mucous membranes and acute swelling of the lymph glands.

(*b*) In acute cases the navel is usually swollen and firm and the navel ring is open. Purulent or putrid exudate may be pressed out. Quite frequently an abscess forms in the abdominal wall. The umbilical vein and one or both umbilical arteries are distended at intervals or throughout their whole length. On palpation they feel firm or fluctuating. When opened a dirty red, often fetid, exudate flows out. The inner surface of the bloodvessels may be covered with a fibrinous coagulum and is sometimes ulcerous. The infection may involve the peritoneum, leading to an adhesive peritonitis, causing adhesions among the abdominal organs. The portal vein and its branches show thrombi extending into the liver.

Metastatic abscesses especially of the lungs, liver and lymph glands are not infrequent. Occasionally there may be present pleuritis and pericarditis. A fibrinous or suppurative panophthalmia is not rare. The affected joints, especially the tarsal and carpal, show suppurative arthritis. The periarticular connective tissue is infiltrated with either a serofibrinous or seropurulent exudate; periarticular abscesses are not uncommon. A communication between the abscess and the diseased joint cavity is not unusual. The adjacent tendon sheaths may also be involved.

(*c*) *Chronic Cases.*—The changes here are frequently in the lungs, such as bronchopneumonia, with fibrinous or serofibrinous pleuritis, and pericarditis. In the later stages caseous foci are found in the lungs. The mediastinal and peribronchial lymph glands are often enlarged and caseated. The cadaver is emaciated and shows symptoms of general anemia and cachexia.

Symptoms.—In pyosepticemia of sucklings the disease may appear clinically as a general septicemia, an affection of the joints or of the navel, and may be complicated with metastatic changes in internal organs (lungs, liver, kidneys, mesenteric glands, brain, etc.). The clinical picture will, therefore, vary. In calves the disease is often less acute than in colts and is less apt to involve the articulations. Both local and general symptoms are observed, the local often being noticed first. Three clinical forms of the disease are recognized:

(*a*) *Septicemic Form.*—In the septicemic form there appear within a day or two after birth the following symptoms: Refusal to suck; the patient is languid, remains recumbent, and, if lifted to its feet, is rarely able to stand, the head held low, the whole body limp. The temperature is elevated (105° F.), the heart beat rapid and the respirations dyspneic. Death occurs in one to two days. In some cases, due to metastasis, symptoms in internal organs (lungs) develop.

(*b*) *Articular Form.*—Most frequently in colts the first symptom noted is swelling of a joint or joints appearing within a few days after birth. The owner generally assumes that the swollen part has been trodden on by the dam. The enlargement occurs in a limb joint (tarsal, carpal, femorotibial) and is inflammatory in character. The surrounding tissue is edematous. While the swelling sometimes promptly disappears, generally it persists, fluctuates and perforates or is lanced, discharging pus. Flexion of the affected joint causes pain and severe lameness. If many joints are attacked the young patient may be unable to stand. Marked suppuration does not always take place. The swelling may never open spontaneously, but lead to a subacute or chronic arthritis with periarthritis, causing temporary or permanent enlargement of the joint.

(*c*) *Umbilical Form.*—In some cases a local inflammation appears, the navel becoming hot, painful, and swollen, the stump moist and discharging pus or ichor. Between the umbilicus and the ensiform cartilage a firm strand, the size of a finger, may be felt in the abdominal wall running toward the liver. The local symptoms may abate in a few days and the patient recover, or, on the other hand, a general septic infection ending in death results. Not infrequently metastatic abscesses form in parenchymatous organs producing symptoms varying with their distribution:

Lungs.—If the lungs are involved the young patient will cough, have nasal discharge and show dyspnea. On auscultation bronchial sounds, rales, and rhonchi may be heard and on percussion areas of dulness and tympany. The animal becomes anemic, emaciated, and extremely weak, remaining most of the time lying on the sternum. In this form it may linger several weeks.

Stomach and Bowels. — The principal symptoms are loss of appetite, abdominal pain, and diarrhea. The feces are like those described under dysentery of newborn animals.

Spinal Cord.—When the cord is invaded by metastatic abscesses, symptoms of paraplegia appear. These may come on suddenly or gradually and follow a period of apparent recovery. In some cases of spinal paralysis the preceding navel symptoms may have been entirely overlooked.

Brain.—The patient is usually very dull and unconscious of its surroundings. It may show forced movements, spasms, and convulsions, opisthotonos, wry-neck, and paralysis.

Diagnosis.—Generally not difficult. When diarrhea is present, a differentiation between joint ill and dysentery is impossible. If the navel is intact and joint swellings are absent, the diagnosis could be made only by a knowledge that the disease exists on the premises (other sucklings showing a more characteristic form of the disease).

Course.—Peracute cases die in twelve to forty-eight hours. In the acute, septicemic form the patients die in two or three days. In subacute cases, especially when the infection occurred late after birth, the navel infection remains local and eventually heals, the patient under proper treatment recovering in two to three weeks. Chronic (lung) cases may last one to two months. If the abscesses in the lungs or liver become fully encapsuled, recovery may even take place.

Prognosis. — Depending on the age attacked and the form the disease assumes, the mortality will vary greatly. It is higher in colts than in calves, relatively more resistant to pus infections. Fully 60 to 90 per cent. die. Most sucklings which do recover remain stunted and do not "do well" for months after the attack (internal abscesses). In cases of joint affection in colts, if the suppuration is mild, recovery is common. When general symptoms appear the prognosis is bad. Bowel symptoms (diarrhea) generally promptly lead to death.

Treatment.—An internal treatment is useless. When general septicemia is present the patient is beyond aid. The most rational treatment is surgical and applied to the primary

seat of infection—the umbilicus. It should be thoroughly disinfected (tincture of iodin), abscesses opened, necrotic portions carefully removed, and drainage provided. At the same time the patient should be kept in a light, well ventilated, clean place. Painting the joint swellings with iodin tincture is recommended. Opening them to evacuate pus, while rarely of therapeutic value, is demanded. If there is no evidence of pus (heat, pain, firm fluctuation, temperature), the knife should be spared.

Vaccination.—The use of polyvalent or autogenic bacterins to arrest the progress of the disease is highly recommended by practitioners. In some cases the results seem very satisfactory. More experiments are desired.

Prevention.—Where infection threatens, the dam about to be delivered should be placed in a clean, light, well ventilated stall (preferably a maternity stall with cement floor and walls) and the bedding sprinkled with some non-poisonous antiseptic (creolin). The genitals may be flushed out with some good antiseptic (creolin 2 per cent.) and the tail and buttocks cleaned with it. As soon as the young animal is born, the navel cord should be gently "milked" with disinfected hands, to remove the Whartonian gelatin, and thoroughly covered with a good antiseptic strew powder (dried alum, camphor, starch, equal parts) to aid in the desiccation of the cord. The application of the strew powder should be repeated often until the stump is completely shrivelled and dried. By frequent application, say once every half hour for the first two hours after birth, the cord becomes thoroughly mummified in two to four hours (Williams).

INFLUENZA OF THE HORSE.

General Remarks.—Under the collective term "influenza" intermittently have been grouped together at least two acute, infectious diseases one of which assumed the form of a general infection of the blood with inflammation of the mucous membranes, subcutis, tendon sheaths and tendons, and the other an infectious lobar pneumonia or pleuropneumonia. From a purely clinical standpoint some authorities

(Dieckerhoff, Bang) have pronounced influenza not to be a clinical entity. They assumed that under this head at least two separate and distinct diseases existed which differed in their pathogenesis, period of incubation and symptomatology. They were therefore given separate names. In Germany the catarrhal form was known as "Staupe" (influenza), and the pectoral as "Brustseuche" ("chest plague"). Dieckerhoff described a third disease, included under the term influenza, which he called "Skalma."

The recent experiments of Gaffky and Lührs, which concerned the etiological factor primarily, indicate that the term "influenza" has been made too inclusive. Their results seem to substantiate the work done by former authorities, especially Dieckerhoff and Bang. Two separate and distinct diseases are therefore recognized in this work.

Influenza of the Horse (*Catarrhal Fever; Pink Eye; Typhoid Fever*).—**Definition.**—Influenza is an acute, contagio-infectious, usually enzoötic, disease of the horse which is characterized by inflammation of the mucous membranes, subcutis, and tendons. It is due to a filtrable virus.

Occurrence.—The disease is very widespread, occurring in all countries. North America was visited by an epizoötic of influenza in 1776 and again in a still severer form in 1870–72 when it swept the Continent from Canada to the Ohio, and westward to California. The disease is now permanent in this country where it is kept alive by the unsanitary stables of horse-dealers and in livery barns, whence it is from time to time spread. It may also attack asses, mules and zebras.

Etiology.—The most recent investigations indicate that the cause of influenza is a filtrable virus which appears in the blood and probably other body fluids. Gaffky produced influenza in healthy horses by subcutaneous injections (5 c.c.) of defibrinated blood from naturally infected horses. The period of incubation is from five to six days. When the blood was injected intravenously the period of incubation was only four days. In artificially infected horses the characteristic symptoms were produced in forty hours. Similar experiments made with filtered blood serum gave positive results.

The infectiousness of the blood was annihilated by the addition of the citrate of ammonia. In no case was he able to obtain growths on culture media from either the blood or blood serum used.

Natural Infection.—The disease is probably spread by the nasal discharge and feces of infected horses. In all probability apparently recovered cases may be "germ carriers" introducing the disease into stables and when brought in contact with susceptible individuals. Indirectly the infection may be carried by contaminated food, bedding, manure, stable utensils, harness or in the clothing of grooms, horsemen and veterinarians. Many sale and livery stables, due to their lack of light, ventilation and cleanliness, may harbor the infection for an indefinite period ("stable miasma") and all horses, especially "green" horses from the country, placed in them fall victims of the disease. The same is true of railway cars and stockyards which have not been properly disinfected. Public watering troughs may also harbor infection and thus contribute to the spread of the disease. Although influenza is commonly enzoötic it not unrarely assumes an epizoötic form. While it appears at all seasons of the year, in the late winter and early spring horses seem most predisposed. This is probably due to the condition of the mucous membranes, which are generally catarrhally inflamed from "colds" in these seasons. Influenza will attack horses of all ages, but it is not so common in colts less than a year old, nor aged horses. Horses three to five years of age are most frequently affected. One attack produces lasting immunity. Horses which have had influenza may, however, take infectious pneumonia, and *vice versa*. The infection is most commonly taken in with the food and water. Infection *via* the respiratory tract is highly probable but not proved.

Symptoms.—The period of incubation is usually less than a week. A minimum period of one day and a maximum of ten days are recognized. The initial symptoms in many cases are moderately developed, but in the majority they are quite intense. Usually the first symptoms noticed by the owner or caretaker are loss of appetite, dulness, and marked languor. The patient stands with head down, eyes closed, ears drooped,

appearing excessively fatigued. The gait is staggering, and crackling of joints is heard when the patient is moved.

The temperature is elevated (103° to 105° F.). It usually drops by crisis on the second or third day. The pulse in the earlier stages is relatively low compared with the fever, but as soon as cloudy swelling of the heart begins it goes up to 60 to 100, and becomes irregular and weak.

Eye Lesions.—Conjunctivitis appears early, and later keratitis and iritis (exudate in anterior chamber, contraction of pupil, etc.). The blood is charged with bile pigment, hence the conjunctiva assumes a yellowish, ochre, or a natural mahogany color. Sometimes a phlegmonous conjunctivitis with marked swelling and eversion of the eyelids occurs. These symptoms generally rapidly subside, provided the cases take the normal benign course.

Respiratory Tract.—Cough is usually present. It is generally strong and moist. Nasal discharge, at first clear but later turbid and more profuse, is a constant symptom. The submaxillary glands are swollen. The patient shows moderate dyspnea; the respirations 20 to 25. Percussion normal. Auscultation, exaggerated vesicular breathing and moist rales.

Digestive Tract.—Lost or impaired appetite; during the fever increased thirst is noted. Usually symptoms of catarrhal stomatitis and pharyngitis are present (coated tongue, warm, congested mucous membranes; regurgitation of water through the nostrils). The gums are swollen ("lampas"), and along their border a marked yellowish discoloration is seen. The bowels during the febrile stage are constipated, the feces passed in the form of hard, small, mucous-covered pellets of sour odor. Later they become soft and fetid, exhaustive diarrhea with tenesmus sets in. In some cases moderate colic symptoms occur (pawing, lying down, slight distention of abdomen).

Urinogenital Tract.—During the height of the fever very little urine is voided. Its specific gravity is high, color dark, reaction acid. It is rich in sediment. Under the microscope tube casts and epithelial cells (renal pelvis, bladder) are found in it. With the falling of the temperature a critical polyuria

develops. In mares the vulva may swell and a mucopurulent discharge be present. Pregnant mares often abort. In stallions the scrotum is often enlarged and orchitis may be a symptom.

Skin.—While there is generally an edema of the hind limbs present in influenza, in some outbreaks it is much more pronounced than in others. With increased heart weakness, edemas appear in pendent portions of the body (under chest, abdomen, udder or scrotum). Occasional cases of tendovaginitis, particularly of the flexor tendons, are noted, and once in a while laminitis occurs. The patients usually lose flesh rapidly, and become very weak and debilitated.

A very dangerous complication is pneumonia or pleuropneumonia, which usually assumes the catarrhal form, and may be hemorrhagic in character. Such cases seriously disturb the functions of the heart and kidneys, and often lead to death.

Diagnosis.—The diagnosis is usually not difficult. The rapid spread of the disease from animal to animal, the icteric mucous membranes, conjunctivitis and skin edemas differentiate it from other diseases attended by high fever. When these symptoms are absent the highly infectious character of the disease, as evidenced by the rapid spread, is significant.

While a differentiation between influenza and infectious pneumonia of the horse can usually be made if the course of the disease can be studied, at first visit it may be very difficult. The prodromal symptoms of the two diseases are very similar. The high initial fever, the swelling and icteric discoloration of the conjunctiva, the contagiousness and the inflammatory swellings in the subcutis and tendon sheaths are common to both. Usually, however, on the second or third day in infectious pneumonia, tangible symptoms of pneumonia develop; in influenza pneumonia occurs as a complication, and usually much later in the course of the disease. Furthermore, influenza is more apt to attack the intestinal tract early. A differentiation between influenza and infectious anemia would become important only in districts in which the latter disease exists. In infectious anemia no catarrhal symptoms develop, the mucous membranes show

petechiæ, the blood serum is opalescent and plainly tinged with red. Infectious anemia is not highly infectious. From strangles influenza is distinguished by the tendency for lymph glands to suppurate, which characterizes the former disease.

Course.—The course is usually about one week or the disease may terminate favorably in less time provided no complications arise. When the disease progress is interrupted by pneumonia, encephalitis, enteritis, or degeneration of the heart muscle the course is protracted and the termination fatal. As a rule; however, influenza is a mild disease, and unless the patient is worked, kept in unsanitary surroundings, or given too much medicinal treatment, recovery in a few days is the rule.

Prognosis.—The mortality is 1 to 4 per cent. During some outbreaks the disease appears more malignant than in others, and complications are commoner. The continuation of a rather high fever for not longer than five to six days is, *per se*, of no significance provided the pulse remains good, the patient does not become dyspneic, and no diarrhea attend.

Treatment.—Most important in the treatment of a self-limiting disease like influenza is to provide the patient with light, ventilation, and cleanliness. In mild sunny weather, if at all feasible, place the patient out of doors during the day. The food should be nourishing and easily digested, and given in small rations (grass, alfalfa, a bran mash with plenty of salt, scalded oats, etc.). To induce the patient to eat, some brown sugar may be strewn over the feed. In case a meal is refused or not entirely eaten it should not remain indefinitely in the feed box, but be removed and the box cleaned. Skimmed milk may be given (three to four gallons daily) if the patient will drink it. Hanging blankets wet with cold formalin solution in the stable helps lower the temperature in hot weather and assists disinfection. A thorough cleaning up and disinfection of the infected quarters will do more to reduce the mortality than drugs. The unequal distribution of the surface temperature should be regulated by proper grooming. The legs should be covered with Derby or flannel bandages, which tend to prevent edema. Medicinal treatment should be employed only when absolutely necessary. Over-drugging

in influenza always increases the mortality. The following conditions may require medicinal aid:

Fever.—The fever should be let alone unless it reaches an unusual height or is continuing rather indefinitely and seriously affecting the heart action. It may be reduced by cold, rectal infusions which not only lower temperature but stimulate peristalsis and unload the bowels. The use of antipyretics, such as acetanilid or phenacetin (℥ij every three hours) is dangerous, as both drugs are powerful heart depressants. They should be administered, if at all, with caution. Sodium salicylate (℥iv) is less dangerous in this regard, but may irritate the stomach.

Heart Weakness.—When the pulse becomes rapid and weak one dose of digitalis (Squibb's fluidextract ℥ij to iv) has a remarkable toning effect. Strophanthus (℥ij to iv), caffein (℥ss to j), camphor oil (subcutaneously ℥ss to j) are also effective. When the pulse is strong enough, alcohol (brandy ℥iij, with ether ℥ss, in a pint of cold water) is of service.

Gastro-intestinal Tract.—Artificial Carlsbad salts (℥ij to iij) to which bitter agents (gentian, nux vomica) are added, are useful. Dram doses of a solution of strychnin nitrate in water (strychnin gr. j, water ℥j) may be used. Bowel disinfectants are also employed, as creolin (℥ss to ℥j) or naphtholin (℥ss). Diarrhea may be fought with astringents like tannin with opium. Starch clysters are helpful. The profuse diarrhea attending septicemia is impossible to check. Calomel (grs. xv) with bicarbonate of soda (℥iss) mixed with powdered licorice root (℥j), smeared over the teeth as an electuary, may be tried.

Skin.—Leg swellings are best reduced by elastic bandages, and if the pulse will permit, moderate exercise. Burrow's solution to which a little gum camphor is added may be used as a leg wash under the bandage.

Eyes.—The eyes should be treated as in conjunctivitis (boric acid solution 2 per cent.) and the pulmonary and pleural symptoms met as recommended under the diseases of the respiratory tract.

Convalescent patients should not be worked until strong enough and full appetite returned. The animal is generally

fully recovered fourteen days after the temperature has become normal.

Infectious Fibrinous Pneumonia of the Horse (*Chest Plague; Pectoral Influenza; Pleurisy; Shipping Fever.*)—**Definition.**—An acute, febrile, contagio-infectious disease of the horse which in typical cases appears as a fibrinous pneumonia or pleuropneumonia with which is associated inflammation of the subcutis and tendon sheaths. In mild cases it may assume the form of a general, febrile disease of short duration.

Occurrence.—Infectious fibrinous pneumonia is widespread, occurring in all countries. Statistically it is difficult to state, however, anything definite in regard to its prevalency as it has been so often confused with influenza. The disease is most apt to appear in large stables in which many horses are kept, and especially during inclement weather. The disease in passing through a stable does not usually affect the horses in the order in which they stand in the stalls, the infection tending to spread unevenly, skipping apparently susceptible horses. Infectious pneumonia does not become epizoötic as readily as does influenza; it is more apt to remain confined to an infected stable, from which it may be spread, however, by a convalescent horse. While infectious pneumonia will not involve in a given country as many horses as influenza, on account of its higher mortality and a greater tendency to leave behind sequels ("heaves," relapsed cases) which may permanently impair the efficiency of the horses attacked, it can assume even greater economic importance.

Etiology.—The cause of infectious pneumonia of the horse has not yet been identified. According to Gaffky and Lührs the bronchial slime of an infected patient contains the virus of the disease, at least in the early stages. In typical cases of infectious pneumonia in which the patients were destroyed on the third or fourth day, in the bronchi was found a quantity of yellow, transparent, viscid fluid which contained no bacteria. In two experiments on twenty-four colts inoculated with this fluid, by painting it upon the mucosa of the nostrils and in the mouth, the colts became typically ill with infectious pneumonia in twenty-three to forty-two days. Undoubtedly

a specific virus, which may be filtrable, forms the true cause of the disease. In all probability, however, other bacteria, principally the Streptococcus pyogenes equi, and the Bacillus equi septicus, are secondary invaders, contributing to the underlying disease process and clinical phenomena.

Natural Infection.—The way in which the disease spreads naturally is at present not known. The infection seems to be spread by more or less intimate contact between the sick horse and susceptible ones. The transmission through intermediate agents, such as food, water, stable utensils, etc., or through persons or insects, has not been demonstrated experimentally. However, practical experience in other diseases and with this disease would not exclude indirect transmission. Sporadic outbreaks in stables could be explained, however, as coming from apparently healthy "germ carriers" or "missed cases" of infectious pneumonia, *i. e.*, where still exist in the lungs or occasionally in other organs unhealed foci of infection. As predisposing factors anything which reduces the resistance of the susceptible animal unquestionably has a bearing on the origin of the disease. Therefore, refrigeration, overwork, bad sanitary conditions, become predisposing factors. The disease is rare in very young or aged horses, and occurs usually in animals in the prime of life.

One attack produces immunity for only a short period. Individual instances are recorded in which a given horse has suffered repeated mild attacks of the disease. An attack of infectious pneumonia in no way influences the susceptibility to influenza infection.

Necropsy.—On postmortem, where the disease has assumed a typical form, the lesions are those identified with fibrinous or even hemorrhagic pneumonia, with a marked tendency to gangrene. The extent and distribution of the inflammation varies. In some cases the pneumonia is of the lobar, in others the lobular type. The exudate is usually of a hemorrhagic character. Yellow-gray areas of necrosis throughout the lung tissues are commonly noted appearing as encapsuled pus centers or gangrenous foci.

The pleuritis is serofibrinous; the thorax may contain several gallons of serous exudate. Adhesions between the

lung and chest wall (adhesive pleuritis) is a common finding. Empyema of the thorax may be present.

In the gastro-intestinal tract appear evidence of catarrh and, especially in the small intestines and cecum, inflammatory thickenings and ulcerations.

Symptoms.—Infectious pneumonia of the horse is characterized by pneumonia and pleuritis. The pneumonia may assume either the lobar (benign) or lobular (malignant) type. (For details refer to the chapter on Pneumonias of the Horse.)

Complications.—The most dangerous complications are: (*a*) Parenchymatous degeneration of the heart characterized by tumultuous heart beat and a rapid, arrhythmic, weak pulse. (*b*) Nephritis distinguished by scantiness of urine (anuria) and albuminuria. (*c*) Septicemia, usually following pulmonary gangrene, and recognized by continued high fever, chills, rapid, irregular and weak pulse, and exhaustive diarrhea. (*d*) Paraplegia is rarely noted, but is usually a fatal complication. (*e*) Paralysis of peripheral nerves (facial, recurrent) which may persist for several weeks. (*f*) Swellings of the limbs and pendent parts of the body, seen especially in the latter stages, are troublesome. (*g*) Tendinitis and tendovaginitis. (*h*) Founder. (*i*) Abortion. (*j*) Decubitus.

Diagnosis.—Usually not difficult. Infectious pneumonia cannot be differentiated from sporadic lobar pneumonias if such occur in the horse. Every case of lobar pneumonia, therefore, should be isolated. Obviously this does not apply to all lobular pneumonias, especially foreign body, medicinal, or hypostatic.

Course.—When the pneumonia assumes the lobar type with mild pleuritis the course is about two weeks. If, however, the lobular form of pneumonia with pronounced bilateral pleuritis is present, the course is prolonged for weeks, with remissions and exacerbations, either ending finally in death or leaving the patient permanently wind-broken (pleural adhesions; roaring). (See Lobular Pneumonia.)

Prognosis.—The mortality is 1 to 4 per cent. The following factors are important in the prognosis; they are all grave symptoms: (*a*) Heart paralysis (rapid, empty pulse, venous pulse); (*b*) extent of pneumonia and pleuritis (bilateral with

great effusion); (*c*) pneumonia centralis (sudden dyspnea with rise in the temperature during the course of the disease); (*d*) hemorrhages (epistaxis, bloody pleural exudate); (*e*) temperature continues high for over a week or is remittent in character; (*f*) diarrhea; (*g*) lung gangrene; (*h*) brain symptoms.

Treatment.—See Pneumonias of the Horse.

PURPURA HEMORRHAGICA. PETECHIAL FEVER.

Definition.—Purpura hemorrhagica is an acute, noncontagious disease the result of a toxemia usually developing as a sequel to a specific infectious disease. It is characterized by marked edematous swellings of the head and limbs and petechiæ in the mucous membranes and internal organs.

Occurrence.—Frequent in the horse. Cases are said to occur in the ox and the dog. The disease usually is seen to follow in the wake of an outbreak of influenza or strangles, individual cases during convalescence developing the symptoms which typify the condition. It may also be a sequel to other debilitating diseases, especially if the patient has been kept in unsanitary surroundings and poorly nourished. It is rare in horses under two years old, but may attack aged individuals.

Etiology.—Purpura in horses, as noted, is a secondary disease, and is probably always associated with a hidden pus pocket or an area of necrosis somewhere on or in the body of the animal attacked. It is, therefore, most apt to follow diseases or conditions attended by pus formation or necrosis (strangles, pharyngitis, empyema of facial sinuses, suppurative tooth diseases, internal abscesses, old castration wounds, necrosis of the skin). The disease is not transmissible either by inoculation or blood transfusion; there are no specific organisms found in the blood. Very probable, therefore, is the theory that toxins, originating in a primary pus or necrotic focus, absorbed by the blood, in time intoxicate the animal the resistance of which has been lowered by disease, unhygienic environment and poor food. While the blood itself seems to suffer little change in physical properties, the

walls of the bloodvessels, especially the capillaries, become porous or rupture, allowing the blood to escape into the surrounding tissue. Thus smaller petechiæ or more extensive ecchymoses are produced. From the larger veins the transudation of serum leads to the characteristic swellings.

Symptoms.—Petechiæ in the mucous membranes of the head. The hemorrhagic spots vary in size from a hemp seed to a bean. Sometimes on the nasal mucosa (septum and turbinates) the areas assume the form of stripes or striations, the membrane being swollen. The petechiæ have a purple color. Sometimes ulceration is noted. There is nasal discharge of a yellowish or reddish serum or fluid blood. In the eyelid conjunctivitis is present with petechiæ and slight sanguineous discharge. The mucous membrane of the mouth, though more rarely, may also be involved.

Swellings of the skin usually appear with the petechiæ or follow in a day or two. These are often at first isolated, flattened, urticaria-like tumefactions from the size of a pigeon's egg to that of a grape fruit, and larger by confluence. They are firm or edematous on palpation. More common, however, are diffuse swellings of the lower portion of the head, ventral part of the thorax and abdomen, udder, prepuce, or scrotum and limbs. In the regions of the nostrils and lips they commonly begin and rapidly extend upward toward the eyes. The swellings are firm, and, especially at the flexion of joints, the overlying skin cracks open and a yellow, viscid, serous fluid exudes, which later dries, leaving a brown scab. The swellings terminate abruptly ("tied off") in the head a short distance below the eyes and in the limbs at the elbow and stifle.

As long as the lumen of the nasal cavities and larynx is not encroached upon by swelling the respirations are not increased. From swelling, especially of the false nostrils and turbinates, dyspnea develops. If a hemorrhagic or inflammatory infiltration of the larynx appear, pronounced inspiratory and expiratory dyspnea with stertorous laryngeal sound follows. (See Glottis Edema).

The leg swellings are painful, and mechanically interfere with locomotion and getting up and down. Usually the

patients remain standing unless greatly fatigued or the attack mild.

From swelling of the head mastication is difficult or impossible. If the pharynx is involved, blood-tinged saliva drools from the mouth and regurgitation through the nostrils is noted. Food is sometimes retained between the teeth and cheeks, where it decomposes and emits a fetid odor. The appetite is good in the earlier stages and may be retained throughout mild attacks. If hemorrhagic or inflammatory infiltration of the gastro-intestinal tract occur, colic symptoms follow. The feces are sometimes blood-stained, and diarrhea may set in as a symptom of some complication (septicemia).

The temperature is usually only slightly elevated and may remain practically normal throughout the course of the disease, provided no complications occur. If complications exist, or the disease which preceded the attack of purpura was a feverish one and still to a degree persist, the temperature is elevated. Purpura and the disease from which it develops may occur concomitantly in the same patient.

In the early stages the pulse remains about normal. As a rule, it rarely goes beyond 50 to 60. If it exceed 80, complications are present.

Complications.—(*a*) Gangrene of the skin with sloughing is not an uncommon complication. The process of healing is slow (sometimes two to three months), and frequently unsightly scars are left behind, greatly reducing the market value of the horse.

(*b*) Foreign body pneumonia: This is apt to follow severe pharyngitis, with dysphagia, or more rarely may be due to the aspiration of necrotic pieces of mucous membrane from the nasal cavities and throat. Pulmonary gangrene usually follows, leading to death in two to three weeks.

(*c*) Septicemia usually attended by a sudden disappearance of the symptoms, profuse diarrhea, recumbent position, and decubitus. Pulse is elevated, temperature high.

(*d*) Gastro-enteritis: Some cases begin with symptoms of colic which yield to proper treatment, the purpura following taking a benign course. If, however, large extravasations occur in the stomach, small or large intestines, severe gastro-

enteritis is noted. Necrosis of the extravasated areas in the bowel may take place and perforative peritonitis result. The patient presents violent colic symptoms and dies in twenty-four to thirty-six hours.

Diagnosis.—Purpura hemorrhagica is characterized by the typical "tied-off" swellings and petechiæ in the visible mucous membranes. The diagnosis is easy if the case is at all typical. When ulcers occur in the nasal mucous membrane it might be mistaken for acute glanders. The crater-like margin and lardaceous base of the ulcers, the nodules, star-shaped cicatrices, enlargement of the submaxillary lymph glands, and the absence of petechiæ in glanders suffice to distinguish between the diseases. Glanders and purpura may both appear in the same patient. With malignant edema the disease would rarely be confused (see this). Urticaria could only be mistaken for purpura in the early stages of the disease. Anthrax is a rare disease in horses in the northern United States. In the South it usually assumes the cutaneous form, the swellings have very little resemblance to purpura. (See Anthrax.)

Course.—Very varied. Mild cases sometimes recover in less than one week. Some are attacked with violent sepsis and die in two or three days after the first symptoms are observed. As a rule, a case of this disease lasts two to six weeks, with many exacerbations and remissions. Recovery is sometimes rapid and unexpected. Some patients give the attending veterinarian great encouragement until some unexpected complication (septicemia, pneumonia) occurs which leads to death. Defects in the skin due to gangrene may take months to heal.

Prognosis.—The prognosis is generally doubtful. When the swellings on the head are extensive, life is always threatened. A high pulse (80), diarrhea, no appetite, constant recumbency, with attending decubitus and severe colic, are bad symptoms. The disappearance of the swellings is not a good sign unless the other symptoms also improve. Blood extravasations may form at any time in any organ (bowels, brain, and spinal cord), precipitating sudden death. The mortality varies from 30 to 50 per cent. and is largely

dependent on the hygienic surroundings and care of the patient.

Treatment.—As yet no successful specific treatment has been devised to combat the disease. The principal factor in bringing cases to a favorable termination is good hygiene. The patient should be placed in a light, clean, well-ventilated, bedded box stall and the head halter removed (prevents necrosis of skin of face). In proper weather it may be let run out of doors during the day. A light blanket may be used for covering. Allow plenty of clean water. The food should consist of oats, grass, and bran with plenty of salt. If there is difficulty in swallowing, gruels or milk may be used or tube-feeding employed. The treatment is surgical, serotherapeutic and medicinal. The surgical treatment consists in a thorough disinfection of the tumefactions, wounds, ulcers, and abscesses (creolin, lysol 3 per cent.). The application of Burrows' solution with camphor (camphor ℥iss, lead acetate ℥vj, alum ℥iij, a tablespoonful to a winebottleful of water) is good to ward off gangrene. Spirits of turpentine applied two or three times is said to have like effect. Scarification of the swellings as usually practised does no good and opens an avenue for further infection.

When suffocation threatens, tracheotomy should be performed. While it no doubt prolongs life, in most cases where called for, death follows.

Good results are reported from the use of antistreptococcic serum in doses from 25 to 50 c.c. It may be given subcutaneously and intratracheally. Naturally, its effect will depend upon the presence of a streptococcus infection which is by no means proved in purpura. Further, the serum is expensive. Perhaps some of the good results reported are due less to the potency of the serum than to the fact, that when used, the resistance of the patient is not being reduced by overdrugging, a common practice before the introduction of modern serum therapy.

The medicinal treatment is very varied and purely empirical. The following suggests some of the possibilities in this regard:

1. Drugs to increase coagulability of the blood:
   (*a*) Calcium chlorid with gelatin (ʒss thrice daily) *via* mouth or rectum.
   (*b*) Turpentine (ʒj thrice daily) in pint of milk.
   (*c*) Adrenalin (1 pro mille, dose ʒiv).
2. Intestinal disinfectants:
   (*a*) Calomel (grs. xx thrice daily).
   (*b*) Ichthyol in form of sodium sulpho-ichthyolate (ʒiiss thrice daily).
3. Other treatments:
   (*a*) Intratracheal injections of Lugol's solution (ʒj intratracheal).
   (*b*) Collargol intravenous (ʒj of a 1.5 per cent. solution in water thrice daily).

Tonic powders assist during convalescence, as artificial Carlsbad salts to which some powdered nux vomica is added.

HOG-CHOLERA. SWINE FEVER.

Definition.—Hog-cholera is a contagio-infectious disease due to a filtrable virus. It assumes a variety of forms, but primarily is a septicemia with secondary pneumonia, and diphtheritic gastro-enteritis. Clinically, acute and chronic types are distinguished.

Occurrence.—The disease is widespread, occurring in all countries. It is especially prevalent in the great corn-belt of the United States in which hog-raising is extensively developed. The first recorded outbreak of hog-cholera occurred in 1833 in Ohio, into which state it was probably introduced with imported hogs or bacon from Europe. Following closely in the wake of the development of transportation facilities, especially railways, it spread from the Middle West to all parts of the country, especially toward the West, where the breeding of swine had become a profitable industry. The losses from cholera are enormous, reaching as high as $200,000,000 (45,000,000 hogs infected) in 1888 and averaging close to $50,000,000 annually. The farmers of the corn-belt naturally suffer the brunt of damage wrought by the disease. Like most infectious diseases assuming an epizoötic form the out-

breaks come in waves. While some years swine are comparatively free from the infection, or it assumes a mild form, in others it is widespread and especially malignant. As far as is known, hogs are the only animals which take the disease.

Etiology.—The cause is an ultramicroscopic organism occurring in the blood, urine, and sometimes in the feces of cholera-sick hogs. The parts played by the Bacillus suipestifer and the Bacterium suisepticum are probably incidental, the former inducing principally the chronic gastro-intestinal and the latter the lung and pleural lesions. (See Swine Plague.)

Natural Infection.—As the organism of cholera is found in the urine and feces, the pens, yards, etc., in which sick hogs have been kept, become contaminated with it. Healthy but susceptible swine become infected largely *via* the digestive tract through food and water polluted with the secretions and excretions of the diseased. Hog-cholera is introduced into a non-infected farm as follows:

(*a*) By infected hogs: These may stray from neighboring infected herds, be borrowed (breeding boars), brought in by purchase, or show-swine returned from fairs, expositions, etc., may bring the infection home. Hogs introduced during the incubative stage of the disease. Such swine may seem healthy at the time of purchase, but a few days later the symptoms appear. Hogs suffering from cholera in a chronic form ("germ carriers"), especially old breeding boars and sows, showing no typical symptoms.

