JOHN A. SEAVERNS

TEXT BOOK

OF

VETERINARY MEDICINE

BY

JAMES LAW, F.R.C.V.S.

Director of the New York State Veterinary College
Cornell University, Ithaca, N. Y.

VOL. III

DISEASES OF THE NERVOUS SYSTEM—GENITO-URINARY ORGANS—EYE—SKIN
CONSTITUTIONAL DISEASES

ITHACA
PUBLISHED BY THE AUTHOR
1901

PRESS OF
ANDRUS & CHURCH
ITHACA, N. Y.

VETERINARY MEDICINE.

DISEASES OF THE NERVOUS SYSTEM.

Nervous control of bodily functions: affected through disease of nerve centres or nerve trunks, sensory, motor, vaso-motor, etc. Modes of impaired nervous function: objective; subjective. Nervous characteristics in different breeds, individuals, sexes, castrated animals. Nervous disorder from microbian toxins, narcotics, nervous stimulants, etc.

All bodily functions are more or less directly controlled by the nervous system, hence nervous troubles are interwoven with the diseases of all other organs. Disorder of the nerve centres or nerve trunks affects the most distant parts over which these preside, or to and from which they convey nervous impulse. In different cases we see this operating through the sensory or motor functions, through lack of coördination or of balance, through modification of the circulation, respiration, secretion, absorption, nutrition, metabolism, special sensation, intellection, emotion, etc. These manifestations are less evident or less diagnostic in the lower animal, because we cannot fully avail of the subjective symptoms. While the human patient can tell us his feelings and experience in their regular order and succession, we can only infer most of these in the animal, through dependent objective symptoms. In many cases we cannot even infer for lack of these dependent symptoms.

The practitioner must carefully watch for and accurately observe all objective symptoms, and seek to rightly interpret them. Among other things he must note the nervous conformation, organization and susceptibility; the hereditary nervous characteristics as seen in breeds, temperament, habit, aptitude to learn, docility, instinct, intelligence, emotions and affections, and judge the case in the light of these. Similarly he must take into account the hereditary, racial and individual irritability, obstinacy, restiveness, vice, alertness, sluggishness, stupidity, moroseness, and diagnose accordingly. Congestion, anæmia, coma, paresis, paralysis, may result from the nervous disorder and offer valu-

able concurrent testimony to the same. Allowance must always be made for the *use* to which the animal has been put, thus *sexuality* tells strongly in the horse, bull, boar, or ram which has been used for breeding and has become relatively indocile and even dangerous; *food* tells in the horse that "shows his corn", and in the dog fed on flesh; the comparatively untrained English race horse is far less docile than the one inured to saddle or harness and the horse fresh from the range, though previously trained, is far less tractable than the one in steady work. The *sexual products* are especially liable to modify the temper, hence the docility of the gelding, and castrated mare, and the undisturbed life and steady growth and fattening of castrated animals from cattle to capons.

The products of certain diseases and many *drugs* derange the innervation and intellection. Of this we have examples in the hebetude of the victims of milk sickness and dourine, in the wild delirium of rabies, in the varied nervous disorders that attend on the use of narcotics, essential oils, alcohol, chloral, sulphonal, trional, strychnia, lead, phosphorus, arsenic, etc.

GENERAL SYMPTOMATOLOGY AND DIAGNOSIS.

Motor disorders: paralysis, paresis, hemiplegia, crossed hemiplegia, spinal hemiplegia, paraplegia, monoplegia, local palsy, pseudo-paralysis, spasm,—tonic, clonic, tremor, hemispasm, monospasm, spasm of eyeball, spasm of head, paraplegic spasm, general spasms—convulsions, local spasms. Incoordination. Staggering. Reflex action. Morbid reflex: increased reflex, reflex tonic spasm.

It seems desirable to note specially some of the more prominent morbid nervous phenomena and conditions, with lesions or other conditions which cause them, before considering what are usually recognized as special diseases.

MOTOR DISORDERS.

Paralysis (*Akinesis*) is loss of voluntary or involuntary muscular movement through defective innervation.

Paresis is a paralysis which is partial in degree; power of motion is impaired but not completely lost.

Hemiplegia is the loss of voluntary motion in many muscles on one side of the face or body. In general hemiplegia, the following muscles are usually excepted :—muscles of the tongue, of mastication, of the eye, of respiration, of the neck and trunk, generally and of the proximal part of the limbs. The hind limbs are usually most affected, and muscles that are most exclusively under the control of the will those dominated by the cortical centres of the brain. When due to a clot on the brain or degeneration it occurs on the side opposite to that occupied by the clot, on account of the motor fibres crossing at the anterior pyramids of the medulla. Hemianæsthesia is a rare attendant and when present is often on the side opposite to the hemiplegia. Sensory fibres cross in the spinal cord, and the lesion is probably spinal.

Crossed Hemiplegia is motor paralysis of certain cephalic nerves (3d, 7th, 5th, 6th, and 8th,) on the same side with the clot or lesion, and of the muscles of the trunk on the other side. The cranial nerves proceed to muscles on the same side as their origin, while filaments going to the trunk through the spinal cord, cross in the pyramids (motor), or spinal cord (sensory). In crossed hemiplegia, hemianæsthesia is common with both forms of paralysis on one side.

Spinal Hemiplegia has the face and head sound (except sometimes the iris), and half the body paralyzed on the side opposite to that on which the spinal lesion (clot) exists. If anæsthesia exists it is on the side opposite to the lesion and posterior to it—the sensory filaments crossing just before leaving the cord.

Paraplegia is loss of voluntary power of one transverse half of the body ; usually the posterior, and affects the tail, and has coincident anæsthesia, being due to a spinal lesion. Anal and vesical sphincters may or may not be paralyzed according as the lesions implicate their respective spinal centres or not. If there is neither anæsthesia nor vesical paralysis the lesion may be cerebral, in the paracentral lobes of both hemispheres (meso-vertix at the fissure of Rolando).

Monoplegia is a circumscribed paralysis, as of one limb, or on one side of the face, one group of muscles or a single muscle. It may be due to cerebral, spinal or nervous lesion. *Cerebral monoplegias* are distinguished by : 1st, initial spasm ; 2nd, ab-

sence of anæsthesia ; 3d, persistence of nutrition ; 4th, paralysis greatest in the distal portion of the member.

Localized Paralysis is usually due to lesion of a nerve, and is both motor and sensory. If due to a spinal lesion it usually affects one or more groups of muscles. In case the lesion is in the nerve, be guided, in investigating it, by Van der Kolk's law, that the sensory fibres are usually distributed in the skin corresponding to the muscles which receive the motor fibres.

Pseudo-paralysis occurs from muscular disease, injury, inflammation or degeneration and has no appreciable central nor nervous lesion nor anæsthesia.

Spasm (*Hyperkinesis*) ; abnormal violent muscular contractions with or without loss of consciousness.

Tonic (*tetanic*) **Spasm** is violent and continuous.

Clonic Spasm is rapidly intermittent :—Contractions and relaxations.

Tremor (*trembling*) consists in small, intermittent, involuntary contractions.

Hemispasm affects the face, or limbs, or both, on one side of the body and may precede hemiplegia.

Monospasm affects one limb, one group of muscles or a single muscle. It may be due to lesion of the brain, of the spinal cord or of the nerves. Thus it may imply commencing disease of the motor centres or tracts.

Spasms of the Eyeballs (rolling of globe to one side), and **Spasms of the Eyeballs and Head**, are important indications of apoplexy. They imply disorder (commencing irritation) of the cerebral motor areas. Advanced disease would probably determine hebetude, coma, drowsiness, or palsy. If epileptiform it turns away from the lesion. If hemiplegic it turns toward the lesion and away from the paralysis. If lesion of the pons it turns away from the lesion.

Paraplegic Spasm is a tonic spasm, partial in degree, causing stiff, tetanoid (spastic) walk. In all four extremities there may be mixed paresis and contraction. This often attends on hemorrhage into the meninges.

General Spasms, convulsions as in Eclampsia, Epilepsy, Chorea, Tetanus.

Local Spasms may be rhythmic or not, in slight cases to be

seen only in the eyelids or superficial muscles as twitching, and occur in neurasthenia, or in poisoning by strychnia, brucia and other motor nerve poisons.

Incoördination (*Dyskinesis*) is the lack of the harmonious balanced movement of the various groups of muscles. Coördination of movement is due to a special mechanism in the spinal cord, and extending forward through the medulla oblongata, pons, and crura cerebri to the floor of the third ventricle. In the form of *ataxia* (lack of power of muscular control) it is usually the result of degeneration (schlerosis) of the superior columns of the cord, of the medulla, pons or crura. It may occur from degeneration or destructive change in the cerebellum, or from disease or section of the posterior roots of spinal nerves, or finally from the action of certain narcotic poisons (ptomaines, toxins).

Staggering (*titubation*) occurs from lesions of the cerebellum, medulla or pons; also from alcohol, opium, and other narcotics.

Reflex Action. The normal stimulation of different functions, motor, secretory, circulatory, etc., depends on the nerve centres in the spinal cord, which are roused into action by a centripetal impulse derived from a distant part. Thus the balanced contraction of the different muscles which preserves the equilibrium of the body, depends on the apprehension by the nerve centres, consciously or subconsciously, of such contractions (muscular sense), and it is largely under the control of the will. Here three impulses act coördinately: 1st, the afferent impulse from the muscle to the nerve centre; 2d, the efferent impulse from the nerve centre to the muscle; and 3d, the inhibitory or controlling, voluntary impulse from the sensorium to the nerve centre involved. In another case, savory odors, sapid flavors and masticatory movements cause a free secretion from the salivary glands. Again, the scratching of a dog's breast causes him to move his hind limb as if he were himself doing the scratching. Again, the pricking of a limb causes the prompt, even if involuntary, contraction of its muscles to withdraw it from the source of irritation.

Morbid Reflex. Reflex action may be modified in various ways as the result of disease or injury. It may become excessive from irritability of the organ from which the centripetal impulse starts, or of the reflex centre in the spinal cord, or of the muscle or other organ to which the centrifugal impulse is directed, or,

finally, from impairment of, or separation from the inhibitory centre in the cerebrum. It may be impaired or abolished from degeneration or destruction of any of the tissues just named, or of the conducting nerves which connect them to each other.

The contraction and closure of the pupil under light is a reflex act from the retina on the optic lobes, etc., and from these through the motor oculi to the iris. This reflex is lost and the iris fails to contract in : anæsthesia of the retina ; atrophy of the optic nerve ; disease of the optic lobe ; superior (posterior) spinal sclerosis ; disease of the motor oculi ; or disease of the iris.

The lumbar reflex is lost in many febrile states in the horse, so that pinching of the loins fails to produce wincing, and this becomes a test of the active persistence of the disorder.

Encreased Reflex is often noticed when the parts, including the spinal reflex centre, are disconnected from the brain : as in lesions or disease of the cord in front (cephalad) of its reflex centre. Here the cerebral or voluntary inhibition is lost.

Reflex Tonic Spasm of muscles around a diseased or dislocated joint, or of those controlling its action, often affords a valuable means of diagnosis, the possibility of nervous, muscular and tendinous disease being excluded.

TROPHIC SYMPTOMS AND DISORDERS.

Degenerative atrophy, in hæmoglobinuria, laryngeal hemiplegia, neurectomy, nerve lesion, brain or cord lesion, lead poisoning, disuse. Dermatitis, ulceration, morbid secretion, polyuria, mellituria, albuminuria, poisonous milk.

Degenerative Atrophy. From section, disease, atrophy or degeneration of nerves or nerve centres, the muscles, which they normally innervate, waste, often to an extreme degree. As examples of this we see the atrophy of the triceps extensor cruris and other groups in hæmoglobinuria, of the intrinsic laryngeal muscles in *roaring*, of the muscles supplied in neurectomy, and of groups of muscles in myelitis, broken back, lead paralysis, and scapular muscular atrophy. True to the law of wasting of physiologically inert organs, the nerves are atrophied and degenerated, and often also the bones, joints and skin.

The degeneration of an active organ applies to the nervous tissues themselves. According to the law of Waller, the nerve fibre (axis cylinder), when cut off from its nutritive centre (cell body with nucleus) degenerates and ultimately perishes. The axis cylinder is a component part of the neuron, which includes also its continuation in the cell and nucleus, and when the latter, which is the source and origin of both nerve impulse and trophic control, is lost, the inactive axis of the nerve fibre degenerates. This law is now availed of in tracing the distribution of nerve filaments, the degeneration being found in those that have been cut off from their nerve cells while those that come into the nerve trunk from other sources, distal of the injury, maintain their integrity.

In addition to this peripheral atrophy, a degeneration centrad of the injury to the nerve is seen under certain conditions, but especially in intrauterine life. In such cases the atrophy may extend up to and include the central nerve cells, causing a secondary central nervous lesion from an initial peripheral one.

By bearing these laws of nerve atrophy in mind, lesions that would otherwise be obscure, may be satisfactorily accounted for.

Eruptions and Ulcerations of Nervous Origin. Herpes or shingles in man is now recognized as a nervous disease, circumscribed to the distribution of given nerves and occurring unilaterally or bilaterally. Deep-seated dermatitis, vesicles, neuralgia, pain, itching and formication are common accompaniments. The whole is traced to disease of the ganglion on the posterior (superior) root of the spinal nerve distributed to the part. This establishes a principle, and in inscrutable and obstinate, circumscribed skin disease the veterinarian should see if it coincides with the distribution of one or more sensory spinal nerves.

Ulcerations are often caused by the lack of protection of a part after paralysis, thus perforation of the cornea will follow section or disease of the trigeminus These may be prevented by carefully covering the part, and even cured by a fine protective covering like collodion.

Alteration of the Secretions often follow on section of the sympathetic trunks, that of the cervical sympathetic in rabbits causing excessive congestion of the facial skin, with exudation and scabby product, also profuse secretion of sweat, tears, and ear cerumen and dry, scaly skin.

Polyuria is determined by section of one point of the medulla behind the root of the vagus, *mellituria* by puncture between the vagus and auditory nerves (the hepatic vaso-motor centre), and *albuminuria* by a puncture in front of the latter. Impairment of the hepatic vaso-motor tracts in the spinal cord, or of the anterior or posterior cervical sympathetic ganglia, or of the first thoracic ganglion equally determines nervous mellituria.

Poisonous milk produced in hard worked mares, or over-excited dams of other species, causing dyspepsia, diarrhœa, arthritis or other trouble in the suckling, must be in part attributed to nervous disorder.

Practically all secretions and nutrition are largely under nervous control, so that modifications in quantity or quality can often be attributed to nervous influence.

SENSORY SYMPTOMS AND DISORDERS.

Hyperæsthesia, cutaneous, thermic, muscular, visceral. Paræsthesia, pressure on nerve. Anæsthesia, partial, drug. Analgesia. Hyperalgesia.

These are necessarily much less obvious to the veterinarian than to the physician of man. Yet in certain cases they may be observed directly, and in others deduced from dependent symptoms.

Hyperæsthesia is a state of exalted excitability of any part of the sensory nervous apparatus.

Cutaneous hyperæsthesia is that condition in which the slightest touch gives rise to an instant and extreme response. Some nervously organized mares which are dangerously ticklish and irritable, afford physiological examples. The surface soreness and sensitiveness which exist in the febrile chill, in wounds, dermatitis and neuralgia give pathological examples. It is further seen in certain cases of meningitis (cerebral and spinal), spinal irritations, rabies, tetanus and neuritis.

Hyperæsthesia to cold is seen in neuralgia, rheumatism, the early stages of many fevers (chill), in myelitis, neuritis, nerve injuries, and in posterior (superior) spinal sclerosis.

Hyperæsthesia of the muscles may be noted in tetanus, muscular rheumatism and neuralgia.

Visceral hyperæsthesia is shown in many cases of spasms of involuntary muscles (colic, arrest of intestinal calculi, gall stones or urinary concretions), and in inflammation of serous membranes (pleurisy, peritonitis).

Paræsthesia. This is a painful or morbid sensation caused by a lesion in the central nervous structures or in the nerves, but referred by the sufferer to some peripheral organ over which such centre presides. It may even be referred to an organ or part that has been amputated or otherwise removed. This may cause lameness of a kind to indicate suffering in a given muscle, tendon or joint, when the cause is purely central. In dourine, sexual acts are excited which have their real source in the nerve centres. The rabid dog snaps at imaginary flies in mid-winter, when such insects are only phantoms of his brain.

Pressure on a nerve trunk induces sensations of tingling, vibration, formication, heat, cold, and paresis, referred by the mind to the part to which that nerve is distributed, and when the pressure is removed these sensations recede in the order in which they came. This may explain some occult cases of lameness.

Itching may be a pure, persistent neurosis without any skin lesion. Treatment should then be addressed to the nervous system.

Anæsthesia, or absence of sensation, is in its degree partial or complete. The latter is familiar as occurring in parts the sensory nerves of which have been cut across, also in parts the sensory nerve or nerve centres of which have become completely degenerated. There is no response to the prick of a needle, the touch of a hot wire, to pinching or cutting. If the nerve remains intact as far as the spinal centres, reflex action may still occur, but the patient himself has no consciousness of this nor of the injury causing it. Accordingly, he makes no movement of head, ears, eyes, or other parts still dominated by the brain.

In **partial or imperfect anæsthesia** the response to irritation is less marked and may be even delayed. In some forms of central lesions the response to a prick may be delayed two, five, or ten seconds, or even more.

Anæsthesia causes awkwardness or uncertainty of movement, especially if the subject is blindfolded.

Anæsthesia may be **induced by medicine**, as in the general

anæsthesia of etherisation, or the local anæsthesia caused by the topical application of cocaine or carbolic acid.

Analgesia, or insensibility to pain, may be present in cases in which ordinary sensations are still felt. It may be caused by cocaine, alcohol, and to some extent by carbolic acid.

Hyperalgesia is the opposite of this condition, and may be seen in certain irritable conditions of the nerve centres.

PSYCHIC SYMPTOMS AND DISORDERS.

Limitation in lower animals. Effects of age, training, race heredity, individual and racial peculiarities, exhaustion, prostration, dementia, cerebral congestion, compression, degeneration, narcotics, ptomaines, toxins. Controlling absorption in another trouble. Delusions, hallucinations, vice, violence, œstrum, fatigue. Cerebral source of motions.

These have a much more restricted field in the lower animals than in man in keeping with the limitation of the mental faculties, and they may often be traced to demonstrated structural disorder. Yet some emotions of joy, fear or rage run very high and are comparatively unchecked by high mental development or mental training. The effect of training is, however, very marked in the more educated animals.

Age modifies by the sobering that comes from experience and habit. The frolics of puppies, kittens, lambs, foals and calves are in marked contrast with the sedateness and stolidity of old dogs, cats, sheep or cattle.

Training is seen in the educated horse which would have been panic stricken at sight of a locomotive, flag or floating paper, at the smell of a lion or bear, at the sound of a gun or drum, and which will now boldly face any one of these with no manifest tremor. The emotional puppy can be trained to soberly fetch and carry, to drive sheep or cattle without biting, to lie sentinel by his master's property, to point at birds without seeking to catch them, or to carry shot birds without devouring them.

Race heredity comes from the training along the same lines in many successive generations. Thus the more domesticated breeds of dogs (shepherd, poodle, and greyhound are very af-

fectionate; other breeds (bull, mastiff, bloodhound) are lacking in this character. All trained races take naturally to the occupations of their ancestors. Some (horses, cattle and sheep) are easily panic-stricken, (stampeded). Some (turkeys, roosters) are not easily stampeded. Some (skunks), having effective sources of defence, have little fear of man.

Individual and racial mental dullness and torpor must also be recognized. Some are stupid and slow, others alert and quickly responsive. Some horses are not *level-headed* and become uncontrollable in difficult situations. Some dogs are so emotional as to endanger their lives from sudden heart trouble. Some horses, dogs and cats will pine and die when separated from their fellows or human friends. Extreme timidity, or sudden rage may be so marked as to constitute a virtual morbid phenomenon. Sluggish cerebral and mental action may result from *exhaustion*, *prostration*, or *dementia*; also from *cerebral congestion*, *pressure* and *degeneration*; or from *poisoning by narcotics*, *ptomaines* or *toxins* (opium, hyoscyamus, Indian hemp, dourine, milk sickness, etc.). It may come from profound absorption in another object, as when the rabid dog bears whipping without a howl.

Delusions or **hallucinations** are shown in the rabid dog snapping at flies, or attacking his friend or master as an enemy, as well as in other forms of delirium. Narcotics, such as opium, Indian hemp, etc., ptomaines, toxins, and (in dogs) essential oils cause delirium by acting on the nerve centres.

Vice in its various forms may become a genuine neurosis, the animal losing control of its actions.

Violence in the form of self-defence or aggression is seen in mares in *heat*, in bulls or stallions under sexual excitement, in animals roused by inconsiderate whipping, or in bulls looking on scarlet clothing.

Some high-spirited animals, under extreme fatigue from overwork, sometimes become violent but resume their docility under rest and food.

In all cases we must know the normal of an individual animal to enable us to properly appreciate any apparent deviation from the psychic norm. No less essential is it to take into account the environment and treatment of the patient.

With regard to localization of cerebral lesions, Sequin thinks emotions are probably generated in the basal ganglia such as those of the pons and thalami, while inhibition depends on the anterior cerebral cortical convolutions.

DIAGNOSIS, SYMPTOMS AND THEIR IMMEDIATE CAUSES. LOCALIZATION OF LESION IN SPECIAL SYMPTOMS.

Spasm, pain, numbness—irritation. Paresis, paralysis, anæsthesia (constant)—destructive lesions. Both combined—variable symptoms, recurrent. Definite, fixed symptoms—structural lesions, usually progressive. Symptoms, variable as to place, time, subsidence and recurrence—functional lesions. Brain lesions. Pressure on brain—pain, spasm, nausea, dullness, blindness, stupor, coma, palsy. Congestion and anæmia synchronous. Lesions of cortex. Encephalic lesions—hemiplegia, with spasms, increased reflexes, spasms follow cranial nerves, vertigo, apoplexy, epilepsy, dementia, coma, little muscular atrophy, or dermal sloughing. Spinal lesions, paraplegia without spasm, reflex reduced or nil, follow spinal nerves, head symptoms less, much muscular atrophy, bed sores. Sensory and motor tracts, in crus cerebri, respiratory centres—inspiratory expiratory, inhibition. Salivation, sneezing, coughing, sucking, chewing, swallowing, vomiting. Cardiac centres, accelerating and inhibitory. Vaso-motor centre. Spasm centre. Perspiratory centre. Pons. Corpora quadrigemini, crura cerebri. Thalamus, corpus striatum. Cerebellum. Cerebral cortex: in ass; in dog. Spinal lesions: lateral half section; central anteroposterior, vertical section: superior columns: inferior columns: cervical lateral columns: respiratory tract: glycogenic centre: pupillary dilator: cardiac accelerator; vaso-motor, sudoriparons: centre for anal spincter: for vesical sphincter: genital centre: vaso motor and trophic centres: muscular sense tract: superior column and Goll's. Table of phenomena from cord lesions.

In **Irritation** of nervous organs the symptoms (spasm, pain, numbness) are usually *intermittent.*

In **Destructive Lesions** of nervous organs the symptoms (paresis, paralysis, anæsthesia) are usually *constant.*

When **irritation and destruction** are associated the symptoms are variable and frequent. The characteristic symptoms of the two may coexist or succeed each other.

Structural Nervous Lesions have symptoms that are definite

in their area of distribution, nature (spasm, paralysis) and permanency. *Objective Symptoms* predominate and the case is likely to be progressive and fatal.

Functional Nervous Diseases have symptoms of indefinite distribution, variable in character, with intermissions and spontaneous disappearances (as under marked excitement) and subjective symptoms predominate. They may, however, last for a length of time without change.

Localisation of Brain Lesions.

Lesions of the cranial nerves and their superficial and deep centres of origin need not here occupy attention. These may be studied in works on anatomy and physiology. Attention may be drawn rather to the remoter effects of ganglia which affect or control distant action, and to general pressure on the encephalon.

General Pressure on the Encephalon, whether through fracture of the cranium and depression of bone, by acute congestion, by blood extravasation, by inflammatory exudation, or by acute abscess, will cause pain, spasms, nausea, dullness, blindness, stupor and coma. After explusion of the cerebrospinal fluid from the cranial cavity, the increasing pressure compresses the blood vessels, reduces or interrupts the circulation and abolishes the functions in the parts deprived of blood. Thus congestion of one portion of the encephalon is usually associated with diminished circulation in another portion. Disorder in the first may occur from hyperæmia and irritation and in another part from a consequent anæmia.

Destructive Lesions of Cortex of One Cerebral Hemisphere may or may not cause permanent symptoms, as shown by the passage of a crowbar through the front of the left hemisphere, yet the man survived for 13 years and showed no loss of intelligence, his disposition and character alone having changed for the worse. The one hemisphere may by itself sufficiently control mental acts, while the other lies dormant or may even have undergone degeneration.

Diagnosis of Encephalic and Spinal Lesions. The following may be taken as guiding principles:

Encephalic: Hemiplegic or bilaterally hemiplegic grouping of symptoms.

Spinal: Paraplegic grouping of symptoms.

Encephalic: Frequent contracture or spasms of paretic muscles.

Spinal: Paralysis more perfect and continuous.

Encephalic: Reflexes in affected muscles increased: Cerebral inhibition absent.

Spinal: Reflex abolished or reduced in parts the seat of the lesion.

Encephalic: Spasms in areas of distribution of cranial nerves (not spinal.)

Spinal: Spasms and paralysis follow distribution of spinal nerves.

Encephalic: Head symptoms frequent (vertigo, apoplexy, epilepsy, dementia, coma).

Spinal: Relative absence of head symptoms.

Encephalic: Comparative absence of marked muscle atrophy.

Spinal: Atrophy in special muscular groups.

Encephalic: Little tendency to form bed sores.

Spinal: Tendency to form sloughs and bed sores.

Sensory (Æsthesodic) and Motor (Kinesodic) Tracts in Encephalon.

In the crus and above, the sensory tract lies dorso-laterad of the motor tract, forming about one fifth of the crus, and extending upward through a white layer bending inward to form an angle and finally diverging to the different cortical convolutions. The motor tract is mainly contained in the inferior pyramids of the bulb, and constitutes the median two fifths and basal two fifths of the crus. Without entering farther into this subject it will be observed that lesions of the outer layer of the crus and its radiating fibres may cause hemianæsthesia of body or head, including the eye, while lesions of the median and basal layers and radiating fibres induce hemiplegia of the head, tongue, fore limb, hind limb, trunk, etc.

Respiratory Centres, Inspiratory and Expiratory are in the floor of the fourth ventricle between the centres for the vagus and accessory nerves, and are directly stimulated by the CO_2 in the blood. Secondary subsidiary centres are in the optic thalamus, in the corpora quadrigemini both anterior and posterior

pairs, and finally in the cervical spinal cord, so that disorder of respiration may occur from lesions in these points as well as in the main oblongata centre.

Respiratory Inhibition and arrest depend on the vagus, the superior and inferior laryngeal nerves.

The Salivation Centre also lies in the floor of the fourth ventricle and stimulation of the medulla causes free secretion.

The Centres for Sneezing, Coughing, Sucking, Chewing, Swallowing and Vomiting are also seated in the oblongata, so that any one of these phenomena may come from a central irritation. In *bulbar paralysis* the loss of power usually extends from the tongue through the lips, cheeks, jaws, pharynx, larynx, to the respiratory muscles and heart. Coughing may be roused by irritation of the external auditory meatus, liver, stomach, bowels, or generative organs as well as from the air passages.

Cardiac Accelerating and Inhibiting Centres are both present in the bulb, the latter receiving its afferent impulse mainly through the vagus nerve. Stimulation of the vagi, anæmia of the bulb through decapitation or through tying both carotids, hyperæmia through tying of the jugulars, a venous state of the blood, and blows on the abdomen all slow or arrest the heart action. Digitalis or muscarin has a similar effect. The heart action is accelerated by febrile and inflammatory affections, by a high or low temperature by section of the vagi, by sipping of cold water, by atropine or curari, and by salts of soda. Potash salts on the other hand restore the inhibitory action of the vagi and lower the heart's action.

The Vaso Motor Center is also in the oblongata and the contraction of the vessels with increase of arterial pressure may ensue from afferent currents in the sympathetic nerve and many sensory trunks. The varying activity is seen in blushing, in the congestion of mucous membranes under rage or excitement, in the capillary contraction in the early stage of inflammation, in the second stage of capillary dilation, in angioma or nævus, and in extensive congestions and hæmorrhages in different organs. The arrest of bleeding under fainting is due largely to the anæmia of this centre.

A Spasm Centre the pricking of which causes general convulsions lies in the medulla oblongata at its junction with the

pons. This is excited by excess of carbon dioxide in the blood, by suffocation, drowning, by anæmia of the bulb from bleeding or ligature of the carotids, by venous congestion after ligature of the jugulars, or by the direct application to the part of ammonia carbonate, or salts of potash or soda. It may also be roused by afferent nervous currents from different peripheral parts (spinal cord, sciatic nerve, etc.).

A Perspiratory Centre is found in the medulla, on each side, which may be roused into action by diaphoretics (opium, ipecacuan, tartar emetic, Calabar bean, nicotin, picrotoxin, camphor, pilocarpin, ammonia acetate, etc.).

The Pons like the medulla is at once a ganglionic and conducting organ, and its lesions may lead to arrest of nerve currents generated above or below it, or to the failure to develop currents in its own centres. Stimulation of its superficial layers may be without effect, but if this is carried into the centre epileptiform convulsions ensue. Lesions of one side of its posterior half cause facial paralysis on the same side and motor and sensory paralysis on the opposite side of the body (crossed hemiplegia). Lesions of one side of its anterior half cause paralysis in both face and body on the same side. This depends on the crossing of the fibres midway back in the pons, which cross again in the medulla (motor fibres) and in the spinal cord (sensory fibres). Lesions of the pons are liable to interfere with the functions of the trigemini, the oculo motor and the superior oblique, and to determine epileptic movements and loss of coördination of sensorio-motor movements. Lesions of the superficial transverse fibres (median cerebellar peduncles) tend to cause involuntary movements to one side.

Lesions of the Corpora Quadrigemina cause disturbance of vision, failure of the pupil to contract to light, blindness, paralysis of the oculo-motor nerves, and lack of coördination of movements. Stimulation of one anterior corpus causes rolling of both eyes to the opposite side, with, if continued, a similar movement of the head and even of the body (horse in mill, or index motion, or rolling on its axis).

The Crura Cerebri are conducting bodies but contain also different nerve centres. Lesions of one crus cause violent pain and spasm on the opposite side of the body, followed by paralysis.

The oculo motor may be paralyzed on the same side, but the face and tongue on the opposite side, owing to the fibres crossing in the pons. There may be turning movements.

The Optic Thalamus transmits sensory currents to the cerebral cortex. Lesions in this organ cause sensory paralysis on the opposite side of the body. Afferent currents that do not traverse the thalamus cause reflexes only. It contains one of the roots of the optic nerve and its destruction will impair vision. Its injuries may also produce turning movements.

The Corpus Striatum transmits motor currents originating in the cerebral cortex. Lesions of its interior (lenticular nucleus) cause motor paralysis and sometimes anæsthesia on the opposite side of the body. Electrical stimulation of this nucleus causes general muscular contractions of the opposite side of the body. Irritation of the surface layers is painless and symptomless.

The Cerebellum has been long credited with coördination, and Flourens, after its removal from a pigeon, found an utter lack of harmonized movement in walking, springing or balancing. Luciani removed the organ from a bitch and, after full healing of the part, found a lack of muscular tone (a cerebellar ataxy), so that no great muscular effort could be satisfactorily accomplished. After months, marasmus set in and proved fatal. The lack of coördination is especially connected with lesions of the vermiform process, those of the posterior portion causing falling forward and those of the anterior portion, falling backward. Injury to the middle peduncle on one side causes turning or rolling to the opposite side. Under slighter injuries there may be only unsteadiness and staggering like a drunken man. Nausea and vomiting, with more or less stiffness of the neck or oposthotonos, may be present. Rolling of the eyes or squinting may occur.

Focal Cortical Centers of the Cerebrum. Cortical Localization. Much has been done experimentally and by observation of morbid lesions to locate functions in the different convolutions, and though the subsidiary implication of adjacent and interdependent parts interferes with a perfectly confident diagnosis, yet certain fundamental facts may be borne in mind as contributing to a satisfactory diagnosis.

Arloing, on the basis of his own experiments and those of his

predecessors, gives the following as applicable to the equine (ass) brain :

1. Stimulation of the origin of the front part of the first frontal convolution, or of the anterior part of the pre-Sylvian convolution, causes approximation of the feet on the opposite side of the body.

2. Stimulation of the superior part of the first frontal convolution or of the superior part of the post-Rolandic convolution causes closure of the jaws and diduction.

3. Stimulation of the anterior end of the upper orbital convolution, or of the anterior part of the pre-Rolandic convolution, leads to movements of the nose and upper lip.

4. Stimulation of the antero-superior part of the lower frontal convolution, or the union of the post-Rolandic with the Sylvian convolution causes movement of tongue and jaws.

5. Stimulation of the union of the vertical and horizontal parts of the orbital convolution or frontal lobe, causes opening of the jaws and bending of head and neck.

6. Stimulation in the front of the union of the frontal and longitudinal convolutions, or at the union of the Sylvian and second parietal convolution causes rolling of the opposite eye.

7. Stimulation of union of the frontal and parietal parts of second parietal convolution leads to closure of both eyelids or, with a strong current, of lids on both sides.

8. Stimulation of the second parietal convolution, above and a little behind the extremity of the Sylvian fissure, causes opening of the eye and adduction of the ear on the opposite side, or, if a very strong current, on both sides.

9. Strong stimulation of the posterior part of the first and second parietal convolutions causes tonic convulsions.

10. Currents through the posterior parts of the third and fourth parietal convolutions gives similar convulsions with violent trembling of the trunk and members.

In the **Dog's Brain** localization is easily made by reference to the crucial fissure which passes outward, right and left, at right angles with the longitudinal fissure about the junction of its anterior with its middle third. Also by four parietal convolutions which run backward from near the crucial fissure, parallel with the longitudinal fissure. They are counted from without inward.

Fritsch, Hitzig and Ferrier have mapped out the following motor areas:

1. The convolution in front of the outer end of the crucial sulcus controls the muscles of the neck.

2. The bend of the same convolution backward, opposite the outer extremity of the crucial sulcus, controls the extensors and adductors of the fore limb.

3. The convolution just behind the outer end of the crucial sulcus controls the muscles which flex and rotate the fore limb.

4. The same convolution behind the middle of the crucial fissure controls the movements of the hind limb.

5. The second convolution back of the crucial fissure controls the muscles of the face.

6. The anterior part of the internal (4th) parietal convolution, just back of the crucial fissure, controls the lateral switching movements of the tail.

7. The posterior angle of the first post-crucial convolution causes retraction and abduction of the fore limb.

8. The outer end of the first post-crucial convolution, directly behind the outer end of the sulcus, causes raising of the shoulder and extension of the fore limb.

9. The anterior end of the third parietal convolution (the second from the longitudinal fissure) controls closure of the eyelids, the rolling of the eyeball upward, and narrowing of the pupil.

10. Stimulation of the anterior end of the second parietal convolution causes partial opening of the mouth with retraction and elevation of its angle.

11. Stimulation of the point of union of the first and second parietal convolutions anteriorly causes opening of the mouth with protrusion and retraction of the tongue.

12. Stimulation of the median part of the second parietal convolution, causes retraction and elevation of the angle of the mouth.

13. Stimulation of the convolution directly in front of the outer end of the crucial sulcus causes dilatation of the eyelids and pupil while the eyes and head are turned toward the opposite side.

14. Stimulation of the convolution behind the crucial fissure causes contraction of the muscles of the perineum.

15. Stimulation of the convolution in front of the crucial fissure, on its anterior and sloping portion, causes movements of the pharynx and larynx (swallowing).

16. Stimulation of motor areas of the cortex, by scraping, irritation, or disease tends to produce spasmodic contractions of certain groups of muscles (Jacksonian Epilepsy). Strong stimulation may cause general epileptiform spasms, which are at first tonic, then clonic. One such seizure strongly predisposes to a second. If, during an attack, the cortical centres presiding over a special group of muscles were sliced off, such muscles relaxed, though the general spasms in the other muscles continued.

Localizations of Spinal Lesions.

Being at once a conductor between the brain and nerves, and a reflex nerve centre, we must consider both rôles in seeking to locate lesions from symptoms. In passing from the nerves to and from the sensorium both sensory and motor currents cross so that one side of the brain presides over the other side of trunk and limbs. This crossing of the motor fibres takes place in the medulla oblongata, while that of the sensory fibres occurs in the spinal cord close in front of the nerve from which they have entered.

Cross-Section of one lateral half of the spinal cord therefore causes motor paralysis and rise of temperature of the whole of that side of the body posterior to the lesion, while it induces sensory paralysis and cooling on the opposite side of the body up to the same point. A very limited sensory paralysis on the same side occurs corresponding to the few sensory fibres passing outward obliquely through the portion injured by the cross-section.

A vertical section of the cord separating the one lateral half from the other does not necessarily affect the motor currents, while it produces a limited anæsthesia on each side in the area of distribution of the nerves, the sensory fibres of which crossed in the seat of the lesion.

Transverse section of the superior columns causes hyperæsthesia and lack of coördination.

Transverse section of inferior columns, or of the inferior horn of gray matter, if close behind the medulla, causes no

motor paralysis, but if farther back induces motor paralysis on the same side of the body.

Transverse section of the cervical lateral columns causes motor paralysis of the lateral walls of the chest (respiratory tract). If the section is made in the dorsal or lumbar region it is the same as lesion of the superior columns.

Among **reflex centres** in the cord the following may be named:

The **Respiratory Tract** in the cervical lateral columns just referred to.

A **Glycogenic Centre** in the anterior cervical section between the bulb and the fourth cervical nerve.

Centres which Dilate the Pupil between the fifth cervical and the sixth dorsal nerve.

Cardiac Accelerator Centres between the three last cervical and the five first dorsal nerves.

Vaso-Motor Sudoriparous Centres in the central gray matter.

Centre for Anal Sphincter between the sixth and seventh dorsal nerves.

Centre for Vesical Sphincter between the third and fifth lumbar nerves.

Genital Centre, opposite the first lumbar nerve.

Vaso-motor and Trophic Centres are found in the inferior horns of gray matter, and their degeneration causes progressive muscular atrophy.

The **Muscular Sense Tract** is located near the surface of the superior columns, so that a certain amount of incoördination and unsteadiness of progression follows its destruction.

The **Deeper Part of the Superior Columns** and the **Column of Goll** which bounds the superior median fissure control muscular sense and coördination, and their disease (posterior lateral sclerosis) entails locomotor ataxy.

TABLE SHOWING PROMINENT PHENOMENA FROM LESIONS OF THE CORD.

	Lesions in		
	Cervical Region.	Dorsal Region.	Lumbar Region.
Paralysis in	Neck muscles; diaphragm; trunk; limbs	Dorsal, abdominal and intercostal muscles. Paraplegia.	Paraplegia.
Sensation	Local hyperæsthesia in fore limbs. Anæsthesia in rest of limbs and trunk.	Hyperæsthesia in lower part of abdominal wall.	Hyperæsthesia in zone around loins; anæsthesia in hind limbs.
Atrophy	Rare in neck; common in fore legs.	Slight in muscles behind lesion.	In hind limbs.
Electric reaction	Lessened in atrophied muscles.	Lessened in dorsal and abdominal muscles; slightly in wasted leg.	Lessened in atrophied muscles; in ratio.
Bladder	Retention or intermittent incontinence reflex, or (later) from overflow. Cystitis common.	Same as cervical.	Incontinence from palsy of sphincter.
Bowels	Involuntary evacuation (reflex spasm) or constipation.	Same as cervical.	Paralysis of sphincter with incontinence; costiveness.
Superficial reflex	Temporary loss; then rapid increase.	Same as cervical.	Lost.
Deep reflex	Temporary loss; then slow increase.	Same as cervical.	Lost.
Priapism	Often present.	Often present	Absent.

HALLUCINATIONS.

Subjective cerebral impressions projected as real. Rabies. Toxins. Poisons. Essential oils. Chloroform.

Hallucinations are subjective impressions which the animal supposes to be real. The disorders in his brain are projected outward and become to him real objects and occurrences.

They may arise from the presence and proliferation of microbes in the brain as in rabies in animals. They may proceed from poisoning of the brain by toxins as in anthrax.

They may be developed, in dogs especially, by the action of certain essential oils on the cerebral cortex. The first two classes will be considered with those special diseases. The mental disorders from drugs have been studied experimentally by Cadeac and Mennier, and may be noticed in this place.

Lavander, fennel and angelica produce in the dog a condition of extreme terror, and overcome all disposition to exercise self-defense.

Mints and origanum induce hallucinations of odor. The dog seeks around with head and nose elevated, sniffs the air, moves cautiously, fixes his eye on some phantom object, and starts to hunt imaginary game. The love of catmint seems to amount to a mania in the feline animal.

Kidney vetch evidently causes a sensation of itching or formication; the dog bites the hair of the tail, the hind limbs or the flank as if to destroy fleas or other vermin.

Dogs under chloroform have sought to hunt, and stallions under ether have shown generative excitement with erection of the penis and movements of coition.

TIMIDITY. PANIC. STAMPEDE.

Timidity. Panic. Timid driver. Impaired vision. Nervous. Irritability. Gadding. Gregarious habit. Absence of natural weapons. Treatment. Habit. Substitution. Absolute constraint. Kindness. Boldness. Work. Moderate diet.

An animal is naturally nervous, and by habit has become timid until it is virtually impossible to utilize it. In a body of animals, fear is quickly transferred from one to the other until all join in a wild panic or stampede. This is common in range cattle or horses, but is found in army horses as well, and a whole regiment will sometimes refuse to longer face the enemy and flee in spite of every effort of the rider. On a smaller scale, two timid horses in a team, scared by some unusual sight, add each to the sense of fear of the other, as they try to escape, until they gallop blindly into any danger. This sense of terror is often fostered by the timid rider or driver, every feeling of apprehension conveyed through the trembling or uncertain hand, or the voice which has lost the element of confidence, tending to undermine the last vestige of trust on the part of the horse. Imperfect sight is one cause of panic, as the perception of common objects in distorted

form or unwonted situations strikes terror to the timid animal, causing shying or bolting. Better absolute blindness than such imperfect vision.

A constitutional timidity tends constantly to increase unless the animal is judiciously accustomed to the object of terror. The horse once scared, seems to become more and more watchful for other objects of dread, and even inclined to bolt from such as are common and of every day occurrence.

Cattle and sheep attacked by the gadfly (œstrus) flee in great terror, and this dread is communicated from animal to animal so that the whole herd or flock is suddenly panic-stricken. The bellow of the ox attacked and the erection of its tail is the signal for every other within reach to join the stampede.

These panics are associated with the instinct of these races toward a gregarious life; they mass together for protection and they learn to heed the slightest indication of approaching danger. This instinct grows more powerful by constant exercise, and is most marked in those genera which have the least natural means of protection. Hence, of all animals sheep are most easily panic-stricken, and once affected, they move in mass, one following its fellow, without object, without definite direction or destination, and without consideration of the other dangers they are to meet. Hence, if one sheep jumps over the parapet of a bridge to certain destruction, the whole flock speedily follows. If one leaps over a fallen tree into a snow bank, all at once follow suit and pile above each other in one suffocating, perishing mass.

While this condition is hereditary in gregarious families, it is essentially a psychosis in those animals that have been often scared until they are continually on the watch for objects of fear.

Treatment. In the case of horses, the best course is to make the animal familiar with the object of dread; let him look at it, approach it slowly, smell it, feel it with his lips. Never turn away his eyes from it and drive him off, as that confirms the impression of dread, and the object retains ever after its dreaded appearance. In this way timid colts become gradually fearless of umbrellas, city sights, street cars, large vans, flags, music, locomotives and the like,—they become, in the expressive language of the horseman, *road-wise*. A paddock or yard beside a railroad will soon accustom a timid horse to the cars, and so with other things, experience will remove apprehension.

A more speedy removal of the habit of dread may often be secured by the principle of substitution. The mind of the animal does not readily attend to more than one matter at a time; if, therefore, we can distract the attention in another direction, the object of fear may be virtually ignored until the eye has become habituated to it, and it will be recognized as harmless. Thus it is that a twitch on the upper or lower lip, a binding of the chin in upon the breast by a Yankee bridle may make the horse temporarily heedless of the object of terror. So also in the bolting horse, the obstruction of the breath by a cord with a running noose around the neck, or the sending of an electric current through wire reins and bit will promptly check him in his wild career.

The result is still better when the animal is made to feel his utter helplessness in the hands of man and the futility of any attempt to escape. On this are based the method of Rarey and of his various successors. With fore limbs strapped up, the animal soon exhausts himself in his efforts to disengage them and escape, and lies down completely reconciled to his fate. He may now be accustomed to his objects of terror—the opening and closing of an umbrella, or the waving of a flag over his head, the discharge of a gun close to his ear, the passing of car or locomotive, or any other object of his dread. When allowed to get up he will usually pay no further attention to these things, especially if patted and spoken to encouragingly, and perhaps fed apple or sugar, or something of which he is fond. As far as is consistent with the thoroughness of the subjection, the animal should be treated throughout with the greatest kindness, so as to retain and even increase his trust in man and sense of dependence, while at the same time he is strongly impressed with the futility of resistance to his will. After the animal has been thus taught to bear with equanimity his former objects of terror, he should not be at once allowed to forget them, but by daily experience he should be confirmed in the conviction that they are harmless, and may be met with safety. This should be carried out, if possible, in the hands of the bold and kind operator who has trained him, as, if returned to a timid driver or rider, he may be easily led back into his former habits of blind terror. A similar and even easier resort is the process of turning as given under balking.

Constant hard work, for a time, is an excellent form of accessory treatment, as the plethora developed by over-feeding and temporary idleness begets an irritability and impatience of control which is quite likely to beguile him into his old habits.

In case of runaway, beside the electric and asphyxiating treatment already referred to, the animal may be blinded and quickly brought to a stand-still. Movable blinds may be used which habitually stand well out from the eyes, but which may be instantly drawn closely over them by the simple pulling of a cord. The sudden darkness and the impossibillity of directing his course, brings an instant realization of the existence of other dangers beside the original bugbear.

BALKING. RESTIVENESS.

Definition. Common in ass and mule. Causes: low condition; overloading; nervousness; sluggish nature; irritable driver; shoulder sores; poor collar; hard bit; sharp or sore maxilla; sores in angle of mouth; mares; racial tendency; going from stable; a psychosis. Symptoms: stands stock still; plunges, but won't draw; will stamp, bite, kick, rear, buck, crowd on wall; lie down. Breach of warranty: sound price; willfulness; balking of raw horse; diagnosis from nervous disorders; sores, etc. Time in which returnable. Treatment: preventive; curative; distract attention; cord on ear; whiff of ammonia or capsicum; closing nostrils; blindfolding; tying up the fore leg; stroking nose, eyes or ears; tapping flexors of metacarpus; move in circle with head tied to tail; coax to go.

In general terms this has been defined as a refusal to obey. Usually in solipeds it is a refusal to move as directed with a load, under the saddle, or in hand. Though essentially a vice, it may become such a fixed habit that it appears to dominate the will of the animal and may thus be called a psychosis—a mental infirmity.

It is much more common in asses and mules than in horses, in keeping with their more obstinate disposition and too often harsher treatment.

There may be simple refusal to pull. This often comes from overloading, and especially when the animal has been sick or idle, and comes back to work with soft flabby muscles unequal to

any violent exertion. After one or two ineffective efforts he sets himself back in the harness refusing to try again and the vice is started. Ordinary loads on bad roads full of holes from which it is impossible to drag the wheels have a similar effect. The danger is greater if the animal is naturally of a nervous or impatient disposition, and if he makes a desperate plunge forward and fails at once to move the load. Such a horse hitched with a slow steady mate is liable to have expended his effort before the latter has had time to join him in the pull, and it becomes impossible to move the load because the two cannot be started simultaneously. The conditions are aggravated if the driver is irritable and by voice and acts further excites the already too excitable animal.

Lesions of various kinds, such as shoulder bruises, abscesses, abrasions and callouses, saddle bruises, callouses, abscesses or fistulæ cause acute pain whenever the effort is made, and render the animal more impatient and indisposed to try again.

Too small a collar or one that fits badly (too narrow, uneven) has often a similar effect.

Among other causes may be named a hard bit harshly used, a sharp edge of the lower jaw bone where the bit rests in the interdental space, sores of the buccal mucous membrane in this situation, and caries or necrosis of the superficial layer of the bone. Also chaps, ulcers, or cancroid of the angle of the mouth.

Young horses, that are as yet imperfectly trained, are more readily driven to balk than old trained animals.

Mares are more subject to the vice than geldings, by reason apparently of a more nervous disposition, but much more because of the excitement to which they are subjected, under the periodic returns of *heat*.

Peuch speaks of rare hereditary cases in which the habit is uncontrollable and the animal incurable.

Friedberger and Fröhner accuse chestnut and sorrel horses as being especially liable to balk.

However started the continued exercise of the act fixes it as an incurable habit a virtual psychosis. Yet the inclination of the animal, his likes and dislikes to a certain extent control its manifestations, thus a horse rarely balks in going home, and shows it mostly in going in the opposite direction, and above all on a new or unknown road.

The *Symptoms* vary greatly in different cases. One animal stands stock-still propping his legs outward and absolutely refusing to budge. This may occur even in the stall when it is attempted to take the animal out. When on the road he is usually willing to turn and go back, but no persuasion by voice or whip can force him forward.

Other horses make ineffective plunges forward but never throw weight enough into the collar to overcome any resistance.

Still others stamp, bite, throw themselves to one side rather than forward, rear up, strike with the fore feet, and if whipped kick with the hind. Some will throw themselves down and struggle in this condition.

Under the saddle the animal may crowd against a wall, rear, kick, buck or even throw himself down in his efforts to dislodge the rider. These violent manifestations however rather belong to vice than mere balking. Trembling, perspiration, frequent rejection of urine, and general acceleration of pulse and breathing may manifest a severe nervous disorder.

Diagnosis. It is often important to pronounce upon the exact nature of this trouble so as to determine whether the seller is responsible for a breach of warranty given or implied. As regards implied warranty a *sound price* for an animal sold to do a given kind of work implies a mutual understanding that the animal is not physically or psychically incapacitated for such work.

The balking horse is one that obstinately refuses to perform a piece of work for which his physical condition seems to be well adapted. The *willfulness* of the refusal is the important feature. In case of such a serious drawback to the value of a horse, the presumption of fraud on the part of the seller is unavoidable, in case he failed to mention the habit to the purchaser, but of course this is even more emphatically certified if he has warranted the animal as a *good worker*, or *kind*, or *true in work*.

On the other hand he cannot be held responsible for the failure to perform an act in case the horse has been overloaded when fat or out of condition, or if he has sores on back, withers or shoulders, a badly fitting collar, a severe or large clumsy bit, or sores on the lower jaw, or indeed any temporary physical infirmity, to which the balking can be fairly attributed.

Balking is not to be confounded with nervous affections

(paretic, spasmodic, congestion) in which the failure to obey is not due to lack of will, but to lack of power. Nor must it be confounded with the inability of the paralysis of lead poisoning. It is perhaps most likely to be confounded with that lack of both sensory and motor power which attends on ventricular dropsy and other chronic affections of the brain. In such cases (immobility, coma) the habitual dullness, drowsiness, general hebetude, and lack of energy contrasts strongly, with the strength, vigor and general life of the animal which suddenly, willfully and incorrigibly balks.

The balky horse which has no such nervous disorder as an excuse, no badly fitting harness, no lesion on shoulder, back, limbs nor mouth, no unsuitable bit, no special softness nor poverty of condition, no slow, ill-adjusted mate, no impatient driver, and no excessive load, nor impassable road, but which jibs without excuse, as a willful disobedience, may well be cause for annulling a sale. In most European countries such a horse can be returned to the seller and the sale set aside within 3 days (Austria), 4 days (Prussia), 5 days (Saxony), 9 days (Hesse).

Treatment. This should be preventive by avoiding the various causes above enumerated, for if the habit is once contracted it is too often impossible to establish a permanent cure. The horse is largely a bundle of habits and the first act of disobedience has given a bias to the nerve cells of the cortex cerebri which like a planted seed tends to reproduce itself whenever an opportunity offers. With every successive act of the kind, the impression on the nerve cells becomes deeper and more indelible and the habit fixed the more firmly.

In slight recent cases in the milder dispositions the vice may be overcome by some resort which engages and engrosses the animal's attention. Among these may be named tying a cord round the root of the ear and tying it down; giving an inhalation of ammonia or a sniff of powdered capsicum; closing the nostrils until the horse struggles to breathe; blindfolding for a few minutes; tying up one fore leg until thoroughly tired; even stroking the nose or ears until the fret is overcome. Immediately following on any one of these methods, move the horse gently to the right and left and call him confidently to *get up.* Some will start if gently tapped with the toe below the knee

until the foot is lifted and repeating this a few times in succession, then, after a few steps, reward with an apple, sugar or piece of bread, and don't push too far at a time but repeat the lesson often. If among the first exhibitions of the vice it may be met by occupying the time in a make-believe fixing of the harness until the animal ceases to fret, then standing by his head, tap him on the croup with a whip and call him to go on. Or he may first be moved to the right and left and then ordered to move. A rather wearisome treatment is to place in the stall with a man behind him who taps him on the rump every few minutes, preventing composure, rest, or sleep, and keeping this up without interval for twenty-four hours or even double that time if necessary. If he goes well when hitched, he is driven but if he balks, he is returned to the stall and the treatment continued.

Magner, who mentions all these methods, reserves his highest commendation for the method of tying the horse's head round to his tail and letting him turn in a circle until he is giddy and falls over. Some stubborn cases get habituated to turning in one direction and continue obdurate until the head and tail are tied around on the other side and the rotatory motion reversed. When thoroughly dazed by this treatment, the animal is hitched up and will usually move on. If there is still an indisposition, stand by his head and tap the croup with a whip, calling on him to start. Or subject him to further rotatory treatment.

DELIRIUM.

A phenomenon in different morbid states; cerebral hyperæmia, anæmia, congestion, inflammation, intoxication, toxin poisoning. Symptoms: horse, ox, sheep, swine, dog. Treatment: adapted to primary disease present, narcotic, poison, and to degree of violence. Anæsthetics, soporifics, cerebral sedations, cold to head, eliminants, depletion or tonics and nutritious food.

Delirium or derangement of emotional or mental functions is usually the result of organic disease of the brain and especially of the cortical gray matter of the cerebrum. It is seen in hyperæmia, anæmia, faults of nutrition, intoxications and variations of

temperature. The derangements of circulation may be in the meninges or in the nervous substance. The intoxications may be with mineral (lead, mercury), vegetable (opium, Indian hemp, belladonna, hyoscyamus, stramonium, strychnia), or other poisions, including the toxic products of microbes (as in pneumonia, scalma, rabies, influenza, Rinderpest, milk sickness, Texas fever, etc.).

Symptoms. These are usually an extraordinary and disorderly nervous excitement. **Horses** take expectant or ready positions of the limbs, plunge with feet in rack or manger, rear, turn, kick, bite, spring violently, neigh, and push or knock the head against the wall. **Cattle** bellow in a loud or frightened manner, attempt to kick and gore, grind the teeth and make movements of the jaws, froth at the mouth, dash themselves in any direction heedless of obstacles, push the head against the wall breaking teeth or horns, and moving heedlessly against fences, or trees, or into water or pits even to their own destruction. **Sheep** stamp the feet, butt, bleat, work the jaws, grind the teeth, leap, and move in a given direction regardless of obstacles. **Pigs** grunt, tremble, champ the jaws, run against obstacles, scratch the ground with their feet or snout and creep under the litter. They may even attempt to bite. **Dogs** are restless, whine, move in a circle, snap at straw, bars, doors, and other objects, and may show a disposition to bite. In all the domestic animals these delirious symptoms may closely resemble those of rabies. This has been particularly noticed in certain forms of poisoning. Pascault has found this in cattle that had eaten garlic and Cadeac in dogs that had eaten tansy.

The animals in such cases become morbid, dull, taciturn, they become usually hypersensitive, sometimes hyposensitive, have a change of voice, and show a readiness to resent and bite if interfered, with, and even to wander away by themselves as in rabies. On the other hand they may be seized with lethargy and torpor as in dumb rabies, and with or without access of convulsions may pass away in a condition of paralysis.

Among other conditions these symptoms have been found to be associated with epilepsy, foreign bodies in the pharynx, gullet, stomach or bowels, with intestinal parasites, or with mycotic poisoning (ergotism, smut, the fungus of coniferous trees, etc.).

The lack of the extreme hyperæsthesia and excitability of rabies, and usually of the mischievous disposition to bite, the presence of foreign bodies in the mouth or gullet, and the evidence of disorder of digestion, with costiveness, tympany, and tenderness, and the history of the case may serve to differentiate. In cases of doubt the inoculation of a rabbit on the brain should demonstrate the absence of rabies by the absence of the characteristic symptoms after sixteen days.

Treatment. As delirium in animals is a deranged innervation from congestion, narcotic drugs, ptomaines, etc., it must be looked on as in most cases a mere phenomenon, pointing to a definite disease, or to a particular intoxication, and treatment must be directed toward the removal of the primary cause. Thus the remedial measures must be directed in the different cases to the encephalitis, meningitis, digestive disorders, contagious disease, or drug to which the affection may be traced. The patient must be put in a strong inclosure or securely tied so that he can do no harm: it will often be desirable to secure shade or cool air, or to apply cold water or ice to the head, and to quiet the nervous excitement by inhalations of chloroform, or ether, rectal injections of chloral, or bromides, or full doses of hyoscine, sulphonal, trional or tetronal. These may be pushed to the extent of inducing anæsthesia, sleep or quiet, as the case may be, and meanwhile other measures should be taken to eliminate the poisons, correct the congestion, or remove the source of irritation. Anæmic cases may demand iron and bitters, with an aliment rich and easily assimilated, while plethoric cases may require purgation, diuresis or even bloodletting. All noise and any cause of excitement must be carefully guarded against.

VICIOUSNESS. AGGRESSIVE VICE.

Subject maliciously using its natural weapons. Horse kicks, bites, crowds against wall, rears, bucks, plunges, treads upon. Cattle use horns or forehead, or kick. Dog bites. Cats scratch and bite. Ticklishness different. Developed or inherited. Revenge. Desperation in pain. Sexual. A psychosis. Responsibility of owner, in selling, toward employe, in exposing in a public place. Treatment: remove source of suffering, treat kindly, secure confidence, castrate, place under absolute constraint, throw *a la Rarey*, Comanche bridle, tie head to tail and circle, etc.

This word is employed to cover only those forms of vice in which the animal shows a malignant disposition to attack or injure man or beast. Each animal uses its natural weapons according to the occasion.

The **horse** strikes with his fore feet, kicks with his hind, bites, crowds his rider's leg against a wall, or his attendants' body against the side of the stall, rears, bucks, plunges, or treads his victim under his feet.

The **ruminants**, large and small, use their horns, and **cattle** their feet as well. In the absence of horns they still use the forehead, but much less effectively and usually only with the purpose of defence.

The **dog** attacks with his teeth and the *cat* with her claws by preference, and uses the teeth as a secondary weapon.

Swine use their tusks to rip or disembowel their adversary or victim.

A very ticklish horse cannot bear to be touched on the flank or hind parts, without throwing the ear backward, glancing back, showing the white of the eye, and lifting the foot. But if this is mere excess of sensitiveness and begets no disposition to kick it is not viciousness.

The vicious horse will in such cases bite or kick repeatedly and with well directed purpose. He will moreover show the movements of ears and eyes and attack his victim in the absence of any such excuse, the simple approach being a sufficient occasion. He will bite and strike with the fore feet at the same time, or he may strike out with one hind foot or with both at once. He may

attack indiscriminately all who approach him, or reserve his ill-will for particular individuals, and then he often acts under a feeling of revenge for ill-usage from this individual or some one he conceives him to represent.

In some cases viciousness is inherited and certain families have a bad reputation in this respect. It may be either a survival of the ancestral disposition of the wild horse, or it may be a trait developed by ill-usage of a team of more immediate ancestors.

In other cases the habit is acquired by the individual himself, and in such cases it may be due to brutal treatment at the hands of man; to a continuous punishment of a high-spirited horse leading to resentment and retaliation; to acute pain in boils, abrasions or other sores in the root of the mane, or the shoulder, or the back, where pressed on by the collar or saddle; or to the generative excitement of mares in *heat*. In many such cases the vice lasts only during the persistence of the cause, in others it becomes permanent. The stallion is much more disposed to aggressive vice than the gelding.

Whether we may consider the vice a disease or not, it becomes a habit engrained in the nature, the nerve centres tending to reproduce their habitual acts indefinitely, so that we may look on the condition as a psychosis which is too often incurable.

Responsibility of the owner. Dangerous aggressive vice is too self-evident to the buyer to constitute a good cause for annulling a sale, but it has this legal bearing, that the owner who keeps an animal known to be vicious, renders himself responsible for whatever injury to man or beast he may perpetrate. Thus the vicious stallion, bull or dog in a public place which damages person or property, renders his owner liable to the extent of such damages. This, of course, must be largely qualified by the attendant circumstances. The man employed to take care of a horse, knows his habits as fully as the owner, takes his chances and should exercise due precautions to avoid danger. The person who enters a stall carelessly without speaking to the horse, seeing that he stands over, or otherwise responds to his call, is himself to blame if he gets kicked. The attendant who does things to a dangerous or questionable horse for mere bravado cannot blame the owner if he gets himself injured. If a person teases a horse so as to tempt him to retaliate, not only is he re-

sponsible for his own consequent injuries, but largely also for the habits of the horse and for such injuries as others may subsequently sustain from him.

A dog or a bull shown in a public place, and which breaks loose and injures spectators or others, manifestly renders his master responsible for all such damage.

Treatment of aggressive vice. In mild dispositions in which the vice is roused by temporary suffering, it may often be cured by removal of the cause of such suffering. Indeed, without the healing of sores under the collar or saddle the vice cannot be arrested. Considerate and gentle treatment, too, will go far to restore confidence and to gradually do away with the aggressive disposition.

In wicked stallions castration will usually restore to a good measure of docility. The exceptional cases appear to be those that are hereditarily and constitutionally vicious, or in which the habit has been thoroughly developed and firmly fixed by long practice.

Mares, too, which become vicious and dangerous at each recurrence of œstrum, can usually be completely cured by the removal of the ovaries especially if this is done early in the disease.

The inveterate cases may usually be subdued and rendered controllable for a time by one of the methods of subjugation employed by the professional tamers, but unless they are thereafter kept in good hands they are liable to relapse into the old habit. Among the more effective methods are the Rarey mode of throwing which may be repeated again and again until the animal is thoroughly impressed with a sense of the domination of man and the futility of resistance; the resort of tying the head and tail closely together and letting the animal weary and daze himself by turning in a circle, first to the one side and then to the other; the application of the Comanche bridle made of a small rope, one loop of which is passed through the month and back of the ears and drawn tightly, then another loop is made to encircle the lower jaw, and the chin is drawn in against the trachea by passing the free end of the rope round the upper part of the neck and again through the loop encircling the lower jaw and drawing it tight; or a similar small rope is passed a number of times through the mouth and back of the ears and drawn tightly so as

to compress the medulla and stupify the animal. This is supposed to be rendered more effective by passing one turn each between the upper lip and gums and between the lower lip and the gums.

CATALEPSY.

Definition. Tetanic and paralytic forms. Balance of flexors and extensors Cataleptoid. No constant lesion. Hysterical. Hypnotic. Subjects: horse, ox, wolf, cat, chicken, Guineapig, snake, frog, crayfish. Causes: strong mental impression, indigestion, etc., in susceptible system. Lesion: inconstant, muscular degeneration, etc. Symptoms: wax-like retention of position given, voluntary movement in abeyance, mental functions impaired, secretions altered. Duration and frequency variable. Treatment: shock; cold; ammonia, pepper, snuff, electricity, amyle nitrite, nitro-glycerine, apomorphine, bromides, purgatives, bitters, iron, zinc, silver, open air exercise.

Definition. This is a functional nervous disorder, characterized by paroxysms of impaired or perverted consciousness, diminished sensibility, and above all a condition of muscular rigidity, by means of which the whole body, or it may be but one or more limbs retain any position in which they may be placed.

Laycock describes two forms in man—the catochus or tetanic form, and the paralytic form. Mills would restrict the name catalepsy to cases in which the muscular tone is such that the affected part may be bent or moulded like wax or a leaden pipe, and will not vary from this when left alone. Other forms in which this waxen flexibility (flexibilitas cerea) is absent or imperfect he would designate as cataleptoid.

The disease is not associated with any constant cerebral lesion, though it may supervene in the course of other nervous disorders, and therefore may own an exciting cause in existing lesions of the brain. The immediate cause must however be held to be functional, and this is in keeping with its most common form in man (hysterical), and with the hypnotic form which is observed both in man and animals. This latter may be looked on as a form of induced or hypnotic sleep, in which the retention of the position given to a limb or part is the most prominent symptom. In all cases there is an impaired condition of the sensory functions of the cerebral convolutions, and an insusceptibility of the motor centres to the control of the will, or the reflex stimulus.

Hering has recorded the disease in the horse, Landel in the ox, and Leisering in the prairie wolf. The hypnotic form has been shown in cats, chickens, and Guinea pigs. The serpent charming of the Indian dervishes and similar effects on frogs and crayfish have been attributed to hypnotic catalepsy.

Causes. Strong mental emotions and diseases which profoundly affect the nervous system have been adduced as causes (fear, excitement, chills). Indigestible food has even been charged with causing it. There is undoubtedly, to begin with, a specially susceptible nervous system, and hence it is liable to prove hereditary, and in man to appear as a form of hysteria, or to alternate in the same family with epilepsy, chorea, alcoholism, opium addiction and other neurosis.

Hypnotism as a cause is claimed by various writers. Azam says that in the fairs in the South of France, jugglers hypnotize cocks by placing the bill on a board, on which they trace a black line passing between the two feet of the bird. Cadeac adds that Father Kircher, in the 17th century, employed a similar method to put fowls to sleep. Alix put cats to sleep by securing them firmly, and then looking steadily into their eyes. The condition attained varies according to the degree of the sleep, the will being dominated first, and later, consciousness of external objects is lost. Hypnotism, however, appears to be difficult and uncertain in the lower animals, in keeping with the limited development of intelligence and will, as compared with the human being. Cadeac states that the very old and the very young are completely refractory to hypnotizing influences.

Lesions. No constant pathological changes are found, though different nervous lesions may serve to rouse the disease in a predisposed subject. Fröhner found in the affected muscles granular swelling, fatty degeneration, hæmorrhages, and waxy (amyloid) degeneration of the cardiac muscles, corresponding to what has been found in tetanus; also hæmorrhages on the stomach and intestines.

Symptoms. The leading objective symptom is the tonic condition of the muscles by which a perfect balance is established and maintained between the flexors and extensors so that the affected part maintains the same position which it had when the attack began, or any other position which may be given to it during the

progress of the paroxysm. The position is only changed when the muscles involved have become completely exhausted. During the attack the affected muscles are swollen and firm, so that their outline may often be traced through the skin, later as the attack subsides they become soft and flaccid. Voluntary movement of the affected muscles is impossible until after the paroxysm. The attack usually comes on suddenly and in this respect resembles epilepsy ; at other times there are premonitory symptoms of nervous anxiety, excitement or irritability. There is usually considerable impairment of consciousness, intelligence, common sensation, and even of the special senses. In a cataleptic dog Fröhner noted mental and motor troubles, considerable anæsthesia, and loss of sight, smell, and hearing. The eyes are fixed, the pupils either contracted or dilated, and the urine passed may be albuminious or even icteric.

Course, Duration. Like other functional nervous disorders this is extremely uncertain in its progress. There may be but one attack or a succession ; they may last from a few minutes, to 7 days (Fröhner), or even several weeks (Hertwig); they may end in recovery or less frequently they may prove fatal usually by inanition.

Treatment. During a seizure a sudden shock will sometimes cut short the attack, douching with cold water, an inhalation of ammonia, of capsicum or of snuff, or the application of electricity in an interruped current through the spine and affected muscles. Ether anæsthesia will not always relax (Sinkler). Inhalation of a few drops of nitrite of amyle has proved effective in man, as has also the injection subcutem of three drops of a 1 per cent solution of nitro-glycerine, apomorphine hypodermically is usually effective (Sinkler). Bromide of potassium has also been advised, and in case of coldness of the surface, a warm bath.

When there is overloaded stomach and gastric indigestion an emetic is indicated, and in constipation a purgative (for speedy action chloride of barium or physostigma subcutem).

In the intervals between attacks tonics and general hygiene should be invoked to build up the weakened nervous system. Quinine, and salts of iron, zinc or silver with a nourishing diet and out door exercise are especially indicated.

INSOLATION. HEAT EXHAUSTION. SUNSTROKE. THERMIC FEVER.

Definition: two forms. **Heat exhaustion.** Causes: prolonged heat, and moisture, overexertion. Impaired vaso-motor centre. Failing heart. Carbon dioxide poisoning. Symptoms: weak, fluttering pulse, perspiration, muscles flaccid, prostration, no hyperthermia. Treatment: stimulant, digitalis, digitalin, subcutem, nitroglycerine, warm baths. **Thermic fever.** Hyperthermia excessive. Causes: insolation, prolonged heat and impure air, furnace heat, moist and dry heat, electric tension, overwork, muscular exhaustion, coagulation of myosin, constant heat on one part (head), excess of carbon dioxide, stiffening of bodies when killed in hot weather, debility, weakness, fatigue, chest constriction, tight girths or collars, short bearing reins, plethora, obesity, open cars and yards, fever, privation of water, heavy fleece. Lesions: right heart and systemic veins full, blood black fluid or diffluent, left ventricle empty, congested meninges, effusions in or on brain, or hæmorrhages. Symptoms: **horse**: dull, stupid, stubs toes, sways quarters, droops head, hangs on bit, props on feet, breathes rapidly, pants, stertor, dilated nostrils, gasping, fixed eyes, dilated pupils, tumultuous heartbeats, gorged veins, epistaxis, perspiration, convulsions: **ox**: parallel symptoms: **sheep**: open mouth, stertor, fixed eyes, pupils dilated, panting, swaying, fall, convulsions: **dog**: dull, prostrate, pants, congested veins and mucosæ, weakness, spasms, syncope, speedy rigor mortis. Overheating. Diagnosis: early excessive hyperthermia, venous congestion, shallow panting breathing, violent heart action, loss of sensory and motor functions, convulsions. Prevention: avoid violent, prolonged heat, and exertion, especially in case of fat animals or those new to hot climate, keep emunctories acting, shade head, water on head and to drink, protect fat cattle, shear sheep, water. Treatment: shade and laxatives; if severe, cold water from hose, ice bags to poll, rub legs, acetanilid subcutem, stimulant enemata, later mineral tonics, iron or zinc.

Definition. A morbid condition produced by the exposure to extreme heat, and marked by profound disorder of the *vaso-motor* and *heat* centres.

The single term of *sun-stroke* or *heat-stroke* has been replaced by two,—**heat exhaustion** and **sun-stroke**, indicating two distinct conditions, brought about by exposure to heat and manifested by different states of the body and distinctive symptoms.

Heat Exhaustion.

This appears as an exaggerated form of the general sense of relaxation, weakness and languor which follows on prolonged

violent exertion in a hot atmosphere. There is more or less impairment of the vaso-motor nerve centre in the medulla, relaxation of the capillary system, and flagging of the heart's action, which loses its customary stimulus, by reason of the defective supply of blood returned by the veins. This may become so extreme that the patient dies by syncope. In other cases the paresis is mainly shown in the vaso-motor system, and its centres in the medulla, the blood is delayed in the distended capillaries and veins, it becomes overcharged with carbon dioxide, the heart's action is accelerated and feeble, the pulse rapid, weak and fluttering, perspiration breaks out on the skin, and the temperature is normal or subnormal. The muscular weakness, the flaccid condition of the facial muscles, and general depression suggest a state of collapse. This condition is not necessarily due to exposure to the intensity of the sun's rays, but may come on in animals subjected for a length of time to artificial heat, and especially if the air is impure, and if the subject has to undergo severe physical exertion.

Treatment. In slight cases of this kind a stimulant is usually desirable and ammonium carbonate in bolus or solution will usually serve a good purpose. In its absence alcohol or spirits of nitrous ether may be given. Digitalis is of great value in sustaining the flagging action of the heart and has the advantage that as digitalin it can be given hypodermically when it is impossible to give ammonia, alcohol or ether by the mouth. For the same reason nitro-glycerine may be resorted to, or even atropia as a vaso-motor stimulant. Active friction of the body and limbs will aid circulation and indirectly stimulate the heart, and in case of subnormal temperature it may be supplemented by a warm bath in the smaller animals, kept up until the normal temperature in the rectum has been restored.

Thermic Fever. Sun-stroke.

This is readily distinguished from *heat exhaustion* by the predominance of the hyperthermia. While in **heat exhaustion** the temperature is usually subnormal, in **sun-stroke** it is excessive, (108°–113° F.).

Causes. The immediate cause of sun-stroke is exposure to undue heat, but this need not be the heat of the sun's rays direct.

A large proportion of cases in the human subject are attacked during the night, and again at sea where an attack in a passenger is practically unknown, it is terribly common among stokers working in a close atmosphere of 100° to 150° F.

The attendant conditions have much influence in determining an attack, thus it is generally held that heat with excess of moisture is the most injurious, yet in Cincinnati, statistics showed a greater number of cases in man when the air was dry. The suppression of perspiration and the arrest of cooling by evaporation in the latter case would tend to a rapid increase of the body temperature, and the condition would be aggravated by the electric tension usually present with the dry air. With the hot, moist air perspiration might continue, but evaporation would be hindered, and there would be arrest of the cooling process and an extreme relaxation of the system.

Again, it is usually found that seizures take place during or after hard muscular exertion in a hot period, and much importance is attached to the attendant exhaustion, the excess of muscular waste, and the alteration of the myosin, which latter coagulates at a lower temperature in the over-worked animal. But on the other hand, experiment shows that the animal confined to absolute inactivity in the hot sunshine or in a high temperature (at 90°), dies in a few hours, whereas another animal left at liberty in the same temperature does not suffer materially. The explanation appears to be that the dog, kept absolutely still, has the continuous action of the heat on the same parts and on the same blood, for the capillaries dilate, and the blood is delayed, overheated, and surcharged with carbon dioxide, and the result is either syncope from heart failure, or asphyxia from excessive carbonization of the blood. Back of these and concurring with them is the paralysis of the vaso-motor and heat generating nerve centres, from the high temperature or the condition of the blood.

The excessive carbonization of the blood deserves another word. The prolonged contact of the blood and air in the lungs is essential to the free interchange of oxygen and carbon dioxide. Vierordt showed that with sixty respirations per minute the expired air became charged with but 2.4 per cent. of this gas, whereas with fourteen respirations it contained 4.34 per cent. Therefore, with violent muscular work (which charges the blood with carbon

dioxide) and rapid breathing (which fails to secure its elimination), the over-driven animal soon perishes from asphyxia. Under a high temperature of the external air, this condition is aggravated since the rarified air contains just so much the less oxygen, the absorption of which is the measure of the exhalation of carbon dioxide.

Dr. H. C. Wood, who has experimented largely on the subject in animals, finds the cause of heart failure in the coagulation of the myosin, which takes place under ordinary circumstances at 115° F., but at a much lower temperature when a muscle has been in great activity immediately before death. As the temperature of thermic fever frequently reaches 113°, or even higher, he easily accounts for the sudden syncope occurring during active work in a high temperature. As an example of such sudden rigor, he adduces the sudden stiffening of the bodies of some soldiers killed in battle during hot weather.

Wood further shows that all the symptoms of thermic fever can be produced in the rabbit by concentrating the temperature on its head, which seems to imply a direct action on the brain and in particular on the heat producing and vaso-motor centres. This becomes the more reasonable that the temperature attained does not impair the vitality of the blood but, leaves the leucocytes possessed of their amœboid motion. He found, moreover, that if the heat were withdrawn before it has produced permanent injury to the nervous system, blood or other tissues, the convulsions and unconsciousness are immediately relieved and the animal recovers.

Other conditions may be adduced as predisposing or concurrent causes of thermic fever. Whatever impairs the animal vigor has this effect. Fatigue, as already noticed, is a potent factor, in man a drinking habit; in all animals a long persistence of the heat during the night as well as the day; impure air in badly ventilated buildings; and mechanical restriction on the freedom of breathing. In military barracks with the daily temperature at 118° F. and the night temperature 105, the mortality became extreme, and in close city car stables the proportion of sun-strokes is enhanced. In all such cases, the air becomes necessarily more and more impure continually. The atmosphere has the same heat as the animal body, so that no upward current from the latter can be established, to create a diffusion. The carbon dioxide

and other emanations from the lungs, the exhalations from the skin, dung and urine, accumulate in the air immediately surrounding the animal and respiration becomes increasingly imperfect and difficult. This condition is further aggravated by the accumulation of the animal heat in the body. The blood circulating in the skin can no longer be cooled, to return with refrigerating effect on the interior of the body, the cooling that would come from the evaporation of sweat is obviated by the suppression of that secretion, as well as by the saturation of the zone of air immediately surrounding the body, and thus the tendency is to a steady increase of the body temperature until the limit of viability has been passed.

The mechanical restriction of respiration should not be overlooked. In European soldiers landed in India and marched in the tight woolen clothing and close stocks a high mortality has been induced and in horses with tight girths or collars and short bearing reins, and oxen working in collars a similar result is observed. Any condition of fever is a potent predisposing factor.

Horses or cattle that are put to violent or continued exertion when too fat or out of condition are especially subject to sunstroke. Fat cattle driven to market under a hot sun, or shipped by rail, crowded in a car and delayed on a siding under a hot sun, with no circulation of air, often have insolation in its most violent form. The same may be seen in the hot stockyard, with a still atmosphere and the fat animals subjected to the full blaze of a July sun. The chafed feet caused by travel, and the muscular weariness caused by standing in the moving car are material additions to the danger.

Similarly horses suffer on the race track when subjected to protracted and severe work in hot weather, or again dragging loads in a heated street under a vertical sun, or on a side hill with the sun's rays striking perpendicularly to its surface.

A change in latitude has a decided effect, the Northern horse suffering much more frequently than the one which is native to the Southern States and which has inherited the habit of heat endurance.

Finally faults in feeding and above all watering are appreciable factors. The privation of water in particular is to be dreaded. Tracy in his experience with American soldiers in Arizona, found

that the command could usually be guarded against sun stroke when a supply of water was kept on hand. It should be used guardedly, but nothing would act better in obviating an attack. On the other hand, when the canteens were empty, under the hot sun the seizures increased disastrously.

Sheep are especially liable to suffer from heat by reason of their dense fleece, which hinders the evaporation of perspiration, and the cooling effect of air on the skin. When the temperature rises, respiration is accelerated and panting, the lungs seeking to supplement the work of the skin. When traveling in a heavy fleece, or in the hot sunshine in July or August sunstroke is not uncommon among them.

Lesions Among the lesions may be named, vacuity of the left ventricle and fullness of the right ventricle and veins with fluid blood or a diffluent clot; congestion of the pia or dura mater, effusion into the ventricles, hæmorrhages into the subserous tissues, and degeneration of the muscles.

Symptoms. **Horse.** When premonitory symptoms are observed the animal fails to respond to whip or voice, lessens his pace, stubs with his fore feet and sways with the hind, depresses his head and hangs heavily on the bit.

Too often these are omitted or overlooked, and the horse suddenly stops, props himself on his four limbs, drops and extends the head, breathes with great rapidity, panting and even stertor, dilates the nostrils widely, retracts the angle of the mouth and even gapes, has the eyes fixed, the pupils dilated and the beats of the heart tumultuous. The superficial veins are distended, the visible mucosæ congested with dark blood, and blood may escape from the nose. Perspiration usually sets in.

The animal may fall and die in a few minutes in convulsions, or, if stopped sufficiently early and suitably treated, he may in a measure recover in 15 to 20 minutes.

Symptoms. **Ox.** The premonitory symptoms are like those in the horse: dullness, rapid, panting breathing, the mouth is opened and the pendent tongue is covered with frothy saliva, a frothy mucus escapes from the nose, the eyes are congested and fixed, the pupils dilated, the nostrils and flanks work laboriously, the heart palpitates, the animal sways or staggers and falls. Death follows in convulsions, or it may be delayed, the animal

struggling ineffectually to rise, or having periods of comparative quiet. The rectal temperature is very high, 107° to 114° F. If able to stand, there is usually blindness and heedlessness of surrounding objects.

Symptoms. **Sheep.** The open mouth, protruding tongue, frothy saliva, reddened fixed eyes, rapid breathing, beating flanks, stertor, and unsteady gait are characteristic when taken along with the manifest causes. Swaying movements followed by a sudden fall and death in convulsions form the usual termination of the disease.

Symptoms. **Dog.** These have been mainly produced experimentally and consisted in hyperthermia, dullness, prostration, accelerated breathing and heart action, congested veins, and mucosæ, muscular weaknesss, convulsions, and syncope or asphyxia. After death the muscles became speedily rigid, and the blood accummulated in the venous system, was fluid or only loosely coagulated. In these animals, if the experiment were stopped in time the animal could be restored to health.

Slighter cases may occur in the different animals, more particularly from overdriving in hot weather, and in such cases the *overheated* animal recovers, but there is liable to remain a special sensitiveness to excessive heat and a tendency to be dull, sluggish and short winded, to hang the head in hot weather, and to seek shelter from the direct rays of the sun.

Diagnosis is largely based on the suddeness of the attack, on the occurrence of high temperature before the seizure, not after as it is liable to be, if at all, in apoplexy, on the dark congestion of the mucosæ, and of the venous system, on the rapidity and shallowness of the respirations, on the tumultuous action of the heart, and on the general loss of sensory and especially of motor function, in circumstances calculated to induce sunstroke. Localized paralysis or spasm would suggest the formation of a cerebral effusion or clot.

Prevention. This will depend on the class of animal and its conditions of life and work. In horses care should be taken to regulate the work by the heat of the season and condition of the animal. When the temperature ranges from 80° to 100° F. the work should be lessened and every attention should be given to maintain the healthy functions (bowels, kidneys, skin) in good

working condition. If the horse is young, fat, or out of condition from idleness or accumulation of fat he must have the greater consideration. So it is with a horse recently come from a colder latitude, and with a heavy draught horse that may be called on to do rapid work. Some protection is secured by wearing a sunshade or a wet sponge over the poll, and much may be expected from an occasional rest in the shade, a swallow of cool water and sponging of the head.

Very heavy fat cattle should not be driven far nor shipped on the hottest days, and the packed car should not be left in the full sunshine in a still atmosphere. Yards with sheds under which they can retreat must be secured if possible.

The heavily fleeced sheep must have equal care and the pastures for fat sheep and cattle should have available shade in form of trees, walls or sheds. Access to water is an important condition.

Treatment. In slight cases (*overheated*) a few days of rest, under an awning rather than in a close stable, with a restricted and laxative diet.

In severe thermic fever the first consideration is to lower temperature. If available turn a hose on the head, neck and entire body for five or ten minutes, or until the rectal temperature approaches the normal. In the absence of such a water supply, dash cold water from a well on the body but especially the head and neck, and if available tie a bag of ice around the poll. Active friction to the legs and body is often of great advantage. A large dose of antipyrin or acetanilid may be given hypodermically. On the other hand stimulants, and especially carbonate of ammonia, or sweet spirits of nitre may be given as an enema. This may be repeated in an hour in case the pulse fails to acquire force and tone.

Should the temperature rise again later it may often be kept in check by cold sponging and scraping followed by rubbing till dry.

In case of continued elevation of temperature, with heat of the head, and perversion of sensory or motor functions, meningitis may be suspected and appropriate treatment adopted.

For the prostration and weakness that is liable to follow thermic fever, mineral tonics such as the salts of iron or zinc may be resorted to.

EPILEPSY. FALLING SICKNESS.

Definition. Frequency. Susceptibility: dogs, pigs, cattle, horses, parrots, sparrows. Divisions: slight and severe: Jacksonian (partial): symptomatic; idiopathic. Lesions: inconstant: of brain, cranium, cerebral circulation, myelon, poisons in blood, dentition, cortical and ganglionic lesions, cerebral asymmetry, stenosis of vertebral canal. Medullar asymmetry, traumas of cranium, anæmia, bleeding, carotid ligation, spinal reflexes, irritation of skin, creatinin, cinchonoidin, lead, ergot, nitropentan, nitro-benzol, ptomaines, toxins, parasites, nerve lesions, local hyperæsthesia (withers of horse, recurrent ophthalmia), indigestion, constipation, sciatic neuritis. Causes: nervous predisposition, heredity (man, cat, dog, ox), sexual excitement, fear, sudden strong visual impression, uric acid in blood, meat diet. Symptoms: **horse,** sudden seizure, bracing feet and limbs, swaying, fall, convulsive rigidity, jaws working or clenched, eyes rolling, salivation, stertor, dyspnœa, sensation absent. Duration. Symptoms of localized epilepsy. **Cattle,** bellow, stertor, rolling eyes, jerking, rigidity, fall. **Sheep. Swine** premonitory malaise, jerking, champing jaws, fall, trembling, rigidity, involuntary discharges. **Dog** trembles, cries, falls, rigidity, clonic contractions, stertor, sequelæ. Diagnosis: sudden attack, unconsciousness, spasms, quick recovery, no spasms in syncope, vertigo has no spasms, thrombosis has symptoms developed by exercise. Jurisprudence: animal returnable after twenty-eight days (Wurtenberg, etc.,) thirty days (France). Treatment: of susceptible brain, and peripheral irritant. Correct all irritation or disease, or expel parasites. Nerve sedations: bromides, opium, valerian, belladonna, hyoscine, duboisine. Tonics: zinc, arsenic, silver, baths, electricity. Borax. Vegetable diet. Castration. Avoidance of excitement. Surgical operations. Trephining. Excision of cortex. Outdoor life. *During a fit:* amyle nitrite, chloroform, ether, chloral, warm bath, cold or warmth to head, quiet secluded place.

Epilepsy is the name given to a class of cases characterized by a sudden and transient loss of consciousness with a convulsive seizure, partial or general. It appears to be due to a sudden explosive discharge of convulsive nervous energy, which may be generated by a great number of causes of morbid irritation—pathological, traumatic, or toxic. As a rule the epileptic seizure is but the symptomatic expression of a complex derangement which may be extremely varied as to its nature and origin.

Frequency in different animals. The affection is far less frequent in the domestic animals than in man, doubtless because of

the absence of the special susceptibility which attends on the more highly specialized brain, the disturbing conditions of civilization, and the attendant vices.

Among domestic animals, dogs are the most frequent victims in keeping with their relatively large cerebral development, their emotional and impressionable nature and the unnatural and artificial conditions in which as house pets they are often kept. Their animal food and the consequent uric acid diathesis is a probable cause, as it is in man. In ten years of the dog clinic at Alfort they made an average of 3 per cent. of all cases. Next to the dog the pig kept in confinement is the most frequent victim, while cattle and horses come last. At the Alfort clinic epileptic horses were not more than 1 per 1000 patients. It is not at all unfrequent in birds, especially canaries and parrots. Reynal has seen it in sparrows.

Divisions. The disease has long been divided into *petit mal* and *grand mal* (*haut mal*). The **petit mal** (slight attack) is usually a transient seizure affecting a group of muscles only and associated with only a momentary or very transient loss of consciousness. The loss of consciousness is uncertain as to many cases. Under partial epilepsies must be included the hemi-epilepsy, or Jacksonian epilepsy, which is confined to one side of the body.

The **grand mal** (severe attack) is one in which the loss of consciousness is complete, and the convulsions are general in the muscles of animal life.

Another division is into **symptomatic** and **idiopathic** cases, and if this distinction could always be made it would be of immense value in the matters of prognosis and treatment as the removal of the morbid state of which epilepsy is the symptom will usually restore the patient to health. Thus the removal of worms from the alimentary canal, of indigestible matters from the stomach, of a depressed bone or tumor from the surface of the brain may in different cases be the essential condition of a successful treatment.

Morbid Anatomy and Pathology. The literature of epilepsy is very rich and extensive and yet no constant lesions of the nervous system can be fixed on as the local cause of the disease. A review of the whole literature leads rather to the conclusion that

irritations coming from lesions of the most varied kind, acting on a specially susceptible brain will rouse the cerebral centres to an epileptic explosion. Thus epilepsy has been found to be associated with lesions of the following kinds :

1st. Brain lesions of almost every kind, including malformations.

2nd. Lesions of the walls of the cranium.

3d. Disorders of the cerebral circulation.

4th. Lesions of the spinal cord.

5th. Morbid states of the circulating blood (excess of urea, uric acid, creatinin, lead poisoning).

6th. Reflected irritation, as from dentition, worms, sexual excesses, injuries to certain nerves, notably the sciatic, or to particular parts of the skin.

1st. **Brain lesions.** Those which affect the medulla and the cortical convolutions around the fissure of Rolando would be expected to be implicated because these centres preside over the principal motor actions of the body and limbs. Yet though these parts are found to be affected with various morbid lesions in a certain number of cases of epilepsy, such lesions are exceptional, rather than the rule. In 20 cases of epilepsy in man, 15 showed no lesion whatever of the brain. Bloeq and Marinesco, pupils of Charcot, recently made a critical examination of the medulla and Rolandic cortex in nine cases that died during the fit. All showed granular bodies (degenerated myelin or blood pigment) in the perivascular sheaths but they found these in disseminated sclerosis and even in healthy brains as well. The neuroglia cells of the first cortical layer contained black granules. Otherwise four cases had no change, while five showed sclerosis of the cortex. The medulla was sound in all cases excepting one which showed punctiform hæmorrhages. Visible lesions may be present in other parts of the brain ; Wenzel long ago claimed constant lesion of the pituitary body. Beside the cerebral cortex, lesions have been found in the bulb, the hypoglossal nucleus, the olivary body, the hippocampi, the thalamus, the corpus striatum, the quadrigemini, the cerebellum, etc. Hughlings-Jackson who made an extended investigation of the subject concludes that any part of the gray matter of the encephalon may become over-excitable and give rise to a convulsive attack. Not

only may the lesion be in any part of the brain, but it may be of any kind: meningitis, cerebritis, softening, tubercle, tumor, hydatid, embolism, or dropsy. Marie Bra found an extreme asymmetry of the cerebral lobes in epileptics. Kussmaul and others found stenosis of the vertebral canal and asymmetry of the two lateral halves of the medulla.

2nd. **Cranial lesions.** These consist largely in blows or falls upon the head, with ostitis, periostitis, fractures with depressions, fibrous neoplasia implicating or not the meninges and pressing on the brain, hæmorrhages from minute arteries, etc. The diagnosis of such lesions will often open a way to a successful treatment. Baker found most of the severe cases from head injuries.

3d. **Disorders of the cerebral circulation.** Burrows, Kussmaul and Turner showed that in animals, loss of consciousness and epileptiform convulsions followed on cerebral anæmia caused by profuse bleeding or by compression of the carotids. The same has been observed in surgical cases after ligation of one common carotid. Hermann caused convulsions in a rabbit by ligating both anterior and posterior venæ cavæ.

4th. **Lesions of the Spinal Cord.** Brown-Sequard determined epileptiform convulsions by transverse section of one half of the spinal cord, or of its superior, lateral or inferior columns. The later development of the doctrine of interrupted spinal inhibition, suggests that, many of the seizures in question are but exaggerated spinal reflexes, which are no longer restrained by cerebral inhibition. That all are not of this spurious kind may be fairly inferred from his further demonstration that bruising of the great sciatic in animals tended to produce epilepsy. In such cases the irritation of certain areas by pinching the skin, served to produce a seizure. Not only so, but the animals in which such artificial epilepsy had been induced tended to transmit the infirmity to their progeny. The prevailing view of epilepsy however, would consider such lesions as sources of peripheral irritation by which the brain is affected sympathetically, while the real explosion is the result of the sudden discharge of the pent up excitement caused in the encephalic centres by the irritation at such distant points.

5th. **Morbid States of the Circulating Blood.** Certain poisons, when brought in contact with encephalic nerve centres produce epileptic seizures. Gallerani and Lussana applied creatinin directly to the cerebral cortex and quickly induced epileptiform convulsions and choreiform movements. Injected subcutaneously it failed to produce the same effect. Cinchonoidin acted on the basal ganglia of the brain producing convulsions but no choreiform movement. Poisoning with lead, ergot, nitro-pentan, nitro-benzol and a number of other poisons brings about intermittent convulsive seizures. The same may be inferred of ptomaines and toxins, in the convulsions that appear in the advanced stages of infectious diseases (canine distemper, hog cholera, etc.).

6th. **Reflex Irritation.** Perhaps no peripheral irritation more frequently causes epilepsy, than parasites. In young dogs worms in the intestines (tænia coenurus, tænia tenuicollis, tænia serrata, tænia echinococcus, and ascarides) have been especially incriminated. Also lingnatula tænioides in the nasal sinuses. In young pigs the echinorrhynchus gigas, ascarides and trichocephalus. In horses ascarides have been principally blamed.

Wounds implicating nerves, and tumors pressing on nerves, have served as sources of nervous excitement which accumulates in the cerebral ganglia and bursts forth as an epileptic explosion. Bourgelat mentions the case of a horse which fell in a fit the moment he was touched on his tender withers, also a case in which a siezure coincided with an attack of recurrent ophthalmia. Gerlach saw a horse which had an epileptic fit the instant he was touched on his sensitive withers. In kittens and puppies the irritation attendant on dentition is a common cause of attacks. In nervous dogs and pigs indigestion or constipation may serve as the occasion of an explosion. In the experimental cases of Brown-Sequard, not only did the injury to the sciatic nerve develop in the brain a latent tendency to epilepsy, but the subsequent pinching of the skin in certain areas (epileptigenous zones) promptly brought about a siezure.

Causes. Most of the causes of epilepsy have been given above under the head of pathology and morbid anatomy. The nervous predisposition may, like any other peculiarity or function, become hereditary. In the human race nothing is more certain

than the tendency to some form of nervous disorder (insanity, dementia, alcoholism, morphinism, epilepsy, chorea, etc.) in a special family line. Reynal records the case of an epileptic cat (belonging to an employe of the Alfort veterinary school) the progeny of which for three generations, became affected with epilepsy and mostly died before they were a year old. Also four epileptic dogs (3 males and 1 female) which produced a number of epileptic puppies. LaNotte records the cases of two bulls affected with epilepsy, in the progeny of which numerons cases of epilepsy appeared ; the cows being attacked after the first calving, and the oxen soon after they were first put to work. Breeding stallions are particularly liable to attacks, the high feeding, lack of muscular work in the open air, and above all the oft repeated nervous excitement attendant on copulation being directly exciting causes. The heredity of the artificial epilepsy induced by Brown-Sequard in Guinea pigs, serves to strengthen the doctrine of heredity in ordinary forms.

Among emotional causes fear easily heads the list. Bernard states that a horse became epileptic in connection with the terror caused by the giving way of a wooden bridge over which he was passing. Bourgelat and Reynal addnce instances, in cavalry horses when first put under fire. Reynal records the case of another which had his first attack when facing a moving locomotive, and which never again could see an engine in motion without suffering another attack. La Notte mentions the case of a horse attacked when frightened by a sky rocket ; Romer, the case of a horse scared by the sudden display of a white sheet in front of him, and Friedberger and Fröhner relate cases of attacks caused by intense rays of light, as in racing toward the declining sun, or the dazzling reflection from the surface of water. Liedesdorf saw it in a dog scared by a locomotive.

A strong impression like that caused by transition from bright light into darkness, by seeing shadows of trees crossing the road, or violent suffering caused by severe forms of constraint have been named as causes.

Speaking in " Brain," of epilepsy in man, Alexander Haig attributes the fits to the fluctuations of uric acid in the blood. Headache (migraine) he finds to be very closely allied to epilepsy and convulsions and to be a result in a susceptible system

of a liberal flesh diet. By a vegetable and fruit diet he reduces the ingestion and formation of uric acid, so that the largest quantity which a patient is likely to get into his blood, shall never or only very rarely, affect the blood pressure and increase the intra-cranial circulation to a dangerous extent. In predisposed subjects, all flesh food, soup, and meat extracts must be avoided, while even tea, coffee, cocoa and other vegetable articles containing zanthin compounds are to be regarded as producing uric acid, and to be denied, or employed only as the merest flavoring.

This position is greatly strengthened by the fact that epilepsy is so much more frequent in the carnivora (dog, cat, bird) than in the herbivora. It also suggests very strongly a light vegetable diet for both prophylactic and curative purposes in our domestic animals. In the same line the frequent and liberal drinking of warm water, the use of diuretics and the flushing of the large intestine are indicated.

For other causes see under pathology.

Symptoms in the Horse. It has been claimed that premonitory symptoms, such as dullness, lack of energy and quick, nervous or startled movements herald an attack, but in animals as in man, the disease usually attacks suddenly without any antecedent indication.

If at work the horse stops suddenly, or if in the stable he ceases eating, seems frightened, stands for an instant immovable, braces his feet, sways, trembles, and falls heavily to the ground. Or he may remain for an instant supported on his rigid limbs, the jaws moving or firmly closed, the eyes rolling, and the facial muscles drawn or twitching. When down there are convulsive movements of the limbs, so that the animal may kick out violently, and tense contractions or twitchings may occur in the muscles of the croup, chest and abdomen. There is usually an increase of the salivary secretion with frothy accumulation about the angles of the mouth. The respiration is stertorous, dyspnœic, and interrupted, the nostrils widely dilated, the nasal mucosa of a dark brownish red, and the superficial veins distended. The pulse is weak, slow, irregular, intermittent and sometimes imperceptible. Sensation seems to be in abeyance. No attention

is paid to loud sounds, nor to pinching, pricking, or even cauterizing the skin. Perspirations may break out on the flank or over the whole surface of the body.

The duration of the attack may be from one to four minutes, or exceptionally ten or fifteen, after which the muscles relax, the twitching ceases, the horse raises his head, extends his fore limbs and finally rises.

After rising some are dull and stupid for an hour or so, and may continue to perspire, some move the limbs, jaws or head automatically, turn in a circle, or seek seclusion and darkness, while some take at once to eating and seem as if nothing had happened.

In *partial* or *localized* epilepsy the spasms are confined to a limited group of muscles like those of the jaws, neck, or fore limbs. These may alternately contract and relax, or they may remain rigid for a minute or less, the mouth being held open or firmly closed with grinding of the teeth, the eyes rolled backward and upward, or affected with strabismus, the face drawn and distorted, the head turned to one side or downward, or the limbs fixed and immovable.

At the conclusion of an attack it is not uncommon to see a discharge of urine or fæces, or in stallions, of semen.

The horse often contracts a fear of the place where the attack occurred, and this contributes, with the re-appearance of the former object of dread (car, locomotive, rifles, cannon, etc.) to precipitate a new attack if he is compelled to go to such a place.

Symptoms in **Cattle.** In cattle the animal is attacked without premonition, bellows, breathes hard and with effort, has dilatation of the nostrils, and squinting or rolling upward and backward of the eyes and falls to the ground rigid and trembling. There may be violent succussions of the limbs, head or neck, movements of the jaws, grinding of the teeth, and the appearance of frothy saliva and elements of food about the lips. The beats of the heart are violent, the pulse slow and small, and sometimes intermittent. Involuntary micturition, defecation, or discharge of semen may occur. In slight cases one or more of these symptoms may be absent, and the victim may not even fall to the ground but support himself against a wall or other object.

The duration of the attack may be from one to five minutes, rarely more, and there is often a slow and progressive subsidence of the spasms. When recovered the animal may get up and go to eating or rumination as in health.

Symptoms in **Sheep.** In sheep the attack is sudden. The animal ceases eating or stops in its walking, and after turning or other involuntary movement falls to the ground, head extended, mouth open, eyes rolling or squinting, and with rigidity or twitching of the muscles of the neck or limbs. There is the same loss of sensation, frothing from the mouth, and grinding of the teeth as in the larger animals. The attack may last 40 to 50 seconds.

Symptoms in **Swine.** In pigs a state of discomfort and restlessness often marks the approach of an attack, referable probably to the digestive disturbance or to parasites which furnish the occasion of the disease. Uneasy, wandering movements, jerkings of head or limbs, rolling of the eyes, and champing of the jaws may first appear. Then the animal falls, extending its limbs and head, with open mouth, retracted lips, and a free flow of saliva. Trembling and jerking of the head, neck and limbs, hurried, short, difficult breathing, and complete loss of sensation may be noted. Discharges of urine, semen, and prostatic fluid are not uncommon. The attack usually lasts 2 or 3 minutes, and exceptionally 10 to 15. In the shorter seizures, frequent repetition is not uncommon, Delafond having observed 5 or 6 attacks in the course of an hour.

Symptoms in **Dogs.** The attack is sudden and unheralded by prodromata. The animal stops, trembles, cries plaintively and falls ; he may manage to rise or to do so in part but instantly falls anew. The limbs stiffen, tremble or twitch, the head is extended or flexed, or jerked, violently striking the ground, the mouth open, with abundant saliva, or firmly closed though the tongue may be between the teeth. The trunk may be firm and rigid or alternately twisted in one direction or the other. The eyes roll or squint, and the breathing is stertorous and difficult. Insensibility is complete. Toward the end of the attack there may be a discharge of urine, fæces or semen, the stools often containing worms. The body is often wet with prespiration during or after an attack.

The attack usually lasts for two or three minutes, then the convulsions gradually lessen in intensity and finally cease, the dog raises his head, opens his eyes, and gazes inquiringly around. Then he gets on his feet shakes himself and may at once resume his customary habits. In other cases the restoration is less sudden. The dog remains for 30 to 60 minutes dull and stupid, or seems to have little power of control over its muscles and staggers as if intoxicated, or as if the muscles were benumbed. It may drop on its knees and then fall with the head on the ground and repeat this several times. In other cases the dog wanders around, or trots off and may snap at any one interfering with him, so that the case is often mistaken for one of rabies. Finally the animal may remain prostrate and fall into a deep sleep marked by stertorous breathing.

Diagnosis. The diagnosis of epilepsy is usually easy. The suddenness of the attack, the loss of consciousness the muscular spasms, the complete temporary recovery and the tendency to recur, form a *toute ensemble*, which is pathognomonic. The danger of confounding this with other nervous disorders is on the whole greatest in the slight cases in which the symptoms are less typical.

From **Syncope** it is easily distinguished by the spasms which are not present in syncope.

From **eclampsia** it is not so easy to diagnose, but the line between eclampsia and epilepsy has not been accurately drawn, and some have even shown a disposition to drop eclampsia from medical nomenclature. Eclampsia may be defined as general convulsions dependent on some eccentric irritation, and which do not recur after such irritation has been removed. This would remove from the category of epileptic attacks the cases of convulsions in which the attacks were due to intestinal or nasal parasites, dentition irritation, tumors pressing on nerves, canine distemper and other infectious diseases. So far the distinction might be made by the diagnosis of the particular disease on which the convulsions depend. There remains however a class of cases in which the centric nervous disorder on which the epileptic seizure depends is present, and also the peripheral source of irritation (worms, etc.). In such a case the presence of the worms or other eccentric source of irritation, even if added to the fact that this was the immediate exciting cause of the epileptic explosion,

could not do away with the fact that the essential conditions of epilepsy are permanently present in the nervous centres. The difficulty therefore of making an accurate differential diagnosis, resides largely in the impossibility of drawing a definite line of pathological separation between eclampsia and epilepsy.

From **Vertigo** epilepsy is distinguished by the absence in the former of marked spasmodic contractions. It is only in the milder forms of epilepsy those in which the spasmodic action is so slight as to be overlooked, that this disease can be confounded with vertigo.

From **Thrombosis** or **embolism** of the iliac or femoral arteries epilepsy is easily distinguished by the absence of exercise as the essential cause in the development of the latter. In thrombosis on the other hand, the loss of control over the hind limbs is developed at will by active motion (walking, trotting). In thrombosis too the absence of pulsation at the fetlocks or at any point below the seat of obstruction is conclusive.

Question of Soundness and Jurisprudence. Manifestly a horse or bull subject to attacks of epilepsy is *not sound*. It is moreover a disease, the symptoms of which are only shown for a very short period at one time, after a long interval of apparently perfect health. It is, therefore, a disease against which a purchaser cannot be expected to protect himself and he should have the right to annul the sale and return the animal in case the infirmity should appear within a reasonable period after purchase. This is provided for in the laws of different countries of Europe, thus in Wurtenberg, Baden and Hesse, a purchased animal may be returned within 28 days; in France within 30 days, and in Bavaria within 40 days. The greatest difficulty arises from the frequent impossibility of obtaining expert testimony on a seizure which is likely to occur at any moment, without premonition, and in which the testimony of a non-expert may easily be misleading. It seems as if complaint having been made within the specified legal time, an extension of guarantee should be given by the court, the animal to be meanwhile kept under the supervision of a veterinarian.

Another question has arisen as to the position of an animal suffering from reflex epilepsy. If the attacks are caused by intestinal worms or nasal acarina which are easily removed, it is

quite evident that this cannot be considered as a permanent unsoundness, and one for which a contract of sale can be justly annulled. But on the other hand, while the eccentric source of irritation which is easily curable may have been the active agent in developing the seizures, it may be none the less true that the central infirmity which determines the abnormal susceptibility, to excessive generation and epileptic explosion of nervous force, may also be present and the animal cannot be considered as *sound* until a sufficient length of time has elapsed after the removal of the peripheral irritation and no new seizure has taken place.

Treatment of Symptomatic Epilepsy. In cases due to an eccentric irritant the first step must be the removal of such irritant. In case of intestinal worms the various vermicides and tæniacides must be resorted to. (See Intestinal Parasites). For the linguatula tænioides the injection of benzine or tobacco water into the nose, or into the sinuses, with or without trephining may be resorted to. In diseased teeth extraction or filling may be demanded. In dentition-irritation, lancing of the gums. In all other cases in which a peripheral nervous irritation can be traced every available means should be taken to remove it.

Treatment of Central Epilepsy. Bearing in mind that peripheral irritation is a frequent exciting cause of a seizure, too much care cannot be given to the conservation of the general health and especially to make the diet wholesome in quantity, quality and time of feeding and watering, and to guard against constipation and indigestion. In dogs a too stimulating meat diet is to be avoided.

The medicinal agents employed have been mainly such as are sedative, or tonic to the nervous system. Valerian was long extolled as a valuable remedy (Gohier, Delafond, Delwart), and this has been improved upon more lately by substituting valerianate of zinc. Belladonna and its alkaloid atropia have been strongly advocated (Tisserant, Bernard, Williams, Friedberger) and it has the recommendation that it causes vaso-motor contraction and tends to lessen cerebral congestion. Hyoscine or duboisine may be used as a substitute. Cynaide of iron has been lauded by Jourdier and Tabourin, as far superior to valerian. Of late years the nerve tonics, zinc compounds (oxide, sulphate,

chloride) and silver salts (nitrate) and arsenic have been used, often with excellent results. Borax strongly recommended for man (1 to 1¾ drachm daily) by Pastena is worthy of a trial for dogs. It is given largely diluted in syrup to avoid gastric irritation.

Of all agents employed up to the present the bromides still claim a foremost place. They should be given in a large dose, on an empty stomach and at such a time as to occupy the system at the hour when the seizure is expected to recur. Thus for morning attacks the dose may be given at night, while for night attacks it may be given in the afternoon. Müller uses sodium bromide in the dog as least liable to disturb the stomach, while Peterson, for man, advises the potassium salt for the same reason. For man, McLane Hamilton advocates a combination of the sodium and ammonium salts, Eulenberg adds the potassium compound, while Berkley uses strontium bromide, and Bourneville camphor monobromide.

Given at night in full dose (30 grs. for dog) the bromides tend to secure a quiet sleep, with brain rest and recuperation. If beneficial they should be repeated daily until a cure or other sign of bromism appears. This may be somewhat checked by arsenic or chloral hydrate.

Wesley Mills finds potassium iodide useful in some dogs when bromides fail. Bromohydrate is advocated by Müller. Flechsig and others have had excellent results in man from the opium bromide treatment. Full doses of opium are given three times a day for six weeks, when they are replaced by full doses of bromides four times a day.

Improvement should be shown in the shortening of the convulsions and the lengthening of the intervals between them. Should the bromides fail in this, resort may be had to other treatment,

Toulouse, Clark and others find that privation of salt, in man, allows the bromine salt to replace the chlorine one in the tissues, and the hydrobromic acid the hydrochloric in the gastric juice, and in this way the bromine can be introduced safely in larger amount into the tissues and is longer retained, though given in half the doses.

A most important element in the treatment is a vegetable diet with or without milk, to obviate excessive production of uric acid. Anything which will disagree and produce gastric or intestinal fermentations with toxins must be carefully guarded against and these will differ in different individuals.

Stallions and other excitable males, and females may often be cured by castration. Patients should be very carefully guarded against all sources of excitement, reports of guns, sight of locomotives or automobiles, waving flags, instrumental music, sudden exposure to sunshine or other bright light, reflection from water, snow, or ice, the contrast of dark shadows, as of trees, alternating with bright light, etc. Dogs, becoming excited at a show, may have a convulsion if not removed, and much more so in presence of another dog in a fit.

A surgical operation often places the disease in abeyance for many months, but, unless in the case of the removal of a diseased organ which has acted as a factor, this is not permanent. Hence in man transient benefit has been secured from operations on the eyes, the brain, the testicles, the ovaries, etc. In local (Jacksonian) epilepsy, which can be traced to a definite cortical area in the brain, the trephining of the skull and the excision of the cortex at that point, has given temporary relief, with a local palsy, but too often the irritation from the resulting cicatrix has in time aroused the disorder anew. Even independently of the removal of the cortex, the trephining has been successfully resorted to, by savage as well as civilized peoples, securing a temporary relief. Though not in practice in veterinary medicine it seems as if this were even more applicable than in man. It would be fully justified if it preserved for a year or more an animal in usefulness which must otherwise be destroyed, even if the disease should return at the end of this time.

Plunge or douche baths (60° to 70° F.) and rubbing dry will often tone up the nervous system, and a course of bitters, or iron, or both, may prove valuable. An out-door life and moderate muscular exercise are important.

During a convulsion the animal should be freed from all harness, halters, girths, etc., that would impair respiration, the jaws may be kept apart with a cloth to prevent biting the tongue, and the animal held with head and neck in natural position.

To arrest the spasms the best agent is amyle nitrite inhaled from a handkerchief. It may be replaced by a mixture in equal parts of chloroform and ether. Or rectal injections may be given of chloral. Nitro-glycerine will sometimes cut short an attack or prevent it. Small animals may have the body immersed in a warm bath, and cold applied to the head. Congested buccal and conjunctival mucosæ would indicate cold to the head, while pallor would suggest warm fomentations.

When the fit is over the animal should be kept in a quiet, dark place until the excitement or stupor has completely passed.

ECLAMPSIA. CONVULSIONS. SPASMS.

Definition: functional convulsions from peripheral irritation. From dentition, helminths, uterine disease, nursing (anæmia). Treatment. Injuries to cranial bone.

Eclampsia (convulsions) is difficult to define as distinct from epilepsy, the present tendency however appears to be to apply this term to cases in which the spasms are of a purely functional nature as far as the brain is concerned and caused by peripheral nervous irritation. Whereas in epilepsy there is some organic disease or disorder of the brain itself. Therefore the convulsions of anæmia, of teething, and of parasitism, would come under this heading being curable by the removal of the distant source of irritation, while the spasmodic seizures, that are due to central nervous lesions and are not exclusively dependent on peripheral irritation would be classed as epilepsy. In other words reflex epilepsies with no central brain disorganization would be classed as convulsions.

Eclampsia from **Dentition** is seen especially in young cats, dogs and pigs when cutting-teeth, and may be obviated by lancing the gums, extracting diseased or milk teeth entangled on the crowns of their successors, and by a slight laxative with bromides.

Eclampsia from **Helminthiasis** has been already referred to under epilepsy. The main object is the expulsion of the worms, after which nerve sedatives and tonics will be valuable.

Eclampsia from **Uterine Disease** has been noticed by Albrecht, in cows shortly after calving, the symptoms being spasms of the neck, persistent extension or turning of the head, grinding of the teeth, loss of consciousness, convulsive movements of the legs, rolling of the eyes, and slow recovery. The same symptoms have been observed in goats and have been supposed to depend on a reflex from the irritated womb. Another supposable cause is the absorption of toxic products from the womb and vagina. Manifestly the removal of the after birth and the disinfection of the womb, should be here employed along with the ordinary nerve sedatives.

Eclampsia in **Nursing Female Dogs** has long been attributed to anæmia by English veterinarians. It occurs especially in high bred bitches, when nursing a large litter and some weeks after parturition when the puppies have grown large and vigorous, with proportionately increased demands on the maternal source of supply. The dam shows an emaciated aspect, with restless anxious eyes, a wearied expression, and a generally exhausted appearance. There is weakness and swaying behind, or complete inability to use the limbs, the animal goes down, trembles violently and shows clonic spasms of the extensors of the legs, the neck, the back, the face and the eye. Breathing is accelerated, stertorous and labored, the heart beats violently the mucous membranes are congested, and the mouth is opened with convulsive movements of the jaws and throat. The attack is readily distinguished from epilepsy, by the retention of sensation, and by the absence of involuntary passages from the bowels, or kidneys. Recovery is likely to be secured if the puppies or most of them are removed early enough and the bitch sustained by nourishing food, and tonics. The spasms may be combatted by the antispasmodics and nerve sedatives employed in epilepsy. Chloroform, morphia, phenacetin, acetanilid, urethane have been specially commended. Beef teas, cod-liver oil, and iron may be resorted to and free outdoor exercise and sunshine should be secured.

General convulsions are common in connection with direct injury to the brain and more particularly of its coverings, (cranial bones, meninges). In such cases the irritation which otherwise starts at a distance and reaches the brain through the afferent nerves, or the modificaton of the circulation acts directly on the

gray matter. It is interesting to note in this connection that the evacuation of the cerebral fluid, which removes the soft support of the water cushion and allows the brain to come in contact with the hard bony walls, determines an access of convulsions. In cases of convulsions attendant on mechanical injury to the cranium surgical interference will be in order.

CHOREA. ST. VITUS DANCE.

Definition. Susceptible animals. Causes: nervous lesions inconstant, youth, debility, anæmia, microbian toxins, cerebral embolism, rheumatism, trophic alterations in nerve cells, fright. Lesions: variable in seat and character, congestion of perforated space, corpus striatum, Sylvian convolutions, gray matter at root of posterior horn of spinal cord, etc., experiments of Chauveau and Wood. Symptoms: **dog**, local twitching, fore limb, one or both, neck, head, maxilla, eyelids, eyeballs, hind limbs, trunk, rhythmic, less when recumbent, usually absent in sheep, roused by excitement: **horse**, head neck, fore limb, trunk: **cattle**, head, neck, limbs: **swine**, hind limbs, neck, head: severe cases lead to exhaustion, emaciation, marasmus, paralysis. Duration: weeks, months, years. Treatment: laxative, tonic, hygienic. arsenic zinc sulphate, strychnia, sedative, belladonna, conium, cannabis Indica, chloral, acetanilid, trional, etc., icebags or ether spray to spine, cold douches, outdoor life.

Definition. A neurosis characterized by constant twitching of muscles or of groups of muscles, and which usually ceases during sleep.

Animals Susceptible. This disease is especially common in the dog, but has been recognized also in the horse, ox, cat and pig.

Causes. Much difference of opinion exists as to the true cause of chorea. In many cases no nervous lesion has been found and therefore the disease has been pronounced purely functional. The victims are as a rule the young, weak and debilitated so that anæmia has been held to be the main causative factor. Then in dogs the affection is a common sequel of distemper and hence it has been attributed to toxic matters (microbian, etc.) in the blood. It should be added that European writers attribute the rhythmic spasms which follow distemper to eclampsia, epilepsy or tic, and claim that the contractions must be irregular or arhythmic in order

to constitute chorea. English and American writers, however, have attributed less importance to this point and consider that the constancy and persistency of the contractions in the dog, differentiate an affection from both eclampsia and epilepsy and relate it rather to chorea. Tic as illustrated in crib-biting is certainly not constant nor rhythmical nor is it a habit beyond the control of the will.

Among other alleged causes of chorea is embolism of the arteries of the brain or spinal cord. Angel Money went so far as to inject a fluid containing arrowroot, starch granules and carmine into the carotids of animals, and produced movements closely resembling those of chorea. Another theory connects chorea with the rheumatic poison. Some English writers find more than 80 per cent. of all cases in man associated in some way with rheumatism, but in Philadelphia, Sinkler found that not more than 15 per cent. showed such a relation. D. C. Wood as the result of necropsies of a number of choreic dogs reached this conclusion : "Owing to emotional disturbance, sometimes stopping of various vessels of the brain, or sometimes the presence of organic disease, there is an altered condition of the ganglionic cells throughout the nerve centres. If the cause is removed and the altered condition of the nerve cells goes only so far, it remains what we call a functional disease. If it goes so far that the cells show alteration, we have an organic disease of the nervous system."

In man the element of sudden fright is awarded a high position in the list of causes.