(*b*) The infection may also be brought into the premises by such intermediary agents as stray dogs, crows, fowls. Persons can also carry it on their boots and clothing. Once the disease breaks out it is spread, as noted, by the urine, feces, and other discharges of the sick, and by careless disposition of the dead (throwing carcasses into waterways or leaving them on the fields; too shallow burial, incomplete cremation, etc.). Hauling the carcasses in wagons through the premises and along the roadways is a further factor in the spread of the disease. It is a common practice when hog-cholera is discovered to exist on a farm for the owner to attempt getting rid of the disease by selling those hogs which are in a market-

able condition. Too often a close discrimination between the infected and uninfected is not made. In transit or soon after arrival at their destination the disease may break out, infecting the railway cars, shutes, pens, etc. Hog-cholera, therefore, is spread over great areas, and especially along lines of traffic.

A transmission of the disease along the course of a flowing stream is probable, the current carrying the infection.

Necropsy.—(*a*) *Septicemic Form.*—Lesions, as in hemorrhagic septicemia. Petechiæ and ecchymoses of the skin, serous membranes and of the mucous membrane of the gastro-intestinal tract, especially of the bowels. In the latter, accumulations of clotted blood may be found; the lymph glands are swollen and blood-shot; the parenchymatous organs congested, the spleen little if at all swollen. The petechiæ over the kidneys give them a spotted appearance, known as "turkey egg" kidney. On the serous membranes fibrinous exudates appear; catarrh of the stomach and small intestine is also noted.

(*b*) *Intestinal Form.*—The most characteristic lesions are noted in the large bowels, especially in the cecum. There appear in the mucosa and submucosa, flat, round, hard, elevated, yellowish, greenish-yellow or gray areas of necrosis. The areas vary in size from a pin-head to a twenty-five cent piece. The larger ones are commonly known as "button ulcers."

(*c*) *Pectoral Form.*—The lungs present the signs of pneumonia (croupous, catarrhal or mixed) with serofibrinous pleuritis and pericarditis. In subacute or chronic cases multiple necrotic foci are present in the lung with caseation which may involve the lymph glands. (Most of these cases are probably swine plague.)

The following necropsy lesions speak for hog-cholera in outbreaks occurring in the United States:

(*a*) Petechiæ and ecchymoses of skin.

(*b*) Petechiæ and ecchymoses of serous membranes, especially of the pleura, epicardium and peritoneum over the kidneys ("turkey egg kidney").

(*c*) Swollen lymph glands.

(*d*) Button ulcers in the bowel (especially cecum and colon).

(*e*) Absence of pronounced splenic enlargement. (In subacute and chronic cases the spleen may be smaller than normal.)

Symptoms.—The period of incubation varies from four to eighteen days, usually it is eight to ten days. The symptoms of hog-cholera are not particularly characteristic. In the beginning of an outbreak the first warning given is the finding of a dead hog in the herd. Later another may be found. These losses may continue for a week or so when a number of swine show signs of disease. The symptoms will vary with the outbreak and the individual, *i. e.*, some cases showing the pulmonary, others the intestinal type of the disease, etc., as follows:

(*a*) *Septicemic Form.*—Barring peracute cases which die suddenly without having shown marked symptoms, the hog with acute cholera loses appetite, seems sluggish, weak, and is apt to crawl off in a corner or bury itself in the straw-pile. They usually do not come to feed when called, and if driven out of their seclusion are disinclined to move, hold the back arched, the curl is gone from the tail, and the ears droop. On the surface of the abdomen, the inside of the thighs, and around the ears and neck smaller petechiæ or larger ecchymoses are noted. A quite characteristic symptom is conjunctivitis, the exudate causing the lids to adhere. Vomiting is not uncommon. At first the bowels are constipated, but later diarrhea sets in, the feces being often blood-stained. The temperature is high.

(*b*) *Bowel Form.*—This type of cholera involves not only the intestines but the whole digestive tract. The symptoms either follow those of the septicemic form or may come on more gradually. They consist in a diphtheritic stomatitis and pharyngitis, leading to dysphagia, and if the larynx becomes involved, pronounced dyspnea. Sometimes in cases with prolonged course on palpation tumefactions due to enlarged lymph glands and adhering bowel loops (adhesive peritonitis) may be felt through the abdominal wall. The hogs eat little or nothing and show diarrhea alternating with constipation. The patients move sluggishly, arch the back

and lie down most of the time. Under symptoms of anemia, cachexia and general debility, death follows in two to three weeks. A few cases recover, but usually remain stunted.

(*c*) *Pectoral Form.*—In this form the symptoms of pneumonia and pleuritis predominate. The hogs show high fever (108° F.), and cough frequently. There is often pronounced expiratory dyspnea and nasal discharge. Conjunctivitis is present. In the skin of the ears, neck, sides and lower portions of the body, tail, etc., appear petechiæ and ecchymoses. The bowels are constipated in the beginning but later diarrhea sets in. Death usually results in one to two weeks, although in a few cases the disease becomes chronic leading to emaciation, capricious appetite, cough, dyspnea and fetid diarrhea. Death may follow in one to two months from exhaustion. Occasionally an encapsulement of necrotic lung foci takes place and the patient recovers. (See Swine Plague.)

(*d*) *Mixed Form.*—While in the beginning of outbreaks of hog-cholera the disease may assume one of the above described forms, usually later both the lung and bowel types occur concomitantly in the individual. The symptoms are, therefore, quite complex, but usually one or the other form predominates. In many outbreaks marked skin lesions appear. Besides the intravascular redness noted, vesicles, pustules, ulcers and marked necrosis, especially of the ears and tail (which may drop off), occur. Not infrequently urticaria and loss of the bristles are observed.

Diagnosis.—The diagnosis of cholera *intra vitam* is difficult, especially in the beginning of an outbreak. Usually after carefully weighing the available symptoms an examination postmortem (see this) must be made. In case of doubt a diagnosis can be made only by inoculating healthy young swine with the filtered blood of the suspect. Hog-cholera may be confused with:

(*a*) *Swill Cholera.*—In districts free from hog-cholera a disease appears very like cholera. It is due to feeding swill containing dishwater in which is a quantity of powdered soap. Even the postmortem lesions resemble those of cholera. Careful inquiry into the mode of feeding usually suffices to explain the origin of the disease which promptly disappears

when the food is changed. Hog-cholera, however, commonly breaks out among swine fed kitchen offal from large institutions (penitentiaries, asylums). The food in these cases is not the cause. Large numbers of hogs are bought up indiscriminately to consume this waste, and with them a few which either have cholera in a chronic form or are cholera convalescents ("virus carriers").

(*b*) *Tuberculosis.*—This is a common disease of swine which follow tubercular cattle, or are fed skimmed milk containing tubercle bacilli, or the tubercular offal from slaughter houses. It would be confused only with chronic cholera. The history and necropsy nearly always suffice to differentiate between the two diseases. In cases of doubt the bacteriological examination (staining for tubercle bacilli) may be employed.

(*c*) *Lung Worms* (Strongylus paradoxus).—These thread worms are sometimes found in the bronchi of young pigs. They induce chronic cough and general unthriftiness. Occasionally death is induced through pulmonary edema. A necropsy reveals absence of cholera lesions and the presence of the strongylus in the foamy mucus of the small bronchi.

(*d*) *Swine Plague.*—In the field the differentiation between swine plague and hog-cholera is most difficult and sometimes impossible. Swine plague appears sporadically and tends to disappear suddenly in a herd. Hog-cholera is epizoötic and usually passes through the whole herd. It must be borne in mind, however, that the two diseases may coexist. On necropsy the characteristic changes of swine plague are either fibrinous or necrotic pleuropneumonia, acute swelling and hemorrhages in the lymph glands. In hog-cholera the characteristic lesions are noted in the bowels (diphtheritic enteritis, "button ulcers"). An accurate differentiation can be made only by filtering the blood and injecting susceptible animals. Negative results from filtration experiments, however, do not disprove the existence of hog-cholera as the virus may have been eliminated from the body before the inoculation material was collected. On the other hand the presence of the bipoled organism in the blood and tissues is not absolute proof of the presence of swine plague, as these organisms have been found in the absence thereof.

Prognosis.—The prognosis varies with the outbreak. In some years the disease assumes a mild form leading to a mortality of not over 16 to 20 per cent.; in others it may reach 80 to 100 per cent. The septicemic form is nearly always fatal. The pectoral type leads to the death of all swine showing marked pulmonary symptoms (dyspnea, cyanosis, etc.). While recoveries in the intestinal form are not uncommon, convalescence is often prolonged which greatly affects the value of the hog. The same is true of chronic cases which seldom regain their former thrifty condition. An exception is formed in old breeding boars and sows.

Treatment.—A medicinal treatment is useless in hog-cholera. Once the disease has broken out it is usually recommendable to kill all swine showing intensive symptoms. The carcasses should be rendered harmless by cremation or deep burial. A thorough disinfection should follow, *viz.*, all litter, droppings, etc., should be burned. Feed troughs, sheds, hog houses, etc., disinfected (cresol 3 per cent.). Lime should be scattered abundantly. Hogs which show mild symptoms or are carrying temperature should be inoculated with Dorset serum (obtainable from some state experiment stations, agricultural colleges, livestock sanitary boards, but not from the Bureau of Animal Industry). There are two ways of applying this serum: One known as the "serum alone" method, used in infected hogs, and the "simultaneous" method, serum and virulent blood being injected simultaneously into the medial aspects of the thighs of swine which show no symptoms of the disease and carry no fever.

CATTLE PLAGUE. RINDERPEST.

Definition.—Cattle plague is a very fatal contagious disease of cattle and buffalo of Oriental countries, which is characterized by a severe croupous and diphtheritic inflammation of the mucous membranes especially of the digestive tract. It sometimes involves the outer skin.

Occurrence.—Cattle plague never existed in the United States. While formerly it was generally distributed throughout France, Germany, England, at the present time, except

for the Balkan peninsula, Europe is free from it. The disease is common in Africa and Asia, however, where it is today notoriously prevalent; in Russia and the Philippine Islands. It is rare in sheep and camels which offer considerable resistance to inoculation. Solipeds and carnivora are naturally immune.

Etiology.—Cattle plague is due to an ultramicroscopic virus found in the blood, tissue fluids, exudates and in the secretions and excretions (bile, urine, feces, saliva, tears, sweat) of the body of an infected animal. The virus is not modified by repeated passing through cattle, although passage through sheep and goats is said to weaken it.

Natural Infection.—Takes place by direct contact with infected animals or indirectly through hides, meat or diseased secretions and excretions, attendants, clothing, food, water, and stable utensils. The disease is readily spread along avenues of transportation by animals which have it in a mild form. The mode of infection is usually through the digestive tract. Transmission through the air does not seem probable, as inclosing an infected patient by a fence, ditch or even a tight stall partition suffices to prevent the further spread of the disease. One attack generally lends immunity for an indefinite period.

Necropsy.—The postmortem changes vary somewhat with the severity and duration of the attack. Most conspicuous are the lesions in the mucous membranes which are swollen, reddened, show petechiæ, croupous and diphtheritic areas. The cadaver is usually much emaciated and commonly an accumulation of a mucopurulent, yellow or discolored discharge is found at the natural openings. The mucous membrane of the mouth and pharynx shows petechiæ, croupous deposits and ulcers, most pronounced in the inner surface of the lips, under the tongue, along the gums and cheeks. While the rumen, reticulum, and omasum present no characteristic changes, the mucosa of the abomasum is swollen, highly reddened, and covered with a viscid, dark-colored exudate. In some cases erosions appear. In the duodenum and ileum, Peyer's plaques and the solitary follicles are swollen, dark red in color and infiltrated with purulent exudate. The changes

noted in the large intestines are similar though usually less marked. The intestinal contents are soft and of a reddish-brown color. The spleen is rarely enlarged; it is usually anemic and smaller than normal. The liver is very light-colored, anemic and very friable. The gall-bladder is greatly distended with a light green, watery or dark-colored bile. The respiratory tract shows catarrhal rhinitis with edema of the submucosa; ulcerous erosions and croupous pseudomembranes are common. The lungs are edematous, sometimes contain catarrhal foci, and frequently interstitial emphysema. The blood is dark and coagulates feebly. In some outbreaks a peculiar nodular and pustular eruption is found on the skin.

Symptoms.—The period of incubation is three to nine days. The first symptom is that of fever, the temperature ranging from 104.9° to 107° F., falling somewhat in the next few days and becoming subnormal shortly before death. The affected cattle are very languid, the whole musculature relaxed, the head held down, ears pendent, back arched, and a tendency to knuckle in the hind fetlocks noticeable. The hair coat is roughened and involuntary twitching of the muscles of the head, shoulders, and flanks is frequent. In some patients there is a pronounced chill lasting several minutes. A common early symptom is a frequent, painful cough which disappears as the severe gastro-enteritis develops. The patients are usually dyspneic. From the nose flows a mucopurulent (rarely bloody) discharge mixed with saliva, which excoriates the skin of the nostrils.

The conjunctiva is either anemic or, on the contrary, it may be congested, the lids closed and swollen, tears flowing down the cheeks. Later there develops a purulent discharge which dries to form crusts about the eyelids. Keratitis may be present. In the mouth the gums are swollen and dark blue or dirty red in color. On the inner surface of the lips irregular, shallow erosions are noted. Commonly the mucous membrane of the mandible and of the dental pad becomes covered with yellow granules as if sprinkled with corn meal. These areas later become confluent and slough, leaving behind readily bleeding ulcers. In swine outbreaks the mouth lesions are little developed.

While in the initial stage the bowels are constipated, after the second day a profuse diarrhea sets in, the liquid feces are mixed with blood and fibrinous clots. The patients lose flesh rapidly. In some outbreaks there appear in the skin of the udder or scrotum, inner surface of the thighs, around the vulva and nose a nodular and pustular exanthema. In milk cows lactation stops, the udder becoming relaxed and smaller. The pulse varies from 50 to 100. From the vulva in some cases there is a dirty, mucopurulent discharge from the inflamed mucosa.

Diagnosis.—The recognition of the first cases of cattle plague is difficult. The disease is characterized by its sudden appearance, the rapid development of the symptoms and the peculiar lesions found on the visible mucous membranes. Of great importance is a clear history of the origin of the outbreak and the opportunity presented for the disease to be introduced on the premises. Cattle plague may be confused with the following:

Malignant Head Catarrh.—Here while the mucous membranes of the head (eyes, nose) are severely involved, the digestive and genital tracts are mildly involved. Keratitis is more pronounced in this disease. Further, malignant head catarrh occurs sporadically and is not contagious.

Foot-and-mouth Disease.—Would only be confused with cattle plague when the mouth erosions following the rupture of the vesicle were deep and covered with a pesudomembrane and gastro-enteritis was present. Foot-and-mouth disease, however, is characterized by vesicle formation, affects the feet and usually does not involve the eyes and nose. Its spread is further much more rapid than the cattle plague.

Course.—The course is usually five to seven days, ending in death. Toward the end of an outbreak a few cases may recover.

Prognosis.—The prognosis is bad, 80 to 90 per cent. of the cases prove fatal. In the gray cattle of the Russian steppes the mortality is not over 50 per cent.

Treatment.—Medicinal treatment is of no value. Good results are recorded from the use of anti-rinderpest serum coupled with a strict sanitation.

Prophylaxis.—In civilized countries cattle plague has been stamped out by the compulsory slaughter of all animals affected and the enforcement of strict quarantine measures against infected districts. As the disease is not transmitted any distance through the air, its eradication is not difficult when coöperation on the part of the cattle owners can be obtained. The period of quarantine should extend ten days after the last patient has died or been killed. Carcasses should be disposed of as in anthrax.

Protective Inoculation.—As one attack of cattle plague confers lasting immunity, efforts have been made to protect against it by inoculating healthy cattle with various secretions (bile, nasal discharge, tears), blood serum and blood from affected animals. The inoculated animals, however, can spread the disease which has led to considerable losses where the method of vaccination has been practised. In badly infected districts, like South Africa, however, fair results have been obtained. (For details see larger works.)

AFRICAN HORSE-SICKNESS. PESTIS EQUORUM.

Definition.—African horse-sickness is an infectious disease of solipeds due to an ultramicroscopic virus. It is characterized by extensive edematous swellings under the skin and hemorrhages in the internal organs.

Occurrence.—The disease is indigenous to South Africa where it occurs epizoötically and leads to enormous losses among horses and mules. It therefore attains economic importance.

Etiology.—The cause is a filtrable virus. The blood of a horse affected with the disease will readily transmit it to susceptible animals no matter what way the inoculation is attempted. The manure is thought not to be infectious. It does not seem possible to infect animals other than horses, asses and mules, although Angora goats have been artificially inoculated.

Natural Infection.—The disease appears during the warm, rainy months of January, February and March. It disappears after the first frost. Horses on pasture during these

months are most often affected. The night season is more dangerous than the daytime in this regard. Probably the infection is carried by insects (mosquitoes, anopheles, stegomyia). Keeping a fire smudge near horses on pasture at night or protection with mosquito-netting is said to prevent the infection.

Necropsy.—Gelatinous infiltration of the subcutaneous and intramuscular connective tissue about the eyes and throat is commonly noted. Evidence of gastro-intestinal catarrh or in some cases hemorrhage and ulceration of the intestine is a common finding. The serous membranes show petechiæ and not infrequently fibrinous exudation. The lungs are edematous, the spleen is normal and the liver and kidneys congested or inflamed. The superficial lymph glands are enlarged.

Symptoms.—The period of incubation is about one week. Four forms of the disease are described from a clinical standpoint: The peracute, acute, subacute, and chronic. The most important are the acute and subacute forms.

(*a*) *Acute Form.*—This form, known in Africa as Dunkopziekte, begins with a high fever (104° to 107.6° F.), dyspnea, pulmonary edema, and heart paralysis. Death usually occurs in one to two weeks.

(*b*) *Subacute Form.*—The subacute form, known as Dikkopziekte, takes a longer and milder course. Characteristic of this form is a marked swelling of the head, principally about the eyes; swelling and prolapses of the tongue which is markedly cyanotic. From this symptom the name "blaw tong" (blue tongue) is given to the disease. The patient shows marked muscular weakness. In this form the majority of the cases recover.

The peracute or apoplectic form produces sudden death; the chronic form assumes an atypical course lasting for weeks and resembling infectious anemia.

Treatment.—A medicinal treatment has to date not proved satisfactory. Internally large doses of creolin have been recommended.

Prevention.—Prevention consists in keeping the horses off infected or suspected pastures during the night season or driving them to higher altitudes during the dangerous months

of the year. Fairly good results have been obtained by preventive inoculation to produce immunity. As in hog-cholera a combined virus and serum vaccination is practised.

Heartwater.—Definition.—Heartwater is a disease which is not identical with horse-sickness. It is a contagio-infectious hydropericardium of cattle, sheep and goats of the Transvaal. The disease is carried by ticks (Amblyomma hebræum).

CHAPTER II.

ACUTE EXANTHEMATOUS INFECTIOUS DISEASES.

VARIOLA. POX.

Definition.—Variola is an acute contagious disease characterized by a typical cutaneous eruption which passes through the stages of papule, vesicle, pustule, and crust. In all animals except sheep this eruption is usually local.

Occurrence.—While cowpox is a very common disease in the United States, horsepox is rare and sheep-pox has never been reported. In Asia, Africa and in parts of Europe sheep-pox becomes epizoötic causing great losses.

Etiological Relationship.—The etiological relationship of the pox of the different animals and of man to one another is not yet clear. There is undoubtedly a close relationship between cowpox (variola vaccina) and smallpox of man (variola humana), although if man be inoculated with cowpox, the eruption resulting usually remains local. Further, cattle are not readily inoculated with smallpox. However, as is well known, if man be inoculated (vaccinated) with cowpox, he is protected against smallpox. Sheep-pox is transmitted with difficulty to either man or cattle, natural infection does not occur and no protection against cowpox is afforded cattle inoculated with sheep-pox virus. Sheep-pox, therefore, is probably a specific disease of sheep which does not spread to other animals including goats. Goatpox affects neither man nor sheep and seems to be a specific caprine disease. Horsepox is a local disease of the skin in the region of the fetlocks. It is probably closely related to, if not identical, with cowpox, being transmitted by recently vaccinated horseshoers to horses while being shod. In the light of our present knowledge smallpox (variola humana), sheep-pox, (variola ovina) and goatpox (variola caprina) are independent diseases, while

the pox of other animals (ox, horse, swine) are merely modifications of these.

It is extremely probable that in the beginning all forms of variola had a common origin. By continued passage for several generations through different species of animals, however, the virus has become so adapted to the specific species in which it has propagated, that transmissibility to other species of animals no longer occurs through natural infection and is difficult to bring about by inoculation.

Etiology.—From recent experiments it has been determined that filtering the virus of the variola of different animals and man does not decrease its virulency unless very fine filters are employed. It is probable, therefore, that the cause is an ultramicroscopic organism.

The virus is contained in the papules, vesicles, and crusts. It is doubtful whether the expirium or blood of the varioloid patient is infectious. The secretions and excretions are infective when contaminated with the contents of the vesicles.

Sheep-pox (*Variola Ovina*).—**Definition.**—A specific infectious disease of sheep characterized by a cutaneous eruption which passes through the stages of papule, vesicle, pustule, and crust.

Occurrence.—Sheep-pox is not known in the United States. While its home is in Asia, outbreaks have occurred in Europe and Africa. When the disease becomes epizoötic it causes considerable economic losses from death and the damage it causes the wool.

Etiology.—The cause is probably an ultramicroscopic organism.

Natural Infection.—The virus enters through the respiratory tract and is carried from the lung alveoli by the blood to the skin and mucous membranes. Intratracheal injections of the contents of the vesicles (lymph) readily produce the disease, while feeding experiments gave negative results provided inhalation was excluded. Impregnating the air a distance from susceptible sheep with a spray of pox lymph or the dried crusts gave positive results.

A flock of sheep is usually infected by direct contact with a variolous sheep, one convalescent from the disease, and in

countries where protective inoculation is practised, from a vaccinated lamb. In rarer instances the contagion is transmitted through intermediary agents (pelts, wool, food, bedding, clothing of attendants, etc.). During the crust and desquamative stages of the disease the affected sheep are especially dangerous to susceptible ones. As the wool will retain the desquamated scales for several weeks, the disease may be spread by a sheep fully recovered from variola, the infection being carried in the fleece. Lambs are much more susceptible than adult sheep.

Intra-uterine infection, the lamb being born with the disease, is not rare. In other instances the lamb born of a variolous ewe, comes into the world healthy but is an immune. The period of incubation is about one week except in cold weather when it may be longer.

Symptoms.—Preceding the eruption there is commonly fever (105° to 107° F.), languor, catarrhal conjunctivitis and rhinitis, loss of appetite and suppressed rumination. The patients seem stiff and extremely sensitive over the back and loins. In one to two days on different parts of the skin usually not covered by wool (eyes, inner surface of the thighs, chest, abdomen, under the tail) small, dark red colored, flea-bite-like spots (papules) appear which in three to five days develop into lentil-sized blisters (vesicles) filled with a clear fluid. The vesicles often present a depression in the center (umbilicated) while some are simply flattened on top. About the seventh day after the appearance of the eruption the contents of the vesicles become turbid, containing pus (pustule). Usually three days later the pustules erupt and dry, forming in the edematously infiltrated, reddened skin about them, firm, gray scabs which later become brown in color. The scabs in three to five days become detached, leaving a pit. All the vesicles do not appear at the same time, but continue to form at intervals, materially prolonging the course of the exanthema in the individual which may present all the successive stages of the eruption at the same time. The temperature which is high during the prodromal stage, falls when the eruption develops but rises again when the pustules form (secondary infection). When the pustules

erupt the temperature becomes normal. The odor of sheep-pox, which is peculiarly sweetish and nauseating, is quite distinctive. It is most marked in the early stages and often of diagnostic value.

The eruption also appears in the mucous membranes, producing when in the mouth, ptyalism; throat, dysphagia; bowel, diarrhea, and in the bronchi, cough.

The following modifications in sheep-pox are observed:

(*a*) *Abortive, Mild Type.*—Either no eruption appears in the skin or only a very few isolated lesions as in "varioloid" of man. The general disturbance is not marked and the course benign.

(*b*) *No Vesicles Form,* the reddish papules in a few days becoming detached and disappearing.

(*c*) *Confluent Pox.*—This is a more severe form, the pustules occurring very close together and finally blending. It is most marked on the head, the skin of which becomes swollen and may slough; secondary pus infections take place leading to pyemia and septicemia. There may be severe pharyngitis and conjunctivitis followed by keratitis and blindness.

(*d*) *Hemorrhagic Pox.*—Between the papules dark red blood spots appear which involve the subcutis. Nasal hemorrhage, hematuria, bloody diarrhea and internal hemorrhage are not infrequent. This form (similar to "black smallpox" of man) is very fatal.

(*e*) *Gangrenous Pox.*—Probably due to a secondary infection (necrosis bacillus?) is not a special form but may attend otherwise usual outbreaks. Portions of the skin, and even the underlying muscle in the region of the eruption become necrotic and slough.

Complications.—(*a*) Dermatitis may develop from rubbing and scratching the vesicles especially in the region of the lips and nostrils.

(*b*) Blindness from ulcerative keratitis and later suppurative panophthalmia.

(*c*) Bronchitis and catarrhal pneumonia.

(*d*) Gastro-intestinal catarrh.

(*e*) Suppurative arthritis of the phalangeal joints, shedding of the claws and general pyemia.

Diagnosis.—The peculiar character of the exanthema and the typical course usually make the diagnosis easy. It would hardly be confused with scab or pustulous eczema as neither of these diseases present marked prodromes and in neither develop well defined, large vesicles.

Course.—The course is acute usually requiring about three to four weeks to pass through the different stages. Through complications, cold weather and in run-down sheep, the course may be considerably prolonged. Sheep-pox may remain in a flock for several months.

The mortality varies with the form the disease assumes. While in the milder forms it may not exceed 4 per cent., the more malignant types claim fully 50 per cent. as victims.

Treatment and Prophylaxis.—Usually a dietetic treatment is all that is necessary (grass, roots, gruels). In mild weather the sheep should be turned out to pasture. Severely infected animals should be slaughtered. The medicinal treatment (disinfectants for eyes, salts for bowels) is expectative and symptomatic. Sporadic outbreaks in uninfected districts are best eradicated by slaughter and proper disposition of the carcasses.

In permanently infected countries "ovination" or protective inoculation with sheep-pox virus has been practised with varying results. The practise is dangerous in districts where the disorder occurs only sporadically as the inoculated sheep can spread the disease.

Cowpox (*Variola Vaccina*).—**Definition.**—Cowpox is a benign, eruptive disease of the ox which occurs enzoötically and is characterized by a vesiculopustular exanthema occurring most commonly on the udders of milch cows.

Occurrence.—Cowpox is widely distributed. It is a frequent disease among dairy cows, to which it is spread by recently vaccinated milkers, attendants, etc.

Etiology.—The cause is evidently an ultramicroscopic organism which passes through coarse but not fine filters.

Natural Infection.—Cowpox is most commonly first introduced into a healthy herd by recently vaccinated persons, especially milkers who convey the virus directly to the udder through the act of milking. It is also usually spread from

cow to cow in the same manner. The disease may also be propagated by contaminated litter, forage, food, etc., which explains its occurrence in heifers, steers, and bulls.

Symptoms.—The period of incubation is four to seven days. The prodromal symptoms are usually so mild as to be overlooked. Occasionally symptoms of general disturbance (fever, loss of appetite, languor) precede the eruption. The first local symptom noted is usually a sensitiveness of the teats during milking. In two to three days on or at the base of the teat and on the udder appear pea-sized papules which in forty-eight hours form vesicles the size of a bean. The vesicles contain a clear fluid and are often pearl-like in color. On the udder they are usually round and on the teats oval in form. They are noted best on the udder, near the base of the teat where they are less liable to be broken by the milker's hands. The vesicles are usually surrounded by a red zone. When intact they are frequently umbilicated. In eight to fourteen days pustules appear that erupt and dry to form crusts which slough leaving shallow pits. As a rule only a few vesicles occur, usually not over a dozen and occasionally only one or two can be found. The eruption does not take place simultaneously, several days may elapse between the formation of individual or groups of vesicles. Those which form later are generally smaller than the earlier ones.

As the vesicles, especially those which form on the teat, are crushed during milking, secondary changes due to traumatism and extraneous infection are usual. From the frequent irritation of milking there form ulcers with eventually indurated borders which heal only after several weeks.

Parenchymatous mastitis is a common complication, especially where milk tubes are employed to draw off the milk from the affected teats. In males the disease is very rare and is said to involve the scrotal region.

A generalization of the eruption is very uncommon. In the few cases recorded, the vesicles appeared in the inner side of the thigh, on the croup, body, chest, neck and muzzle.

Diagnosis.—The typical exanthema, its spread from cow to cow and to the hands of the milkers, the absence of general symptoms and the benign course characterize the disease. In

isolated cases where no vesicles can be found and the only lesion apparent a teat ulcer, the diagnosis is impossible.

Course.—The disease usually lasts several weeks in a herd, depending upon the number of susceptible animals and the precautions taken to prevent its spread.

Prognosis.—Good. The disease is benign and does not cause death. Economic loss is occasioned through the diminution and pollution of the milk, the persistency of the teat ulcers and the cases of mastitis which develop during the course of an outbreak.

Treatment.—No internal treatment is necessary. Washing the teats and udder after each milking with a 1 per cent. solution of sodium hyposulphite generally suffices. Sterile milk-tubes may be used when the teats are sore. The ulcers heal readily in ten to fourteen days provided they are not irritated during milking.

Protective Inoculation.—Vaccinating cattle with calf lymph (vaccine points) intended for human beings has been practised in Europe with apparently good results.

Horsepox.—Definition.—Horsepox is a local vesiculopustular eruption which occurs in the region of the fetlocks.

Etiology.—It is probably cowpox transmitted to horses by recently vaccinated horseshoers.

Symptoms.—It is most frequent in young horses. The region of the fetlocks becomes edematous and sensitive. In a few cases there may be mild general symptoms (fever) which are usually not noticed. In one to two days, on the posterior surface of the fetlocks, there develop pea to bean sized vesicles which soon burst and form crusts. Occurrence on other portions of the body is rare, although cases are recorded where the vesicles appeared on the head, about the mouth and nostrils and, exceptionally, the buccal mucous membrane was invaded. On the limbs a temporary change in the color of the hair, which becomes lighter, has been noted. The course is benign.

Swinepox.—Occurrence.—Swinepox is a rare disease and has not been reported in the United States.

Etiology.—It is probably transmitted to swine from man and cattle. Experiments to transmit sheep-pox to swine have resulted only in a local infection.

Symptoms.—Swinepox affects usually young pigs. It is characterized by a pox eruption involving most of the body. The initial symptoms are those of fever, languor, loss of appetite, labored gait, and catarrhal symptoms such as conjunctivitis. The eruption usually occurs about the snout, eyelids, inner surface of the thighs and over the abdomen. The parts of the skin well covered with bristles are not usually involved. The eruption at first consists of small, red spots which become papules and in about three days pea-sized vesicles which rapidly turn to pustules to be followed by dark-brown scabs which are thrown off to leave pits.

Diagnosis.—The diagnosis depends upon the rapid development of the typical pox lesions in the skin. In doubtful cases inoculations may be made. Swinepox should not be confused with eczema or the skin eruption which sometimes accompanies hog-cholera. From squamous eczema ("soot" of young pigs) it is readily distinguished by the brown or black crusts which form in this disease.

Prophylaxis.—The prophylaxis consists in protective vaccination as in sheep-pox.

FOOT-AND-MOUTH DISEASE.

Definition.—Foot-and-mouth disease is a very contagious disorder of cloven-hoofed animals, due to an ultramicroscopic organism and characterized by the formation of vesicles which occur on the mucous membranes and skin. The mouth, the interdigital space and the teats and udder are most often affected.

Occurrence.—The disease has been an occasional visitor to the United States. In 1870 foot-and-mouth disease spread from Canada into New England and New York; in 1884 there was a limited outbreak in Portland, Maine; in 1902 again in New England, in the states of Connecticut, Rhode Island, Massachusetts and Vermont; in 1908 it was introduced through contaminated smallpox vaccine into New York, Pennsylvania, Michigan and Maryland; in 1914 the most widespread outbreak in the history of the country occurred. Within three months the disease spread from the

Chicago stockyards to Michigan and Ohio and ultimately infected twenty-one states and the District of Columbia. The origin of this outbreak is in dispute but it probably came from contaminated anti-hog-cholera serum. In Europe, Asia and Africa and in South America foot-and-mouth disease is prevalent. While not an especially fatal disease it occasions enormous losses due to its rapid spread, the damage it causes the milk industry, the losses entailed through quarantine and the sequels (loss of claws, mastitis, etc.) which follow in the wake of an outbreak. The damage done the cattle industry amounts to an average of $20 per head for each animal affected.

Etiology.—The cause is evidently an ultramicroscopic virus which passes through coarse but not the finest bacterial filters. The virus is present in the vesicles and is found in the blood only in the early part of the fever stage. The saliva, tears, milk and nasal discharge are infectious from contamination with the contents of the vesicles. As the disease progresses the virulency of the virus decreases.

Natural Infection.—The virus of foot-and-mouth disease is taken up by the digestive tract with food, water, bedding, litter, etc., which have become contaminated principally by the saliva of affected animals. Such intermediary agents as stable utensils, mangers, watering troughs, clothing and the hands of attendants, etc., may also harbor the virus. Railway cars, stockyards, cattle pens, manure, hides, wool, milk, veterinarians, butchers, cattle dealers, herders, etc., are also carriers of the contagion. Hay, straw, feed and the like imported from infected districts often spread the disease. In 1908, as noted, foot-and-mouth disease was introduced into the United States by some calves used for the propagation of vaccine virus, which had been inoculated with contaminated lymph imported from Japan. It is very probable that recovered animals ("germ carriers") may harbor the virus for an indefinite period which accounts for sporadic outbreaks of the disease in uninfected districts.

While cattle are most predisposed, the disorder also attacks sheep, swine, goats and buffalo. It is rare in horses, dogs and cats.

One attack produces immunity for only a short period (usually not over one year). In certain individuals no immunity is conferred, the animal suffering within a few months repeated attacks. Calves born of cows attacked in advanced pregnancy are sometimes (not always) highly resistant to either natural infection or artificial inoculation.

Symptoms.—The period of incubation is two to seven days, although it may be longer.

The prodromal symptoms are those of fever, the temperature reaching 106° F., lasting one or two days, and falling to normal as soon as the vesicles appear. Unless complications arise from secondary infection, no further rise in temperature occurs during the course of the disease.

With the fever there is a period of loss of appetite, suppressed rumination and languor. The mouths of the patients become sore, causing them to masticate slowly and in an interrupted fashion. The mouth is usually held closed, saliva hanging from the commissures in long strands. When opened a peculiar smacking sound is made. Where a number of affected cattle are housed together the noise produced is marked.

On examining the mouth one to two days after the beginning of the attack, the mucous membrane, especially of the lips, gums, dental pad and tongue, shows a vesicular eruption, the individual vesicles varying in size from a pea to a walnut. The large blisters rupture in about one day, leaving behind an excoriated surface of a brown-red color, which is often covered with a gray deposit. The smaller vesicles persist for two to three days. On the back of the tongue one to three walnut-sized vesicles are often noted. As the organ is extremely sore and therefore little moved, the blisters remain intact for two to three days. The contents of the vesicles are clear or yellowish. Where they erupt a very sensitive, highly reddened, shallow erosion is left which becomes covered with new epithelium in two to three days. When healing is well under way the eroded area appears as a brown-yellow spot which eventually disappears. As soon as the erosions are sufficiently covered with epithelium, the emaciated patient begins to eat.

In some cases the muzzle (snout of swine), base of the

horns (very rare), nasal mucous membrane, conjunctiva, pharynx (dysphagia, cough, regurgitation) and even the cornea are similarly involved.

The foot lesions induce lameness with knuckling of the fetlock of the limb attacked. If two or more feet are affected the patient lies down most of the time and is made to arise with difficulty. The coronet is hot and swollen, especially in front and between the bulbs of the heels. In some cases the swelling extends up the leg to the middle of the canon. On the second to third day of the attack pea- to bean-sized vesicles appear in the swollen area. The vesicles rupture very early and leave behind sores covered with a tough, brown scab. Healing usually requires one to two weeks. In sèvere cases, from secondary infection, shedding of the claws results.

In swine and sheep only the feet may be attacked, no mouth lesions being apparent.

In cows the skin of the teats and udder is frequently affected, most often the former. The vesicles are from the size of a pea to a hazel nut and are generally ruptured during milking. The teats are swollen, sometimes phlegmonous (secondary infection) and extremely sensitive. Later the sores become covered with scabs and heal.

Catarrhal mastitis frequently attends the exanthema leading to changes in the milk, which becomes colostral, has an acid reaction, coagulates readily and is difficult to make into butter or cheese.

Complications and Sequels.—The most serious complications are phlegmons of the digits which lead to suppurative inflammation of the tendon sheaths, tendons, joints and horn matrix inducing severe general disturbance (fever, high pulse), inability to stand, decubital gangrene, septicemia and death in one to two weeks.

Septic infection of the udder (parenchymatous mastitis) is not an uncommon complication which may cause the loss of a quarter or a half of the organ.

Gastro-enteritis is a fatal complication in calves leading to death in two to three days.

Diagnosis.—In typical cases, during the vesicular stage of the eruption, the diagnosis is not difficult. The presence of vesicles, the foot lesions, the rapid spread of the contagion and the ease with which it may be transmitted artificially characterize the disorder.

During the early stages (before the eruption) and at the end of a sporadic outbreak (vesicles healed or only secondary changes present) the diagnosis can be extremely difficult.

Foot-and-mouth disease may be confused with various forms of stomatitis in cattle, none of which are contagious but some of which are attended with vesicle formation. The most important are the following:

(*a*) *Traumatic Stomatitis* ("tooth cuts") presents lesions on the bars, lips and dental pad. There is no vesicle formation and the wounds have sharp borders and are deep.

(*b*) *Mycotic Stomatitis of Cattle,* a non-contagious foot and mouth affection widespread in the United States. Vesicles rarely appear and are never well developed. The mouth lesions are more ulcerous in character than in foot-and-mouth disease and are more apt to involve the deeper structures. There is more swelling of the limbs but no vesicles occur at the coronets. A peculiar reddish-brown coloration of the muzzle, udder and teats is considered characteristic by some authorities. The disease cannot be transmitted by inoculation and does not affect sheep or swine. Often only a few animals in a herd are attacked.

(*c*) *Ergotism.*—Ergotism produces gangrene of the distal portions of the extremities (feet, ears, tail), the necrotic parts sloughing. Blisters are not common and when present not well marked. This poisoning is not contagious and cannot be transmitted artificially from animal to animal. It occurs only among cattle which have eaten ergot-of-rye.

(*d*) *Necrotic Stomatitis.*—Nearly always seen in calves ("calf diphtheria") and pigs ("sore mouth"). There is no vesicle formation but a necrosis of the mucous membrane, yellow-gray patches developing in the mouth (cheeks, tongue).

(*e*) *Foot-rot of Sheep.*—A contagious disease of the interdigital space which may later induce suppurative pododerma-

titis, tendovaginitis, open joint, etc. There are no vesicles and the disease spreads slowly through a flock.

(*f*) *Foul-in-the-feet of Cattle,* due to filthy stables and barnyards, does not affect the mouth; there are no vesicles and no contagion. A malignant type of foul-in-the-feet due to the necrosis bacillus and appearing in cows soon after parturition or in advanced pregnancy, assumes the form of a necrosis of the interdigital space which may involve the deeper structures (matrix, tendon sheaths, tendons, joints). It is attended by fever when secondary infection is present. The mouth is not involved.

(*g*) *Infectious Vesicular Stomatitis.*—This disease, while it occasionally attacks cattle, is most often seen in horses. It does not seem to affect sheep and swine. It rarely attacks the feet. Its spread through a herd is not so rapid and does not involve so high a percentage of animals as does foot-and-mouth disease. The mouth eruption in infectious vesicular stomatitis is continuous or progressive, different stages coexisting, which is not true of foot-and-mouth disease. Differing from foot-and-mouth disease, straining the vesicular fluid through a Berkefeld filter causes it to lose its virulency.

The indications which point to foot-and-mouth disease in a recently recovered animal are: Ptyalism (usually profuse); yellow cicatrices or areas on gums and dental pad; small, red spots and erosions in the gums and borders of the muzzle. These traces are said to persist for several weeks.

Course.—In most outbreaks the course is benign. Individual vesicles usually heal in five to six days, but as they do not all erupt at the same time, the duration is often extended two to three weeks. The mouth lesions heal more rapidly than do those of the feet. As all animals are not infected simultaneously, an outbreak will last in a given barn one to two months.

In calves (under two months) the course is more rapid and fatal (toxemia, septicemia, pyemia, gangrenous pneumonia, heart muscle degeneration) leading to death in three to four days.

From resulting foot troubles (panaritium, suppurative tendovaginitis, open joint, interdigital ulceration), loss of

flesh and milk (udder complications) the course is not only prolonged but the patient's economic value may become permanently reduced.

Prognosis.—The disease is benign except in very young animals, when it assumes a malignant form and when complications due to secondary infection occur (loss of claws, decubitus, septicemia, etc.). It is rare for the mortality to exceed 1 per cent. in the benign type. It may exceed 50 per cent., however, in the rarer, malignant type.

Treatment.—In countries, like the United States, which are not permanently infected, no treatment should be tolerated. The extreme contagiousness of the disease and its rapid spread justify the most radical methods of eradication. These consist in the immediate slaughter of all affected animals, the proper disposition of the carcasses (deep burial, cremation) and a thorough disinfection of the premises.

In countries where foot-and-mouth disease is widespread and radical methods of stamping it out cannot be employed, the following indications in the way of therapy are followed:

The cattle should be fed only soft, sloppy food (bran mashes, root pulp, grass) and allowed plenty of water. The mouths may be irrigated twice daily with some non-toxic disinfectant (pyoctanin 1 to 1000). The stable floors should be kept clean and dry and the feet treated with disinfectants (may be stood in a shallow bath of 2 per cent. creolin solution). To the teats and udder boroglycerin, camphor ointment, etc., are useful. Mastitis should be treated as such. Complications (shedding of claws, tendovaginitis, etc.) are handled according to surgical principles.

The milk should not be used during the outbreak. In a raw state it is fatal to sucklings and when sterilized it is not wholesome.

General symptoms (heart weakness, high fever) are met as they occur (oil of camphor subcutaneously, digitalis, alcohol, acetanilid).

As foot-and-mouth disease is a self-limiting disease which usually takes a typical course, in uncomplicated cases not much medicinal treatment is needed.

Formerly, it was common practice, when foot-and-mouth

disease appeared on a premises, to inoculate all cattle still healthy by rubbing saliva from an infected animal into the mucous membrane of the mouth. This method infected the whole herd simultaneously and lessened the virulence of the attack. It is seldom permitted nowadays, although it has much to recommend it.

Protective and Therapeutic Inoculations.—Various methods (for which see larger works) of preventive vaccination have been tried with as yet indifferent success.

INFECTIOUS VESICULAR STOMATITIS.

Definition.—Infectious vesicular stomatitis is a communicable infectious disease of horses, mules and cattle, characterized by the formation of vesicles which appear upon the mucous membrane of the mouth, in horses particularly the tongue.

Occurrence.—The disease has long been recognized in Europe and South Africa and occasional sporadic outbreaks have occurred in the United States. It first attracted attention in this country during the Great War when large numbers of horses and mules were collected for shipment to the allied armies. At remount depots the best opportunity was afforded to spread the disease. From these centers it spread rapidly throughout the west until in the states of Nebraska, South Dakota, Colorado and Wyoming, it became alarmingly prevalent. Horses and mules are most often attacked from which the disease may spread to cattle. Natural infection has not been observed among hogs and sheep. While infectious vesicular stomatitis is a benign disease, unless complications occur, it occasions economic loss in the animal attacked from emaciation, reduction in milk supply in cows, and the incapacitation of the patient. Furthermore, especially in cattle, its resemblance to foot-and-mouth disease is apt to lead to error in diagnosis, causing genuine cases of the latter to be considered infectious vesicular stomatitis. This might lead to serious consequences in that an unrecognized focus of foot-and-mouth infection is

overlooked and the disease thus permitted to become enzoötic or even epizoötic.

Etiology.—The cause of infectious vesicular stomatitis is unknown. During the stage of vesicular eruption it seems most transmissible. Fresh vesicular fluid strained through a Berkefeld filter loses its infectious properties. The virus therefore is evidently not filtrable. Susceptible horses may become infected by inoculation with the fluid contained in the vesicles of diseased animals. Frequently, however, inoculation experiments will fail to develop the infection. In one experiment made by the Bureau of Animal Industry the disease was successfully transmitted to only three out of nine animals inoculated. This experience has led some observers to believe the disease non-communicable. The virus is probably short-lived and is transmitted only by close contact. The disease does not seem to spread from one corral to another, provided a double fence separates the two. A field which had contained cases of infectious vesicular stomatitis did not infect horses and cattle placed therein three weeks later. Susceptible animals did not become infected when placed in a stable which sixteen days before had housed active cases.

Transmission through intermediary agents does not seem common. Direct contact seems necessary for spread from animal to animal. Inoculation experiments on rabbits, guinea pigs, rats and mice, resulted negatively.

As far as is known one attack produces immunity for at least three months. Blood serum, however, from immune animals failed to produce resistance to the disease when injected into susceptible animals. The milk of infected cattle has been fed to hogs without ill effect.

Symptoms.—The period of incubation is usually two to five days. A minimum period of thirty-six hours and a maximum of nine days have been recognized. The initial symptoms consist in the appearance of red areas in the mucous membrane of the mouth especially the tongue. There rapidly develop vesicles of grayish-red color, slightly elevated, filled with a clear or yellowish fluid and varying in size from a ten-cent piece to a silver dollar. Smaller vesicles by coalescence

form larger ones. The vesicles rupture in a very short time and therefore are often not found even in the early stages of the disease. When the vesicle ruptures a red erosion persists partially covered with grayish-white fragments of mucous membrane. The erosions may become confluent forming raw areas, particularly on the dorsum of the tongue, as large as the palm of the hand, or the whole upper surface of the tongue may be involved. Besides the tongue the tooth surface of the lips, commissures of the mouth, and gums may be attacked. In cattle the principal lesions occur on the dental pad, lips, gums, and sometimes extending to the muzzle. In fresh milch cows vesicles occasionally appear on the teats probably due to infected calves sucking them. Foot lesions have not been observed in these cases. In horses the lips are swollen and itchy causing the animal to rub them.

With the eruption of the vesicles there is moderate fever which rapidly subsides. As in all marked stomatites, a profuse flow of saliva occurs which hangs from the mouth in thin strands or may be churned into foam. Sometimes the first symptom noted of the disease is salivation. The patients refuse food and in cattle the jaws are champed producing a smacking noise as in foot-and-mouth disease. In horses the patient frequently grits its teeth. Healing usually occurs in from eight to fifteen days.

Diagnosis.—Infectious vesicular stomatitis in cattle may be confused with the following:

(*a*) *Foot-and-mouth Disease.*—To distinguish accurately between foot-and-mouth disease and infectious vesicular stomatitis requires close observation for several days and inoculation tests. From the field standpoint the following differences are noted: Horses are rarely infected with foot-and-mouth disease. In the United States during the epizoötics of this disease no horses were attacked. While foot-and-mouth disease spreads to swine on the same pasture infectious vesicular stomatitis does not. Sheep take foot-and-mouth infection readily but do not seem susceptible to infectious vesicular stomatitis. In infectious vesicular stomatitis the lesions appear to be continuous or progressive,

different stages coexisting, which is not true of foot-and-mouth disease. Furthermore, the feet are rarely attacked in infectious vesicular stomatitis. Calves rarely take the disease other than in a mild form while in foot-and-mouth disease they are often seriously and fatally attacked. Infectious vesicular stomatitis does not spread through a herd and community as rapidly as foot-and-mouth disease nor is the percentage of animals attacked in a given herd as high. Filtering the vesicular contents in foot-and-mouth disease does not reduce their virulency while in infectious vesicular stomatitis it does. The passage of the virus of infectious vesicular stomatitis through a series of calves attenuates its virulency; in foot-and-mouth disease the opposite is true.

(*b*) *Mycotic Stomatitis.*—This disease occurs, usually following a drought, among cattle on pasture in the late summer or early fall. It does not run a regular course, and rarely infects more than 10 to 15 per cent. of the herd. It tends to produce croupous membranes rather than a vesicular eruption in the mucous membrane of the mouth, and attacks the feet. It does not spread to horses and gives negative results when inoculated into calves.

(*c*) *Necrotic Stomatitis.*—This disease affects pigs and sheep as well as cattle and calves and is much more infectious than infectious vesicular stomatitis. It leads to a necrosis of the mucous membrane with a formation of yellowish, cheesy patches involving the mucous membrane particularly of the cheeks and tongue.

(*d*) *Contagious Pustular Stomatitis.*—This disease is confined to horses and is characterized by the formation of nodules which turn to pustules and ulcers involving usually only the tip and borders of the tongue, lips, gums and cheeks.

Treatment.—The treatment of infectious vesicular stomatitis consists in separating the diseased from the healthy and completely isolating the former. Gentle animals may be treated medicinally by placing on the tongue twice daily borax ($\frac{1}{2}$ tablespoonful) or syringing out the mouth with permanganate of potash (1 per cent.). Animals which cannot be handled may be given borax (2 tablespoonfuls), potassium chlorate (1 tablespoonful) dissolved in a bucket of water

which should be kept accessible to the patient. Only soft foods should be fed for the first few days. Outbreaks handled in this manner rapidly disappear.

CONTAGIOUS STOMATITIS OF THE HORSE.

Definition.—Contagious pustulous stomatitis is an acute, benign, infectious disease of the horse characterized by its marked contagiousness and the appearance of pustules in the mucous membrane of the mouth. Sometimes the nasal mucosa and the skin of the lips are involved.

Occurrence.—The disease is widely distributed, appearing enzoötically, chiefly among colts or young horses herded together in barns or on pastures. While the disease is usually benign, the patients lose flesh and older horses are often unable to work during the attack. Occasionally foreign body pneumonia may complicate the disease (pneumonia medicantaria from needless drenching).

Etiology.—The cause is unknown. The virus seems to be contained in the nodules and pustules; the saliva is therefore virulent. The disease is readily transmitted artificially. A spread to other animals (sheep, cattle, swine) and even man is recorded. This is rare, however, as the disease is seen ordinarily only in the horse.

Natural Infection.—Contagious pustulous stomatitis is spread by the saliva of the sick which contaminates the food and water. Transmission through polluted stable litter, utensils, sponges, hands of attendants, etc., also occurs. The disease does not assume an epizoötic form probably because the virulency of the virus rapidly attenuates in passing from animal to animal. Usually all horses exposed do not become infected.

The disease is generally introduced into a stable or pasture by an infected animal.

Symptoms.—The period of incubation is three to five days. The first symptoms noted are usually impairment of appetite and ptyalism. The patient holds the mouth shut; if opened a quantity of saliva spills out. The saliva is very viscid and

hangs in long threads from the mouth. The lips and cheeks are somewhat swollen and tender. On examination of the mouth, which the patient often resists, the mucosa appears congested and streaked with red. On the mucous surface of the lips, gums, the hard palate and particularly beneath the tongue about the frenum, appear firm red papules the size of a pea. The papules do not all erupt at the same time but successively, a fresh crop appearing after two or three days. From the papules vesicles form. The smaller vesicles break and heal while the larger ones turn to pustules which perforate, leaving behind small, round ulcers. Sometimes two or more ulcers coalesce, forming large, irregular areas of ulceration. The ulcers usually heal in a few days, leaving behind a white scar.

In some cases the pustular eruption may involve the lower part of the nasal mucosa, especially of the wings of the nostrils. Nasal discharge may be present which dries and adheres as brown crusts to the borders of the alæ. In rare instances the lacrimonasal canal is invaded, the infection spreading through it to the conjunctiva.

Sometimes the external skin is involved. There appear on the lips, nostrils and cheeks papules, vesicles and pustules like those observed on mucous membranes. Cases are recorded where the eruption appeared on other parts of the body (breast, shoulder, thigh).

The lymph vessels and glands adjacent to the eruption frequently swell, the vessels forming cord-like strands. There is no tendency, however, for abscesses to form along their course as in strangles. The submaxillary lymph glands are swollen.

Diagnosis.—The diagnosis is usually not difficult. If the case is at all typical, it is exceedingly easy. The contagious character of the disease and the formation of papules ending in pustules and ulcers is indicative. In vesicular stomatitis which develops only in the mucous membrane of the lower jaw, especially in the region of the frenum of the tongue, vesicles (clear fluid contents) appear, but never papules. Small abscesses and ulcers which occur in the mucous membrane of the mouth as the result of traumatism or caustics,

do not form pustules, the ulcers are usually deeper seated, and there is no evidence of contagiousness. In contagious acne the mucous membranes are not involved, although large pustules appear upon the skin in the region of the harness rests. It could be confused only with those rare cases of contagious pustulous stomatitis which do not involve the mucous membranes. Coital exanthema affects the genital organs. Horsepox is confined to the region of the fetlocks. If the nasal mucosa is involved, contagious pustulous stomatitis might be confused with glanders. Glanders, however, does not affect the buccal cavity. The ready healing of the ulcers and the rapid spread to other horses are not noted in glanders.

Course.—The disease usually lasts about two weeks and ends in complete recovery. It requires three to six days for the pustules to develop, they remain about four or five days and form ulcers which heal in about the same time. Severe cases may take a more protracted course in that one eruption follows another in succession. Fatal cases are rare. They result usually from secondary infection with pus organisms or the necrosis bacillus or in that pharyngitis develops from the specific process extending back to the pharynx, causing dysphagia and occasionally foreign-body pneumonia.

Treatment.—Internal medication is rarely indicated. The patient should be fed soft food, such as grass, gruels, bran mashes, etc., and allowed constant access to fresh water. The mouth may be syringed out two or three times daily with a 2 per cent. lysol solution. Skin ulcers may be treated with compound alum powder.

Prophylaxis.—If only a few horses are affected they should be isolated and given separate attendants. If, however, the disease is generally distributed and further spread inevitable, to shorten the course of the outbreak it is recommendable to inoculate the still healthy animals. This is easily performed by taking a clean towel, passing it through the mouth of an infected animal, and then through the mouth of those animals which have not yet taken the disease. A thorough disinfection of the premises should follow.

COITAL EXANTHEMA.

ERUPTIVE VENEREAL DISEASE. GENITAL HORSEPOX.

Definition.—Coital exanthema is a benign, acute, contagious disease of the external genital organs of cattle, horses, sheep and swine. It is characterized by a vesicular eruption which is transmitted from animal to animal by the act of coitus.

Occurrence.—The disease is very common in horses and cattle, outbreaks occurring during the spring and summer. It is sometimes confused with variola with which it has probably no connection. While the disease is benign it interferes with breeding, lactation in cows, and by affecting a number of animals in a district, attains economic importance. It is quite common in the United States.

Etiology.—The cause of the disease is not yet known. The virus seems to be contained in the lymph of the vesicle or the pus of the pustule or ulcer from which it may be transmitted by artificial inoculation.

Natural Infection.—Coital exanthema is nearly always transmitted by coition whereby the male animal infects the female and *vice versa*. The disease is spread usually by an infected stallion or bull. It is claimed that the male may transmit the disease without himself becoming infected in that the infectious secretions from a diseased female adhere to the penis and are lodged in the vulva or vagina of the female served by him soon after. This naturally would only apply where the service was performed within an hour or two following copulation with an infected female.

The disease once in a while occurs in females in advanced pregnancy, also in animals which have not been bred. This would indicate that occasionally intermediary agents, such as contaminated stable litter, utensils, sponges, etc., may carry infection.

Cattle seem to be more susceptible than horses; the disease is less commonly seen in sheep, goats, and swine. One attack produces a transient immunity, although cases are recorded where one and the same animal has been attacked repeatedly.

Symptoms.—The period of incubation is usually from two to five days. A minimum period of twenty-four hours and a maximum period of nearly two weeks have been recorded. The first symptom in females is an inflammation of the mucous membrane of the vulva and vagina in which appear small, red papules which in two to three days form red, pea-sized vesicles containing a clear, yellow fluid. The lesions are commonly found in the upper commissure of the vulva and in the neighborhood of the clitoris and the mucous surface of the lips of the vulva.

Similar eruptions may invade the skin around the vulva, anus, and the perineum. In a short time, from secondary infection, the vesicles become pustules, which erupt, leaving behind shallow ulcers which heal readily by granulation. In some cases, however, the ulcers are deeper, and when they heal, there is left behind a white or yellow scar which in time tends to disappear. In some instances, after the eruption of the pustule, tough, yellow crusts nearly a quarter of an inch thick remain. When these crusts are removed or drop off, white areas are left which are usually small and circular, but by confluence larger and irregular in form.

The lips of the vulva are edematously swollen as may be the surrounding parts. In mares repeated blinking of the vulva is noted. There is a vaginal discharge of a mucopurulent character which soils the tail and buttocks.

In male animals a similar eruption appears upon the penis, prepuce and sheath. The penis itself is swollen, and from the urethra flows a mucopurulent discharge. The above described depigmented areas noted in female animals are also seen on the penis in males. Urination is frequent and accompanied by considerable straining, the patient often switching its tail, arching the back and kicking against its abdomen. In males, especially stallions, the penis is often erected and a yellowish, mucopurulent discharge flows from the urethra.

Ordinarily the general condition of the animal is not impaired. The appetite is usually retained and there is no loss of flesh. On the other hand where the eruption has been extensive and associated with much pruritus, there may be

restlessness, frequent straining as if to urinate, the patient seeming much disturbed.

Pruritus, however, is not a constant symptom.

The usual benigh course of the disease may be perverted by secondary infection or through the patient rubbing the ulcerous surfaces. Thus in individual cases general pyemia or septicemia may follow.

Diagnosis.—The diagnosis is usually not difficult. The appearance of the vesicles and the ready spread by coition usually suffice for a diagnosis. If, however, no vesicles can be found, the condition might be confused with ulceration due to other causes. In this regard the rapid healing of the ulcer is suggestive. In granular vaginitis of cattle, red-gray nodules appear which give to the affected parts of the vagina a granular appearance. Vesicles are very rarely noted. The disease could hardly be confused with dourine, as it is much more contagious, its course more rapid and termination benign.

Course.—The course of the disease is usually brief and mild. Spontaneous recovery occurs in the majority of cases in two to four weeks. In severe cases or where proper treatment has not been applied, the secondary lesions developing from traumatism or infection, may take months to cure. In rare instances chronic vaginal catarrh, metritis, cystitis and the like may follow. In bulls abscess of the penis may permanently deform the organ or so weaken it that the animal is incapable of performing the coital act.

Treatment.—In mild cases no special treatment is necessary, although it is advisable to wash off the affected parts of the genital organs with disinfectants (phenol ℥j, tannin ℥j, glycerin ℥vj, water one gallon). Before applying disinfectants to the penis it should be washed thoroughly with soap and water, rinsed and dried. Obstinate ulcers which remain behind may be treated with nitrate of silver or copper sulphate. Some recommend flushing out the urethra with mild antiseptics. This is, however, rarely necessary. That an infected animal should not be bred is obvious.

CHAPTER III.

ACUTE INFECTIOUS DISEASES LOCALIZED IN CERTAIN ORGANS.

STRANGLES. CORYZA CONTAGIOSA EQUORUM.

Definition.—Strangles is an acute, contagio-infectious disease of horses, asses and mules, due to a specific streptococcus, which leads to catarrh of the upper air passages and abscess formation in lymph glands.

Occurrence.—Strangles is a disease of colthood and is, therefore, most prevalent in breeding districts. Adult or aged horses are more rarely attacked. The disease is most common in colts from six months to five years of age, although cases in patients under two months are not rare. The disease is widespread, but Ireland and the Argentine Republic are said to be free from it. Generally speaking strangles is a benign disease, but by checking the growth and sometimes causing the death of colts, it attains economic significance. Furthermore, some outbreaks are malignant in character and lead either to considerable losses from death (internal abscesses) or permanent injury through sequels (roaring, pharyngeal paralysis).

Etiology.—The Streptococcus equi of Schütz found in the lymph glands and nasal discharge of affected animals.

Natural Infection.—The streptococcus probably enters the body through the intact nasal mucous membrane, although other mucous surfaces (throat, bowel, vagina) may form ports of entry. Nasal discharge and pus from lymph gland abscesses or more rarely skin lesions are taken in with the food and water, reaching the upper air passages.

In breeding establishments strangles is usually introduced by a horse which has not yet fully recovered from the disease.

By direct contact with such an animal or one showing pronounced symptoms of the disease susceptible horses are readily infected. In a barn where strangles exists the air will be more or less impregnated with globules of discharge containing streptococci, especially if the ventilation is bad. Mangers, water buckets, troughs, stall partitions and floors contaminated with nasal or abscess discharge, may under favorable conditions retain the infection for several months. Therefore, horses placed in stables where the disease has existed or if they are permitted to eat or drink out of infected buckets, watering troughs, etc., may ingest the streptococci of the disease. Livery and dealers' stables are thus more or less permanent sources of infection, each year outbreaks occurring in them.

Colts may infect their dams by nursing; stallions mares during the act of coitus. Intra-uterine infection may also occur.

Some outbreaks which are not traceable to any recognizable source, may possibly be due to a saprophytic life which the streptococcus is thought to assume.

One attack of strangles usually affords life-long immunity. There are, however, many exceptions, individual animals suffering two or more attacks of the disease.

Any factor (refrigeration, overwork, poor food and care, etc.) which reduces the resistance of the horse predisposes it to the disease.

Necropsy.—The postmortem lesions are those of pyemia with abscesses in lymph glands (mediastinal, bronchial, mesenteric) or in any of the parenchymatous organs. Bronchopneumonia or suppurative pneumonia with lung abscess alone or combined with pleuritis and pericarditis is commonly noted. In the abdominal cavity the mesenteric lymph glands are found involved. In some instances a spontaneous rupture of the abscess has occurred, leading to a diffuse, purulent peritonitis. In others a more chronic condition is noted (adhesive peritonitis) with adherent loops of bowel. Where there has been general metastasis, multiple abscesses may form in any organ of the body (brain, muscle, thymus, heart, etc.).

Symptoms.—From a clinical standpoint it is useful to classify the cases in two types: (*a*) Regular strangles, and (*b*) irregular ("bastard") strangles.

Cases of regular strangles are those which take a relatively rapid and benign course, while those spoken of as irregular have a protracted course or suffer threatening complications or sequels. The period of incubation is from one to eight days, usually three to four days. The symptoms of the first type are:

Cough from an affection of the larynx or bronchi. The cough may persist a week or two after the other symptoms have subsided.

Nasal Discharge.—At first the discharge is watery but soon becomes mucopurulent. It is usually viscid and copious. If dysphagia is present, food, water or saliva are mixed with it. The discharge usually lasts eight to fourteen days, gradually becoming less.

Fever.—The temperature is usually elevated in the beginning of the disease to 102.5° to 104.8° F. Other symptoms of fever, such as languor, lowered head, disinclination to move, rough hair coat and sometimes chill are noted. When the abscesses are evacuated the temperature drops.

Swelling of the Lymph Glands.—Concomitant with the catarrhal symptoms a swelling appears, usually in the intermaxillary space. In most cases the swelling is edematous, hot, painful and fills the space between the jaws, and tends to suppurate, becoming "ripe" in four to eight days. In other cases, notably in older horses, and in patients which have suffered previous attacks of strangles the swelling is not marked and does not lead to abscess. Commonly the abscess opens spontaneously in six to ten days.

Dysphagia.—From the attending pharyngitis the patient will hold the head extended and through the nostrils water, saliva or even solid food particles are discharged. From the mouth saliva drools. The appetite is more or less impaired depending on the severity of the attack. The patient will often refuse concentrates (oats, corn) but eat some hay or grass. The thirst is not increased.

The Respirations.—If the disease is confined largely to the nasal mucosa, the respirations remain about normal. When the laryngeal or bronchial mucous membrane is much swollen, dyspnea with dilation of the nostrils occurs, the frequency often as high as 25 per minute.

The Pulse.—The pulse in the beginning of the disease is not much affected but later may reach 76 or higher.

The Conjunctiva.—Frequently in the earlier stages there is a flow of tears, and later a thick, mucous discharge from the inner canthus of each eye. In mild cases this symptom may not appear.

Irregular Strangles (*Bastard Strangles*).—In nearly every enzoötic of strangles some cases develop which differ in symptomatology from the typical form described. The principal difference between the regular and irregular types of strangles lies in the fact that in the latter the abscesses develop by way of metastasis in parts of the body other than in the submaxillary region. In irregular strangles any part of the organism may be selected as the seat of an abscess, although lymph glands are most apt to be involved.

For practical reasons only the most common seats will be considered. They will be grouped according to the part of the body in which they appear.

Head.—(*a*) Abscess in the pharyngeal lymph glands. When these glands are involved, there develops in the parotid region a swelling which may be quite extensive. It may reach the upper surface of the larynx, displacing the organ ventrally, and cause dyspnea. As the pharyngeal lymph glands lie on the lateral surface of the pharynx, their enlargement may interfere with swallowing. A spontaneous rupture of the abscess may occur, the pus discharging into the pharynx (sudden, profuse nasal and mouth discharge), or into the guttural pouches (sudden disappearance of the swelling, copious nasal discharge, dysphagia).

(*b*) Abscess in the anterior cervical lymph glands. This causes swelling in the region of the thyroid gland. Fluctuation is usually present. The abscess usually bursts outwardly although it may rupture into the gullet and lead to esophageal fistula. If the adjacent omohyoid muscle becomes infiltrated

with pus, dysphagia and aspiration (foreign body) pneumonia can follow.

(*c*) Abscess in the sublingual glands lead to swelling of the tongue. The organ may be so swollen as to protrude from the mouth. After bursting of the abscess, the edema of the tongue rapidly subsides.

(*d*) The superficial lymph glands and vessels of the skin of the head may become involved. In this case in the region of the eyes, nose, cheeks and lips, painful strands, the size of a slate pencil, and surrounded by indurated tissue, are noted. Later nodules form along the course of the swollen lymph vessels. They finally erupt and discharge pus. In individual cases considerable swelling of the head occurs leading to severe dyspnea.

(*e*) Abscesses in the facial sinuses may form and lead to empyema or very rarely to meningitis.

Body.—(*a*) Abscesses in the subcutaneous lymph glands of the body rarely occur in the region of the withers, flanks, etc.

(*b*) Abscesses may form in the mammary glands involving one or both halves of the udder. In stallions scrotal abscesses have been observed.

(*c*) The anal lymph glands are sometimes involved causing retention of feces and symptoms of proctitis.

Limbs.—(*a*) Abscess may form in the axillary, inguinal and popliteal lymph glands. While they usually heal readily, occasionally they are obstinate and induce lameness of considerable duration. This is especially true of popliteal abscesses.

(*b*) Abscess of joints (purulent arthritis) is not common. One or more joints may be attacked. When the leg joints are involved, there is severe lameness.

Internal Abscesses.—(*a*) Abscesses of the posterior cervical lymph glands (prepectoral) sometimes rupture inwardly producing purulent pleuritis and pneumonia. Pronounced dyspnea and the usual signs on percussion and auscultation of the thorax are noted on physical examination.

(*b*) Metastatic lung abscesses. The symptoms of pneumonia (cough, purulent nasal discharge, temperature, auscultation and percussion) indicate the condition.

(*c*) The mesenteric lymph glands. These glands may be primarily or secondarily involved. The symptoms are fever, mild colic and a history of exposure to strangles. The condition leads to death either gradually or suddenly through rupture of the abscess and the resulting peritonitis. Similar symptoms may arise from abscess formation in any other of the lymph glands of the abdominal cavity whether they be parietal or visceral.

Diagnosis.—The recognition of the usual case of strangles is not difficult. The purulent nasal discharge and abscess formation in the submaxillary region are characteristic. Cases occur, however, which are so atypical that from the clinical symptoms alone, a diagnosis is impossible. The history of exposure to strangles, the age of the patient and the existence of more typical cases on the same premises may assist to establish a diagnosis. The microscopical examination of the pus (nasal discharge) for streptococci and the inoculation of experimental animals (white mice) are frequently of service in this regard. Very puzzling are those cases in which the abscesses form in internal organs (lymph glands of abdomen), as the symptoms are often vague. The history of the case, the tendency to attacks of mild colic, the irregular temperature and the presence of albumoses in the urine are suggestive of hidden pus of probable strangles origin.

Course and Prognosis.—Most cases of strangles heal readily in two to four weeks without complication. This is especially true of the usual benign type ("regular form") of the disease which is confined largely to the nasal cavities and lymph glands of the submaxillary space. On the other hand, when the disease assumes an atypical ("irregular") form, affecting the pharynx and its lymph glands or involving internal glands or organs (lungs, mediastinal glands, mesenteric, lumbar glands, brain, etc.) the course is greatly protracted, and the prognosis doubtful to bad. When, after the submaxillary abscess is evacuated, the temperature continues up or rises again after a temporary fall, the development of further abscesses is probable. In some cases, as the abscesses heal, fresh ones appear, prolonging the course and complicating the prognosis. The resistance of the patient and its surroundings

are important factors in the prognosis. In very young colts, especially if lacking in vigor and growth, the disease is much more apt to be fatal than in colts over one year of age. Sucklings are not infrequently attacked in the pharyngeal lymph glands, leading to severe dyspnea and serious dysphagia followed by foreign-body pneumonia. When the patients are neglected, kept in damp, dark, dirty stables and poorly fed, the course is longer and the prognosis not so good as when opposite conditions prevail.

In some outbreaks the irregular form of the disease dominates, involving the internal lymph glands, especially of the abdomen and leading to death from peritonitis.

The mortality is 1 to 3 per cent. in the benign form but may reach 20 to 30 per cent. in malignant outbreaks.

The most frequent sequels to strangles are: (*a*) Purpura hemorrhagica; (*b*) roaring; (*c*) empyema of the sinuses of the head; (*d*) immobility from encapsuled brain abscess; (*e*) tabes mesenterica from chronic, adhesive peritonitis leading to thickening of the mesentery, bowel adhesions, emaciation and finally cachexia.