Lesions. Constant morbid changes of structure have not been established in chorea. On the contrary in the many careful necropsies of choreic subjects some lesion of brain or spinal cord has been almost always found. Dickinson always found congestion of some part of the brain or spinal cord, but most constantly of the substantia perforata, the corpora striata and the beginning of the Sylvian fissure. In the cord the cervical and dorsal regions were the most commonly affected, and preëminently the gray matter at the root of the posterior horn. The bilateral symmetry of the contractions in cases of unilateral lesions, has been held to discredit the theory of embolic origin, yet this may

be explained by mutual relation of the ganglia of the two sides and their coördination of function.

The question of the relative importance of the encephalic and spinal lesions has been also debated. Chauveau believed that by section of the cord in choreic dogs, he had proved that the spasms were of medullary origin. Wood on the other hand found that the choreic movements persisted after section of the cord, and seemed warranted in the conclusion that the movements originated in the cord. The probability is, that with the cord intact, the primary source of the morbid movement may reside either in the encephalon or the cord. In chorea, following distemper, I have found marked congestion of the encephalon and its meninges.

Symptoms. In the dog the twitching may be confined to one fore leg, or it may extend to both and then usually implicates the neck and head. In other cases the lower jaw, the head, the eyelids or even the eyeballs may be the seat of the twitching motions and in still others the hind limbs and trunk are also implicated. In nearly all cases the tendency is to a continuous rhythmic action, which may moderate without actually ceasing while the animal lies down, but which usually stops altogether during deep sleep. When it intermits at other times it can commonly be roused into activity by exciting the animal in any way.

In the horse the muscles affected may be those of the head and neck, of the fore limb (Hering), of head, limbs and trunk (Leblanc).

In cattle the movements have affected the neck and head and the fore or hind limbs (Anacker, Schleg).

In young pigs the hind limbs, the neck and head have been chiefly involved in the spasms (Hess, Vervey).

If the affection is slight it may not seriously impair the genera health, but in proportion to its severity and the constancy and generalization of the spasms and the consequent muscular waste and exhaustion, the animal becomes gradually worn out and emaciated and dies of paralysis and marasmus.

Duration. In favorable cases recovery may ensue in a few weeks; in others the disease becomes chronic and will last for months or years.

Treatment. Among the first indications are fresh air and wholesome easily digestible food. The removal of any source of intestinal irritation should be sought by bland laxatives. As the health is usually low, a course of iron tonics is nearly always in order.

Special nervous tonics are next demanded. No agent has a better reputation than arsenious acid. Ten drops of a 1 per cent. solution of arsenite of soda may be given daily to the dog or one ounce to the horse. If gastric irritation forbids the use of this agent, sulphate of zinc may be given in 1 to 3 grain doses twice a day to the dog. In other cases strychine $\frac{1}{80}$ to $\frac{1}{60}$ of a grain may be given in the same manner.

Nerve sedatives often have a good effect in calming the nervous irritability and in this way belladonna, conium, cannabis Indica, chloral, chloroform, acetanilid, trional, etc., have been employed.

In the same line are ice bags or ether spray applied to the spine for ten minutes at a time, and douche baths of cold water, the animal being afterward rubbed dry. This last partakes of the nature of a stimulant and may be classed with gentle exercise which at once distracts the patients' attention from the nervous infirmity, trains him to control the muscles, gives normal exercise and tone to the enfeebled organs and improves the general health.

CONVULSIVE TWITCHING OF THE FACE.

Nature of phenomenon, arhythmic, hyperexcitability of nerve centres or skin. Nerve section. Head jerking: **horse,** hard bit, severe check, internal pain, exertion, hypersensitiveness. Treatment: nerve section. Tongue lolling, etc. Flapping of lower lip. Nasal rhythmic movements. Weaving, from impatience, rhythmic. Rocking on hind limbs. Resting foot on coronet. Pawing. Treatment: eliminate irritation, nerve tonics, sedatives, hygienic measures.

This has been observed especially in the dog and may be easily confounded with chorea. The muscles on one side of the face, are twitched at more or less regular intervals, or in other cases there are sudden opening and closing of the lower jaw.

The affection has not been satisfactorily connected with any special brain lesion, though as in chorea proper and epilepsy, we must invoke a special disorder or hyper-excitability of the nerve centres presiding over the affected muscles. The clonic spasm may in some cases be due only to a motor impulse from such excited nerve centre, while in others it may be traced backward along the afferent nerves to an oversensitive part of the skin or other organ. In these last purely reflex forms of the trouble it may be possible to correct it by section of the sensory nerves involved.

Convulsive Movements of the Head.

Convulsive movements of the head as a whole constitute a frequent form of chorea in the dog. It is especially common in horses and shows itself in different forms. A horse with a tender mouth, or which has been used with a hard bit, or with a heavy hand on the reins, or which has been driven with a check rein so short as to be unsuited to its conformation, is liable to indulge in annoying elevation and depression of the head when under the saddle or in harness. The same phenomenon may be shown in connection with violent internal pains, as in strangulated hernia, intussusception, or twisting of the bowels. The habit once formed is not easily corrected, so that careful treatment with the view of prevention is especially to be given.

Another more objectionable, dangerous, and less voluntary motion is the sudden jerking of the head upward, or to one side when excited under the saddle or in harness. The disorderly movements are not, as a rule, seen while the animal is at rest, but seem to be produced under the stimulus of exertion. They appear to be quite involuntary, and suggest the dread caused by the settling or buzzing of an insect about the nose or ears, but occur in the depth of winter in the absence of insect life, as well as in midsummer. The suddenness and involuntary nature of the movement is suggestive of epilepsy, but there is no indication of attendant unconsciousness. From choreic movements it is apparently distinguished, by its presence only when ridden or driven. It is unquestionably associated with hypersensitiveness of the nerve centres, and yet in many cases it appears to be a reflex originating in a specially tender or sensitive part of the

skin or mucous membrane. In more than one instance in this college clinic the trouble was corrected by the section of both facial branches of the 5th cranial nerves as they emerged from the infra-orbital foramina.

Abnormal Movements of the Tongue.

Some horses double the tongue downward, others upward of the bit; others protrude the tongue and give it a sinuous, serpentine motion which causes alternate protrusion and retraction.

Flapping of the Lower Lip.

This habit of rapid opening and closing of the lower lip so as to produce a disagreeable flapping noise by striking it against the upper, is seen in many horses and proves a most objectionable trait in harness or saddle animals.

Rhythmical Movements of the Nose.

Certain horses apply the protractile end of the nose against the lower lip and spend hours in succession in moving it rhythmically forward and backward, or from side to side.

Weaving. Movement like a Bear in a Cage.

This consists in a lateral rocking of the head and neck, and sometimes of the chest as well with alternate stepping on the right and left fore feet. It has been supposed to represent the movement of the weaver in working a hand loom, or still better the movement of a caged wild beast in constant turning toward the right and left of the front of his cage. The motions are as regular as a pendulum, and involve the contraction of corresponding groups of muscles on the two sides of the body.

They seem, in some cases, to begin in impatience in waiting for the feed, while other horses in the same row are being attended to first, but when the habit has been formed it may be continued most of the time in the intervals between feeds as well. Nervous horses and those that are hearty feeders are the most subject to this infirmity.

Disorderly Movements of the Limbs.

Some horses have a habit of continuously raising one hind limb, others raise the right and left alternately, rocking the hind quarters from side to side, others stand with the heel of one hind foot resting on the front of the coronet of the other, while still others paw continuously with the fore feet while standing in the stall.

Treatment. These various conditions even when begun as an expression of impatience, soon become fixed habits, that prove in the end virtually uncontrollable by an animal, which has no strong will and no consciousness of anything to be gained by resisting the impulse. They become virtual psychoses. In cases in which the habit can be traced to a peripheral irritation, the cutting off of this by complete section of the afferent nerves leading to the irritable nerve centre will sometimes succeed in effecting a cure. In other cases in which the source of the disorder is probably largely central in the cerebral ganglia, nerve tonics, and sedatives, and generally corroborative treatment are the most obvious means of palliation. Such measures are, however, rarely successful. Nourishing food and invigorating outdoor exercise are useful auxiliaries.

VERTIGO. MEGRIMS. BLIND STAGGERS.

Disadvantage of lack of subjective symptoms. Causes, varied, narcotics, overloaded stomach, cerebral anæmia or hyperæmia, degenerations, parasites, tumors, jugular obstruction, valvular heart disease, disease of internal ear, plethora. Susceptible animals, horse, ox, dog, pig, sheep. Direct causes: tight collar, or throat-latch, flexion of head, heart disease, pulmonary disease, embolisms, gastric distension, hepatic disorder, optic vertigo, aural vertigo, injections into ear, rhigolene, chloral, acariasis, seasickness, railroad sickness, cholesteatoma, cœnurus, concussion, degeneration, softening, œstrus, linguatula, narcotics, essential oils: essential vertigo. Symptoms: in irritable animal, highly fed, and without exercise, crowds pole, his mate or a wall, shakes or jerks head, staggers, trembles, rears, plunges, falls, struggles, sweats, rolls eyes, recovers. In gastric or hepatic cases, dullness, pendent head, swaying gait, dull eye, dilated pupil, pendent lids and lips, leans on adjacent object, staggers, falls. In optic cases are obvious cause in transition to light, etc., and palliation by covering the eyes. In aural cases, roll-

ing eyes, constrained position of ear, deafness, pharyngeal or Eustachian trouble, wax or acari in ear, tender or itchy ear. Plethoric cases in spring, in overfed, etc. Brain lesions may have fever and disordered innervation, but retained consciousness, and no marked spasm. Duration. Sheep : parasitic vertigo. Turning. Rotation. Treatment : according to cause : restrict ration, give exercise, purgative, adjust collar, breast strap, check, avoid sudden transitions of light, overdraw check, blinds, treat nasal, pharangeal, ocular or aural trouble ; during attack, stop in shade, cold to head, deplete, bleed, purge, shady pasture or light work, bromides, blisters, etc.

In dealing with vertigo or giddiness in animals we are confronted by the impossibility of realizing the subjective feelings of the animal, as we can so easily ascertain by interrogation in the case of man, and thus our conclusions are largely inferences drawn from certain unsteady, reckless or uncontrollable movements, or from an apparent inability to maintain a stable equilibrium. The condition is rather a symptom of a variety of morbid conditions, functional and structural, than a disease *sui generis*. It may be due to alcoholic or other narcotic intoxication, to overloaded or otherwise deranged stomach, to shock, to a stroke of lightning, to disturbances—anæmic or hyperæmic—in the circulation in the encephalon, to degenerations, parasites or tumors in the brain, to compression of the jugular veins, to valvular or other disease of the heart, to disease of the internal ear, to the plethora of spring or early summer, to the qualms of sea sickness, to insolation.

The purely toxic cases are more clearly defined and temporary so that they may be eliminated from consideration at present, yet their possible occurrence must always be borne in mind by the practitioner especially when called to pronounce upon cases of vertigo in connection with veterinary legal questions. The cases that are due to a persistent neurosis, or to circulatory troubles may well be placed in a list by themselves, yet in their legal relations it is highly important that the practitioner should as far as possible discriminate among these as well.

Susceptible Animals. Vertigo undoubtedly exists among all domestic animals. The symptoms by which it is recognized have been noted especially in the horse and much less frequently in ox, dog, pig and sheep.

Among *horses* it especially attacks the mature or aged, and family harness horses, pampered and irregularly exercised ; (saddle horses rarely suffer); it is more likely to appear for the first time in spring though when established it happens at all seasons ; it may come on when a horse is driven in blinders and fail to appear in the absence of these.

Causes. 1st. **Compression of the jugular veins** by a too tight collar is the cause of one of the simplest forms of vertigo and is observed, in growing or fattening animals in which the neck has become gradually too large for the collar. The supply of a larger and well fitting collar will soon confirm the diagnosis by a complete and permanent removal of the trouble. In other cases the veins may be compressed by undue flexion of the head, the chin being drawn toward the breast, or by a throat latch buckled too tightly. The substitution of an overdraw check rein, or a loose throat latch will show the true source of the trouble.

2nd. **Disease of the valves** *of the heart* or their insufficiency from cardiac dilatation is a common cause of vertigo, and may be recognized by auscultation and by the general symptoms of chronic heart disease.

3d. **Disease of the lungs** interfering with the flow of blood through the right heart and more distantly with the return of blood from the brain. It further effects the brain functions through the circulation of a highly carbonized blood, which fails to maintain the normal functions of the ganglia.

4th. **Disease of the blood vessels**, it may be by emboli washed on from clots in the pulmonary veins or the left heart and arrested in the vessels of the brain ; it may be by aneurism of the anterior aorta as reported of a horse (Lustig); it may be by phlebitis and thrombosis of the jugulars ; it may be by adjacent tumors pressing on the vessels.

5th. **Gastric Vertigo, Abdominal Vertigo,** is a complication of gastric or hepatic disorder with giddiness and unsteady movement. The abdominal disorder may be at once a cause and result of the vertigo and it is not always easy to decide which predominates. The unsteady movements in certain cases of overloaded stomach, in the horse are illustrations of purely abdominal vertigo, while on the other hand in vomiting animals nausea, retching, emesis, and other gastric disorders promptly attend on

the primary cerebral disorder. There is also a special tendency to vertigo in the fat, idle, gorged horse and in those with torpor or other disorder of the liver occurring in pampered horses in spring and early summer.

6th. **Optic vertigo** is a reflex disorder, determined in the excitable nerve centres by the visual influence. Thus it has been seen in horses and sheep from the intense glare of the sun's rays, reflected from a lake or river or from white snow or ice, or even from the glistening inner surface of the blinds. The effect is intensified if the animal has just emerged from a dark stable or a darker mine. The overdraw check may be a factor by reason of its turning the eyes upward and exposing them continuously to the full glare of the sun. The sense of motion conveyed through the eyes contributes to bring on giddiness and a sense of swimming. In man this is notorious, the sense of nausea and vertigo being precipitated by looking at the nearby, moving objects in cabin or on deck, while it may be retarded by directing the eyes to steady distant objects. As dogs, horses and other animals suffer from seasickness, and even railroad sickness, this attendant factor may be logically accepted. The mere limitation of the field of vision, by the use of blinds, and the disappearance in rapid succession of near objects behind this narrow screen probably has an influence similar to the visible motions in the ship between decks, in cases in which these portions of the harness are manifest factors.

7th. **Aural Vertigo** is determined by irritations of different kinds affecting the external, middle or internal ear. Experimental sections show that this is especially due to injuries of the semicircular canals. If the *horizontal canal* is divided there are pendulum-like movements of the head alternately to the right and left, also lateral rolling of the eyes. If the *posterior canal* is cut there is a vertical movement, or nodding of the head and vertical rolling of the eyes. If the *superior vertical canals* are injured there are pendulum-like vertical movements of the head and the animal tends to fall forward. Injury to the *anterior canal* causes diagonal rolling of the eyeball. In destruction of *all the canals* various pendulum-like movements are performed, and standing often becomes impossible. Stimulation of one auditory nerve is followed by rotation of the eye and rotation

of the body on its axis toward the injured side. The passage of a galvanic current through the head *between the mastoid processes*, or *from one external auditory meatus to the other*, causes rolling of the eyeballs. *Injection of water violently into a rabbit's ear*, or of *iced water* or of a *rhigolene* jet, causes rolling of the eyes, and rotation of the body toward the side operated on. Dr. Weir Mitchell had a similar experience in his own person. If the injections are repeated a permanent vertiginous condition is induced, and the rabbit or Guinea pig, which has been kept in darkness for a few hours and is then suddenly exposed to sunlight, is unstable on its limbs for a few seconds. Lucæ found that with perforation of the membrana tympani, an *ear air douche*, at 0.1 atmospheres caused abduction of the eyeball, dyplopia, giddiness, sense of darkness, and disturbed respiration. Vulpain found that a 25 per cent. solution of *chloral hydrate dropped into the ear* of a rabbit caused vertiginous movements. McVey records the case of a music teacher who had intense vertigo induced by the *low bass notes* of a piano. Crum Brown noticed that if a person with bandaged eyes, is rotated for some time as on a potter's wheel, he can at first estimate the degree of rotation, but after a time he fails to do so, and the rotation may be stopped, without checking his sense of whirling. The familiar method of subduing an intractable or vicious horse by *running* him rapidly around *in a very narrow circular course*, or by tying head and tail together and letting him circle around until he staggers or falls, is another manifest example of this aural vertigo. Rabbits and dogs suffering from *acariasis of the external ear* move around in a circle, or even turn somersaults tending toward the affected side. Trasbot has found larvæ of insects (simulium cinereum?) in the ears of vertiginous horses, which he successfully treated with injection of chloroform. Even hard pellets of wax pressing on the tympanic membrane have been found to give rise to vertigo.

The explanation of cases of aural vertigo, has been sought in the physiological action of the endolymph and perilymph on the end filaments of the nerve in the membranous labyrinth, the turning of the head from one side to the other having the effect of changing the pressure in different parts and establishing currents by which the change of position is recognized; on the

other hand any injury to the canals, by disturbing the pressure of the perilymph and interfering with the relative position of the canals, and the direction and force of the currents of the endolymph and perilymph, destroys all proper sense of balance. The rotation of the subject as on a wheel or in turning in a narrow circle, is held to cause circular currents in and around the membranous labyrinth which temporarily destroy all sense of equilibrium. Seasickness and railroad sickness are doubtless in part due to the swaying motions causing disturbance in the canals. The intimate relation between the root of the auditory nerve and that of the vagus in the medulla, may serve to explain the mutual interdependence of derangement of the stomach and liver on the one hand and the occurrence of vertigo on the other. Again the relation of vertigo to visual troubles, both as to cause and effect, has been attributed to the close relations of the ganglia presiding over the 3d, 4th, 5th and 6th nerves and those of the nerve of hearing.

There is a degree of deafness in nearly all cases of aural vertigo, a circumstance which may be utilized in the diagnosis of such cases, the presence of disease of the guttural pouch, or Eustachian tube, and evidence of deafness on one side rather than the other, may be taken as corroborative evidence of the affection. On the other hand James shows that the stone deaf are much less subject to both seasickness and vertigo than those that hear. The disorder that leads to vertigo implies a retention of a measure of the normal function of the internal ear, and therefore of hearing, whereas the disease that has caused complete destruction of the internal ear and consequent loss of hearing has equally destroyed the function of the labyrinth in maintaining a sense of balance, and has obviated the aural sensations of equilibrium and loss of balance.

8th. **Cerebral Vertigo** may be associated with derangement of the circulation, or disease in the brain or its meninges. Anæmias and congestions resulting from disease or impaired function of heart, lungs, arteries or veins have been already noticed. It remains to note the presence of cholesteatomata and other tumors, and of parasites (cysticercus cellulosa in pig; cœnurus cerebralis in sheep) in the encephalon, and of injuries from concussion, degeneration and softening of the brain substance.

9th. **Nasal Vertigo** has been noted by Cadeac in connection with irritation in the nose and especially by the larva of oestrus ovis (sheep), and the linguatula (dog, horse). In certain of the cases manifested by jerking of the head and diverging to one side, the recovery after section of the pes anserina indicates a nasal origin.

10th. **Vertigo from Narcotics** (alcohol, lolium temulentum, belladonna, solanum, various essential oils, carbon monoxide, etc)., have been already referred to.

11th. There remains to be noted cases in which no narcotic poison, no mechanical disturbance of the circulation, no visual, aural nor nasal trouble, no gastric nor hepatic disorder, and not even a distant nervous lesion can be found; the disease may in such cases be ranked for the present as **essential vertigo.**

Symptoms. As usually seen in the horse, vertigo often attacks the nervous, irritable animal in which the slightest occasion of irritation or disturbance causes intense suffering and quick response. This is often aggravated by the plethoric condition of the animal, kept on a liberal ration of grain and having little exercise. In some forms of the affection, however, and especially the gastric and hepatic, the subject is dull, carries the head low, and lacks vivacity and energy.

In the first form (the most common in the horse) the animal which has been full of life and vigor, slackens his pace and the tension on the reins, or stops suddenly, shakes the head, vertically or horizontally, or jerks it to one side, trembles, staggers, props his legs outward for more stable support, presses against the pole, or the other horse, or a wall, leans on the breeching or hangs on the breast strap, plunges forward, or to one side, or rears up and even falls backward, and comes to the ground. Profuse perspiration ensues, the eyes roll, the face is pinched and drawn, the prostrate animal may struggle in a helpless way, and, if the harness is loosened, he may get up in a few seconds and slowly recover. Often, however, he remains for an hour or more, nervous, sensitive, bedewed with sweat, trembling and with anxious expression. Such are the more common manifestations of what is familiarly known as blind staggers. The symptoms will vary however, with the cause.

In **gastric** or **hepatic** cases there may be more particularly

dullness and lack of energy, low carriage of the head, unsteadiness of gait, lack of lustre in the eye, pupils dilated, semi-closed eyelids, pendent lips, a tendency to lean on the stall or hang on the harness, and though the animal may stagger and fall, there is not the abrupt transition from life and energy to the active excitement and uncontrollable movements. The same remarks apply in a measure to narcotic vertigo.

With **optic vertigo**, the attendant conditions will help to a diagnosis. The animal has come from darkness to full sunshine; there is the white, icy or snowy reflection everywhere which the animal was facing when attacked, or the glistening lake or river, the overdraw check rein, or the blind with perhaps a shining inner surface. The pupil is closed, and the eye is rolled back or oscillates in one direction or another. The symptoms are checked by covering the eyes or removing the subject into a dark building or even into the shade of a tree or shed.

With **aural vertigo** there may be similar rolling of the eyes, without the pupillary closure, the ear may be drawn down or back, and the shaking or jerking of the head is likely to be a marked feature. If there is more motion of one ear than the other, if the head is jerked to the one side, if there is a measure of deafness in the one ear (to be ascertained rather in the intervals between attacks), if there is disease of the pharynx, the Eustachian tube or pouch, or swelling about the root of the ear, if there is wax, scurf, or acarus in the ear, if the animal rubs it frequently, aural vertigo may be suspected.

Nasal vertigo. Those forms in which the head is jerked horizontally, vertically, or diagonally, the animal pressing against its mate or the pole, or outward in the harness, and getting out of its track, even if it should stop short of falling, and which appear only during work, or are aggravated by exertion, have been attributed to lesions of the ear (Fleming), but in some cases they can be warded off by wearing a net over the nostril, and can be entirely stopped by complete transverse section of the pes anserina, so that in a certain number at least they must be accounted nasal. These are not usually attended by sneezing. The simple expedient of driving with a rather close net over the nostril may enable one to diagnose many of the purely nasal forms.

Plethoric vertigo may be suspected when the attack comes on

in spring, in a fleshy or fat horse, over-fed and little exercised, when there is dark red congestion of the nasal mucosa and conjunctiva, and a subsidence with rest.

In the *vertigo* of brain lesions, the acute forms are attended by fever and marked signs of delirium or disordered nervous functions, while in the chronic forms there may be permanent hyperæsthesia or anæsthesia, general or with rather diffuse limits, and the vertiginous attacks repeat themselves frequently irrespective of weather, though they may be precipitated by faults of feeding, indigestion, severe exertion, or some of the other exciting causes above mentioned.

These cases are to be distinguished from epilepsy by the absence of any spasmodic contraction, aside from the jerking of the head and rolling of the eyes, and by the fact that consciousness is retained throughout. During the attack the animal may fail to respond to irritation of the nasal mucosa, but this appears to be due to the fact that his whole attention is engaged with a more serious trouble.

The duration of an attack is from one to two, or exceptionally five minutes. The form which is represented by jerking of the head and deviation from the direct line of motion may continue so long as exercise is kept up.

In the **nasal vertigo** of sheep and dogs, due to parasites, sneezing, and congestion of the mucosa are to be looked for.

In the **cerebral parasitic vertigo** of sheep and pigs, the symptoms vary according to the seat of the parasite. These may be blindness, turning in a circle, moving straight ahead regardless of obstacles, jerking upward of the head with nose protruded, hemiplegia, hemianæsthesia, cross hemiplegia, cross hemianæsthesia, and any one of the many forms of paralysis, or exaggerated nervous action. The animal usually turns to the side on which the parasite lies and is paralyzed on the opposite side of the trunk. A peculiarity of these cases is that while the symptoms are continuous, yet there are periodic aggravations which bear no relation to feeding, exertion or excitement, but depend on the protrusion at intervals of the heads of the parasites into the brain substance. If there are several parasites in the brain and they do this at different times the symptoms are liable to vary according to their seat, and the special organ which is irri-

tated. This variability of symptoms is suggestive of parasitism.

While turning around in a circle has been already noticed there remains, in certain cerebral forms, the peculiar phenomenon of the animal rotating rapidly on its longitudinal axis. The patient falls on its side and rolls over and over. Among the brain lesions with which it has been experimentally identified are injuries to the *middle peduncles of the cerebellum*, or of the *supero-external portion of the cerebral peduncles*, or of the *posterior part of the encephalon*, or of different parts of the hemispheres.

Cases of vertigo that occur without any appreciable lesion have been named *essential vertigo*. Guibert has attributed some cases to irritation of the lower part of the limbs by contact with the litter but this could only occur in an animal in which the nervous system was in a morbidly excitable condition.

Treatment. The prophylactics and therapeutics of vertigo will vary with the cause. The diagnosis of the cause is therefore the most important step. In pampered, overfed, idle horses a reduced ration and daily exercise or work will often suffice. It is usually desirable, however, to remove intestinal irritants and deplete the vascular system by an active purgative. If the attacks appear only in Spring this care should be especially given at such seasons. A tight or badly fitting collar should be corrected, also a position of the breast strap which causes it to press on the jugular veins. A short bearing rein causing undue flexion of the head must be lengthened or abandoned. A too dark stable should be avoided, also the sudden exposure to white, dusty roads and, still more so, to the glare of snow, ice, or water. A short, overdraw check rein, turning the eyes up directly into the sun's rays, or blinds with a glistening inner surface may require correction. It may be better to abandon blinds altogether, or to cover the eyes by a piece of leather, 2 to 4 inches wide, extending across the forehead from one eye to the other; or a sunshade attached to the headstall may be worn so as to protect the eyes. Horses which become seasick or carsick may sometimes be helped by covering the eyes. Other indications would be to treat any existing trouble which interferes with a normal circulation in the brain (pulmonary congestion, aneurisms, tumors pressing on carotids or jugulars, phlebitis, etc.), and such as affect the ear (disease of the pharynx, guttural

pouches, adjacent glands, petrous temporal bone, membrana tympani, external ear). Indurated wax, insects or insect larvæ may be removed by careful irrigation with warm water, and perhaps by chloroform. Nasal parasites must be washed out or destroyed by benzine, and any hyperæsthesia of the nasal mucosa may be met by covering the nostril with a net, or radically by cutting the facial branch of the 5th nerve as it emerges from the infra-orbital foramen.

When attacked the horse should be at once stopped and put under the shadow of a roof or tree, or in their absence a blanket or lap robe may be used to cover his eyes. If there is danger of falling remove the harness, and secure a soft piece of ground, free from stones or other hard bodies. Cold water applied to the head will sometimes check. A common practice is to bleed from the palate, and in plethoric cases especially, and in such as are dependent on congestion, tumors or other lesion of the brain it is to be commended. The action will be rendered more prompt and effective if the blood is taken from the jugular. A laxative diet, and carefully regulated work are desirable to obviate the tendency to the affection, and this may often be accomplished by a run at pasture. Otherwise daily small doses of Glauber salts in the feed may suffice. Bromides may be used to calm nervous excitement.

In cases of **gastric vertigo** an active cathartic, followed by smaller laxative doses or a laxative diet and a course of bitters may prove useful. Such cases should never be worked on a full stomach but should be left at rest for at least an hour after a meal.

In **aural vertigo** special attention must be given to the throat, and external ear. Bromides may often be useful, and sometimes benefit may be derived from an occasional blister or light firing back of the ear.

In **cœnurus cerebralis** in sheep the only resort is to trephine and remove the parasite.

CONCUSSION OF THE BRAIN.

Definition. Causes: leaps, trips, falls, blows. Symptoms: fall, insensibility, flaccidity, suspended respiration, tumors, vomiting, recovery, signs of cerebral congestion. Pathology: anæmia followed by congestion. Diagnosis: from fracture, epilepsy. Treatment: quiet, rubbing of limbs, ammonia, cold to head, or heat; for congestion, bromides, depletion, ice pack, derivatives.

Definition. Concussion is the condition produced by mechanical jar or shock of the cerebral mass, and manifested by modification of the brain functions of any grade from a simple dazed condition to that of complete unconsciousness,

Causes. The most familiar cause is the stroke of the butcher's pole axe, producing sudden and absolute insensibility. A horse in leaping, trips and falls on his head or running against a wall sustains a concussion, which leaves him for some seconds without any signs of life. The same will happen to other animals, but above all to rams which in their combats, back for a number of yards and running together meet with a shock from the effect of which even their thick skulls cannot save them. Other blows upon the head operate to the same end.

Symptoms. Concussion is manifested by different grades of symptoms. At first there is usually a fall with complete insensibility. The animal lies flaccid, utterly insensible to external irritation and there is suspension of respiration. The heart continues to beat and a frequent weak pulse may be often detected. In slight cases, breathing may be reestablished at the end of a minute or two, with muscular tremors and movements of the limbs; then the animal rises, shakes his head, neighs, and walks at first unsteadily and afterward with greater and greater firmness. In vomiting animals, emesis occurs.

In some cases this may be followed, after an hour or two, by signs of congestion, heat of the head, redness of the eyes, irritability, or dullness and stupor and perhaps muscular twitching. This may improve or it may terminate in death preceded by spasms, general convulsions, rolling of the eye balls, and stupor or coma with general muscular relaxation.

The primary condition is usually an anæmia of the cerebral

matter as seen in the brain of the animal suddenly killed by the blow of a hammer. The return of consciousness or semi-consciousness is connected with the resumed freedom of the cerebral circulation. The later convulsions, stupor or coma, usually imply active congestion or the effusion of blood on the brain surface, or in its substance.

Diagnosis from fracture must be made mainly by manipulation of the bone in the seat of the blow, and by the absence of the increasing stupor and coma which attend on pressure from a gradually increasing blood clot. From epilepsy it is to be distinguished by the evidence of mechanical injury, by the absence of spasms at the first, by the suspension of breathing and the absence of froth about the lips.

Treatment. Keep the patient still and prostrate until there are signs of returning respiration and free cerebral circulation. This may be hastened, however, by active rubbing of the limbs and body, by giving guarded inhalations of ammonia, or even by friction of the skin with ammonia and oil. Sometimes reaction is favored by dashing cold water on the head, while in other cases hot water to the poll will prove more effective, or the two may be used alternately with good results.

If, after partial recovery, there is marked restlessness, or irritability it may be met with bromides. If secondary unconsciousness supervenes effusion of serum or blood is to be feared, or extreme congestion, and blood may be drawn from the jugular or by cups from the cranium, and ice bags or cold water may be applied to the head. Hot foot baths or mustard embrocations applied to the limbs, and even derivation toward the bowels may be used. The indications for treatment come to be for meningo-encephalitis.

LIGHTNING STROKE. ELECTRIC SHOCK.

Fatal. Non-fatal. Herbivora at pasture under tree. Symptoms: dazed for a few minutes, unconscious for hours, permanent paresis or paralysis. Lesions: lines of burned hair, skin or muscles, rigor mortis slight, decomposition rapid, bluish black venous and capillary congestion, extravasations, blood fluid. Diagnosis. Treatment: ammonia, ether, alcohol, caffein, nerve stimulants.

While a stroke of lightning is usually fatal, yet in certain cases, the victim is but temporarily stunned and recovers with more or less remaining paralysis. The subject has also great importance in connection with the the claim of the owner against a company which may have insured his stock against lightning.

Any animal may be struck, but the herbivora which are turned out to pasture are especially liable to such injuries, because they seek shelter under trees, which operate as lightning rods.

Symptoms. In slight cases of shock whether by lightning or the current of a hanging live electric wire, the subject may be simply dazed and may or may not fall to the ground, and recover itself in a very few minutes. In other cases there is a more violent shock which prostrates the animal to the earth, where it may lie unconscious for some hours and yet quickly and completely recover. In still other cases after such prostration recovery is incomplete and the animal remains affected with paresis or paralysis of one or more, commonly of both hind, or all four limbs. In the more violent shocks death is instantaneous.

Often the impact and course of the current are marked by visible lesions. Sometimes the skin is wounded exposing a bluish black tissue beneath. More commonly there is an area of burnt hair, or straight, radiating or angular lines of raised and frizzled hair marking the course of the current. In a horse killed by an electric light wire in Ithaca recently the current had burned to a depth of several inches in the muscles of the shoulder which rested on the wire.

Lesions are often rather indefinite. There may be no appreciable change in the nervous system. Rigor mortis is slight; it passes off rapidly and decomposition sets in early. The venous system and capillaries are usually filled with liquid blood of a

dark bluish black color, and at intervals are points, spots and patches of blood extravasation. The uniformly liquid state of the blood is one of the most marked phenomena of death from electricity. The dark blue congestion of the radical veins is also very pathognomonic, the part struck or traversed by the main current, being the seat of the most elaborate arborescent network. This arborescent appearance of the dark colored veins, and the petechiæ are often marked in the internal organs (brain, kidneys, liver, lungs).

Diagnosis. The environment of the animal will often clear the diagnosis. The patient is found helpless, or dead under a tree, by a pole, or under a hanging wire, and if a tree there are evidences of the electric shock in scattered leaves and branches, stripping off of the bark, or perhaps rending of the tree in pieces. In case of wires attached to or passing near such a tree, the supporting poles show similar splitting and rending. Add to these the fluidity of blood in the carcase, the thickly ramifying network of the minute dark bluish, red veins, the petechiæ and the comparative absence of cadaveric rigidity, and we have a picture very significant of lightning stroke.

Treatment in such cases is according to the condition. The primary unconsciousness is met by inhalations of ammonia or ether, or the injection of brandy or alcohol subcutem. Caffein, atropine or hyoscyamin may be used as substitutes. If consciousness returns recovery is usually rapid and complete. Should paresis or paralysis remain it must be treated like any ordinary case of these affections.

INTRACRANIAL HÆMORRHAGE AND THROMBOSIS. APOPLEXY. SOFTENING OF THE BRAIN.

Definition. Causes: Nature: intracranial rupture, with pressure, serous effusion, excessive congestion, experimental cases, anæmia from pressure, comparative immunity of horse, heart disease, Bright's disease, atheroma, degeneration, emboli, age, blood tension, severe exertion, excitement, concussion, insolation, venous obstruction, toxins, neoplasms. Lesions: blood clots, small and multiple, large and solitary, brain absorption, cavities, cysts. Symptoms: dullness, swaying, trembling, elevation of head, turning in circle, sudden fall, spasms, unequal dilated or contracted pupils, eyes turned to affected side, congested or anæmic mucosæ, stertor, puffing cheeks except in solipeds, pulse slow, soft, full, vomiting, stupor, coma, unconsciousness, paralysis, monoplegia, hemiplegia, sequelæ. Diagnosis: sudden unconsciousness, with little spasm, but paralysis, history, sign of trauma, deep coma, eyes turned to one side, pupils unequal, stertor, slow breathing and pulse; from uræmia, pulmonary apoplexy, œdema or anthrax. Treatment: bleeding, ice pack, snow, cold water, rest, derivatives to limbs, later purge, bromides, potassium iodide, tonics, open air life.

Definition. Cerebral apoplexy has been defined as a sudden loss of sensation and voluntary motion, from pressure originating within the cranium and followed by paralysis, often unilateral. The definition is somewhat insufficient as regards the early symptoms as the same conditions attend on convulsions and epilepsy (haut mal), and it is only by excluding these by their characteristic features of sudden seizure with clonic spasms and their intermittent and paroxysmal habit that we reach an easy and satisfactory distinction. Later the paralysis tends to identify the apoplectic attack.

Causes and Nature. The immediate cause and essential lesion of apoplexy has been generally held to be the rupture of an intracranial artery and the formation of a considerable blood clot which presses upon (and abolishes the functions of) the brain. There are cases, however, in which the characteristic symptoms are present, and yet a complete recovery ensues at an early date, too early to allow for the absorption of a considerable clot. Moreover, in fatal cases perhaps no blood clot is to be found, but in place a serous effusion, or an internal congestion which exercised the fatal pressure on the brain. So far, therefore, as clinical phe-

nomena are concerned, we must allow that apoplexy may arise from any sudden pressure on the brain substance. Pagenstecher produced the symptoms of the disease by injecting, at a regulated pressure, melted wax and tallow between the skull and dura mater in the dog. In the moderate cases there were drowsiness, psychic depression and general muscular weakness. In the more severe ones there were added sleep and unilateral paralysis. In the more extreme cases death followed in a few hours after coma set in, though in some of these a partial recovery ensued if the waxy mass was scooped out before the fatal symptoms appeared. Cases ended fatally only when the injection pressure equalled that of the blood, and convulsions occurred only when the pressure was unsteady. The temperature fell as it does in apoplexy in man, at the outset, but it continued falling to the fatal issue contrary to what takes place in man.

Duret injected water into the cranium of animals so as to produce great tension of the occipito-atloid membrane causing thereby arrest of the respiration and slowing of the heart's action. On tearing the membrane so as to allow escape of the water, respiration began anew and consciousness was gradually restored.

Edes sustains the view that apoplexy is directly due to anæmia of a lesser or greater portion of the brain substance, and that this need not be in any one particular seat nor of any definite extent. This anæmia is usually induced by pressure and may be caused by effused blood, or serum, or by the extreme congestion due to narcotic poisons, or other cause. Embolism of a cerebral vessel, however, by cutting off the blood from the part of the brain which it supplies may give rise to the apoplectic phenomena.

Friedberger and Fröhner found apoplexy quite frequent in sheep, ox, and dog, and rare in the horse, although more subject to the violent exertion which they put in the front of all causes. It is probable that the sluggish, pampered life of the first three animals, and the tendency to fatty degenerations and heart disease introduces a special predisposition as it does in man, while the horse, inured to an open air life and a vigorous muscular condition, is comparatively immune. Bright's disease is a common cause in the human subject, with its resulting cardiac hypertrophy. The degenerations attendant on these conditions and especially fatty change (atheroma) in the walls of the cerebral arteries,

pave the way for their rupture and for blood effusion. Emboli also carried from the diseased heart not only cut off the blood from the parts supplied by the plugged arteries, but increase the blood tension on the cardiac side of the obstruction and endanger rupture at any weak part. Thus they may cause apoplexy from anæmia without rupture or apoplexy from the pressure of effused blood.

Age which is such a notorious factor in man is not without its influence in the lower animals. It is in the old that we mostly see disease of kidneys and heart and the degenerations of the tissues, including the brain and its vessels; in these, therefore, rupture and extravasation are the most frequent.

The other causes are mostly connected with increased blood tension with or without a debility of the vascular walls. Violent exertions as in racing, coursing, dragging heavy loads up hill or on heavy ground, severe excitement, cerebral concussion, insolation, and intense congestion of the brain substance have all been recognized as causative factors. The compression of the jugulars by a small collar, the violent straining attendant on parturition, or constipation, and even the retrocession of blood from the surface when exposed to extreme cold, may contribute to the final rupture.

In infectious diseases in which the toxic products tend to produce profound modifications in the blood and tissues, extravasations are met with in the brain as in other organs. Thus they are seen in anthrax, Texas fever, petechial fever, etc.

Then the formation of **neoplasms** in the brain may be the occasion of the rupture of the vascular walls and apoplexy. **Hæmatoma** of the dura in the dog (Friedberger and Frölmer), **cholesteatomata** in the horse, and **carcinoma** may be apparent causes.

The effect of **mechanical injury** must be admitted, as blows on the head, injuries from an ox yoke, and concussions during the battles of rams and bulls.

Lesions. Blood extravasations may be found at any part of the brain: a. into the the brain substance; b. into the ventricles; c. from the pia mater; d. into the arachnoid sac; e. between the skull and dura mater. It is especially common in connection with the ganglia adjoining the ventricles; the corpus striatum,

optic thalamus, the corpora quadrigemini, the fornix. In other cases the crus cerebri, pons, medulla oblongata, corpus calosum. In other cases the convolutions of the cerebrum or cerebellum suffer. The amount of effusion may be limited to a few drops or it may cover an extensive area and cause considerable flattening of the brain substance.

When capillary hæmorrhages are present—the size of a millet seed or a pea—Friedberger and Fröhner have usually found them multiple, but when large enough to form distinct clots they are usually single and confined to one side. If a clot, involving the brain substance, is small, it merely separates the nervous fibres, but if larger, the cerebral tissue is broken down in the mass of clot, discolored, torn and softened. If the patient has survived the first attack the clot passes through the different stages of discoloration, brown, brownish yellow, yellow, and may become fibrous forming a distinct cicatrix, with loss of brain substance. In connection with the partial absorption of the effused blood, cavaties may be filled with a serous fluid (apoplectic cysts), and these may show multiple loculi. The nerve fibres which lead to an old standing lesion are usually degenerated.

When effused into a ventricle, blood is less readily absorbed and tends to remain as a flattened discolored layer.

Extravasation between the dura mater and the cranium is probably always the result of direct mechanical violence.

Symptoms. Premonitory indications of apoplexy are less commonly recognized in the lower animals than in man, doubtless largely because of the impossibility of appreciating subjective symptoms. The first observed indications are usually dullness, some lack of coördination of movement, swaying, unsteady gait, trembling and a tendency to deviate to one side or to move in a circle. In the majority of cases, however, the first symptoms noticed are a complete loss of consciousness or nearly so, a sudden fall and often more or less convulsive movements of the limbs aggravated by any excitement. The eyes remain dilated, the pupils enlarged or sometimes contracted, and in case of unilateral effusion the axis of vision of both eyes is turned to the affected side, right or left. The pupil of one eye is likely to be more widely dilated than that of the other. Roll-

ing of the eye-balls is not uncommon. Convulsions may occur, the head and hind limbs being drawn back forcibly as in opposthotonos, or the animal may lie flaccid and comatose from the first. The nasal, buccal and orbital mucous membranes are usually congested, deep red or livid, yet sometimes they are anæmic and pale (Shock). The breathing is usually characteristic, being deep, slow, labored, irregular and stertorous and accompanied by puffing out of the cheeks at each expiration (except in solipeds). Yet there are cases in which stertor is absent. The pulse is usually slow, full and soft, and, in the carotids, throbbing, but it may be weak and imperceptible. There may be complete unconsciousness, and again from the first, or nearly so, there may be a slight response to a stimulus, which cannot be referred altogether to reflex action. In vomiting animals, emesis may ensue. Stupor and coma are more or less marked, though liable to intermissions under any cause of irritation.

Along with the above symptoms the spasms and sequent paralysis, are significant. If confined to given muscles or groups of muscles (monoplegia) it usually implies pressure on some special cortical convolutions presiding over these muscles, and convulsions are to be expected. If there is hemiplegia it is suggestive of implication of the medulla or pons on the opposite side, or of a clot on the corpus striatum or extensively on one side of the cerebrum. A clot in the lateral ventricle tends to profound coma. So liable, however, is pressure to be extended from one side of the brain to the other, and irritation on the one side to rouse a corresponding condition on the opposite side, or in related ganglia, that deductions of this kind cannot always be implicitly relied on.

Though an animal should recover from an attack there is liable to remain some modification of the nervous functions, partial anæsthesia, circumscribed paresis, dullness, lack of energy, irritability, or muscular atrophy.

Cerebral embolism and thrombosis and their sequelæ, infarction and softening, give rise to corresponding symptoms, according to the seat of the lesion, and like lesions of the blood vessels predispose to subsequent attacks.

Diagnosis is based largely on the appearance, usually sudden

but sometimes slow, of a more or less profound unconsciousness, attended or followed by paralytic troubles. The history of the case may assist, any blow on the head, or sustained by falling, striking a wall or post, or wearing a yoke, is to be noted. Any extraordinary exertion or excitement must be considered. Any sign of injury about the head; the congestion of the cephalic mucous membranes in contrast with the pallor of *shock*; the onset of the attack without convulsions (or with them as in epilepsy); the deep coma indicating cerebral hæmorrhage or narcotic poisoning; the absence of the odor of alcohol, opium, or other narcotic from the breath; the turning of the eyes to one side and the inequality of the pupils on the two sides; the turning of the head to the same side as the eyes; the slow, labored, usually stertorous breathing; the slow, full, soft pulse; the occasionally rigid condition of the muscles and finally the paralysis, hemiplegic, and less frequently monoplegic or paraplegic, make up the diagnostic picture.

Uræmia and *diabetic coma* may be excluded by examination of the urine, *pulmonary apoplexy* or *œdema* by the predominance of respiratory troubles, and fulminant *anthrax* by the examination of the blood and by the fact that this disease does not prevail in the locality

Treatment is very unsatisfactory in the lower animals, as the disease is very fatal, and unless recoveries are complete, they are not pecuniarily desirable. It is only in the slighter cases, therefore, that treatment can be recommended. At the very outset nothing is better than a full bleeding in a large stream from the jugular vein or temporal artery. Ice, snow, or cold water should meanwhile be applied to the cranial region. Absolute rest should be given, any harness that would impede circulation or respiration removed, and hot water or stimulating embrocations applied to the limbs.

When consciousness returns and the patient can swallow, an active purgative may be administered, or barium chloride or eserine may be given subcutem. Any recurring heat of the head may be met by renewal of cold applications, and the force of the circulation may be kept in check by small doses of bromides or aconite. In case of the formation of a clot, iodide of potassium and other alkaline agents may be resorted to. Quiet and the

avoidance of all excitement together with a laxative non-stimulating diet must be secured throughout. A course of vegetable or mineral tonics and an occasional blister to the side of the neck may prove a useful sequel.

CEREBRAL HYPERÆMIA.

MENINGO—ENCEPHALIC CONGESTION.

Passive and active hyperæmia. Causes: *passive:* obstacles to return of blood: anæmia: *active:* brain excitement, sunstroke, violent exertion, fear, abdominal tympany, ptomaines, narcotics, lead, darnel, millet, leguminous seeds partly ripened, tumors, parasites. Symptoms: *horse:* variable, vertigo, stupor, convulsions, apoplexy, irritability, disorderly movements, strong, hard pulse, congested mucosæ, heat of head, dulness, drowsiness, lethargy, coma, alternating periods of violence, aggravated by what tends to increase vascularity of brain, congested optic disc: *cattle:* parallel, with special heat of horns: *dogs:* similar, with desire to move, or wander, or has nausea, howls, snaps. Treatment: cold to head, derivation to limbs and bowels, chloral, bromides, ergot, bleeding, darkness, coolness, nonstimulating food.

Congestion of the encephalon is treated here as a pathological entity, though it cannot always be distinguished clinically from some forms of vertigo on the one hand and from the milder types of apoplexy or encephalitis on the other. It has been divided into *passive* or *venous hyperæmia* and *active* or *arterial hyperæmia*.

Passive hyperæmia, as shown under vertigo and apoplexy is a common result of a tight collar, a tight strap used for cribbiting, a too short bearing rein, dilation or valvular disease of the right heart, or disease of the lungs, violent efforts in running, draught, etc. It tends to be associated with arterial anæmia on the principle that the closed cranial cavity can only admit a certain amount of blood and if an excess accumulates in the veins and capillaries, this must be compensated first by the movement backward to the spinal canal of the cerebro-spinal fluid, and second by the diminution of the blood in the cerebral arteries.

Active hyperæmia may be brought about by any excitement which especially affects the brain. This has been already noted in connection with insolation (sunstroke). It may result from

severe exertion during hot weather, in a violently contested race, in drawing a heavy load up hill, or in harsh training. Violent exertion just after a meal is especially injurious. Also the excitement of travelling by rail, or that caused by proximity to locomotives, to discharges of firearms and to other causes of great fear; encreased blood tension in the cerebral vessels in connection with hypertrophy of the left ventricle, or obstruction in other vessels (of the limbs) so as to direct the force of the current into the carotids, the expulsion of blood from the splanchnic cavities by gastric or intestinal tympany, or overloading of the paunch, and irritation of the brain by ptomaines and toxins in certain infectious diseases (rabies, canine distemper, etc.) In the same way vegetable narcotics (opium, etc.) produce congestion. Among the most common causes of congestion are lead, poisoning by lolium temulentum, partially ripened lolium perenne, millet, Hungarian grass, and partially ripened seeds of the leguminosæ (chick vetch, vicia sativa.) Other causes are the presence of tumors (cholesteatoma) and parasites (cœnurus, cysticercus) in the brain.

Symptoms. Cerebral hyperæmia, like other brain disorders may give rise to a great variety of symptoms, according to the condition of the animal and the susceptibility of its nerve centres. Some cases have the characteristic seizures of vertigo, others the manifestations of heat stroke, and others, epileptic explosions or apoplectic symptoms. For these see under their respective headings. In other cases the symptoms are those of encephalomeningitis but moderate in its type and often tending to a transient duration, or to prompt resolution and recovery.

Horse. There is manifest change of the nervous and intellectual conditions, which may show itself by irritability or restlessness, by pushing against the wall, by hanging back on the halter, by trembling, shaking the head, neighing, pawing and, in exceptional cases, by rearing, biting or kicking. The pulse is hard and full, the heart's impulse strong, the beats in the carotids and temporal arteries being especially forcible, and the buccal, nasal and orbital mucosæ are strongly congested. Heat of the head is usually a marked feature. While usually very sensitive to touch, noise or light, the animal may be dull or drowsy, and in spite of its marked sensitiveness, it is then inert or lethargic and indis-

posed to any active exertion. Freidberger and Fröhner say that the habitual comatose condition alternates at intervals with periods of violent excitement during which the animal pushes or dashes against the wall, grinds the teeth, rears, paws, kicks, bites, etc., and then relapses into the state of coma. When the disease reaches this stage it may be questioned whether we are not dealing rather with acute encephalitis.

In active congestion the symptoms are always aggravated by whatever tends to increase the vascular tension in the brain. Active exertion, draught, the pendent position of the head, the recumbent position on the side with the head as low as the body or lower, aggravate all the phenomena and render the animal more helpless.

The following table slightly modified from Spitzka serves to point out the distinctions between anæmia and hyperæmia :

Symptoms.	In Cerebral Anæmia.	In Cerebral Hyperæmia.
Pupils.	Usually dilated and mobile.	Usually small or medium.
Respiration.	Often interrupted by a deep breath or sigh, even when at rest.	Normal or nearly so.
Activity.	Lassitude.	Restless, but indisposed to exertion.
Temperament.	Lethargic with exceptions.	Irritable with exceptions.
Intelligence.	Senses impaired.	Impaired.
Elevation of head.	Aggravates symptoms.	No effect, or improvement.
Recumbent, dependent head.	Amelioration.	Aggravation.
Straining.	Not necessarily aggravated.	Aggravated.

Cattle show the same general congestion and heat of the head, ears and horns, congested mucosæ, fixed eyes, and pupils, indisposition to follow the herd, irritability, and dulness with often a disposition to lie down. This may go on to violent bellowing, pushing against the wall, grinding of the teeth, working of the jaws, rolling of the eyeballs, and violent dashing in different directions regardless of obstacles.

Dogs show the same restlessness and excitability, congested head, eyes and nose, frequent movement from place to place, a desire to wander off, and it may be spasms. If there has been any gastric disturbance vomiting usually supervenes. As in the

larger animals the disease may go on to more violent symptoms, and the animal howls, rushes in different directions, and may snap at imaginary objects, or at any one who interferes with him. His movements are liable to be unsteady, uncertain and swaying.

In all cases the ophthalmoscope reveals a congestion of the optic disc.

In the different animals too, acute cerebral hyperæmia tends to merge early into encephalitis with exudation and pressure, attended by stupor, coma, somnolence or profound lethargy.

Treatment. In slight cases of cerebral hyperæmia, it may be sufficient to apply cold to the head with a stimulating fomentation to the limbs, and an active purgative, with chloral or bromides. Ergot in full doses has often an excellent effect.

In the more acute types of the disease, bleeding is the first and most efficient measure. A full abstraction from the jugular will relieve the vascular tension and relieve the circulation on the brain. It has been counselled to avoid this when comatose symptoms have set in, and in some prostrate conditions a large and rapid abstraction of blood may fatally increase the prostration. In other cases, however, the less rapid abstraction will improve at once the intracranial circulation and nutrition, and solicit the reabsorption of the exudate which produces sopor and coma.

A purgative is one of the most efficient derivatives, the determination of an excess of blood to the bowels and of an abundant serous discharge into their interior acting as a valuable depletion, and abstraction of blood from the over-excited brain. At least a half more than the usual dose must be given, and may be supplemented by an injection of glycerine or a hypodermic exhibition of eserine. It is best to avoid too drastic or irritant purgatives as the cerebral congestion may be aggravated by the irritation, as it often is induced in severe indigestions. For the horse, aloes and podophyllin, or for ruminants, omnivora and carnivora castor oil may be resorted to.

The patient must be placed by himself in a dark, cool, well aired building, and when able to resume feeding must receive an easily digested, non-stimulating diet; for horses or cattle gruels, wheat bran mashes, pulped roots, or green food; for dogs and pigs, gruels, mush or milk.

Any sequent paralysis must be treated on general principles.

MENINGO—ENCEPHALITIS. STAGGERS.

Divisions. Causes: traumas, faulty diet, highly nitrogenous, leguminous seeds, undergoing ripening, cotton seed, gluten meal, forced feeding, buckwheat, ryegrass, lupins, cryptogams, trefoil, equisetum, narcotics, microbian ferments, experiments with spoiled food and epizootics in wet years, high temperature, violent exercise, railroad travel, climatic change, complex causes, embolisms, infections, lead, phosphorus, tumors, parasites. Symptoms: with *meningitis*, fever, hyperæsthesia, active delirium and convulsions predominate: with *encephalitis*, dullness, stupor, somnolence, muscular weakness, anæsthesia, paralysis, coma; usually complex, hyperthermia, periods of benumbing, followed by excitement; drowsy, stupid, semi-closed eyelids, drooping lips, ears, and head, latter resting on manger or wall, walks unsteadily, limbs out of plumb, hangs on halter, wont back, turns in circle, costive, indigestion, tympanies, rumbling, abnormal (often slow) pulse and breathing, congested optic disc; alternate with trembling, excitement, pawing, rearing, plunging, pushing against the wall, trotting motions, etc.: uncontrollable violence; severity and frequency of paroxysms indicate gravity; recovery: sequelæ. Duration: death in 24 to 36 hours: or weeks. Prognosis: one-quarter recover, with increased susceptibility; nervous animals worst. Lesions: extravasation, congestion, exudates, pus, thickened meninges; choroid plexus: brain matter gray or red, puncta, infiltrated, softened, excess of leucocytes, red softening, yellow softening, sclerosis, cicatrix, abscess. Diagnosis: from rabies, cerebral congestion, immobility, influenza. *Cattle.* Symptoms: evidence of trauma, indigestion, lead poisoning, narcotism, parasitism; dullness, stupor, somnolence, stertor, grinding teeth, spasms, twitching, restless movements, blindness, violent actions, bellowing, hebetude, palsy. Relation to causation. *Sheep:* Symptoms. *Swine:* Symptoms. *Dog:* Symptoms. Diagnosis from rabies. Treatment: quiet, darkness, coolness, restraint, ice or cold irrigation, elimination, derivation, depletion, diuretics, potassium iodide, antipyretics, laxative diet, cool water, evacuate abscess. *Cattle*, similar, saline laxatives, for lead sulphuric acid, for cœnurus, operation, for œstrus, benzine. *Dog*, parallel treatment, milk diet or gruels, for linguatula, benzine.

The inflammatory affections in the cranial cavity have been divided primarily into the following:

1. *Meningitis.* Inflammation of the coverings of the brain, and
2. *Encephalitis* (*Cerebritis*). Inflammation of the nervous substance. These are further subdivided into:

A. Pachymeningitis. Inflammation of the dura Mater.

B. Leptomeningitis. Inflammation of the pia Mater.

C. Purulent Meningitis.

D. Serous Meningitis.

E. Tubercular Meningitis.

F. Traumatic Meningitis, etc.

G. Cerebro-Spinal or Infective Meningitis.

H. Acute Meningitis.

I. Chronic Meningitis.

J. Polioencephalitis Corticalis. Inflammation of the brain cortex.

K. Polioencephalitis Superior. Inflammation of convolutions around the Sylvian fissure, palsy of the eyeball.

L. Polioencephalitis Inferior. Inflammation of the Medulla, bulbar palsy.

M. Interstitial Inflammation of the Brain. Resulting often in sclerosis.

In the lower animals, however, where we cannot avail of subjective symptoms, such fine distinctions can rarely be made in diagnosis and except in case of an uncomplicated meningitis, or a circumscribed encephalitis, which affects only a limited group of muscles like those of the eye, arm, or leg, we have to fall back upon a more general diagnosis. Again meningo-encephalitis is more common than the uncomplicated affection of the brain, or the membranes, and therefore, we shall follow Trasbot in dealing with the combined affection, and noting incidentally the distinctions that can be made in the more purely limited affections.

Causes. **Mechanical Injuries.** Pachymeningitis occasionally results from blows or other injuries upon the head, especially in stallions and vicious horses struck with a heavy whip or club, cattle and sheep injured in fighting, and oxen hurt by the yoke. These injuries may also affect the brain as in concussion, or by the extension of the disease into the nervous tissue. In the cranium of a stallion in the New York State Veterinary College Museum the whole of the meninges are greatly thickened by a traumatic meningitis of old date and the subjacent cerebral convolutions of the right hemisphere are deeply encroached on, flattened and absorbed over an area of 1¾ inches in the longest diameter.

Diet. Among the most common causes of encephalitis in horses is an injudicious dietary. Overfeeding with grain, but

especially with grain and seeds that are rich in albuminoids deserve the first mention. The various leguminous seeds, peas, beans, tares, vetches, and the ripened leguminous fodders, clover, alfalfa, and sainfoin, are especially to be incriminated. These are usually most dangerous when in the stage of advanced ripening and yet not fully matured, evidently indicating the development of narcotic poison at this stage. Such poisons are found habitually in certain species, like the chick vetch (vicia cicera) which produces paralysis when fed to the extent of more than one-twelfth part of the ration. This danger is not, however, confined to the leguminosæ; an over abundant ration of cottonseed meal has a similar effect, and indeed this rich alimentary product has been practically discarded from pig feeding, and largely as the main constituent from the ration of dairy cows. Gluten meal, another product rich in proteids, is attended by similar dangers. But it is not alone the seeds that are rich in nitrogen that are to be dreaded, forced feeding even on the carbonaceous maize induces disorder of the digestion and brain, especially in dairy cows. Buckwheat, also, and indeed all the heating carbonaceous grains tend to similar disorders, and are especially injurious in internal ophthalmia (recurring ophthalmia) which is so closely related to brain congestion. With sound judgment and in well balanced rations, all such agents can be fed to advantage; it is only when fed exclusively or to excess as the heavy ration that they are to be feared.

Narcotics. Next must be noted those alimentary matters which are hurtful by reason of narcotic constituents. At the head of this list may be placed the lolium temulentum or intoxicating ryegrass. Like the vicia sativa or cicera, the seeds of this are always poisonous, hence its significant name. Then the other ryegrasses, perennial and annual (Italian), though perfectly safe in ordinary circumstances, develop at the period of ripening a narcotic principle, which produces cerebral congestion or inflammation in whole stables of horses at a time. The lolium temulentum is poisonous to man and animals alike. Baillet and Filhol obtained from the seeds an etherial extract containing a bland oil to the amount of two-fifths and a yellow extract to the extent of three-fifths. The amount of this extract derived from three ounces of the seeds often developed the most violent

symptoms in the dog, while that furnished by six pounds of the seeds proved fatal to the horse. Pigs and cattle seemed to be unaffected by the agent when given by the month. Sheep suffered more but required large doses. Ducks and chickens were practically immune, being affected only by very large doses. Rabbits were not poisoned by the yellow etherial extract, but succumbed to a watery extract. Brydon found that lambs suffered extensively from eating the heads of the ryegrass.

Lupins on certain lands produces an icteric disorder accompanied by cerebral symptoms but the result is not the same under all conditions and it has been suspected that the symptoms were caused by cryptogams and their products. The same remark applies to the brain symptoms sometimes produced by trefoil, equisetum and other plants.

A great number of **narcotic** and **narcotico-acrid** plants produce nervous symptoms indicating cerebral congestion or inflammation such as ranunculus, wild poppy, digitalis, fennel, œnanthe crocata, hellebore, veratrum, conium, yew, tobacco, box, aconite, cicuta virosa, even buckwheat at the time of flowering, vetch and flax.

Fodders affected with **cryptogams** or **bacterial ferments** are undoubtedly at times the cause of encephalitis. Veterinary records furnish many instances of wide spread attacks of stomach staggers, abdominal vertigo, and cerebro-spinal meningitis in wet seasons, when the fodders have been harvested in poor condition, or when from inundation or accidental exposure they have become permeated by cryptogams and microbes. Among comparatively recent accounts of this are those of Martin and Varnell (musty oats), Lombroso, Depre, Erbe, Pellizi, and Tireli (smuts), Bouley and Barthelemy (musty fodder), and Ray (fermented potatoes). One of the most extended local outbreaks of cerebro-spinal congestion I have seen, occurred in the pit mules of the Wilkesbarre coal mines, while fed on Canadian hay which had been soaked with rain in transit and had undergone extensive fermentation. It should be noted that there were the attendant factors of overwork, in anticipation of a strike, and a Sunday's holiday above ground in a bright summer sunshine.

The experimental administration of moulds, smuts and mi-

crobes, have in the great majority of cases led to little or no evil result (Gamgee, Mayo, Dinwiddie, etc.) and there is a strong tendency to discredit the pathogenic action of these agents in reported outbreaks. The safer conclusion perhaps would be, to recognize the fact that they are not equally pathogenic under all conditions of their growth and administration. The oft-recurring epizootics of brain disease in connection with wide spread spoiling of the fodders in remote and recent times, probably imply that cryptogams or microbes and their products, plus some condition not yet fully understood are efficient concurrent factors. If we can discover this as yet unknown factor and demonstrate that it operates with equal power in the absence of the cryptogams and ferments, as in their presence, it will be logical to pronounce these latter as nonpathogenic under all circumstances. Until then cryptogams and bacteria must be held as probable factors.

A continuance of **high temperature** is an undoubted factor and becomes more potent, if conjoined with a close, damp, ill-aired stable.

Violent exertion especially in hot weather produces active congestion of the brain and occasionally merges into meningo-encephalitis. If the animal has been for sometime confined to the stable on rich aliment the condition is aggravated.

Railroad travel is another recognized cause.

Any considerable **change of the conditions of life** may operate in the same way. A sale and transport to a distance with change of feed, water, work, stabling and even of climate is at times a potent factor. Prietsch has seen a horse attacked three times in a single year, and on each occasion after a change of ownership and locality. Trasbot quotes an Algerian veterinarian to the effect that many of the percheron horses imported into the Mitidja are attacked by encephalo-meningitis during the extreme heats of summer.

A careful observation of cases will however show that in the majority of cases an attack comes not from one individual factor alone but from a concurrence of several operating together.

Other cases are caused by **embolisms** and **infections** from diseases localized in other parts of the body. Thus we have cerebral abscess in pyæmia, strangles and omphalitis, and cerebral

congestions and inflammation in canine distemper, equine contagious pneumonia, laminitis, and angina.

Among mineral poisons, **lead** is notorious as a cause of acute cerebral disorder often leading to inflammation. Other mineral poisons like **arsenic** and **phosphorus** may lead to encephalitis symptomatic of gastro intestinal irritation, or caused by the toxic products of indigestion.

Rapidly growing **tumors**, like cholesteatomata, are liable to induce recurrent attacks of encephalitis in connection with periodic irritation.

Finally **parasites** in the cranium are sufficient causes of attacks. In the New York State Veterinary College Museum is the brain of a cat with a nematoid wound round the hypophysis. In equine subjects suffering from the strongylus armatus the larval worm or clots caused by its presence in other arteries sometimes invade the encephalic blood vessels causing disturbances of the circulation, embolism, inflammation or degeneration. (Albrecht, Von Heill). The larvæ of the œstrus has also been found in the brain substance producing inflammatory or degenerative foci (Brückmüller, Megnin, Siedamgrotzky). Their presence in the nasal sinuses at times cause encephalitis by contignity. The cestoid worms, cœnurus in sheep and other ruminants, and cysticercus in swine, find their natural larval habitat in the brain and by their movements produce more or less congestion and inflammation. Cases of cœnurus in the horse have been described by Rousset, Frenzel, Zundel, and Schwanefeldt.

Symptoms. The symptoms of uncomplicated meningitis on the one hand and encephalitis on the other are rarely seen, the disease usually implicating more or less both brain and meninges, in a common inflammation or the symptoms of the one involving those of the other through proximity or interdependence of function. And yet in traumatic lesions of the cranial walls, the symptoms may be those of pure meningitis, and in thrombosis, embolism or parasitism of the brain, and in certain tumors they may be those of simple encephalitis. The distinction consists largely in the predominance of fever, hyperæsthesia, active delirium and convulsions in *meningitis*, and especially in its earlier stages; and the prominence of dullness, stupor, somnolence, muscular weakness, paralysis, anæsthesia, coma, and the clouding of special senses, with much less pronounced febrile reaction, or vascular excitement in *encephalitis*.

There is usually, however, a mixing of symptoms so that the benumbing or paralysis of the nervous functions alternates with periods of their exaltation, and with both conditions hyperthermia exists, though usually higher with meningitis.

The manifestations of benumbing or paresis may be continuous or interrupted, and are exhibited in stupor, coma, somnolence, lethargy, paresis or paralysis. The manifestations of excitement are not continuous but occur in paroxysms or at least exacerbations, which may show in visual or mental illusions, active, violent delirium, trembling, rigors, clonic or tonic spasms. The onset is usually abrupt, the animal passing in a few hours from apparent health, to pronounced nervous disorder. The horse seems drowsy and stupid, standing with semi-closed eyes, often drooping lower lip and ears, head pendent and resting in the manger or against the wall in front, the back arched and the limbs drawn together. When moved, it walks unsteadily and often the limbs are left out of plumb, one extending unduly forward, backward or to one side, and often crossing over its fellow. Some cannot be made to back, others back spontaneously hanging on the halter. Turning short in a circle is difficult or impossible and tends to throw the patient down. Yet some exceptional cases will turn around spontaneously to the right or left, and an animal tied to a post goes around it at the end of its halter in its effort to pass straight forward. The circling movement may be due to the irritation on the one side of the brain or to irritation of particular ganglia and nervous tracts as noticed under cerebral hyperæmia.

Appetite is usually lost, or, more properly, the animal no longer takes notice of surrounding things, not even of its food. In some cases, however, in which stupor or coma is not extreme the animal will eat a little during his quiescent intervals. In ryegrass and other dietetic poisoning, the animal may still eat and fall asleep with the mouth full. The digestion is impaired or suspended, the bowels costive, and fermentations with tympanies and rumbling are frequent complications. When originating from poisonous food this often contributes to these abdominal complications.

Respirations in the comatose condition are deep and slow, sometimes not more than four or five per minute. The heart

usually beats strongly, often tumultuously, and the pulse varies greatly—infrequent or frequent, strong or weak, full or small. With cerebritis it is often abnormally slow.

Hyperthermia is always present to a greater or less extent, being often more marked in the more violent forms or those in which meningitis appears to predominate than in the purely cerebral forms. The temperature may vary from 101° to 106°.

The optic disc is congested.

Probably in all cases or nearly all there is a preliminary stage of excitement, in which the eye is clear, the eyelids open, the aspect alert and the whole skin affected by a marked hyperæsthesia. In some cases the symptoms of excitement are much more violent at the ontset of the disease, as marked by trembling, nervous movements, pawing, pushing the head against the wall while the motions of walking or trotting are performed by the limbs, or those of plunging forward, rearing up, drawing back on the halter, etc.

But even when the disease seems to have started with stupor and coma, these paroxysms of excitement almost invariably appear at intervals as it advances. Some, however, plunged in stupor or coma at the first, remain in this condition until they end in paralysis or death, or start in convalescence.

During one of the paroxysms the trembling animal may push his head against the wall as if pulling a heavy load; at other times he will plunge with his feet in the manger and recoiling, fall to the ground, where he struggles violently in an apparent effort to rise; others rear up, pulling on the halter or breaking it and falling back over; some pull back on the halter and throw themselves down; some grind the teeth, or seize the manger, or strike blindly with the fore limbs. When seized out of doors the horse may be quite uncontrollable and refuse to return to the stable even when led by two men with double halters. In all such cases the eye has a fixed, glaring aspect which is the more pronounced when the pupils are dilated, the conjunctiva is deeply congested, of a deep, brownish red with a tinge of yellow. This is usually greatly enhanced by the bruises and extravasations caused by pushing or knocking the head against the wall. The same violence may lead to serious bruises and injuries elsewhere, even fractures of the orbital process or zigoma, of the

ilium or ischium, of the poll or the base of the brain; also of the incisor teeth.

These paroxysms may be so frequent that they seem to be subject to remissions only, and not separated by complete intermissions. During the paroxysms breathing and pulsations are both greatly accelerated.

The gravity of the attack may be judged in part by the violence and frequency of the paroxysms. Yet some cases, marked by profound coma from the first, prove the most rapidly fatal, and the paroxysms of excitement and violence are not incompatible with recovery. Improvement may usually be recognized by the increased length of the intervals between the paroxysms, and by the shortening and moderation of the periods of excitement. After the paroxysms have ceased the drowsiness or stupor gradually disappears, and the hyperthermia subsides.

Even after recovery from the acute or violent symptoms there is liable to remain some aberration or perversion of function, due to the persistence of some encephalic or meningeal lesion. The general hebetude known as *immobility* may bespeak dropsy of the ventricles, pressure of a tumor or clot, or degeneration of ganglionic centres. Diseases of the eyes (amaurosis, glaucoma, cataract), or of the ear (deafness, disease of the internal or middle ear) are less frequent results.

The supervention of general or facial paralysis or of hemiplegia during the active progress of the malady, is an extremely unfavorable symptom.

Duration. A fatal result may take place at any time by self inflicted injuries (dashing the head against a wall, or falling backward and striking the head on a solid body). Apart from this, death may come within twenty-four or thirty-six hours. If the animal survives two to seven days recovery is more probable. Hering records a case of recovery after five weeks illness. Hot weather hastens a fatal result, while cool, cloudy weather is favorable.

Prognosis. Under rational treatment about one-fourth recover. One-half of the victims make a partial recovery but remain in a condition of dementia or hebetude, blindness, deafness, local or general paralysis which renders them more or less useless. Not more than one-fifth or at most one-fourth of all cases recover.

Even in these there is left an increased predisposition to recurrence. It is noted by Trasbot that the mortality is higher in highbred, nervous, irritable animals, which show a tendency to greater frequency, force and duration of the paroxysms of excitement. When decubitus is constant, death may take place from septic poisoning starting from bed sores, and gangrenous sloughing. In other cases there is fatal starvation from inability to eat.

Lesions. In **pachymeningitis** due to mechanical injury there is usually cutaneous and subcutaneous, blood extravasation, and there may be fracture of the cranial bones. The dura mater is dark red, hyperæmic, thickened, covered with exudation and small blood clots mixed with pus cells, and has contracted strong adhesions to the cranial bone. Bony spicula may project into the fibrous neoplasm.

Leptomeningitis usually coexists from extension of the inflammation into the adjacent arachnoid and pia mater. There is then a reddish serous effusion into the arachnoid and beneath it, and the substance of both membranes is thickened by exudate, and discolored by congestion and minute hæmorrhages. Whenever the pia mater is thus inflamed, the superficial layer of the brain is implicated, œdematous, soft and doughy. The extension is also made into the ventricles and a serous effusion takes place often to two, three or more times the normal amount (82 grammes Schütz). The choroid plexus forms a yellowish gelatinoid mass, and the ganglia (corpora striata, optic thalamus, etc.), are flattened.

In **encephalitis** the affected superficial gray matter of the ganglia or convolutions, is deepened in color, usually in limited areas corresponding to the disease of the meninges. Sometimes the color becomes of a distinctly reddish tinge, and when cut into shows unusually prominent red points where the capillaries have been cut. Somewhat larger areas of blood staining indicate hæmorrhagic extravasations. The nervous substance is more or less infiltrated with liquid and softened. The nerve cells are swollen, and in process of granular degeneration and the same is true of the myelin, while the axis cylinder is uneven in its outline. Apart from the numerous minute petechial hæmorrhages there is an abundant migration of leucocytes which are found scattered in the degenerating and softened nervous tissues.

The softening of the nervous tissue may result in a pulpy material, which in the comparative absence of blood is grayish (**gray softening**), if abundantly infiltrated with blood is red (**red softening**), if older and discolored is yellow, as in an old extravasation, (**yellow softening**), if thick and viscous is **gelatinoid softening**. If the exudate becomes organized into fibrous material it is a **connective tissue sclerosis** or a **cicatrix**. If the softening exudate becomes purulent it constitutes a **cerebral abscess**. Cerebral abscess is especially common as secondary abscess in strangles or contagious rhino-adenitis in the horse, but may occur as the result of the presence of any pyogenic germ.

Diagnosis. While there is a certain similarity to **rabies**, the horse with encephalo-meningitis is distinguished by the absence of the extreme hyperæsthesia and irritability, of the persistent neighing and squealing, of the rapid alterations of the voice, hoarse and shrill, of the hallucinations, as following imaginary objects with the eyes, of the readiness to attack with teeth or heels when in any way disturbed or excited, of the disposition to get violently excited when a dog is brought near, or in the case of a stallion to show generative excitement.

From **cerebral congestion** it is to be distinguished by the greater severity of the paroxysms, or the deeper character of the stupor, but above all by the presence of the hyperthermia and other indications of fever.

Immobility which presents the symptoms of drowsiness, stupor and hebetude, is also unattended by fever, or anorexia, shows a healthy condition of the functions, of respiration, digestion and assimilation and a restful condition when left quiet and still.

The *cerebral excitement* that sometimes appears in **influenza** is really an encephalitis complication, but its specific cause is recognized in the local prevalence of the infectious disorder, and the inflammatory or catarrhal condition of the mucous membranes.

The diagnostic manifestations of *meningitis* and *encephalitis* respectively are given under symptoms.

Symptoms in Cattle. In cattle encephalo-meningitis supervenes on congestion, and sometimes comes on abruptly in connection with traumatic injuries, acute gastric disorder, lead-

poisoning, or narcotism. The cases of cerebral parasitism are usually slow in their onset.

Upon the preliminary dullness and somnolence there supervenes excitement, manifested by loud bellowing, pushing the horns, forehead or teeth against the wall, labored often stertorous breathing, a fixed eye often with dilated pupil giving it a peculiar glaring appearance, movements of the jaws, frothing at the lips, tremors, muscular spasms, twitching, or a restless disposition to move, in a circle, in a straight line or less frequently backward. The patient seems to see nothing and is utterly regardless of obstacles. Sometimes the animal plunges violently into manger or rack, against or through the partition of his box, through fences, into ponds, pits, quarries and other dangerous places that may be accidentally in his way. The paroxysms may be intermitted by intervals of comparative calm, and tend to merge into a condition of dulled sensation, staggering, stupor, hebetude and paralysis. The congested conjunctiva and, when it can be seen, the optic disc will correspond to the cerebral congestion. These cases usually proceed to a fatal issue in a few hours. Some cases, however, make a good recovery after a few days of dullness and prostration. In cases that are connected with lead poisoning, or the toxic action of narcotics in the fodder, the attendant circumstances will assist in the diagnosis. From malignant catarrh implicating the encephalon, it may be distinguished by the absence of the catarrhal inflammation of the conjunctiva, pituita, sinuses, buccal mucous membrane, and genito-urinary passages. Also of the tendency to implication of the hair follicles and the keratogenous tissue of the frontal horns.

In the *Edinburgh Veterinary Review*, Dundas describes a form of alcoholism in cows caused by feeding these animals on "burnt ales" in the vicinity of distilleries. The ale is given by steeping straw in it, and the animals will also drink it freely. They often sleep soundly after such a beverage or give evidence of intoxication. The head is turned singularly to one side and slightly elevated. The pupils are widely dilated, and the eyes have a remarkably wild appearance. On being approached the animals wink rapidly and tremble. There is marked heat of head, horns, and ears. When pressed with the finger in the axilla they fall instantly and when pulled by the head they incline to turn over.

The pulse is 70 to 80 per minute. Delirium and loss of coördination of the muscular movements set in, and in case of survival various forms of chronic brain disease are manifested. In one cow the violent symptoms came on with the near approach of parturition. The post mortem lesions consisted in ramified redness and punctiform blood extravasations in the pia mater and meninges. The brain substance was softened and clots of blood were found in the lateral ventricles. Congestion and extravasations were also found around the cervical myelon. (See Alcoholic Intoxication).

Symptoms in Sheep. The sheep is often drowsy, dull and stupid, lying by itself with head low or laid backward. During the periods of excitement it works the jaws, froths at the mouth, carries the head turned in one direction, upward or lateral, bleats piteously, pushes against the wall, has uncertain, stiff or staggering gait, or convulsions, and finally paralysis. The head is hot, and the eye fixed, congested or sometimes rolled upward or squinted. Symptoms in the goat are nearly the same. The cœnurus disease is more gradual in its onset, and produces periodic paroxysms corresponding to the activity of the heads of the parasite when protruded into the brain substance. It is mainly confined to sheep of one year and under and that are kept where dogs have access.

Symptoms in Swine. Pigs may at first have a period of dullness or restlessness, the latter merging into active delirium. The patient champs his jaws, froths at the mouth and nose, sometimes vomits, squeals, raises himself with fore feet on the wall, walks round and round, or falls and rolls over, has tremors or convulsions.

Symptoms in the Dog. There may be preliminary indications of illness, anxiety, restlessness, irritability and a desire for seclusion. Vomiting may occur. This is liable to merge into prostration, a dullness of the special senses, utter inattention to calls, yet a disposition to resent any interference, a readiness to bite, at least to howl, when handled. Some will constantly howl or moan. The eye is fixed, the pupils dilated, the conjunctiva deep red, the head and roots of the ear are hot. The expression of the face is pinched and drawn, the muscles may twitch, the eyes roll, twitching of the neck or limbs may appear, and even epilep-

tiform attacks. In exceptional cases the symptoms approximate to those of rabies, in the tendency to seek seclusion, to wander off, to bite on any interference, and even to gnaw the bars of the cage or any object within reach. After more or less of such excitement, the period of stupor, coma, paresis, or paralysis comes on, and the animal dies in a state of complete nervous prostration. In cases associated with the lingnatula tænioides the sneezing, nasal discharge and nasal congestion, even in the early stages, betray the true character of the disorder.

The rabiform cases usually lack the intense heat of the head, the deep conjunctival congestion, the depraved appetite, the alteration of the voice, and the mischievous desire to attack without reason which characterize rabies.

Treatment. In all cases of phrenitis, quiet, darkness, and coolness are especially demanded. For the horse a roomy, loose box or a well fenced yard may be secured, and if he can be secured by a halter from a point above the level of the head and in the centre of the box it will obviate the increase of congestion by hanging of the head. The application of cold to the cranium in the form of wet cloths, ice bags or irrigation is always in order, and should be continued so long as heat of the head and other indications of cranial hyperæmia last.

In the *horse* suffering, as is so often the case, from narcotic poisoning an active purgative is one of the first considerations to clear away any remains of the poison from the *prima vie*. An ounce of aloes may be safely given, as there is in this case little danger of superpurgation, and, to secure an even more prompt response, eserine (1½ grain) or barium chloride (½ drachm) may be given subcutem. Or an ounce or two of glycerine by the rectum might be used as a substitute for these last. The action of the purgative proves not only eliminant and therefore antidotal, but it is a most effective derivative from the brain. When the restlessness or excitement is very great we may use acetanilid, trional, sulphonal or some one of the many brain sedatives and antithermics. Sedatives, or anodynes like opium, which tend to increase cerebral congestion are dangerous.

Bleeding from the jugular or temporal artery, has been objected to on the ground that it tends to increase the exudate and therefore the pressure and cerebral anæmia. On the other hand it

often proves of great value in vigorous, muscular and plethoric horses in temporarily lessening the blood-pressure in the brain, and affording the walls of the overcharged capillaries an opportunity to resume a more normal tone and to control that very exudation which is so much dreaded. It is most effective in the early stages when little or no exudation has taken place and may then be pushed to the extent of producing a perceptible softening of the pulse (4, 6 or 8 quarts). Even in the advanced stages when exudation has led to stupor or coma a moderate and carefully guarded bleeding may favor reabsorption of the liquid exudate. In weak and anæmic cases in which general bleeding appears to be contra-indicated the shaving of the cranial surface followed by leeching or wet cupping can be safely resorted to.

Counterirritants like bleeding are denounced and advocated by different practitioners. In cases of extreme hyperæsthesia where excitement and fever would be dangerously increased by their use, they must be discarded, or used only in the modified form of soothing hot fomentations to the extremities. Where there is less sensitiveness mustard poultices or pulp applied on the sides of the neck, or upon the limbs, or even more energetic blisters will be of great service.

After the action of the purgative the bowels may be kept free by calomel in ½ drachm doses twice daily and as much sulphate of soda as may be necessary.

Iodide of potassium (1–2 drs. twice a day) is beneficial as an antithermic a circulatory sedative, an eliminant, and probably at times as an antidote but it cannot be given while calomel is used. Certain it is that it often seems to act well in succession to the purgative, in cases of poisoning by ryegrass and leguminosæ.

When fever runs very high it may sometimes be admissible to give aconite, but the coal tar products are much more prompt and powerful, and may therefore be more hopefully employed for a short time.

In conditions of extreme prostration, stupor, or coma, stimulants are resorted to, but too often with no good effect, the exudation and compression which many times cause such symptoms being rather aggravated than benefited by such agents.

During convalescence a restricted, non-stimulating laxative

diet (bran mashes, gruels, apples, potatoes, carrots) is demanded. Pure cool water should be always accessible.

In other forms of meningo-encephalitis the same general principles should be applied, due attention being paid to the removal of the active cause when that can be discovered.

When indications point unequivocally to abscess, and its seat can be accurately located by a circumscribed paralysis, an operation for its evacuation is fully warranted. Otherwise death or permanent uselessness is almost certain.

In *cattle* and *other ruminants* the same general principles of treatment must be applied. As a cathartic Epsom or Glauber salts are preferred to aloes and may be supplemented by barium chloride or eserine. Croton, sometimes useful, is liable to dangerously increase the gastric irritation in cases in which this is a marked determining factor. When the animal is down, raise the head by bundles of straw, or by a halter tied to a beam overhead. In lead poisoning, sulphuric acid largely diluted may be added to the sulphates so as to precipitate the insoluble sulphate of lead. Potassium iodide is of value to dissolve the lead in the tissues and lead to its elimination. Cases of cœnurus require trephining and extraction; the larvæ of the œstrus should be washed out with tobacco water or destroyed by benzine.

In *dogs* the stomach is usually emptied spontaneously by emesis. A purgative of castor oil, followed by daily doses of calomel may be given, and attention given to the cooling of the head and general system. Antipyrin or acetanilid may be usefully employed. The diet should be restricted to milk or thin, well-boiled gruels.

The linguatula in the nose must be met as are the œstridæ of the sheep, and intestinal worms must be got rid of by active vermifuges.

CHRONIC HYDROCEPHALUS. DROPSY OF THE VENTRICLES. IMMOBILITY.

Horse especially suffers. Enzootic in given Alpine Valleys, along Rhone, in Mississippi Valley and bottom lands. Acclimatizing fever. Old, lymphatic, large heads, narrow foreheads predisposed. Geldings. Causes, heredity, cerebral and meningeal congestion, cranial traumas, venous obstruction, tumors, false membranes, fodder or water poisoning, overwork, insolation, prolonged moist heat, hepatic, gastric, and pulmonary disorders. Symptoms: form of head, stupid expression, irresponsive ears, pendent lips, sluggish movements, crossed legs, slow mastication, dips face in water, intractable by halter or rein, unable to back with rider, or wagon, drags back fore limbs, worst in hot damp weather, in sunshine, or after work, or with full stomach. Paroxysms of excitement. Lesions: excess of arachnoid, subarachnoid or ventricular fluid, atrophy of ganglia and convolutions, ependyma thick, opaque, sclerosis, brain anæmic. Tumors, nature. Experimental cases. Prognosis, incurable, better in cool season. In cattle, sheep, swine and dogs. Treatment: derivatives, nerve stimulants (nux), puncture, pilocarpin, purgatives, Jurisprudence. Notify seller in 9 days (France), 15 (Bavaria), 21 (Wurtenberg, Baden), 28 (Hesse, Prussia), 30 (Austria). Examination by expert.

Dropsy of the ventricles is common in the horse in certain countries and districts, yet even there it is uncommon in cattle, sheep, swine and dogs, save as a congenital affection. It is reported as enzootic in some Alpine valleys and along the Rhone, attacking especially the mares and immature horses so that breeding becomes impossible. Manener who reports this says that in the same localities encephalic diseases are more common in man. In America it appears to be most frequent in the rich bottom lands of the Mississippi valley and of the Southern States. Northern horses taken to the Gulf States though they may not suffer to this extent, are liable in the first year to show weakness, debility, and lack of vigor which is spoken of as the **acclimatizing fever**. Elsewhere the affection is one of the old horse in which the vital powers begin to fail. Common breeds of horses with lymphatic temperament, large head and narrow forehead have been found to be especially predisposed. Geldings are said to be most liable on account of the arrested development of the brain, but with the great preponderance of geldings among work horses, it is dangerous to generalize too far.

Causes. Acute encephalitis may lapse into the chronic form and then assumes the symptoms of this disease. Cases that come on slowly and imperceptibly appear at times to be hereditary, as might be expected from the fact that it usually goes with a lymphatic temperament. The conditions which cause cerebral or meningeal hyperæmia in chronic form conduce to the affection. Injuries to the cranial vault from traumatism or disease are infrequent causes. Renault records a case associated with two bony tumors, each as large as an egg, projecting inward from the frontal bone and which had produced extensive absorption of the convolutions and increase of the cerebro spinal fluid. In a case of my own with an abscess in the diplöe above the frontal sinus, and pressing inward on the brain a similar condition existed. In other cases Renault noticed that the cerebro spinal fluid was largely in excess.

Much more commonly, however, the accummulation of liquid takes place in the ventricles, and is associated with different causes: as tumors or false membranes near the base of the brain pressing on the veins returning blood from the ependyma, tumors in the ventricles (cholesteatomata, etc., of the ependyma or choroid plexus) obstructing the circulation or giving rise to local hyperæmia, and chronic congestions from the other causes such as faulty conditions of fodders, or water, exposure to undue heat, overwork, etc. The enzootic prevalence of the disease in certain localities, (Alps, Rhone Valley, bottom lands) would suggest that local conditions in food or water are factors, though we cannot as yet fully explain the mode of causation. In the same way we must recognize the influence of hepatic and gastric disorders, which arise from such faulty regimen and affect the brain by nervous sympathy and by the action of toxic elements thrown into the circulation. Then again we must take sufficient account of the congestions resulting from obstructions in the lesser circulation, disease of the lungs, and of the right heart, and compression of the jugulars by a tight or badly fitting collar, or compulsory curving of the neck as set forth under vertigo and cerebral congestion.

Symptoms. Among the symptoms must be recognized the conformation with which it is usually found associated. The predisposed animals are usually low bred, common horses, with

narrowness of the cranium and space between the ears and with a retreating of the head from the orbits to the poll. Other horses suffer but the majority are of this conformation, and thus the disease acquires a hereditary basis.

The expression of the face is characteristic. The eye is dull, often sunken, lacking in vivacity and life, the eyelids are semi-closed, the ears do not prick up to sounds, the muscles of the face are relaxed, so that the lips hang flaccid, and the nostrils fail to dilate freely and rhythmically. The animal is apparently unconscious of all that goes on around him, and is not aroused by the entry or exit of men or horses, by voice or slap, by food or water. His head is probably dropped and resting in the manger, and he raises it sluggishly when compelled ; when moved from side to side of the stall his legs may retain a position turned outward or crossed one over the other ; if energetically roused he wakes up slowly, and almost immediately relapses into his former lethargy, without accomplishing what was called for. When left with legs crossed he often remains so until wearied by the constrained position, or in danger of falling from loss of balance. Not only the legs but the head will retain for a time an abnormal position given to it,—bent, dropped, turned to one side or the other.

This same lethargy extends even to mastication, which is usually performed slowly and indifferently, and is often interrupted in the middle of the trituration of a morsel which remains in the cheek, on the tongue, or between the teeth, and perhaps hanging out of the mouth. Hence the horseman's expression, *he smokes his pipe*.

His mode of drinking is no less singular. Usually the lower part of the face is dropped deeply into the water, and he will only withdraw it when it becomes necessary to breathe. He may continue to masticate while drinking.

When walked or trotted he may move a short distance all right ; he may even hasten his progress for a short distance without refusing meanwhile to respond to the rein, then he may stop and for a short time longer resist all efforts with voice, whip, or spur to start him anew. In other cases he will turn to one side, getting into ditches or fences by an apparently involuntary action and in defiance of whip or reins.

One of the most striking features of the disorder is the difficulty of backing. In some cases he will back a few steps and then prove unable to back farther; in others he will show it best when heated with a journey; in other cases still he will back well enough under his own weight, but prove utterly unequal to the act if a heavy man is placed on his back, or if hitched to a loaded wagon. In backing with or without a rider the horse, pulled back by the reins, inclines backward with his hind limbs extended forward beneath the abdomen, his forefeet extended in front, and his back arched; he extends his head or turns it to one side, and when the change in the centre of gravity endangers his equilibrium, he draws back his forefeet without lifting them, each making a groove in the ground, and at the same time he makes a disorderly motion of the hind limbs to one side to restore the balance. In default of this he may drop his quarter on the ground and perform a back somersault on his rider. Even when he succeeds in balancing himself after dragging the forefeet back, the difficulty of further backing is rather increased, as the nervous irritability is enhanced by a continuance of the excitement.

When hitched in a carriage the phenomena are virtually the same; when backed he extends or flexes the head, inclines the body backward, and after a time loses his equilibrium, sometimes executes a few disorderly steps backward, or throws himself violently to one side, or turns over backward in the shafts.

The symptoms are always worse during hot, damp weather, and when the animals are exposed to the full glare of the sun. The milder cases can be worked without great inconvenience in winter, while they become utterly useless in summer.

Active exertion and increased rapidity of the circulation has a similarly injurious effect. After a period of rest nothing amiss may be noticed, while after a period of work in the sunshine the symptoms become well marked and the difficulty of backing pronounced. Plethora or full feeding aggravates, while spare, laxative diet, laxatives, rest or bleeding relieves.

The affection may become complicated by more active inflammatory action leading to paroxysms resembling those of meningo-encephalitis;—pushing against the wall, rearing up with the fore feet in the manger, acting as if walking or trotting, etc. In other cases the paroxysms resemble those of vertigo; the animal

plunging forward, starting to one side, or rearing up and falling back.

Lesions. The pathological anatomy of this disease is that of chronic hydrocephalus. Renault records cases in which the subarachnoid and arachnoid fluids were under the normal while the fluid in the ventricles was increased to a marked extent. This accumulation is often so great that the whole of the surrounding nervous matter is greatly attenuated, the convolutions of the cerebral hemispheres are flattened so that the sulci are all but effaced, the water may shine through at points and even bulge after the manner of a hernia, the ganglia in the ventricle (corpus striatum, optic thalamus, hippocampus) are flattened and atrophied, the base of the cerebrum is thinned and bulges downward, and the olfactory lobes may have their internal cavity greatly distended so that they look like little bladders of fluid. The ependyma may have lost its normal thinness and translucency, having become thick and opaque, and sometimes its surface is granular and rough. The choroid plexus is congested and swollen with infiltration. The brain tissue adjacent is firmly adherent and there is a hyperplasia of its connective tissue constituting a veritable sclerosis. At some points, however, the compressed nervous tissue has undergone degeneration and softening. As might be expected from the pressure of the liquid, anæmia of the brain tissue is a marked feature of the morbid condition.

Other conditions have at times been found in chronic hydrocephalus.

Renault found two long tumors each as large as a hen's egg projecting from the dura mater into the cerebral hemisphere. In other cases there have been fibrous thickening of the dura mater, exudations on the pia mater, and false membranes on the arachnoid (Röll). Chabert and more recent writers have observed cysts and tumors of the choroid plexus in such cases, but these have been met with not unfrequently in the entire absence of the characteristic symptoms of this disease.

Nature. The affection before us is evidently one in which the majority of the higher brain functions are profoundly depressed or debilitated, and this is accounted for by the accumulating intraventricular liquid pressing on the ganglionic centres in the cerebral hemispheres, and in the floor of the lateral and third ventricles.

Experimentally an approximate loss of sensation, intelligence, spontaniety, will, and muscular power is produced in birds or mammals deprived of their cerebral hemispheres. Colin's heifer, which had been thus mutilated, would lie in torpor, and though it could be made to get up and walk, it struck its head heedlessly against the wall, and retained in its mouth unchewed, the food that had been placed there. He says of such cases: "they live a long time, move automatically, respire, digest, but they lose, with the sensations, memory, judgment, will, and the most vital instincts of their kind."

In the dropsy of the ventricles the attenuation and atrophy of the cerebral convolutions produce symptoms which approximate closely to those resulting from their experimental ablation, so that one may fairly attribute the general symptoms in the two cases to the loss of their function. Many of the attendant symptoms, and especially the aberration of smell, sight, hearing and taste, may be referred to the concomitant injuries of the basal ganglia of the brain.

We need not seek in one general answer to resolve the question whether the dropsy or inflammation is the initial lesion. For our present purpose it must suffice, that dropsy with anæmia and atrophy of the cerebral convolutions and basal ganglia produce the symptoms of immobility.

At the same time it is only logical to conclude that any morbid condition of the cerebral circulation or of the brain or membranes which leads to a corresponding amount of ventricular effusion, or atrophy or destruction of the nerve centres already designated, will produce the symptoms characteristic of this disease. Thus the different forms of meningitis, traumatic injuries to the cranium, chronic encephalitis, cerebral softening or degeneration, sclerosis, neoplasms of all kinds affecting the brain (cysts, cholesteatoma, psammoma, melanoma, etc.), and parasites may occasion this disease.

Prognosis. The disease is essentially incurable. It may last for years with little change except the winter improvement, but it rarely subsides permanently. It is only in those cases in which the symptoms have been determined by a transient or removable cause, as a moderate exudation or a parasite with a short term of life that a favorable result may be looked for. Usually the im-

provements seen in cool seasons or stables, under good hygiene, are not recoveries but temporary amelioration only.

Symptoms in other animals. Corresponding conditions produce similar symptoms in cattle, sheep, swine and dogs, but the disease receives less attention in these animals because they are not called on for steady work. The animals are lazy, dull, insensible to excitement, stupid, show a lack of muscular power and control, stagger or move disorderly and show tympanies or other indications of indigestion.

Treatment. Majendie and others had a few apparent recoveries after violent counter-irritation over the spinal cord (cervical and dorsal). Coculet and Lafosse claimed recoveries from the prolonged use of nux vomica in large doses (up to 5 drachms). Hayne attempted evacuation of the fluid by puncture through the perforated plate of the ethmoid bone, but had evil results from the ensuing hæmorrhage and encephalitis. Aseptic puncture through the plate of the frontal bone would be much more promising. Klemm suggested hydrochlorate of pilocarpin (15 grains), and this would promise better than any other measure to induce absorption of the liquid. The fatal drawback to this as to other measures is that it is not applied until the slow, steady pressure has caused such extensive cerebral atrophy that, even if the liquid could be removed and its reproduction prevented, the lost functions can never be restored. If the disease could be diagnosed and treated before this change of structure had taken place, the hope of recovery would be much better founded. Even in cases which make a temporary recovery during cold weather one would be warranted in using active derivatives toward the bowels and kidneys, also pilocarpin, counter-irritation to the spine and even tapping of the ventricles.

Legal Aspect of Chronic Hydrocephalus.

To claim relief in case a horse affected in this way is sold as sound, the seller must be notified at an early date. In the different countries of Europe a limit is set after which such notification will have no legal value. The seller must be notified, in France in 9 days, in Saxony in 15 days, in Bavaria, Wurtenberg and Baden in 21 days, in Hesse and Prussia in 28 days, and in Austria in 30 days.

Diagnosis. The veterinarian called to act as expert in such cases must examine the suspected animal along the different lines in which the cerebral aberration is manifested in the disease. He will see the animal standing quietly in the stall apart from all sources of excitement. See if there is a defect in the breadth of the cranium, or a deflection backward of this region from the straight line of the front of the face, together with a heavy clumsy head. Is the head pendent, resting on the manger, with dull eye, drooping lids, lack of expression, loose hanging lower lip? Does he hold morsels unchewed projecting from the mouth or over the tongue, or in the cheek? Does he plunge both mouth and nose in the water to drink, and masticate meanwhile? Are his legs found crossed or in abnormal positions, and if put in such positions, does he fail to rectify them at once? Is the head left in an abnormal flexed, depressed or lateral position if placed in it? Does the subject find it difficult or impossible to back? Does he pay the customary attention to the going or coming of other horses, to feeding, etc.?

If no distinct symptoms are found he should then be examined under other conditions. In taking out of the stable how does he turn in the stall or back out of it, and in what way does he approach the door, clumsily or with difficulty? When moved in a circle, does he sway or stagger? Can he back when mounted or attached to a heavy carriage? Can he execute all these movements satisfactorily after the respiration and circulation have been excited by walking, trotting or galloping in hand or otherwise? In case of difficulty in backing, in the absence of the other diagnostic symptoms, he must see that this does not arise from other causes. Some untrained horses have not been taught to back and cannot be made to do so at once. Some refuse to back from indocility or stubbornness, but can perform the act if induced in other ways, as in having a narrow stall. Sometimes a sore mouth, from a hard bit may make a horse nervous and obstinate so that he will seek to escape in any other way rather than by backing under steady pressure of the bit. Sometimes he will back all right under a halter. In all such cases of simple obstinacy or fear of pain, the absence of the other symptoms is strongly suggestive. If the horse has a well developed cranium, a full bright, alert eye, firmness of lips, intelligent expression, readiness to

appreciate and respond to all noises, words, touches or other causes of excitement, plenty of fire and spirit, and an absence of any apathy, dulness, awkwardness of movement or position of the limbs, or of any other sign of failing nervous power he may be considered free from this affection, even if he refuses to back in a docile manner. In other cases there is a distinct physical incapacity quite apart from any brain disorder. Sprains or anchylosis of the back or loins or anchylosis or painful arthritis of the hocks, may hinder backing.

The diagnosis from encephalitis and other inflammatory affections associated with stupor, rests on the absence of hyperthermia, of the congestion of the orbital and nasal mucosæ, of the heat of the head and of the paroxysmal attacks of excitement which characterize these diseases.

CEREBRO-SPINAL MENINGITIS.

Definition. Epizootic manifestations. Faulty hygiene, insanitary stables, impure air, defective drainage, fermenting food, overwork, overfeeding, excitement, heat exhaustion, electric tension. Probably complex. Horse, ox, sheep, goat, dog. Microbian factors in man and rabbit. Lesions: meningeal, brain and spinal congestion, effusion, suppuration, circumscribed necrosis, softening, petechiæ. Blood dark, fluid or a diffluent clot. Symptoms: *horse:* paresis, anorexia, dysphagia, mucous congestion, reddish brown: in severe cases, chill, stupor, apathy, debility, palsy, tonic spasms of neck, back or loins, hyperæsthesia, twitching, trismus, hyperthermia, delirium, coma, convulsions, and early death. Duration averages 7 to 15 days. *Ox*, as in encephalo-meningitis. *Sheep*, microbes. *Dog*, dulled senses, stupor, coma, palsy, hyperthermia, heat of head, spasms, etc. Diagnosis: by brain and spinal symptoms; cases in groups. More sudden than tetanus, or rabies, and shows no mischievous purpose, nor depraved appetite: from tubercular meningitis. Treatment: Avoid suspected stable, food, water, or suspicious environment, disinfect, correct local diseases, unload bowels, belladonna, atropia, chloral, bromides, ergot, phenacetin, potassium iodide. Bleeding. Cold to head or back. Derivatives. Sling. In convalescence, regulated diet and tonics.

Definition. Concurrent inflammation of the meninges of the brain and spinal cord.

This appears at times in many horses in the same locality, as in New York in 1850 (Large), in Denmark since 1852 (Stockfleth, Bagge), and in Egypt in 1876 (Apostolides). In Cairo alone

about 6,000 horses, mules and donkeys perished. Hence the disease is known as *epizootic cerebro spinal meningitis*. But again it is often seen in scattering or sporadic cases. Add to this that no evidence has ever been adduced that the disease is communicated from one animal to another, and in these days of the parallelism of *epizootics* and *pathogenic microbes*, we may well hesitate about continuing to use such a qualifying term. Friedberger and Fröhner claim "that a large number of clinical facts have been erroneously reported under the name of spasm of the neck. Rabies, tubercular basilar meningitis, apoplexy, simple encephalitis, and certain poisonings have been confounded with that disease." They assure us that "cold, damp, chilly weather, hot stables, clipping and overfeeding are of but secondary importance," but they fail absolutely to tell us what is of *primary importance* in a causative sense. American writers who have attempted to account for the disease have groped somewhat blindly for causes in the idea of poison. Large charged it on insanitary conditions, poisonous gases, and defective sewerage in cities, and lack of drainage and deficient stable ventilation in the country. J. C. Michener attributes it to foods undergoing fermentation and considers it as a paralysis due to toxic fungi. W. L. Williams, in Idaho, found the greatest number of cases in winter had been fed hay made from alfalfa (lucerne) and timothy, though some had small grains and native grasses. The soil was dry, porous, gravelly, devoid of humus, and lying on lava rock. The altitude and clearness of the atmosphere were supposed to exclude the idea of cryptogams, yet the crops generally were raised by irrigation. The water was from clear mountain streams. Stables were generally low and full of manure, with thatched roofs, but hardly tight enough to be called close. In these cases the defective stable room, the irrigation, the leafy hay (lucerne), and the probable presence of ferments (bacteria), are the only suggestive conditions. In a fatal outbreak which I saw among the Wilkesbarre, Pa., pit mules, rain-soaked and badly fermented timothy hay, overwork in view of a strike, and a Sunday's holiday in an unshaded yard under a hot July sun, in contrast with the previous darkness and coolness of the pits, coincided to disturb the general health. In several of the Southern States it is attributed to worm-eaten corn. Trum-

bower thinks it should be traced to the parasitic fungi that grow on plants, grains, and vegetation. In many instances the disease has appeared simultaneously with the feeding on certain specimens of brewer's grains, oats and hay, so that to use Trumbower's words these were the carriers if not the prime factors of the disease.

In recognizing how much cryptogams and bacteria vary under different conditions of life, and what various products they elaborate at different stages of their growth, we can theoretically explain the absence of the disease at one time and its presence at another under what seem to be identical circumstances, as also the variety of symptoms shown in different outbreaks. While this causation cannot be said to be absolutely proved, it is not antagonistic to the facts in many of the best observed outbreaks, and may serve as a hypothetical working theory until actual demonstration can be furnished. The affection suggests a narcotic poison introduced from without, rather than a disease due to a germ propagated in the system.

This need not, however, exclude the operation of attendant conditions such as over work, plethoric feeding, excitement, close stables, heat exhaustion, etc., which tend to bring about cerebro-spinal congestion. Even the electric tension of Idaho, of the United States generally, and of Egypt, in connection with their comparatively dry atmosphere, should not be overlooked in considering the possible causative factors.

In all probability as we learn more of the true pathology of the disease, we shall come to recognize not one, but several toxic principles, and several different affections each with its characteristic phenomena in the somewhat indefinite affection still known as cerebro-spinal meningitis.

The malady has been described in horses, oxen, sheep, goats and dogs, attacking by preference the young, which are not yet inured to the unknown poison, and by preference in winter and spring, the periods of close stabling, dry feeding and shedding of the coat.

In the absence of bacteriological data from the horse, it may be noted that in man cerebro-spinal meningitis, has been commonly found to be associated with the presence in the meningeal exudates of the *micrococcus pneumoniæ crouposæ*, (Micrococcus lanceolatus

encapsulatus). This is frequent in the mouths of healthy persons so that some additional accessory cause must be invoked to increase the susceptibility or lessen the protective power of the tissues. This has been thought to be found in the concurrent presence of other bacteria, the staphylococcus pyogenes aureus, pneumobaccillus of Friedländer and the streptococcus pyogenes. Mosny appears to have established this for the staphylococcus in the case of rabbits. With a given dose of the micrococcus pneumoniæ death was always delayed for a fortnight, while with the same dose thrown into one thigh, and the staphylococcus aureus in the other, the rabbit died in one day. This enhanced potency resulting from the presence of the golden staphylococcus has been invoked to account for the germ making its way from the mouth to the brain in cases of otitis, suppuration of the Eustachian tube, tonsilitis or nasal catarrh. These remarks are intended to be suggestive, rather than conclusive, as we have as yet no certainty that cerebro spinal meningitis in the horse is caused by the same germ as it is in man.

Lesions. The lesions are usually those of leptomeningitis, or congestion of the brain and spinal cord and often effusion into the ventricles, with a serous exudation under the pia mater or into the arachnoid cavity. This may be transparent and yellowish, or grayish and turbid, or milky. In the sheep, Roloff has found purulent products under the pia mater, around the roots of the spinal nerves, and in the surface layers of cerebral gray matter. The marked hyperæmia on the surface of the gray matter is a striking feature, and circumscribed areas of necrotic nervous tissue and softening are not uncommon. Petechiæ are frequent on the meninges, the brain, heart, lungs and kidneys. Granular and fatty degenerations are also met with in these parenchymatous organs. The blood may be dark and liquid or diffluent.

Symptoms in the **Horse.** The mildest attacks are manifested by paresis, or loss of perfect control over the limbs, or loss of power over the tail, impairment of appetite and some difficulty of swallowing, together with some congestion or reddish brown discoloration of the orbital and nasal mucosæ. In other cases paralysis of one or more limbs may supervene but without marked fever or coma.

The more severe forms are ushered in by violent trembling, or by stupor, apathy, and extreme muscular weakness, or actual paralysis. In such cases the animal may stagger or fall. Dysphagia or inability to swallow is often a marked symptom, the saliva falling in strings from the lips. Another common phenomenon is the rigid contraction of the muscles of the neck, back and loins, the parts becoming tender to the touch and a more or less prominent oposthotonos setting in. Twitching of the muscles of the shoulders and flanks may be noticed. Trismus also is sometimes seen. The breathing is usually rapid and catching and the temperature 104° to 106°. The pulse may be accelerated and hard, or weak and soft, or alternating. The eyes are violently congested, of a brownish or yellowish red color, and the eyeballs may be turned to one side. Paroxysms of delirium may set in, when the animal will push against the wall, or perform any of the disorderly movements described under meningo-encephalitis. Sooner or later coma and paralysis supervene, and death occurs in from five to forty-eight hours. In the most acute (fulminant) cases the animal falls and dies in convulsions. On an average the disease lasts from eight to fifteen days. In the more favorable cases, without any supervention of coma, recovery may begin on the third or fourth day.

Symptoms in the **Ox.** These are largely those of encephalomeningitis. If they do not come on with the customary violence, there may be at first difficulty in prehension, mastication and swallowing of food; a rigid condition of the muscles of the neck, back, and sometimes of the jaws, and twitching of the muscles of the limbs, neck, lips, or eyes. For a time there may be hyperæsthesia, restlessness and irritability, stamping of the feet or shaking of the head, then there is liable to follow, dullness, apathy, stupor, coma and paralysis. As in the horse, the distinction from ordinary encephalo-meningitis will at times rest on the prevalence of the epizootic disease in the locality.

Symptoms in **Sheep.** The attack is described as coming on with weakness, dullness, lethargy, salivation, convulsions, opposthotonos, grinding of the teeth, succussions of the body and limbs, heat of the head, and stupor or paralysis unless death ensues during a paroxysm. The congestion of the head and of the encephalic mucous membranes, and the deviation of the eyes

are constant features. Wischnikewitsch describes an extended outbreak in sheep in which the brain lesions were complicated by hepatization of the lungs, and bacilli were found in the various exudates. This reminds one of the presently accepted cause in man, which is, however, a micrococcus rather than a bacillus.

Symptoms in **Dogs.** These are described as some aberration of the senses, which gradually merges into stupor, coma and paralysis. While the animal is able to keep on his feet he sways and staggers, runs unconsciously against objects, or walks in a circle. There is heat of the head, injected eyes, sometimes drawn back or squinting, opisthotonos, and general spasms occurring in paroxysms. The duration of the disease is about the same as in the horse.

Differential Diagnosis. From other forms of meningitis this is easily distinguished. Fulminant cases almost all belong to this type, the fact of the coincident implication of brain and spinal cord is strongly suggestive of this form, and the occurrence of many cases at once, without any demonstrable toxic or thermic cause, is tolerably conclusive. From tetanus there is this added distinction, that the disease does not set in so slowly, the spasms of the neck and back are not so persistent, and stupor sets in early, in a way that is unknown in lockjaw. Rabies is recognized by the slow onset, the characteristic prodromata, the mischevious disposition, the depraved appetite, and by the history of its local prevalence. Tubercular meningitis in cattle has a similar association with tuberculous animals in the same family or herd, and often by the local indications of tubercle elsewhere, emaciation, unthriftiness, cough, flocculent and gritty nasal discharge, enlarged lymph glands, pharyngitis, mammary disease.

Treatment. With a disease so fatal *prevention* should be the first consideration and especially when it appears in an enzootic form. Even in the absence of a definite knowledge of its germ or toxin, it is logical to avoid the locality, condition, food or water by which such germ or toxin has presumably entered the system, together with every unhygienic condition, which may have reduced the resistance of the system and laid it open to the attack. The animals should be removed to a clean, airy, building and the old one should be thoroughly emptied, purified and whitewashed, the lime-wash containing 4 ozs. of chloride of lime, or 1 dr. of mer-

curic chloride to the gallon. Drains and gutters should have special attention and the animals should not be returned until the stable is thoroughly dry. A change of feed is imperative when there is any suggestion of damp, mustiness or fermentation, and even in the absence of such indications, since the ferments and their products may still be present in a dried condition. It should also be an object to correct any morbid or pyogenic condition of the pharynx, Eustachian pouch, nose or ear, by appropriate measures and the inhalation of sulphurous acid or chlorin may be resorted to with advantage.

As medicinal treatment Large advises to give at the ontset 1 oz. aloes with one or two drs. of solid extract of belladonna and as an eliminant, derivative and nervous sedative there is much to be said for it. In case the difficulty of swallowing should prove a serious barrier a hypodermic injection of ½ dr. barium chloride, 1½ to 2 grs. eserine, or 2 grs. hydrochlorate of pilocarpin together with ¼ grain of atropin, may be employed. As a substitute for atropin, ergot, potassium bromide, chloral hydrate, chloroform, or phenacetin have been tried in different cases with varying results. Iodide of potassium has been employed with advantage in the advanced stages and in convalescence, and may be usefully employed in the early stages as a sedative to the nervous system, a deobstruant and an eliminant, if not as a direct antidote, to the toxins.

Bleeding is generally condemned, yet in acute cases where there are indications of active brain congestion, threatening convulsions or coma it may tend to ward off a fatal result.

Cold applications to the head are generally commended. Bags of ice or snow, irrigation with cold water, or cooling by running water at a low temperature, through a pipe coiled round the head or extended along the spine, will meet the purpose. Applied continuously this constringes the bloodvessels within the cranium as well as on its surface, lessens the exudation, and controls the pain and spasms. This may be advantageously associated with warm fomentations to the feet and limbs, friction, or even the application of stimulating embrocations to draw the blood to these parts. In the smaller animals even warm baths may be resorted to as a derivative, cold being meanwhile applied to the head and spine. This not only lessens the vascular pressure within the cranium,

but secures elimination of toxic matters by both skin and kidneys. Cold pure water should be constantly within reach.

A most important thing in the horse is to put him in slings, if he is at all able to stand with their assistance. In decubitus he rests on his side, with the head on the ground, and lower than the splanchnic cavities. The result is a gravitation of blood toward the head. In the sling, with the head fairly raised the gravitation is the other way and the head is depleted. If the patient is too ill to be maintained in the sling, he may be packed up with bundles of straw on each side, so that his breast may lie on a thickly littered bed, and his head may be elevated.

When convalescence sets in care must be taken to nourish with non-stimulating, easily digested food, gruels, soft mashes, pulped or finely sliced roots. In vomiting animals rectal alimentation may become necessary. The rise of cranial temperature or the aggravation of brain symptoms should be met as needed by the local application of cold, and potassium iodide and iron or bitter tonics may be given if they do not interfere with digestion.

ABSCESS OF THE BRAIN.

Infection, in traumas, meningitis, encephalitis, strangles, etc. Symptoms: evidence of trauma, chill, hyperæsthesia, irritability, drowsiness, giddiness, stupor, spasms, paresis, coma, dilated pupils, congested mucosæ, vomiting. Location indicated by muscular groups involved. Treatment: As in meningitis: trephining in hopeful cases.

The formation of abscess in the cranial cavity has been referred to in connection with injuries to the cranium and meningo-encephalitis. It may here be said in general terms that this abscess is a product of infection. In the horse the most common cause is strangles, and especially such cases as run a tardy or irregular course with imperfect softening and limited suppuration in the submaxillary or pharyngeal region. It is to be looked on as an extension of the purulent infection so as to cause a secondary abscess. The same may occur in case of ordinary abscess in any distant organ. In the brain as elsewhere suppuration may result from direct local injury as in the case of blows by clubs, or yokes, running against walls or posts, falls, the effects of but-

ting, injuries by bullets and otherwise. In these cases, as noticed under concussion, there may be two points of injury (and two abscesses) one, in the seat of the injury, and one in a deeper part of the brain, at the opposite wall of the cranium. Again abscess may result in the brain from extension from a similar process going on in the vicinity. Thus otitis extends through the middle and internal ear to the brain, and its starting point may have been more distant, namely, in the Eustachian tube, or pouch, or in the pharynx.

The *symptoms* vary according to the size of the abscess, the rapidity of its formation and the amount of attendant congestion. In the common cases resulting from strangles, I have usually found the animal down, unable to rise, blind, amaurotic, with dilated pupils, congested mucous membranes, and occasional spasmodic movements of the limbs, neck and head. The symptoms may, however, vary through hyperæsthesia, irritability, drowsiness, giddiness, stupor, local or general paralysis with occasional spasms or convulsions. There may be an initial shivering, and a rise of temperature, yet as pressure on the brain increases it may become normal or subnormal. In circumscribed abscess the symptoms may be much less severe, not perhaps exceeding irritability, drowsiness, and some paresis or local paralysis.

In some such cases one can trace the connection to some primary disease, (traumatic injuries to the cranium, abscess of the diplöe or sinus, parasites in the sinus, otitis, or pharyngeal disease) which serve as an indication of the true state of things. In others there may be circumscribed local manifestations (anæsthesia, hyperæsthesia, hemiplegia, paralysis of special muscular groups, or spasms of the same) which may indicate more or less accurately the exact seat of the lesion. When well defined, this localization of the resultant phenomena, serves to distinguish this and other local lesions, from meningitis which is apt to be much more general in its diffusion. In the carnivora and omnivora vomiting is a marked symptom.

Treatment of brain abscess is usually hopeless, yet the attendant inflammation may be met as in other cases of meningitis. If the seat of abscess can be ascertained surgical interference is fully warranted.

TUBERCULAR MENINGITIS.

Little seen in cattle. Acute and chronic cases. Miliary tubercles in pia. Hydrocephalus. Progresses slowly. Irritability, hyperæsthesia, photophobia, congested conjunctiva, grinding teeth, spasms, squinting, dilated pupils, congested disc, drowsiness, stupor, coma, palsy.

Tubercle of the encephalon has been little noticed in the lower animals, partly because it is especially a disease of early life, while animals usually contract tubercle later in life, and partly because subjective symptoms are inappreciable, and the cranium is seldom opened in post mortem examinations. As the affection usually appears as a secondary deposit, the tubercles elsewhere go a long way toward identifying the nature of the disease in the brain. It has usually been found consecutive to pulmonary tuberculosis.

In a case reported by Fischœder as seen at the Bromberg abattoir, in a 350 lbs. calf, the animal had shown weakness, stupor and a tendency to fall toward the right. The brain lesions consisted of small foci of tubercle on the posterior pillars of the fornix (trigone) and adjacent parts. The left eye had on its inner aspect, near the junction of sclerotic and cornea, a firm mass with tubercular centres, extending inward as far as the retina. The bronchial mediastinal, prepectoral, brachial and precrural glands were tuberculous.

In a case in a cow reported by Lesage there were unsteady gait, impaired vision, and great timidity. Necropsy showed a suboccipital tubercle extending into the frontal sinus and cranium, and invading the brain near the parietal lobe for more than an inch. There were retro-pharyngeal and pleural tubercles as well.

In a case of Routledge's, with extensive recent exudate, the condition advanced from apparent health to extensive paralysis in three days, while in a case which the author obtained in slaughtering a tuberculous herd no special nervous symptoms had been noticed during life. Much therefore depends on the rapidity as well as the seat of development.

The primary lesions in the brain are of the nature of miliary tubercles in the pia mater which becomes congested, rough, gran-

ular, and throws out a free serous secretion. Thus hydrocephalus is a usual concomitant of the affection.