Treatment.—The principal factors in treating strangles are: (*a*) Isolation and care of the sick and subsequent thorough disinfection of the premises; (*b*) proper hygiene (light, cleanliness and ventilation), and (*c*) the early opening of the abscesses. The patient should be placed in a roomy, light, airy box-stall. In mild, sunny weather it may be turned out during the day. The feed box should be kept clean and free from accumulations of nasal discharge. The diet should consist of easily digested, laxative foods (steamed oats, bran mashes, grass, carrots). If the patient refuses all food and is growing weak, milk, eggs and alcohol may be used. Sometimes appetite may be stimulated by feeding small quantities of oats over which a little brown sugar has been strewn. Sucklings should be assisted at nursing, or in case they cannot suck (dysphagia) rectal feeding may be employed. Friction to the skin through good grooming is helpful. The nostrils may be cleaned with cotton, soaked in a $1\frac{1}{2}$ per cent. creolin solution and the canthi of the eyes kept free from pus accumulation.

The abscesses should be encouraged to "ripen" by hot fomentations. Blisters (biniodid of mercury in lard one to four) are occasionally dangerous (dyspnea from increased swellings). As soon as the first signs of fluctuation appear, the abscesses should be opened, using a long incision, and the pus discharged. Once evacuated, and thorough drainage provided, too much after-treatment with the syringe is contraindicated, as it retards healing. If, following the opening of the abscess, the temperature does not sink in the next twelve to twenty-four hours, further abscess formation may be looked for. If the swelling does not decrease in size, a second abscess is forming in the neighborhood. It is sometimes possible to thwart its development by puncture with a finger or blunt instrument from the original abscess cavity. Abscesses in the parotid region may be opened through Viborg's triangle. Pharyngeal abscesses are frequently deep-lying and can be reached only with a long, blunt instrument, such as the metal nozzle of a syringe. (See Surgery.)

If dyspnea develops and becomes serious, tracheotomy should be performed. In profuse nasal discharge, steaming the head is useful. It should not be repeated too often as it tends to produce atony of the mucous membranes of the nose and throat. The use of intralaryngeal and intratracheal injections (subnitrate of bismuth 5 per cent., tannin 2 per cent., Lugol's solution) are rarely necessary and sometimes dangerous.

Internal medication plays a secondary part in the treatment of strangles. Drug treatment should be expectative and contingent upon the arising symptoms. The fever and heart are treated as in influenza (see this). When the bowels are inactive, small doses of artificial Carlsbad salts may be used (tablespoonful in feed). The catarrhal symptoms are treated as in catarrh of the air passages (see this).

Of late antistreptococcus serum is much used in strangles. It is claimed it lessens the nasal discharge, shortens the term of fever and prevents complications arising. Further substantiation is desirable.

PROTECTIVE INOCULATION.—SERUM THERAPY.—As an attack of strangles usually produces immunity lasting for

several years, attempts have been made to immunize artificially horses against the disease.

(*a*) *Immunization.*—A serum produced from horses hyperimmunized with graduated intravenous doses of virulent strangles streptococci, is recommended to produce a passive immunity. It is given in 20 to 30 c.c. doses subcutaneously. The immunity produced, however, lasts only about a month. When injected during the incubation period of the disease it does not prevent its development but is said to reduce the severity of the attack and favorably modify sequels, fewer horses being left unsound.

(*b*) *Therapy.*—Daily doses (20 to 30 c.c.) of the immunizing serum seem to have a good effect upon the course of strangles when given to animals suffering from the disease. After its use the patient appears brighter, the nasal discharge less and the abscessing of the lymph glands prevented.

A vaccine prepared from the Streptococcus equi, so treated as to kill the organism, is used therapeutically in strangles. The dose is 1 to 10 c.c. subcutaneously, repeated in seven to ten days if necessary. Its use is most valuable in subacute or chronic cases in which it promotes the healing of abscesses and reduces the nasal discharge.

MALIGNANT HEAD CATARRH OF THE OX.

GANGRENOUS CORYZA. CORYZA GANGRÆNOSA BOVUM.

Definition.—A non-contagious, acute, infectious disease of the ox and buffalo, characterized by an inflammation of the mucous membrane of the head, leading to ulceration and the formation of pseudomembranes. The eye is also involved and nervous symptoms are present.

Occurrence.—Usually occurs sporadically or as a stable miasma, especially in unsanitary barns. The disease is relatively rare in the United States, but outbreaks have been reported from Ohio, New York, New Jersey and Minnesota. The disease is common in Europe.

Etiology.—The specific organism is yet unknown. It is assumed that the virus which causes the disease reduces the

resistance of the body tissues, especially mucous membranes, through toxins which it eliminates. The ulcers and pseudo-membranes may be due to secondary invading bacteria.

Natural Infection.—The mode of infection is not yet understood. Probably the germs harbored in damp, dirty stables contaminate the food and water and thus are taken in through the digestive tract. Occasionally the disease appears among cattle on pasture, but usually only when the available drinking water is foul.

The disease has not been transmitted by inoculation. It does not spread by contact. In some stables, as noted, it exists for years, appearing each spring and fall and in time causing considerable losses.

Young, fat cattle (one to three year old steers) are most often attacked. One attack does not produce immunity against subsequent ones. A given animal may suffer successive attacks a few weeks or months apart.

Symptoms.—The period of incubation is from twelve hours to two days.

The disease usually begins with symptoms of fever (104° to 107° F.). The surface temperature is unevenly distributed, the poll of the head hot, the muzzle hot and dry. The fever drops on the second or third day. Before death it becomes subnormal.

Nervous Symptoms.—The head is held low or rests on the manger. In some patients on the second or third day there is marked stupor, the animal lying on the ground unconscious.

In other patients there is excitement, the animal bellowing, rearing, and plunging. Locomotion is difficult, the gait uncertain and staggering. Twitching of the muscles of the neck, shoulders and body may occur. Epileptiform spasms have been observed. The excitability of the patient is increased by such external influences as bright sunlight, sudden noise, etc.

The eyes on the first or second day show conjunctivitis, which may extend to the cornea causing keratitis. There are photophobia and profuse lacrimosis. The cornea becomes turbid or "milky," the clouding beginning at the periphery. Iritis and cyclitis are also observed, the anterior chamber

being filled with yellow exudate. The episcleral blood-vessels are congested. From the fibrinous iritis the animal is often blind.

The Respiratory Tract.—There is at first a thick, viscid, purulent, later more fibrinous nasal discharge, mixed with blood, which finally becomes discolored and fetid. The croupous masses forming in the larynx narrow its lumen, cause noisy dyspnea, and at times distressing cough. The sinuses of the head may become filled with exudate and the horn core inflamed leading to the horns becoming loose. In thrashing about, the patient often knocks off the loosened horn.

The Digestive Tract.—The buccal mucosa is early reddened. Later in the course of the disease the gums, lips and hard palate become eroded. Croup membranes are sometimes noted on the mucous surface of the lips, cheeks and palate. The animals drool and fetid croup masses are occasionally ejected from the mouth. There may be dysphagia. The appetite is impaired. Water is drunk in small quantities. The bowels are constipated during the height of the fever, the feces tar-like. In the latter stages there is diarrhea. The feces are said to contain fibrinous masses and blood in some instances.

Skin.—An eczematous eruption (papulovesicular) has been observed. The skin of the head, especially at the nostrils is most apt to be involved, although the coronets may be elected.

Udder.—In cows the milk secretion stops. Rarely a coincidental mastitis occurs.

If the patient live several days it emaciates and becomes anemic.

Course.—The course is acute. The initial symptoms usually last one day. In three to four days the disease is fully developed. The patients rapidly emaciate and generally become unconscious and die in from four to eight days. In some cases this disease may be prolonged three to four weeks and end in recovery.

Prognosis.—Mortality 60 to 90 per cent. Marked nervous symptoms, high fever after the second day (pneumonia) are

bad signs. Those animals which do recover are left with such sequels as blindness, chronic nasal or gastro-intestinal catarrh.

Treatment.—This is largely symptomatic. (*a*) Cold applications to head and base of horns; (*b*) injecting out the nostrils with antiseptics and removing manually the membranous obstructions to breathing; (*c*) tracheotomy in apnea (don't be in a hurry to remove tube);[1] (*d*) trephining the frontal sinuses or even sawing off the horns and irrigating the cavities through the opening; (*e*) alcohol may be used in great mental depression or strong coffee added to the drinking water; (*f*) fever may be reduced by antipyretics; (*g*) constipation and diarrhea as in bowel catarrh.

The poll axe is indicated in severe cases (long convalescence; blindness, etc.).

NECROTIC STOMATITIS OF CALVES. CALF "DIPHTHERIA."

Definition.—Necrotic stomatitis is a contagio-infectious disease of suckling calves in the course of which there develop on the buccal mucous membrane croupous-diphtheritic patches and ulcers. It is usually attended by a general toxemia which leads to death.

Occurrence.—The disease is very common in Europe, and serious outbreaks have been reported in the United States, particularly in Colorado, Wyoming, South Dakota, Iowa and Southwestern Texas.

Etiology.—The cause of the disease is the Bacillus necrophorus of Bang, a thin, long rod which forms in tissue and in artificial media slender, undulated filaments.

Natural Infection.—Probably comes from the calves getting into the mouth soiled straw or litter which is contaminated with necrosis bacilli. Healthy calves in contact with diseased ones are infected by licking the sick or picking up matter contaminated with the nasal and mouth discharges of the affected. The disease is very easily transmitted, spreading from calf to calf.

Very young calves (three to five days old) become infected.

[1] Of very questionable value in practice.

Necropsy.—On postmortem in the mucous membrane of the mouth and throat are found areas of dirty, gray or brown color, irregular in form and well circumscribed. They are frequently found on the cheeks and borders of the tongue, but may also involve the upper respiratory passages and occasionally the intestinal canal, or even the region of the coronet. In some cases large surfaces of the mucous membrane are covered with a yellowish-gray, friable, fetid, cheesy mass. Sometimes the lungs are infected, in which purulent or caseous foci develop. Occasionally there may be pleuritis. In some instances the small intestines are catarrhally inflamed and dotted here and there with small, pea-sized, grayish-yellow, caseous deposits. The spleen is not enlarged, though the lymph glands are swollen.

Symptoms.—The period of incubation is from three to five days. As noted, very young calves are most often attacked. The first symptoms observed are languor, disinclination to suck, and slight increase in temperature. There soon develop drooling from the mouth and slight swelling of the cheeks. The examination of the mouth, which is painful to the patient and therefore resisted, reveals that the mucous membrane of the cheeks, tongue, hard palate, and fauces shows areas of redness and erosion. These areas represent patches of yellow or grayish-yellow pseudomembranes, or ulcers. The patches are from the size of a five-cent piece up to a silver dollar and quite irregular in form. The necrotic mass is very adherent to the underlying tissue and can be removed only with difficulty. It may be an inch in thickness and involve the muscle or even bone.

The disease frequently involves the nasal cavities, producing a yellowish or greenish-yellow, sticky discharge which adheres closely to the border of the nostrils. Occasionally the nose is obstructed by accumulations of exudate causing difficulty in breathing. If the larynx and trachea are involved there will be cough and dyspnea. Besides these local symptoms there are those of general toxemia, such as loss of appetite, extreme languor, weakness and temperature ranging from 105° to 107° F.

Diagnosis.—The diagnosis is not difficult, and depends upon the fact that in this disease well marked, thick, necrotic areas and deep ulcers occur upon the mucous membranes in the regions noted. In no other disease of the calf are these deposits so marked.

Course.—The disease, if left to itself, in most cases leads to death in five to eight days. Some individuals may live as long as three weeks and then die of pneumonia. Cases which recover, do so very gradually, convalescence lasting for weeks. The healing of the ulcers is an extremely slow process.

Prognosis.—Calves affected with necrotic stomatitis if left to themselves usually die, or if they recover, remain permanently stunted in growth. On the other hand, if proper treatment is administered early, most of them recover, healing requiring about two weeks.

Treatment.—The treatment consists in thorough cleansing and disinfection of the mouth cavity. Twice daily the mouth should be syringed out with a 2 per cent. creolin solution in warm water. The patches of necrosis may be scraped off or curetted away in so far as this is feasible. After the sores are irrigated, it is recommended to cover them with a paste made of one part salicylic acid and ten parts glycerin applied with a stick or brush. Lugol's solution of iodin is also recommended. The calf may be allowed to suck its mother, or if a weanling, should be fed liquid food, best milk. Plenty of cool water should be kept within reach.

Prevention.—The sick calves should be separated from the healthy ones. The mouths of exposed calves should be examined once daily for lesions of necrotic stomatitis. Disinfection of the stalls, pens, buckets, etc., should be made, using a 3 per cent. creolin solution.

NECROTIC STOMATITIS OF PIGS.

Definition.—Necrotic stomatitis is a contagio-infectious disease of suckling pigs characterized by a necrosis of the mucous membrane of the mouth and occasionally of the skin of the snout.

Occurrence.—The disease is widely distributed and occurs most commonly among pigs kept in unsanitary quarters or which are allowed to root in soil polluted with the bacillus of necrosis.

Etiology.—The bacillus of necrosis.

Symptoms.—The symptoms very much resemble those described for calves although the tongue is not so commonly involved. The inflammation usually begins in the gums about the incisor and canine teeth, which become swollen, very sensitive, bleed easily and assume a bluish-red color. In two or three days the involved areas are covered with diphtheritic patches of yellowish, cheesy material which may later slough, leaving behind ulcers. The inflammation may extend to the snout causing it to become swollen, sensitive and later necrotic. A fetid odor is exhaled from the mouth. The process may spread to the pharynx, causing dysphagia; or to the larynx, inducing dyspnea and cough. Later in the disease blood-stained saliva flows from the mouth and the incisor teeth become blackened and loose ("black tooth").

The affected pigs frequently attempt to nurse, but on account of the sensitiveness of the gums, fail to get much milk. They are usually dull, languid and remain lying most of the time. In some cases a fetid, black diarrhea is manifest. The udder of the mother sow may also be involved.

Diagnosis.—The diagnosis is not difficult and depends upon noting the characteristic necrotic lesions and the fetid expirium.

Course and Prognosis.—The course varies from a few days to several weeks, depending upon the virulency of the infection. The prognosis varies. In milder infections, where treatment is promptly applied, it is good; if neglected and allowed to become chronic, or if the intestines are involved (diarrhea), the prognosis is bad.

Treatment.—Treatment, to be successful, must be applied early. Once the disease is well advanced, it usually proves fatal in spite of treatment. Antiseptics, such as silver nitrate (1 per cent.), zinc chlorid (3 to 5 per cent.), creolin (3 per cent.), or potassium permanganate (2 per cent.), applied daily are recommended. A simple method of treatment is

to extract the loose teeth, scrape away the necrosed tissue and apply iodin tincture to the raw areas remaining. If persisted in, healing can be produced usually in ten days.

LUNG PLAGUE OF CATTLE. CONTAGIOUS PLEURO-PNEUMONIA OF CATTLE.

Definition.—Lung plague of the ox is a specific contagio-infectious pleuropneumonia which may assume an acute, subacute or chronic form and is due to a filtrable although visible virus.

Occurrence.—The disease does not exist in the United States at the present time. In 1843 an outbreak occurred in New York, which had its origin in an infected cow imported from England. In the years following the disease spread throughout the country, getting as far west as the Mississippi River and south to Virginia. It was not until 1892 that it was finally entirely stamped out.

Lung plague is now well under control in Europe, although still quite prevalent in Spain. Throughout Asia and Africa the disease is general. Australia, New Zealand and Tasmania are infected. In the Philippine Islands the disease is widespread.

Etiology.—Lung plague is due to a very minute microorganism which passes through coarse porcelain filters but may be seen under the microscope when magnified over 1500 diameters. It is the smallest visible microörganism as yet known, being almost ultramicroscopic. The organism has been grown in artificial media. The virus is found in the pleural exudate, diseased lung and in nasal discharge. The blood rarely is infectious.

Natural Infection.—Probably through the respiratory tract. Experiments to produce typical lung plague in susceptible animals by inhalation or feeding have not been successful.

The disease spreads usually by direct contact with infected animals. Infected and emptied stables in which within a year sick cattle had been kept are common sources of infection. Where cattle are crowded together in a barn the disease spreads most rapidly. On the other hand, however, a

rapid spread among cattle on pasture has been frequently noted.

Very commonly a supposedly recovered ox with an encapsuled lung focus is the infecting agent. So long as the sequestered center in the lung is completely encysted, no virus will be thrown off, but once a communication is established with a bronchus giving the virus exit the patient becomes a source of danger to healthy cattle. The virus may remain virulent for two or three years in a lung sequestrum of an apparently healthy ox.

The disease is more prevalent in stable-fed than among pastured animals. It affects all breeds, although some individuals possess natural immunity.

Necropsy.—As a rule only one lung (left) is involved. Usually a large area is hepatized. On cross-section a clear, yellow fluid, which quickly coagulates, flows over the cut surface. The interlobular connective tissue is greatly thickened, forming yellow, gelatinous strands one-half an inch or more in thickness, which divide the lung tissue into islands of varied color. Characteristic of the lung lesions are the different degrees of inflammation which appear at the same time, fresh areas of congestion lying beside those showing red or gray hepatization or even necrosis. As a result the cut surface presents a distinctly variegated, marbled appearance very characteristic of the disease. The bloodvessels are distended and thrombosed. The corresponding lymph glands are swollen and edematous.

In chronic cases the proliferated interlobular connective tissue is almost white in color and very firm. The portions of the lung they surround are necrotic and sometimes calcified. In such cases a thick, connective-tissue capsule may inclose areas from the size of a walnut to a grape fruit—so-called sequesters. A zone of reactive inflammation surrounds the capsule. The pleura shows serofibrinous pleuritis which can involve the pericardium. Rarely there is serofibrinous peritonitis especially in the region of the diaphragm and liver. In calves arthritis of individual joints and a gelatinous infiltration of the subcutis (dewlap, chest) may exist.

Symptoms.—The period of incubation is from one to four weeks. In many instances it appears shorter, the early symptoms (temperature) being obscure and passing unobserved. In very hot weather the attack is often more sudden and severe than in the cold season.

The symptoms are very varied. From a clinical standpoint usually two stages can be recognized. During the first stage (so-called occult stage) a peculiar, short, weak, painful cough is heard, especially after drinking or eating, or when driven up or out of the barn. The patients are languid, show capricious appetite, suppressed rumination and stand with back arched, head down and ears pendent. Driving the animal induces dyspnea. The temperature is usually somewhat elevated (one or two degrees). This condition may exist from two to four weeks, and lead to recovery or the symptoms may become more pronounced. Second stage: The temperature ascends to 104° to 108° F., and severe dyspnea develops, the mouth is held open, tongue protrudes, and each expiration is accompanied by a loud moan. The patient usually stands with its neck extended and elbows turned out; if it lies down it does so on the affected side. There is usually complete loss of appetite, suppressed rumination and cessation of milk flow. There is often a mucopurulent nasal discharge stained with blood. The feces are dark and dry; later a profuse, fetid diarrhea develops. On percussion (which is painful) over the affected side an extensive area of dulness may be detected. On auscultation bronchial tones and rales and occasionally frictional sounds are heard. If the consolidated area is not near the lung surface, however, percussion and auscultation are negative. The abnormal respirations are best heard after exercising the patient.

In fatal cases the patient rapidly emaciates, becomes hidebound, anemic, cachectic and dies of exhaustion in three to six weeks.

If recovery occur, it generally takes place gradually and is not always complete, the patient remaining unthrifty for a long time.

Diagnosis.—*Intra vitam* a positive diagnosis is very difficult or impossible. Usually only a careful necropsy will

establish beyond doubt the existence of the disorder. A clear history of the prevalence of the disease in the country or community, the insidious onset, the fact that only a portion of the cattle are attacked at the same time and the clinical evidence of pneumonia are very suggestive if not convincing. Lung plague can be confused with:

(*a*) *Hemorrhagic Septicemia* (*Pectoral Form*).—While this disease usually has a sudden onset, is much more acute and attacks larger numbers of animals simultaneously, some acute cases of lung plague may greatly resemble it, not only clinically but on necropsy. In doubtful cases only a bacteriological examination (finding bipoled bacillus; animal inoculation) will determine.

(*b*) *Tuberculosis* (*Pulmonary*) may be confused with a chronic case of lung plague. The absence of continued fever, lack of pleural symptoms, the even more chronic course of tuberculosis and the results of the tuberculin test usually suffice to differentiate between the two. However, lung plague and tuberculosis may occur concomitantly in the same animal.

(*c*) *Pneumomycosis* (*Aspergillosis of the Lungs*) is rare in cattle, affects most often birds. The development is very slow; little fever. On necropsy the presence of Aspergillus fumigatus in the bronchi and consolidated lung tissue is conclusive.

(*d*) *Verminous Bronchitis.*—Affects mainly calves. Coughing prominent. Parasites or eggs occur in the ejections.

Course.—The course of the disease is varied. Many cases recover during the early stage and after only a few days illness. In others recovery is slow and often imperfect, pulmonary sequestra remaining. Such an animal, as noted, is a dangerous source of infection.

The course is generally more stormy in young, well nourished patients, although calves are quite resistant. The mortality is usually from 60 to 70 per cent. Not over 20 to 30 per cent. fully recover. A number remain chronically affected, but may fatten.

Depending on circumstances, the disease may persist on a given premises for years. This is particularly true where

only those showing clinical symptoms are disposed of, no thorough disinfection practised and later new cattle brought in to replenish the herd.

Treatment.—Palliative measures are rarely successful and generally lead to the further spread of the disease. To wipe out lung plague all sick and exposed animals should be slaughtered and the premises they occupied (barns, shed, etc.) thoroughly disinfected. Once this drastic method is enforced the disease is soon entirely eradicated. In the United States and in other countries where it no longer exists, no cattle should be imported from an infected country without passing through a strict (ninety-day) quarantine.

Preventive Inoculation.—In countries, as in South Africa, where it is impossible to enforce drastic measures of eradication or even efficient quarantine, preventive inoculation against lung plague of cattle has been practised with considerable success. Among the different methods employed the following are described briefly:

(*a*) *Nocard Method.*—Nocard employed living cultures of the causal organism which were injected subcutaneously in the dorsal aspect of the tail three or four inches from the tip. One to four weeks later the animal reacts with symptoms of slight fever, increased respirations and the formation of an inflammatory swelling about the size of a hen's egg which appears at the point of inoculation. Of nearly one thousand cattle so inoculated none died and only three suffered loss of the end of the tail.

(*b*) *Pasteur Method.*—The material for inoculation is obtained from a local subcutaneous swelling, produced artificially by injection of the yellow, fibrinous exudate from the interlobular connective tissue of the lung of a natural case. This material is injected into a healthy three- or four-months-old calf in the dewlap or behind the shoulder. From the resulting swelling the "lymph" is collected after the calf has been killed. A few drops are injected under the tail tip. This method has not proved very successful as the virulency of the "lymph" becomes too attenuated to produce the desired immunity in all cases. A modification of this method

is to inject the "lymph" intravenously. The danger of phlebitis, however, makes its use rather hazardous.

(*c*) *Old Method.*—The material was collected by expressing from the interlobular connective tissue of a hepatized area of an affected lung a quantity of the liquid exudate which was filtered through clean linen to remove solid matter. The injection was made under the tail tip after the hair had been shaved from an area and the skin disinfected. A more primitive method was to soak a piece of woolen thread in the exudate and insert it as a seton under the skin. In one week to one month a swelling occurred at the point of inoculation and the animal would show slight fever. If the material used was contaminated or infection allowed to enter the inoculation wound, necrosis of the tail and sometimes even pyemia result. The mortality from this method is about 2 per cent. and about 10 per cent. suffered loss of tail.

CHAPTER IV.

INFECTIOUS DISEASES INVOLVING PRINCIPALLY THE NERVOUS SYSTEM.

TETANUS. LOCKJAW.

Definition.—Tetanus is an acute, infectious disease due to an anaërobic microörganism which produces in the body a toxic product resembling strychnin in its physiological action. The disease is characterized by tonic spasms of the muscles. The mind of the patient remains undisturbed.

Occurrence.—While tetanus has a wide general distribution it is confined to infected districts. Where the soil has become contaminated with the germ of the disease, it is of common occurrence. For this reason it is more prevalent in the tropics than in northern climes; in some parts of the country commoner than in others. Horses, swine and sheep are more often attacked than other domestic animals. The disease is more prevalent in the spring and fall than during other seasons. Since the use of antiseptics has become more general, tetanus is not as frequent as formerly.

Etiology.—The disease is caused by the Bacillus tetani, an anaërobic, rod-shaped germ, usually carrying a spore at one end. The germ occurs in the spore form in earth, putrifying fluids and manure. In infected districts tetanus spores are normal inhabitants of the intestines of ruminants.

Natural Infection.—Infection takes place through a fresh wound into which the spores of the specific bacillus have gained entrance. Obviously wounds so situated as to be contaminated with soil or manure are most apt to become infected. Therefore wounds in the feet, scrotum, umbilical cord, compound fractures of the limb bones, tooth cuts and eye wounds are most dangerous in this regard. As the original wound may be very small and heal by first intention, it

cannot always be found. This led to the former belief in "idiopathic" tetanus. In cattle tetanus most commonly follows parturition where rough manipulations have been made to relieve dystocia. Newborn animals may become infected through the navel. The practice of docking lambs leads to tetanus the infection entering the fresh tail stump. Limited enzoötics of tetanus have occurred among horses, swine and sheep where castration without sufficient precaution was practised in infected districts.

Necropsy.— There are no constant lesions found on postmortem. The brain and cord present nothing characteristic. The condition of the wound through which the infection entered is very varied. Usually it is not granulating well and there is little pus discharge. The nerves are often bruised, congested and swollen. If the infection was through the umbilicus (tetanus neonatorum) the navel may be inflamed.

One attack of tetanus does not produce immunity. A given animal may suffer more than once from the disease.

Symptoms.—The period of incubation is usually from one to two weeks. A minimum period of twenty-four hours has been noted in very young animals and exceptionally in older ones. As a rule the disease reaches full development in one to two days. During the prodromal stage the patient is stiff, does not care to move and shows loss of appetite or at least slow mastication. The ears are held erect. If the head of the patient is elevated a protrusion of the nictating membrane over the eye occurs, a symptom most pronounced in the horse. Tetanus may be partial, involving only parts of the body (partial tetanus) or it may be general affecting the whole body (universal tetanus). In some cases the muscular spasms are confined to the head and neck; in others the hind parts; in still others the whole body is involved.

Horse.—When the symptoms are fully developed and the reflexes stimulated by excitement, the patient assumes a characteristic attitude: The legs are spread and stiff, the neck and head are extended and the tail elevated. The ears stand erect approaching each other, the eyes retracted and in part covered by the nictating membrane. The pupils are

dilated, the nostrils distended and the nasal wings trumpeted. The mouth is held shut with the commissures drawn upwardly. From a spasmodic contraction of the masseter muscles it may be impossible to open the mouth more than a fraction of an inch (trismus). Due to a contraction of the constrictors of the pharynx, dysphagia and ptyalism are present. While in most cases the back is held straight and rigid, occasionally it is arched downwardly (opisthotonos) or still more rarely curved laterally (pleurothotonos). Locomotion is difficult, the limbs being advanced stiffly and the feet barely raised from the ground. It is almost impossible to back the horse. The muscles are tense and hard, individual muscles standing out prominently. Twitching of the muscles is a symptom often noted. These symptoms may subside temporarily provided the patient is in no way excited. However, any sudden noise, a flash of bright light or an unaccustomed sight will cause the spasms to return. If the patient is struck with the hand a paroxysm of muscular contraction passes over the body. The mind of the patient is clear although the face shows anxiety and a peculiar rigid stare. Sometimes in stallions the penis is erected. The pulse is small, the artery hard. In severe cases the heart beat is rapid and often palpitating. The respirations are increased three to five times their normal frequency. As the blood is charged with CO_2 a cyanosis of the mucous membranes appears. The lungs are commonly congested and edematous so that rales are heard on auscultation. In rare instances the respirations are noisy or pronounced roaring occurs. As swallowing is difficult, saliva, food or drugs may enter the windpipe causing foreign-body pneumonia and gangrene of the lungs. The temperature is affected only in severe cases and usually just before death or if some complication has set in (pneumonia, septicemia). One to two days before death it may reach 110° F. As has been observed in other diseases accompanied by severe muscular spasms, the temperature remains high for several hours after death (postmortal temperature). The appetite usually remains good, although mastication is labored. Food is often retained in the mouth or coughed out into the manger. From a decomposition of the

unswallowed food and saliva the expirium becomes fetid. A regurgitation of liquids and solids through the nostrils is not uncommon. The peristalsis is suppressed and defecation is difficult. Strangury is occasionally present; the specific gravity of the urine is high.

In tetanus the patient usually stands during the course of the disease. If it should fall to the ground it rises with great effort or must be assisted to its feet. In the last stages the patient usually falls to the ground where, after showing violent muscular spasms, it dies in a few hours.

In local tetanus the muscular symptoms are confined to the muscles nearest the point of infection and the spasms are not severe. Generalized tetanus is usually preceded by local tetanus.

Ox.—In cattle, tetanus most often follows obstetrical operations. The symptoms are usually not so marked as in the horse and are sometimes quite vague. The reflexes are not much increased, the animal appearing stupified rather than excited. From a contraction of the paunch muscles bloating commonly occurs. From the vulva there is often a putrid discharge. Emprosthotonos has been observed.

Sheep.—In lambs, following umbilical infection or as the result of castration and docking, tetanus may assume an enzoötic form. The symptoms in sheep are much like those in the horse. Opisthotonos is usually well developed.

Swine.—In swine the disease commonly follows castration or ringing. There is usually marked trismus.

Diagnosis.—The characteristic tonic muscular spasms, the normal mind and the absence of temperature speak for tetanus.

From strychnin poisoning the disease is distinguished by the fact that the symptoms of this poisoning are much more acute and between the paroxysms there is no rigidity. Trismus is further rarely present in strychnin poisoning except in the last stages.

The disease might be confused with an acute muscular rheumatism. However, this is not apt to occur if the symptoms are carefully noted. There is no prolapse of the nictating membrane and the muscles are tender on palpation in rheumatism.

Tetanic symptoms have been noted in cases of intestinal irritation in the horse due to the presence of ascarides. The symptoms, however, are very mild and the case usually yields to proper treatment, such as giving a vermifuge.

Tetany is a rare condition in animals. It may occur when a torn or severed sensory nerve heals in the lips of the wound (castration).

Course.—The course is very varied. Some cases die in two to three days, while others may live two to three weeks and the disease terminate fatally. Death usually occurs, however, in three to ten days after the appearance of the first symptoms. Cases which terminate fatally usually grow steadily worse from the beginning. There are, however, exceptions. Sometimes the patient dies suddenly from respiratory arrest or the aspiration of food (oats) when recovery seems probable. The course in local tetanus is benign provided it is not complicated with trismus.

Where the termination is favorable, the contractions of the muscles become less after the second week. Convalescence usually lasts four to six weeks.

Prognosis.—The mortality in tetanus is 55 to 90 per cent. The disease is not so fatal in the ox as in other animals. In sheep the mortality is 95 to 100 per cent.

The earlier the disease appears after infection and the more rapidly and severely the symptoms develop, the more fatal the attack. Fever is a bad sign. Where the patient is unable to eat on account of trismus, the termination is usually fatal. Severe dyspnea may lead to hypostatic congestion of the lungs and death. On the other hand, if the case develop gradually, after a long period of incubation, and the symptoms of generalized tetanus are not severe, no fever is present and the appetite retained, the outlook is more favorable. Even in these cases, however, the prognosis should be made with caution as fatal complications may occur at any time.

Treatment.—*Hygienic.*—The patient should be removed to a quiet, darkened stall. There is no objection to a mare being allowed her foal or a horse its teammate. Idle curiosity seekers should be kept away. The animal should be given soft food, and water kept within constant reach. Slings

should be used only when absolutely necessary to keep the animal on its feet and where the temperament of the patient permits of their use. If there is difficulty in defecation the feces may be removed from the rectum. If the bladder is distended it should be emptied best by careful pressure. While theoretically the primary wound should be curetted and disinfected, in the horse this is often a difficult procedure. Furthermore it is not always possible to find the wound.

Medical.—Internal medication is of little avail. On account of the danger of drenching, drugs should be given as far as possible with the food and water. To keep the bowels open, salts may be administered. Opiates such as morphin, chloral hydrate and the bromides afford only temporary relief. Inhalations of chloroform and ether just before a meal undoubtedly assist mastication by temporarily relieving the trismus.

Subcutaneous and intramuscular injections of phenol solutions are highly recommended by some authorities. Subcutaneously 1 ounce of a 2 per cent. solution or 2 drams of a 10 per cent. solution of phenol in glycerin may be administered twice daily. One dram of a 5 per cent. solution may be injected into the muscles of the neck and shoulders. The injection may be repeated once every three hours for the first thirty-six hours.

Tetanus Antitoxin.—The administration of tetanus antitoxin, while it confers temporary immunity against the disease, has not proved valuable as a curative agent. It is most effective when used early and in subacute cases. As a curative agent 3000 to 20,000 units should be given. This amount may be split into several doses: For instance, 20 c.c. may be used for the first dose followed by 15 to 20 c.c. in five to ten hours.

Prevention.—Tetanus may be prevented by thoroughly disinfecting all fresh wounds and by the use of small doses of antitoxin. In infected districts the use of antitoxin to produce immunity prior to important surgical operations, treatment of fresh wounds (especially punctured feet in horses, castrations, etc.) has proved extremely valuable. In coal mines, where general tetanus commonly follows foot injuries,

and in the Panama Zone, a badly infected district, good results have been obtained from antitoxin used as a preventive. The immunity produced lasts about one month.

RABIES. LYSSA. HYDROPHOBIA.

Definition.—Rabies is a contagious, generally fatal, infectious disease, transmitted by the bite of an infected animal and characterized by delirium, nervous excitement, and finally paralysis. Its incubation period is very varied and no characteristic macroscopic lesions are found on postmortem.

Occurrence.—The disease is most common (80 per cent.) in dogs which, when infected, inoculate by biting other animals (horses, cattle, sheep, and swine) or human beings, thus spreading the disease. Rabies occurs in nearly every country in the world. Australia is said to be free from it, and since the introduction of the last muzzling law in 1895 the disease has disappeared in Great Britain. Within the past ten years rabies has become widespread in the United States. No state is free from it, but accurate statistics as to its prevalency are not available. From 1900 to 1910 it was reported in 73 cities, causing the death of 230 persons.

Etiology.—Rabies is due to an organism which in certain stages at least is ultramicroscopic and passes through bacterial filters. The virus is found in the tissues and fluids of the infected body, especially in the central nervous system. It also occurs in the saliva, pancreatic juice and milk, occasionally in the aqueous humor and has been found to exist in the blood. The muscles seem free. In 1903 the Italian investigator, Negri, discovered in the protoplasm of certain nerve cells of rabid animals small, stainable bodies which are now called "Negri bodies." They were demonstrated in 95 to 98 per cent. of the cases of rabies examined, and are rarely found in old, healthy dogs (immunes). It is probable that these bodies are protozoa which in some stages of their development are small enough to pass through bacterial filters. Of this, however, there is as yet no scientific proof available.