The disease is characterized by its slow advance in keeping with gradual increase of the tubercle and is thus distinguishable from the more acute congestions and inflammations. The earlier stages are usually marked by nervous irritability, hyperæsthesia, intolerance of light, closed eyelids, congested conjunctiva, grinding of the teeth and even spasms partial or general. The second stage shows somnolence, deepening into stupor or coma, or there may be going in a circle or other irregular movement. Squinting usually convergent, dilated pupils and congestion of the optic disc frequently occur. The sleep, stupor, paralysis or coma may set in early and is usually largely due to the amount of exudation and the rapidity of its effusion.

TUMORS OF THE BRAIN. NEOPLASMS.

Existence inferential with similar external tumors. Cholesterine tumors on plexus of lateral, third or fourth ventricle : pea to egg : in old ; concentric layers with abundant exudate. Symptoms : slight, or excitability, dullness, vertiginous paroxysms with sudden congestions, as in encephalitis, sopor, stupor, paresis, coma. Melanoma : mainly meningeal ; pea to walnut ; with skin melanomata in gray or white horses. Cases. Pigmented sarcomata. Diagnosis, inferential. Psammoma : advanced cholesteatoma, melanoma, fibroma, etc. : osteid tumors. Nervous irritation, delirium, spasms, nervous disorder, and paroxysms. Myxoma : contains mucin : cells (in homogeneous matrix) round, spindle-shaped or stellate. Changes to fat (cholesterine). Œdematous connective tissue, neoplasm. Myxolipoma. Myxo-cystoid. Symptoms.

Tumors in the brain are not marked by distinct pathognomonic symptoms, so that their presence is to be inferred as a probability rather than pronounced upon as a certainty.

The most common forms in the horse are cholesterine (cholesteatoma), melanotic (melanoma), sandy, gritty (psammoma), and fibrous (fibroma).

CHOLESTEATOMA.

These are tumors formed largely of the peculiar fat which is found in bile and brain matter, and that crystallizes in flat oblong scales with a notch at one corner. The tumors are usually connected with the choroid plexus and developed beneath the pia mater, and may be of any size from a pea to a hen's egg, or in exceptional cases a sheep's kidney. As a basis there is a stroma of connective tissue permeated by bloodvessels from the plexus. Groups of spherical or polygonal cells fill the interstices while fusiform cells are found in the stroma. There is a variable amount of phosphate or carbonate of lime which in oldstanding cases may give a cretaceous character to the mass. These constitute sandy tumors (psammomata).

Cholesteatomata are especially common in old horses and are manifestly connected with congestion of the choroid plexus and exudation. In a recent case or in a case which has shown a recent cerebral hyperæmia, we may find a central mass of yellowish cholesterine, and surrounding this an abundant yellow gelatinoid exudation. This latter is rich in cholesterine which fails to dissolve along with the rest of the exudate on the occurrence of resolution, and is therefore laid up as the solid fatty material. For the same reason the fatty element is usually laid on in layers, one corresponding to each access of local hyperæmia and exudation. The great tendency to calcareous degeneration has been attributed to the abundance of phosphate of lime in the cerebral exudate.

The *symptoms* of these tumors are exceedingly uncertain. Many such tumors of considerable size have been found after death in animals in which no disease of the brain had been suspected during life. In these it is to be inferred that the accretions were slow, gradual, and without any serious congestion. In other cases the tumor is attended by paroxysms of vertigo, or indications of hyperæmia or meningitis, which will last for several days and gradually subside. It is reasonable to suppose that the tumors are largely the result of such recurrent attacks of encephalitis, and are no less the cause of their recurrence. The

intervals of temporary recovery correspond to the subsidence of hyperæmia and the reabsorption of the liquid portion of the exudate. The manifestations during an access correspond directly to those met with in encephalitis. As in that affection there is usually an initial period of excitement and functional nervous disorder tending to more or less somnolence, stupor, paralysis or coma, with long intermissions of apparently good health. In other cases the stupor or paretic symptoms may persist up to the fatal issue.

MELANOMA OF THE ENCEPHALON.

Black pigment tumors have been found in connection with the brain and especially the meninges, varying in size from a pea to a walnut, and as a rule, secondary to similar formations elsewhere. They are most common in gray horses which have turned white, and may give rise to gradually advancing nervous disorder. Bouley and Goubaux record a case of this kind attended with general paralysis. W. Williams reports the case of an aged gray stallion with melanomata on the meninges and in the brain substance which were associated with stringhalt of old standing. Mollereau in a vertiginous horse found a pigmented sarcoma in the right hemisphere between the gray and white matter, and like an olive in size and shape. There were melanomata around the anus. (Annales de Medecine Veterinaire, 1889). So far as such have been examined they follow the usual rule in melanomata in having a sarcomatous structure.

While it is impossible to make a certain diagnosis without opening the cranium, the condition may be suspected, in gray horses, when melanotic tumors are abundant in the usual external situations (anus, vulva, tail, mammæ, sheath, lips, eyelids, etc.), and when brain symptoms set in and progress slowly in such a way as to suggest the gradual growth of a tumor.

Treatment is hopeless, since if they have invaded the brain, the tumors are likely to be multiple in the organ, and numerous and widely scattered elsewhere.

PSAMMOMATA (GRITTY TUMORS) OF THE BRAIN.

As already noted these sandy tumors are often the advanced stage of cholesteatomata, the abundance of the phosphate of lime leading to its precipitation in the neoplasm. The same cretaceous deposit often takes place in old standing tumors of other kinds, as in melanoma, and fibroma so that the sandy neoplasm may be looked upon as a calcareous degeneration of various forms of intracranial tumors. The same tendency to calcareous deposit is seen in the tuber cinereum (pineal body) of the healthy brain which has taken its name from the contained gritty matter. This tendency to the precipitation of earthy salts may be further recognized in the osteid tumors which occasionally grow from the dura mater.

The gritty tumors are especially found in the older horses in which the tendency is greatest to extension of ossification and calcic degenerations.

Like other tumors these may attain a considerable size before they give rise to any very appreciable symptoms, but having attained a given development—often the size of a walnut, they become the occasion of nervous irritation, delirium and disorder, as indicated under encephalic hyperæmia and inflammation, cholesteatomata, etc. There may, however, be drowsiness, stupor, coma, or paralysis as the exclusive symptom, or there may be spasms and convulsions.

MYXOMA OF THE BRAIN.

Myxoma is a tumor in which mucoid elements or a gelatinoid degeneration and infiltration containing mucin is a prominent feature. The *mucous tissue* which constitutes the tumor may differ little from ordinary connective tissue except that the intercellular spaces contain mucin. Histologically the tissue consists of cells embedded in a homogeneous matrix. The cells may be of various forms, round, (in recent formations) and spindle-shaped or star-shaped, but especially the latter, in the older. When incised a fluid containing mucus escapes in greater or less abundance.

While this has properties resembling albumen it is distinguished by the fact that the precipitate thrown down in it by alcohol is softened and redissolved on the addition of water. The precipitate thrown down in an albuminous liquid is insoluble in water.

The formation of this mucous exudate is liable to be followed by fat so that Virchow considered it as antecedent to fat formation. This is especially noticeable in the early stages of the cholesteatomata of the choroid plexus of the horse, in which, as observed by Fürstenberg, Lassaigne, and Verheyen, the new formation is at first a myxoma, which later becomes filled up with cholesterine.

Recent observations tend to discredit the alleged distinctive character of myxoma. The meshes of all connective tissue contain a perceptible amount of mucin. Œdematous subcutaneous connective tissue contains this mucin in greater proportion and approximates to the condition of mucous tissue. The umbilical cord, which has been long advanced as the physiological type of mucous tissue, has been shown to consist of ordinary connective tissue with an abundance of fluid in its meshes.

Koster denies that the myxoma is a special type of tumor, and holds that it is only a condition that may arise in any tumor which contains connective tissue. In other words, myxoma is only an œdematous condition of the connective tissue neoplasm—fibroma, sarcoma, carcinoma, etc.—due to passive congestion or other circulatory disturbance.

As seen in the brain of the horse the formation is usually of the nature of a myxo-lipoma, as the final outcome is usually the cholesterine bearing mass. In other cases the connective tissue spaces become further distended with the viscous, gelatinoid liquid and form veritable cysts—myxoma-cystoides.

In tumors of this kind affecting the choroid plexus the chain of symptoms is essentially the same as given under cholesteatoma and the prognosis is nearly equally grave. It need only be said that in recent cases in which there is as yet little permanent tissue, measures may sometimes be hopefully adopted, to secure the reabsorption of liquid constituents, and even perchance to remove some obvious cause of passive congestion upon which the effusion depends.

ACROMEGALY. HYPERTROPHY OF THE PITUITARY BODY.

Like other portions of the brain the pituitary body is subject to degenerations and diseases of various kinds. This is particularly mentioned here because of the occasional association of its hypertrophy with the trophic processes of different parts of the body. Along with an over-development of the limbs, and less frequently of the body, an enormous increase of the hypophysis has been found, and the one condition has naturally been set down as the result of the other. In some such instances, of overgrowth, however, some other blood glands, such as the thyroid or thymus, have been found to be hypertrophied, so that at present it is difficult to do more than notice the association observed between the two conditions.

CEREBELLAR DISEASE.

Cerebellum and coördination. Pressure on adjacent parts renders results uncertain. Generic symptoms, ataxia, titubation. Marked symptoms with rapid morbid progress. Treatment: tonic, hygienic.

Whatever functions are exercised by the cerebellum there is no doubt of its control over muscular coördination. It is quite true that disease of any other part of the brain causing effusion, exudation or intracranial pressure will more or less completely arrest the functions of the cerebellum just as disease of the cerebellum producing intracranial pressure will derange the functions of other parts of the encephalon. The general symptoms produced in this way cannot therefore be accepted as indicating the precise localization of an intracranial disease. Dullness, stupor, coma, dilated pupils, choked discs, optic neuritis, and vomiting, are in this sense generic symptoms, which may in the absence of fever indicate dropsy, exudation, apoplexy, tumor, concussion or other lesion, and with hyperthermia may indicate encephalitis or meningitis. But if in the absence of these symptoms and of aural

disease there should appear ataxia, swaying unsteady gait, and staggering, there is a strong presumption of cerebellar disease. This may also be manifested by the other and generic symptoms already mentioned only the diagnosis is not then so certain. Again cerebellar disease may exist without the ataxia and lack of balance, but probably only in cases in which the progress is slow and the organ has had ample time to accommodate itself to the as yet comparatively restricted lesions. The result may be a mere defect of muscular tone, or it may extend to an almost absolute loss of contractility, or it may be of any intermediate grade.

Treatment, which is eminently unsatisfactory, consists in improving the general health and tone, by corroborant medicines and conditions of life, and training the muscles by carefully graduated exercise and even electricity.

BULBAR PARALYSIS. DISEASE OF MEDULLA OBLONGATA.

Impaired innervation of bulbar nerves. Paresis of lips, tongue, and larynx. Roaring. Rapid pulse. Glycosuria, albuminuria. Ptosis. Twitching eyelids. Dysphagia. Paralysis. Treatment, rest, cold to head, laxatives, nerve stimulants, tonics, electricity.

The bulb is intimately connected with the origin of the hypoglossal, glosso-pharyngeal, spinal accessory, vagus, facial, and trifacial nerves and active disease in the bulb is therefore likely to entail impairment of the function of several of these nerves. In man this is recognized in *chronic progressive bulbar paralysis*, which almost always affects the lips, tongue and larynx advancing steadily though slowly to a fatal termination. In degenerative lesions there is modified voice, difficulty of swallowing, rapid pulse, and laryngeal paralysis (especially of the arytenoid muscles). The implication of the root of the vagus may be inferred from the arrest of inhibition of the heart, and from glycosuria or albuminuria. Occasionally the ocular and palpebral muscles are involved causing ptosis, or twitching of the muscles. When the facial (7th) nerve is implicated, paralysis of one or both sides of the face may be marked, including often the ears. When

the glosso-pharyngeal, the difficulty of swallowing is a prominent feature, and when the spinal accessory, spasm or paralysis of the neck. In the worst cases death supervenes early, by reason of interference with the respiratory and cardiac functions.

The *treatment* of these affections is usually very unsatisfactory, though in meat producing animals it may sometimes be desirable to preserve them in preparation for the butcher. Rest, in hyperæmic cases, cold to the head and purgatives, and in those in which fever is absent, small doses of nerve stimulants (strychnia) and tonics (phosphorus, phosphates, ammonia-sulphate of copper, zinc sulphate, silver nitrate) may be tried. A course of arsenic and carefully regulated electrical stimuli may at times give good results.

LOCO POISONING. OXYTROPIS LAMBERTI. ASTRAGALUS MOLLISSIMUS.

Astragalus Hornii: A. Lentiginosus: A. Mollissimus: Oxytropis Lamberti: O. Multifloris: O. Deflexa: Sophora Serecia: Malvastrum Coccinium: Corydalis Aurea. In dry regions. Cause, a psychosis. Emaciation. Lassitude. Impaired sight. Illusions. Vice. Refuses other food. Contradictory views. Experiments by Dr. Day.

The term *loco* is of Spanish origin and has come to us through the Spanish speaking residents on the cattle raising plains and the Pacific Coast. The word is defined to mean *mad, crazy, foolish*. It has been applied indiscriminately to a disease in stock manifested by these symptoms, and to a variety of leguminous plants, found growing on the western lands and supposed to cause the disease in question. The plants complained of are Astragalus Hornii, and A. Lentiginosus (Griesbach) in California, A. Mollissimus (Torrey) and Oxytropis Lamberti (Pursh*iana*) in Colorado and New Mexico. Other allied species, and like these found also in the other Rocky Mountain States, Sophora Serecia, Oxytropis Multifloris, O. Deflexa, Malvastrum Coccinium, and Corydalis Aurea var. Occidentalis have been less confidently charged with producing the disease.

These plants grow on poor, dry, sandy or gravelly soils, and having great power of resisting drought, are often in fair growth,

and present an abundant mass of leaves when surrounding vegetation is withered up. Hence, it is alleged, the animals are driven to use it when nothing else is obtainable and once accustomed to it, the desire for more becomes a veritable craze or neurosis, and the victim searches for it and devours it to the exclusion of other food.

The following quotations may serve to illustrate the effects alleged :

Among the symptoms first noticed are loss of flesh, general lassitude and impaired vision ; later the animal's brain seems to be affected ; it becomes vicious and unmanageable and rapidly loses both flesh and strength. Frequently when approaching some small object it will leap into the air as if to clear a fence. The patient also totters on its limbs and appears as if crazy. After becoming affected it may linger many months, or a year, but usually dies at last from the effects of the complaint. (Dr. Vasey. Report of Dept. of Agriculture, 1884).

"I think very few if any animals eat the loco at first from choice ; but as it resists the drought until other food is scarce they are first starved to it, and after eating it a short time appear to prefer it to anything else. Cows are poisoned by it as well as horses, but it takes more of it to affect them. It is also said to poison sheep. As I have seen its actions on the horse, the first symptom apparently is hallucination. When led or ridden up to some little obstruction, such as a bar or rail lying in the road, he stops short, and if urged, leaps as though it were four feet high. Next he is seized with fits of mania in which he is quite uncontrollable and sometimes dangerous. He rears, sometimes even falling backwards, runs or gives several successive leaps forward, and generally falls. His eyes are rolled upward until only the white can be seen, which is strongly injected and as he sees nothing, is as apt to leap against a wall or a man, as in any other direction. Anything which excites him appears to induce the fits, which, I think, are more apt to occur in crossing water than elsewhere, and the animal sometimes falls so exhausted as to drown in water not over two feet deep. He loses flesh from the first and sometimes presents the appearance of a walking skeleton. In the next and last stage he only goes from the loco to water and back, his gait is feeble and uncertain, his eyes are

sunken and have a flat, glassy look, and his coat is rough and lustreless. In general the animal appears to perish from starvation and consequent excitement of the nervous system, but sometimes appears to suffer acute pain, causing him to expend his strength in running wildly from place to place, pausing and rolling, until he falls and dies in a few minutes." (O. B. Ormsby, Report Dept. of Agriculture, 1874.)

"Animals are not fond of it at first, or don't seem to be, but after they get accustomed to the taste they are crazy for it and will eat little or nothing else when loco can be had. There seems to be little or no nutrition in it as the animal invariably loses flesh and spirit. Even after eating of it they may live for years, if kept entirely out of its reach, but if not, they almost invariably eat of it until they die." (Mrs. T. S. Whipple, San Luis, Cal. Report Dept. of Agriculture, 1874).

"Cattle, after having eaten it," Oxytropis Lamberti, "may linger many months, or for a year or two, but invariably die at last from the effects of it. The animal does not lose flesh apparently, but totters on its limbs and becomes crazy. The sight becomes affected so that the animal has no knowledge of distance, but will make an effort to step over a stream or an obstacle while at a distance off, yet will plunge into it or walk up against it on arriving at it." (Dr. Moffat, U. S. Army.)

"The term *loco*, simply meaning foolish, is applied because of the peculiar form of dementia induced in the animals that are in the habit of eating the plant. Whether the animals (horses chiefly) begin to eat the plant from necessity (which is not likely) or from choice, I am unable to say. Certain it is, however, that when once commenced, they continue it, passing through a temporary intoxication, to a complete nervous and muscular wreck in the latter stages, when it has developed into a fully marked disease, which terminates in death from stavation or inability to digest more nourishing food. The animal, toward the last, becomes stupid or wild, or even vicious, or again acting as though attacked with blind staggers." (Dr. Rothrock. Report of Dept. of Agriculture, 1884).

Dr. Isaac Ott, of Easton, Pa., gives the following as the physiological action of the Astragalus Mollissimus: "It decreases the irritability of the motor nerve, greatly affects the sensory

ganglia of the central nervous system, preventing them from readily receiving impressions. Has a spinal tetanic action. It kills mainly by arrest of the heart. Increases the callory secretion. Has a stupifying action on the brain. Reduces the cardiac force and frequency. Temporarily increases arterial tension, but finally decreases it. Greatly dilates the pupil." (*Amer. Jour. of Pharmacy*, 1882).

In opposition to these statements Professor Sayre, of Kansas, after an extended observation, arrived at the conclusion that "it is a grave question whether loco weed is a poison at all; upon chemical examination no poisonous principle of any kind was discovered; no toxic effect was observable when administered to frogs, cats, dogs, or the human species, ... the point cannot be accepted as a settled one whether loco is poisonous to cattle or not."

Dr. G. C. Faville found in *locoed* sheep in Colorado bunches of tapeworms in the gall ducts. Dr. Cooper Curtice, who subsequently studied the subject, found the tænia fimbriata, and believes that to these the symptoms are exclusively due. "The affected lambs are large headed with undersized bodies and hide-bound skins. Their gait is slightly like that of a rheumatic. They seem to have difficulty in cropping the shorter grass; they also appear to be more foolish than the other sheep, standing oftener to stamp at the sheep dogs or the herder than the healthier ones. Others do not seem to see as well, or are so affected that they seem to appreciate danger less. In driving, they are to be found at the rear of the flock." (Animal Parasites of Sheep.)

It is altogether probable that the tæniasis of sheep has been mistaken for *loco*, but this can hardly account for the remarkable symptoms found in other genera of animals, as a concomitant of an acquired and insatiable fondness for these leguminous plants. The tænia fimbriata has been found in sheep and deer, but there is no record of it as a parasite of cattle and horses.

Dr. Sayre's failure to find any poisonous principle in the plants, or any toxic action on frogs, dogs or cats, cannot be received as conclusive in face of the results reached by others. Perhaps Dr. Sayre's specimes were not grown under the proper conditions, or were not collected in the proper season to secure the toxic ingredient.

Miss C. M. Watson, of Ann Arbor, Mich., succeeded in separating a small amount of alkaloid from the root of Oxytropis Lamberti, but did not apply the crucial test of physiological experiment. In the Report of the Department of Agriculture for 1879, are given analyses of Oxytropis Lamberti, Astragalus Mollissimus and Sophora Speciosa, in each of which a small amount of alkaloid was found.

In 1888–9, Dr. Mary Gage Day, of Wichita, Kansas, made careful experiments on cats and rabbits, under the supervision of Dr. Vaughan in the Michigan Laboratory of Hygiene. She used a decoction of roots, stems and leaves of plants gathered in September and gave 60 to 70 c.c. of this to a half-grown vigorous kitten daily, along with abundance of milk and other food. In two days the kitten became less active, showed rough coat, increased desire for the *loco*, with partial loss of appetite for other food, diarrhœa came on, and retching and vomiting occasionally occurred. The expression became peculiar and characteristic. These symptoms increased, and emaciation advanced, and on the 18th day periods of convulsive excitement supervened. These were sometimes tetanic, the head being thrown backward and the mouth frothing. At other times the kitten stood on its hind limbs and struck the air with its fore paws, then fell backward and threw itself from side to side. There were short intervals of quiet, life being indicated by breathing only. After 36 hours of these intermittent convulsions paraplegia set in, and the kitten died in two hours. There was no apparent loss of consciousness before death.

Post mortem examination revealed gastric and duodenal ulcers, some of which were nearly perforating. The heart was in diastole; brain and myel appeared normal; the entire body anæmic.

To a *vigorous adult cat* 60 c.c. to 70 c.c. of a more concentrated solution were given with other food. The results were essentially the same. By the twelfth day the cat was wasted to a skeleton and very weak. Paralysis of the hind limbs came on and the cat died on the thirteenth day.

As a test experiment, two strong young cats were confined in the same place, fed from the same dish, and treated in every way the same, except that the one was fed daily a decoction of the *loco*.

The one fed *loco* acquired the *loco* disease with the symptoms described above while the other, eating ordinary food only, remained healthy.

Subcutaneous injections of the concentrated decoction thrown into frogs and chickens at the Michigan Laboratory of Hygiene, under direction of Dr. Victor C. Vaughan, caused nervous twitching and in large doses, death in 1 or 2 hours from heart paralysis. The same symptoms were produced in frogs by injection of an alcoholic extract of the residue left after evaporation to dryness of the decoction.

The loss of appetite, acquired liking for the "*loco-weed*", rough coat, emaciation, peculiar expression, rearing, plunging, and a staggering uncertain gait are among the symptoms given in the earliest published observations on the loco disease, and agree with the statements universally made by ranchmen. An ulcerated condition of the intestines was also pointed out by Professor Sayre in a locoed cow upon which he made a necropsy (Dodge City Times, July, 1887): but the diarrhœa which was so marked a symptom in the cats experimented on, is not mentioned as a characteristic symptom in horses and cattle.

"From the close agreement of the symptoms in the cats with those universally recognized in locoed horses and cattle, I conclude that the cases described above were genuine cases of the "loco disease" and are, so far as can be ascertained, the first that were ever experimentally produced."

"The craving for the "loco" is soon acquired. The kittens would beg for it as an ordinary kitten does for milk, and when supplied would lie down contented. To determine whether a herbivorous animal would easily acquire the "loco habit" a young "jack" rabbit was captured and fed a few days on milk and grass; then fresh "loco" was substituted for the grass. At first the "loco" was refused, but soon it was taken with as much relish as the grass had been. After ten days of the milk and "loco" diet the rabbit was found dead, with the head drawn back and the stomach ruptured."

"With reference to the character of the plants at the different seasons of the year, I am convinced by numerous experiments, on material gathered in different months, that the greatest amount of poison is present in the autumn and winter." The

scarcity of other food at that period of the year is only a partial explanation of the number of deaths occurring at that season.

Conclusions :

"1st. There is some poison in "loco weed" which may cause the illness and, if sufficient quantity is taken, the death of an animal."

"2d. This poison is contained in the decoction obtained from the plants, and by systematically feeding it to healthy cats cases of "loco" disease may be produced."

"3d. Taste for the green "loco weed" may be experimentally produced in the jack rabbit."

"4th. From the large quantity of the plant or the decoction required to produce the disease, the poison must be weak, or if strong, it must be in very small amount."

LEAD POISONING. PLUMBISM.

Physiological action on nervous system. Sources : near smelting furnaces on vegetation ; paints ; paint scrapings in manure and on soils ; lead packing of pumps, engines, etc. ; sheat lead ; bullet spray ; wall paper lead ; leaden water pipes or cisterns ; lead acetate ; painted buckets ; painted silo ; lead compounds in arts. Experiments on animals. Accidental poisoning : **horse,** fever, gray nasal discharge, salivation, convulsions, paralysis, dyspnœa. **Cattle,** emaciation, dyspnœa, palsy, tonic spasm of flexors of limbs, swollen carpus, death in a few months. Young worst. **Sheep,** lambs paretic. **Swine** in pens escaped, those at large suffered. Post-mortem ; lead or lead compounds in stomach, or shown by analysis, in gastric contents, liver, spleen, kidney, etc. Tests. Treatment : hydrosulphuric or sulphuric acid, sulphate of magnesia or soda, antispasmodics ; in chronic cases, potassium iodide, bitters.

The physiological action of lead is exerted on the nervous system, so that lead poisoning may be appropriately enough treated of as a disease of the nervous system.

Sources. The sources of lead as a poison for animals are extremely varied. In England in the vicinity of lead mines and smelting furnaces it is deposited from the air in a fine powder, and consumed with the vegetation. Herapath found that the deposit, in dangerous amount, began half a mile from the chimney of the smelter and extended about half a mile further.

A second source is in lead paints used about farms and the scrapings of paint pots thrown out with manure and spread upon the fields. These lead combinations will last for years in the soil or on the surface, being plowed under one year and turned up again the next when the occasion of their presence has been completely forgotten. In one case I found the red lead paint marked by the tongues of cattle at the back of an abandoned cottage the fence around which had been broken down. In another the scrapings were found in an orchard which had been near and convenient for throwing them out. In a third case a paint can hung on the branch of an apple tree, well out of the way of the stock as the owner fondly supposed, showed in its contents the marking of the barbed tongues of the cattle. In a fourth case a barrel of paint was set under the barn where there was not height enough to admit the matured cattle, but it bore the marks of licking by the young stock, and they alone died but in such numbers that the owner concluded it must be the "Rinderpest."

The lead packing from the joints of pumps, engines and other machinery, thrown away around works and mines, is a common source of the trouble. I once found large quantities in the gastric contents of cows that had died around a coal mine in Ayrshire, Scotland.

Sheet lead—tea-chest lead—is another common source of the poison. This is thrown out, scattered with the manure on the field, and will resist the elements for years but dissolves when taken into the acid stomach of the animal.

The spray from bullets in the vicinity of rifle butts is another common cause of the poisoning.

In one instance I have seen a cow poisoned by eating some lead-impregnated wall paper which had been carelessly left in the stable.

Less frequently the poisoning comes from drinking water carried in leaden pipes, or left to stand in a leaden cistern. The softest waters—rain, snow, distilled water—are the most liable to this impregnation. The hard waters containing carbonates, sulphates or phosphates, tend to be decomposed, the acid uniting with the lead to form comparatively insoluble carbonates, sulphates or phosphates of lead, which protect the subjacent lead

against solution. The hardness of the water is not, however, a sufficient safeguard, as iron, solder, and other agents present in the lead as an impurity or merely resting upon it, are sufficient to set up a galvanic action resulting in solution.

The salts of lead may find direct access to the animal, as in the case reported by Gamgee in which a farmer used a barrel which had contained acetate of lead for mixing the feed given to his stock. A somewhat similar source of poisoning is found in the use of buckets or silos which have been painted inside, and scale off in contact with hot water, etc.

Blythe enumerates the following compounds of lead as employed in the arts:

1st. Hair dyes which have a basis of litharge, acetate or carbonate of lead in combination with lime and other agents.

2nd. White lead in its various forms is carbonate of lead.

3rd. Newcastle white is white lead made with molasses vinegar.

4th. Notingham white is white lead made with sour ale.

5th. Miniature Painter's white is lead sulphate.

6th. Pattison white is an oxychloride of lead.

7th. Chrome Yellow is impure chromate of lead.

8th. Turner's Yellow, Casella Yellow, Patent Yellow is oxychloride of lead.

9th. Chrome Red is a bichromate of lead.

10th. Red Lead is the red oxide of lead.

11th. Orange Red is an oxide obtained by calcining the carbonate.

12th. Nitrate of Lead is much used in calico printing.

13th. Pyrolignite of Lead is an impure acetate used in dyeing.

14th. Sulphate of Lead is a by product in the preparation of acetate of aluminium for dyeing.

Forms. Lead poisoning occurs in acute and chronic forms. The two forms, however, merge into each other and are largely convulsive and paralytic.

Experimentally. Harnack found that 2 to 3 mgrms. in frogs and 40 mgrms. in rabbits caused increased intestinal peristalsis, diarrhœa, and paralysis of the heart. Dogs had choreic symptoms. Gusserno gave 1.2 grm. to rabbits and dogs respectively, and produced emaciation, shivering and paralysis of the hind extremities. Rosenstein with 0.2 to 0.5 grm. obtained in dogs

similar symptoms with epileptiform convulsions, and Heubel had symptoms of colic in a few cases.

Casual or Accidental Poisoning. Metallic lead is slowly dissolved and therefore large doses of this may be taken in without visible ill effect. Shot has often been given to relieve the symptoms of broken wind in horses, and a dog at the Lyons Veterinary School took four ounces without visible ill effect. When finely divided, however, as in sheet lead or the spray of bullets it presents a much more extended surface to oxygen and acids, and in the acid stomach of monogastric animals, or even in the organic acids of the rumen it is dissolved in quantity sufficient to prove poisonous.

Symptoms in Horses. Shenton thus describes his cases. "There was a rough, staring coat, a tucked up appearance of the abdomen, and a slightly accelerated pulse; in fact, symptoms of febrile excitement which usually, however, passed away in about a week. About this time large quantities of gray colored matter were discharged from the nostrils, and saliva from the mouth, but at no time was there any enlargement of the submaxillary, lymphatic, or salivary glands. Nor was there constipation of the bowels, which appears to be nearly always present in cases of lead poisoning in man. Fits and partial paralysis came on at intervals; and when the animals got down they often struggled, for a long time ineffectually, to get up again. The breathing up to this time was pretty tranquil, but now became so difficult and labored that the patient appeared in danger of suffocation. The pulse was in no case above 60 or 70, and I ascribe the difficulty of respiration to a paralyzed state of the respiratory apparatus. The animals did not live more than two or three days after these symptoms appeared. The *post mortem* appearance varied but little. The lungs and trachea were inflamed; the lungs engorged with large quantities of black blood; the trachea and bronchia filled with frothy spume. In all cases but two the villous part of the stomach presented isolated patches of increased vascular action, and in all cases the intestines, and especially the large ones, were inflamed. The blind pouch of the cæcum was nearly gangrenous. There was nothing remarkable about the liver, spleen or kidneys, except that they were of a singularly blue appearance."

Symptoms in Ruminants. These are described by Herapath as following the erection of lead smelting furnaces in the Mendip Hills in Somersetshire. There were stunted growth, emaciation, shortness of breathing, paralysis of the extremities, particularly the hinder ones, the flexor muscles of the fore limbs affected so that the animals stood on their toes, swelling of the knees and death in a few months. Even if removed to a healthy locality the victims failed to thrive. The effects were most pronounced in the young. Lambs were born paralytic; at three weeks old they could not stand, and palsy of the glottis rendered it dangerous to feed from a bottle. Twenty-one out of twenty-three died early. The milk of cows and sheep was reduced in quantity and quality, and contained traces of lead. The cheese had less fat in it. The dead showed the mucous surfaces paler than natural and the lungs had large areas with abruptly circumscribed margins of a dark red color, surcharged with fluid. A blue line appeared on the gum close to the teeth, and from this a globule of lead could be melted under the blowpipe.

In the cases that have come under my observation paralysis of the hind limbs, emaciation and low condition, have been most prominent in the chronic forms, while these have been complicated by torpor of the bowels, blindness, stupor, coma, and more or less frequent paroxysms of delirious excitement or convulsions in the acute. In the chronic cases the blue line on the gums is an important symptom.

Herepath noticed that near the smelting furnaces pigs escaped if kept in the pen but suffered if allowed to go at large. This is explained by the presence of lead in the forms of oxide, carbonate and sulphate on the herbage, hay and hedge rows, and in short, on all vegetation.

In *post mortem* examination the stomach should be carefully searched for lead in the metallic form as sheet lead, bullet spray, etc., for the different forms of paint of which lead forms an ingredient, for the discarded white lead packing of pipes and machinery, and even for solid masses of metallic lead. This is especially necessary in the case of cattle in which the morbid habit of eating non-alimentary matters is so common, and for which the sweet taste of some of the lead compounds seems to offer an attraction. The lead being long retained in the first

three stomachs in contact with acetic and other organic acids is especially liable to be dissolved and absorbed in dangerous amount.

In the chronic cases especially, the test by electric current may furnish a valuable pointer. In lead poisoning the muscles respond much less actively to the stimulus than in the normal condition.

In resorting to analysis the following table from Heubel of the amount of lead in the different organs of a dog may offer a guide to the selection of an organ for examination :

Liver	.03 to .10 per cent.
Kidney	.03 to .07 " "
Brain	.02 to .05 " "
Bones	.01 to .04 " "
Muscles	.004 to .008 " "

Professor George Wilson found the lead very abundant in the spleen, and used it for analysis. He dissolved it in aqua regia over a slow fire, cooled, filtered, evaporated, cleared, and boiled with dilute nitric acid. Then filtered and dried again, dissolved in dilute muriatic acid, and finally applied the color tests. With hydrosulphuric acid it gives a black precipitate, with sulphuric acid, a white, and with potassium iodide or bichromate a bright yellow. Or from the solution of the chloride the lead may be obtained as a metallic deposit on zinc from which it can be fused into a minute globule on charcoal.

In the *treatment* of lead poisoning the first object is to prevent the further solution of lead in the alimentary canal and to carry it off. To fill the first indication, hydrosulphuric acid or sulphuric acid may be administered to form respectively the insoluble sulphide or sulphate. As a purgative, sulphate of magnesia or soda should be preferred, as favoring at once elimination and the formation of an insoluble precipitate. Large doses are usually desirable, especially in ruminants, because of the bulky contents of the stomach and the torpor of the alimentary canal. If griping is a prominent symptom opium or other antispasmodic must be added.

In chronic cases, after the evacuation of the contents of the alimentary canal small daily doses of potassium iodide will serve to dissolve the lead out of the tissues, while sulphates may be given in small doses to assist in elimination from the bowels and to prevent reabsorption. The treatment by potassium iodide is equally applicable, to assist in the elimination of the lead that

has passed into the circulation and tissues. The doses, however, should in any case be small to avoid the sudden solution of a large amount of lead which had been deposited in the tissues in a comparatively insoluble form. The sudden entrance into the circulation of any large amount of such lead would induce a prompt return of the toxic symptoms. A continuous exhibition of small doses is the course of wisdom and safety. The bowels should meanwhile be kept somewhat relaxed by small doses of sodium or magnesium sulphate. As a general tonic a course of bitters may be called for, especially when torpor or emaciation is pronounced.

ALCOHOLIC INTOXICATION.

Beer in pigs, alcohol in dogs, absinthe in horse, alcohol and burnt ales in cows, alcoholized grain in fowls, also fermented raisins. Symptoms, lack of coördination, staggering, flushed mucosæ, full pulse, stertor, sopor, coma, alcoholic breath, chill, muscular twitching, delirium. Treatment: ammonia acetate, or carbonate, apomorphia, pilocarpin, warm water, coffee, stomach pump, electricity.

Poisoning by alcohol is less common in the lower animals than in man, yet the veterinary journals record a considerable number of cases. We have seen pigs suffer from drinking soured beer; the smaller breeds of dogs (English terriers) which are systematically dwarfed by feeding alcohol are often kept for a length of time in a condition of semi-intoxication. Bissauge records the case of a horse inebriated by a glass of absinthe and a pint of white wine, and that of a cow which died intoxicated 24 hours after she had been given three quarts of pure alcohol (Rec. de Med. Vet. 1895). Dundas records intoxication in cows fed on *burnt ales*. Intoxication of barnyard fowl and wild birds from eating grain soaked in strong alcoholic liquids has been frequently noticed, and Bissauge reports fatal drunkenness among our domestic fowls from eating raisins and other fruits which had undergone fermentation.

The *symptoms* are too suggestive to require notice in detail. There is a lack of coördination of movement, a staggering gait, a disposition to lie, dilated pupils, dark red flushing of the visible mucous membranes, a full pulse, stertorous respirations, drowsi-

ness, stupor, and finally coma. The breath exhales the odor of alcohol, and the temperature usually falls, especially if the subject has been exposed to cold. It may rise later in connection with inflammation of the stomach or brain. Muscular twitchings and delirium are sometimes found, and may occur paroxysmally.

Treatment. A pint of liquor of the acetate of ammonia to horse or ox may quickly relieve the symptoms, or 1 oz. carbonate of ammonia may be given in solution in a pint of vinegar. If more convenient the hypodermic injection of 1 or 2 grains of apomorphia, or of 5 grains of pilocarpin may be employed. Warm water is of the greatest value in securing elimination. A strong infusion of coffee is very effectual. If the patient is a vomiting animal an emetic may be employed, and in case of coma the stomach pump may be resorted to. Cold applied to the head or galvanism may be used to rouse the patient.

In case of gastritis or encephalitis following the attack these must be treated according to indication.

ANILINE POISONING.

Composition. Source. Uses. Toxic action on skin, by inhalation, and by stomach. Symptoms: acute: chronic. Test. Treatment: emesis, purgatives, stimulants, tonics, enemata, bleeding, normal salt solution.

Aniline (Amidobenzene, Phenylamine, $C_6 H_7 N$) is a product of coal tar produced in the manufacture of benzole and of aniline dyes. Being an object of large production and consumption in the arts, its toxic action is seen not infrequently in man, and less so in animals. As used in confectionery it is so diluted that it is rarely or never injurious. On textile fabrics, however, it often causes cutaneous irritation, and when eaten by animals may be toxic. Workmen in the factories usually suffer from its inhalation.

Turnbull gave ½ dram sulphate of aniline to a dog, inducing vomiting in 2 ½ hours and purging one hour later. There were accelerated pulse, labored breathing and paraplegia, followed by recovery in five hours. Other objective symptoms are coldness of the surface, and a bluish or purple color of the visible mucosæ,

the blood failing to take up oxygen. In chronic aniline poisoning in man the following symptoms have appeared: papular, vesicular or pustular skin eruptions, or ulcers on hands feet and scrotum; an odor of coal tar; anorexia, nausea and vomiting; headache, vertigo, stupor, ringing of the ears, amblyopia, muscular spasms, muscular weakness, anæsthesia and motor paralysis especially of the extremities. The fatal dose is 1½ dr. and upward.

Aniline may be extracted from the tissues by petroleum ether, and on the evaporation of the solution, it is left as an oily yellowish mass which gives the following reactions:—with a few drops of sodic hypochlorite a blue or violet blue; with acids a rose red; with bromine a flesh red.

The *treatment* must be by elimination by emesis, or purgation, by removal from aniline fumes or mixtures, and by stimulating and tonic agents. In place of emesis a stomach tube and lukewarm water in large amount may be employed to wash out the stomach. For vomiting animals ipecacuan may be employed. Copious enemata may be given, along with purgatives, to clear out the bowels. As stimulants strong coffee, caffein, camphor, or strychnia may be employed. In case of profound stupor, prostration, or paresis it may be desirable to reduce the amount of aniline in the system by free blood-letting, care being taken to inject subcutem, or into a serous cavity, a nearly equivalent amount of normal salt solution.

POISONING BY NITRO-BENZOL.

Composition. Source. Uses. Characters. Toxic qualities. Convulsions: paralysis: cyanosis; weak pulse; bitter almond odor; dark red urine; sopor, giddiness; reduced size of red blood globules; congested brain, stomach, intestines. Treatment: emesis, purgation, stimulants, electricity, derivatives, bleeding, normal salt solution.

Nitro-benzol (Nitro-benzine, $C_6H_5NO_2$) is a coal tar product, formed in large quantities in the manufacture of aniline dyes and extensively used as a flavoring agent for soaps, sweet meats, etc. It is formed by the addition of strong nitric acid to benzine, and appears as a yellow fluid with an odor resembling, yet somewhat different from, that of prussic acid or oil of

bitter almonds. It may prove deadly to man or dog in a dose of fifteen drops, though most commonly it enters the system by inhalation. In animals the prominent symptoms are convulsions and paralysis, supervening on a period of weak circulation and pulse, and blueness of the visible mucous membranes. The characteristic odor resembling the oil of bitter almonds exhales from the lungs and skin. In man there are dilatation of the pupils, blueness of the lips and nails, pallor of the face, weak pulse, slow breathing (often in the end Cheyne-Stokes respiration), a dark maroon or port wine color of the urine, and amblyopia. In the chronic cases the skin is yellowish, and there are weariness, a dragging walk, headache, morning anorexia, drowsiness, giddiness, numbness of the hands or other parts and emaciation. The blood is chocolate color with red globules reduced in size, in number and in hæmoglobin, but containing an excess of carbon dioxide. The brain is often congested and the gastro-intestinal mucosa like the skin may be yellow (from alleged formation of picric acid). In chronic cases disseminated sclerosis may be seen.

In *treatment* emesis, purgation, stimulants (ammonia, camphor), galvanism, sinapisms to the chest, and phlebotomy, with injection of normal salt solution, may be resorted to, as in aniline poisoning.

POISONING BY CARBON DISULPHIDE.

Used to kill insects in grain, etc., in barns. Locally anæsthetic, and irritant. Inhaled, toxic, causing excitement, anæsthesia, collapse. Large doses, excitement, reckless movements, incoördination, giddiness, sleep, stertor, paraplegia. Small doses, weakness, emaciation, tremors, paraplegia, polyuria, melliturla; convulsions, death. Distortion and varicosity of axis cylinder, and unequal staining of cytoplasm. Treatment: pure air, good diet; massage, electricity, tonics, phosphorous.

This agent is largely used in vulcanizing and other factories where the employes are liable to suffer, and also in granaries, barns, etc., for the destruction of insects in grain and other objects and where animals are liable to suffer.

Locally it acts like chloroform, when confined to the surface, as under a glass or covering, producing very active irritation with anæsthesia.

Inhaled it produces intoxication, excitement, general anæsthesia and finally collapse. In rabbits it causes intense excitement, giddiness, swaying from side to side, and reckless leaps forward, followed by profound sleep with deep stertor, and paraplegia for half an hour after the return of consciousness (Oliver). When taken for a long time in smaller quantity it caused weakness, emaciation, tremors, paraplegia, and death in convulsions. There was polyuria, with excess of sugar but neither urea nor albumin. The large cells in the motor areas of the brain, when stained by Golgi's method, showed the axis cylinder distorted and varicose, and the cytoplasm stained unequally. The action on dogs was essentially the same, and in neither animal were changes in the blood globules observed (Oliver).

In man slow poisoning caused headache and exhilerant intoxication, followed by depression, mental apathy, dullness, loss of memory, impaired vision, hearing, sexual desire and muscular power. Cramps are common (Delpech, Curtis).

Treatment consists in giving pure air, good food, massage, galvanism, tonics, and for the persistent nervous failure phosphorus.

TETANY.

Definition. Casual and experimental cases in animals. Causes: Excision of the thyroid, indigestions with fermentation, rheumatism, infection, malaria, rachitis, want of hygiene, hereditary or developmental irritability, microbian poisons. Symptoms: intermittent spasms with semi-flexed limbs, tremors. Diagnosis: by the complete intermissions of spasms, and by pressure on nerve or artery, rousing them. No fever. Like spasms of ergot. Treatment: thyroid extract, grafting thyroid; remove sources of irritation, anti-spasmodics, warm or tepid baths, electricity.

Definition. Tetany is the name given to a limited contraction of a group of muscles usually in the extremities occurring paroxysmally with intervals, during which it may usually be roused into activity by compression of the nerve or artery proceeding to the muscles in question.

The disease has not been accorded a place in systematic works on veterinary medicine, though cases have been recorded which are supposed to have been of this nature, and in cases occurring

in man and associated with dyspepsia and gastric dilation, Bouveret and Devic have extracted from the contents of the stomach a toxic substance which caused tetanic convulsions in animals. The total removal of the thyroid gland, or even of four-fifths of it (Eiselsberg), in the cat is found to be invariably followed by tetany.

Causes. Beside the origin from the removal or general disease or degeneration of the thyroid, it has been attributed to digestive troubles, associated with fermentations and the production of toxic matters, to rheumatism, infection and malaria, to rachitis and unhygienic conditions. The systemic changes and trials of growth and development, of pregnancy and lactation, seem to be factors in certain cases. A peculiar irritable nervous organization transmitted by heredity is undoubtedly a potent cause, and upon this, bacteridian, leucocytic and other poisons operate so as to rouse the paroxysms.

Symptoms. There are usually prodromata in the shape of dullness, prostration, weariness, and some dullness of the special senses. Fever is commonly absent and the contractions tend to affect both flexors and extensors, but as the force of the first predominates, the affected member is usually held more or less rigidly semi-flexed. The spasm appears suddenly, often taking occasion of some voluntary movement, and may last for several minutes or hours. It is followed by an interval of relaxation of equally uncertain duration. Though usually attacking the limbs and causing the victim to walk on the toes, it may extend to the face, neck or trunk, and constitute an intermittent trismus, opisthotonos, or emprosthotonos.

Diagnosis. Tetany is to be distinguished from tetanus by the complete intermissions of the spasms, and by the voluntary development of these by compression of the presiding nerve or artery. Pressure on the nerve arouses its excitability, and compression of the artery shutting off the supply of blood from the disordered and susceptible muscles, tends to increase their irritability. Ligature of an artery supplying healthy muscles causes simple trembling of such organs. From the spasms of cerebral, spinal or meningeal inflammation tetany is distinguished by the absence of fever, and the complete intermissions of the paroxysms. The spasms of ergotism bear the closest relation to those of tetany

and in the absence of proof of the ingestion of ergot, might well be confounded with them.

Treatment. This consists mainly in doing away with the causes, when these can be ascertained. Portions of thyroid may be grafted if complete thyroidectomy has been performed, or thyroid extract may be given. In the human subject recoveries have followed the expulsion of intestinal worms, the cure of gastric dilation, dyspepsia, fermentations, diarrhœa, rachitis, menstrual irregularity, or auto-intoxication, Fíré has seen recovery follow the extraction of a carious tooth.

The spasms may be met by the internal administration of anti-spasmodics (chloral, belladonna, bromides, opiates), and the external application over the affected muscles of anodynes and anti-spasmodics (belladonna, opium, chloroform, oil of cajeput, oil of peppermint, menthol, etc.). Warm or tepid baths are often of great value and a mild electric current has been found useful.

CONGESTION OF THE SPINAL CORD IN THE HORSE AND COW.

Under this heading Trasbot describes hæmoglobinuria and parturition paresis, but this tends to cover up the more important causes and phenomena of these diseases, which should be kept in the foreground. Spinal congestion is undoubtedly a feature of both these affections, and the sudden onset and rapid recoveries often seen, indicate the absence of inflammatory action, yet this is but an accompaniment of a constitutional morbid state which we think fully warrants a special consideration of each elsewhere (see Hæmoglobinuria; Parturition paresis).

Apart from these affections congestion habitually merges into myelitis or spinal meningitis, and may be considered as the initial stage of these disorders. It owns the same causes and is manifested by closely allied symptoms, but these are less persistent, and may subside abruptly into a condition of health. The treatment will be on the same general lines as for myelitis, but with much better hope of success.

ACUTE MYELITIS. POLIOMYELITIS. INFLAMMATION OF THE SPINAL CORD.

Causes: Stimulating food to excess, sexual over-stimulation, violent over-exertion, hot sun, chill, rheumatism, traumas, injury to spinal nerves, vertebral caries, microbian infection, narcotics, vegetable poisons, cryptogams. Lesions: discoloration of white or gray matter, swelling, friability, softening, extravasations in points, leucocytes in excess, nerve cells cloudy, granular, nucleus enlarged, stain highly, chromophile granules irregular, neuroglia thickened. Symptoms: Hyperthermia, rigor, hyperæsthesia, tonic contractions in neck and limbs, intense lameness, paresis, palsy, muscular atrophy, areas of heat followed by coldness, such parts may not perspire, palsy less complete than in broken back, circulation and breathing accelerated or slow, paraplegia in large herbivora. Diagnosis: progressive onset, hyperæsthesia or rigidity merging into palsy, retention and later incontinence of urine, extreme spinal tenderness, rapid atrophy of affected muscles, skin sloughing. Prognosis, always grave. Treatment: purgation, bleeding, hot fomentations, ice bags, compresses, derivatives, bromides, chloral, potassium iodide, atropia, ergot, electricity, strychnia, soft laxative food, bitters, phosphates.

Causes. Like congestion this may be a result of **plethora** in overfed animals, in those subjected to specially stimulating food like gluten meal, cotton seed meal, beans, peas, vetches in excess, animal food for herbivora (the waste of hotels and restaurants for cows, compressed meat products for pigs), a period of absolute rest on full rations in horses habituated to hard work and full feeding; of **sexual over-stimulation** in males (stallion, bull, ram); of **violent over-exertion**, especially if **under a hot sun**; of **sudden chill** when over-fatigued and perspiring; of **cold rain storms** (Freirier); of **rheumatism** (Kowalski); of **traumatism** (fractures, sprains, slipping with over-distension); of **falls upon the point of the ischium**; of blows upon the back (Cruzel, Trasbot); of **tumors implicating the cord**; of **too violent efforts in serving** by stallions; of **injuries of the great nerve trunks** passing off from the cord (Gull, Trasbot, etc.); of extensions from **caries or suppurations of the vertebræ** (Decoste, Trasbot); of **microbian infection**, as in rabies, distemper, tubercle, dourine, louping ill, milk sickness, contagious pneumonia, influenza, and suppurations; of **narcotic**

poisoning, as from ergot, smut, the poisons of the cryptogams and bacteria of mouldy bread, musty fodder, spoiled meats, fish, etc.; also the poisons of **lolium, vetch, lupin, astragalus, oxytropis, arsenic**, etc.

Lesions. These consist in a yellow or pink discoloration of the white and especially of the gray matter, and a special prominence of the puncta vasculosa in the affected part. Swelling or distortion of the part is not usual. This may involve only a single gray horn, the two horns on the same side, the two inferior horns, or all four at once, or the white matter adjacent may also show the rosy tint, the large puncta, and a characteristic softness and friability. Minute blood extravasations are very significant. Microscopically examined leucocytes are found in abundance in the perivascular spaces and in the neuroglia. The neurons (nerve ganglion cells) are degenerated, being cloudy, swollen, with enlarged nucleus, stain highly, and show enlargement of the chromophile granules. In a more advanced stage the cell has an indefinite outline and the nucleus is indistinct and may fail to take a stain; the chromophile granules are irregular and do not radiate evenly from a centre and many vacuoles appear. This may lead to fatty softening, or to fibrous increase of the neuroglia, and sclerosis.

Symptoms. These vary greatly in different cases according to the part involved, the meninges or some special region of the cord, to the essential cause of the inflammation and its acuteness. Usually the attack sets in slowly in contradistinction to the abrupt attack of congestion. **Hyperthermia** and **rigor** are usually among the first symptoms, though in many cases **hyperæsthesia** is the most marked early symptom. The skin covering the muscles which derive innervation from the affected section of the cord is the most sensitive. This is often so extreme along the vertebral column that percussion on the spinous processes or pinching between the fingers and thumb causes the most pronounced wincing and dropping of the back. Copland and Laposso have noticed that a sponge of hot water drawn along the line of the vertebræ causes acute pain and contractions of the muscles of the back and limbs, which are almost tetanic in their force. This probably implies the existence of meningitis, since the absence of rigidity of the muscles of the neck, back and

limbs, usually implies the absence of meningeal inflammation. It may, however, occur in localized or commencing myelitis. The existence of unilateral lesions and rigidity determines intense lameness, which is further characterized by the most marked hyperæsthesia.

The **morbid phenomena of the motor system** are more characteristically paretic or paralytic than spasmodic. When rigidity or spasm ushers in the attack it is superseded in a few hours or in two or three days by flaccidity of the muscles of the affected part, with imperfect control or even complete paralysis. The muscles affected will depend on the seat of the spinal lesion. If in the neck it may affect fore and hind limbs, and even the chest and abdomen; if in the back or loins it will induce paraplegia, the anterior limit of which will correspond to the seat of the lesion; if near the caudal extremity of the cord, (lumbar portion), paralysis of the tail and of the sphincters ani and vesicæ may be prominent features. Retention of urine and fæces (spasm) may precede incontinence (palsy).

Common sensation may be dull or abolished on one or on both sides. If on one side only, the other may show hyperæsthesia.

Trophic modifications are very marked though they may not be noticeable at first. The paralytic muscles waste rapidly and the impaired nutrition is manifested in the rapid formation of sloughing and intractable sores where pressure comes in recumbency (the hips, stifles, hocks, shoulders, etc.). This is especially noticeable on parts supplied by the cord at or behind the seat of the lesions.

Vaso-motor changes are usually marked by a preliminary hyperthermia of the affected parts, followed by a corresponding hypothermia. Sometimes the affected part of the skin will remain quite dry while the rest of the body is covered by perspiration.

Choked optic disc and retinitis are sometimes present.

The **febrile reaction** which is at first moderate, gradually increases in force; the animals become dull, drowsy, careless of food, and the hyperæsthesia merges into paresis or paralysis. This is rarely so complete as in fracture of the vertebræ. If the inflammation is restricted to the lower columns only, there may be

akinesis without change of the sensitiveness or with hypersensitiveness. If restricted to the upper columns there may be sensory paralysis only on the opposite side.

The **heart sounds and pulse** are usually altered, palpitations may appear early with acceleration and sharpness of the pulse, and this may alternate with a tardy slow pulse with intermissions. **Breathing** also becomes accelerated and in violent cases with trembling, though in moderate inflammation with effusion, softening and degeneration, it is liable like the heart beats to become slow and tardy.

When **vertigo** appears it may be attributed to extension to the bulb or cerebellum, or to the sympathetic implication of these organs.

The frequency with which paraplegia occurs in the large herbivora suggests a special susceptibility of the lumbar portion of the cord, probably in connection with severe muscular effort of the hind limbs.

In protracted cases the fever may run very high, being complicated by septic poisoning from the numerous cutaneous sloughs and sores, as well as by cystitis and nephritis.

Diagnosis. This may be based on the progressive onset, unlike the sudden attack of congestion; on the occurrence of primary fever with hyperæsthesia or even muscular rigidity, merging into a later paresis or paralysis; on the retention of urine, followed by incontinence; on the torpor of the rectum; on the extreme tenderness of the spine in the region of the inflammatory lesion; and on the tendency to rapid atrophy of the affected muscles, and the death and sloughing of the skin under pressure over the prominent parts of the body. The definite localization of the muscular symptoms, and the different temperature and secretion of the affected part of the skin, from the unaffected, are further confirmatory of myelitis.

Prognosis. While always grave, myelitis induced by narcotic elements in the food which can quickly be eliminated from the system, and that which has not caused compulsory decubitus, or persistent retention of urine and fæces, may be considered as hopeful. When, on the other hand, the nature and extent of the lesions have entailed a prolonged paralysis, or in the large animals, (especially solipeds), a persistently recumbent position, there is

little to be hoped for. The degenerated myel, and the badly wasted muscles, combine to prevent rising and the use of the limbs, the sloughing bed sores quickly poison the blood and general system, and the animal sinks beyond hope of remedy. Again, if the fæces accumulate in the rectum causing general retention of the bowel contents and fermentation, the shock to the nervous system and the toxins absorbed add materially to the prostration and danger. Finally the retained urine infected through the blood or by a catheter, quickly passes into ammoniacal fermentation, with softening and detachment of the cystic epithelium, septic infection of the mucosa, and the extension of this infection through the ureters of the kidneys. The complication of infective inflammation of bladder and kidneys introduces one of the most dangerous conditions possible.

Treatment. In an acute case, at the outset, elimination of any extraneous poison should be sought as the first step toward a restoration of the normal spinal functions. Purgatives may be employed to this end, and if the case is urgent and without spasms immediate action may be sought by a hypodermic injection of 1½ gr. eserine and 2 grs. of pilocarpin. Meanwhile the horse may receive a dose of aloes or the cow one of Epsom salts. Abundance of watery or demulcent liquids given by the mouth, or as enemas, should not be omitted.

When plethora has been a prominent factor and symptoms are urgent, a free bleeding (4 to 5 quarts for horse or cow) from the jugular vein may serve to relieve the vascular tension, dilute the vital fluid, and moderate the inflammation. Hot fomentations or sinapisms to the limbs, and even cupping on the neck and chest, may contribute to relieve the tension on the spine. When the temperature is already high, bags of ice may be applied to the tender parts of the spine or those indicated to be the inflamed parts by the groups of rigid or paretic muscles. Wet compresses or evaporating lotions may be substituted. In the absence of mustard, tartar emetic, biniodide of mercury, or euphorbium may be used, or even croton oil in a carefully guarded manner, but cantharides, oil of turpentine, and other agents calculated to irritate the kidneys are to be avoided.

Bromides, hydrobromic acid, potassium iodide, chloral, or belladonna may be availed of. Some prefer ergot, but this, like

strychnia, is of doubtful effect or positively injurious in most cases in the early stages. Even in the early stages electricity may be used in the form of a constant current, which tends to vasomotor contraction and a better tone of the capillaries. The electrodes may be applied along the affected side of the spine so that the current may traverse the affected part. It may be kept up for ten to twenty minutes at a time and repeated daily. Any undue suffering under the current may be accepted as a demand for the reduction of its force or its suspension for the time being.

When the hyperthermia has subsided and the occurrence of paresis or paralysis demands nervous stimuli, these may be sought in counterirritants, strychnia, and interrupted currents of electricity. The blisters already mentioned may be used. Strychnia may be used internally (horse or ox 2 grs., sheep $\frac{1}{4}$ gr., dog $\frac{1}{30}$ to $\frac{1}{16}$ gr.) or hypodermically (horse 1 $\frac{1}{2}$ gr., sheep $\frac{1}{8}$ gr., dog $\frac{1}{100}$ to $\frac{1}{30}$ gr.). Should this excite the animal or aggravate the symptoms it must be stopped and deferred until the inflammation shall have more completely subsided. The same remark applies to electricity which may be tried in the interrupted current, and graduated to the endurance of the patient or entirely abandoned for the time.

If the patient is able to support itself on its limbs, it is best kept in a sling to avoid the formation of sloughs and sores. If it cannot so support itself a very thick soft bed of litter is essential to avoid the sloughing and septic poisoning. Food must be laxative and easily digestible such as mashes, hay tea, and boiled or pulped roots. Fresh green grass may be employed when obtainable.

During convalescence a course of bitters with calcium phosphate and carefully regulated exercise are important. In tardy cases Trasbot especially recommends cauterization.

SPINAL MENINGITIS.