Natural Infection.—Rabies is essentially a disease due to the bite of a rabid animal, the saliva of which contains virulent virus. Such saliva coming in contact with any fresh wound could produce infection. The saliva of an animal may be virulent as early as eight days before the termination of the period of incubation and before symptoms of the disease develop. The virus, as in tetanus, extends along the nerves to the brain and cord. It may also be carried by the blood and lymph. The danger of the bite of a rabid animal depends upon the virulency of the saliva, character of the wound and the number of lymph vessels and nerves injured. Bites inflicted by carnivorous animals are more dangerous than those produced by herbivora on account of the form of the teeth and the character of the wound they produce. Wounds near the brain and cord are especially apt to be followed by infection. In horses, bites in the lips, nose, and cheeks are therefore dangerous. The length and thickness of the hair or wool covering the part bitten are important factors, a heavy, thick growth catching most of the saliva and preventing its entering the wound. Recently shorn sheep are thus much more susceptible than when wearing the full wool coat. The infected wound usually heals as any other wound, quite often by first intention. Infection through the intact skin has not been demonstrated. Not over 30 to 50 per cent. of the animals bitten by rabid animals take the disease.

Necropsy.—There are no characteristic lesions of rabies. In herbivorous animals fairly constant are the empty stomach, congested lungs, and larynx. The other organs are either normal or show secondary lesions not directly due to rabies.

Symptoms.—The period of incubation is very varied. Generally the disease breaks out two to eight weeks after inoculation. Much longer periods have been observed. An incubation period of one to two years is probable. The length of the period is determined by the virulency of the virus, the character and location of the wound, and the age of the animal. Young animals are more susceptible than adult or aged ones. In general the symptoms of rabies are much the same in all animals. They are modified only by the natural peculiarities of the different species. In practically all animals

are observed psychic, sensory, and motor-nervous disturbances, the absence of fever, lost or perverted appetite, rapid emaciation, and fatal termination. The psychic changes are shown by hyperesthesia and the tendency to attack other animals or even persons by biting, kicking, or horning and the continued bellowing in cattle; the sensory by the licking, gnawing, tearing, or rubbing the part of the body which was bitten (itching of part, neuralgia), and the motor by hyperkinetic symptoms, such as clonic spasms or twitching of muscle groups, or, on the other hand, by akinetic phenomena, as sudden dropping, paresis or paralysis, change in voice, etc. The perverted appetite is recognized by the fact that rabid patients often eat their own dung and drink their own urine in preference to normal food. In animals two clinical types of rabies have long been recognized: (*a*) The furious form, and (*b*) the paralytic (dumb) form. They are more pronounced in the dog than in other animals. Between the two types, however, intermediate forms are noted, so that clinically many cases occur which do not clearly belong to either type. The dumb form may suddenly change to the furious, and *vice versa*. In dogs, further, three stages of the disease are fairly well presented, *i. e.*, the stage of melancholia, the stage of mania, and the stage of paralysis. They are best observed in typical cases of the furious form of the disease. In the other domesticated animals they are rarely well defined. Rabid animals rapidly emaciate and almost always die within ten days after the first symptoms appear.

Horse.—The patient is first noticed to rub or gnaw the healed bitten wounds (lip, nose, forelimb). At first the friction thus applied is moderate, but later in the disease deep excoriations and severe injury to the part rubbed or gnawed are induced. The skin of the metacarpus may be torn, exposing the underlying tendons and bones. Naturally swelling of the part results. The patient is usually quite excitable, restless, pawing, alternately lying down and getting up, symptoms not infrequently mistaken for colic. Biting into the manger, stall partitions, etc., is a common occurrence. The lips, gums, and even the teeth are thus injured, as the patient disregards caution in its destructive delirium. If a

stick be presented, the horse snaps at it or seizes it with his teeth. A water pail may be seized and smashed. In some cases slight dysphagia is an early symptom, saliva drooling from the mouth; in drinking, regurgitation of water through the nose follows. In occasional patients marked symptoms of fury are noted, the animal rearing wildly into the manger, and with mouth and hoof seemingly trying to tear the stable down. Blankets, feed boxes, studding, in fact anything which may come in the way are torn or splintered. Other horses or even men are attacked during the paroxysm. In stallions and mares increased sexual desire is noted. The appetite is perverted, the horse ingesting dung and urine. A change of voice also occurs in horses, but is not as marked a symptom as in dogs and cattle. About the second or third day paralytic symptoms appear, the patient remains down, and dies in convulsions or coma. In some cases the paralytic symptoms are not preceded by a stage of fury, the course of the disease resembling the dumb form of rabies of dogs.

Ox.—Rabid cattle are restless, excited, and particularly aggressive toward dogs and fowls, which animals they pursue with avidity. Even inanimate objects, if in motion (a rolling pumpkin), are chased by them dog fashion. In milch cows milk secretion stops. Not infrequently they attempt to bite, seizing the coat sleeve of a person standing near. The patients have an anxious, mischievous expression, and quite frequently a peculiar movement of the muzzle, like that observed in the healthy rabbit. Sometimes violent contractions of the abdominal muscles, as if to defecate, are seen. Anything which attracts their attention they rapidly approach and try to gore and climb upon with their forefeet. A common symptom is a sudden loss of muscular coördination or power which causes the animal to drop to the earth as if "pulled down" by a rifle shot. They remain down but a moment and spring to their feet again. The appetite is vitiated, dung and feces being licked up in preference to good food. Water is not refused, but it may be swallowed with difficulty. Continued bellowing is a prominent symptom, the sound of the voice becoming gradually hoarser and fainter. The patients from day to day grow weaker and more emaciated and finally get down and are unable to rise.

Death usually occurs about the seventh to ninth day. In some outbreaks the disease takes a fatal termination in three to six days.

As in the horse, paralytic symptoms (dysphagia, bloating, constipation, paraplegia) may appear without being preceded by a furious stage. The patients get up from a recumbent position with difficulty, walk with a staggering gait, show marked ptyalism, bellow continuously; food and water are regurgitated through the nose and mouth; finally they lie prone on the ground, unable to rise, show spasms of the diaphragm and other muscles and, the temperature dropping below normal, die in five to seven days.

Sheep.—In general, the symptoms in sheep are similar to those in cattle, although, as a rule, the patients are not as aggressive and destructive in their tendencies. A very common symptom is increased sexual desire, the affected sheep mounting their fellows. Occasionally aggressive symptoms are observed, the otherwise shy animal attacking by butting the other sheep, dogs, or even persons who enter the pasture. Occasionally they try to bite. The disease usually lasts three to five days, and ends in paralysis and death.

Swine.—In hogs the symptoms are quite similar to those observed in dogs. The patients are very restless, keep running around the pen, and squealing in a hoarse voice. They bury themselves in the straw and gnaw the parts where bitten. Sudden noises arouse them, and occasionally they will attack other animals and man. There is usually profuse ptyalism. Water they attempt to drink, but usually cannot swallow. Quite commonly young pigs will come together head on and push each other around the inclosure. In a few instances the disease resembles the dumb form in dogs, and the patients show no aggressive symptoms but are simply paralyzed, unable to swallow, show changed voice, and die in two to five days.

Diagnosis.—Where there is history of the animal having been bitten and the symptoms of the different stages well developed a diagnosis *intra vitam* is usually not difficult. In the furious form of the disease the aggressive and destructive tendencies of the patient are very suggestive. However,

these symptoms are not always present. Occasionally the diagnosis is extremely difficult and cannot be made during the life of the animal. Generally the psychic, sensory, and motor disturbances, the fatal termination, and the negative post-mortem are indicative. As a rule, however, a positive diagnosis can be made only by microscopic examination or experimental inoculation. In cases of doubt it is best to confine the animal for a day or so for observation, during which time there usually develop sufficient symptoms to make the diagnosis highly probable.

The microscopic examination, which is highly valuable, consists in the examination of properly prepared and stained brain tissue, particularly of the hippocampus, medulla oblongata, and cerebellum. In practically 98 per cent. of the cases of rabies which died or were killed in the advanced stages of the disease, peculiar cells, the so-called Negri bodies, are found. The presence of the Negri bodies indicates rabies while their absence tends to disprove its existence.

Diagnostic Inoculations.—An emulsion is usually obtained from the medulla oblongata of an animal which died or was killed because rabies was suspected. This is injected subcutaneously or subdurally into rabbits or sometimes pups. Intra-ocular and intramuscular inoculations have also given successful results. Usually in from two to three weeks after the injection the experimental animal dies of typical rabies provided the material used came from a rabid animal.

Course and Prognosis.—The disease usually lasts four to seven days. It is extremely rare for it to exceed ten days in any animal. While a few recoveries have occurred in cases produced by artificial inoculation, authentic records of recovery from natural infection are wanting. The disease is generally fatal.

Treatment.—Once the disease is fully developed no treatment is of any avail. To prevent rabies the fresh bitten wound should be thoroughly disinfected with a 3 per cent. carbolic acid or a 1 per cent. bichlorid of mercury solution. If the wound is older and granulating the actual cautery or caustics, such as strong hydrochloric acid, sulphuric acid, strong ammonia, etc., are indicated. Subcutaneous injec-

tions into the tissues adjacent to the wound may be helpful. Bichlorid (1 to 10,000), or 1 per cent. carbolic acid, may be used. The prompt application of a ligature above the bite, if applicable, is often life-saving. Generally speaking, however, the prevention of rabies by the treatment of the bitten wound is successful only where it has been applied promptly and within the first fifteen minutes after the injury is made.

The preventive treatment as commonly practised in man consists in subcutaneously injecting the patient daily for a period of fifteen to twenty-one days with an attenuated virus the virulency of which is increased with each successive injection. This is commonly spoken of as the Pasteur treatment. Since the cost of production has been lowered, it is now applied in veterinary practice. The success following its use in animals does not seem as good as in man. Where promptly applied, and the attenuated virus good, excellent results are recorded in human practice.

Prophylaxis.—Rabies may be absolutely prevented by doing two things: (*a*) Inforcing a dog tax and keeping the public thoroughfares free from stray dogs; (*b*) by muzzling all dogs which are allowed to run at large. That these measures are only successful when applied to an extensive territory is obvious. Applying them only to a small district will not give beneficial results, as a rabid dog during the prodromal stage of the disease may wander far and wide, biting any live stock with which it may come in contact.

CHAPTER V.

CHRONIC INFECTIOUS DISEASES.

TUBERCULOSIS. CONSUMPTION.

Definition.—Tuberculosis is a chronic, contagio-infectious disease due to a bacillus tuberculosis and characterized by the formation in the different organs of the body, of small nodules, nodes, or larger irregular areas which tend to caseate, undergo fibroid degeneration, or calcify.

Occurrence.—Tuberculosis occurs in all domesticated animals, although it is very rare in sheep. In fact all warm-blooded animals and many cold-blooded ones (fish) are susceptible to it. In man one-seventh of the race die of it (150,000 annually in the United States alone). In animals cattle, swine, and fowls are most commonly infected. The prevalency of bovine tuberculosis, as in other contagious diseases, depends upon the opportunity for infection and spread. It is, therefore, most common in large herds confined in stables and less frequent in small herds living in the open. In the western ranges of the United States and in the great open grazing districts of other countries (steppes of Russia, South American pampas) tuberculosis is comparatively rare. On the other hand in the more densely populated parts of this country (Atlantic seaboard, Middle West, neighborhood of large cities) it is very prevalent. In practise a greater percentage of tuberculosis will be found in dairies and in herds of full-blood cattle maintained and sold for breeding purposes (stud or "seed" cattle). In both instances the opportunity for infection is great (indiscriminate purchase of new, non-tuberculin tested animals) and the close contact in which cattle of these classes are kept still further favors the spread of this contagion. While every state in the Union is infected,

in some districts it is much less common than in others, and even in badly infected districts whole herds of cattle are found free from the disease. Modern transportation facilities are important agents in spreading the disease among cattle. The illegitimate use of tuberculin by unscrupulous persons is a further factor in the spread of bovine tuberculosis. Cattle reacting to the test are frequently sold as healthy to unsuspecting buyers, who thus introduce the disease into their herds. Tuberculosis of animals is not yet as prevalent in the United States as in other countries. It is constantly increasing, however, especially in states where no adequate measures have been inaugurated to combat it. Swine are infected from tubercular cattle in two ways, *viz.:* (*a*) By being fed milk containing tubercle bacilli, and (*b*) by feeding on the excrements or offal of tubercular cattle.

The prevalency of animal tuberculosis can be estimated with approximate accuracy from abattoir statistics and the results of tuberculin testing. In Germany nearly 21 per cent. of the cattle and 3 per cent. of the swine killed for food have been found affected. The tuberculin test showed over 50 per cent. reacting. Fully 25 per cent. of the cattle of Germany are infected, and in France over 10 per cent. In the United States 1 per cent. of the cattle are found tubercular on slaughter and 2.5 per cent. of the hogs. Results from tuberculin tests on 400,000 head of cattle gave 10 per cent. reacting. It is very probable that 1 per cent. of the beef cattle and 10 per cent. of the dairy and stud herds of this country are tubercular.

Etiology.—The cause of tuberculosis is the Bacillus tuberculosis of Koch. Three types of this bacillus are fairly well defined, *viz.:* (*a*) Typus humanus, (*b*) typus bovinus, and (*c*) typus gallinaceus. (See Bacteriology.)

Natural Infection.—A tubercular animal can spread the disease only by throwing off tubercle bacilli. Such are spoken of as cases of "open tuberculosis." On the other hand, where the animal is tuberculous but no tubercle bacilli are passing from it, the case is one of "closed tuberculosis." Bacilli may not pass continuously from "open" cases. "Closed" cases may at any time change to "open" ones.

Modes of Infection.—Tubercle bacilli are taken into the body: (*a*) *Via* digestive tract with contaminated food and water; (*b*) *via* respiratory tract by the inhalation of tubercular spray ejected by coughing or lowing infected cattle; (*c*) *via* genital organs during coitus; (*d*) *via* udder through teat canal; (*e*) *via* wounds (very rare; may follow castration), and (*f*) congenital tuberculosis has been noted only in isolated cases.

(*a*) Calves and swine are commonly infected through milk from creameries, especially skimmed milk obtained by centrifugal separation. The ingestion of cattle excrement by swine is a pregnant source of infection, especially in America, where the practise of allowing hogs to follow cattle is much in vogue. The feeding of the offal of slaughter houses to swine is likewise dangerous, as tubercular lungs, livers, lymph glands, gastro-intestinal contents, etc., are consumed.

The bronchial exudate of tubercular cattle, coughed up or otherwise raised from the lungs, may mix with the saliva, and thus tubercle bacilli are carried to feed or watering troughs, bedding, etc. As most of the exudate is swallowed by the patient (not expectorated as in man), the feces become polluted, scattering bacilli wherever dropped. Susceptible animals (hogs and cattle) eating or drinking substances contaminated by such discharges become infected. The same would apply to any other secretion or excretion containing tubercle bacilli (vaginal discharge, urine, etc.).

(*b*) Infection through the respiratory organs comes from the inhalation of either globules of bronchial exudate, mixed with mucus and saliva, which are coughed out or otherwise forcibly ejected from the nose and mouth of tubercular cattle; or of tubercle bacilli which have become partially dried and are adhering to dust particles floating in the air. In the former case cattle immediately next to an "open" case of tuberculosis acquire the infection by close contact, and in the latter, which is far less frequent, by breathing in the dried bacilli which contaminate the inspired air.

(*c*) Infection by coitus may occur provided the genital organs of the bull (penis, prostates, testes), or cow (vagina,

uterus) are diseased. This form of infection is relatively rare, but more common than generally supposed.

(*d*) Through the teat canals tubercle bacilli from contaminated bedding, manure, etc., may reach the udder, inducing a primary tubercular mastitis. A general infection from this source is seldom noted.

(*e*) While infection through skin wounds is not infrequent in man, in animals it rarely occurs. In swine and cattle it has been observed to follow the use of raw milk applied to fresh castration wounds. Accidental wounds of the prepuce in bulls and udder in cows, in contact with contaminated litter, bedding, etc., can form ports of entry for infection.

(*f*) Congenital (intra-uterine) infection is rare. It may occur if tuberculosis of the uterus is present and in advanced generalized tuberculosis (uterus intact).

Conceptional or germinal tuberculosis (infected sperm or ova) has not been proved.

Modes of Elimination.—In cases of "open" tuberculosis the bacilli may be eliminated from the body through the following channels: (*a*) By coughing out or otherwise ejecting infected bronchial exudate through the nose and mouth; (*b*) with the feces contaminated with swallowed bronchial slime or from the discharge of tubercular ulcers in the mucous membrane of the digestive tract; (*c*)[1] the milk will contain tubercle bacilli if the udder is infected, or when advanced, generalized tuberculosis is present, and the udder seems intact; (*d*) the urine contains tubercle bacilli when the renal pelvis or parenchyma is tubercular or in tuberculosis of the reproductive organs (vagina, uterus; prostates, epididymis), the contaminated exudate or secretions afterward mixing with the urine.

Tuberculosis is essentially a stable disease in that the opportunity for infection and spread is greatest where ventilation, light, and cleanliness are inadequately provided. Further, in stables the animals are in closer contact with one another than in the open. However, tuberculosis is observed in cattle which are never housed and hogs are frequently

[1] The milk of apparently healthy cows which react to the tuberculin test only occasionally contains tubercle bacilli.

infected when out of doors following tubercular cattle. As in other contagio-infectious diseases, darkness, dirt, and foul air are conducive to the propagation of tuberculosis, while the opposite conditions tend to inhibit its development. For these reasons life in the open is a useful preventive and curative measure especially in the earlier stages of the disease. In advanced cases it helps relatively little. (See Treatment.)

Susceptibility.—As noted, tuberculosis is the most widely prevalent disease of cattle and very common in swine and fowls. On the other hand, horses, dogs, and cats are seldom infected, and in sheep the disease is extremely rare. A high resistance offered to tubercular infection may be racial or individual. It is believed that certain breeds of cattle, for instance, are less predisposed than others. The semi-wild strains from the Russian steppes and the native cattle of Japan seem more resistant than those of more refined origin. The long-horn of Texas and the West was apparently an immune. However, the opportunity for original infection and subsequent spread of the disease has been much more difficult among these cattle than it is in the more thoroughly domesticated European breeds from which our better American cattle sprang. Among the tamer breeds (Jerseys, Shorthorns, etc.) no racial differences in resistance have been noted. The manner of caring for and the use to which the animal is put probably has as much to do with the susceptibility to tuberculosis as any racial peculiarity. Selection in breeding operations with only precocity in development, or an unnaturally great milk production in view, to the exclusion of other factors (good constitution, etc.), will produce a race of low resistance to any infection. If tuberculosis happens to be the disease to which this race is exposed, infection is the more apt to take place.

Individual immunity against tuberculosis is commonly observed. In notoriously infected herds a few animals will sometimes remain healthy, although surrounded by every opportunity to take the disease. Whether this immunity is acquired or congenital is difficult to state. Accurate experiments to determine whether or not it may be handed down to future generations are wanting.

Necropsy.—The lesions of tuberculosis may appear in any organ in the body with the exception of the teeth. The location of the lesion may depend upon the kind of animal, mode of infection, and whether the disease is primary (local), or secondary (generalized). In the ox, tuberculosis is usually confined to the lungs, serous membranes, and lymph glands. In swine the digestive tract with corresponding lymph glands is most frequently involved. In the horse the lymph glands (mesenteric, retroperitoneal) are generally elected. However, exceptions to this rule are frequently noted; in generalized (spread *via* blood) tuberculosis the nodules may occur in any organ, even being found in muscle. The influence of the mode of infection is difficult to determine, since it has been proved that subcutaneous inoculations in calves (even at the tail tip) with tubercle bacilli were followed by pulmonary lesions.

The most characteristic lesion in tuberculosis is the tubercle which has undergone caseous degeneration. The lesion may vary in size from a small millet seed (miliary tubercle) to a cheesy mass larger than a human head, due to the confluence of numbers of smaller foci. While the individual tubercle is at first of a translucent, gray appearance, later from the degeneration, which begins in its center, it assumes a yellow color. The formation of nodules tending to caseate, particularly if corresponding lymph glands are similarly affected, is characteristic of tuberculosis.

Ox.—As noted, the lungs, serous membranes, lymph glands, especially the bronchial and mediastinal, are most commonly involved.

Lungs.—In the lungs nodules or nodes of varied size, of firm to fluctuating consistency, with usually well-defined outline, invade the tissue. On cut surface the dry, yellow, friable caseation surrounded by a thick capsule is found, or, on the other hand, the contents are soft, puriform, thick-fluid, covered by a thin, connective-tissue layer. The color is grayish-yellow to pronounced yellow. The size will vary from that of a millet seed to a clenched fist, or, by confluence, a whole lobe of lung tissue may be found changed to a caseous mass. In old cases calcification of the tubercle occurs,

whereby it grits under the knife when cut through. Usually in the neighborhood of a larger node, small tubercles are present. While the lung tissue between the tubercles is often normal, sometimes the intervening alveoli are filled with tubercular exudate and the interstitial tissue thickened. By the confluence of smaller nodes great tubercular masses form. Sometimes the center of the mass is hollow (caverns), but more often filled with friable, dry caseation or moist pus. Between some of the cavities and bronchi a communication forms through which a secondary infection with pus cocci or saprophytic bacteria takes place, in some instances changing the character of the caseous or puriform mass to that of ichor, causing the color to become grayish and giving it a fetid odor. Tuberculosis of the superficial parts of the lung often extends to the pleura, leading usually to circumscribed, pleuritic adhesions. Very rarely a diffuse, serofibrinous pleuritis develops.

In calves pulmonary tuberculosis assumes the form of a catarrhal pneumonia. On cut surface the affected area of the lung is strewn with small, round, reddish-gray to yellow areas, which, by confluence, are enlarged to caseous centers the size of a hickory nut or larger. Bronchitis almost always attends pulmonary tuberculosis, therefore lesions in the bronchi are noted on postmortem. The signs of bronchial catarrh with bronchiectasis are common findings, the dilated air tubes filled with mucus or cheesy masses. At times the bronchial mucous membrane is ulcerous. The trachea is less liable to ulceration than the larynx. In the latter organ tumor-like, connective-tissue growths occur, sometimes almost entirely occluding the lumen. On section the neoplasm is found to contain small, gray or yellowish tubercles.

Serous Membrane.—The pleura is most frequently involved. In the earliest stages reddish-gray, small granules develop surrounded by masses of connective tissue. On section of these masses areas of caseation from a millet seed to a pea in size are revealed. By confluence and simultaneous connective-tissue proliferation, round nodes or cauliflower-like excrescences protrude from the serous membrane. The protuberances may have a broad base or be pediculated. While in

consistency they are at first soft, later they become firm to hard. The tubercles are imbedded in the growth of connective tissue and new-formed bloodvessels, where they caseate and calcify. By confluence great polypoid masses several centimeters thick occur, appearing not unlike a bunch of grapes, hence the old name "grape disease." Not infrequently these nodular protuberances will involve the pericardium and epicardium, leading to adhesion between them. The endocardium and valves of the heart are rarely involved.

Lymph Glands.—The lymph glands corresponding to the affected organ are almost always tubercular. Not infrequently only the lymph glands are diseased. This is especially true in young animals in the earlier stages of the disease, and in older individuals condemned by the tuberculin test. In some cases the lymph glands on the surface of the body, particularly the submaxillary, subauricular, prescapular, and precrural are involved. In other infections the supramammary glands are elected. The tubercular lymph gland is often enlarged to many times its normal size and presents a nodular surface. On section it will be found to contain tubercles which appear either as round or irregular-shaped, radiating areas of caseation, sometimes surrounded by a capsule of connective tissue, and often calcified. In pulmonary tuberculosis especially the mediastinal and peribronchial lymph glands are diseased. In the digestive tract the suprapharyngeal, mesenteric, and portal lymph glands are elected. The glands may attain the size of a double clenched fist, and in some instances interfere with the functions of organs with which they come in contact. Partial occlusion of the esophagus when mediastinal lymph glands are involved is often observed. The mucous membrane of the digestive tract may show nodules or ulcers. As a rule the borders of the tubercular ulcer are thickened and the base caseous. They extend into the submucosa or muscularis. Usually the environing mucous membrane is thickened and catarrhally inflamed.

Liver.—Besides tuberculosis of the peritoneum covering the liver, in the parenchyma of the organ small tubercles or larger, dry, caseous or softer, puriform areas are noted. As

a rule the nodes and abscesses are surrounded by connective tissue capsules. In some instances from connective-tissue proliferation the liver may attain several times its normal weight.

Spleen.—Tuberculosis of the spleen is usually confined to a few small tubercles scattered through the parenchyma of the organ. The spleen is usually affected in young cattle only.

Kidneys.—A tubercular nephritis is common in old cattle. In the parenchyma of the kidney caseous tubercles are noted surrounded by connective tissue capsules. By confluence larger nodes form which may proliferate into the pelvis of the kidney. The ureters, bladder, and urethra may be involved.

Genital Organs.—In male animals the epididymis and the testes are most frequently elected; in the female the uterus and uterine tubes. In the uterus round tubercles may be palpated in the early stages, but later, due to connective-tissue proliferation, the walls becoming greatly thickened and rigid, isolated tubercles may not be felt. Tubercles, caseous or calcified, of grayish-white color, are found on section. In some instances superficial tubercles may lead to ulceration of the uterine mucosa.

Udder.—Tuberculosis of the udder appears as caseous or calcified nodes in the parenchyma of the organ, usually in the neighborhood of which smaller foci are present. As a rule only the hind quarters are affected. In some cases an enormous enlargement of the tubercular quarter or quarters occurs, while in others, on the contrary, an atrophy is noted. In the wall of the milk ducts, milk cistern and even teat canals, small tubercles find their seat, the lumen being filled with a cheesy detritus and sometimes a turbid, yellowish-green fluid.

In recent cases of embolic infection the lobules of the quarters concerned are swollen, and on section are found strewn with grayish tubercles, the intervening connective tissue showing numerous caseous areas from the size of a millet seed to that of a pea. The supramammary lymph glands are in all cases tubercular.

Tuberculosis of the central nervous system, bone, joints

and muscle is relatively rare in cattle. It is seldom that the skin, tendons, penis, prostates or eye form foci of infection.

Acute Miliary Tuberculosis.—This form of tuberculosis is usually seen to accompany a primary lesion from which it sprang by way of thrombosis or direct eruption into a blood-vessel. Not infrequently, in the same lung, along the course of a bronchus is found a large, irregular-shaped, caseous or calcified primary focus, and throughout the rest of the lung tissue, a number of small, round tubercles all of about the same size and alike caseous (secondary foci). These tubercles are usually evenly distributed, and each surrounded by a red zone. In the liver, spleen and kidneys similar lesions may be present. The corresponding lymph glands in miliary tuberculosis are always acutely swollen and their cortical substance abnormally reddened.

Symptoms.—Fully 90 per cent. of the cases of tuberculosis in animals present no clinical symptoms. As long as the disease is local and does not seriously involve the gastro-intestinal tract, or if there is no general intoxication of the organism with the toxins of secondary infection, a remarkable destruction of parenchymatous organs may follow and the patient appear healthy. In generalized tuberculosis or, as noted, if the bowels are much involved, or sapremia is attending, symptoms develop. The character of the symptoms is, however, so indefinite that they cannot be relied upon with any degree of certainty. Any of them may be caused by other diseases and none is pathognomonic of tuberculosis.

Fever.—The temperature of the body in tuberculosis is usually not disturbed until the late stages of the disease, when fever of an intermittent or remittent type sets in. Sometimes the temperature is higher in the morning than in the evening. As a rule, following a period of fever, there may be several weeks of normal temperature. Only in the last stages is the fever of a continuous type. As the symptoms of tuberculosis vary in the different domesticated animals, each kind of animal will be considered separately as follows:

Ox.—The period of incubation after artificial infection in bovine tuberculosis is two weeks or more. Following natural infection it is probably much longer. As a rule months or

years elapse before appreciable symptoms appear. In cattle tuberculosis inducing clinical symptoms affects the following organs or tracts:

(*a*) *Lungs.*—Cough is often a noticeable symptom. The cough is usually short, dry, and infrequent, occurring at first early in the morning when the cattle are driven up to feed or milk. Sometimes moving the animals, a cold drink of water, or a chilly draft of air (opening the stable door) induces it. In an occasional case the cough is paroxysmal. During the act of coughing a fine spray is ejected from the nose and mouth, and following it a viscid, bronchial exudate is swallowed. Sometimes a portion of this exudate is retained for a time in the mouth and pharynx, from whence it may be removed with the hand. (See Diagnosis.)

Dyspnea is usually not a prominent symptom when the patient is at rest. After brisk motion, however, the respirations become abnormally rapid and labored.

Percussion.—As the tubercles in the lung are generally surrounded by air-containing alveoli, percussion is usually negative. Only when large areas (at least 10 cm. broad) of solidification are superficially located and the thoracic wall relatively thin, is dulness noted.

Auscultation is very often negative. Especially after exercise in some cases, bronchial breathing and rales are heard. The rales are either dry or moist, depending upon whether the exudate is tough-viscid or more fluid in character. Dry rales, as a rule, predominate and are heard over the whole field of auscultation.

If the pleura is also involved (tubercular pleuritis, pearl disease) the patient may show pain on pressure over the ribs and percussion induces coughing. Friction sounds on auscultation can rarely be distinguished. Generally the clinical symptoms of tubercular pleurisy are too vague to be of diagnostic value.

Appetite.—In the earlier stages the appetite is retained, but toward the end (when the animal becomes emaciated) it is lost.

Loss of Flesh.—In the later stages of pulmonary tuberculosis the animal begins to lose flesh notwithstanding good

food and care. The hair coat lacks luster, becomes erect and the skin feels leather-like and thick. In time emaciation is in evidence, the patient very anemic and, toward the end (usually after months), cachectic.

(*b*) *Larynx.*—In tubercular laryngitis palpation of the larynx readily causes coughing. In some cases tubercular growths develop in the lumen of the larynx, inducing great dyspnea and even suffocation. As swallowing becomes difficult the animals eat little, and hence fall off in flesh.

(*c*) *Lymph Glands.*—The lymph glands may be primarily diseased or in association with other organs (lungs, udder, bowel, etc.), which are also tubercular. The following superficial lymph glands are most commonly elected: Submaxillary, parotid, prescapular, precrural and supramammary. The glands enlarge to form tumor-like growths, plainly visible on the surface of the body. They may attain the size of a large potato, are round or oviform, little sensitive, firm, nodular, not readily movable and the overlying skin not adherent. In calves they may show fluctuation and when incised discharge a thick, white pus.

Internally, tubercular lymph glands may interfere with the functions of organs with which they are in contact. The mediastinal glands, if much enlarged, may depress the dorsal wall of the esophagus, constricting its lumen, and thus indirectly lead to intermittent bloating. The enlargement of the suprapharyngeals causes dysphagia.

(*d*) *Udder.*—Usually secondary. In the latter stages there appear in the hind quarters firm, painless, not well-defined, nodular enlargements which may develop into hard, tumor-like growths as large as a human head. In some cases the whole quarter or quarters may be swollen to enormous size and be almost of the consistency of stone. Smaller enlargements (lumps) are best palpated after the udder is milked out. The supramammary lymph glands are increased in size to sometimes that of a clenched fist. In not a few cases only these glands seem tubercular, the udder appearing intact.

The milk is usually normal in appearance for a long time after the udder is involved. In the late stage, however, it

becomes mixed with tubercular exudate, is watery and of greenish color, or contains white flocculæ.

(*e*) *Bowels.*—The only tangible clinical symptom of intestinal tuberculosis is a persistent and incurable diarrhea. The patient usually soon emaciates, becomes cachectic, and dies.

(*f*) *Genital Organs.—Peritoneum.*—A remarkable development of peritoneal tuberculosis can be present and the patient not only appear in health but gain in flesh. In cows tuberculosis of the peritoneal covering of the ovaries may induce nymphomania, the animal showing almost continuous estrum. Later the patients fall off in flesh.

(*g*) *Uterus.*—The most constant symptom is a vaginal discharge of a mucopurulent, yellowish, ichoric, fetid character. Rectal examination may reveal the greatly thickened, rigid wall of the body and horns of the uterus. Failure to conceive (perpetual bulling) and abortion may be attending symptoms. In the later stages sexual desire is lost.

(*h*) *Vagina.*—Hickory-nut-sized, yellowish nodules occur in the vaginal walls often near the vulva.

(*i*) *Testes.*—There appears a non-painful, firm swelling of the epididymis, later the testes become enlarged forming a swelling of considerable size. Hydrocele is a common attendant and perforation with pus discharge is not uncommon.

Swine.—As a rule there are no clinical symptoms of diagnostic value. The disease may involve:

(*a*) *The Lymph Glands,* especially those of the throat, neck and prescapular regions (submaxillary, pharyngeal, prepectoral, etc.). In pronounced cases a firm, nodular, non-sensitive swelling of the throat and neck appears which may be extensive enough to interfere with mastication and the movements of the head. Occasionally fluctuation and perforation with the discharge of thick pus or cheesy masses occur in some part of the swelling. A tendency for fistulæ to remain is noted.

(*b*) *Lungs.*—The symptoms are much like those of the pulmonary form of hog-cholera and consist in cough, dyspnea, emaciation, anemia, cachexia and death in about one month.

(*c*) *Bowels.*—A primary intestinal tuberculosis in pigs follows feeding with infected skimmed milk or slaughter house offal. The symptoms are similar to those observed in cholera but the course is generally longer, the animal wasting gradually. It is sometimes possible to palpate through the abdominal walls firm, nodular enlargements which are either tubercular lymph glands or adherent loops of diseased intestine. Death follows months of decline.

(*d*) *Bones and Joints.*—The vertebræ and ribs and the joints of the legs are most commonly attacked. Rarely are symptoms noted in bone tuberculosis. In tubercular arthritis the affected joint is chronically swollen but presents no symptoms of acute inflammation. Lameness is usually present.

HORSE.—Tuberculosis is rare in the horse and the symptoms are usually too vague to be more than suggestive. Briefly, they are those of chronic cough, dyspnea, early fatigue when at work and intermittent nasal discharge (sometimes bloody). Percussion and auscultation are generally negative. Finally the animal becomes emaciated, anemic and cachectic. In colts (fed infected cow's milk) a tuberculosis of the bowels and mesenteric glands has been observed. The symptoms are not characteristic. The colt remains stunted in growth, pot bellied, may show periodical attacks of colic, and constipation alternating with diarrhea. On rectal examination enlarged lymph glands may be palpated.

In adult horses polyuria has been observed in some cases. As in the ox enlargement of the superficial lymph glands occurs. Tubercular ulceration of the nasal mucous membranes is very exceptional. The temperature in equine tuberculosis is much as in the ox—intermittent or remittent. Morning exacerbations and evening remissions in the course of the fever have also been observed.

Diagnosis.—As noted, in the majority of cases tuberculosis is a local disease in animals and presents no clinical symptoms. A physical examination of the tubercular patient will, therefore, reveal nothing to indicate the presence of the disease. Even in those cases of advanced tuberculosis the symptoms

are too vague and indefinite to be depended upon with any degree of certainty. A positive diagnosis of tuberculosis, therefore, from the clinical symptoms alone is not tenable. Other aids to diagnosis must be employed, the principal ones of which are the following:

A. *The Tuberculin Reaction.*—Tuberculin may be applied in several different ways. The most important methods of application given in the order of practical importance are the following: (*a*) The subcutaneous; (*b*) conjunctival, and (*c*) dermal.