Complex cases. Microbian invasion. Lowered vital tone. Traumas. Poisons, parasites, tubercle, rheumatism, neoplasms, poisonous food. Symptoms: Stiffness, tonic contraction, spasms, hyperæsthesia with warmth, enuresis, paralysis later. Treatment: parallel to myelitis; cold, anodynes, nerve sedatives, and anti-spasmodics, saline purgatives, diuretics. Iodine, electricity, cauterization.

It is often difficult to distinguish between spinal myelitis and meningitis in the lower animals, and the danger of confusion is greater because the two affections are often conjoined. Attacks appear to be often associated with microbian invasion of the membranes, but in its turn this is often favored by the lowered tone of the membranes through mechanical injury, circulatory disorder, trophic changes, or the action of poisons in the blood. Thus the condition may supervene on fractures, partial dislocations or sprains of the neck, back or loins, abscesses pressing on the spine, extension of septic inflammation from poll evil, fistulous withers, or arthritis of the vertebræ, penetration of the membranes by sharp pointed bodies (Reindl found a darning needle in a cow's spinal canal), invasion by microbes in influenza, brustseuche, dourine, rabies, milk sickness, distemper, pyæmia, septicæmia, strangles, louping ill, or Texas fever. The toxins of tetanus may start similar trouble. The larva of cysticercus cellulosa may cause meningitis in dogs or pigs, the sclerostoma in the soliped, the filaria in dogs and strongle in a variety of animals. Tubercle of the meninges is not unknown, and rheumatism is alleged as a cause. Neoplasms commencing in the cord act in a similar way, and the poisons of rye grass, millet, loco, lupins, tares and vetches may act on the membranes as well as on the myel.

Symptoms. In the main these resemble those of myelitis and are often present at the same time, and it is only necessary to note those which are especially pathognomonic. The early rigors are followed by stiffness of the back shown in rising or walking and aggravated by motion. There may be tonic contraction of the dorsal and lumbar muscles amounting at times to opisthotonos. The muscles of the limbs, chest or abdomen or some part

of them may be the seat of tonic or clonic spasm. The skin is usually hypersensitive and this is aggravated by heat. The urine is liable to be retained because of the pain of stretching to micturate. Paralysis usually follows and implies extension to the myel, compression of the cord by reason of exudation, or implication of the spinal nerves at the points of exit. In myelitis on the other hand the spasms may be entirely absent, and paralysis sets in early and extends rapidly according to the seat and extent of the lesion.

Treatment. This will be along the same lines as in myelitis, being aimed at elimination of toxic matters, and the counteracting of the existing inflammation. Anodynes such as bromides and chloral and cold water or ice are especially called for to alleviate pain and hyperæsthesia, and anti-spasmodics like ether, chloroform, chloral, belladonna, etc., to allay the spasm. Saline purgatives too, and diuretics may be availed of to limit effusion and favor reabsorption. In the advanced stages iodine may be freely applied to the spine, and an occasional electric current, or cauterization may be availed of.

ACUTE MYELITIS IN THE DOG. MENINGO-MYELITIS.

Causes : distemper, in long-haired pets, pyæmia, exposure to cold, violent over-exertion, traumas, vertebral caries or abscess. Lesions : in lumbar enlargement, horns yellowish, red, friable, pultaceous, leucocytes in excess, punctiform extravasations, neurons opaque ; granular, filaments diffluent, varicose, sclerosis. Meninges congested, thickened. Symptoms : as in horse, extreme hyperæsthesia, later anæsthesia, tremors or twitching, later paraplegia. Treatment : Laxatives, ice bags, sedatives, later derivitives, cauterization. During convalescence, phosphates, iron, zinc, strychnia. Attend to bladder and rectum, light, laxative diet, pure air and water.

Causes. This disease is a common result of distemper and according to Trasbot, is much more frequent in long-haired and pet dogs than in the short-haired and mongrels. The shelter of the hair, like the warm indoor atmosphere, seems to contribute to a special sensitiveness of the cutaneous and nervous tissues. The infective inflammation of the myel is also seen in pyæmia, rabies and milk sickness. It appears to be further induced by ex-

posure to cold draughts when heated, or excited, by plunging into ice cold water, by lying on cold, damp, stone pavement or metallic plates. Violent over-exertion, excessive fatigue, and a variety of traumatisms are further factors. Kicks, blows on the back, concussion from falling from a window or other height, and sprains received in fighting or otherwise, are common causes. Disease of the vertebræ or abscesses in their vicinity will sometimes extend to the meninges and cord.

Lesions. These are like those in the larger animals, being to a large extent determined by the cause and nature of the lesion, concussion, sprain, fracture, pyæmic, septicæmic, or other infection. The implication of the myel to the exclusion of the meninges is very frequent and the lumbar enlargement is the most common seat of disease. Localization in the brachial enlargement or in one lateral half of the cord is uncommon. The gray matter towards the extremity of the horn is the most commonly involved, reflecting a yellowish, grayish red or deep red color, and breaking down into a pultaceous mass on the slightest pressure. At an advanced stage the altered coloring matter gives to the tissue a brownish yellow color without altering its consistency. The still vital and vascular area around the centre of softening may be slightly swollen and abnormally firm. The neuroglia is the seat of leucocytosis, and minute (usually punctiform) extravasations of blood. The red globules are crenated or otherwise distorted and the white are granular and opaque. The neurons are swollen, granular and opaque and the nerve fibres are more or less diffluent, moniliform and in their substance show no clear outline of white substance and axis cylinder. Interruptions by granule masses and vacuoles are common. In old standing or chronic cases the liquid exudate and granular debris have been largely absorbed and the thickening of the neuroglia by fibrous neoplasm, has restored the firmness or even approximated the part to a condition of sclerosis.

In case the meninges are involved there is thickening by exudation into their substance or on their surface, there may be adhesion between the outer and inner layers of the arachnoid and a serous fluid, red, milky or clear, distends the arachnoid or subarachnoid space. The false membranes, here as elsewhere, are usually red if recent, and increase in pallor with age.

Symptoms. These are in the main the same as in the larger animals. The early excitement usually takes the form of hyperæsthesia. When lifted, pressed, touched or only approached the dog may growl, howl, snap, cringe, cower or tremble, glancing up meanwhile with anxious or pleading eyes. When later, this gives place to anæsthesia no such interference will draw a response. The motor disorders at the outset are mostly of the nature of tremors or twitching of the muscles of the limbs or of those parts of the trunk corresponding to the seat of the lesion. In exceptional cases spasms or convulsions may be shown. Trasbot records a case of very acute myelitis of the brachial enlargement in which there were clonic contractions of the muscles of the neck, jaws and eyeballs, and grinding of the teeth, which condition lasted for thirty-six hours. When this motor excitement merges into paralysis it usually attacks the hind limbs which are extended backward helpless while the animal pulls himself forward by his fore limbs. Some such cases are restless and in continual movement while others are dull, apathetic and indisposed to move. The precise seat of the paresis or paralysis will be determined by the seat of the lesion as in the larger animals. Thus paraplegia is most common, less frequently hemiplegia, palsy of the fore limb, palsy of a single limb, and monoplegias, about in the order named. Palsy of the tail and sphincters implies a lesion of the lumbar section of the cord and is very offensive in the incontinence of urine and fæces especially in long-haired subjects.

Treatment. The abstraction of blood is rarely called for in myelitis in the dog. If admissible at all it is in the case of strong, vigorous, plethoric animals which have been attacked in connection with sudden exposure to cold or accidental concussion, and which are presented for treatment at once. Then leeches may be applied to the abdomen or inside of the thigh, or the jugular may be opened with a lancet. Usually on the other hand the patient is fat, lymphatic, and, if a few days have elapsed, even anæmic, while if he has been the victim of an accident the shock and prostration would forbid any depressive measures.

Derivation toward the bowels may be sought by purgative doses of calomel or jalap. In case of high fever, cold may be applied (in the form of icebags, evaporating lotions or wet cloths) to the

tender portion of the spine. If the attack has followed exposure to cold, salicylate of soda may be given, otherwise the bromide of potassium or camphor. Acetanilid and other antithermic agents may be used with caution.

With the abatement of the high fever and the supervention of paresis, if not before, counterirritants are demanded.

Owing to the propensity to lick and the danger of absorption, poisonous agents are virtually proscribed. Yet Möller advises cantharides, croton oil, mercuric iodide, and oil of mustard, and Trasbot restricts the choice to tartar emetic one part to sixteen parts of lard. This the latter rubs softly along the spine for several minutes. If the dog is closely watched or muzzled this or the mustard or croton oil may be admissible. If otherwise, a long-haired dog may be rubbed on the spine with a combination in equal parts of strong aqua ammonia and olive oil ; or it may have applied for some minutes wet cloths rather hoter than the hands can bear ; or a light cauterization may be made with a Paquelin cautery. At this stage, too, bitters and phosphate of lime may be given. Trasbot has long used with the best results 1 grain doses of neutral, gelatinoid phosphate of lime, repeated two or three times a day. Iron may also be resorted to, or sulphate of zinc. Strychnia and electricity are also of great value as soon as the irritability of the spinal centres will allow of their safe employment. Massage and gentle exercise are important.

From the first, attention must be given to obviate the retention of urine and fæces, and the strict antisepsis or asepsis of the catheter adopted to prevent infective cystitis and nephritis.

Throughout the disease abundant nourishment of an easily digestible quality is demanded. Cleanliness, pure air and general comfort must not be forgotten.

CHRONIC MYELITIS. SCLEROSIS.

Sequel to acute. Result of sprains and spinal injuries. Symptoms: paresis on exertion, lameness in one or more limbs, knuckling, circumductive movement of feet, uncertain planting, dropping, worse if blinded, phenomena progressive. Lesions: sclerosis of cord; absorption of nerve cells and fibres, in gray horns, and columns, superior, lateral and inferior, cord, altered in color, unduly firm, in points softening. Stains deeply in carmine, lightly in osmic acid or hæmatoxylin. Meninges thickened, nerve roots atrophied. Diagnosis: previous acute myelitis; later muscular weakness, and paresis, under exercise; from embolism. Treatment: hopeless if advanced: progress delayed only. Good hygiene, tonics, open air, gentle exercise, pure water, grooming, succulent pasture, nourishing food, alkalines, common salt, phosphates.

Cases of this kind have not been satisfactorily diagnosed, and as a rule domestic animals affected with partial paralysis are rarely allowed to live in a condition in which they are offensive to themselves and owners, a source of constant expense with little or no hope of recovery nor profit. Again, in the case of the large mammals, the prolonged recumbency and the low grade of nutrition in the semi-paralyzed parts, usually entail unhealthy sores and septic poisoning which sooner or later prove fatal. It is only, therefore, in the slighter cases, in which a fair measure of control over the limbs remains, that these cases are likely to survive. Trasbot suggests that many cases which pass for lumbar sprains are really chronic myelitis and on careful examination will show spinal sclerosis.

Causes. These are largely speculative, yet doubtless the same causes which determine the acute form, will produce the chronic when acting with less force and greater persistency. The lesions that are left after an acute attack are calculated to keep up a measure of vascular and trophic disorder which will be found associated with more or less sclerosis.

Symptoms. In Weber's case in the horse (Recueil de Med. Vet., 1884, p. 432) the advance was slow, so that for nearly a year the manifestations were not diagnostic. At first there was weakness of the hind limbs when worked to fatigue. Perfect rest led to improvement, and work, to aggravation which became steadily worse and worse. For a length of time the horse main-

tained good condition, glossy skin, elevated head, alert expression, keen sight and hearing, and normal breathing and pulse. Standing in the stall there was no abnormal position of the limbs, nor evidence of lack of perfect control.

But when moved all this was changed. He showed first lameness in the right fore limb and soon in all four members. The feet were swung and planted uncertainly, the animal swayed and staggered, the limb would knuckle over at the knee or fetlock, or bend at the hock, and be recovered with difficulty. After going slowly for a few steps he moved with greater freedom though still with difficulty, and the trouble was greatly aggravated when the eyes were blindfolded. Then every step threatened to precipitate him to the ground. The symptoms were essentially those of locomotor ataxy.

The tactile sensibility was unimpaired, the loins had the normal sensibility, urination and defecation were natural and the appetite remained good. After ten months he showed loss of condition, dullness of the special senses, stupor, and a special sensitiveness about the head, and resented its handling.

Lesions. Thirteen months after the commencement of the attack this horse was destroyed and the cord was found to be profoundly altered by fibroid degeneration of the neuroglia and absorption of the nerve elements (cells and fibres), the lesions affecting different portions of the gray horns, and the columns—superior, lateral and inferior. The affected portion of the cord usually shows in man a grayish, opaque or translucent appearance, with in some cases a shrunken aspect and undue firmness of texture, with at points, centres of softening. If hardened, the sclerosed sections take the carmine stain deeply, but the osmic acid or hæmatoxylin stain very slightly, contrary to what holds in health. In recent cases there is only slight thickening of the neuroglia, but when the disease is advanced the trabeculæ are thick, dense, and firm, and the nerve fibres have largely disappeared. The coats of the blood vessels adjacent to the sclerosis are thickened and their lumen is narrowed. Thickening of the meninges is not uncommon, either confined to those covering the diseased portion of the cord or extending completely around it. Atrophy of the nerve roots is often appreciable by the naked eye.

Diagnosis. This depends largely on the fact that the condition

follows an acute attack of myelitis, on the supervention of muscular weakness and lack of muscular control, whenever the animal is exercised to fatigue, the morbid symptoms subsiding promptly when he is allowed to rest, the aggravation of these symptoms when the patient is blindfolded and a gradual though slow advance of the symptoms with the lapse of time. From arteritis and embolism it is to be distinguished by the absence of the local symptoms of pain and tenderness, and by the absence of pulsation in the same artery distal of the obstruction and of improvement by the lapse of time or a run at grass.

Treatment. Unless in the very early stages even a partial recovery is not to be looked for. By a run at grass or by gentle well regulated exercise the impaired nerves and muscles may be educated to a better control for a limited period but the progress of the disease is not really arrested and the final issue is likely to be ruinous. Even in man, where 90 per cent. of the cases are connected with syphilis, the fibroid hyperplasia (sclerosis) is not remedied as gummata are, by mercury and iodides. In the soliped, where no such specific disease can be charged, the repair of the structural changes is no more hopeful. The many different methods of treatment in man,—electricity, blisters, firing, stretching of the spine, stretching, of the sciatic and crural nerves,—though inducing transient improvement in many cases, produce no real permanent benefit, and are to be remanded to the region of psychic inferences which have little or no place in the therapeutics of the lower animals. Strychnia, veratrin and other spinal stimulants are of little permanent value. A general hygienic and corroborative treatment may be used with the view of retarding the progress of the disease rather than of curing it. Open air exercise, sunshine, succulent pasturage, an ample supply of pure water, and active grooming are valuable. Nourishing food is all important. Lecithin or the hypodermic injection of spermin or other rich albuminous animal product is useful. A course of bicarbonate of soda and carbonate of iron with or without bitters may be tried. When the animal must be kept on dry winter food, he should have free access to common salt and water. This favors at once absorption, assimilation, and elimination, and by fostering nutrition and the removal of waste matters, it contributes to keep

the disease in abeyance. Phosphoric acid and the various phosphates have been largely used and largely rejected, their main value being in the tonic effect on the spinal centres. Trasbot especially recommends the neutral gelatinoid phosphate of lime as having proved especially valuable in his hands. He gave from 1 grain upward to dogs twice a day.

ARTERITIS (THROMBOSIS, EMBOLISM) OF THE SPINAL CORD AND MEMBRANES.

Conditions of spinal circulation favorable to embolism and microbian invasion. Slow currents. Blood stasis. Free anastomosis a compensation. Symptoms. Treatment.

Facts are wanting with regard to these lesions in the domestic animals, but anatomical, physiological and pathological consideration are strongly suggestive of their occurrence. The vascular network of the spinal cord favors a tardy circulation, and this in turn is favorable to the arrest of solid bodies and the delay, proliferation and colonization of microbes. The median spinal artery receives a supply of blood by two trunks, right and left, entering by the intervertebral foramina at each intervertebral articulation. It has not, therefore, one continuous, equable, onward flow, but rather numerous independent currents corresponding to the entering vessels, and with intervening eddies or areas of comparative stagnation. The nervous material of the cord admits no large arteries but only capillary trunks which anastomose freely in its substance. This would seem to entail a sluggish flow, which would favor microbian arrest and colonization, even if the small size of the vessels serves to shut out clots of any material size. Finally the abundant venous plexus, and especially the two lateral venous sinuses, communicating freely with each other and, through each intervertebral foramen, with the extra spinal veins determine a similar tardy flow that should be favorable to morbid processes. If we pass back of these vessels, we find the posterior aorta to be at once the largest and the most direct channel for the entrance of emboli coming from the left heart or lungs. This danger is counteracted in greater part by the fact that the greater

part of this blood passes into the large vessels which supply the liver, spleen, kidneys, stomach, bowels, and hind limbs, and while embolism is well known in these parts it has not been demonstrated as yet in the spinal cord. The toxins produced in infectious diseases and circulated in the blood can often lead to destruction of the endothelium, and inflammation of the deeper structures. In this way any circulating microbes find a ready infection atrium. Hektoen seems to have demonstrated this in the case of tubercular meningitis. By pressure of the neoplasm on the vessel or by fibroid thickening and contraction of the walls of the vessel, the subsidiary cord is denied its full supply, and degeneration of the nervous substance is invited. In the human subject degeneration of the cord has been shown to follow the line of such diseased arteries. Thrombosis follows in every case in which the serous coat is involved, and embolism can easily occur from clots small enough to enter the capillary vessels. Lamy's experiment of blocking the small arteries with inert powder, shows that this will give rise to foci of hemorrhagic softening, which commence in the gray substance. The blocking, however, must be multiple to produce any material effect, as the free anastomosis of the spinal capillaries otherwise secures an abundant blood supply to adjacent parts. In case of an infective embolism the disease will advance even if the obstruction is single.

The general symptoms of these conditions would depend on the exact seat of the lesion, and treatment would have to proceed on general principles, the object being to check the inflammatory conditions, and trust to the *vis medecatrix naturæ* in connection with rest and good hygienic conditions.

HEMORRHAGES INTO THE SPINAL MEMBRANES.

Meningeal Spinal Apoplexy. Hæmatorrachis. 2. Hemorrhage into the Spinal Cord. Spinal Apoplexy. Hæmatomyelia.

Definition. Causes: violent exertion, blows, falls, morbid blood, fractures, caries, tumors, tubercle, aneurisms. Lesions: Clot between or outside membranes in meningeal hæmorrhage, in gray matter and even in white in myelon bleeding. Cord bulges. If survives, nervous matter absorbed. Symptoms: Sudden stiffness or palsy of given areas; spasms more common in meningeal extravasation. Rapid muscular wasting. No fever at first. Treatment: cold to part; slings; atropia, ergot, lead acetate. Later as for myelitis. Large clot may warrant surgical interference.

In the first of these forms the bleeding takes place between the arachnoid and the two contiguous membranes—pia and dura, or outside the dura. In the second it takes place into the substance of the cord though it may encroach on the pia mater. Both conditions have been attributed to violent muscular efforts or contractions as in draught, racing, fighting, leaping, tetanic convulsions, also to blows on the back, or falls from a height. Morbid states of the blood in which there is a hemorrhagic tendency (scurvy, purpura, hæmophilia, anthrax) may be contributory causes. Spinal fractures, aneurisms, caries, tumors, and tubercle may be additional causes.

Lesions. In meningeal bleeding the clot is found outside the dura, or between the dura and arachnoid which may or may not be ruptured. A clot on the pia mater may press seriously on the cord or may cause rupture of the arachnoid. In hemorrhage of the cord, the effusion usually begins in the gray matter, though it may extend far into the white. It may be circumscribed to half an inch in diameter or affect almost the entire length of the cord. The cord may be distinctly enlarged at the point of effusion, and in exceptional cases the blood may have broken through to the membranes. If the patient survives, absorption and degenerations of the cord are inevitable.

Symptoms. In both forms there is a sudden attack, with

stiffness or paralysis of given muscles and without hyperthermia. Rigidity and spasms of the muscles are more characteristic of meningeal hemorrhage, and early paralysis of the spinal. An early hyperæsthesia is also most significant of an effusion in the cord. Rapid muscular atrophy is also characteristic of this. The two conditions resemble meningitis and myelitis but come on much more suddenly and are unattended by fever.

Treatment. Such cases are not hopeful. Cold to the affected part of the spine, keeping the patient in slings to solicit the good effect of gravitation, and giving ergot or lead acetate internally are among the first indications. Later, the treatment would be practically the same as for meningitis or myelitis. In case of complete paralysis from the sudden formation of a large clot, it has even been advised to cut down on the seat of the injury and evacuate the blood, using antiseptic precautions.

SPINA BIFIDA.

This is an elastic swelling consisting of the spinal meninges enclosing a liquid, and in some cases the spinal cord as well, and protruding between the unclosed laminæ of the vertebral rings, usually in the region of the sacrum or loins. It is essentially an arrest of development on the part of the vertebræ and enveloping muscles and skin, and an extension of the cord and effusion of the meningeal and central spinal liquids, in the absence of their bony and muscular support. Hydrocephalus is no uncommon complication.

The *diagnosis* may be based on the presence of an elastic, somewhat transparent tumor, projecting from the vertebral spines, at birth. If it contains a segment of the cord it is usually flattened, depressed on the summit and often associated with paralysis.

As occurring in the lower animals this need not be treated. In man, careful antisepsis and evacuation followed by injection of a solution of iodine 10 grs., iodide of potassium 30 grs., and glycerine 1 oz., have, in favorable cases, secured obliteration of the sac, but even then the recovery has rarely been complete and permanent. Paralysis and death have usually supervened.

SPINAL CARIES. TUBERCULAR OR OTHER INFECTIVE DISEASE OF THE VERTEBRÆ.

Spinal caries in old horses, sprains, fractures, infections; caries often confined to articulating processes: anatomical form: Oxoido-atloid caries from poll evil, concussions, fights, rachitis. Tubercle of bone and intervertebral cartilage in cattle. Symptoms: distortion, stiffness, rigidity; stands day and night, sensory and motor paralysis: localization of lesion; dyspnœa; spastic palsy back of lesion; effect on tail, sphincters. Diagnosis: progressive tenderness and stiffness of spine, distortion, localized exaltation or depression of nervous function, osteoporosis, rachitis, caries, tubercle, melanoma, abscess, infectious disease. Treatment: Sling in narrow stall, good food, pure air, sunshine, tonics, phosphates.

As seen in the human subject spinal caries is usually tubercular and is known as "*Pott's disease.*" As seen in old horses it appears to be rather a result of other infections, especially purulent, and may have started in connection with traumatic or mechanical injuries to the bones and ligaments. In such cases I have seen it repeatedly in hollow backed horses in which the line of the spine descended abruptly and extremely from the withers and rose again to the sacrum. The distortion was so great that the back appeared as if it could barely sustain the weight of the animal and yet the patients were kept at work and proved useful for light driving. Post mortem examination showed extensive caries and suppuration of the vertebral bodies, confined, however, to one or two segments as if due to a mechanical lesion. In the region of the loins it is much more likely to affect the articulations of the vertebral rings, because of the manner in which these are wedged into each other when the spine is pressed downward. In the neck where the normal movement is so much freer such injuries are much less common. Caries affecting the articular surfaces of the axoido—atloid joint is by no means uncommon. It may follow poll evil, or injuries sustained when a horse runs against a wall, or in the fights of bulls or rams. Disease of the vertebræ may be of the nature of rachitis occurring in this case ife.

In cattle the disease is liable to be tubercular in which case, not only is the bone invaded but the morbid process extends to the

intervertebral cartilage and projecting, presses on the spinal meninges and cord. It may even encircle the entire spinal cavity and strangle the cord. If the pus should extend downward it may form abscess under the spine, and rupture into an internal cavity or externally.

Symptoms. Injuries and disease of the vertebræ may last for a length of time without implicating the spinal cord or nerves. They may then cause only distortion, with stiffness or rigidity of the spinal column. When, however, the displacement of the injured, carious or tubercular bones, the distension of the abscess or the increase of the hyperplasia leads to pressure on the nerve roots, the meninges or cord, nervous symptoms are likely to be developed. Compression of the nerve roots—sensory or motor—may cause sensory or motor paralysis or both, limited to particular areas the outline of which will point to the precise seat of the lesion. If in the recti of the head and other muscles of the neck, it points to the anterior cervical vertebræ. If in the fore limbs, it points to the posterior bones of the neck. The implication of the diaphragm would incriminate the fourth and fifth cervical vertebræ. If in the crural muscles or those of the quarter and thigh, the lumbar vertebræ must be looked to. The implication of the nerves of the back, while impairing the functions of the intercostal and abdominal muscles, produce less marked symptoms than when the limbs are involved.

When the disease extends deeper so as to implicate the meninges and especially the cord, there is evidence of impairment or interruption of conduction in the cord in addition to the simple involving of the nerves that emerge at that point. Thus serious disease or pressure on the cervical part of the cord in front of the fourth vertebra will make respiration difficult or impossible and speedy asphyxia may ensue. The paralysis of all parts behind the lesion is overlooked, in view of the fatal nature of the paralysis of the intercostals and diaphragm. If the interruption of conduction is incomplete there may be spastic paralysis and hyperæsthesia in the limbs and trunk back of the lesion.

If the dorsal cord is involved so as to render conduction imperfect there will be at first imperfect control of parts posterior to

the lesion, and when still further implicated, flaccid or spastic paralysis, especially of the hind limbs and tail. When it implicates the lumbar region in addition to the paralysis named for the dorsal, there will be incontinence of urine and even relaxation of the anal sphincter. In a case of acute tuberculosis in a cow, supervening on an ancient tuberculous lesion, as seen by the author, there were imperfect control of the hind limbs and uncertain gait, with tenderness of the dorsal region as if the animal had sprain of the back.

Diagnosis of these cases of vertebral disease may not always be possible in the early stages, yet the symptoms of progressive tenderness and stiffness in the region of the spine, the distortion in some instances of the spinal column, the subsequent appearance of localized motor and sensory symptoms, and later still the spasms or spastic paralysis in all parts behind the seat of the lesion, will be strongly suggestive of such a disease. When indications exist of osteoporosis, rachitis, or tuberculosis, of caries, abscess, or infectious lesions of the cervical or dorsal spinal region, the inference is still stronger. Then if reaction occurs under the tuberculin test, or if the urine contains an excess of phosphates in the herbivora, the case may be diagnosed with certainty.

Treatment. This will rarely be admissible on account of the expense and uncertainty of result. Some meat animals may be killed for food. If otherwise, keep in narrow stall where the animal cannot turn even the head, feed from moderate level to avoid movement of the spine by the upward and downward movements of the head. Gentle brushing is useful as a means of cleanliness, and of toning up the muscular system. Nourishing food of an easily digestible kind is essential, and pure air and sunshine are important auxiliaries. A course of cod liver oil with bitters may be given to improve the general health, calcium sulphide may be tried in case of suppurative caries, and calcium phosphate will usually be desirable to improve the nutrition and consistency of the osseus system. In case of a valued patient which it is desirable to preserve for reasons of sentiment or affection, or for breeding purposes, one might be warranted in continuing a long and expensive course of treatment, but in the regular run of cases considerations of humanity and economy would counsel the prompt destruction of the animal.

SLOW COMPRESSION OF SPINAL CORD. PARALYSIS.

Causes: Caries, vertebral diseases and lesions, neoplasms, actinomycosis, tubercle, abscess, organizing exudates, parasites. Melanoma, cholesteatoma, sarcoma, papilloma, lipoma, glioma, chrondroma. Symptoms: advance insensibly, or by sudden leap with exudate, spasm, paresis, transverse, senses clear, muscular atrophy, advance from behind forward. Cervical, dorsal, lumbar lesions. Bladder, sphincters, tail. Symptoms increased by movement. Treatment: according to lesion. Tumors, hopeless. Blood clots, Actinomycosis. Analgesics. Electricity.

Causes. Slowly progressive compression of the cord has been already noted as resulting from caries and other diseases of the vertebræ. It remains to notice such as result from the growth of tumors and other neoplasms in the spinal canal. In the horse these are commonly melanoma (in white horses), sarcoma, encephaloid, papilloma, cholesteatoma, and osteoma; in cattle, beside tubercle and actinomycosis, have been found sarcoma, lipoma, osteoma and glioma; and in dogs sarcoma and chondro-sarcoma. Chronic abscesses may be met with in all animals determining the same class of symptoms by slow pressure. In the same manner exudates in process of organization contract, and are liable to compress the myelon. Cadeac draws attention to a calcic degeneration of exudates in the dura mater of the dog (ossifying pachymeningitis), and of ossification of the intervertebræ cartilages with vegetations on their surfaces. Parasites also exercise a growing pressure, especially echinococcus, in cattle cysticercus mediocanellata, in sheep and dogs coenurus, and in pigs and dogs cysticercus cellulosa.

Melanoma. In gray and white horses, with disseminated melanosis, the spinal canal is often involved, the pigmentary sarcoma appearing in small formations and sometimes large enough to determine injurious pressure. In the early stages these may cause stiffness and lameness referable to particular muscles or groups, varying in situation, even as to the limbs affected, at successive dates, and finally merging into paraplegia.

Cholesteatoma is less common than in the encephalon, yet one is reported by Dexler as attached to the pia mater and possessed of great firmness, crisply crackling under the knife. It

doubtless secures accretions under meningeal exudates like those of the choroid plexus.

Sarcoma and **encephaloid** are usually found in connection with the dura mater, and of small size, but numerous. They often surround the roots of the spinal nerves, and here as on the cord exert sufficient pressure to impair nervous function.

Papilloma has been found connected with the pia mater and of marked vascularity. The author has found one in the ewe in a case of the neurasthenia of advanced gestation.

Lipoma is also rare. Osseous growths are common, being favored by sprains and injuries. All show a marked predilection for the lumbar and last dorsal vertebræ. This may be partly explained by the liability to injury and to disease invasion through the interlocking of the joint surfaces of the rings. *Osteophytes* growing from the intervertebral cartilage are common in the dorsal region as well.

Glioma has been found in cattle, occupying the substance of the cord itself and growing to the size of a hen's egg or even of the closed fist. The cord is gradually atrophied and paraplegia is inevitable.

Chrondro-sarcoma has been found growing from the intervertebral fibro cartilage of dogs.

The *Symptoms* may be deferred for a length of time on account of the accommodation of the myelon to the slowly increasing pressure. When they do become manifest, it is usually at first by insensible gradations so that for a time their existence is questionable. Yet a case will sometimes reach a sudden climax, by reason of a blood extravasation or inflammatory exudate, and the signs of sudden pressure or acute myelitis or meningitis supervene. In the absence of sudden access of trouble, the symptoms are those of a slow increase of *motor troubles* (local paresis, paralysis, paraplegia), or *sensory* (hyperæsthesia, anæsthesia). Spasms may occur early or even later in the disease. From disorders due to cerebral lesions the morbid phenomena are distinguished by being paraplegic rather than hemiplegic; sensori-motor rather than sensory or motor; local rather than general; with intelligence and special senses clear, rather than dull or abolished; associated with marked muscular atrophy in the affected parts; advancing from behind forward rather than uniform throughout the body.

The area of nervous disorder points more or less clearly to the seat of the lesion. Early implication of the fore limbs, and then later of the hind, suggests lesion of the cervical region. Dyspnœa tumultuous heart action, or vertigo may coincide. Tardy movements of the hind limbs, imperfect balancing, dragging, swaying, knuckling, involuntary flexions of stifle or hock, flexor contractions, standing on toe, cramps, paraplegia, indicate lesion in the dorsal or lumbar region. There may be palsy of the rectum, anus, bladder, sphincter vesicæ, penis, and vulva. Paralysis or other nervous disorder of the tail and sphincters ani and vesicæ, without implication of the hind limbs or quarters, may bespeak lesion in the terminal end of the spinal cord.

With paralysis of the bladder the penis may be pendent out of the sheath, or being retained within it, the urine may dribble constantly into and from that cavity, and the vulva may be soft and flaccid. When the anus is involved, the adjacent part of the rectum usually participates becoming overloaded, the sphincter is soft and lax and allows a constant oozing, and the exposure of the mucosa. The paralytic tail hangs between the thighs, limp and flaccid, and becomes saturated with manure and in females with urine.

Even in the earlier stages the symptoms are usually greatly aggravated by compulsory movements like turning in a circle, walking up hill, or (in dogs) up a stair, the arched back, the pendent head, and hesitating planting of the foot suggests walking on pins. For a more exact localization of the lesion the reader may consult the table indicating the functions of the different parts of the spinal cord. The early fatigue under exercise grows as in other progressive spinal lesions.

Treatment. In most cases this is hopeless. Tumors, bony and calcic growths, tubercles, degenerations and absorption of nervous tissue are practically beyond remedy. A blood extravasation may be largely absorbed, leaving only the permanent changes in the nervous tissue. In this time is the main element. Actinomycosis may sometimes be successfully met by a course of potassium iodide, when, if the nervous lesions are slight, a fair recovery may be secured. In the majority of cases, however, the practitioner is limited to measures for palliation of suffering by atropia, chloral, phenacetin, etc., or by nerve stimulants like

nux in small doses, or by weak currents of electricity. In meat producing animals, it is often the best course to fatten rapidly, or to turn over at once to the butcher.

DILATATION OF THE CENTRAL CANAL OF THE SPINAL CORD. SYRINGOMYELIA.

This means literally a cavity in the spinal cord but is applied to cavities formed by dilatation of the central spinal canal, or by an excavation in the nervous tissue immediately adjacent and usually communicating with a dilated segment of the canal. In man it is usually the result of an active proliferation of the epithelial cells of the canal, blocking the same, or extending into the adjacent nervous tissue in the form of a glioma. In different cases in dogs it occurred as the result of pressure. It has been seen in dogs, cats and Guinea pigs, as a casual lesion and as the result of experiment.

In a case reported by Lienaux it extended for practically the whole length of the cord, varying in form and size at different points. In the lumbar portion it was only slightly dilated, in the dorsal it was very irregular with prolongations into the gray matter, toward the cervical enlargement, its transverse section resembled an inverted V, and in the anterior cervical part it was unevenly rounded. Notable changes were cell proliferation and subsequent degeneration with the formation of cavities, thickening of the neuroglia, and compression and even obliteration of the vessels with circumscribed areas of necrosis, terminating also in cavity formation.

Symptoms. These vary with the nervous structures invaded, atrophied or destroyed. Invasion of the anterior horns of gray matter, causes trembling and muscular wasting. The implication of the superior horns determines more or less marked anæsthesia. Hyperæsthesia, spasms, paresis and paraplegia are also seen but no symptom nor group of symptoms is diagnostic of the exact lesion.

Treatment is manifestly hopeless.

NEURASTHENIA IN PREGNANT EWES.

Causes: inactivity, lowered muscular and nervous tone, twin pregnancy, approach of parturition, dry (clover hay) ration, concurrent diseases. Symptoms: moping, anorexia, depression, stupor, blindness, paresis, lethargy. Prevention: open air life, exercise, high muscular condition, avoidance of debilitating and relaxing conditions. Treatment: hygienic, nerve tonics, attend to concurrent diseases.

Neurasthenia has been defined as an incompetency of the nervous system, leading to early fatigue, and inability to recuperate from the prostrate condition. Pending a better knowledge of the affection, I have given this name to an asthenic affection seen in pregnant ewes when nearing the completion of the period of gestation.

Causes. In a large number of cases I have found several conditions so constant, not to say invariable, that they seem to deserve special attention in the list of causes:

1st. **Enforced inactivity.** In every instance that has come under my notice the ewes have been confined for several months to a barn or at most a confined yard so that exercise became impossible. The muscular system was flabby and soft, although as a rule there was abundance of fat, and the number of red globules did not vary much from the normal. In an animal that is naturally so active, and so accustomed to outdoor life the reduction of tone and vigor is to be expected. The same evil shows in other directions, thus after a snowy winter and close confinement the crop of lambs will sometimes perish of goitre without exception, while in subsequent years, with enforced exercise of the ewes, practically all escape.

2d. **Twin lambs** have been found in the womb of almost every case examined. The extra drain upon the system, and the depressing action of the load on an atonic ewe together with the symptomatic irritation are to be noted.

3d. The near approach of the **completion of gestation**, the cumulative effect of a long pregnancy, and perhaps the absorption of metabolic products from the fœtal membranes in course of preparation for detachment, and of leucomaines from the physiologically

active or developing mammæ, doubtless have a prostrating influence on the susceptible nervous system.

4th. All had been fed on **clover hay** either as an exclusive diet or as part of their ration. This is sufficiently nutritive, as testified by the fat condition of the patients, but it may be that it was too nitrogenous for such an inactive life. Again the clover hay usually abounds in cryptogams and bacteria and their products, which may have contributed somewhat to the asthenia.

5th. **Concurrent diseases**, in individual cases or flocks, manifestly contributed to the general loss of nervous power. In some the bowels were studded with the nodules of the œsophagostoma, in others œstrus larvæ had extensively invaded the nasal sinuses, one had congestion of the mucosa of the small intestines, some had congestion and fatty degeneration of the liver, others had fatty kidneys, and one had a papilloma pressing on the spinal cord. Manifestly diseases and degenerations of various kinds would still further undermine nervous energy and add to the atony.

Cold and heat did not seem to dominate, as most were kept in warm barns, and wore heavy winter fleeces, while one clipped early in December, and kept in an atmosphere of 40° to 55°, was attacked in the second week of January.

As this experience was had in a goitre district it may become a question whether the poison of this disease was a causative factor. Goitre was not a prominent feature in either ewes or lambs.

Symptoms. Variable. The most prominent are, leaving the flock, moping alone, grinding the teeth, drooping and trembling of head and ears, temperature normal or subnormal (100.5°), respiration 24, pulse 80, feeds and ruminates sparingly, bowels normal, buccal mucosa pale, conjunctiva hyperæmic, in some cases stupor and partial blindness, the animal walking against racks or fences, walk is slow and unsteady, the muscles feel soft and flabby, the abdomen may be full, but its walls are quite flaccid so that the lambs can be easily felt. As the disease advances all symptoms are aggravated, food is no longer taken, rumination ceases, the ewe remains recumbent, cannot be made to rise, and when lifted and carried makes no struggle. After 24 hours of this helpless condition death supervenes. In some

instances labor pains have come on and the ewe has perished in a vain effort at delivery. Illness lasted about a week.

Prominent *lesions* have been noted under causes. It need only be added that no notable difference from the normal was found in making a count of the red globules, and the size of the individual globule was normal or only perceptibly smaller as is to be expected in a dense plasma. In different cases there was found congestion of the abomasum, small intestine, liver and brain.

Prevention. The most important measure is to maintain a strong, well-developed muscular system, and a vigorous nervous tone by a sufficiency of out door exercise during the winter months. Half a mile or a mile at least should be given daily to the breeding ewes, no matter what the attendant difficulties. If clover hay is musty it should be replaced in whole or in part by another kind. Parasites and other diseases which tend to lower the general tone should be appropriately treated.

Treatment. When once established, the disease has not been successfully treated. Nerve tonics are indicated.

NEURITIS. PERINEURITIS.

Definition. Causes: traumas, poke, stanchions, collar, yoke, interfering, neurectomy, fractures, tumors, callus, rheumatism, gout, violent over-distension. Lesions: nerve sheath red, swollen, exudate, leucocytes in excess, fibroid thickening, nerve atrophy, degeneration, axis granular, myelin in oily globules, peripheral extension. Muscular degeneration and atrophy. Symptoms: tenderness, swelling, muscular atony, wasting, spasms, twitching, decreased excitability, paralysis, in section swelling on proximal end. Prognosis: disability for weeks, months or year; response to electric current, operability of tumors, curability of rheumatism or gout, hopeful conditions; long standing degeneration, etc., unpromising. Treatment: rest, soothe, anodynes, splint with soft pad, essential oils, lead and opium lotion, ice, snow; derivatives; laxatives; diuretics, anti-rheumatics, Faradisation.

Definition. Inflammation of a nerve leading to paralysis of the parts to which it is distributed.

Causes. Traumatism is the most common factor. Among the common examples are injury of the seventh nerve above the

angle of the lower jaw, by a poke worn in pasture by the horse, or by stanchions in the cow. Hogs may suffer from blows of the triangular neck gear worn to prevent them from breaking through fences. Blows by the yoke, incised and contused wounds implicating the nerve, such as neurectomy, and the blows received in interfering, and compression by tumors or bony growths, are familiar examples. Fractures with displacement, notably those of the sacrum and proximal end of the coccyx with caudal paralysis, are not uncommon. In fractures of the limbs the pressure upon or wounding of a nerve. Again, the callus on the seat of fracture may induce neuritis by pressure, as may also the projection of the end of a bone in luxation. Rheumatism affecting the nerve sheaths and, in birds and swine, gout, are additional factors. Violent overdistension, and even chronic muscular spasm, are quoted as causes.

Lesions. The early changes are mainly in the connective tissue sheath, which becomes hyperæmic, red and swollen, with a gelatinoid exudate and a great multiplication of leucocytes. Later, the interfibrillar connective tissue is involved and the nervous substance proper undergoes hyperæmia and degeneration. The axis cylinder undergoes granular degeneration and the myelin breaks up into oil-like globules. The lesions are at first limited in extent, though there may be more than one focus, and the resulting degeneration of the nervous filaments advances toward the periphery in accordance with Waller's law by which disease changes proceed rapidly in parts cut off from their trophic cells.

The muscles supplied by the inflamed nerves also rapidly degenerate. The fibres shrink in size, and lose their striated appearance, becoming distinctly granular, and pale. Round cells are formed in excess in the sarcolemma and muscular fibre, and if the morbid condition persists there is fibroid degeneration, cirrhosis and contraction.

Symptoms. In the absence of the subjective element of pain, which is the most constant symptom in man, we must rely mainly on the exquisite tenderness on pressure along the line of the nerve, but localized at some particular point, on the swelling at such tender point and on the loss of muscular power or even of sensation in the tissues corresponding to its peripheral distribution. The muscles may be hypersensitive and are usually flaccid

if not from actual paralysis, still from the pain which attends on their contraction. In some cases they are the seat of clonic spasms or twitching. Under a current of electricity they show a decreased irritability which bears a direct relation to the grade of degeneration which has occurred in the nerve fibres. In cases of deep-seated neuritis paralysis may be the only appreciable symptom. In traumatic injuries like bruises of the seventh nerve or fracture of the sacrum the local swelling and tenderness are marked initial symptoms, upon which supervene the paralysis and atrophy of the muscles cut off from full innervation. In neurectomy the tender swelling in the stump which is still in connection with the nerve centre may amount to a distinct neuroma, while the peripheral and detached portion of the nerve steadily loses its irritability as shown by electric stimulus.

Prognosis. This will depend on the nature of the lesion. A single transverse section of a nerve, without loss of substance may be repaired in a few months, while with loss or degeneration of a considerable part of its substance it may maintain a paralysis for years or even permanently. Lesions due to slight bruises may recover in a few weeks, while the more severe ones will persist for months or years. The response to electric stimulus distal of the lesion, is a guarantee of the absence of degeneration and a feature hopeful of recovery. Pressure by bony displacement or neoplasia must be done away with as the first condition of improvement in such cases. Rheumatic and gouty cases will persist until these constitutional infirmities are corrected.

If the neuritis and paralysis have lasted for any length of time, the degeneration of the muscles will keep up a degree of muscular weakness (and if in the limbs lameness) after the repair of the nerve has been completed.

Treatment. Rest is the first consideration accompanied by soothing and anodyne application to the inflamed nerve. When neuritis exists in a limb a softly padded splint may be useful at first. The skin over the inflamed nerve may be rubbed by one or a combination of the anodyne essential oils, (oil of cajeput, oil of peppermint, oil of lavender). If the pain and tenderness are extreme, a bag of ice or snow may give relief and should be kept applied for a length of time. Or hot fomentations with a lotion of lead and opium may be preferred especially in rheumatic cases.

If blisters seem to be called for, aqua ammonia and oil of turpentine may be added to the essential oils, or muriatic acid may be applied with a glass rod in points along the line of nerve. A laxative of Epsom or Glauber salts will often prove of great value at the outset and may be followed by diuretic doses of potassium iodide, potassium nitrate or acetate, and in rheumatic cases sodium salicylate. In these last forms, as also in gout, the carbonates and acetates of the alkalies, colchicum, and salicylates are especially to be persisted with. In these, too, rubefacients and blisters are often of essential value and may be repeated again and again.

Faradism is of little account during the active stage of neuritis excepting as a test of the progress and extent of the degeneration, but when inflammation has subsided nothing contributes more to the restoration of the tone and healthy nutrition of both nerve and muscle. The current is to be sent along the line of the paretic nerve and muscles for ten or fifteen minutes at a time and not less than once a day.

NEURALGIA.

Intermittent or remittent pains, in line of nerve without inflammation, or other structural lesion. Diagnosis: lameness, stiffness of particular muscles having a common nerve. Unnatural position habitual. Pain of inflammation and of neuralgia. No functional change. Rheumatism. Tumors. Causes: lead, rheumatism, gout, auto-poisons, cold, anæmia, reflex. Facial neuralgia, occipito-cervical, dorso-intercostal, lumbo-abdominal, sciatic. Treatment: elimination, of lead, etc.; intestinal antiseptics, tonics, hot water, anodynes, arsenic.

This is characterized by pain paroxysmal, intermittent or remittent situated in the course of given nerves. It must be a pure neurosis and unaccompanied by any specific structural lesion like inflammation, degeneration, atrophy, hypertrophy, tumor or the like. It is therefore manifested subjectively and cannot be easily identified in the lower animals. Nevertheless, Lafosse, Zundel, Genée, and others have recorded cases, their conclusion being deduced from symptoms which were held to indicate nervous suffer-

ing in the absence of any structural lesion whatever. *A priori* one can with difficulty escape the conviction that neuralgia must exist in the lower animals as in man, and the only drawback to its recognition is the difficulty of diagnosis.

The first step in such diagnosis must usually be the presence of lameness, stiffness or indisposition to free movement of some particular muscle or group of muscles deriving their innervation from a particular nerve. Or there may be a particular position habitually assumed such as semi-closed eyelids, drawn back ears, laterally inclined neck which strongly suggests nervous suffering. Next, there must be the exclusion of any appreciable structural cause and especially of inflammation. The three prominent features of the pain of inflammation is that it is aggravated by pressure, it is heightened by movement, and it is accompanied by some decided alteration of the function of the part. If there are at the same time exudation and swelling, inflammation is all the more certainly indicated. In a neuralgic pain on the contrary pressure does not increase the pain: it may even alleviate it: movement of the part may be rather satisfactory to the patient than painful; and the disturbance of function, contractile, secretory, trophic, is not perceptible. There is no local exudation nor swelling to account for the nervous disorder.

The liability to confound the affection with a neuritis more centrally situated, but the pain of which is referred to the periphery of the nerve, is to be obviated by a tracing of the nerve along its course to the nerve centre so as to identify any centre of tenderness, and also by the implication of all the peripheral branches coming off ectal of that point.

Again, rheumatism may be easily confounded with neuralgia, but here the affected nerve and muscle and even the skin over it is liable to be very tender to the touch or pinch, and if at all acute some hyperthermia is present. Like rheumatism, neuralgia shows a tendency to shift from place to place.

Pains due to pressure on the nerves by tumors, aneurisms, and other swellings, are constant, whereas neuralgic pains are marked by remissions and aggravations and even by intervals of complete relief.

Causes. The toxic neuralgias are illustrated by chronic lead poisoning, in which, in man, there are wandering pains like those

of rheumatism, and in the lower animals muscular stiffness and contractions which suggest a similar condition. In man, too, gout is a common factor, and in pigs and birds in which this condition exists, stiffness and evidence of suffering may well be at times attributed to a similar cause. How many other forms of chronic metallic poisoning and poisoning by morbid autochthonous products of indigestion are attended by disorders of innervation and nutrition, it is as yet impossible to say. The direct action of cold, an anæmic condition of the nerves, and reflex action from distant sources of irritation are among the other invoked causes. Inflammation in the *nervi nervorum* is also invoked as a factor, but in this case the symptoms would not accord with the rule given above, since the nerve trunks would be very tender to touch or pressure, and the suffering would be unshifting and shown permanently in the one seat.

Facial Neuralgia. Lafosse and Zundel describe as cases of this kind those in which periodically the horse's eyes are fixed and shining, the ears drawn back and depressed as in vice, the head at intervals bent on the neck, with plaintive neighing, rubbing the head on the stall and pawing. Those cases of twitching of the head or rapid jerking of the ears in horses, when they have been driven for some distance, and which are relieved by wearing a close net over the nostril or by section of the trifacial nerve at the infra-orbital foramen, manifestly partake of this character.

Cervico-Occipital Neuralgia. Lafosse speaks of this as often mistaken for torticolis, the head being turned to the affected side during the paroxysms. In man this is often a result of cold draughts on the back of the head, and associated with tender points on the course of the nerve, between the mastoid and the median line.

Dorso-intercostal neuralgia causes pain in deep inspiration, and **lumbo-abdominal neuralgia** develops tenderness in the loins, in one testicle, or in one lip of the vulva according to Lafosse. Diagnosis between such cases and neuritis, spinal disease, and other obscure nervous affections must be very problematical.

Sciatic Neuralgia. This is described by Zundel as causing jerking and lameness in the affected limb, sometimes aggravated

and sometimes improved by work and associated with muscular weakness or paresis. Sciatica in man is, however, rarely a simple neuralgia, but partakes rather of the nature of a neuritis, and there is no good reason for supposing that the disease of this nerve in the lower animals is other than an inflammatory condition.

Leclainche after consideration of the testimony adduced, is of the opinion that we still lack absolute evidence of uncomplicated neuralgia in the domestic animals.

Treatment. For **toxic** cases elimination of the poison is the first consideration. For *lead* carefully graduated doses of iodide of potassium to carry off the offending agent without increasing its poisonous action must be continued as long as the metal is passed by the urine. It may be followed by a course of strychnia, by electricity, massage and blisters. Gouty subjects may be treated with salicylate of soda, alkalies, or colchicum. The victims of Bright's disease must be treated for the kidney affection.

Where there has been **trouble of the digestive organs**, intestinal antiseptics (salol, sodium salicylate, bismuth-salicylate, beta napthol) and small doses of arsenious acid will sometimes benefit.

In **anæmic conditions** a course of tonics (cod liver oil, iron, quinine, nux vomica) are indicated, and, to improve the local blood supply, nitro-glycerine. A rich stimulating ration, currying, an open air life, and sunshine (in summer a run at grass) are called for.

In man with a suspicion of **traumatic origin**, W. H. Thomson strongly advocates a persevering use of the hot water douche to the parts first affected, the hypodermic use of morphia and atropia, and in case of local anæmia nitroglycerine every three hours. Where there is a suspicion of inflammation he successfully employs absolute rest, with opium narcotism so as to abolish the pain, for twenty days if necessary. Aconite, antipyrin, acetanilid, phenacetin, exaglin, and gelsemium have their advocates, and may benefit in individual cases. A course of arsenic is often successful, and phosphorus and ergot have each proved of value.

ATROPHY OF NERVES.

From arrest of function, from lesions, pressure, distal, but at times central of lesion. Symptoms: Loss of function advancing to paralysis. Muscle atrophy. Prognosis: in absence of incurable cause, is hopeful. Union of divided ends, restoration of function. Treatment: time, ligature of divided ends.

This is usually the result of arrest of function. It may be due to transverse section of the nerve, as in surgical neurectomy when the separated peripheral end of the nerve gradually wastes. It may come from contused wounds implicating the nerve and causing destruction of its substance. It may be from tumors or other neoplasms pressing on the trunk of the nerve and preventing the passage of nerve currents. Or, inflammatory effusion may press on the nerve, as happens often to the crural in hæmoglobinuria. Or the pressure may come from enlarged mediastinal glands, or even from the distended posterior aorta under habitual violent exertion so as to permanently incapacitate and atrophy the left recurrent laryngeal nerve as in chronic laryngeal paralysis(roaring). Similar wasting occurs in other nerves under corresponding conditions. Atrophy may, however, extend centrally from the peripheral end of a nerve when it can no longer remain functionally active. We find an example of this in the atrophy of the optic nerve up to the commissure when the eyeball has been excised. A similar condition is often seen in horses in which the integrity of the eye has been completely destroyed in connection with recurring ophthalmia.

The *symptoms* attendant on atrophy of a nerve are those of impaired function gradually advancing to complete paralysis of motion or sensation. In cases of a complete breach of continuity as in section or severe traumatism the entire loss of function necessarily precedes the atrophy. Again, when it comes from destructive changes in the coats and media of the eye, and of the ganglionic cells of the retina, the atrophy of the nerve trunk proceeds simultaneously with the lesions of the organ of vision.

The *diagnosis* will in many cases be easy as deduced from the traumatic or surgical lesion. In other cases it may be made with

certainty from the complete muscular paralysis, wasting and degeneration of the muscles supplied by the nerve, and by the history of the case (hæmoglobinuria in atrophy of the triceps extensor cruris, roaring in atrophy of the laryngeal muscles and recurrent nerve). In other cases, as in the eye, we have the atrophy of the eyeball, the distortion or complete paralysis of the iris, the opacity of the lens, or the exudation into the vitreous, choroid and retina when these can still be observed.

Prognosis will depend on the cause. With a nerve severed with a knife or crushed in a part of its course and atrophied, without destructive changes in the organs in which it is distributed, repair is possible and to be expected in time.

Treatment is expectant, yet inflammation must be subdued, tumors removed, divided ends ligatured, etc.

DISEASES OF THE URINARY ORGANS.

Relative prevalence in man and animals. Causes of difference. Kidneys as eliminating organs for nitrogenous material, toxins, bacteria, mineral, vegetable and animal poisons, diuretic drinking water, condition powders, cantharides, urea, etc. Suppression of urine, precipitation of urine. Filtration through kidney. Secretion. Urinary solids. Nervous control of secretion. Excess.

Diseases of the urinary organs are less prevalent in the lower animals than in man, owing largely no doubt to the greater simplicity of their habits of life and to the comparative shortness of the lives of those that are kept for meat producing. It is a mistake, however, to suppose that they are so infrequent as would appear, since the absence of subjective symptoms in the animal allows a number of the milder forms of renal disease to be passed over without recognition.

In man the excessive consumption of animal food, the lack of exercise, the abuse of alcohol, the prevalence of venereal diseases, conduce largely to renal troubles, while animals in general escape. Yet animals suffer much more extensively than is generally supposed. The kidneys are, as in man, the eliminating organs for superfluous and waste nitrogenous matter, and in overfed animals may be overcharged with this work. They are the general emunctories for the soluble poisonous products of bacteria and plants, which may stimulate the urinary secretion, and from these irritation may result. It is through the kidneys that the bacteria themselves largely leave the animal body, and trouble is liable to come during their passage. Further, exposure to cold tends to **increase the urinary secretion,** over-stimulating the kidneys, and the same may come from diuretic drinking waters and condition powders, also from cantharides and other diuretic agents applied to the skin. Urea and many toxins are diuretic, hence the occurrence of polyuria at and after the crisis of fevers.

On the other hand **suppression of the urinary secretion** may occur in connection with profuse perspirations in hot weather, with prolonged diarrhœa, or with privation of water, and in such cases the liquid becomes concentrated and irritating and there is a disposition to precipitate its solids under slight disturbing causes. As conducive to such precipitation may be named foreign solid bodies, bacterial ferments and probably the goitre poison since gravel and calculus are common in goitrous regions.

There are two forms of elimination through the kidneys. 1, *filtration*; 2, *secretion*.

1 *Filtration* is referred to the glomeruli, and is determined by the relative blood pressure. Increase of pressure causes increase of watery transudation. Digitalis increases heart action and arterial pressure, and accidently urination. Excessive consumption of water and watery liquids increases intravascular tension, and the amount of urine.

2. *Secretion* is referred to the columnar epithelium of the convoluted tubes. It is by the elective affinity or selective power of this epithelium that the solids of the urine are abstracted from the blood and passed into the urine. Crystals of uric acid have been found in these cells and it is supposed that the abundance of water furnished by the glomeruli, irrigating these convoluted tubes, dissolves and washes on the various solids and other products with which the epithelial cells are charged. The protoplasm of the cells becomes saturated with the urea, uric acid, hippuric acid coloring matter (indican, urochrome, etc.), and this is washed out, passing by exosmosis to the liquid of lesser density with which the tubes are filled.

Nervous Control of Urinary Secretion.

An electric current through the renal plexus of the sympathetic (vaso-motor) lessens, or suppresses urinary secretion (inhibition).

Cutting the nerves of this plexus causes excessive vaso-dilation, renal pulsations synchronous with heart beats and arterial pulse, and great increase of urine. A similar increase comes from the application of cold to the surface, from fatigue, from heat exhaustion, from irritation of the floor of the fourth ventricle

just in front of the origin of the vagus and from section of the splanchnic nerve. This last is, however, much less marked and more transient than from section of the renal nerve noted above; the latter causing dilation of the renal vessels only, and increased pressure, whereas the former causes dilation of the abdominal organs generally, diverting the blood largely to other parts than the kidney and preventing the same increase of pressure in the vessels of the latter. For the same reason transverse section of the medulla oblongata, or of the spinal cord as far back as the seventh cervical vertebra, lessens or interrupts the urinary secretion, the pressure in the kidney being reduced by the diversion of much of the blood elsewhere. This influence of the nervous system on the urinary secretion seems to be mainly or entirely one of increase or decrease of blood pressure in the kidney. For this reason a weak heart tends to lessen urinary secretion.

Excessive increase of urine is only important when continuous and in the absence of visible cause, such as diuretics.

PHYSICAL PROPERTIES OF THE URINE.

Color, yellow, red, brown; horse, ox, calf, sheep, goat, dog, cat, bird. In disease: pale yellow, with water in excess; deep yellow, red, brown with solids in excess, urobiline, biliverdin, hæmoglobin. Extraneous colors. Bilharzia. Translucency: Turbidity: horse, ruminants, carnivora, pig. In disease, horse, other animals. Consistency, viscous, stringy, tarry; odor, horse, dog, cat, ammoniacal, fœtid, drug odor. Specific gravity, estimate of solids; reaction, acid, alkaline, neutral; morbid chemical changes, sodium chloride, phosphate, alkaline, earthy, indican, urea, uric acid, hippuric acid, phenol, creatinin, acetone, oxalic acid, allantoin, xanthin, hypoxanthin, cyanuric acid, leucin, albumen, glucose, bile salts and pigments, blood, hæmoglobin, epithelium, pus, casts.

Color. In estimating the color we must note the various shades of yellow, red and brown and compare these with the normal in different genera of animals, on different food and water, and in different conditions of health. Grades of color may be stated as follows:

Yellow: Pale, clear and deep yellows.

Red: Reddish yellow, yellowish red, and red.

Brown: Brownish red, reddish brown and brownish black.

Color of Normal Urine. This varies with the species of animal, food, quantity of water drunk, and time of retention in the bladder.