(*a*) The subcutaneous application of tuberculin, which consists in injecting the tuberculin in proper dosage into the loose connective tissue under the skin, is the best known and most reliable method. The reaction following is general in that it induces in tubercular animals (best in cattle) a febrile temperature which usually begins in six to eight hours, reaches its acme in twelve to twenty hours, and lasts for twenty-four to forty hours after the injection. (For detail of technic, interpretation, etc., see Malkmus's Clinical Diagnostics.) Tuberculin properly used is a very reliable diagnostic agent. In 98 per cent. of the cases a positive reaction indicates the presence of a tubercular lesion which can be determined on necropsy. Tubercular animals may not react to the test under the following conditions:

1. When the disease is in the period of incubation (Moore).
2. When the progress of the disease is arrested.
3. In advanced, generalized cases and the condition of the patient is bad.
4. When the animal has been just previously (within four weeks) injected with tuberculin.

In the last two instances cited (3 and 4), by increasing the dose of tuberculin, a reaction is apt to follow.

(*b*) The conjunctival application consists in instilling into the conjunctival sac a few drops of tuberculin (undiluted). In tubercular cattle in six to twenty-four hours symptoms of conjunctivitis develop (congestion, swelling, lacrimosis) with the accumulation of a yellow, flocculent exudate in the inner canthus of the eye. The reaction may last two to four days. It is spoken of as positive when the conjunctivitis is pro-

nounced and the exudate purulent. Milder reaction (simple catarrhal conjunctivitis) may occur in healthy individuals, especially where full strength tuberculin has been used. Further, in healthy cattle the application of this test at spaced intervals will often induce an apparent reaction which is very confusing. Sometimes tubercular cattle will not react. Negative results, therefore, are not decisive.

(*c*) The cutaneous applications of tuberculin consist in either rubbing the tuberculin (*a*) into the intact (shaved and cleaned) skin (dermic), (*b*) painting it upon the scarified skin (endermic), or (*c*) injecting it into the substance of the skin (intradermic), probably best into one of the skin folds extending from the tail-root (tail elevated) on each side downward to the anus. In tubercular cattle the positive reaction, modified somewhat by the method of application, consists in an inflammatory (edematous) swelling which usually appears within twenty-four hours and often lasts for two or three days. When the skin has been scarified, small vesicles sometimes appear. While the dermal application of tuberculin may furnish valuable contributory evidence in detecting tuberculosis, it is often not conclusive enough to be relied on. Negative results are not always indicative of the absence of tuberculosis. However, this method of applying tuberculin is still in the experimental stage.

B. *Microscopic Determination of Tubercle Bacilli in the Secretions and Excretions or in Tissue from the Suspected Patient.*—While in man the examination of sputum for tubercle bacilli is commonly practised, in animals, which do not spit, the method is not so feasible. However, milk, manure, urine and tissue (portion of abscess walls, udder, superficial lymph glands, tubercular growths, etc.), may be subjected to microscopic examination for tubercle bacilli. Of late bronchial slime collected with special devices from the gullet (gullet dipper) and trachea (tracheotomy tube, wire carrying sterile gauze at end) has been used. In cattle this method has proved of service in detecting open cases. In this connection it should be borne in mind that there are many other bacilli which are "acid-fast," and so closely resemble

the tubercle bacilli morphologically and in staining properties, that a differentiation with the microscope is impossible. (See Bacteriology.)

C. *Diagnostic Inoculations.*—The inoculation of experimental animals is always advisable where the microscopic examination has been indecisive. The guinea pig is usually chosen, as it is very susceptible to tuberculosis. In practice, negative results in this animal speak for the absence of tubercle bacilli in the suspected material injected. Positive results are recognized by the formation of a true tubercle which appears not only at the point of injection but spreads to the neighboring lymph glands and internal organs (liver, spleen, lungs). Acid-fast, paratubercle bacilli, on the other hand, induce in guinea pigs merely a local lesion at the point of injection which does not tend to spread from the primary focus. The inoculations may be made subcutaneously, intramuscularly, intramammarily (in nursing females), and intraperitoneally. (See Bacteriology.)

Course.—The course of tuberculosis in animals is chronic. An infected calf may show no clinical symptoms until it has reached maturity or even old age. Too frequently advanced tuberculosis which has led to the near destruction of important organs (lungs, liver) or caused great areas of the pleura or peritoneum to be invaded, is first discovered in the slaughter house. Only in the last stages of the disease, and then usually due to the invasion of secondary organisms (pus cocci), is it possible by the customary methods of physical examination to recognize the disease during life. A sudden generalization of the disease which may follow parturition, an attack of some acute disease, exposure and privation is sometimes noted. It may lead to death in a few weeks. As a rule, however, for months or years following infection the tubercular ox seems in good health. Even cases of open tuberculosis may appear in normal health and condition. When clinical symptoms finally appear their development is slow and the decline of the patient gradual.

Tuberculosis at first spreads slowly through a herd, but with each new victim another source of infection is supplied and the spread becomes more rapid. Finally, but usually

after years, unless something is done to check its advance, a large percentage or the whole herd, irrespective of age, is infected.

Prognosis.—Generally speaking, the prognosis in animal tuberculosis is unfavorable. While undoubtedly in some cases the process never develops beyond a few local lesions, and in others it may become arrested (encapsulement of foci of infection), in the majority of cases the disease progresses continuously and finally leads to clinical symptoms, decline, and death. That the sanitary conditions surrounding the tubercular animal are of influence in the earlier stages of the disease, there is little doubt. It is frequently noted, however, that infected cattle kept under ideal conditions as to light, cleanliness, and ventilation not only do not improve in health, but actually become worse, the disease making recognizable progress in the individual and in the herd. This is especially true if open cases are not eradicated but kept to infect and reinfect their companions.

Treatment.—A medicinal treatment is useless. In man, light, cleanliness, and ventilation, coupled with rest (especially if the patient has fever), have checked or even healed the disease, provided it was not too far advanced. Whether similarly good results are obtainable among tubercular cattle has not been adequately demonstrated. Too few scientifically conducted experiments in this regard have been made. Furthermore, the trouble, expense and danger of treating individual animals would be prohibitive, and when compared with the preventive measures now employed to control and eradicate the disease (see Prophylaxis) become insignificant. Repeated injections of small doses of tuberculin have given good therapeutic results in man. In cattle the expense and trouble of administration make its use prohibitive.

Prophylaxis.—(*a*) Tuberculosis may be kept out of a healthy cattle herd by preventing infected individuals from coming in contact with it. The disease is practically always introduced by a tubercular animal. One open case of tuberculosis may in time infect a whole herd. Therefore, all cattle brought into the herd should be proved free from tuberculosis by the

tuberculin test. (*b*) Secondary to this is to promote a high resistance to disease in the individuals constituting the herd. This may be accomplished by proper breeding, feeding, good sanitary surroundings and an outdoor life. It would be still safer once yearly to test the herd with tuberculin.

Eradication.—Tuberculosis is a disease which is spread practically only by infected individuals. To remove these individuals is to remove the source of infection. While it is true that not all tubercular animals are at all times passing tubercle bacilli and thus spreading infection, in practice any attempt to differentiate in favor of one case of the disease as against another (to determine whether "open" or "closed", is not feasible. A tubercular ox is a menace and constant source of danger to the rest of the herd. It should, therefore, be removed from all contact, direct or indirect, with its susceptible companions.

Generally speaking, there are two methods of eradicating the disease in cattle, both of which are based upon the permanent separation of the diseased from the healthy.

A. *Radical Method.*—Commonly used in the United States, and where the disease has made limited progress. This method consists in testing the entire herd with tuberculin and killing the reacting animals either on the premises, where the carcasses are rendered innocuous, or preferably in a central slaughter house, in which the carcasses are passed upon in regard to whether or not fit for food by competent veterinary inspectors. Subsequently the premises (stables, barns, etc.) are thoroughly cleaned and disinfected. This method is certain in its results but not always applicable. It does away with the necessity of keeping two herds (reacting and healthy), and the rearing of the calves is not so cumbersome as with the palliative methods.

B. *Palliative Methods.*—(*a*) The Bang method. Where the number of animals in a given herd is too great, or it is the desire to preserve valuable blood lines, a less drastic method of control has been suggested. It is as follows: All clinical cases of tuberculosis (lung, bowel, uterus, udder) are taken out of the herd and destroyed. Reacting cattle which show no clinical evidences of the disease are kept separated from

the cattle which do not react to the tuberculin test, and the progeny of the herd is reared on milk which is free from tubercle bacilli, either by feeding it sterilized or allowing the calves to suckle only healthy dams or nurse cows.

(*b*) The Ostertag method differs from that of Bang only in that the original herd is not tested with tuberculin and no separation of the diseased from the healthy is made. Clinical or known open cases are removed from the herd and all calves are reared and kept entirely isolated. Twice a year the herd is inspected by a veterinarian, but not tuberculined, and clinical cases which may have developed in the interim removed. The calves are tuberculin-tested every six months. Reactors are removed or forbidden to be bred. The milk and feces are frequently examined bacteriologically.

Protective Inoculation.—Depending upon the well-recognized fact that most strains (not all) of tubercle bacilli of the human type are little virulent to cattle, attempts have been made to produce immunity against bovine tuberculosis by inoculations with human tubercle bacilli. Other investigators have used attenuated bovine tubercle bacilli or have enclosed the bacilli in vehicles (colloidal sacs) to prevent their being taken up by the organism. A degree of immunity has thus been produced, but it is of short duration, nine to eighteen months, and in practise, as yet, has contributed little toward either the control or eradication of the disease. A certain danger attends inoculating cattle with tubercle bacilli of human type in that they sometimes produce lesions of tuberculosis, or at any rate are so slowly eliminated from the body (may remain alive in the body from two to two and one-half years) that the use for food of the animal so inoculated might lead to infection of human beings. There is a further possibility that the milk of cows so treated might contain human tubercle bacilli. The following methods of bovovaccination are in vogue:

1. von Behring's Bovovaccine.—The vaccine is made by drying tubercle bacilli (typus humanus) in a vacuum and injecting it at intervals into the jugular veins of calves. A marked resistance against subsequent artificial inoculation with either bovine or human tuberculosis was acquired, but it lasted no longer than twelve to eighteen months. To

repeat the vaccination each year would be expensive, and the consequent elimination of bacilli highly infective to man makes the method hazardous.

2. Koch-Schütz Method.—Consists in injecting an emulsion of tubercle bacilli of human type into the veins of cattle. Nearly six months later three cattle so treated were still resistant enough to overcome highly virulent cultures of bovine tubercle bacilli with which they were inoculated. Subsequent experiments showed the immunity produced to be short-lived, not lasting a year.

3. Klimmer's Method.—Two strains of human tubercle bacilli are employed, one which has been attenuated by heating to 52° C., and the other by being repeatedly passed through the salamander. The bacilli used are non-virulent (even to guinea pigs) and passage through animals does not revive their pathogenic properties. The vaccine (called "antiphymatol") is injected subcutaneously (5 c.c.). The injection should be repeated at least once a year. In infected individuals, where it is said to have therapeutic value, it is given every three months. Hygienic measures (separation, removal of open cases, feeding calves sterile milk, etc.), are recommended to accompany the vaccination.

4. Heyman's Method.—Consists in inserting under the skin of cattle a gelatin capsule containing tubercle bacilli (human or bovine). The metabolic products of the bacilli diffuse through the walls of the capsule and are taken up by the lymph, eventually impregnating the whole organism. Immunity is produced and in tubercular animals a curative effect is claimed. Cattle of any age, whether tubercular or not, may be treated by this method (once yearly for durable immunity), for which good results are attested by the originator.

INTESTINAL PARATUBERCULOSIS. JOHNE'S DISEASE.

Specific Chronic Enteritis of the Ox. Chronic Bacterial Dysentery.

Definition.—A chronic, contagious disease of the bowels of cattle, which in the majority of cases leads to intermittent

diarrhea, anemia, cachexia, and death. It is due to an acid-fast bacillus.

Occurrence.—The disease was discovered in 1895 by Johne and Frothingham in Germany. It is not uncommon in the United States. Sporadic outbreaks and enzoötics have been reported from several states. England and the continent of Europe are badly infected. In Switzerland and Denmark it causes considerable losses. It is probably much more prevalent than usually suspected, being confused with other chronic enterites and bowel tuberculosis.

Etiology.—An acid-fast bacillus resembling the tubercle bacillus of avian type. The bacilli are found in the intestinal mucous membrane, and in the mesenteric lymph glands. The germ does not grow artificially unless cultivated on a special medium. It is probably distinct from the tubercle bacillus. (See Bacteriology.)

Natural Infection.—The causal organisms are eliminated with the feces. They enter the body of a susceptible animal *via* the digestive tract. The contagiousness of Johne's disease has been proved by feeding experiments and successful transmission intravenously. This is further confirmed by the practical observation, that when once introduced into a herd it spreads.

Necropsy.—The cadaver is usually emaciated. The lesions are confined to the bowels and mesenteric lymph glands. In typical cases the mucous membrane of the small intestines and occasionally the colon and cecum is greatly thickened (sometimes four-fold) and thrown into folds or convolutions, some of them transverse, some longitudinal, giving the bowel a corrugated appearance. Coating the affected mucosa is a turbid, grayish-yellow, slimy exudate which is readily scraped off. The surface of the folds is usually smooth; the crevices between ragged. Nodules and ulcers do not occur. The mesenteric glands and Peyer's patches are somewhat swollen.

Symptoms.—As a rule, only adult cattle are affected, although occasionally it may attack yearlings or even younger animals. The disease develops gradually and may go on for a year and not be noticed. The most prominent symp-

toms are progressive emaciation and anemia. An intermittent diarrhea is an almost constant symptom. The feces are thin, gruel-like, or watery, and discharged without straining. In a few cases there may be no diarrhea although the emaciation and anemia are present.

The general condition of the patient gradually becomes bad. The animal grows very weak, the appetite capricious, lactation ceases, the hair coat is dull and erect, the eyes sunken, the body thin and wasted.

Diagnosis.—As the symptoms are not especially characteristic, in the absence of a necropsy the diagnosis is difficult. The microscopic examination of the feces and scrapings from the wall of the rectum for the specific bacillus is helpful, but not very reliable, as often the rectum is not involved and from the feces usually only a few bacilli can be obtained which must be differentiated from tubercle bacilli and nonpathogenic acid-fast bacteria.

O. Bang recommends testing the suspected cattle with tuberculin prepared from avian tubercle bacilli. Cattle affected with Johne's disease react to this form of tuberculin somewhat as do tubercular cattle to bovine tuberculin. The post-injection temperatures may reach as high as 105.8° F. As a rule, however, the post-injection temperatures are lower than in tuberculosis. Often associated with the rise in temperature occur constitutional disturbances, such as chill, diarrhea, etc. While tubercular cattle react to avian tuberculin, those affected with Johne's disease do not react to bovine tuberculin. It is recommendable, therefore, to employ both tuberculins in suspected cases, that tuberculosis be excluded. By thus testing and destroying the reactors the disease has been eradicated from a few herds in England.

Course.—The course is prolonged, extending over several months. It seems to be favorably influenced by an open-air life, but close confinement, advanced pregnancy, and parturition affect the course adversely.

Prognosis.—The prognosis is bad. Cases which may be recognized clinically, die in a few weeks to a few months.

Treatment.—No successful treatment has yet been devised. The destruction of the diseased animals prevents further spread.

CASEOUS LYMPHADENITIS OF SHEEP.

CHEESY BRONCHOPNEUMONIA OF SHEEP. PSEUDO-TUBERCULOSIS OF SHEEP.

Definition.—Caseous lymphadenitis is an infectious disease, due to a specific bacillus, which sometimes manifests itself as a subacute or chronic bronchopneumonia and at other times occurs as an affection of the superficial lymph glands.

Occurrence.—The disease appears in the form of an epizoötic in the western part of the United States, where it induces, especially among lambs, a large mortality, over two-thirds of the lambs dying of the disease. It therefore attains economic importance. The disease is not uncommon in Europe, Australia, and in the Argentine Republic.

Etiology.—The cause is the Bacillus pseudotuberculosis ovis, a non-sporebearing, immotile rod which may be easily stained with anilin dyes. It also stains according to Gram. The germ is pathogenic to swine, guinea pigs, and rabbits. Pigeons and fowls are not very susceptible.

Natural Infection.—Infection probably takes place through the digestive tract, although it may enter through wounds (docking, castration), or the unshrivelled navel of young calves. The droppings of infected sheep contain the bacillus in large numbers, and therefore form an important factor in spreading the disease.

Necropsy.—The principal lesions are found in the lungs, lymph glands of the thorax, and the external lymph glands. Throughout the lung tissue numerous small, gray or grayish-green nodules occur which through coalescence may form large, cheesy areas. There is frequently also present an adhesive pleuritis. The corresponding lymph glands may be intact. In many cases the lesions are confined to the external lymph glands, which are enlarged, and when incised, show greenish-yellow, sticky, smeary, cheesy masses often arranged in concentric layers and surrounded by a capsule of connective tissue. Often the caseous mass undergoes partial calcification changing it to a grayish-white, gypsum-like mass. In rarer instances the abdominal organs are

involved, especially the liver, spleen and kidneys, and more rarely the mesenteric lymph glands.

Symptoms.—The symptoms are rather vague. Most cases are discovered in the slaughter house. Where the affection involves superficial lymph glands, tumors appear on the surface of the body, most frequently in the prescapular and precrural regions. They are about the size of an average potato and are not sensitive on palpation. The condition of the sheep otherwise may be normal except that the enlarged lymph glands may interfere with locomotion.

When the lungs are affected the patient shows cough, dyspnea, anemia, emaciation and ultimately cachexia and death.

Course and Prognosis.—The course is prolonged, the disease lasting for weeks or months with a gradual wasting of the patient. The mortality varies but may reach, especially among lambs, as high as 70 per cent.

Treatment.—Medicinal treatment is of no avail. The disease may be presented to a certain extent by a thorough disinfection of the navel immediately after birth and of wounds surgical and accidental. Vaccination has been successfully practised, using an attenuated strain of the bacillus. When feasible the infected droppings should be rendered innocuous by disinfection or removal to fields not used for sheep.

ACTINOMYCOSIS. LUMPY JAW.

Definition.—Actinomycosis is an infectious disease due to a specific fungus and characterized clinically by the appearance of either connective-tissue enlargements, bone growths, or abscesses which usually occur about the head of the animal affected. The disease is not contagious.

Occurrence.—While most common among cattle, actinomycosis occurs occasionally in swine and rarely in horses. Cattle which have access to straw stacks, especially of barley or bearded wheat, are most often attacked. It may happen that a whole herd of steers or heifers running about a straw stack, which serves them for shelter and in part

food, will become victims. Stable-fed cattle are often infected by forage fed in a dry state, but which was grown on lowlands subject to overflow. While clinically the disease is more important to the surgeon than to the internist, it not infrequently affects internal organs (lungs, liver, stomach), and leads to the condemnation of the carcass in the abattoir.

Etiology.—The cause of the infection is a thread-like fungus known as the Streptothrix actinomyces or the Actinomyces bovis. A common name for it is the ray fungus. This fungus grows on various kinds of grasses, particularly on the awns and glumes of barley and related plants, especially when these have grown on bottom lands subject to overflow.

Natural Infection.—Infection takes place through the mucous membrane, usually of the mouth, or through wounds in the skin. Forage infested with the fungus, provided it contains sharp awns or glumes or the sharp cut ends of coarse straw (stubble), easily cuts the mucous membrane introducing the fungus into the wound at the same time. Cattle from the Southwest suffer from eating cactus, the sharp spines of which wound the mouth. Obviously when the mucous membrane of the mouth is edematous and tender as occurs when the deciduous teeth are being shed, the opportunity for infection is greatest. Sometimes the infection enters the alveolus of a tooth leading to the development of disease of the bone of the jaw (periostitis, osteitis, osteomyelitis). If the mucosa of the cheek is infected, either abscesses (young animals) or connective-tissue growths appear about the head. If the tongue is invaded, either a diffuse, connective-tissue proliferation follows or multiple, circumscribed, actinomycotic foci result. In swine sharp-pointed, plant particles may enter the crypts of the tonsils and set up infection. Otherwise swine are most commonly infected through skin wounds. Sows with pendent udders pasturing on stubble fields often suffer from udder actinomycosis, and pigs are infected through castration wounds especially if allowed access to straw stacks. The infection of internal organs may be primary or secondary. Actinomycosis may involve the

udder, spermatic cord of castrated animals, vagina, and *via* metastasis the liver, spleen, muscle, and brain. A generalization of the process is, however, rare.

Symptoms.—CATTLE.—Actinomycosis in cattle usually appears in one of the following forms: (*a*) Enlargements about the head particularly in the neighborhood of the lower jaw (angle and between rami) and in the parotid region—the so-called actinomycoma. (*b*) An affection of the tongue. (*c*) Disease of the lips. (*d*) In the form of growths in the mouth, pharynx, and larynx.

(*a*) In young cattle the enlargement may appear as an abscess which, when evacuated, is found to contain, mingled with the pus, numbers of pale or sulphur-yellow-colored granules (fungi). Such an enlargement presents the characteristics of a subacute abscess. The connective-tissue growths are firm, non-sensitive, movable, the overlying skin partially adherent; their development is slow. In time they undergo puriform softening and perforate at one or more points. Out of the sinuses is discharged a viscid, mucopurulent exudate. If the bone is involved, the infection leads to periostitis, rarefying osteitis and osteomyelitis. The enlargement is hard, non-movable, somewhat sensitive and usually involves one or more of the premolar teeth; it also tends to perforate. At first several openings appear which later merge into one large, crater-like cavity from which issues a foul smelling, discolored discharge.

(*b*) If the tongue is seriously involved, prehension and mastication are interfered with. It will be noted in the earlier stages that the patient does not lick the corners of the manger or feed-box clean, as the tongue is stiff and cannot be protruded sufficiently. There is more or less ptyalism and a gradual decline in the condition of the animal is observed. If proper treatment is not given, the animal may become reduced to a skeleton. On opening the mouth the changes seen in the tongue will depend upon whether the organ is diffusely infiltrated or the process confined to isolated areas. In the former case, the tongue appears larger than normal, is stiff, moved very little, and feels firm or hard. The organ may become entirely useless to the animal

(so-called "wooden tongue"). In the second case throughout the tongue, especially along the dorsum, nodules from the size of a shoebutton to a hickory nut are felt. Occasionally ulceration appears and usually just in front of the dorsal prominence of the tongue. Surrounding the ulcer cavity polypoid growths are often noted. The ulcer may be covered with accumulations of food, hairs, etc., and obscured from view.

(*c*) Actinomycosis of the lips, while common in some parts of Europe, is rare in this country. As with the tongue there occurs a proliferation of the connective tissue especially of the upper lip which becomes firm and rigid and greatly increased in size. In other cases multiple nodules from the size of a pea to a walnut appear in the connective tissue of the lip.

(*d*) Actinomycosis of the pharynx leads to severe dyspnea, dysphagia and swelling in the parotid region, which in some cases is quite marked. The pharyngeal enlargement may be palpated from without (head extended) or through the mouth. In calves firm growths the size of a fist may be palpated in the thyroid region. They produce dysphagia, dyspnea with wheezy respirations, and general unthriftiness; marked rales may be heard on auscultation over the throat.

Diagnosis.—The diagnosis of actinomycosis depends upon finding the ray fungus under the microscope. The lesions described are suggestive. Swellings due to injury might be confused with actinomycomas. However, these appear suddenly, show an inflammatory character and a benign course. Occasionally foreign bodies (bones, shoe soles) may lodge between the teeth and the cheeks, causing a protrusion of the latter which resembles slightly an actinomycoma. Actinomycosis of the larynx or pharynx might be easily confused with tuberculosis. However, the affection of the corresponding lymph glands, which occurs in the latter and rarely, if ever, in the former and the use of the tuberculin test, should suffice for differentiation.

Course and Prognosis.—The course in actinomycosis is chronic, the disease gradually progressing from month to month until it terminates fatally. A few cases which are

mild may recover spontaneously. As a rule, where bone is not involved and the location of the lesion permits of operation, surgical intervention produces a cure; or if taken early and the lesion internal (tongue, pharynx) or external, healing is possible through the use of iodin.

Treatment.—Superficial actinomycomas are treated surgically by extirpation and subsequent cauterization or tincture of iodin applied to the wound. Bone enlargements are usually incurable. Tongue and throat lesions and inoperable actinomycomas are successfully treated with iodin in the form of iodid of potash. This is administered in doses of $2\frac{1}{2}$ drams per day for each 1000 pounds animal. Each dose of iodid of potash is dissolved in a pint of water and given as a drench, repeated daily for ten days or two weeks or until symptoms of iodism appear (discharge from nose and eyes, peeling off of superficial layers of skin, loss of appetite). The use of the drug is then discontinued until all symptoms of iodism subside, when it may be again administered. It usually requires from three to four weeks to produce a cure. A few animals show no reaction to the treatment. These had best be slaughtered. In addition it is recommended to paint the tumors with tincture of iodin or to inject into them Lugol's solution. With the iodin treatment on the average about 75 per cent. of the cases recover.

GLANDERS. MALLEUS.

Definition.—Glanders is a contagious, usually chronic, infectious disease of horses, asses, and mules. It is characterized by the formation of nodules which tend to degenerate and form ulcers in the mucous membranes, skin, and internal organs, especially the lungs. The disease occasionally attacks man and carnivorous animals. Sheep and goats may be inoculated artificially.

Occurrence.—Glanders is generally distributed throughout the world. It is commonest in cities or on the ranges where large numbers of horses are congregated together, giving it greater opportunity for spread. In the United States it is especially common in the larger cities, and has occurred on

the ranges in the Northwest. As glanders is a local disease in its incipient stages, presenting no clinical symptoms, and usually takes a chronic course, horse owners and persons ignorant of its character not only resist efforts to eradicate the disease, but disregard its contagious character. It is not uncommon in the United States to find glanderous horses housed, fed, watered, and even worked with healthy horses. Through this neglect, glanders is probably more widespread in this than in any other country in the world. Scandinavia and Australia are free from it.

Etiology.—Glanders is due to the Bacillus mallei, a straight or slightly curved, aërobic bacillus, which has a characteristic growth on potatoes and is essentially an obligatory parasite.

Natural Infection.—Susceptible animals are infected with glanders: (*a*) Through the digestive tract with the food and water which has been contaminated with the discharges (nasal, farcy-bud) or more rarely with manure and urine of glanderous animals. (*b*) Through skin wounds. Infection through skin wounds is very rare. It may follow the use of an infected harness which rubs and chafes the skin. (*c*) Through the respiratory tract. It is exceedingly uncommon for glanders to be transmitted in this way, especially if the mucous membranes are intact. The inhalation of the moist spray coughed or sneezed out by a glanderous patient is not a common occurrence; in the dry state the glanders bacilli have a very low virulency. At any rate, primary nasal and lung glanders are exceedingly rare forms. (*d*) By the act of coitus. Occasionally instances of transmission of the disease from an infected stallion to a mare through copulation are recorded.

Glanders is nearly always introduced into a stable through an infected individual, usually a horse suffering from chronic pulmonary glanders, and which shows no symptoms of either nasal or skin glanders. From this animal it usually spreads to the ones next adjacent or sometimes to animals farther removed in other parts of the stable. When the horses are permitted to drink out of a common trough or fed out of a common crib, the infection spreads more rapidly than

under opposite conditions. It is a notorious fact that an apparently sound horse may infect a large number of horses with which it comes in direct or indirect contact. Dealers' stables and livery barns may be more or less permanently infected. Strange horses brought there to be fed or watered are thus exposed to the infection. Public watering troughs are particularly dangerous in this regard. The horse is not as susceptible to glanders as the ass or the mule. In fact, horses offer a remarkable resistance to infection, the disease in them usually assuming a chronic form, and sometimes ending in recovery. In asses glanders usually takes an acute course with rapid and fatal termination. The mule in this regard seems to stand between the two. Obviously anything which will reduce the resistance of the horse, such as overwork, poor food, exposure to weather, etc., will render the animal more susceptible.

Symptoms.—Following natural infection, weeks or months may elapse before clinical symptoms appear, although during this time the patient may show an occasional rise in temperature. In not a few instances prominent clinical symptoms never occur during the life of the patient.

The period of incubation is usually placed at two weeks.

For convenience it is customary to classify glanders from a clinical standpoint as: (*a*) Nasal glanders; (*b*) skin glanders[1]; (*c*) pulmonary glanders. In this connection it is well to bear in mind, however, that any two or all three of these forms may be combined. Further, it is very rare not to find pulmonary glanders present either alone or associated with skin and nasal lesions.

Nasal Glanders.—The first symptom noted is usually nasal discharge, which is very commonly unilateral. The quality and amount of the discharge vary greatly. In chronic glanders it is at first serous or mucoserous; later it becomes more copious, quite viscid, and often mixed with blood. The discharge tends to adhere to the wings of the nostrils, where it dries to form brownish crusts. Coughing or sneezing momentarily augments the discharge, which is rarely odorous.

[1] Skin glanders was formerly known as farcy, which term is now practically obsolete.

The nasal mucosa is swollen, of a leaden hue, and the veins much distended. If low enough down in the nasal cavity, nodules from the size of a shot to a pea may be seen and felt. They are of a gray or yellow color, and often surrounded by a red zone. These nodules soon break down, forming ragged ulcers with a dirty, yellow base. By confluence large, irregular areas of ulceration develop, especially on the septum, but also on the turbinals. In some cases the whole mucosa becomes an ulcerated surface. The favorite seats of the ulcers are the septum nasi, turbinals, and the nostril margins, particularly the internal surface of the internal wing. As the ulcers age, their walls become thickened, bolster-like, and the base paler. Often between the ulcers, peculiar stellate, radiate or irregular, elongated, elevated proliferations of connective tissue appear, the so-called "star-shaped cicatrices." Sometimes they are not associated with ulcers, the latter having healed. With the development of the cicatrices and the disappearance of the ulcers the nasal discharge ceases. Occasionally an ulcer occurs on the apex of the scar. When the mucosa is much thickened, due to the chronic, indurative inflammation, the lumen of the nasal passages is so encroached upon, that pronounced nasal, inspiratory dyspnea with wheezing, blowing sounds, is heard on exercise. Ulceration of the lower part of the nose may extend to the skin of the lips, which becomes swollen. Nodules and ulcers may appear in the swollen area.

The submaxillary lymph glands of the affected side are always enlarged in nasal glanders. At first the glands are diffusely swollen, somewhat hot and tender, but later they become well circumscribed, painless and nodular. In time they adhere to the jaw, the skin over them becoming immovable. With the eruption of fresh nodes or ulcers in the nose the submaxillary glands may show temporary inflammatory symptoms. Spontaneous rupture of the swellings is very rare. Occasionally conjunctivitis and keratitis may accompany nasal glanders.

Skin Glanders.—In skin glanders so-called farcy nodes (farcy-buds) and ulcers (chancres) occur in the subcutaneous connective tissue and skin. The nodes vary in size from a

pea to a walnut. They soon break down forming ulcers. They may appear without infiltration of the adjacent connective tissue, but very commonly a zone of reactive inflammation surrounds them. The ulcers are irregular in shape with ragged edges which overhang the base. The base of the ulcer is usually of a dirty, gray color, although it may be covered by a brownish scab. The discharge may be scant and thin or more copious and thick. In the former case it has an oily appearance and a very viscid consistency. In the latter, a thick pus is discharged which does not adhere readily to the hair. While the ulcers are generally indolent, they not infrequently heal leaving behind small scars which do not entirely become covered with hair.

The lymph vessels in the neighborhood of the nodes and ulcers are frequently swollen, appearing like cords or ridges under the skin. They are usually hot and sensitive. After remaining for a time they may gradually disappear, or along their course ulcers erupt. The superficial lymph glands (inguinal, popliteal, prepectoral) may become swollen, hot and tender. Later they are hard and less sensitive.

In chronic skin glanders there is a tendency for the skin and subcutis of one or more limbs (especially the hind ones) to undergo fibrous thickening (elephantiasis), which ends abruptly at the hoof. Quite often the patient is lame.

Pulmonary Glanders.—Nearly every case of glanders begins in the lungs. As a rule, however, the earlier symptoms of pulmonary glanders are so vague that a diagnosis from physical examination alone is impossible (so-called "occult" glanders). As in tuberculosis of the ox, months may elapse before the infected patient shows clinical evidence of the disease. In the meantime a number of horses may become infected by the unsuspected "occult case." The patient may show occasional cough, which is usually dull, weak, and dry. Less often periodical, slight, nasal hemorrhages (epistaxis) are noted. The horse may tire easily at work and show dyspneic symptoms resembling "heaves." From time to time the temperature may be elevated 1° or 2°. Percussion of the thorax usually gives negative results. In rare cases dulness over an area of the lung may be determined, which

speaks for a superficially located glanders tumefaction at least the size of a double clenched fist. Auscultation is usually negative, although after a smart gallop moist rales are heard in some cases, especially if the ear is placed over the lower end of the trachea. The trachea is sensitive on palpation in individual instances (tracheitis). If the larynx is involved, spasmodic cough and inspiratory dyspnea with stenotic noise are noted. As a rule, in time the general condition of the patient becomes bad. It loses weight, the hair coat appears dull, and the mane and tail hairs become loose. In some cases there is a tendency for passive edemas to develop on the limbs and pendent portions of the body (sheath, udder, ventral part of the abdomen). Following a remission in the course of the disease these dropsical symptoms may temporarily disappear.

Diagnosis.—Provided the clinical symptoms are well developed the diagnosis of glanders is not difficult. The occurrence of the nodules, characteristic ulcers, and stellate cicatrices on the mucous membrane of the nasal cavity, the enlargement of the submaxillary lymph glands, and the symptoms of fever are almost pathognomonic. However, in many cases the lesions are too high up in the nasal cavity (or they may be in the adjacent sinuses) to be seen or felt. In these cases, unless there is a history of glanders infection, or other horses or mules on the premises showing typical lesions, the diagnosis from ordinary physical examination may be impossible.

The skin glanders is characterized by the indolent ulcers which often are not surrounded by a zone of acute inflammation. However, any persistent edematous swelling, nodular thickening or ulcer formation on any part of the body especially under the abdomen, sheath or udder should be looked upon with suspicion.

Differential Diagnosis.—There are a number of diseases producing nasal discharge, lesions on the nasal mucous membranes, swellings of the submaxillary lymph glands, and nodules and ulcers in the skin. At times some of these appear strikingly like glanders. Formerly when a differentiation was only possible by carefully weighing the clinical

phenomena, these diseases were extremely important to consider. However, we have now available several accurate methods of diagnosis. When doubt exists in clinical cases or when there is no clinical evidence of the disease, although the patient has been exposed, the following methods of diagnosis may be employed.

A. *The Mallein Tests.*—Mallein may be employed in several different ways: 1. It may be injected *into* the skin of the eyelid (intradermic, intrapalpebral). 2. Instilled into the conjunctival sac (ophthalmic). 3. Applied subcutaneously (hypodermic). 4. Rubbed into a disinfected area of the scarified skin (endermic). The intradermic method seems to be the most accurate and reliable and has been officially adopted by the armies of the world.

1. The intradermic method consists in injecting a special mallein *into* the skin of the lower eyelid about one-quarter of an inch from the margin and midway between the inner and outer canthus and parallel with the lid. The best syringe for this purpose is of the Luer type with glass barrel and colored glass piston and of either $\frac{1}{2}$ or 1 c.c. capacity, graduated in tenths and holding five to ten doses. The syringe is fitted with a slip needle of 25 gauge diameter and approximately one-quarter of an inch long. The dose is $\frac{1}{10}$ of a c.c. of intradermic mallein. The method of application is as follows: With the left hand encircle the right eye with the thumb and fingers, drawing the skin of the lower lid down tightly with the thumb and allowing the fingers of the hand to be free so as to act as a covering for the eye. With the syringe in the right hand, the back of which is resting against the head of the animal in order to follow any movement, the needle may be introduced *into* the skin from before backwards and parallel with the margin of the lower lid. The free index finger of the right hand is then placed on the end of the plunger and $\frac{1}{10}$ of a c.c. is injected. As the mallein is injected, a small elongated swelling is seen to appear within the skin, which insures that the injection has been made into the dermis and not subdermally. The injection may be made in either eye, the relative position of the operator, assistant and animal remaining the same. While

many animals may be injected with merely a halter on, others must be restrained with a twitch and very obstinate animals placed in stocks.

Reactions.—A small, "puffy," non-sensitive swelling is seen in practically all animals in from two to three hours after injecting. It may remain in a few cases for twenty-four hours or rarely for forty-eight hours. This swelling should not be confused with a typical swelling of a positive reaction.

(*a*) A positive reaction will be indicated at forty to forty-eight hours by a marked swelling and edematous infiltration of the skin and subcutis, both upper and lower lids usually being involved and nearly or completely closing the eye. In some animals the edema will extend downward along the side of the face. The swelling is usually hot, sensitive and pits readily on pressure. In some cases, in addition to the swelling, lacrimation, photophobia and purulent conjunctivitis occur. All typical swellings in positive reactions will remain for from three to four days, disappearing gradually until the eye presents a normal appearance. Some infected animals will show a decided reaction at the tenth or twelfth hour which will increase until it reaches its maximum at the thirty-sixth to forty-eighth hour. Other reactions appear slowly.

(*b*) Doubtful Reactions.—A few animals may show at the time of reading a slight edematous swelling of the lid. These cases should be considered doubtful and retested immediately in the other eye and checked by means of the complement-fixation or other tests.

Reading the Test.—The test should be read for all animals within a period of from forty to forty-eight hours after injection. All animals showing no reaction at this period should be considered free from glanders. All doubtful cases should be removed, carefully observed and examined at least three times during the next twenty-four hours. Animals showing a positive reaction should be considered glanderous and destroyed.

2. The ophthalmic method of using mallein is quite simple. It consists in dropping into one of the eyes of the animal to be tested 3 to 5 drops of concentrated mallein; or the mallein

may be introduced into the conjunctival sac with a camel-hair brush. The reaction usually begins five to six hours after the instillation of the mallein and lasts from twenty-four to thirty-six hours. A positive reaction is manifested by an accumulation of yellow exudate at the inner canthus of the eye to which the mallein has been applied. In some cases the discharge is very slight, in others profuse and usually associated with severe conjunctivitis; at other times the conjunctivitis is absent. The intensity of the reaction is not an index of the extent of the disease. Ordinary mallein used for subcutaneous testing is not adaptable. The Bureau of Animal Industry prepares a special mallein for ophthalmic tests, which contains no glycerin as a preservative. Some experimenters have used dry mallein (mallein siccum). As a rule positive reactions are not attended by fever or systemic disturbances. Some glanderous horses, however, are so hypersensitive to mallein that they give a thermic reaction. It is therefore advisable to take the temperature just before the mallein is instilled and again when the eye is being examined to determine the reaction. When the reaction is doubtful the complement-fixation test may be used as a control. The test may be repeated within twenty-four hours on the same, or control eye. If another retest is necessary it should not be made in less than three weeks.

3. The subcutaneous method is applied as follows:

The normal rectal temperatures of the horse to be tested are first determined one or two days before the injection of mallein is made, best taking them each morning, noon, and evening.

The mallein is injected in doses of 1 c.c. into the side of the neck.

Beginning four to eight hours after the injection, the post-injection temperatures are obtained every two hours until the twentieth hour after injection, and carefully recorded.

Interpretation of Results.—A typical reaction consists in an elevation of temperature of at least 3.6° F., and must exceed 104° F. The temperature curve usually remains at an elevation for some time, or it may make a slight drop and

rise again later in the day. Such a reaction is spoken of as positive. On the second and sometimes on the third day a second temperature curve, though usually less pronounced, may occur.

When the post-injection temperature exceeds the highest pre-injection temperature, 2.7° F. and reaches 103.1° F., or over, and a marked swelling occurs at the point of inoculation, the swelling being hot, sensitive and at least 5 to 10 cm. in diameter, the reaction is positive. The swelling should persist for at least twenty-four to thirty hours.

The reaction is doubtful even if the temperature exceeds 3.6° F. and no local reaction appears.

The reaction is negative when, notwithstanding the height that the temperature may reach, the febrile condition does not last at least four to six hours.

The reaction is negative when the temperature elevation is not more than 1.8° F. and does not exceed 102.5° F.

A positive reaction denotes that the patient is affected with glanders. An atypical reaction indicates that the case should be considered suspicious. A negative reaction denotes the absence of glanders. Cases of doubtful reaction should be retested but not sooner than fifteen days to six weeks following the next previous test.

4. The endermic method of applying mallein is very rarely used. It does not compare in accuracy with the intradermic.

B. *Serum Diagnosis by Means of Agglutination.*—The so-called agglutination test for glanders is a fairly reliable laboratory method in which the serum of the blood of a suspected horse is prepared in various dilutions by means of the addition of physiological salt solution. In order to determine the agglutinating power equal quantities of emulsions of glanders bacilli which have been attenuated by heating at 60° C. (test fluid) are added to the serum solutions. Blood serum which will agglutinate glanders bacilli in dilutions 1 to 1000 or in greater dilutions must be considered as coming from a glanderous horse. Agglutinations occurring only in dilutions ranging from 1 to 500 to 1000 are doubtful. Agglutinations in dilutions of less than 500 indicate the absence of glanders. The test often fails in chronic glanders,

the serum in such cases having a very low agglutinating power. On the other hand some healthy horses possess an agglutinating power as high as that found in some glanderous horses. Healthy horses recently malleinized (within three months) may give a positive agglutination reaction.

Since it is the degree of agglutination and not agglutination itself that determines whether or not infection is present, misinterpretations are unavoidable. When the agglutination test is to be employed, the veterinarian usually only collects the serum under proper precautions and sends it to the laboratory.

C. *Serum Diagnosis by Means of Complement Fixation.*—This is a laboratory method for the diagnosis of glanders which seems to be very accurate. Practically, it is the application to glanders of the Wassermann test for syphilis in man. The test should be made by an experienced manipulator in a properly equipped laboratory. The practitioner usually only collects the serum as in the agglutination test. The results so far obtained from this method are very encouraging (for details see Bacteriology).

D. *Inoculation of Experimental Animals.*—For this purpose a young, male guinea pig is chosen which is inoculated intraperitoneally with an emulsion in sterile water of nasal or skin ulcer discharge from a suspicious case. One to 2 c.c. are injected into the abdominal cavity of the guinea pig. If the bacilli of glanders are present, swelling of the scrotum, followed by adhesion of the testicles, will occur in two to three days. Sometimes only a skin abscess at the point of inoculation appears. The danger of general septicemia may be avoided by keeping the material in a refrigerator for a few days before inoculation. Potato cultures should always be made from the lesions in the scrotum. On potato the glanders bacilli produce yellow colonies resembling honey, while the pseudoglanders bacilli produce white colonies. Positive evidence obtained from this method is of course much more valuable than negative. Occasionally the discharge collected, even though it comes from a glanderous animal, may not contain glanders bacilli. The agglutination and complement-fixation tests have largely superseded this method.

Course.—The course in glanders is very varied. Like tuberculosis of the ox its duration is usually a matter of months or years. The chronic course may be interrupted by acute exacerbations and remissions, until finally the disease assumes a clinical form in which either nasal or skin glanders or both become manifest. The patient either dies or is destroyed. In rare instances death may result from inanition or occur suddenly following pulmonary hemorrhage. The periods of fever which occur during the course of the chronic disease are probably due to the development of fresh foci.

To a certain extent the course depends upon the food and care which is given the patient. Poorly fed, overworked horses more readily succumb to the disease. The nasal discharge which becomes mixed with the food and water and is ingested by the patient produces continual reinfection, which increases the development of the disease.

A few cases of pulmonary glanders and even nasal and skin glanders recover. Such instances, however, are exceptional in temperate climates. It is said that in tropical countries glanders sometimes assumes a more benign form, and that patients showing marked clinical symptoms of the disease ultimately recover. Similar observations have been made in the western United States. They belong to the exceptional rather than the rule.

EPIZOÖTIC LYMPHANGITIS.

JAPANESE FARCY. SACCHAROMYCOSIS.

Definition.—Epizoötic lymphangitis is a chronic, communicable disease of solipeds which manifests itself as a suppurative inflammation of the subcutaneous lymph vessels and regionary lymph glands.

Occurrence.—The disease occurs in Southern Europe, but has also been reported from Finland, Russia, and England. It is common in Asia (Japan, India) and Africa. Whether or not true cases have occurred in the United States is problematical. The ones so diagnosed are probably "sporotrichosis" presenting similar symptoms.

Etiology.—The cause is supposed to be the Cryptococcus farciminosus, large oval bodies very difficult to stain, found in the discharge from ulcers. In the cases which occurred in Pennsylvania bacteriological investigations failed to reveal the cryptococcus but did show the presence of a sporothrix identical with that isolated from man. Evidently the Pennsylvania outbreak, and very probably the other outbreaks in this country, were not identical with the epizoötic lymphangitis first described by Tokishiga (1896) and Pallin (1904) in horses in Japan and India.

Natural Infection.—Evidently occurs through small lesions in the skin. The cryptococcus is probably carried by intermediate agents, such as harness, bedding, stable utensils, etc. It is also possible that insects may be carriers of the infection. The disease is most common in cold, damp weather. Asses and mules seem more predisposed than horses. Cattle are very rarely affected.

Symptoms.—The disease usually first attacks the limbs, particularly the forelimbs, but may also occur on the scrotum or udder or more rarely the body and neck. Usually the disorder originates in a wound or fresh cicatrix. A wound so infected does not heal but is converted into an ulcer with exuberant granulations. From a cicatrix a painful nodule the size of a pigeon's egg forms, which later erupts, discharging a thick, yellow pus. Soon the inflammation involves the lymph vessels, which become swollen, corded, and very painful, and along their course fresh abscesses develop. The abscesses rupture, forming ulcers which heal slowly. The ulcers show a tendency to exuberant granulation, and by confluence are spread and may produce great ulcerous surfaces. The regionary lymph glands are involved in the process; not infrequently abscesses form in them. As a rule the infected limbs swell; particularly about the joints and in the overlying skin superficial ulcers develop.

In rare instances the morbid process may involve the nasal mucous membrane, on which form white nodules and later ulcers which tend to coalesce. The submaxillary lymph glands are involved and may suppurate. Nasal discharge is rare. Usually the appetite and temperature remain normal.

Diagnosis.—The disease closely resembles skin glanders, especially chronic cases. In doubtful instances the usual tests for glanders may be applied. Otherwise a microscopic examination of the pus from a true case of epizoötic lymphangitis will show the characteristic parasites. Ulcerous lymphangitis takes a much milder course and the pus contains the characteristic bacillus.

Course.—The course is chronic. Mild cases last one to two months. Remissions and exacerbations are not uncommon. The mortality varies from 7 to 10 per cent. Patients which recover are usually left with thick legs.

Treatment.—The treatment is largely surgical (extirpation of the nodules, early opening of abscesses, antiseptic treatment of ulcers). Iodid of potash improved but did not cure the condition.

Prophylaxis.—Prevention consists in separating the sick from the healthy, and a thorough disinfection of the premises.

ULCEROUS LYMPHANGITIS OF THE HORSE.

Definition.—Ulcerous lymphangitis is a chronic, infectious disease of horses characterized by a progressive, suppurative inflammation of the subcutaneous lymph vessels along the course of which there form ulcers. The regionary lymph glands are not involved.

Occurrence.—The disease was first described by Nocard in France. No outbreaks have been reported in the United States. A similar disease has been observed in the Philippine Islands.

Etiology.—The disease is due to a bacillus resembling the bacillus of caseous lymphadenitis of sheep.

Natural Infection.—The bacillus evidently enters through small wounds, particularly in the skin of the legs. The disease is not communicable.

Symptoms.—The first symptom noted is a diffuse swelling of the hind limbs. In the swollen limb develop circumscribed, painful nodules which undergo puriform softening forming ulcers with thin borders from which is discharged at first a creamy, later a thinner pus. The ulcers tend to heal readily,

especially if antiseptics have been applied to them. Usually following the healing of the first ulcers a new crop of nodules and ulcers form between which the lymph vessels swell to strands the thickness of a finger. Along the course of these swollen lymph vessels continually new nodules and ulcers develop. In this manner the disorder may continue for several months. In rare cases the forelimbs, body, neck and even the head may be attacked, leading to the death of the patient. In some instances the disease takes a chronic course, the nodules and ulcers appearing in the winter, heal during the summer months, to recur again the following winter. The regionary lymph glands, while swollen, do not take part in the suppurative process.

Diagnosis.—The disease resembles in some respects skin glanders. There are, however, differences from a clinical standpoint. The ulcers are not indolent as in glanders but heal readily; the regionary lymph glands are not involved; the nasal mucous membrane is intact, and no reaction occurs to mallein. The pus from the nodules and ulcers contains the short Gram-positive bacillus, which will not grow on acid potato. In guinea pigs a very rapidly developing periorchitis follows intraperitoneal injection, the purulent exudate containing the characteristic bacillus. Compared with epizoötic lymphangitis, ulcerous lymphangitis is a mild disease. In the former the round or oval cryptococci are found in large numbers. The disorder resembles contagious acne, which, however, is found usually only where the saddle or harness comes in contact with the skin.

Treatment.—The treatment consists in washing out the ulcers with antiseptics. If the process tends to spread the nodules may be opened and disinfected. Some cases resist treatment obstinately. Good results are reported from the subcutaneous injection of diphtheria antitoxin (50 c.c. daily).

INFECTIOUS ABORTION OF CATTLE. ABORTION DISEASE.

Definition.—Infectious abortion is a specific inflammation of the mucous membrane of the uterus, which in pregnant

animals leads to an affection of the fetal membranes and often to the premature birth of the fetus.

Occurrence.—Infectious abortion occurs frequently among cows. It is widely distributed, forming one of the commonest diseases with which we have to deal. In many of the eastern states, where cattle breeding is carried on extensively, fully 70 per cent. of the herds are infected. The disease is chronic and does not seem to affect the general health of the pregnant cow. Infectious abortion attains economic importance not only in that it leads to the loss of large numbers of calves, but because of the sterility, retention of afterbirth, chronic uterine catarrh, and diseases of the udder which follow in its wake. It not only destroys the young but may greatly lessen the value of or even extirpate the dam as a breeder.

Etiology.—In cows the disease is caused by the Bacillus abortus.[1] This is a small, non-motile, non-sporebearing bacillus which stains irregularly with anilin dyes but is Gram-negative. In cultures the bacillus is originally anaërobic but gradually assumes aërobic characteristics. It is found in the uterine exudate, fetal membranes, and in the fetus; also in the udder (milk) of infected cows. The germ is highly resistant and may remain virulent in the uterus of infected cows for several weeks (usually three) after they have aborted. The disease is spread primarily by the uterine discharge, fetal membranes, and fetuses of infected cows. Spread through infected milk can also occur.

Natural Infection.—The infection is taken up by the susceptible mother: (*a*) Through the digestive tract, the causal germ contaminating the food and water; (*b*) through the genital organs which may become infected by the stable litter, manure, etc., or by contact with such utensils as buckets, milking stools, ropes, halters, sponges, douching hose, obstetrical instruments, etc. The bull may also be a carrier of infection as he can transmit by coitus the bacilli

[1] The experiences of Theobald Smith, M'Fadyean and Stockman indicate that occasionally bovine abortion may be due to causes other than the Bang bacillus. The former found in a limited number of outbreaks a Spirillum, and the latter a Vibrio. However, in the light of our present knowledge, most bovine abortion is caused by the Bang bacillus.

which have collected on the penis during copulation with an infected female.

Whether the bull is merely a mechanical carrier or the abortus bacillus may proliferate in the seminal vesicles, causing him to become also infected, and therefore a permanent distributor, remains undecided. In a few cases the seminal fluid contained abortion bacilli, but no infection was transmitted to cows served by these bulls, the service taking place on neutral ground.

Experimentally abortion has been induced in pregnant animals by introducing pure cultures of the abortus bacillus into the vagina, stomach, and veins. The disease is practically always brought into a herd through an infected female which has either recently aborted or is eliminating the causal germ in her vaginal discharge and milk. The tendency for cattle owners to dispose of cows which have aborted insures a wide dissemination of the disease. In rare instances the premises may become infected through a contaminated bull to which the cows of the herd have been brought for service. In still rarer instances a very young calf from an infected mother may introduce the infection. That persons, dogs, fowls, and birds of the air may too carry infection from infected to non-infected premises is probable.

Symptoms.—The period of incubation following natural or artificial inoculation is varied. It averages from natural exposure about four months (33 to 230 days). Artificial transmission of vaginal discharge from diseased to healthy cows was followed by abortion in 9 to 21 days. The symptoms which indicate the presence of the disease in a cow herd are: (*a*) A number of cows are dropping their calves prematurely; (*b*) cows which have aborted show an abnormal vaginal discharge, and (*c*) the appearance of symptoms of premature labor especially in heifers.

The first few cases of abortion in a herd may be entirely overlooked as they are apt to happen during the first weeks (five to seven) of pregnancy when the fetus is very small. A given cow may thus abort, be rebred, conceive and abort again without the owner's attention being attracted to the condition. Finally after aborting twice or more times she

may carry to full term and be delivered of a viable, fully developed calf. Such a cow is spoken of as an "immune" in the sense that she will not abort again although she is still diseased and can infect other cows. Abortion seems most common in the fifth to seventh months of pregnancy, usually occurring on or about the 190th day, but varying from the 149th to the 254th day. Quite often before the actual abortion occurs the cow shows prodromal symptoms, such as filling of the udder, edema of the vulva, colostral milk, sinking on each side of the tail root, congestion of the vaginal mucosa and the discharge of a reddish or yellow, odorless, viscid fluid. The expulsion of the uterine contents usually occurs, however, without marked labor pains and the fetus comes dead. After the abortion the placenta is often retained and a vaginal discharge persists. For two or three weeks or longer the discharge is of a dirty, reddish-brown color, odorless or odorous, the flow either continuous or interrupted. In time the discharge usually diminishes. When bred during this period the cow may not conceive. It occurs occasionally that cows, especially heifers, may show all of the premonitory symptoms of abortion, fail to abort and carry to full term.

Necropsy.—On postmortem the uterus appears externally normal. Between the mucosa and the chorion is found an exudate which is fluid to semi-solid and of light brownish-yellow color. The fetus appears normal. In other cases symptoms of hydropsy and mummification of the fetus are present, conditions which can begin in the third month of pregnancy.

Diagnosis.—On account of its great prevalency the diagnosis from the physical signs alone is not difficult. All cases of multiple abortions in a cow herd should be looked upon with suspicion, and until disproved considered cases of infectious abortion. As contributory to diagnosis a bacteriological examination of the vaginal discharge, the uterine exudate, the placenta or the fetus may be made. However, this is rarely feasible in practice. Of late the complement-fixation test has been extensively employed. While this test is not so accurate as the complement-fixation test for glanders, it nevertheless forms a valuable contribution to the diagnosis. In cases of doubtful

reaction (incomplete hemolysis) a retest should be made in four to six weeks. A negative reaction does not necessarily exclude the abortion bacillus as the infection may have occurred so recently that the immune bodies have not yet formed in sufficient amount to bring about the reaction. The agglutination test is also used. It is found that the blood serum of cattle suffering from infectious abortion possesses an average agglutinating value of 1000 and may cause agglutination in dilutions as high as 16,000. In healthy cows the agglutinating value of the serum is rarely above 50. So-called "abortin," which is prepared from the abortus bacillus much as tuberculin is prepared from the tubercle bacillus, has failed to give uniformly satisfactory results. The reaction is a thermic one with which are sometimes associated constitutional symptoms.

Course.—Infectious abortion usually persists in a herd for years. After the first abortions, often overlooked or attributed to other causes by the owner, new cases occur with a few weeks' interval between. Finally the abortions become more and more frequent until a full-term calf is a rarity in the herd. In time, however, the abortions occur at less frequent intervals. Cows which have aborted one or more times carry to full term (become tolerant—so-called "immunes"). Clean cows introduced into the herd may be the only ones to abort. Ultimately, in two or three years, the abortions cease entirely, provided the herd has been kept intact. However, there are exceptions to this rule and not infrequently individual cows fail to produce full-term calves.

Treatment.—Once the disease has gained a foothold in a cow herd, treatment is rarely successful. The reason for this is that the germs of the disease are within the uterus and obviously in the pregnant animal cannot be reached with disinfectants. Very fashionable is the administration of phenol. It may be administered subcutaneously in doses of 10 c.c. of a 2 per cent. solution during the fifth to seventh months of pregnancy or it may be given with the food. A simple method of dosage is to make up a 3 per cent. solution of phenol, giving to each cow daily four ounces of the solution

in the food. In ten days the dose can be increased to sixteen or even twenty-four ounces daily provided this quantity is given in two or three feeds.[1]

The fundamental principles underlying the control of infectious abortion are the same as for the eradication of any infectious disease, *viz.:* (*a*) The separation of the sick from the healthy. (*b*) The disinfection of the premises including the safe disposal of the dead. (*c*) The care of the sick.

(*a*) Cows which are about to abort or have aborted should be removed from the rest of the herd and placed in maternity stalls.

(*b*) The premises should be thoroughly cleaned and disinfected and the dead fetuses and afterbirths rendered innocuous by burning, boiling, or deep burial.

(*c*) As the herd bull may be a transmitter, it is advisable to disinfect his genital organs before and after each service by flushing out the sheath with some antiseptic (lysol 1 per cent.). The long hairs at the end of the sheath should be cut away and the hair around and in front of the sheath's opening removed with clippers. Bulls should be allowed contact with cows only at time of service. Permit service only on neutral ground, ground ordinarily not occupied by cattle.

(*d*) Cows which have aborted and are still discharging should be treated by irrigating with an antiseptic solution (lysol 1 per cent.; creolin 2 per cent.). At first this may be done every other day, later every third day and afterward once or twice a week until all discharge ceases. The cow should not be bred for about ten weeks after she has aborted and not then if still discharging. It is usually advisable to flush out her genital passages just before service, with a bicarbonate of soda solution (2 per cent.).

Prevention.—To prevent the introduction of the disease from the outside all newly purchased, pregnant animals should be isolated until after calving. No cows should be added to the herd with any abnormal vaginal discharge. No bull should be patronized unless he is known to be clean.

[1] If gradually brought up to it cattle will stand enormous doses of phenol. A thousand pound bull was given without injury in one day nearly 2 pounds of phenol divided into four doses.

Aborting herds should not be allowed to exhibit at fairs or cattle shows which is a common way of distributing the virus of the disease.

Cows which have aborted should not be sold but kept in the herd until they become "ceased aborters," unless they fail to conceive when bred. Keeping together the original herd will lead to a more rapid eradication of the disease than if the infected animals are sold and replaced with new, susceptible ones.

Immunization.—Recent investigations point to the probability of immunizing cattle against infectious abortion. Repeated intravenous injections of living cultures in 10 c.c. doses two months before conception, were employed with the result that the fetus was carried to full term. The effect of the vaccination on the animal, however, was not favorable. Later attenuated cultures were used with partial success. In England experiments with 150 c.c. of a virulent culture injected two months before breeding gave encouraging results.

INFECTIOUS ABORTION OF MARES.

Definition.—Infectious abortion of the mare, differing from the cow, is an acute disease, probably a septicemia. It is characterized by the premature delivery of the foal, or, if the pregnancy is completed, by the birth of a colt unable to stand and nurse and which soon dies.

Occurrence.—Infectious abortion of mares was not recognized in this country until 1886. In that year in one county in Illinois twenty-five hundred foals, valued at $150,000, were lost through it. The disease is now widely distributed, but obviously attracts more attention in studs where a large number of foals is lost on a single farm. Isolated cases may escape unnoticed.

Etiology.—According to Ostertag abortion in mares is caused by a Gram-negative streptococcus. From the investigations of Good and of Meyers, substantiated by Schofield, it would seem that the outbreaks in America are produced by a specific bacillus which has been isolated from a number of different studs of aborting mares and one stud of aborting

jennets. The bacillus belongs to Subgroup II of the colon typhoid group and has been named the Bacillus abortivo-equinus.

Natural Infection.—The disease is usually introduced by an infected mare whose discharges pollute food, especially grass. The disease therefore spreads readily among mares on pasture, especially where an aborted mare has soiled the grass with fetal membranes and discharges. The part played by the stallion as a transmitter is a matter of dispute. Obviously intermediary agents, such as grooming utensils, blankets, harness, etc., may carry the infection.

Symptoms.—The period of incubation is about two weeks. The symptoms of the disease are in general similar to those in the cow. During the early stages of pregnancy they may pass unnoticed, as the small fetus, dropped on the pasture, may not be found. Even in the advanced stages the first symptom noticed is usually the aborted dead fetus. In some mares, in the latter stages of gestation, restlessness and colicky pains occur. As the fetus is not usually fully developed, it is expelled easily and as in normal parturition. Occasionally, however, probably due to the weakened condition of the mare, the last stage of parturition is not completed and the fetus is found lodged in the *os uteri* and vagina. In a large stud a few mares will show symptoms following abortion, such as fever, marked depression and vaginal discharge.

The aborted fetus and the fetal membranes usually present pathological lesions. A peculiar "sour" odor emanates from the placenta, and also from the stomach contents of the fetus. The fetal ovaries (testes) are much enlarged, the lungs often hepatized, spleen swollen, the mesenteric lymph glands congested, and petechiæ occur on the serous membranes. Small abscesses may invade the myocardium.

Following the abortion, and often prior to it, a chocolate colored, fetid discharge from the vagina is noted. Retention of the afterbirth is a common sequel, in some cases leading to peritonitis and septicemia. Mares do not usually abort several times in succession. As a rule, a live foal is delivered the following year.

Diagnosis.—The diagnosis is not difficult as usually several mares in a stud are losing their foals, showing vaginal discharge and retained placenta. As a result, many of them are unthrifty, thin and weak. Foals carried the full period of gestation, which are able to stand and nurse, frequently develop knuckling of the anterior fetlock joints, painful joint swellings and diarrhea.

Treatment.—The fundamental principles suggested to control abortion in the cow apply equally well to the mare. A bacterin made of the Bacillus abortivo-equinus injected subcutaneously into pregnant mares in proper and increasing doses has been found harmless. Whether or not it produces immunity has not been demonstrated.

INFECTIOUS GRANULAR VAGINITIS OF CATTLE.

COLPITIS GRANULOSA INFECTIOSA BOVUM.

Definition.—Infectious granular vaginitis is a disease of cattle characterized by catarrhal inflammation of, and the presence of peculiar nodules in the vaginal mucosa.

Occurrence.—The disease is widely distributed in the United States. In some communities nearly every dairy herd is infected. It is also very prevalent in England and on the continent of Europe. As the disorder tends to prevent conception and in some cases induces abortion (?), it attains great economic importance.

Etiology.—The cause seems to be a specific streptococcus which is Gram-negative.

Natural Infection.—The disease is spread by the bull during the act of coitus. It may also be disseminated by contact of healthy with infected cows. Stable litter, utensils, syringes, etc., or even the hands of attendants contaminated with infectious vaginal discharge, are important factors of spread. The disease, therefore, is not confined to cows, but attacks heifers, calves and even males. In the bull the mucous membrane lining the sheath, external surface of the penis, or even the urethra becomes infected which accounts for the rapid dissemination of the disease in a cow

herd or community (community bull). Horses, sheep, and swine are immune to infection.

Symptoms.—The period of incubation from natural infection is usually three to five days. A minimum period of one day is recorded. The first symptom is an acute, purulent colpitis with congestion, swelling, and sensitiveness of the mucosa of the vagina, accompanied by some mucopurulent discharge. Later there appear on the lateral surfaces of the vagina and in the neighborhood of the clitoris a number of small, firm nodules or granules about the size of a hemp seed. At first they are dark red, but later become paler (enlarged lymph follicles). Concomitant with the granular eruption occurs an odorless, mucopurulent, or purulent vaginal discharge, which soils the vulva and the tail, where it dries to brownish crusts. The general condition of the animal is not much disturbed. In about one month the symptoms of acute inflammation subside, the discharge becomes more mucous, and the granules fade to light red or reddish-yellow. The process tends to extend forward in the vagina and may enter the uterus, causing endometritis. Sterility and abortion are apt to result.

Diagnosis.—The recognition depends upon the contagious character of the disease and the characteristic appearance of the granules which do not break down to form pustules or ulcers. In the vaginal mucous membrane of perfectly healthy cows a few enlarged lymph follicles may be found. Therefore conclusions should not be arrived at hastily. In infectious abortion the lymph follicles may also become swollen but as a rule the nodules are larger, more discrete and less numerous. From vesicular exanthema of cattle, infectious granular vaginitis is distinguished by the fact that no vesicles, pustules or ulcers occur, the general condition is not usually disturbed and the bull is also pronouncedly affected (vesicles and ulcers in the sheath and on the penis; mucopurulent discharge from the urethra). Furthermore, coital exanthema occurs also in the horse, sheep, and swine.

Course.—The course is prolonged, the disease usually lasting for weeks or months. As an attack does not produce immunity, reinfection commonly occurs. As a general

proposition, infectious granular vaginitis is a stubborn disease, which, unless treated in the early stages, becomes a most obstinate malady.

Treatment.—Treatment consists in a thorough cleansing of the affected genital passages with disinfectants which are not too irritant. The disinfection of the premises should also be made. Where feasible a separation of the infected from the non-infected should be practised. The vagina may be flushed out with a lukewarm solution of bicarbonate of soda (2 per cent.) or lysol solution (1 per cent.). Vaginal tampons made of gauze impregnated with some antiseptic in fluid, ointment, or powder form are considered more serviceable than mere irrigations, as the antiseptic is kept in longer contact with the inflamed part. Success does not depend, however, so much upon the disinfectant used as the thoroughness of its application. Bulls may be treated as recommended in infectious abortion.

Prevention.—Prevention is accomplished by practically the same means suggested for infectious abortion.

CHAPTER VI.

INFECTIOUS DISEASES DUE TO PROTOZOA.

PIROPLASMOSES.

Definition.—Piroplasms are one-celled protozoa which assume various shapes, some of them pear-, some round-, and some ring-shaped. When introduced into the body of a susceptible animal they enter the red blood corpuscles which cells they destroy leading to anemia, hemoglobinemia, and icterus. Piroplasms are transmitted from the infected to the susceptible animal by insects known as ticks. In the tick they probably pass through an evolutionary stage. The most important pathogenic piroplasms and the piroplasmoses they produce are the following:

(*a*) Piroplasma bigeminum, causing Texas fever.

(*b*) Piroplasma parvum, causing East African fever of cattle.

(*c*) Piroplasma equi, producing biliary fever of horses.

(*d*) Piroplasma ovis, causing so-called malarial fever of sheep.

(*e*) Anaplasma marginale, producing gall sickness of cattle.

Relationship of the Tick to Piroplasmosis.—Animals become infected when on pasture from being bitten by ticks. These insects, of which there are a great many varieties, belong to the group Ixodinæ and the family Ixodidæ. From a pathological standpoint the most important belong to the genus boöphilus and the genus ixodes. The ticks become fully developed either on the animal which serves as host (Texas fever) or they leave the host as nymphs (East Coast fever) or both as larvæ and nymphs (European piroplasmosis). The most important varieties are the following:

(*a*) Ixodes ricinus (European piroplasmosis).

(*b*) Boöphilis (or margaropus) annulatus (American, Australian, East Asian and South European piroplasmoses).

(*c*) Rhipicephalus appendiculatus (East Coast fever).

(*d*) Dermacentor reticulatus (biliary fever).

(*e*) Rhipicephalus bursa (malarial fever of sheep).

Texas Fever.—(*Piroplasmosis of Cattle. Southern Cattle Fever, Tick Fever*).—**Definition.**—Texas fever is a specific blood disease of cattle, due to the protozoön Piroplasma bigeminum, and characterized by fever and hemoglobinuria. The causal germ is transmitted by ticks.

Occurrence.—The disease is indigenous to districts infested with certain varieties of the cattle tick. In the United States Texas fever exists permanently in the southern states. In the North, due to the cold winters which kill the ticks, only sporadic outbreaks take place in the summer season, the infection being carried from the South by ticky cattle. Native southern cattle are tolerant to the disease, but northern cattle brought South are very susceptible. The disease also occurs in Australia, South America, India and throughout Europe (different types). Before the recognition of the tick as a carrier of the infection, and the consequent establishment of a Texas fever quarantine line across the United States, the disease practically prevented the interchanging of northern and southern cattle.

Etiology.—The cause of Texas fever is the protozoön Piroplasma bigeminum. The transmitter of the infection is the cattle tick of which there are several varieties. In the United States the Margaropus annulatus is the carrier; in Europe the Ixodes ricinus, in South America and Australia the Boöphilus argentinus and australis, and in South Africa the Boöphilis decoloratus and appendiculatus.

The Piroplasma bigeminum is found in the red blood corpuscles during the fever stage of the disease. They are pear-shaped or round and usually occur in pairs. Depending upon the number of protozoa introduced, the type of the disease may be severe (acute) or mild (chronic). During the height of the Texas fever season (late in August, early in September) the acute form prevails, while earlier than August and later than September the milder, chronic type is met with.

The blood of an ox containing the piroplasm is virulent when injected into susceptible cattle (subcutaneous, intravascular, intraperitoneally), but feeding such blood fails to produce the disease. The parasite will remain virulent in a recovered animal for years.

How Texas Fever is Spread.—As noted, the cattle tick is the carrier of the causal protozoön. As far as is known it is the only carrier. These ticks are essentially parasitic; they cannot attain full development unless they have access to cattle. Their life history is important as the control and eradication of the tick is naturally followed by a cessation of the disease. The life history is simple: A pregnant female falls to the ground from the skin of an ox and soon lays 2000 to 4000 eggs. Depending upon the temperature and moisture present, in from two to six weeks a fully developed embryo breaks the egg shell and becomes free. When hatched the little seed tick is a very active, six legged, spider-like insect which crawls up the grass blades and lies in wait for an ox to the skin of which it attaches itself. The larval tick can grow only when on cattle. In moist soil, leaves, etc., it can remain alive, however, for many months, even living through the mild southern winters, and be capable of infecting cattle the following spring. In the more rigorous northern winter, however, it perishes. Once attached to the skin, the tick moults, attains sexual maturity, copulates and fills itself with the blood of its host. While withdrawing the blood of the host the causal protozoön is introduced and the infection brought about.

Susceptible cattle placed in tick-infested pastures thus become infected. This is commonly observed when susceptible cattle are turned on the usually tick-infested southern pasture or are placed in northern pastures or enclosures in which tick-laden southern cattle have been grazing or kept. In permanently tick-infested districts the native cattle are wholly or partially immune. If they contract the disease at all, the attack is usually mild. One attack of Texas fever confers a relative immunity, but the blood of an immune animal remains virulent for several years. The disease can therefore be spread by an immune or recovered animal provided the purveying tick is present.