Horse: Urine is normally clear yellow, brownish yellow, or deep citron yellow, and the color is deepened by rich and abundant food (excess of solids) and by exposure to the air (changes in pigments). It may be sulphur white and sedimentary from precipitation of $CaCO_3$ when on green food.

Ox, Calf, Sheep and Goat: Normal urine clear yellow to wine yellow. In the ox especially it is a pale straw tint, but varies to a deep brown on nitrogenous food (clover, peas, beans, cotton seed, lentils, pea or bean straw). Color may be due to indican and sometimes to indicanin or indigo blue, which explains the blue urine sometimes described.

Dog: Normal urine is yellow, straw-colored, aniline yellow, honey yellow, to brownish yellow in hot season or on dry nitrogenous food. Is always relatively deeper than in ruminants.

Cat: Straw yellow to honey yellow, with variation as in the dog.

Pig: Very pale yellow, more highly colored on dry feeding, nuts, peas, etc.

Birds: White or yellow, sedimentary. Mixed with fæces in cloaca.

Color of Pathological Urine: **Pale yellow** with excessive secretion glycosuria, polyuria, cryptogamic polyuria, chronic interstitial nephritis, under diuretics, or after excessive drinking. The free secretion of a crisis in a fever is pale yellow.

Deep yellow, deep red, deep brown color, indicates excess of urinary pigment (urobiline) and is deepened by nitric acid. This is seen in all hyperthermias with suppressed or diminished secretion, in privation of water, or food. This urine is *acid* even in herbivora.

Yellow, saffron yellow, brownish yellow, greenish, olive, or brownish red indicate the presence of bile pigments (biliverdin, bilirubin) as in jaundice or cholyuria. Bile salts should be tested for. A similar coloration may come from free consumption of carrots, or other yellow pigmentary matters.

Red, brownish red, blood red, or deep brown color implies the presence of blood or blood coloring matter in the urine

(hæmaturia, hæmoglobinuria). Exposed to the air this becomes brown or chocolate in ratio with the amount of blood or blood pigment present. Some such cases are complicated by blood clots.

Color due to Foreign Constituents.

Bronze or black color may come from injection of **phenic acid.**

Deep green or olive green may come from **tar, carbolic acid, salol, creosote,** or **derivatives of benzine** taken in.

Brownish green comes from **thallin** and reddens with iron chloride.

Brown or blood red from **rhubarb** or **senna.**

Purple red from santonin, if alkaline (if acid, is reddish yellow).

Red from **madder** (it is alleged from indigo).

Yellow from **carrots.**

Blue (indigo blue) may occur in urine of horse or ox when exposed to the air.

Bluish green will come from feeding **indigo.**

White or yellow color will result from the presence of **pus.**

White, chylous urine occurs with a hæmatozöon (**Bilharzia Crassa**) in the blood of cattle.

Translucency. Urine may be passed clear and become turbid by standing. The presence of colloids hinders precipitation and prevents clearing.

Horse: Urine is generally turbid, especially what has been long in the bladder, and that which is last passed. The turbidity is largely due to precipitation of calcium carbonate and bicarbonate, and increases on green food, or if the liquid stands exposed to the air and is cooled. Not unfrequently the salts are thrown down as fine spherical granules, or there may be a white pultaceous mass. They are sometimes entangled in extremely mobile cylindroid masses coming from the uriniferous tubes during convalescence from fevers or during fasting. A fine pellicle on the surface is normal in horse's urine left in the air.

Ox, Sheep and Goat: Urine is passed clear. May become turbid through the change of lime carbonate into bicarbonate in cattle but always more slowly than in the horse.

Carnivora: Urine is passed clear but becomes turbid on decomposition, or if concentrated. With excess of fat in the food it

may become opaque from floating oil globules, apart from the classic chyluria.

Pig: Fed on raw fresh vegetables the urine is clear, but if on cooked or dried vegetables, and especially if nitrogenous, it may show opacity.

Pathological: The **horse's** urine is limpid and acid in polyuria; limpid and alkaline or neutral with modified phosphates. It may be morbidly turbid from excess of lime phosphate or sulphate, urea or other acid salts, exudates, leucocytes or pus. These usually indicate nephritis. Mucus and muco-purulent exudate suggest pyelitis or pelvic nephritis. Blood elements indicate nephritis, cystitis or urethritis. Debris of kidney tissue may indicate tuberculosis; tumors, etc.

Turbidity in **other animals** than solipeds is abnormal: examine the urine.

Consistency of Urine. Morbid urine may be gluey, sizy, syrupy, mucous, oily. If a **horse's** urine is scanty a **slight siziness** may be normal and due to tenacious mucus from the pelvis of the kidney, and from the solution of mucin and epithelium in the alkaline fluid. **Viscous, sizy, stringy,** and **tarry** (**pitchy**) urine is found in pyelitis, pyelo-nephritis, or cystic catarrh, but not in polyuria owing to the presence of the solvent acid.

Odor of Urine. This is somewhat **aromatic** in **horse** and **ox, disagreeable** in the **dog**, and **repulsively heavy** in the **cat.** With polyuria the odor is less. If the urine has been **retained** and **fermented** it is **ammoniacal,** if there are **ulcers** or **tumors** it is **fœtid,** in **diabetes** it smells of **acetone,** after taking **turpentine** it has a **violet odor,** and after phenic acid, camphor, ether and other drugs it is variously modified.

Specific Gravity of Urine in ratio to water 1000:

Horse,	1020	to	1050	(1040)
Ox,	1025	"	1045	(1030)
Sheep; Goat,	1015	"	1065	(1040)
Dog,	1020	"	1060	(1040)
Pig,	1005	"	1015	(1010)
Cat,	1020	"	1040	(1030)

In the horse the urine may be 1001 to 1010 in polyuria, in chronic interstitial nephritis, and in a crisis of fever attended by

diuresis. It may be 1050 to 1060 in glycosuria. Undissolved solids that are merely suspended in the urine do not affect its density.

A rough estimate of solids may be made by multiplying the last two figures of a specific gravity expressed in four figures by 2.33. The result approximates to the number of grammes of solids in 1000 cc.

Chemical Reaction of Urine. The liquid is tested by litmus paper, red and blue, weakly impregnated. The normal reaction is determined by the food: the urine of **carnivora** and **sucking herbivora** is **acid** turning blue litmus red: the urine of **vegetable feeders** is *alkaline* turning reds blue. In the **horse** the **alkalinity** is mainly due to excess of lime bicarbonate, passing, with standing, into lime carbonate, the carbon dioxide being derived from organic acids (lactic, malic, citric, etc.), by oxidation. The hippurates are also alkaline in reaction. In **dogs** the acidity is due to lime and soda phosphates, sulphates, urates and oxalates.

Pathologically we find the urine strongly **alkaline** from the evolution of ammonia from urea, in fermentations occurring with prolonged retention in the bladder or in cystitis. The urine is **acid** even in herbivora in all fevers in which appetite is lost or seriously impaired, and in which the metabolism is excessive.

Chemical Changes in the Urine in Disease. Sodium Chloride, is present in large amount in health (horse 25 to 35 grammes, dog 0.25 to 5 grammes daily) is **diminished** in fever, anæmia, visceral and exudative inflammations. It is **increased** during the absorption of false membranes and exudates. It is thrown down by adding solution of nitrate of silver, the curdy white precipitate being insoluble in nitric acid.

Phosphates of lime, soda, potash and, scantily, of magnesia are normally present (horse 0.08 to 0.60 gramme phosphoric acid daily) and are present in excess in digestive disorders and in malnutrition of bones (rachitis, osteoporosis and rheumatoid arthritis. The **alkaline phosphates** are very soluble and never precipitated. **Earthy phosphates** dissolve in acid urine, but are precipitated from alkaline. To a little of the urine add a few drops of acetic acid, followed by a few drops of uranium acetate. A yellow precipitate of uranium and ammonium double phosphate is thrown down.

Indican ($C_8H_7NSO_4$) is formed from indol which passes successively through the forms of indoxyle and indoxylid potassio-sulphate. This is normally present in the urine, the horse excreting 1 to 2 grammes daily, the dog 0.15 gramme. It is present in excess in intestinal indigestions, constituting indicanuria. It is tested by adding a drop of muriatic acid and one of a solution of chloride of lime to the urine, when it will show a blue ring, the depth of which indicates the relative amount.

Urea (CON_2H_4) the principal waste product of nitrogenous matter, is always present in considerable amount. The sound horse may eliminate 100 to 200 grammes daily, the dog 5 to 180 grammes. It is present in excess in all fevers and inflammations unless urination is suspended or impaired, in cryptogamic diuresis, in mellituria, uræmia, nephritis and cystitis. Test: The addition to a filtered solution of urine, freed from phosphates, of solution of acid nitrate of mercury, precipitates it as nitrate of urea. A simpler test is to add to a drop or two of urine on a glass slide a drop of nitric acid and heat gently. The nitrate of urea is precipitated in the characteristic rhombic or hexagonal crystals as seen under the microscope. Heat urea crystals in a test tube: biuret is formed and ammonia escapes. Add a trace of a copper sulphate solution and a few drops of a 20 per cent. solution of caustic potash: a rose-red color is produced—the biuret reaction.

Uric Acid ($C_5H_4N_4O_3$). Traces only of this are found in the normal herbivorous urine, yet it is more abundant when on a full dry grain diet, on milk (suckling) or on animal food. The dog kept on animal food has a large amount.

Pathologically it is produced in the dog and even in the horse in fever, overwork and starvation, the animal living on his own tissues. Interference with oxidation in the lungs seems to produce it as an arrest in the transformation of albuminoids to urea. The neutral urate of soda remains in solution: the acid urate of soda is precipitated. *Test:* To the urine add one-fourth its volume of muriatic acid and set aside for 24 hours in a cool place. On the bottom and sides of the glass and on the surface of the liquid will be found the yellowish red acicular crystals of uric acid.

Hippuric Acid ($C_9H_9NO_3$) is normally present in all urine, but is especially abundant in that of herbivora. The horse elim-

inates 60 to 160 grammes daily. It has been found to be increased by feeding on dandelion, carrots, clover, asparagus, apples, plums, benzoic acid, oil of bitter almonds, toluol, cinnamic or kinic acid. It is absent in sucking calves, and horses fed on grain devoid of husk. **Pathologically** it is increased in hyperthermia, icterus, some liver diseases and diseased kidneys. Test: Precipitate any albumen by nitric acid and boiling, then add hydrochloric acid which precipitates the hippuric acid in long needle-like crystals. Heated in a small glass tube it forms an oily liquid, and heated to redness gives off an odor of hydrocyanic acid (nitro-benzol) and carbon is left. This distinguishes alike from uric acid and benzoic.

Phenol is produced by intestinal fermentation. The horse normally excretes about 3 grammes daily. Pathologically it appears in excess in indigestions, abscesses, softened discharging tubercle, pyæmia, and septicæmia. Test: Dilute solutions of ferric salts give a blue coloration.

Creatinin, a product of metabolism of albuminoids, is found especially in the urine of carnivora and omnivora in health. It is pathologically increased when oxidation is interfered with, as in diseases of the lungs. Test: Add to the urine a very dilute solution of sodium nitro-prusside and then drop by drop some solution of caustic soda, when a ruby red color is shown and disappears again on boiling. Acetic acid changes to blue.

Acetone (C_3H_6O) is found in the urine of healthy omnivora and carnivora and increased by excess of nitrogenous food. Pathologically it has been found in fevers with much blood change, in inanition, in cancer, in indigestions, and auto-intoxications. *Test:* To several c.c. of urine add a few drops of iodo-potassic iodide solution and caustic potash when iodoform will be abundantly precipitated with its characteristic color and odor.

Oxalic Acid ($C_2H_2O_4$) appears to be secreted in small amount by healthy kidneys and it may also come from the splitting up of uric acid after secretion. It is augmented by feeding agents rich in oxalic acid (beets, fresh beans, asparagus, tomatoes). Pathologically it abounds in certain indigestions, and is associated with lameness and emaciation. Test: Add

lime water to the urine, and the white oxalate of lime is precipitated.

Allantoin ($C_4H_6N_4O_3$) is found in the urine of sucklings (calves) during the first few weeks of life, in pregnancy and when on a meat diet. It diminishes with the increase of vegetable food.

Xanthin ($C_5H_4N_4O_2$) is found in urine as a result of imperfect oxidation of nitrogenous matters especially, which would otherwise pass into uric or hippuric acid. Its immediate antecedents in such transformation are guanin and hypoxanthin or sarkin. It is a rare constituent of urinary calculus.

Hypoxanthin ($C_5H_4N_4O$) is produced from fibrine in gastric and pancreatic digestion and in putrefaction, and is especially abundant in leucæmic subjects.

Cyanuric Acid ($C_{20}H_{14}N_2O_6$) occurs in dog's urine.

Leucin ($C_6H_{13}NO_2$) and **Tyrosin** ($C_9H_{11}NO_3$) are products of pancreatic digestion of proteids, and the former occurs normally in the spleen, thymus, thyroid, liver, salivary glands, and urine. Both are present in large amount, in the urine, in acute atrophy of the liver. Test for leucin: Evaporate carefully to dryness with nitric acid: the residue, if leucin, will be almost transparent and turn yellow or brown on the addition of caustic soda. If now heated with the soda it forms an oily drop. Test for tyrosin: treated with strong sulphuric acid, gently warmed and chloride of iron added, it gives a violet color.

Albumen is an important morbid constituent of urine, which appears in a great variety of diseases (nephritis, pneumonia, epilepsy, anæmia, leucæmia, diabetes, hæmaturia, hæmoglobinuria, hydræmia, infectious lung diseases, cardiac obstruction, venous stasis in the kidney, dermatitis, burns, lesions of the crura cerebri, floor of the fourth ventricle, spinal cord, or renal vaso motor nerves). It also occurs after violent exertion, in poisoning by strong acid, phosphorus, arsenic, lead, mercury, opium or alcohol, and when an excess of albumen is injected into the blood. All forms of albumen may enter the urine, but the most common are serum albumen, globulin of serum, propeptone and peptone. A simple test is to acidulate the urine with acetic acid and boil: if the precipitate does not dissolve on addition of nitric acid, it is albumen. Sulphosalicylic acid added to the urine will cause a precipitate in urine containing only $\frac{1}{5000}$ of albumen.

Glucose ($C_6H_{12}O_6$) is often normally present for a short period in small amount after a full meal of farinaceous material. It is permanently present in excess in glycosuria, which may result, among other conditions, from diseased liver, punctured medulla, suppression of milk secretion on weaning the calf, oil of turpentine, nitrobenzole, nitrotoluol or amyle nitrate. Test: Add yeast to the urine and keep at 15° to 20° C. when if glucose is present, it becomes cloudy and gives off carbon dioxide, or add a little caustic potash solution, and a few drops of cupric sulphate solution until it is blue: then heat and a red precipitate of cupreous oxide is thrown down. The amount gives the ratio of glucose. Uric acid, hypoxanthin or mucus causes brown precipitate in the absence of glucose: peptone, creatin, creatinine, pepsine and urinary pigment prevent its formation though glucose be present.

Bile Salts and Pigments are present in excess in cases of icterus, where these characters may be studied. See Icterus.

Blood and Hæmoglobin in Urine. In a variety of diseases (anthrax, hæmaturia, nephritis, Texas fever, hæmoglobinuria, etc.) blood or blood coloring matter escapes in the urine. When blood escapes one finds the reddish color, and under the microscope red globules, normal or crenated (especially in alkaline urine), free, aggregated in masses, in small clots, or embedded in casts of the uriniferous tubes. Under the spectroscope the spectrum shows two dark absorption bands, one in the yellow and one in the green. When the color is due to hæmoglobin the urine shows under the microscope numerous masses of amorphous brown pigment, and the spectrum shows one dark line in the yellow, and three others less deep, (but one of them very broad) on the limit of the green and blue. Urine which contains the elements of blood is usually turbid and thick or glairy, by reason of the presence of salts, albumen and fibrine. There may also be crystals of urinary salts (calculi), fragments of broken down tissue (tumors) or the ova of worms.

Epithelium in Urine. The slight cloud seen in healthy urine contains epithelial cells. The source of these may be often determined under the microscope. The *bladder epithelium* are the most numerous, the largest, and are squamous. Those from the *ureters* and *renal pelvis* are also squamous, but neither so large nor so numerous. The *epithelium from the uriniferous*

tubules are polyhedral with large nucleus or columnar. The cells from the *male urethra* are also largely columnar. In cases, however, in which these cells are passed in large amount because of catarrh of the mucosa all alike tend to assume the globular form with large nucleus so that their true source cannot be certainly stated. It is only from such cells as have become detached without change of form that the seat of desquamation can be determined. If an excess of cells approximating to the kidney type are associated with albuminuria and cylindroid casts they become diagnostically significant. Polygonal cells darkly granular with large oval nucleus and nucleolus suggest kidney inflammation. If the granules are freely soluble in ether there is probably fatty degeneration. If hard, tough and glossy they suggest (but don't prove) amyloid degeneration.

Pus Cells in Urine. Pus cells, with mutiple nuclei revealed by adding dilute acetic acid, may be found in small numbers in apparently healthy urine. When present in large numbers, they usually indicate a catarrhal affection of the mucosa, and especially pyelitis, cystitis, or urethritis. There is always cloudiness, excess of mucin, and, in the alkaline herbivorous urine, the liquid may be glairy or stringy.

Casts of the Uriniferous Tubes. These usually indicate the existence of nephritis, yet they may be present in small numbers in the urine of healthy individuals under a slight toxic action such as alcohol.

Unorganized casts of urinary salts or hæmatoidin found in sucklings appear to have no pathological significance. *Organized* casts, on the other hand, usually imply renal troubles, and especially inflammation. As these will be fully described under Bright's disease, it need only be noted here that they may be composed in great part of *red globules*, *leucocytes*, *epithelium*, *bacteria*, *granules*, a *homogeneous wax-like matter*, *fat globules*, *hyaline matter*, or *urinary salts*. The predominance of one or other of these determines the nature of the cast.

The observations of Mayer, Knoll, Bovida, Von Jaksch and others seem to show that the basis substance of urinary casts differs from all our familiar proteids and must be considered as a distinct nitrogenous compound, a derivative of one of the common proteids.

GENERAL SYMPTOMS OF URINARY DISEASE.

External symptoms, arched back, stiff gait, straining, tender loins, backing, turning, dropping under weight, urine checked, dribbled; in dogs and cats, palpation of kidney; bladder, urethra, pains in different animals. Internal symptoms, rectal exploration, vaginal, urethral, straining, ureters, bladder, calculi, neoplasms, prostate, urethritis.

External Symptoms. With inflammatory or painful affections of the urinary organs the animal tends to roach the back or loins, tuck up the abdomen, move the hind limbs stiffly and with a straddling gait, protract and withdraw the penis which may be semi-erect, retract and drop the testicles alternately, and stretch himself and strain to pass urine without success. Lying down and rising may be accomplished with marked effort and groaning. The loins along the spines or beneath the outer ends of the transverse processes may prove tender to tapping or pinching, the animal drooping to excess. Backing or turning in a narrow circle may be accomplished awkwardly and stiffly though usually more easily than with lumbar sprain. The animal drops when mounted but less than with sprained back. Urine may be passed in excess or in diminished amount, or it may be entirely suppressed. It may be abruptly interrupted when in full stream, suggesting calculus or polypus, or it may be passed often in mere dribblets, or finally it may ooze away constantly partly lodging in the sheath and partly trickling down the thighs.

In dogs or cats with flaccid walls of the abdomen external manipulation may detect in the kidneys, differences in size, position, and tenderness as well as the presence of tumors. The distended bladder also may be distinctly felt, and the pyriform area of flatness on percussion will serve to map out its size and outline.

In the horse the urethra is superficial and easily traced over the ischiatic arch and for some distance downward, when it becomes deeper and is less easily felt. In the bull the urethra is deep over the ischiatic arch but becomes more superficial lower down and can be easily felt at the sigmoid flexure and below. In sheep and dog it is easily followed from the ischium to the end of the penis.

As a rule the penis is easily drawn from its sheath in the horse

and dog; this is more difficult in the sheep and goat and still more so in the bull and boar. In the small animal protrusion is favored by setting him on his rump, with his back between the operator's legs, and the pelvis doubled forward toward the sternum. The penis of the bull may be extended in presence of a cow in heat, and promptly seized, or it may be seized through the sheath back of its first bulging part and skillfully worked out. In the ruminant, calculi may be felt at the sigmoid curve, and in the ram, in the vermiform appendix at the fore end of the penis.

Internal Exploration. This is accomplished in the larger animals with the oiled hand in the rectum, the nails having been pared short and even to avoid injury to the mucosa. In ponies and yearlings the kidney may be felt, and this may be true also of mature animals of larger species in cases of hypertrophy or floating kidney. The ureters, bladder and intrapelvic urethra are easily felt in the male. The empty bladder lies on the anterior border of the pelvis; when full, it projects forward into the abdomen but retains its pyriform or, in the very young animal, its fusiform shape. In the female the sensation is somewhat modified by the presence on its upper surface of the uterus dividing into its two horns anteriorly. The single enlarged horn of pregnancy is especially misleading.

The female urethra, cervix and bladder may be explored through the vagina. To explore the cervix vesicæ and urethra the fingers are slowly drawn back from the bladder along the median line of the floor of the vagina. In the *mare* the cervix and adjacent portion of the bladder can be further explored with the index finger introduced through the opening of the urethra in the floor of the pelvis and at the junction of the vagina and vulva. In the cow the urethra is too small to be readily explored from within, and the orifice is still further guarded by the two lateral blind canals of Gærtner, into which the unskilled fingers more readily pass. Success only attends the careful search for the small central lower orifice. In the *smaller animals* the finger only can be introduced into vagina or rectum and the urethra, cervix and bladder only can be felt. *The result of such exploration* is straining even in healthy conditions but which becomes excessive in nephritis, pyelitis, renal, uretral, vesical or urethral calculus, cystitis, rectitis or enteritis.

The *ureters* are tender when inflamed, and they are swollen in calculous obstruction with an elastic feeling in front of the stone.

The *bladder* is very sensitive when overdistended, inflamed or pendent on the abdominal floor, or when the seat of calculus. In the absence of any liquid contents a calculus is felt as a hard solid mass firmly clasped by the contracted vesical walls. If liquid is present the solid hard calculus is felt movable in the fluid. An empty contracted bladder is firm and pyriform. An empty flaccid bladder, resulting from rupture or exhaustion, is flabby, with indefinite form and, if the seat of a lesion, tender. It varies in consistency with neoplasms (papilloma, sarcoma, carcinoma, or epithelioma). These have not the free mobility of the calculus floating in urine, and their point of connection with the wall may often be made out. When a solid body is felt, or suspected to be in the contracted bladder, an injection of sterilized water will usually facilitate diagnosis, and a differentiation of calculus and neoplasm.

Hypertrophy of the prostate is felt as a swelling of uneven outline over the cervix vesicæ. It is to be looked for especially in old dogs.

Urethritis is indicated by swelling and tenderness along the median line of the pelvic floor, back of the cervix. With a *calculus* in the urethra the swelling is more strictly localized and the canal in front of it may be full and elastic.

HÆMATURIA.

Symptoms of different lesions of kidneys and constitutional states, of poisoning by irritant plants, common on moors and in woods. In puerperal cow fed on turnips raised on mucky, unreclaimed, sour lands. Bacteria. Toxins. Anæmia. Poor wintering. Limed new soils. *Symptoms:* in plethoric, congested mucosæ, vascular tension, hurried breathing, colics, straining, red urine; in vegetable irritants, depression, weakness, coldness, trembling, stiffness behind, scanty red or black urine, diarrhœa, constipation; in anæmia, poverty, debility, red urine, pink tinge in milk, emaciation, hide-bound, anorexia, colics. Chronic or intermittent. Lesions: in plethoric, congested enlarged kidney, without softening; in irritant poisons, congestion also of throat, stomachs, intestines, liver with hæmorrhagic extravasations; in anæmia, kidneys pale, flaccid, hydrœmia, liver enlarged, softened, reddish

liquids in serous cavities. Treatment: avoid the injurious soils, drain, cultivate, feed products of such soils with other food, oleaginous or saline laxatives, antiferments, tonics, astringents, flax seed, farinas.

The passage of blood or blood elements in the urine.

Causes. A symptom of a variety of diseases, producing lesions of the secreting structures of the kidneys; acute congestion, tumors, calculi, parasitism. Also as a manifestation of diseases of distant organs—hæmoglobinuria, southern cattle fever, anthrax, poisoning by irritant diuretics, wounds of the bladder, pelvic fracture with injury to bladder or urethra, cystitis with varicose cystic veins, etc.

Among the irritant plants charged with producing the affection are the young shoots of oak, ash, privet, hornbean, alder, hazel, dogberry, pine, fir, and coniferæ, generally. Also ranunculus, hellebore, colchicum, mercuriales annuus, asclepias vincetoxicum, broom, etc. The disease is common in spring in cattle turned out too early to get good pasturage and which, it is alleged, take to eating the swelling buds and young shoots of irritant plants.

The disease has occurred mostly in woods and wild lands and has accordingly been vulgarly named the wood evil, (maladie de bois, holzkrankheit), and moor ill.

In England, as occurring in the puerperal cow, Cuming, of Ellon, attributes it to a too exclusive diet of turnips. His analysis showed that turnips contained 10 % sugar and 1 to 1½ % vegetable albumen. The sugar is held to stimulate unduly the milk secretion, but fails to supply the nitrogenous materials needful to form it, and the cow is speedily rendered anæmic, with solution of the blood globules or of the hæmatin and its excretion by the urine. No attempt was made to produce hæmaturia by an exclusive or excessive diet of sugar, and cows fed on turnips grown on well drained lands never suffered from the disease.

Williams says that urine in such cases had a strong odor of rotten turnips. This argues not an anæmia determined by sugar, but rather an intestinal fermentation, perhaps superinduced by ferments introduced along with the turnips. Add to this the notorious fact that the offending turnips are usually such as are grown on wild, damp, undrained, swampy, or mucky lands, and

we have the suggestion of a bacteridian poison, or a toxic product of bacteria. Williams and Reynal practically agree on the point that the common hæmaturia is the result of anæmia. It has long been noticed that the herds which suffer from the affection are those which have come out of the winter in low condition, the victim is the poor man's cow, and the symptoms are most likely to appear when turned into the fields in spring before the pastures have come up. The anæmic condition of the carcasses is quoted in support of this view, but perhaps without making sufficient account of the extraordinary destruction of blood globules during the progress of the malady.

Pichon and Sinoir see in the liming of soils and the production of larger crops, a cause of anæmia in the rank and aqueous growth of the meadows, and their overstocking in order to eat them down, or to consume their products. They found that an abundant artificial feeding was the most efficacious mode of treatment.

Reynal, who endorses this view, tells us that in the anæmic and liquid blood the globules become smaller and can pass more readily through the walls of the vessels. But this is exactly the opposite effect from what we see when the blood is diluted with water. The globules in such a case are distended and enlarged, and may finally have their protoplasm and hæmatin dissolved and diffused through the liquid. If the blood globules are shrunken, then we must look for a cause very different from anæmia.

Reynal further assures us that plethora is a common cause of hæmaturia in cattle. "Under the prolonged influence of a very assimilable diet, the blood becomes more plastic, circulates with difficulty in the capilaries, and may even rupture them, with a resulting capillary renal hæmorrhage, and bloody urine." He further intimates that this occurs especially in spring after the animals have been turned out on very rich pastures, and that in Normandy certain pastures of unusual richness are notorious for producing hæmaturia.

Apart from the fact that the rich grasses of spring produce at first intestinal congestion, and diarrhœa, with consequent disorder of the liver and kidneys, this spring affection on particular pastures suggests some special poison in the pasture as the unknown cause of the disease.

In all forms alike of this affection the nature of the soil appears to have a preponderating influence. It is the disease of the woods, and waste lands, of damp and undrained lands, of dense clays, of lands underlaid by clay or hard pan, of lands rich in vegetable humus, or vegetable moulds the decomposition of which has been hastened by the application of quicklime.

Pottier, Salomé, Wiener, and Reynal especially testify to the prevalence of hæmaturia on soils that are either dense and impermeable, or that have a subsoil of clay or hardpan.

The disease has not been traced to any definite microbe nor toxin, but there is much to suggest the necessity for inquiry in that line. The special susceptibility of animals that may be plethoric on the one hand, or in low condition on the other, would be entirely in keeping with such a view, as the debility or derangement of health would lay the system open to attack.

Symptoms. In the plethoric animal there are congested mucosæ, full, strong pulse, forcible heartbeats, full veins, accelerated breathing, colicy pains, dullness, straining frequently and the discharge of thick, red or bloody urine.

If from irritant buds and shoots, or plants, there is more depression, weakness, fever, dry skin, staring coat, coldness of the surface, tremblings, stiffness or weakness of the hind limbs, diarrhœa, followed by constipation, frequent straining and the passage of colored urine with pain. In violent cases the expulsion of bloody urine may be excessive, and the cow may die in 24 hours. From irritant plants however the quantity of urine is liable to be small, but frequently passed.

As occurring irrespective of plethora or irritants there may be at first only poor condition and debility with the passage of blood. A pink tinge may show on the froth in the milk pail, and a red precipitate on its bottom. If not anæmic at the outset they soon become so, and the pulse which was at first bounding becomes small and weak, the heart palpitates, the red mucosæ become pale. The subjects become tucked up, emaciated, weak, rough coated, the skin adherent to the bones, and the appetite and rumination impaired or lost. Sometimes colics are present.

In the milder anæmic forms it may continue for months before it causes death. In such cases it may prove intermittent.

Morbid Anatomy. In the hæmaturia of plethora the kidneys

are large, congested and of a dark red, but, preserve their normal consistency and texture.

In the form associated with ingestion of irritant plants, there is congestion of the pharynx, stomachs, and intestines with hæmorrhagic spots, congestion of the liver, violent congestion of the kidneys which are of a blackish red color, and enlarged to perhaps twice the normal size, with hæmorrhagic exudations, the convoluted tubes filled with fibrinous exudate and blood globules, the pelvis red and like the bladder containing some reddish urine. The vesical mucosa may be black.

In anæmic cases the kidneys are pale, flaccid and colorless, with a reddish liquid in the pelvis and bladder. The vascular system is comparatively empty, and the blood, thin, and watery, and often coagulates loosely or not at all. As noted by Herland globules are greatly reduced in numbers and size, and often crenated or partially broken down. Slight serous effusions in the serous membranes are common. The liver is softened and enlarged, the lacteals have reddish contents, and the ingesta are dark colored.

Treatment. Preventive. Avoid hæmaturia pastures and the fodder grown on such lands. Drain and cultivate such soils. When animals must feed on the products of such soils supplement the food by grain, oil cake, cotton seed meal, etc. Avoid stagnant waters draining from such soils.

Therapeutic Treatment. Give oleaginous or saline laxative to clear out poisons and ferments from the bowels and may add an antiferment (salol, salicylic acid, carbolic acid, turpentine oil, chlorate of potash, sulphites or hyposulphites), no matter if diarrhœa is present. Follow with tonics (copperas, chloride of iron) and stimulant antiseptics (ol. terebinth, potas. chlorate), and sound food. Flax seed, linseed meal, farinas. Bitters may be added (gentian, quinine, quassia). As a calmative, camphor (2 to 4 drs.) 2 or 3 times a day has proved useful.

In case of nephritis treat as for that affection.

Weiner lauds empyreumatic oil and oil of turpentine with camphor.

In chronic cases, nourishing food with change of locality and water are very important.

A course of iron tonics should wind up the treatment.

ACUTE CONGESTION OF THE KIDNEYS IN SOLIPEDS.

Definition. Causes: bacteria, toxins, irritant diuretics, musty oats or fodder, foul water, cantharides, turpentine, aqueous grasses, onions, moulting, cold, chills, injuries to loins, over driving. Lesions: kidney enlarged, red, black, softened, capsule loose, cut surface drops blood, brown, softened necrosed areas, gorged capillaries of glomeruli and convoluted tubes, granular or fatty changes in epithelium, may be ruptures. Symptoms: sudden; weak tender loins, slow dragging straddling gait, accelerated pulse and breathing, anxious countenance, colics, sweating, urine from limpid to black, with red globules, and casts. Prompt recovery or nephritis. Diagnosis: from nephritis, hæmoglobinuria, laminitis, indigestion. Prevention: Treatment: bleeding, laxatives, diffusible stimulant diuretics, bromides, diluents, mucilaginous agents, fomentations, sinapisms, rectal injections, clothing, friction to the skin, restricted laxative diet.

Definition. Active congestion of the renal capillaries, especially of those of the glomeruli and convoluted tubes, with colicy pains, and free discharge of urine, in some cases bloodstained.

Causes. It may be determined by local irritation caused by the passage of the bacteria and toxins of infectious diseases such as influenza or contagious pneumonia. In the same way irritant diuretics, medicinal, alimentary and toxic, operate. Diuretic balls and condition powders given recklessly by stablemen and grooms, saltpeter, resin, oleo resins, turpentine, rue, savin, colchicum, squill, anemone nemorosa, adonis, cynanchum vincetoxicum and other species of ascelepias, hellebore, mercurialis annua and bryony are examples. The young shoots of the coniferous plants, fir, balsam fir, pine, white and yellow, and hemlock, are at times injurious.

In the same way, damp moldy oats or fodder produce renal congestion and excessive polyuria, also corrupt, stagnant water and that of marshes which often contains complex toxic products of fermentation. Water of ponds in which cantharides or potato beetles have been drowned, is dangerous. The cantharides, euphorbium or oil of turpentine applied too extensively to the skin as a counterirritant, is another factor.

Even the rich aqueous grasses of spring succeeding to the dry winter diet, stimulate the kidneys, determining an active congestion with polyuria and in bad cases hæmaturia. In many such

cases there are superadded the acrid diuretic plants already referred to. In Denmark where onions are grown on a large scale, the tops fed to animals have produced renal congestion.

There appears to be an extra susceptibility in spring when the winter coat is being shed, and at this time especially, but also independently of this and at other seasons, exposure to cold and the occurrence of chills tend to induce an attack. Exposure to cold storms of rain or sleet when perspiring or fatigued, standing tied out of doors in zero weather without a blanket, wading or swimming deep rivers in cold weather and while fatigued, standing wet and unblanketed in a cold stable when returned from work, exposure to draughts between open windows or doors, the continuous falling of cold rain, from a leaking roof, on the loins, the cold of a damp stable newly finished in brick or stone, the cold and damp of an undrained floor in a wet retentive soil, all have a tendency to drive the blood from the surface, to increase the tension of the blood in the heart and internal organs, to stimulate the kidneys to extraordinary secretory activity, and at the same time to temporarily debilitate the whole system and lessen the power of resistance and recuperation. The factor is especially potent when it involves the nervous interdependent sympathy between the chilled loins or abdomen and the kidneys. Sprains and other injuries of the loins have long been charged with producing renal congestion and inflammation, and even Trasbot, who doubts the reality of this, acknowledges that the already diseased kidneys are seriously injured in this way. Cadeac and Schmid record cases of actual rupture of the horses' kidney from violent movement, and other cases of congestion and bloody urine have been traced to kicks on the loins, falls, sprains and the carrying of unduly heavy loads. The overexertion which produces albuminuria, casts and sanguineous transfusion in athletes has a similar effect on the overdriven race horse, trotter or draught horse.

Lesions. The congested kidney is enlarged, sometimes to two or three times its natural size, softened, and red, especially in the cortical portion which may be so dark as to appear cyanotic. The capsule is also the seat of ramified redness, and is very loosely adherent to the cortex. Beneath it may be considerable yellowish exudate especially abundant in the vicinity of the hilus. On section the cut surface is very bloody, the cortex

literally dropping blood, though brownish spots may appear at intervals representing areas of necrosis, which under pressure break down into a pulpy débris. Microscopically the glomeruli appear hæmorrhagic, the capillary vessels being gorged to excess, while blood globules and even minute blood clots are found in the intervascular spaces. The epithelium covering the glomeruli and lining the convoluted tubes show granular or fatty changes, and granular matter is found outside the vessels.

The congestion is less in the medullary portion and even in the convoluted tubes and the tubes of Henle, though these may be the seat both of hyperæmia and exudation.

In case of very violent congestion, extensive sanguineous extravasation may occur, leading even to rupture of the capsule and the escape of blood into the perirenal adipose tissue or into the abdominal cavity. Cases of this kind in the soliped are recorded by Caroni, Cadeac, Moussu, Kitt, Zundel, Mollereau and Porcher. Averons describes in the *Revue Veterinaire* (1897) a case in which both kidneys were surrounded by an immense black clot, and weighed no less than 36 lbs. Leblanc records a similar case affecting the one kidney. The mass measured about 10 inches by 8.

Symptoms. These are liable to appear suddenly, often while the patient is at work, and are manifested by weakness in the loins, slow gait or sudden stopping, the hind limbs are held in abduction, and advanced with apparent stiffness and pain. There is much excitement and anxiety, the face is pinched and strained, the respiration accelerated, the pulse hard, tense and rapid, and the eyes or nose may be turned toward the flank or loins. There may be colicy pains, with uneasy movements of the tail and hind limbs, pawing, and even lying down and rolling. The visible mucosæ are strongly injected and in bad cases the skin may be drenched with sweat. There is at first little or no hyperthermia.

At first there may be no micturition but in an hour or more, urine may be discharged in excess, sometimes as much as 25 quarts, and of a low specific gravity (1001 to 1005). If there has been no blood extravasation it is usually clear and limpid but with extravasation it may be of all shades of pink or red to black. In the latter case the suffering is liable to be acute (Cadeac), and contrary to the condition in hæmoglobinuria, the urine contains

blood globules and even tubular coagula representing the uriniferous tubes and entangling the blood cells. This is complicated by albuminuria.

Course. Duration. The congestion is short lived. It speedily undergoes resolution with the passage of normal, clear urine, and the recovery of appetite and spirit, or it becomes rapidly aggravated, with continuous suffering and colic, complete loss of appetite, dullness, constant decubitus, weakness, debility, small or imperceptible pulse, palpitations, darker color and perhaps complete suppression of urine, and stupor or other nervous disorder. Death may occur on the fourth to the sixth day. It may be delayed by a partial recovery followed by a relapse.

Diagnosis. Acute renal congestion is distinguished from *nephritis* by the suddenness of the onset, the absence of fever and the comparative absence of tenderness of the loins, and of tubular casts.

From *hæmoglobinæmia* it is distinguished by the absence of the conditions under which that affection appears:—the previous heavy work and full rich feeding, the day or more of complete rest on full ration, and the sudden exercise following. The hind parts in hæmoglobinæmia are benumbed, paretic, or paralytic and not unfrequently rigid and swollen, and the brownish or reddish urine contains hæmoglobin in amorphous particles, and not red blood globules and sanguineous tubular casts as in renal congestion.

From *laminitis* it is distinguished by the absence of high fever, by the absence of the advance of the fore feet resting on the heels, of the heat and tenderness of the feet, by the ability to bear the lifting of one fore foot, or the tap of a hammer on the toe, by the lack of improvement after the first few steps as is seen in laminitis, and by the absence of the strong pulsations in the digital arteries.

From *indigestion* it is distinguished by the absence of the history which leads up to that condition, of abdominal tympany, of rumbling, of impaction and of frequent attempts to defecate, and by the presence of the stiffness, straddling, and the blood globules and albumen in the urine of low density.

Prevention. This must be sought by the avoidance of all the factors of causation:—autointoxication in contagious diseases,

excessive renal irritation from the injudicious use of diuretics, or the accidental ingestion of irritant or acrid diuretic plants or waters, or musty fodders, or the sudden change to the succulent, watery, first vegetation of spring, or of exposure to cold, wet, or damp, in all their forms, or of direct injury to the back or loins by blows, shocks, or violent exertions.

Treatment. Trasbot and Cadeac strongly recommend venesection, and at the very outset in specially acute cases the sudden lessening of the arterial and capillary tension, by this potent means, may furnish the opportunity for the capillaries of the glomeruli and tubes to regain their normal tone, and thus contribute to a speedy abortion of the affection. If resorted to at all it should be made in a full stream from the jugular, so as to secure the fullest and most prompt result with the least possible effusion of blood.

Much, however, must depend on the attendant conditions. In toxin poisoning following on an infectious fever, the already existing debility will sufficiently forbid a resort to the lancet, and we must seek elimination by the bowels, the skin or even the kidneys. Antiseptics, too, are in order if there appears any ground for suspicion of the action of infecting agents. Some cases will recover promptly under diffusible, stimulant diuretics such as spirits of nitrous ether, which by stimulating the circulation in other organs and especially the skin, appears to relieve the kidney and solicit normal secretion. But most veterinarians dread the stimulus and irritation and prefer small doses of refrigerant diuretics: bicarbonate of soda 4 drs., saltpeter 2 drs. or the tartrates, citrates or acetates of the alkaline bases. In case of irritation by acrid diuretics, but especially by cantharides, camphor 2 drs., has been found to be particularly soothing, and next to this, bromide of camphor or bromide of potassium 1 to 2 drs. may be resorted to. Zundel prescribes acidulated camphorated drinks. The free use of mucilaginous drinks, such as boiled flax seed; and the persistent application of fomentations or wet compresses to the loins are of equal value in soothing irritation. Sinapisms may advantageously follow the local emollients.

Laxatives act with less promptitude than diuretics, but on the whole constitute a safer treatment, since they secure elimination and derivation without risk of irritation to the kidneys. The

oils: castor 1 to 2 pints, linseed 2 pints, or olive 2 pints, are especially to be recommended in this respect, but l'Homme advises manna, and calomel may also be used as a substitute. Injections of warm water are valuable in unloading the rectum and colon, soothing the kidneys and soliciting peristalsis.

A restricted amylaceous diet is essential, and a warm stall or abundant clothing. Grooming or active rubbing of the skin tends to active derivation and often materially relieves. The case should not be abandoned until a day or two after the urine has returned to the normal, and for some time special care should be taken of the diet, stabling and work.

ACUTE CONGESTION OF THE KIDNEYS IN CATTLE.

Causes: infection, toxins, etc., irritant diuretics, chills, moulting, swill. Lesions: cortical kidney congestion, red to black, softening friability; urine limpid to red, with blood globules, albumen, and crystals. Symptoms: chill, tender loins, colic, straining, recovery in four days. Diagnosis: from hæmoglobinuria, cystitis, calculus. Prevention: diet, etc. Treatment: laxatives, flax seed, wet compresses, bromides, camphor, disinfectants, bitters.

Causes. In cattle this malady is largely traceable to the same causes as in the horse, and is very often but a complication of some other affection. The renal congestion of infectious diseases is seen in the advanced stages of lung plague, in anthrax, in malignant catarrh, in hæmoglobinæmia, and implies an accumulation of irritant toxins in the system. The abuse of diuretics, the ingestion of acrid diuretic plants, including the early shoots of the coniferæ, the introduction through any channel of cantharides or potato beetles, the drinking of stagnant water charged with deleterious fermentation products, the consumption of musty or spoiled fodder, and the sudden change to the succulent grasses of spring, operate as in the horse. So it is with cutaneous chills, cold stone floors, cold wet storms, draughts and dropping from a leaky roof. The shedding of the coat in spring is an undoubted predisposing cause.

Cattle in the swill stables of breweries and distilleries are the subjects of a constant renal congestion and polyuria, which, however, does not prevent rapid fattening. This diet, how-

ever, unfits the animal for a future vigorous life, and any concurrent injurious influence may easily bring on active kidney disease.

Lesions. There is redness and swelling of the kidney, it may be to two or three times its normal size, the enlargment being especially referable to the cortical portion, which may be mottled in different shades of red up to black extravasations. The lack of firmness in its connection with its sheath, and the softening and friability of the parenchyma resemble the same conditions in the horse. The urine may be clear or more or less tinged with blood, and contains blood globules, albumen, and crystals of carbonate of lime and urate of ammonia, which seem to indicate the presence of a bacterial ferment.

Symptoms. The patient usually shows some indication of chill, with staring coat and arched back, which is very sensitive to pinching. There is impairment of rumination and appetite, decrease of milk in dairy cows, uneasy movements of the hind limbs and tail, frequent straining to urinate, and the passage of urine often in small amount and sometimes of a pink or reddish tinge. In bad cases this may become deep red, or black, and the pulse becomes weak, with palpitations, marked muscular weakness and a tendency to lie down most of the time.

With early improvement recovery may be complete in from four to six days. In the more severe and fatal cases death may occur as early as the sixth day. Unless under the influence of violent irritants or a persistence of the original poison the prognosis is favorable.

Diagnosis. It is especially important to distinguish this from **hæmoglobinæmia**, which shows an uniform red or brown discoloration of the urine and an entire absence of blood globules as such. In congestion the reddish material tends to precipitate and is found to consist largely of blood globules. It is further associated with albuminuria.

Hæmorrhagic cystitis and **cystic calculi** are both chronic affections, and identified on rectal exploration by the tenderness of the bladder and the presence of the stone.

Prevention consists in the avoidance of the various causative factors, and especially those that find access among alimentary

matters. Cattle turned out in early spring should be fed before going and should be returned from the pasture in an hour or two. This repeated day by day, allows the digestive and urinary organs to accommodate themselves to the fresh spring grass and to any vegetation to which the animals have not been accustomed. Chills, draughts, injuries and other disturbing conditions must be guarded against.

Treatment. Bleeding is strongly recommended by Cruzel and Cadeac. In Germany, England and America derivation toward the digestive organs is more generally relied on. Laxatives should be, as in the horse, oleaginous (castor, olive, linseed) or manna, rather than agents that may perchance act on the kidneys. Free purgation should be secured. Flaxseed tea, and wet compresses over the loins are valuable adjuncts, and anodyne agents like camphor, bromide of camphor, or other bromides may be added, and when there is any suspicion of infection, salicylates, or iodide of potassium may be employed. Finally a course of bitters (salicin, quinine, nux,) may be employed to restore tone and iron carbonate with sodium carbonate as a reconstructive tonic. The diet must be changed to wholesome food, but not too stimulating, and the animal kept quiet.

ACUTE CONGESTION OF THE KIDNEYS IN SHEEP AND GOAT.

Causes: irritant food. Lesions: Symptoms: separates from its fellows, arched back, stiff straddling gait, straining, muscular weakness, recumbency, urine red, with blood globules and albumen. Prevention: care in feeding and watering, change of pasture and treatment as in the ox.

Causes. As in cattle, the smaller ruminants appear to suffer especially from an alimentary renal congestion, showing itself mainly in animals that are unaccustomed to the particular toxic aliment. Thus, Cornevin finds that the Pyrennean sheep thrives on the leaves of the Quercus tosa, while Southdown sheep taking it in any considerable quantity perish of renal congestion or nephritis. Similarly Weith fed four sheep on cynauchum vincetoxicum and developed renal congestion in the course of

three days. Other causes doubtless contribute in individual cases but have not been specially traced to their effects.

Lesions are in the main the same as in cattle, the kidneys being bluish red, soft, flaccid and friable.

Symptoms. The sheep lags behind the flock, frequently lies down and rises, strains to urinate, and passes often considerable quantities. The back is arched, the loins tender, the walk stiff and straddling, the pulse small and weak. If the disease advances, there come on extreme muscular weakness, a disposition to lie, an uncertain, gait, with frequent stumbling, dullness, stupor, and it may be coma. The urine is usually tinged with blood or of a deep red or black, and contains well-formed blood globules and more or less albumen.

Prevention and treatment should proceed on the same lines as in the ox, but in dealing with a large herd it becomes difficult to treat each separate case with special care. The avoidance of sudden change of food as in turning out in spring, the feeding of grain before turning out, the return to the fold after a short freedom, and the gradual transition to the new food are important. When the disease has developed, an entire change of pasture or food, the use of roots, ensilage, or grain, or of freshly cut meadow grass, is indicated, and an oleaginous laxative (castor oil 2 to 3 ozs.) are indicated. Oilcake or flaxseed meal will often prove a most valuable article. Beyond this the same agents would be indicated as for the ox.

ACUTE CONGESTION OF THE KIDNEYS IN SWINE.

Causes: infection, toxins, fermented food, traumas, crowding, cold. Symptoms: stiff loins and quarters, frequent micturition, urine limpid or red. Treatment.

Renal congestion in pigs has been seen mainly as the result of toxin poisoning in swine erysipelas, hog cholera or caseous pneumonia. It is also liable to occur from putrid or overfermented food, and in fat, heavy animals from injuries sustained in shipping by rail by trampling on or squeezing each other. Kicks and other injuries may at times contribute to its occurrence.

Exposure to cold storms, to which swine are especially sensitive, a wet, cold bed, or a leaky roof, are additional causes.

The *symptoms* are more or less stiffness of the loins and hind parts, frequent urination, the secretion being often passed in excess, and though at times clear yet at others pink or bloody and precipitating blood clots or at least containing blood globules.

Treatment is mainly prophylactic. If therapeutic measures are desirable for valuable animals, they should follow the same lines as for sheep: rest, fomentations, aqueous food, anodynes, weak alkaline diuretics, laxatives, and balsams.

CONGESTION OF THE KIDNEYS IN CARNIVORA.

Causes: acrid diuretics, loss of kidney, catheterization, dermatitis, burns, traumas, overexertion. Lesions: enlarged, blood-gorged kidney, red or black, petechiated. Symptoms: stiff, arched, tender loins, tardy, dragging of hind limbs, urine passed often, clear to bloody, albuminous, anorexia, nausea, vomiting, diarrhœa, dullness, stupor. Treatment: stop cause, give emetic, laxative, in surgical cases antiseptic, for cantharides, camphor, bromides, vegetable food. Warm clothing or building.

Causes. This comes most commonly from the ingestion of acrid or diuretic agents, saltpeter, turpentine oil, cantharides and, according to Cadeac, various essential oils including oil of mustard. It takes place in the remaining kidney after the one has been extirpated, or had its functions abolished by disease or uretral obstruction. Again, surgical operations on the urinary organs, even the simple passing of a catheter, will cause sympathetic renal congestion. Extensive acute dermatitis, and burns of the skin may have a similar sequence.

Falls, kicks, blows, or crushing beneath a wheel or otherwise are additional causes.

Finally violent overexertion as in coursing, causes congestion with albuminuria, and blood globules and even casts in the urine. This is common to the human athlete, who undergoes a violent and continued overexertion, race and draught horses, and dogs.

Lesions. When congestion is produced experimentally by cantharides the kidneys are found to be enlarged and the cortex gorged with blood so that it has a deep red or blackish port wine

hue, with here and there spots of ecclymosis. The veins capillaries and glomeruli are especially congested, and the epithelial cells of the convoluted tubes have become laterally distended, so that they approximate to a globular form. A loose coagulum containing blood globules may be found in the capsule of the glomerulus and in the convoluted tubes.

Symptoms. These are arching and stiffness of the loins, a tardy, dragging movement of the hind limbs and tenderness of the loins. The urine may be scanty or in excess, and tends to be passed frequently, in small quantities and with evidence of pain. It may be clear, pinkish or bloody, and shows albumen and frequently casts, and blood globules or small clots. There is some impairment of appetite, and, in severe cases, nausea and vomiting, with, it may be, diarrhœa, nervous depression, dullness and stupor. When due to poison or other transient cause the symptoms improve when this factor has been stopped.

Treatment. When due to poison taken by the stomach this must be stopped, and the stomach and bowels evacuated by an emetic (ipecacuan) and laxative (sodium sulphate). Appropriate treatment must be made in case of burns or skin eruptions. When surgical cases are due to infection rather than simple shock or sympathetic irritation, antiseptic injections of the bladder are indicated. In all cases alike a warm bath is an important adjunct. When irritation is due to cantharides, it may be calmed by camphor, 2 grains every three or four hours. Other anodynes may be given as required. Rest is essential and, as appetite is recovered, a moderate amount of amylaceous puddings. A warm building or comfortable clothing is desirable.

NEPHRITIS.

The renal inflammations have not been fully investigated in the domestic animals, and even in man, the pathology of several of the forms is still enveloped in some measure of doubt. *In man* the following conditions have been noted:

1st. *Acute parenchymatous nephritis* with enlarged kidney and degenerated tubules.

2d. *Chronic parenchymatous nephritis* with enlarged kidney and degenerated tubules.

3d. *Acute diffuse (desquamative, interstitial) nephritis* with enlarged kidney and glomeruli, tubules and connective tissue degenerated.

4th. *Chronic diffuse (desquamation, interstitial) nephritis.*

5th. *Suppurative nephritis and pyelo-nephritis*, infection may be from injury.

6th. *Perinephritis:* infection of connective tissue with adjacent disease.

In domestic animals the following distinctions have been made:

1st. *Acute nephritis.*

2d. *Chronic nephritis.*

3d. *Purulent nephritis and pyelo-nephritis.*

4th. *Perinephritis.*

ACUTE NEPHRITIS. ACUTE BRIGHT'S DISEASE.

Animals affected. Causes: hyperæmia, traumas, cold, chill, fever, bacteria, toxins, overfeeding, nitrogenous food, raw potatoes, xanthin products, acrid diuretics, diuretic insects, suppression of micturition, skin lesions, burns, embolism, calculus. Symptoms: colic, trembling, rigor, arched, stiff, tender loins, stiffness in quarters, drags hind legs, urination frequent, movements of penis and testicle, costiveness, grinding teeth, anorexia, vomiting (in dogs, cats and pigs), fever, dropsies, uræmic convulsions, urine scanty, high colored, red or bloody, thin, cloudy or turbid, albuminous, purulent, oxalates, urates, hippurates, hæmatoidin, epithelium, mucus, casts. Prognosis: resolution in three days, or uræmia, suppuration, degenerations. Lesions: kidney enlarged, softened, friable, red, yellow, black, purulent, glomerulitis, tubular nephritis, interstitial nephritis. Treatment: rest, warm building, warm clothing, green or sloppy, amylaceous food, bleeding, cupping, skin friction, fomentations, warm bath, hot air bath, sinapisms, anodynes, laxatives, diaphoretics, heart tonics, alkaline diuretics, paracentesis, bitters, iron, phosphates, hydrogen peroxide, cubebs, etc.

Genera affected. This has been seen in horse, ox, dog, sheep and pig.

Causes. It is ascribed to the most varied causes, such as: hyperæmia, blows and injuries on the back and loins, sprains of

the loins, abrupt wheeling when in galop, exposure to cold winds, and storms, especially when perspiring and fatigued, sudden suppression of perspiration, extreme terror, bacterial infection and infection by toxins (in septicæmia, pyæmia, influenza, contagious pneumonia, uterine sepsis, omphalitis, infectious angina (Friedberger), bronchitis (Siedamgrotzky), glanders, tuberculosis. Among dietary causes are named: a rich nitrogenous food (grains, beans, peas, vetches, cotton seed, clover), raw potatoes in excess, cotton seed meal, agents that increase the nitrogenous and xanthin bodies in the urine.

Dr. Alfred C. Croftan, in his experiments with **xanthin bodies** on rabbits, found that xanthin and hypoxanthin produced great increase of arterial pressure, atheromatous changes in the vessel walls consisting in thickening of the intima, with small celled infiltration and necrotic changes in different areas of the vessel walls. This in the kidney produces the primary interstitial form of nephritis known as gouty kidney and associated with retention of uric acid and other xanthin bodies. The accompanying cardiac hypertrophy, so common with such kidneys, he attributes to the increased intravascular pressure.

Irritant vegetables that are resinous or diuretic, and irritant diuretic insects are incriminated (cantharides, caterpillars in grass, or on plants, etc., lice on cabbages particularly,—Cruzel, Neubert). Irritant drugs that are eliminated by the kidneys have been equally charged (tar, carbolic acid, iodoform, chlorate of potash, nitrate of potash, phosphorus, arsenic, lead, mercury). Compulsory suppression of micturition is undoubtedly injurious in house dogs shut up, mares kept long in harness, or horse on railway car, above all if this follows a diuretic or drinking abundantly. In such cases it is altogether probable that bacteria already exist in the blood or kidneys and take occasion to attack the tissues weakened by the overdistension or other inimical cause. This is all the more probable seeing that the kidneys are a favorite channel for the elimination of bacteria present in the system. It should be noted that nephritis is liable to supervene on extensive skin burns, chronic dermatitis and other skin diseases. Some cases are traceable to embolism, the clots coming from the lungs, heart or arteries, in others the irritation is due to calculi in the renal pelvis or tubules, and their attendant bacteria.

These are especially common in cattle that are winter fed on dry food. Again, the infection may have travelled forward through the ureters from a pre-existing infective cystitis.

Symptoms. There may be obscure or intense colic; trembling or rigor may occur, yet is often omitted or unobserved; the loins are arched; the hind feet are advanced under the belly, or there is frequent shifting of the weight from one foot to the other; the walk shows stiffness of the back and hind limbs which appear to straddle or drag behind; urination is frequent in small amount, or there are frequent ineffectual attempts to urinate; the patient is indisposed to lie down, and if he does so it is carefully, with difficulty and groaning; the testicles are drawn up and dropped alternately, the penis is often protruded from and retracted within its sheath, the loins are sensitive to pinching, percussion, or electric current; when mounted the animal drops under the weight; he carries the head low and refuses to go fast. In bad cases there is constipation, grinding of teeth, anorexia, and in dogs, vomiting. Temperature may be normal or there may be considerable fever. Dogs may lie curled up, with occasional tremors. Dropsical effusions are frequent in the form of anasarca under the chest or abdomen, or beneath the lower jaw, or as stocking of the limbs, or the effusion may occur into an internal serous cavity. Convulsions may occur from brain poisoning by urea or other retained urinary product.

In the slighter forms the severe symptoms may be absent, and the *condition of the urine* must be investigated as affording *the most constant and characteristic* phenomena.

The *urine* is usually scanty, high colored, of a high specific gravity and is passed often with pain and groaning. At the outset of an acute attack it may be bloody; later it may be only cloudy or turbid from the excess of epithelial and pus cells, leucocytes, salts and albumen. Early in the disease the casts may contain red blood cells, and renal epithelium, later leucocytes, nuclei, granules, pus cells, crystals and other matters. Albumen is usually abundant as demonstrated by boiling and nitric acid.

Soda carbonate crystals, rhomboid, rosette-shaped or spherical and effervescing with acetic acid, abundant in normal herbivorous urine may be greatly reduced or absent in nephritis.

Soda oxalate crystals, tetrahedral and insoluble in acetic

acid, and normal in herbivora and carnivora, are increased if the urine is acid as in severe nephritis, but also in rheumatism, tetanus, septicæmia, angina, heaves, and other affections with defective æration of the blood.

Ammonio-magnesian phosphate crystals, rhomboid but insoluble in acetic acid, are found in alkaline (ammoniacal) or neutral urine, and appear to be often due to intestinal fermentations.

Cystine crystals, flat hexagonal plates, precipitated in healthy urine, but dissolved by ammonia are absent in retained and fermented specimens.

Uric acid crystals, rhomboids and plaques, brick red, and normal in the urine of carnivora and flesh-fed omnivora, may be present in herbivora not only in acute nephritis, but in other extensive inflammations attended with anorexia and the consumption of the animal tissues.