Symptoms.—After a period of incubation of eight to ten days following infestation with ticks, symptoms of fever appear. The temperature ranges from 104.9° to 108° F., the patient is dull, stupid (in rarer instances may show excitement), the muzzle dry and hot and the appetite is impaired or fails. Quite often the patients assume unnatural attitudes when standing or lying. The mucous membranes are icteric, pulse rapid, breathing dyspneic, bowels constipated and the feces, which later become softer, tinged with blood and bile. On the skin of the scrotum (udder), inner surface of the thighs, escutcheon and sometimes over the whole body ticks may be felt and seen. Toward the end of the attack, and especially marked in fatal cases, is the appearance of hemoglobinuria, the urine assuming a claret-wine color. The blood is anemic (number of red corpuscles sinks from eight to less than three million), poikilocytes appear and numbers of pear-shaped or round protozoa can be seen in the red blood corpuscles.

Necropsy.—In acute cases the condition of the cadaver is usually good, but where the course has been protracted it is emaciated. Ticks are found on the skin especially in the region of the scrotum (udder), inner surface of the thighs and escutcheon. The subcutaneous tissue is usually anemic and icteric. The spleen is enlarged, often weighing six or seven pounds and its parenchyma has become a dark, purple colored, disintegrated mass. The liver is enlarged, has lost its natural brown color and is yellow on its surface. When incised it shows a mahogany brown color; from fatty degeneration the color is still lighter yellow. The gall-bladder is distended with flaky bile mixed with mucus of a viscid, stringy consistency. The urinary bladder is filled with red urine the shades varying from pale red to a deep red. The kidneys are hyperemic, the lungs are intact and the serous coverings of the heart show ecchymoses.

Diagnosis.—In the United States Texas fever might be confused with anthrax, blackleg and hemorrhagic septicemia. The presence of the ticks (in non-tick-infested districts) on the skin and the microscopical examination of the blood should clear away all doubt. Clinically the

symptoms of general anemia are very suggestive of Texas fever. In anthrax anemia does not develop. Blackleg attacks only young cattle which show characteristic, crepitant swellings and no splenic enlargement. The course of hemorrhagic septicemia is usually very rigorous and rapid (die in a few hours), no ticks are present, the spleen is not enlarged and microscopically the bipoled bacteria are visible between the blood corpuscles.

Course.—The course of the disease is varied. Acute cases (height of the Texas fever season) die in three to five days. Subacute cases may linger for three weeks and terminate fatally. Chronic cases often recover after weeks of illness. In calves the disease is often benign, ending in recovery. As noted, outbreaks during very hot weather are more malignant than when cooler. Chronic cases are subject to relapses. The mortality is from 5 to 90 per cent.

Treatment.—The patient should be removed at once to a non-tick-infested, shady place, and if feasible, the ticks removed from them (hand-picking, antiparasitic agents). Internal medication avails little. Much recommended is quinin (ʒij to v per os). Intravenous injections of formalin (100 to 500 grams of a 1 per cent. solution), followed by lysol internally (℞—lysol, ʒij; spiritus frumenti, ʒiij; aqua, Oj; M. D. S.; hourly one tablespoonful until urine becomes clear) have given some results. Trypanroth given subcutaneously or intravenously (up to 200 c.c. of a $1\frac{1}{2}$ per cent. solution; make up fresh in distilled water) will reduce the number of parasites but usually only temporarily.

Prevention.—The prevention of Texas fever depends upon the eradication of the carrier-tick. Once a field is rid of these insects, non-tick-bearing cattle may be turned into it with impunity. The task of ridding the United States of cattle ticks is now well under way. In the southern states an area, formerly tick-infested, larger than two ordinary states, has already been freed. Various methods of eradication are in vogue. Which one to employ will depend upon local conditions. The following briefly describes the most practical methods:

(*a*) *Hand Picking.*—When only a few cattle are kept on the farm or in the case of ox teams, removing the ticks by picking, currying or brushing three times a week from May till December effectually protects the pastures from reinfestation with pregnant females and also betters the condition of the cattle themselves.

(*b*) Spraying with crude petroleum or some coal-tar dip (5 per cent.) from May till December serves the same purpose in small herds. Sometimes where no spraying mechanism is at hand, the fluids are applied with sponges, brushes or with a syringe.

(*c*) *Dipping.*—Dipping is the most practical method of ridding animals of ticks. It is recommended that all cattle, and also horses and mules if they harbor ticks, be dipped regularly every two weeks during the warm season of the year and until the ticks have disappeared. If dipping were properly carried out and persisted in, a complete eradication of the cattle tick would follow and with it the disappearance of Texas fever. In many tick-infested districts community dipping vats made of wood or concrete are erected to serve a number of farms. The most reliable solution in which to dip ticky animals is arsenic trioxid combined with sodium hydroxid and sodium carbonate. Pine tar may be added to increase the adhesive properties of the bath which gives greater effectiveness against the ticks and reduces the danger of blistering the cattle. For details as to mixing and application appropriate bulletins should be consulted.

(*d*) "*Soiling Method.*"—The ticky cattle are placed in a tick-free pen for three weeks. At the end of this period they are removed to a second tick-free pen and kept in it a further three weeks. If at the end of this time they are found free from ticks they may be placed on a non-infested pasture. If not, they are returned to a pen for two weeks longer. The success of this plan depends upon the fact that all of the ticks drop off the cattle while in the pens and as the animals are removed to a new pen before a new crop of seed ticks can hatch, no opportunity for reinfestation is offered. Obviously the pens must be thoroughly disinfected before using again.

Freeing Pastures from Ticks.—There are several methods of eradicating ticks from infested pastures.

1. If the pasture be cultivated for one year, and all ticky cattle kept out of it, it will become rid of ticks.

2. Burning ticky pastures each spring and fall will keep them free so long as no ticky cattle are permitted on them in the interim.

3. Early in September the cattle are moved from the infested pasture and cleaned of ticks. They are then placed on a non-infested pasture and all contact with ticky animals prevented. The original pasture is kept free from animals until the following April when it will be free from ticks. In the eight months during which the field has not been used for pasture, the seed ticks which hatch in the fall have died of starvation having had no access to cattle.

4. *Feed-lot Method.*—A field of corn or other forage crop is fenced off into three different enclosures. Around each enclosure a furrow is plowed and a board placed so as to prevent the escape of ticks. The cattle are placed in this field for a period of sixty days, spending twenty days in the first enclosure, twenty in the second and twenty in the third. At the end of this period they are free from ticks, as they were not allowed to remain in any one of the enclosures long enough for reinfestation. In moving the cattle from one enclosure to another they should be driven over plowed ground and after they are taken out the furrow should be sprayed with crude petroleum. Obviously the cattle should not be fed hay nor given water from tick-infested pastures.

Protective Inoculation.—Susceptible cattle shipped to tick-infested regions, especially animals from six to eighteen months old, may be immunized against Texas fever by one of the following methods:

1. The animals are confined in a tick-free enclosure and a small number of (25 to 50) virulent seed ticks placed upon them. A month later a greater number of seed ticks (200 to 400) is used. This will often produce a non-fatal type of Texas fever which renders the animal immune to natural infection.

2. The susceptible young cattle are injected subcutaneously with the defibrinated blood of a native calf or a recovered adult animal. Usually eight to ten days after the

injection the animal develops symptoms of anemia, hemoglobinuria and sometimes bloody diarrhea. Microscopically the blood will show a great diminution of red blood corpuscles and will contain a few piroplasms. In eight to ten days these symptoms temporarily disappear but a month later usually a second reaction sets in which lasts only eight to ten days but is milder in type, the red blood corpuscles showing only a few piroplasms of atypical form. If cattle so treated are two months later turned into infested pastures, a large proportion of them will resist natural infection. Some of them, however, will show symptoms of fever which is usually followed by recovery. The losses from this method of immunization are about 10 per cent. Some animals of low resistance die during the process of immunization. This method seems more controllable than the former one and is now much employed to prevent losses among imported susceptible cattle.

Quarantine Line.—The United States Government has established a quarantine line which extends from the sea coast east of Norfolk, Virginia, across the country to the coast of California near San Francisco. This line is a very irregular one and is varied from time to time as counties just south of it are freed from ticks. Roughly, at present, the line extends through the center of Virginia, westward along the north boundary of North Carolina, through the State of Tennessee, along the north boundary of Arkansas to the center of the north boundary of Oklahoma, where it drops suddenly taking a southwesterly course until it reaches the Mexican line along the north border of which it proceeds westward until it reaches the east boundary of California which it follows north as far as the latitude of San Francisco where it again turns westward to the coast.

Piroplasmosis of European Cattle (*Infectious Hemoglobinuria of the Ox. "Red Water"* [1]).—**Definition.**—Piroplasmosis of European cattle is an infectious blood disease very similar

[1] The "red water" of British Columbia and of the Northwestern United States, according to Hadwin, is not due to protozoa, but to poisoning with oxalic acid from the wet, undrained and infertile pastures. The disorder, which is very fatal, usually occurs only among adult cattle.

to, if not identical with, American Texas fever. The disorder is transmitted by a variety of the cattle tick.

Occurrence.—This piroplasmosis is generally distributed throughout Europe (Germany, Russia, Finland, Rumania, etc.), where it assumes usually an enzoötic form, outbreaks occurring most commonly among cattle on pasture in the spring and summer months. From tick-infested forage, stable-fed cattle are occasionally infected. Cows and yearlings are most susceptible. The disease also attacks sheep and goats. Badly tick-infested pastures are permanent sources of infection, particularly wet woods, pastures and boggy fields which adjoin brush and timber lands. Native calves are more resistant than adults and native cattle withstand the attack better than imported animals. One attack does not produce permanent immunity as the same animals may repeatedly suffer from the disorder.

Etiology.—The cause of the disease is the protozoön Piroplasma bigeminum, which is transmitted by the European cattle tick, the Ixodes ricinus (I. redubius), which is harbored in grass, brush, bushes, etc., especially on low swampy lands. The life history of this tick is somewhat different from that of the American cattle tick. The female does not lay so many eggs and the hatching period is longer (six weeks). The larvæ leave the cattle three to five days after attaching themselves to the skin and on the ground develop into nymphs in about four weeks. They then reattach themselves to the skin of an animal, remain three to five days, drop to the ground and in the following eight weeks develop to sexually mature ticks which again attach themselves to a host, suck its blood and copulate. Therefore, the larvæ, nymphs and sexually developed ticks are capable of carrying the infection. The time which elapses between the laying of the eggs and the dropping off of the pregnant female is about nineteen weeks under average conditions.

Symptoms.—The period of incubation is ten days. The first symptom is high fever (106° F.), which is soon followed by diarrhea. About the second day the characteristic hemoglobinuria appears and the urine becomes red. The shade of red may vary from a light claret wine to a dark tar-like

color. The patient rapidly becomes anemic and the mucous membranes icteric. In some cases there is marked weakness of the hind quarters. The blood of the animal is thin, very dark colored, and its serum is stained red (hemoglobinemia). Microscopically (blood drawn from an ear vein) pear-, round- or rod-shaped protozoa are seen in the red corpuscles with proper staining.

Prognosis.—If the cattle are immediately taken off the infested pastures and all ticks removed from them, recovery usually follows in about two weeks provided the attack has been acute. On the other hand, where the animals are left on the infested fields or where the outbreak has been very severe, the patients die, the disease assuming a chronic form with symptoms of anemia, emaciation, and cachexia.

Treatment.—The removal of the cattle from the infested pastures and the eradication of the ticks (oil dips) are essential. Internally individual patients are treated symptomatically.

Prophylaxis.—Tick-infested pastures should be avoided. Tile draining and tilling such fields are indicated. Where this is not possible, the brush should be cut off and the fields burned over as recommended in Texas fever. Dipping the ticky cattle is indicated.

Protective Inoculation.—The value of protective inoculation (3 c.c. of fresh calf's blood) is still in dispute. In some outbreaks the inoculation material seemed too weak and in others too strong.

East African Coast Fever (*Rhodesian Red Water*).—**Definition.**—East African fever is a form of piroplasmosis in cattle due to the Piroplasma parvum. Contrary to Texas fever it can not be artificially transmitted by blood.

Occurrence.—The disease occurs along the East African coast where it has existed for a long time in a latent form. It has spread into the interior and has caused great losses among the cattle of Transvaal and Rhodesia.

Etiology.—The disease is caused by the Piroplasma parvum, a small, rod-like protozoön. Several varieties of ticks, which in the earlier stages of their development have sucked the blood of infected cattle (Rhipicephalus appendiculatus, R.

Evertsi, sinus, nidens and capensis) are responsible for its spread. The parasite does not pass through the egg as in the case of Texas fever, and the disease cannot be transmitted from immune animals to healthy cattle. The infection takes place only among young cattle when on pasture. Adult animals are immune. As noted, the disease cannot be transmitted by the blood, although transmission has been accomplished to susceptible animals by introducing intra-abdominally large pieces of spleen.

Symptoms.—The period of incubation is ten to twelve days. The first symptoms are those of high fever, salivation, bloody diarrhea, swelling of the lymph glands of the throat, emaciation and weakness. Anemia and hemoglobinuria are usually not present. The appetite of the patient may be retained until the last stages.

Prognosis.—The disease is very malignant, the mortality among young cattle reaching 60 to 90 per cent.

Prophylaxis.—Is similar to that of Texas fever and depends upon the eradication of the tick. The value of blood and serum inoculations is very questionable.

Piroplasmosis of the Horse (*Biliary Fever*).—**Definition.**—The piroplasmosis of horses is an infectious, blood disease which occurs chiefly in Italy, Russia and also in Africa and India. It is due to the Piroplasma equi.

Occurrence.—The disease occurs so far as reported only in the countries noted above. In Russia it is most common among young, native horses which run on low, swampy pastures. Aged horses imported from non-infected localities are, however, susceptible.

Etiology.—The disease is due to a Piroplasma equi, a small, polymorphous parasite found in the red blood corpuscles. The carriers of the infection are several varieties of ticks. In Russia the Dermacentor reticulatus and in Africa the Rhipicephalus Evertsi are the carriers. Besides horses, asses, and mules, zebras and quaggas take the disease. The disease may be transmitted by blood from immune horses. While one attack produces immunity this immunity is readily overcome by anything which decreases the resistance of the animal (hard work, other diseases). Imported horses are much more susceptible than natives.

Symptoms.—The period of incubation is about fourteen days, after which there develops a remittent fever with great heart weakness, rapid pulse, icteric discoloration of the mucous membranes, hemorrhage from the conjunctiva, great mental depression, dyspnea, constipation followed by diarrhea, emaciation, polyuria and yellow discoloration of the urine. Microscopically the protozoa are found in the red blood corpuscles. The course of the disease is very varied. In acute cases death may result in two to five days. In chronic cases the course is two to four weeks or it may extend over many months.

Diagnosis.—From horse-sickness piroplasmosis of horses is usually distinguished by the presence of icterus and the absence of edematous swellings. A positive diagnosis can only be made by finding the piroplasma in the red blood corpuscles.

Treatment.—No successful medicinal treatment has been found. As a prophylactic measure, keeping the animals off infested pastures during the hot months is recommended. The importation of solipeds should be made only during the cold season and confined to adult animals. Protective inoculation with 1 c.c. of infected colt blood seems to be successful.

Piroplasmosis of Sheep.—Definition.—This is an infectious, blood disease of sheep occurring mostly in the bottom lands along the Danube River in Rumania. Low, swampy pastures are also infectious. Following floods the disease is observed to a marked extent.

Etiology.—The cause is the Piroplasma ovis which very closely resembles the Piroplasma bigeminum. The disease is spread by the tick Rhipicephalus bursa. The period of incubation is eight to ten days.

Symptoms.—The symptoms are those of fever, languor, anemia, icterus, hemoglobinuria, hematuria and bloody diarrhea. Death usually results in two to five days. Occasionally the disease assumes a milder form and manifests itself by symptoms of bowel catarrh, fever and anemia. One attack produces immunity. Transmission by blood can be made. The mortality is 50 to 60 per cent. of the adult sheep,

and all young lambs (three to four months) die. Convalescence consumes several weeks.

Treatment.—Internally, sulphate of quinin (grs. viij twice daily) and Glauber salts (℥j to ℥ij) are recommended. Prevention consists in keeping the sheep from infested pastures.

TRYPANOSOMIASIS.

Dourine.[1]—**Definition.**—Dourine is a specific, infectious trypanosomiasis of breeding horses and asses, spread by coitus. It is characterized by two distinct clinical stages, *viz.*, a primary stage which is a local disease of the genital organs, and a secondary stage of general infection, which induces nervous symptoms (polyneuritis), skin lesions, and emaciation.

Occurrence.—Dourine probably originated in the Orient, from where it spread with the Arabian horse to Europe. It is widely prevalent in Russia, Rumania, Spain, and Algiers. The disease has invaded Germany, Austria, France and Switzerland from time to time but vigorous veterinary police regulations have held it in abeyance. The United States has witnessed sporadic outbreaks, the infection evidently spreading from imported European stallions. In 1885 it was reported in Illinois, in 1892 in Nebraska, in 1901 in South Dakota (Pine Ridge and Rosebud Indian reservations), in 1903 in Iowa and in 1911 again in Iowa. Since this date no further outbreaks have been noted. By the vigorous methods of suppression employed by the United States Bureau of Animal Industry, all of the above cited outbreaks were effectually controlled and the disease eventually stamped out. As dourine is a chronic disease, often difficult to diagnose and usually fatal, which spreads readily among breeding horses, its economic importance is great.

Etiology.—The cause of dourine is the protozoön Trypanosoma equiperdum discovered and described in 1896 by Rouget. In the United States the presence of this para-

[1] From the Arabic, meaning unclean.

site was first demonstrated in 1911 (Iowa outbreak) by Dr. John R. Mohler of the Bureau of Animal Industry, which conclusively established the identity of the American with the dourine of Europe, Asia and Africa.

Natural Infection.—Susceptible mares are infected by diseased stallions during copulation, the urethral discharges containing the causal trypanosomes. Likewise a mare suffering from the disorder may infect a stallion while he is serving her. A transmission by the stallion from a diseased to a healthy mare, without the stallion himself becoming infected, also occurs. The trypanosomes penetrate the intact mucous membranes of the genital tract and enter the blood. Insect transmission is probable, but so rare, if it occur at all, as to be negligible. Dogs, cats, rabbits, rats, white mice and sheep have been successfully inoculated. Dogs die in two to three months after becoming greatly emaciated. Rabbits emaciate and die in two to eight months, while white mice succumb to general septicemia in three to five days following intraperitoneal inoculation.

Symptoms.—The period of incubation varies from five to thirty days or longer (probably several months). Following the incubative period the local symptoms of the first stage appear.

Primary Stage.—In stallions there is swelling of the penis first noticed in the glans but later involving the whole organ. The prepuce becomes edematous, but is not sensitive to the touch. The edema may involve the ventral abdominal wall and scrotum. The testes may also swell. From the urethra is discharged a thin, yellow, serum-like fluid which drips away more or less continuously (in European outbreaks the urethral discharge is thicker, more purulent). In three to four days small vesicles appear on the penis. In twelve to thirty-six hours the vesicles erupt, discharging a thin, yellow fluid leaving behind raw ulcers which tend to coalesce with those adjacent. The ulcers heal rapidly but leave behind white, non-pigmented, permanent scars. The stallion may show strangury and increased sexual desire (frequent erections). They may attempt to cover mares, but usually full erection of the penis fails. The preputial and inguinal

lymph glands become swollen. In some cases abscess of the testicle with sloughing has been noted. In mares the earlier symptoms may be easily overlooked (range horses). There is edema of the vulva, constant erection of the clitoris, and a mucopurulent discharge which soils the tail and buttocks. The discharge is similar to that from the penis of the male. In a short time papules, vesicles, and ulcers appear on the external skin of the vulva and on the vaginal mucous membrane. The ulcers, although angry looking at first, heal readily, but leave behind permanent, puckered, pitted scars lighter (white in skin) in color than the surrounding skin or mucosa. The mares show estrum-like symptoms (switch tail, urinate frequently). Sometimes edema of the udder and ventral wall of abdomen is present.

In some cases the above described local symptoms may be mild and remain unobserved. In such cases the general symptoms of the second stage of the disease are noted first and the traces of the local stage then looked for.

Secondary Stage.—The second stage affects stallions and mares alike. Sometimes several months may elapse between stages. Anything which tends to lower the resistance of the patient (exposure, fatigue, breeding) may, however, precipitate the second stage. In European outbreaks the first symptom of the second stage is the appearance of urticaria-like swellings (so-called plaques) of about the size of a silver dollar, usually round or half round in form with the centers depressed. The hairs over the swellings are commonly erect. When punctured, a blood-stained serum exudes from the wound. The favorite seats of the plaques are the croup (near tail root), chest, wall, neck, under the belly and chest. The plaques usually disappear in one to eight days to be followed by a new crop. The alternate appearance and disappearance of the plaques may continue for several months and form a very characteristic symptom of dourine. In some of the American outbreaks, however, no plaques were observed (overlooked?). There is generally pruritus causing the patient to rub and scratch the skin.

Nervous Symptoms.—The nervous disturbance of the second stage consists largely in motor paralysis principally

of the peripheral motor nerve (polyneuritis, perineuritis). The following nerves are most commonly affected: Facial nerve, producing symptoms of unilateral facial paralysis; the (recurrent) inferior laryngeal nerve leading to roaring; the femoral nerve causing symptoms of crural paralysis; the great sciatic nerve to a swinging leg lameness, the limb being dragged; peroneal nerve causing knuckling in the hind fetlock and stumbling behind; the obturator nerve to spreading of the gait behind with abduction of the limbs; oculomotor nerve to paralysis of the upper eyelid; pudic nerve to paralysis of the penis. Naturally not all of these paralyses are noted in any one case, but in practically every case some of them, especially those involving the hind limbs, will be observed. Associated with the peripheral paralysis symptoms of hyperesthesia are often present. Many patients are extremely sensitive when the skin is touched or pricked with a pin. In some cases if the back be stroked, the horse suddenly arches it downwardly and seeks to evade the examiner. Quite frequently, during rest, the animal may show cramp-like contractions of the muscles of a leg, holding the member in the air (as in "straw cramp"). Not infrequently atrophy of paralyzed muscular groups follows. While in stallions the genetic instinct is well preserved, yet on account of the partial paralysis of the penis which prevents complete erection of the organ, they are unable to serve mares.

Spinal paralysis is not a common complication, although some patients become completely paralyzed behind.

An important symptom of the secondary stage is marked emaciation. In some instances the patients emaciate to skeletons, losing fully 50 per cent. of their normal weight and presenting a peculiar tucked up appearance of the flanks. As further symptoms may be noted swelling of the lymph glands in the throat and inguinal regions and decubital gangrene of the skin. Occasionally, nasal catarrh, conjunctivitis, fibrinous pneumonia, arthritis and tendovaginitis, iritis and albuminuria are observed. The temperature often remains normal throughout the attack although an atypical fever is not infrequent. The appetite of the patient is usually well preserved.

Diagnosis.—In isolated cases with uncertain history the diagnosis from the clinical symptoms alone is often difficult. In the United States where no other trypanosome disease among horses is known, the microscopic determination of the Trypanosoma equiperdum is clinching. However, in our climate this protozoön, which exists in the urethral or vaginal discharge, plaques, and edematous swellings, is very scarce and difficult to find. Negative evidence, therefore, in this regard would not be convincing. Of diagnostic value are the plaques, nerve symptoms (paralysis), and the marked emaciation of the patient. All breeding horses and asses showing these should be considered suspects. Animal inoculations (dog, mouse, rabbit) may also be employed. When in the primary stage dourine might be confused with coital exanthema which sometimes looks strikingly like it. However, this is a benign disease, healing readily and leaving behind no permanent scars and is followed by neither plaques nor nerve symptoms. Traumatic lesions of the vagina from excessive copulation (range mares) are characterized by wounds and ecchymoses (purple patches) rather than by vesicles and ulcers. The mallein, guinea pig or complement-fixation tests suffice to differentiate between glanders of the sexual organs and dourine. Paralysis due to infectious anemia, forage poisoning, etc., affects geldings as well as stallions and mares.

Course.—In northern latitudes the course is chronic; in southern usually acute. In some cases the second stage, beginning with the development of the cutaneous plaques, follows closely the first stage. The nerve symptoms (paralyses) may not develop, however, until weeks or months later. The duration of the whole attack may extend over one or more years. Obviously any factor which reduces the resistance of the patient (poor care, insufficient food, breeding, etc.), will shorten the duration. Exacerbations and remissions during the course are common.

Prognosis.—The prognosis is bad. Fully 50 to 80 per cent. of the animals attacked die. A few recover when in the first stage of the disease, the second stage failing to develop. It is doubtful whether cases in the second stage ever recover.

Treatment.—Internal medicinal treatment (arsenic, atoxyl, bichlorid of mercury, intravenous injections of tartar emetic, arsenophenylglycin, cacodylate of soda, etc), has been tried with indifferent success in countries permanently infected with dourine. In the United States no attempt to treat the patients should be made. All affected animals should be killed. In some instances the castration of stallions suffering from the first stages of the disease has been followed by recovery. Apparently cured animals can harbor virulent trypanosomes for months. All suspected and true outbreaks should be promptly reported to the State authorities.

Surra.—Definition.—Surra is a specific, blood disease of Asiatic horses, mules, camels, and dogs, due to the Trypanosoma evansi, transmitted by biting insects. It is characterized by high fever (early stages), edemas of the skin, urticaria, petechiæ of the mucous membranes. Later the patients become anemic, weak, and emaciated.

Occurrence.—Surra exists in Asiatic countries, and is a common disease in the Philippine Islands, where it affects horses and occasionally carabao and cattle causing considerable losses. The disease is most prevalent in swampy districts in the hot months where horse-flies abound.

Etiology.—The Trypanosoma evansi, which is introduced into the blood of susceptible animals by species of horse-flies (Tabanus tropicus, T. lineola, Stomoxys calcitrans). Cattle and zebras, which are only slightly susceptible to the disease, are virus carriers.

Symptoms.—The period of incubation is four to thirteen days. The disease begins with fever (105.8° F.), which lasts four or five days, the hair coat becomes rough, the joints swell, and later edemas appear under the belly and in males in the prepuce. Petechiæ appear in the mucous membranes (eye), followed by paleness, and in the latter stage the conjunctiva presents a marked grayish-white appearance. The fever usually becomes intermittent, the patient very anemic and notwithstanding good appetite, loses flesh rapidly. In the blood the Trypanosoma evansi is found, especially in the beginning of a febrile attack.

Course.—The course is usually one to two months, rarely does death ensue in one to two weeks.

Prognosis.—Bad. Nearly all cases die.

Treatment.—In permanently infected districts, arsenic (grs. x to xv daily for ten days in powder form as a bolus or electuary) has given good results. Atoxyl (℥j to v of a 4 per cent. solution subcutaneously) given at the same time with arsenic (grs. x to xv per os) was less successful.

Prevention.—Ridding the infested districts of fly harbors (cutting brush, draining fields, tillage, cleanliness about stables), pasturing only at night when the flies do not swarm, applying agents to the skin to keep flies away (creolin, petroleum), and encouraging the growth of citronella grass are helpful.

In non-infected localities quarantine should be established against animals from infected countries. The killing and cremation of all infected animals are indicated. By taking the temperatures frequently during fresh outbreaks, making blood examinations and animal inoculations (rats, mice) the animals harboring the trypanosomes may be identified and destroyed leading to the eradication of the disease in districts and countries where it has not yet gained a firm foothold.

Nagana (*Tsetse Disease*).—**Definition.**—Nagana is an African trypanosomiasis of horses and cattle, more rarely in sheep, goats, and dogs, due to the Trypanosoma brucei and transmitted by the tsetse fly. In character it much resembles surra.

Occurrence.—Nagana was formerly widespread in Central and South Africa, but of late years has grown less common (disappearance of virus-carrying game?). Low, swampy, river countries are most infectious.

Etiology.—The Trypanosoma brucei, which is transmitted from animal to animal by the bite of the tsetse fly (Glossina morsitans) or other closely related flies (Gl. fusca, Gl. pallidipes, etc.).

Symptoms.—In horses the period of incubation is two to ten days. The symptoms are high fever (104° to 105.8° F.); congestion of mucous membranes (eye); edematous swelling

of conjunctiva (with profuse lacrimosis), throat, legs, under belly, prepuce (in males); the trypanosomes being found in the blood during the fever stages. Later the fever assumes an intermittent or remittent type, the patient becomes anemic (pale, icteric mucous membranes), emaciated and very weak, especially in hind parts, finally gets down and dies of inanition. The appetite is well retained throughout the disease. In some cases keratitis, corneal abscess, iritis and blindness develop during the attack. Urticaria (plaques) may also be noted occasionally.

Diagnosis.—Depends upon a knowledge of the prevalency of the disease in the district, the symptoms of remittent fever, edemas, emaciation and weakness (in spite of good appetite), and the demonstration of the trypanosomes in the lymph glands, blood and edematous swellings. When the microscopic findings are negative, dogs should be inoculated.

Prevention.—Same as in Surra.

Mal de caderas.[1]**—Definition.**—Mal de caderas is a South American disease of horses due to the Trypanosoma equinum. It is characterized by fever, paralysis of the hind parts, urticaria, edemas of pendent portions of the body, keratitis, anemia and emaciation.

Occurrence.—The disease is found in Brazil, Batavia, Argentine, Paraguay, and on the islands of the delta of the Amazon. Like most trypanosomiases, mal de caderas is found in swampy localities. In infected districts in Argentine the disease plays havoc with the horse industry.

Etiology.—The cause of mal de caderas is the Trypanosoma equinum (T. elmassiana), which is very similar to the Trypanosoma brucei of nagana.

The mode of transmission of the disease from animal to animal is as yet unsolved. Formerly horse-flies (Stomoxys brava), tabanidea and even mosquitoes were accused.

Doubt has been cast upon this assumption by the fact that a wire fence is sufficient to prevent the spread of the disease. It is probable that the disease is transmitted indirectly by the capybara (a large rodent). The capybaras

[1] Mal de caderas, "hip disease," from a prominent symptom.

are said to die in great numbers and are eaten by dogs from which a spread to horses is assumed.

Symptoms.—The period of incubation is about ten days. The most prominent symptom is weakness of the hind parts with loss of coördination, the hind legs being dragged and the fetlocks knuckling over when walking. The patient rapidly emaciates and becomes so weak (paraparesis) that when down, it rises from the ground with great difficulty. When standing, the limbs are spread apart. Finally the patients remain down, show paralysis of the rectum (fecal stasis), or relaxation of the anal sphincter and incontinence of feces. The fever is atypical, urticaria-like swellings appear on the skin, and individual joints become swollen. Edemas of pendent parts of the body are not so common as in surra and nagana. Albuminuria and hematuria are not rare. Conjunctivitis and keratitis are common complications. The appetite is retained to the end. In acute cases the trypanosomes are found in the blood in numbers; in chronic cases their determination is only possible by inoculation of rats and mice.

Course.—The course is usually one to two months; chronic cases may linger for months, the emaciation and paresis developing very gradually.

Prognosis.—Bad. Nearly all cases die.

Treatment.—No medicinal treatment is of value. Trypanroth has been tried in experimental animals (mice) with some success.

Prevention.—As a mode of transmission is still in doubt, well directed preventive measures are wanting. Keeping susceptible horses off low-lying, undrained fields and killing the diseased animals are recommended.

COCCIDIOSIS.

Coccidial Dysentery of Cattle. Red Dysentery.

Definition.—An inflammation of the intestines of cattle due to a coccidium and characterized by a bloody diarrhea.

Occurrence.—The disease is found principally among young cattle on pastures, especially in wet seasons and during the months of June to September. As the disease assumes an enzoötic or sometimes an epizoötic form, affecting a large number of animals, and is not infrequently fatal, it assumes economic importance.

Etiology.—The cause is the Coccidium zuerni, which is found in the feces of sick animals as round or oval protozoa, varying in length from 10 to 25 microns. Under the microscope they are highly light refractive and unstained appear greenish-violet in color.

Natural Infection.—Infection takes place through the digestive tract, the coccidia being taken up with water from pools and swamps and also probably with infested food. Occasionally infection occurs in barns, especially when damp and dirty. Young animals are much more susceptible than older ones which, however, are by no means immune. The disease is rarely seen in calves under six months, as they are seldom exposed to infested pastures. Cattle ranging from six months to two years of age are most often attacked.

Necropsy.—The lesions are usually confined to the large bowel, especially its posterior portion. The mucosa is swollen, congested, often thrown into transverse folds (corrugated), partially denuded of its epithelium, and spotted with hemorrhages. Frequently masses of adherent epithelium hang in shreds from the eroded mucous membrane. The contents are a thin fluid, and vary in color from greenish to reddish-gray or reddish-brown. Sometimes blood clots are mixed with them. Over the mucosa a yellowish or grayish fibrinous exudate is often noted. Due to secondary infection the small intestines may show hemorrhagic inflammation or even necrosis. In the intestinal contents and mucosa (especially in the loose shreds) numbers of coccidia are found. The cadaver is usually anemic and emaciated.

Symptoms.—The period of incubation varies from one to three weeks. A number of young cattle on pasture may be simultaneously affected; more rarely a whole herd may develop symptoms in a single day. The principal symptom is a suddenly appearing diarrhea, the feces thin, copious,

discharged with considerable tenesmus, and after a few hours streaked with blood. If the tenesmus is severe, prolapses of the rectum may result. In adult cattle the symptoms disappear in three or four days, and after showing capricious appetite for a time, recovery follows. In young animals, however, the condition gets worse, the feces becoming very fetid, admixed with clots and shreds of mucus—often ichorous in character. The patients are greatly depressed, have no appetite, rapidly emaciate, and show an uncertain, staggering gait. The pulse becomes increased and the temperature elevated. Death may occur within one to three days.

Diagnosis.—The occurrence of the disease among young stock on pasture, its acute course and the foul hemorrhagic diarrhea with rapid emaciation of the patient are very suggestive. The determination of the coccidia by microscopic examination of the feces clinches the diagnosis.

Course and Prognosis.—The course is acute, lasting from one to ten days. Animals which recover do so very gradually. Individual patients are left with a profound anemia which may lead to death in a few months. Mild cases which assume the type of a simple gastro-intestinal catarrh recover promptly in eighteen to twenty-four hours. The prognosis in young animals is less favorable than in older ones. Obviously weak and debilitated patients succumb more rapidly than those with higher resistance. The mortality is about 5 to 10 per cent.

Treatment.—Treatment is only successful in mild cases. It is important to take the animals from the pasture and place them on dry feed in the stable where they should be provided with pure water. Internally intestinal disinfectants and astringents have been recommended. A mixture of tincture of opium (℥ij), oil of eucalyptus (℥ss), dilute acetic acid (℥ss), mixed with water as a drench, is recommended. The above dose may be given hourly until the symptoms begin to subside, afterward three times daily. Intrarectal injections of tannic acid (1 per cent.) or alum (1 per cent.) have given good results. As the appetite remains impaired for some time the patient may be fed milk and eggs as a substitute for its usual food.

Prevention.—Prevention consists in keeping young cattle away from the polluted water of swamps and stagnant pools. Once the disease breaks out, the cattle should be removed to the stable and placed on dry feed with pure water, or at least to dry pastures provided with a pure water supply. The coccidia in the feces are killed with a 3 per cent. solution of sulphuric acid.

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