Hippuric acid crystals, right rhombic prisms and their derivatives, and insoluble in hydrochloric acid or ether, are greatly increased in all febrile diseases in herbivora, nephritis included.

Hæmatoidin crystals, fine needles or bundles of the same, yellowish red, are found in nephritis, hæmaturia, heaves, etc.

Epithelium, if columnar, points to disease of the kidney tubes, though very similar cells are derived from the urethra in both male and female. Squamous epithelium points to the cystic mucosa and is not increased in nephritis.

Mucus in cylindroid form may point to nephritic congestion or inflammation, but this may be present in health, and may show in irregular masses derived from the renal pelvis or the bladder. Mucous casts are always extremely elastic and mobile, and lack the even clear cut margins of the casts of nephritis. They are much more common in horse's urine than in that of other animals.

Tube casts are especially indicative of nephritis and exudation into the uriniferous tubes. They are much firmer than the mucous cylinders and have smoother and more even margins. If relatively thick and straight they probably come from the straight tubes; if sinuous or twisted, from the convoluted tubes. With a similar basis substance they often enclose different solid bodies and have been named accordingly:—**epithelial casts** when containing cylindroid, or polyhedral cells may be unhesitatingly referred to the uriniferous tubules:—**granular casts** in

which the homogeneous cast is impregnated with granular cells and free granules of proteid, fatty, or mineral matter, point directly to inflammation affecting the uriniferous tubules and their epithelial lining :—**blood casts** enclosing red blood globules imply hemorrhage, or congestion or inflammation of the tubules, with blood extravasation or diapedesis :—**casts containing leucocytes and pus cells** bespeak suppurative inflammation of the tubules :—**calcareous casts** entangle numerous crystals and granules, mainly of lime carbonate, and effervesce with acetic acid :—**hyaline casts** are homogeneous, clear, so transparent that it is sometimes necessary to stain them with iodine or aniline to make them distinct ; they are found in nephritis and especially in the chronic forms :—**colloid casts** or **waxy casts**, or **amyloid casts** may designate a class of firmer cylinders, clear, homogeneous and refractive, and often bearing fatty or blood globules, crystals or fungi. They may have a yellow color, or they may give the amyloid mahogany reaction with the iodo-potassic iodide solution (even in the absence of amyloid degeneration of the kidney ; Jaksch).

Progress Acute nephritis may advance for three days or more and then terminate in resolution, or go on to complete anuria with coma, to suppuration, gangrene or chronic nephritis.

Resolution is marked by general improvement of pulse, breathing and expression, clearing of the urine, and return of appetite. The urine may remain albuminous for eight days longer.

Complete suppression of urine has persisted five days in cattle (Funk), and seven days in horses (Friedberger), accompanied by intense fever, dullness, stupor and coma ending in death from uræmia.

Purulent urine is white, milky, albuminous, granular, with epithelial cells and casts and pus cells, showing their double nuclei with acetic acid. There are usually rigor, hyperthermia (106° F.), thirst, intermittent colics, diarrhœa, perspiration, uncertain walk, and stocked legs. Convulsions have been noticed in the horse (Didie), cow (Pflug) and bitch (Trasbot). The horse may turn in a circle (Friedberger) or have amaurosis (Didie). Death usually occurs in two weeks.

Gangrene is likely to prove fatal. Berger has seen death oc-

cur in three days in the horse, and Trasbot in four days in a cow, after a large cantharides blister.

Pathological Anatomy. The kidney is enlarged, soft, friable, dark red, yellow with red spots, or having areas of hemorrhage. When fatty it is marbled, pale yellow or white and red. The capsule is easily detached. On section it is bloody, oozing or even dropping blood, or a pale creamy fluid. The pelvis contains urine, thick, gelatinoid, bloody or purulent. The latter condition must not be confounded with the thick pus-like mucus which normally occupies the renal pelvis in the horse.

The lesions of the secreting portions of the kidney will vary with the concentration of the inflammation in one or other of the separate tissues.

In **glomerulitis** from toxic irritants, the capsules enclose an albuminous liquid exudate, the capillaries are overdistended, their walls thickened and cloudy, and thrombi with an excess of red globules and leucocytes block them at intervals. This capillary obstruction extends to the plexus surrounding the convoluted tubules.

In **tubular nephritis** there is congestion of the plexus covering the convoluted tubes, and the epithelium shows cloudy swelling, with fatty granules and hyalin droplets in the desquamating cells.

With **interstitial nephritis** there is an exudate into the interstitial connective tissue between the tubules, and into the tubules, forming hyalin casts. The epithelium of the tubules are swollen, granular, opaque and desquamating.

In **suppurative nephritis** may be found all stages of abcedation from minute points, gray or yellow, and only just visible to the naked eye, in the midst of the deep red congested tissues, through the larger white suppurative areas, to the extensive abscess formed by a coalescence of the many, the intervening tissue having broken down by a necrotic disintegration. In the earlier stages the pus infiltrates the parenchyma so that it may be comparable to a sponge filled with this liquid.

Treatment. The first consideration is rest, with a warm building or clothing to solicit the action of the skin and lessen the work of the kidneys. Warm summer weather is favorable, or we should secure a sunny, comfortable, loose box, or a building

heated by a stove. In default of this, warm woolen blankets, hood and leg bandages should be secured. If the case is mild enough to allow of appetite, the food for herbivora may be green food in summer and carrots, beet, turnip, potato or ensilage in winter. The dog may have buttermilk or sweet milk or mush and milk. Meat is objectionable because of the amount of urea and other urinary products which it produces.

Trasbot strongly recommends general bleeding in strong, vigorous horses and cattle, attacked by the disease in an acute form, but deprecates it in the lymphatic, fat, or debilitated.

Omitting the general bleeding, one can always find a good and safe alternative in bleeding the animal into his own tissues. Shaving the loins and cupping has often an excellent effect. An approach to this may be had by vigorous rubbing by several men at once, of the limbs and the whole surface of the body, by warm fomentations over the loins by means of spongio-piline or surgeon's cotton covered with dry blankets, or by winding a hose round the body through which warm water is forced, or finally by a bath of steam or hot air, or in small animals of warm water. The dog may be placed in a bath of 80° or 90° F., which is allowed to gradually cool to 65° or 70°. In all these cases the greatest care must be taken to avoid chill when the animal is taken out. He should be quickly rubbed dry in a warm room and blanketed.

Counter-irritants act in the same way, and mustard or hot water hotter than the hands can bear may be applied. Turpentine, cantharides and other diuretic counter-irritants must be carefully avoided. An old practice of laying a freshly removed sheep skin over the loins, with the flesh side inward, often causes a distinct exudation, thickening of the skin and derivation.

A damp cloth, laid across the loins and thoroughly covered with dry to prevent any evaporation and chill, will usually give great relief and may be kept on for days.

Internal medication must at first be mainly anodyne, laxative and diaphoretic. The two latter classes are at once derivative and eliminating, carrying out through other channels, waste products that would otherwise have taxed the kidneys.

Among anodynes, the bromide of camphor (horse, 1–2 drs., dog, 2 to 5 grs.), bromide of potassium (horse, 1 dr., dog, 1 to 3 grs.), or hyoscyamus may be used, and repeated twice daily.

Purgatives must be restricted to such as have no tendency to act on or irritate the kidneys. Castor oil, or sweet oil for the larger animals, or for the dog senna or jalap, may be given every morning to secure free movement.

As diaphoretics, ipecacuan, Dover's powder, tartar emetic, and even pilocarpin may be used. The last named agent is especially useful when dropsy sets in, or uræmic stupor or coma threatens (horse 3 grs., ox 7 grs., dog $\frac{1}{2}$ to $\frac{1}{3}$ gr. according to size). If the heart shows weakness it must be sustained by digitalis, strophanthus, caffein or nitroglycerine, and the pilocarpin withheld.

In a sufficiently strong subject the stupor or coma may be met by the abstraction of blood, which benefits by the dilution of that which is left.

Eclampsia may be further met by the inhalation of ether or chloroform, or the rectal injection of chloral or bromide solution.

As the inflammation abates, if the action of the kidney is still insufficient in spite of the free drinking of pure water, alkaline diuretics may be given in small doses (tartrate, acetate or citrate of potash, bicarbonate of soda, saltpeter).

In excessive dropsy avoid sloughing by lancing the most tensely swollen parts to allow drainage, and keep the parts disinfected with carbolic or other antiseptic lotion. For ascites or hydrothorax, aspirate, and apply a compressory bandage.

During convalescence a course of bitters (cinchona, salicin, gentian, nux vomica) and iron (phospho-tartar, iodide or phosphate) will often be called for. Anæmia may be met by doses of peroxide of hydrogen or the inhalation of oxygen.

In the advanced stages benefit may accrue from the use of small doses of cubebs, copiaba, oil of turpentine or buchu, which have a tonic action on the renal mucosa.

PURULENT NEPHRITIS.

Causes: general, traumatic, metastatic, infective, wounds, shocks, strains, blows, falls, crowding, heavy loads, calculi, infective embolism. Lesions: miliary or large abscesses, diffuse suppuration, softening, disintegration, fistula. Symptoms: obscure, nephritic symptoms following distant abscess, chill, hyperthermia, general symptoms of nephritis, pus in urine, anæmia, emaciation. Treatment: to external wound, antiseptics, evacuate abscess, extirpate kidney with pyonephrosis, in dog or pig, calcium sulphide, sulphites, copiaba.

Causes. Aside from the main causes of nephritis, the suppurative form may be determined by traumatic or metastatic infective conditions. Under *traumatic* factors may be named punctured or gunshot wounds, shocks and strains connected with falls, blows, crowding, compression, too heavy weights on the back (pack, rider, two wheeled cart loaded too heavily forward and going down hill), and finally calculi in the uriniferous tubules. Under *metastatic* factors come all infections, pyæmia, omphalitis, any suppurative affection of the lungs, (abscess, pneumonia, broncho-pneumonia), pharangitis, etc. Embolic renal abscess may start from endocarditis, arteritis, or pulmonary phlebitis,

Lesions. There may be a circumscribed renal abscess like a good large orange, or many small gray spots like millet seeds, peas or hazel nuts, having purulent centres and containing pus cocci or bacilli. In other cases a diffuse inflammation suppurates throughout till the whole gland becomes a pulpy mass of pus, blood and broken down kidney tissue (pyonephrosis). In traumatic cases the pus centres around the wound or injury, perhaps invading adjacent parts, and even communicating through the skin externally along the line of the original wound. The pus may burrow in different directions in the cortex or under the capsule with abscess at intervals (perinephritis), or along the vessels to the medullary structure.

Symptoms. These are often obscure. The sudden appearance of kidney disease in the course of a suppurative affection elsewhere, the extension being ushered in by a chill or rigor or attended by a succession of these, and the course marked by

a variable hyperthermia is very suggestive, Stiffness and weakness of the hind parts and tenderness of the loins are significant ; also, in carnivora and omnivora, nausea and vomiting. When the kidney can be felt by the hand in the rectum or in the small animals, through the flaccid abdominal walls, the manifest enlargement, the tenderness, and in some cases even fluctuation will assist in diagnosis. In such cases, puncture by a large hypodermic needle, or a small trochar may betray the presence of pus and complete the diagnosis. If the pus escapes into the pelvis of the kidney it may be recognized in the urine. The case is very liable to become chronic, and is then marked by anæmia and emaciation.

Treatment. When an external wound exists it must be treated, antiseptically, with boric acid, potassium permanganate, or other antiseptic lotion. If a single large abscess exists, puncture evacuation through needle or trochar, and washing out with an antiseptic solution is the obvious resort. Any foreign body must of course be removed. If the suppuration is diffused through the whole mass of softened kidney, the resort of extirpation may be considered. This is always dangerous as provocative of infectious peritonitis, but it is less so in dogs and swine than in other animals owing to their natural antagonism to pus microbes. The operation should be attempted extraperitoneally, the incision being made beneath the anterior lumbar transverse processes and carried inward through the sublumbar connective tissue. The renal artery will require ligature with antiseptic catgut and all manipulations should be aseptic or antiseptic. Even if successful, this operation leaves the subject in a dangerous state, as in case of kidney disease at any future time, there is no second kidney to compensate for the temporary loss of function and uræmic poisoning is to be dreaded.

Apart from surgical measures the general treatment would be largely the same as for acute infectious nephritis, As antiseptics calcium sulphide, the different sulphites, copiaba, etc., will be indicated.

PERINEPHRITIS.

Definition. In cattle on low damp lands, acrid plants, sprain, blow, calculus, from purulent nephritis, in anæmia. Symptoms: of nephritis, soiling of tail or prepuce, albumen, pus or blood in urine, lameness, unilateral or bilateral, lumbar swelling, in small animals fluctuation, history. Lesions: abscesses around kidney, under capsule, intercommunicating. Treatment: as in purulent nephritis.

Suppuration in the connective tissue between the kidney and its capsule is seen in cattle in low condition, on damp, unimproved soils like undrained river bottoms and estuaries, abounding in acrid and diuretic plants. Even among such animals it is rare and has probably a directly exciting cause in a sprain or blow on the loins, or the presence and movement of a renal calculus. It may extend from suppurations in the substance of the kidney and to such extension the weak or anæmic condition materially contributes. In man, in which such conditions have been more frequently observed, a weak or cachetic condition is considered as an essential accessory factor along with the traumatic lesion (R. Harrison). Similar conditions may be expected to bring about perinephritis in any one of our domestic animals. The author has observed it especially on the low lands on the banks of the Ouse in Yorkshire, England.

Symptoms. These are mainly those of nephritis in general, shivering, stiff movement in the hind limbs, straddling, frequent passage of urine, straining, difficulty in lying down and rising, tenderness of the loins, dropping when mounted, groaning when turned in a short circle. If the suppuration communicates with the pelvis of the kidney there may be, in females, soiling of the tail, and in males of the prepuce. Blood may be passed with the urine, and pus cells and albumen are found when it is examined. If one kidney only is affected, there is lameness in the corresponding hind limb, the special feature being inability to extend it backward. A swelling on the one side of the loins, and beneath the lumbar transverse processes just posterior to the last rib, is likely to be a marked symptom, and if this persists and is especially prominent at one point, an exploratory incision or

puncture will detect the presence of the pus. Fluctuation can rarely be detected, yet in small animals with very flaccid abdomen, the swollen, tender kidney and even fluctuation should be detected at times. The history of the case, the low, damp pasturage, the access to acrid plants, the alimentation with hay or grain covered with cryptogams, the fact of an injury and the low, weak, anæmic condition of the animal should contribute to a satisfactory diagnosis.

Lesions. In bovine kidneys affected in this way we have found general inflammation and exudation around the entire kidney and inside the capsule, with numerous small abscesses, in many instances communicating with each other. They may extend through the capsule and invade surrounding organs.

Treatment. In the treatment of cases of this kind the general principles of therapeutics for nephritis are about all that can be attempted in the lower animals. Fomentations over the loins are especially desirable as a means of relieving the suffering, and moderating inflammatory action. To the same end is the allowance of plenty of pure water as a diluent. Then the various agents that antagonize suppuration may be thought of, and some one selected for use. Beside the antisuppurants already mentioned one may use copiaba, cubebs, or turpentine in small doses, salicylates, or the sulphide or sulphite of calcium. Surgical interference by puncture or incision and antiseptic irrigation can only be thought of when the abscess is single and circumscribed; never when the whole periphery of the organ is involved. In the latter case the only rational surgery would be the desperate resort of the removal of the entire kidney.

PYELITIS. PYELONEPHRITIS. INFLAMMATION OF THE RENAL PELVIS.

Definition. Causes: primary from vegetable irritants in food, toxins, cryptogams, pelvic calculus, strongylus gigas; secondary from renal calculus, parasite, tubular uretral or vesical infection, infecting deposits, metastasis. Symptoms: as in nephritis, pus, blood, or albumen in urine, tender, arched loins, purulent polyuria, with spheroidal epithelium. Diagnosis. Lesions: inflammation, calculus, etc., in pelvis. Treatment: pure water, sodium bicarbonate, antiseptics, balsams, fomentations, piperazin, extraction of calculus.

Suppurative inflammation of the mucosa lining the renal pelvis may occur in the acute or chronic form.

Causes. It may be either primary or secondary. As a **primary** disease it may be the result of poisoning by irritant diuretics such as cantharides, turpentine, colchicum or balsams, or shoots of the coniferæ, it may be due to the passage of the irritant products of cryptogams found in musty fodder or grain, or it may come from the irritation caused by the toxins of bacteria developed in the system or in food or drink. Cases that develop from the irritation of a pelvic calculus or precipitate, and from the presence of the strongylus gigas (in dogs) may also be placed in this class.

As **secondary** causes are those in which the inflammation starting in the uriniferous tubes extends down to and implicates the pelvis, and the still more frequent instances of extension of purulent infection upward from the ureter, bladder or prostate, so as to involve the pelvis. So in blocking of the urethra by strongylus, stricture, clot or calculus, and in spasm of the sphincter vesicæ, the delayed urine is liable to undergo fermentation with evolution of ammonia, and not only the bladder but the ureter and renal pelvis may suffer. Again, the occurrence in the kidney of the hyperplasia of cancer, glanders (horse) and tuberculosis (cow) may be the direct cause of pyelitis. Similarly foci of infection in the kidney may be found in distemper in the dog, and in the contagious pneumonia and influenza of the horse. In man infection from the bowel through the migration of the bacillus coli communis to the devitalized kidney and contents in hydronephrosis, has been traced, and the liability to this must be still greater after the surgical insertion of the ureters in the rectum as a substitute for the obliterated bladder and urethra. Again, in the intra-arterial migrations of the strongylus (sclerostoma) armatus pus microbes may be carried to the kidney and reach the pelvis.

Symptoms. These are in the main those of nephritis with marked rigors. The presence of pus cells and albumen in the urine may come from suppuration in the substance of the kidney itself, or sub-capsular abscess opening into the pelvis, or it may come from cystitis, prostatitis or urethritis. The special stiffness and tenderness of the loins, polyuria in which the liquid is purulent, but free from uriniferous casts, and in which it is charged with the spheroidal epithelium of the pelvis (not the columnar of

the tubules), may afford presumptive evidence of pyelitis. But pyelitis is usually combined with nephritis or cystitis and the complications prevent diagnosis. In some cases the urine is scanty and strongly albuminous, and in others a round calculus will block, at intervals, the opening of the ureter giving rise to obstruction of the flow from that kidney and the occurrence of violent renal colic lasting until the stone is again dislodged backward.

Diagnosis cannot often be certain. Purulent urine, with a considerable number of the spheroidal cells of the pelvis, and the general signs of nephritis may be taken as diagnostic. A great excess of such epithelial cells would on the contrary point to cystitis.

Lesions. In the early stages the mucosa of the pelvis is congested, red, and sometimes, with calculus, hæmorrhagic. Later it becomes thickened by exudate, which fills also the submucous tissue. In some instances the pelvis is distended by an impacted calculus, in others the obstruction of the ureter by an impacted calculus or a swelling has led to overdistension of the pelvis, and ammoniacal fermentation of its contents. Coincident inflammatory lesions of the kidney, ureter, or bladder are common.

Prognosis is not hopeful. Where it has resulted as a descending infection from the kidney, the severity of the primary lesion renders the case a grave one, while if it has been an ascending inflammation from the bladder it is no less so.

Treatment. Diluent (watery) diuretics are especially indicated. Pure water may be given *ad libitim.* To this may be added if necessary moderate doses of bicarbonate of soda or potash with such non-irritating antiseptics as salicylic acid, salicylate of soda, sulphite of soda, sulphide of calcium, quinia or chamomile. Trasbot even recommends small doses of vegetable astringents, balsam of Tolu, or Peru or of Copiaba, or oil of turpentine or tar water. Apart from simple water, the diuretic agents may be used with greater freedom if the solid parts of the kidney are little or not at all involved and if the urine contains no casts of the uriniferous tubes.

Benefit may also be obtained from fomentations, or cupping of the loins, and even from the application of mustard and counter-irritants.

If the active symptoms subside the continued use of tonics would be indicated, especially quinia, and also of the balsams with the view at once of antisepsis and toning up of the mucous membrane.

In case of calculus of the pelvis surgical extraction is virtually the only resort, though a very desperate one. Its increase may be retarded or prevented by antisepsis, a liberal use of water, and the exhibition of piperazin or some of the essential oils.

CHRONIC NEPHRITIS.

Cases destroyed as eating their heads off. Causes : sequel of acute nephritis, swill, lead, experimentally, microbian invasions, toxins, metastatic embolism, extension from aortic disease, sclerostoma, nitrogenous overfeeding, toxins of putrid food, or cryptogams, valvular disease of the right heart, rheumatism, heaves, calculus, starvation, debility, retention of urine. Symptoms : emaciation, flabby muscles, lack of vigor, stiff loins and quarters, short step, straddling, fatigue under slight exertion, groaning in trot, or in turning, droops when mounted, slow to rise on hind limbs, poor capricious appetite, anæmia, stocked legs, dropsies, urine of lower density, albuminous, with granular epithelium and casts, abundant in early stages, scanty with weakened heart and degenerated kidneys. Secondary palpitations, bronchial catarrh, pneumonia, hemorrhage, stupor, lethargy, vertigo, etc. Lesions : recent cases, kidney large, cortex firm, capsule adherent, with granular fatty debris, and tubular casts ; old cases, kidney contracted, fibroid, glomeruli and tubules atrophied. Bronchitis, pneumonia, hepatic cirrhosis, heart enlarged, fatty, dilated, insufficient valves. Prognosis unfavorable. Treatment : gentle exercise, warmth, succulent food, amylaceous, tonics, iron, bitters, mineral acids, heart tonics, for polyuria bromides or iodides, balsams, pilocarpin, fomentations or sinapisms to loins.

Chronic nephritis has received little attention in the lower animals for various reasons. The lower animals largely escape the causative factors of alcoholism and chronic lead and copper poisoning, and when suffering from any chronic affection that disqualifies the animal for use and renders it anæmic and emaciated it is naturally sacrificed to save the cost of maintenance. In spite of this a considerable number of cases have been recorded in horses, and cattle and especially in dogs and cats.

Causes. Cases of acute nephritis sometimes improve and give promise of recovery without completing the work of convalescence.

Trasbot notes such cases in the dog, and Dickinson in the ox. Alcoholic nephritis and degenerations are to be sought for especially in cattle kept on distillery and brewery dregs. Lead taken in small quantities in soft water that has run through lead pipes or stood in leaden cisterns produces in cows and other animals chronic affections of the kidney. Ellenberger and Hoffmeister have produced the disease experimentally with lead and copper respectively.

Microbian invasions of the kidney that advance slowly like glanders and tubercle are further causes of chronic nephritis. Other secondary microbian infections of the kidney are complications of infections diseases in other parts, including abscess, pyæmia, septicæmia, ulcerative endocarditis of the left heart, bronchitis, pneumonia (Fröhner), and of others less directly in the line of the circulation, as omphalitis, uterine phlebitis (Lustig), abscess of the nasal sinuses, bones, and fistulæ (Trasbot).

In other cases the nephritis is evidently a result of the irritation caused by toxins in process of elimination by the kidneys, as there is no evidence of a nephritic infection.

In some instances minute emboli originating in the lungs or heart, become the starting point of the nephritis, which slowly extends by reason of infection or low condition and special susceptibility. Disease of the aorta or renal artery may lead to this condition as noticed by Cadeac and Lustig. Cadeac has also noticed its association with aneurism of the mesenteric arteries so that the strongylus (sclerostoma) armatus may be considered as a factor. Again in old horses and dogs it has been associated with atheroma of the aorta and renal vessels (Trasbot).

Overfeeding is not without its influence, especially when on animal food, which charges the kidneys with excreting an excess of the irritating urea and uric acid, and this is one reason why it is far more frequent in house dogs than in other domestic animals. When the meat is already decomposing and putrid there is the added evil of a quantity of toxins and even of microbes to be eliminated from the system by the much abused kidneys. Add to these that the dog's urine is even in the normal condition more dense and contains more irritating ingredients than that of herbivora, and that owing to the slight activity of his perspiratory apparatus he can obtain less relief from the skin, and we find a substantial ground for the prevalence of chronic nephritis in this animal.

Disease of the valves of the right heart or dilatation with insufficiency of the auriculo-ventricular valves is a potent cause of nephritis, the reflux of blood into the veins and the increased venous tension, speedily producing passive congestion and a slow type of inflammation in the kidney. This factor is especially liable to operate in dogs, which are particularly obnoxious to rheumatism and valvular ulceration, and are very subject to nervous cardiac disorders ; in horses that have contracted heaves ; and in beef breeds of cattle which suffer from fatty degeneration of the heart with dilatation.

The influence of calculi must not be overlooked, whether they are lodged in the pelvis, the chalices, or the uriniferous tubules. Their tendency is to induce local irritation and exudation, with fibroid degeneration and thickening of the walls of the tubules or pelvis and of the adjacent tissue.

When to one or more of the above conditions there are added overfeeding or what is worse a low condition from starvation or unwholesome food (permeated by bacteria or cryptogams or containing vegetable acids), and when to crown all there are frequent exposures to cold or wet, we have a vicious combination especially conducive to kidney trouble.

Habitual retention of urine in mares in harness, in house dogs, or in horses in railway cars, and violent exertion, or sprains of the back are among the remaining accessory causes.

Symptoms. These are often slight or obscure, so that not only owners and attendants but even veterinarians are liable to overlook them. Loss of flesh, flabbiness of the muscles and a lack of spirit and energy are among the first symptoms. The *horse* appears stiff, especially in his loins and hind limbs, and fails to advance the hind feet as far under the belly as formerly, and straddles more. When put to work he is early fatigued and appears unfit for sustained exertion. His movements are slow and if urged to a trot he may even groan with every step and quickly settles back to his sluggish pace. If turned sharply round on himself he does so with difficulty and often groans. When he is mounted or when the loins are pinched he may droop to excess. If you come on him lying down, and urge him to rise he may rise on his fore limbs and sit on his haunches until urged before he makes any attempt to raise himself on his hind. The *dog* may

spend most of his time in the kennel, and show little disposition to run, play or hunt. On the contrary the owner may have to call him several times before he will come out and then he moves listlessly, wearily and even weakly.

In all animals the appetite is poor or capricious, and the patient gradually loses condition, at first slowly and later, after a few weeks or months, more rapidly. The advance of anæmia is also steadily progressive.

Dropsical effusion is not uncommon. It is often prominent in the horse as stocked limbs, but may be absent for a length of time. In other animals it is more likely to appear later in the disease and under the chest or abdomen or in one of the internal serous cavities. Trasbot has found it absent for months in the nephritic dog.

The exploration of the kidney through the flaccid abdominal walls in small animals, and through the rectum in small horses and cattle, may reveal renal tenderness and even swelling. If there is a tendency to frequent passage of urine in small quantities, or to straining without micturition, the indication is of value.

There may be little or no fever, and, when left at rest, little evidence of discomfort.

Any indication of urinary trouble, and especially with dropsy, weakness, flabbiness and anæmia and a subnormal temperature, should lead to examination of the urine, as a crucial test. A high density is good ground for suspicion. But this is not constant. In advanced cases (chronic interstitial nephritis, small white kidney, atrophic nephritis) it may be 1015 to 1025, in exceptional advanced cases with polyuria, it may be 1010, 1005, or even 1001. With such a condition, however, there is great anæmia, pallor of the mucosæ, and prostration. Tested with nitric acid and heat, the urine throws down an abundant precipitate of albumen. Under the microscope it shows a profusion of granular, degenerating epithelial cells, and casts of the uriniferous tubes.

Progress. The course of the disease is usually slow, extending over several months, but with a tendency to constant advance. The thirst increases and the urine increases in amount, clearness and levity. There may supervene extreme sluggishness, drop-

sies, anæmia, and weakness, irritability of the heart, and palpitations on slight exertion. So long as the heart's action is strong, elimination may be maintained and life prolonged for months (in cow, Dickinson), or years (Friedberger and Fröhner). When the heart's action becomes weak, elimination is rendered imperfect and the animal shows catarrh of the lungs or bowels (common in dogs), local inflammation of the lungs, pleura or pericardium, or oedemas, or hæmorrhages. The toxic effect on the nerve centres is shown by stupor or lethargy, or vertigo. When an abcess forms it is associated with a temporary rise of temperature (Trasbot). The patient may die in convulsions, in a state of coma, or by gradually advancing debility and failure of the heart.

Lesions. In cases of comparatively **short standing** the kidney is usually of full size, or somewhat enlarged, with firmly adherent capsule and rough or even nodular surface. The surface of the cortex may be red or grayish or parti-colored, pink and gray. The cortical portion is firm and it may even be attenuated somewhat, while the medullary portion, naturally lighter, has often grayish streaks converging toward the hilus. When the gray streaks are scraped with the knife a serous fluid, mixed with fatty granules or globules, is obtained. The glomeruli may be still about the normal size with some increase of the epithelial tuft cells. The tubules contain casts (colloid, hyaline, granular), and their epithelium normally columnar, are flattened down to cubes and are swollen, granular or fatty.

In cases of **older standing** the connective tissue has usually undergone a marked increase. The capsule is thick, dense and adherent. The cortical substance is shrunken with a great increase of the fibrous elements, and the same holds true of the medullary portion. In consequence of this, even in the cortical substance the white or gray color predominates. The parenchymatous tissue (glomeruli, tubules) have greatly shrunken. In connection with the contraction of the forming fibrous hyperplasia, there is a general shrinkage of the kidney in size, it may be to one-half its original volume. Trasbot reports a case of nephritis, of 8 months standing, in the dog, with a kidney half the normal size. In the end the parenchyma may have practically disappeared, and the kidney may have shrunken to a small, firm,

white, fibrous mass. Abscess of the kidney is exceptionally met with (Laurent, Lafosse).

Lesions of distant organs are not uncommon. Bronchitis, pneumonia, pleurisy, insufficiency of the tricuspid or mitral valves, dilated heart, hypertrophied or fatty heart, congested or fibroid liver, arteritis, and dropsies are among such morbid conditions.

Prognosis. This is almost always unfavorable. Death may be delayed for months or years, and partial transient recoveries may take place but a restoration to normal structure and function is not to be looked for.

Treatment. This cannot be expected to be much more than palliative. The avoidance of overwork, and of the exposure to cold and wet, and the securing of a free action of the skin by warm buildings and clothing, are essential. The diet should be easily digested and non-stimulating, for *herbivora* green food, carrots, roots, apples, silage, with a moderate allowance of oats to counteract weakness and anæmia; and for *carnivora*, milk, buttermilk, mush made of oat, wheat or barley meal, with, if necessary, a slight allowance of tender raw meat. Tonics fill a similar need. Iron and bitters may be combined. Or hydrochloric acid or nitro-muriatic acid with bitters (nux, calumba, salicin, quassia) may be tried. These acids are especially valuable when the case has originated in or is maintained by calculi, indigestion or hepatic disorder. When the heart is defective in tone, it may be stimulated by small doses of digitalis, strophanthus, spartein, caffein, or nitro-glycerine, or to a certain extent by strychnia or nux. These, however, must be used with judgment, if it is found that they aggravate the case by increasing the arterial tension. In those cases in which there is an excessive secretion of watery urine, the possible source of this in musty aliment should be avoided, and the flow checked by nux vomica, in moderate doses, and bromide or iodide of potassium in full doses. When, on the other hand, the urine becomes scanty and dense, the great danger of a toxic action must be met by agents that favor excretion. Pure water at will is perhaps the least objectionable of such agents, but potassium or sodium acetate or citrate, or even sodium chloride, in weak solution, may be given. In some cases benefit will come from a moderate use of the balsam of copiaba, or the

leaves of buchu, which may improve the tone of the secretory elements. The most promptly effective of these agents is pilocarpin (Friedberger and Fröhner), but it has the serious drawback of inducing profuse and dangerous depletion and debility. Yet in careful hands, and with good cardiac tone, it may often be used to advantage.

Fomentations over the loins, warm baths and mustard embrocations, may at times be beneficial. Attempts have been made to check the hyperplasia by the use of arsenic, mercury or the compounds of iodine, but their use in such cases is based on theory rather than accomplished results.

HYPERTROPHY OF THE KIDNEY.

Hypertrophy of both kidneys has not been recorded in domestic animals. On the other hand the extraordinary development of one in compensation for the loss or atrophy of the other is not uncommon. In this the organ follows the general law of adaptation, seen in the double symetrical organs (testicle, etc.) and the more so that its functional activity is indispensable to life. Among *causes* are: blocking of an ureter by calculus, worms, neoplasm, nephritic abscess, gangrene, etc. The enlargement of the remaining kidney is a vicarious act and essentially a physiological one.

If compensation is perfect, it may be impossible to detect symptoms apart from those of the primary disease.

Prognosis. Life is endangered in case of any subsequent kidney disease.

ATROPHY OF THE KIDNEY.

Result of hyperplasia of connective tissues and compression and absorption of parenchyma. Unilateral or partial. Causes: chronic productive inflammation, calculus in tubes, ureter, or pelvis, tumor, retention cyst, embolism. Lesions: sclerosis of kidney, firmness, pallor, anæmia, lack of glomeruli and tubules, cysts, congenital, urinous retention, colloid. Symptoms: reduced secretion, palpation of kidney. Treatment: Prevention: arrest conditions, abundance of water, succulent food, parasiticides, operation on cysts, counteract nephritis.

Unlike hypertrophy, this is constantly the result of a pathological process. So long as a normal functional activity of the secreting elements is carried on, such parts must maintain their size and healthy characters. But with the compression of such secreting elements (glomeruli and convoluted tubes) by a hyperplasia of connective tissue, by pressure from without or from the damming back of the urine in the pelvis and tubes, the secretory elements are absorbed and removed, and the final result is a general atrophy. If such atrophy appears in both kidneys at once it can only be very partial in extent, as extreme atrophy of both, with loss of their secretory function, would entail poisoning and death from the retained urinary products. The comparative frequency of the disease may be inferred from the reports of the numbers of specimens found by Barrier and Moussu in old horses in the dissecting rooms. The latter observed a dozen cases in a single winter, other examples are recorded by Cadeac (horse), Soula (swine) and Trasbot (in various animals).

Causes. The most common source of the condition is the occurrence of chronic productive inflammation. The new product in such cases, if not pus, or a growth that rapidly passes into fatty or granular degeneration, or into gangrene, tends to form tissue of a low organization, especially fibrous. The resulting increase of the fibrous trabeculæ, in undergoing subsequent contraction necessarily compresses the secretory tissue and the final result is a visible and, it may be, extreme wasting. Hence any slowly advancing productive inflammation is liable to result in absorption and removal of the kidney parenchyma, and distinct atrophy of the gland.

Again the obstruction of the ureter by a calculus in the pelvis which falls into the infundibuliform entrance, or a stone arrested at any part of the duct (or even of the urethra) or by worms, hydatids, cysts or tumors, throws back on the kidney the secreted urine, which distending the pelvis and uriniferous tubes leads to direct compression and absorption of the secretory parenchyma. Direct compression of the kidney by an adjacent tumor will act in a similar manner. Retention cysts by their gradual increase and augmenting pressure cause absorption of the gland tissue.

The blocking of individual uriniferous tubules by minute calculi, which is so often seen in cattle, kept on dry feeding in winter, is a cause of partial nephritis, and absorption, as noted by Röll.

A somewhat rare cause of atrophy is the diminution of the blood supply by arteritis and embolism of the renal artery, or by pressure of tumors on that vessel. Arteritis and blocking suggests at once the possible agency of the strongylus (sclerostoma) armatus in the horse. Trasbot records a striking instance of compression of the renal artery and kidney by an enormous sublumbar melanoma. This occurred in an aged horse and led to atrophy.

Lesions. In cases due to productive inflammation with sclerosis of the kidney, the firmness, pallor and bloodlessness of the organ is a marked feature. When incised it is found to be composed mainly of fibrous tissue, while the glomeruli and tubuli have to a large extent disappeared.

If there has been simple lack of circulation the kidney becomes flaccid, pale and small in size. The secretory elements (glomeruli and uriniferous tubes) are first absorbed, leaving the fibrous network, which tends to shrink and form a hard resistent mass. In extreme cases there may be absolutely no glandular tissue left, and the dense shrunken mass represents only the hyperplasia of the original fibrous network. In the different successive stages of this process the glomeruli and tubules become flattened, the epithelial cells become granular, or contain colloid casts and refrangent elements like oil globules and finally they are represented by a small mass of fibrous material.

Of all the atrophies caused by the pressure of tumors perhaps

that caused by cysts is the most characteristic. There may be a single cyst or they may be multiple ; they may range in size from a pea to the size of the two fists the total size exceeding that of the normal kidney. In all such cases the cysts project visibly from the surface of the organ. They vary according to their origin and nature. Congenital cysts are said to have resulted from distension by retained urine of the capsule of the glomerulus. The arterial tuft is atrophied and flattened against the wall. Serous cysts with clear contents are found in the old. Urinous cysts again form by distension of the tubules that are obstructed by cysts or minute calculi. Colloid cysts are found in certain forms of nephritis formed by the dilatation of the capsule of the glomerulus or of the uriniferous tubules. The liquid often contains leucin, tyrosin and cholesterine. In all such cases the walls of the cyst become thick, and the glandular parenchyma is compressed leading to progressive degeneration and atrophy.

Symptoms of atrophy of the kidney are necessarily those of suppression of urine, with, in certain cases, the passage of casts of the uriniferous tubes and of crystals of salts. There are, however, no absolutely pathognomonic symptoms. When the kidney can be reached through the flaccid walls of a comparatively empty abdomen, or through the rectum, its hard, shrunken condition may assist in diagnosis.

Treatment is not successful in advanced cases. *Prevention* is to be sought by obviating or treating the conditions on which the atrophy depends. Nephritis must be treated on general principles. Calculi must be avoided by a liberal supply of water, by soiling, or by pasturage. Strongylus parasitism should be dealt with by destroying the parent worms in the bowels, and by securing pure drinking water free from their eggs and embryos. Cysts, and tumors are only amenable to surgical measures and not often open even to these.

FATTY DEGENERATION OF THE KIDNEY: STEATOSIS OF THE KIDNEY.

Causes: age, overfeeding, idleness, atony, retention of urine. Lesions: kidney enlarged, pale yellow, capsule loose, cut surface glistening unctuous, oil globules in scrapings, granules soluble in ether. Symptoms: in idle, overfed, obese, improved meat producing breeds, closely confined, starchy or saccharine food, fatty granules in urine, finally dropsies, anæmia, debility, sluggishness. Prognosis unfavorable in advanced stage. Treatment: butcher, restricted regimen, open air exercise, nitrogenous diet, crossing, diuretic food or drugs, oil of turpentine, balsam copiaba. Palliation only.

Fatty degeneration of the kidneys is by no means unknown in the domestic animals. It has been observed in dogs and cats (Rogers, Gonbaux, Vulpain, Trasbot). In dogs it has been erroneously set down as a characteristic lesion of rabies. Like fatty degeneration of other organs, it is also met with in old and over-fed individuals of meat producing breeds of animals, in which the tendency to early maturity and rapid and excessive fattening has been fostered from generation to generation. In man small, granular, fatty kidney is a common result of chronic parenchymatous nephritis, and often coincides with fatty liver. Chronic poisoning by arsenic or phosphorus is another cause, as it is of fatty degeneration in other organs.

Vulpain has attributed it to a lack of active exertion and of general tone, associated with excessive amylaceous feeding, sluggish, shallow breathing and tardy elimination. Gonbaux and Trasbot attach great importance to the compulsory retention of urine in house dogs, cats and horses. The damming back of the urine in the convoluted tubes and glomeruli, temporarily arrests secretion, and the inactive and compressed cells tend at once to granular and fatty degeneration.

Lesions. The gland is sensibly increased in size, and pale, yellowish or straw yellow. The capsule is easily detached from the cortical substance, contrary to what is the case in chronic productive inflammation. The cortical substance is increased in thickness, and pale, the pallor being largely in ratio with the duration or extent of the fatty degeneration. The cut surface may be glistening and unctuous to the touch. It is softer than usual,

rather friable, and if scraped, furnishes a serous or grayish pulp in which oil globules are prominent features, together with granular epithelium and free granules that dissolve readily in ether. Tubules are varicose and unequal at different parts. The medullary portion has undergone little change. It may be paler at certain points, with some shrinking of its substance and increase of firmness.

Symptoms. As a rule the disease occurs in pampered, overfed and obese animals, and in those of the improved breeds which have great power of digestion, assimilation and fattening. It is especially to be looked for after close confinement on full, stimulating, amylaceous diet. Symptoms are not usually recognized during life. There is, however, a lessening of the urinary secretion, and, as the disease advances, albuminuria. When examined microscopically this is found to contain characteristic elements, such as granular epithelial cells, the granules soluble in ether, oil globules, and at times crystals of cholesterine (Beale). A diagnosis based on the mere presence of oil globules may, however, be fallacious, as these may be present in animals that have just been heavily fed on oleaginous food, and again the oil used to smear the catheter may float in the urine and prove misleading. Under such circumstances vaseline or glycerine may be substituted on the catheter. Scriba induced fatty urine by injecting fat or oil emulsion into the blood, and Chabrie by ligating the large intestine. Trasbot says that cylindroid casts may be present. As in other grave kidney affections, dropsies supervene as the disease advances. These may show in the limbs, in the abdomen, or in other serous cavities. A steadily advancing anæmia with pallor of the mucosæ, listlessness, weakness, debility and sluggishness are to be noted.

Prognosis. Since the disease is rarely diagnosed until it has reached an advanced stage, it usually progresses steadily to a fatal issue. If, however, it can be detected at an earlier stage, it may be palliated, or held in abeyance, for a length of time varying with the extent of the lesions. As it is very largely a disease of meat producing animals and as the subject is at first in a condition of marked obesity, it can usually be turned over to the butcher without material loss.

Treatment. If the disease has resulted from the inbred pro-

pensity to fattening, the family that shows the disposition must be subjected to a somewhat different regimen, open air exercise must take the place of confinement in warm stables, a rather bare pasturage is valuable for herbivora, and a restricted diet in which the oleaginous, saccharine, and amylaceous constituents do not predominate, is strongly indicated. Crossing with a strange male having many of the desirable qualities of the herd, but which is more vigorous may be resorted to. When the secretion of urine becomes scanty an abundance of pure water, or a diet of succulent grass or roots or ensilage or even small doses of alkaline diuretics may be resorted to. Any source of arsenic or phosporus poisoning should be cut off, and as an antidote to phosporus, oil of turpentine may be given in small doses. This agent may, indeed, replace the alkalies as a diuretic, bringing in an element of tone for the mucosa which is not to be despised. Or balsam of copaiba or buchu leaves may be substituted.

When the small white kidney (granular, fatty) results from chronic nephritis, the prevention and treatment would be as for that disease. Little hope is to be entertained of entire restoration to health.

AMYLOID KIDNEY. LARDACEOUS OR WAXY KIDNEY.

This condition of the kidney has been found in the ox (Gerlach) and dog (Rabe, etc.). There are usually similar degenerative lesions in the liver, pancreas, intestines and other organs. It is usually a concomitant of some chronic wasting disease (chronic nephritis, tuberculosis, etc.).

Morbid Anatomy. The kidney is usually enlarged, pale and on section waxy or glistening. Soaked in dilute compound tincture of iodine it shows spots of a walnut or mahogany brown color. The glomeruli are well marked and show the earlier changes, later the tubes do so excepting the epithelium. The latter is swollen, granular, fatty.

Symptoms. There may have been those of chronic nephritis. Rabe has noticed in dogs dropsy of the limbs, ascites, emaciation, anorexia, followed by uræmia, coma, weakness, vomiting, and if the kidney alone was affected great lowering of temperature

(35.9°C). With hepatic complication there was greater weakness, giddiness, and higher temperature (39.6°C). Urine is usually increased (in man albuminous) and the casts have shown the anyloid reaction. They tend to be fatty or finely granular. Casts may, however, show anyloid reaction when the kidney, post-mortem, does not (Jaksch).

Diagnosis from Bright's disease is often impossible.

Treatment is essentially the same as in chronic nephritis, and is not hopeful.

Trasbot recommends KI 3 to 7 grs., or tinct of iodine 3 drops for shepherd dog. Ol. terebinth and alkaline diuretics are also commended.

RENAL CALCULUS.

This is much more common than is supposed. Small calculi formed in the tubuli uriniferi of cattle on dry winter feeding often pass without recognition, and habitually disappear on rich spring and summer grass.

If retained in the pelvis until increasing size forbids their passage through the ureter they form pelvic calculi.

If retained in the bladder so that they cannot enter the urethra they form cystic calculi.

Pelvic calculi or concretions are often (in cattle and swine) mere scales lying in chalices. They may fill the whole pelvis and send branching processes into chalices.

Causes. They are attributed to phosphaturia, lithæmia or uric acid diathesis, oxaluria, etc. In cattle they are associated with dry feeding and are common on all *magnesian* limestone soils. There are usually catarrh of the kidney and the presence of bacterial ferments and colloids (pus, albumen, etc.). (Sharing and Ord.) Calculi or gravel is preceded by renal catarrh, but this is aggravated by the crystalline deposit. Bacteria act also in producing NH_3O, which instantly precipitates ammonio-magnesian phosphate. Retention of urine greatly favors the precipitation.

Symptoms. A white or brownish yellow deposit in the last urine discharged collects on the floor. Cloudy urine. Passage of crystals—round—or angular. Colic. Lameness in one or both hind

limbs. Arched back. Sensitive loins. Pain paroxysmal. Attempts to urinate. Little passed but often with drops of blood. Sudden relief when the calculus enters the bladder.

Retained in the kidney it may cause no suffering in meat producing animals, but in horses it usually causes stiffness or lameness especially under violent effort. Also hematuria ; blood globules are found in the deposit when placed under the microscope. There may be sepsis and specially cloudy offensive urine.

Diagnosis : May be confounded with renal tuberculosis, or sarcoma or oxaluria. Examine for bacillus, small cells, or oxalate of lime or oxalic acid.

Prophylaxis. In the early stages give succulent, watery food, ensilage, roots, potatoes, spring grass, and water ad libitum.

Treatment. Salt may tempt the patient to drink. Nitro-muriatic acid is a solvent and antiseptic. Or alkalies with salicylate of soda. Also tonics. Quiet pain by morphia and other anodynes. Use piperazine.

These failing, an operation on the kidney may be considered.

HYDRO-NEPHROSIS.

A common result of calculus or other obstruction, causing increasing pressure of urine in the pelvis and absorption of the parenchyma, and finally leaving a mere urinous sac.

RENAL TUMORS.

1. *Non-malignant :* Fibroma.
   Lipoma.
   Angioma.
   Adenoma.
   Papilloma.
2. *Malignant :* Sarcoma.
   Carcinoma.

RENAL PARASITES.

Echinococcus : Herbivora, Omnivora.
Bilharzia Crassa : Egyptian cattle.
Strongylus Gigas : Horse, ox, dog, man.
(Cysticercus Tennicollis : Ruminants : Pig).
Tænia serrata : Dog. Pelvis.

Sclerostoma equinum : (renal arteries, *kidney pelvis*), soliped.
Stephanurus dentatus : Pig, (pus cavities).
Trichosoma plicata : (Urinary bladder), dog.
T. felis : (Cat), bladder.
Indetermined embryos : Kidneys, dog ; small tumors.
Cytodites nudus : Kidneys ; hens.
Œstrus, (Gast. Hemorrhoidalis) : Bladder walls? horse.
Mucorimyces : Kidneys ; dog.
Coccidia : Kidney, Horse, dog, goose.

INJURIES OF THE URETERS.

Lesions by bullets, arrows, stabs, bruises and lacerations in parturition, treads, wheels, tumors, ulcers, calculi, tubercles, parasites. Course. Pathology : transverse division may cause hydronephrosis, or septic peritonitis. Symptoms : uncertain, traumatism, bloody urine, arched, stiff, tender loins, straining, recumbency, groaning in turning or rising, rectal palpation of distended ureter, of ascitic fluid, pitting on pressure of loin, flank or groin, liquid drawn through a cannula is urinous, urine still discharged by normal channel. Crystals in urine, worm ova. Treatment : compresses, fomentations, sinapisms, anodynes, balsams, antispasmodics, extraction of calculus, lateral implantation of urethra.

From their deep and protected position it might be plausibly concluded that the ureters were secure against every kind of traumatism, This however, is not the case, since in both man and animals they have been known to have been injured by bullet wounds, arrow wounds, and stab wounds of various kinds. In distokia with laceration of the womb, vagina or bladder the ureter is liable to be injured. By blows and kicks it may even be ruptured or torn across, and also by sudden and severe mechanical compression of the abdomen as when run over by a wagon or trodden on by a horse, ox, or other large animal. Tumors of various kinds may grow in, or press upon the ureter, ulcers with thick indurated margins or base may obstruct the passage, or calculi, or worms may block and give rise to overdistension and even rupture. Kopp describes obstruction by multiple calculi with saccular dilation in front, close to the kidney in a cow. Cadiot records cases of thickening of the mucosa by

numerous cysts as small as hempseed. Intra-abdominal tumors of the spermatic cord have been known to block the passage. Again tubercles have formed on the urethra, and polypi on the trigonum vesicæ have blocked the ureter and produced all the evil consequences of calculus, parasites, etc.

Course. The progress of the disease will vary greatly according to the nature of the lesion. With complete rupture of the ureter the urine as a rule escapes into the peritoneal cavity. If the urine is aseptic this may not lead to serious results and the edges of the wound cicatrizing, the urine is imprisoned in the ureter and pelvis of the kidney, and leads to final atrophy of the kidney and hydro-nephrosis. When on the other hand infecting matter escapes with the urine, as in perforating ulcer, tubercle, glanders, cancer, infective catarrh of the kidneys or ureter, such infection is brought in contact with the whole peritoneal surface, where secondary infections follow. In cases associated with penetrating wounds, wounds resulting from dystokia, calculous or parasitic obstruction, similar infection is to be dreaded.

The *symptoms* are by no means clear, unless the injury result from external traumatism, or when it can be detected by rectal exploration. There may be blood staining of the urine, stiffness of the loins, lameness in one hind limb, tenderness of the lumbar vertebræ and of their tranverse processes on pinching, frequent straining to pass urine, a disposition to remain recumbent, pain and groaning when rising, or when turned in a narrow circle. Under rectal examination the blocked ureter may sometimes be recognized, its cystic end shrunken and empty, up to the seat of obstruction, and its renal end, from the hypersensitive seat of obstruction forward, full, rounded, elastic and firm. If the ureter has been ruptured, it may be impossible to distinguish it, but the presence of urine free in the peritoneum may be detected through the rectum as it may often be through the abdominal walls. Sometimes the urine infiltrates the subperitoneal connective tissue, and forms a pasty swelling on the loin or flank. In either case a hollow needle inserted will draw off a liquid having an urinous odor. That this urine has escaped in front of the bladder may be safely inferred from the continued discharge of urine by the urethra and by the absence of cystic swelling, heat and tenderness. If the kidney can be reached by rectal exploration it is

felt to be firm and resistent up to the period of rupture of the ureter.

In case of obstruction by calculus, crystals and even small calculi may be passed in the urine, there is usually a history of previous attacks of renal colic, and the suffering is manifestly extreme. In case of worms (strongylus gigas) the use of the centrifuge on the urine, may possibly secure in the sediment specimens of its ova. The existence of tumors or tubercles can usually be clearly made out.

Treatment will be as varied as the lesion. Simple ureteritis may be met by wet compresses, sinapisms, and internally by balsams and anodynes. Calculi and parasites may be passed with some aid perhaps from fomentations, antispasmodics, and diluents. Obstinate cases can only be successfully met by surgical interference. The resulting wounds in the ureter, like ruptures, perforating ulcers and strictures may be met by Van Hook's "*lateral implantation*": the divided cystic end of the ureter is ligatured and on the cystic side of the ligature a longitudinal incision is made, large enough to admit the divided extremity of the renal portion, and through each of the two sides of this last a fine ligature is passed; these ligatures are then passed into the lumen of the cystic portion of the ureter through its longitudinal incision and brought outward through its walls; next the renal end is inserted into the incision in the cystic end and the two are firmly sewed together by the two ligatures. When a small portion of an ureter must be excised it may sometimes be possible to save a valuable animal by such an expedient.

In some cases of rupture into the vagina or uterus an available fistula may sometimes be established into one of these.

URETERITIS.

From wounds, calculus, parasites, infection, injuries in parturition. Symptoms: in wounds of ureter. Course: danger of infection of kidney or bladder. Treatment: for calculus, antispasmodics, anodynes, fomentations, for parasites arsenious acid, for catarrhal conditions, balsams, buchu, salicylates, etc. Operation. Uretero-vaginal fistula.

This may arise from the passage of a rough calculus, from wounds of the ureter sustained in kicks and blows or by being run over by wheels (dogs, cats), it may be due to the blocking of the tube by a parasite such as strongylus gigas, echinococcus, etc., or it may be the result of extension of an infectious inflammation backward from the kidney or forward from the bladder. Again it may be the result of a lesion of the ureter in cases of dystokia.

The *symptoms* are obscure but there is likely to be frequent straining and passage of urine, tenderness of the loins, all the more significant if confined to one side, lameness or halting on the corresponding hind limb, and on examination through the rectum the swollen and tender cord representing the ureter may be recognizable. In case of calculus or other obstruction the ureter may be felt to be swollen, elastic and tender back to a slight nodular, painful, firm swelling at the seat of obstruction.

Course. In all such cases there is always danger of inflammation (infections or otherwise) of the kidney with degeneration and loss of structure and function, the organ being reduced to a simple urinous cyst (hydronephrosis). In some cases, however, the obstruction (calculus, parasite) may escape into the bladder and a recovery follow. Slight infections, too, may improve and advance to complete convalescence.

Treatment will depend much on the causative factor: Calculus must be treated by anodyne anti-spasmodics, and fomentations, and in case of relief by measures calculated to prevent its formation anew: parasites may be treated by arsenious acid, oil of turpentine, and other parasiticides which are secreted by the kidneys: catarrhal and infected conditions may be met by balsams, buchu, salicylic acid and even peppers. In case of calculus which does not give promise of passing, even a surgical operation may be thought of, especially in the smaller house animals.

In rupture of the ureter in dystokia the walls of the womb or vagina have usually suffered, and a recovery with a uretero-uterine or uretero-vaginal fistula is not unknown.

ACUTE CATARRHAL CYSTITIS.

Acrid diuretics, by mouth or skin, microbian infection, retention of urine, urethral calculus, parasites, spasm, enforced suspension of micturition, unclean catheter, adjacent infection, chill. Lesions: hyperæmia of mucosa, thickening, vascular distention, clouding of epithelium, muco-purulent secretion, alkaline fermentation, ammonia, liquefaction of cells, erosion. Symptoms: Slight fever, stiff, straddling gait, urine scanty, cloudy, alkaline, penis or clitoris semi-erect, smearing of tail or prepuce. Crystals of triple phosphate. Treatment: Antiseptics, boric or salicylic acid, gum arabic, astringent antiseptics, laxatives, flax seed, slippery elm, anodynes, diluents, piperazine, drainage, rest, restricted laxative diet, warmth, avoid stimulants.

Causes. Cystitis is caused in all animals by irritant diuretics like cantharides, copaiba, or oil of turpentine given by the mouth or applied to an extensive cutaneous surface. It is an error, however, to conclude with Williams that this is the sole cause. The very existence of calculi virtually implies bacterial infection, and fermentation. The presence of free ammonia in the urine usually implies fermentation, and fermentation must be looked upon as practically synonomous with microbian invasion. That bacteria may be present without serious injury is undoubted. The protective power of the healthy mucosa is very great. But when the mucosa is weakened, microbes that would otherwise be harmless, find a ready infection atrium, and triumph over the weakened tissues. Hence retention of urine and overdistension of the bladder as in urethral calculus, blocking of the urethra by a parasite, spasm of the sphincter vesicæ, compulsory retention as in the mare in harness, the dog kept indoors, or in railway car on a long journey, or in mares so travelling, may become the occasion of cystitis. Even in cases in which no microbe is present at first, this reaches the bladder by the introduction of an unclean catheter, or by extension from an uretheritis, vaginitis or metritis, or even from a peritonitis, or infected urachus. Or the infection

may descend from a suppurating kidney. Another occasion of microbian invasion is the congestion which attends on exposure to cold.

Lesions. Hyperæmia of the cystic mucosa, with dilation and tortuous deviations of the larger vessels, thickening of the membrane, and distension and clouding of the epithelial cells, with a thick covering of tenacious mucus containing epithelial, pus, or white blood cells. As the disease advances epithelium is desquamated abundantly, and degenerates with production of free nuclei and pus. Along with these are microbes, usually the bacillus coli communis, and various cocci. In the fully established disease there is liable to be alkaline fermentation, and the liberated ammonia dissolves the epithelial cells, leading to extensive desquamation and raw granulating surfaces, so that the disease tends to run in a vitiating circle, the alkali dissolving the epithelium and increasing the microbian development and fermentation, which in its turn produces an increasing quantity of ammonia.

Symptoms. There is slight hyperthermia or none, stiff or straddling gait, frequent passage of urine in small quantities and cloudy, or straining without passage, the penis or clitoris is semierect, eversion of the lips of the vulva is frequent, and the bladder is tender (through prepubian wall, vagina or rectum). If a finger is inserted into the bladder in the mare the thickening of the walls can often be recognized. The urine often contains precipitated crystals of ammonio-magnesian phosphate, and even clots of blood. It has an alkaline reaction even in herbivora.

Treatment. The danger centres around the bacteridian fermentations, and a main object must be to disinfect the bladder. This will be all the more effectual if the lotions used are of an acid reaction. Thus boric acid or salicylic acid in 3 per cent. solution, injected after evacuation of the bladder and repeated a number of times a day may soon establish a healthy action. If the bladder is especially irritable a boiled weak solution of gum arabic will form a suitable medium. Other antiseptics are often used as creasote (0.5 : 100), carbolic acid (3 : 100), chloride of zinc (3 : 100), chlorate of potash (3 : 100), mercuric chloride (1 : 5000), silver nitrate (0.5 : 100), or astringents are often better: PbA, $ZnSO_4$ tannic acid, ferri chloridi in dilute solution so as not to cause pain.

The bowels should be kept open by an occasional saline laxative, pain moderated by codeine, and abundance of pure water and a laxative diet enjoined. Linseed tea, and infusions of slippery elm or marsh mallow have long been employed, and by soothing and relaxing the bowels they act favorably on the urinary mucosa. Stimulants of the urinary track like buchu, uva ursi or copaiba in small doses, or antiseptics like creosote, boric acid, salicylic acid, peperazine, are available in slight cases or when the acute symptoms have subsided somewhat. With prior infection of the kidneys, the latter may be used. Constant drainage may be necessary to avoid distension.

Perfect rest is absolutely essential, a restricted laxative diet, and a careful avoidance of cold, and stimulants.

When urine is retained it should be removed with a thoroughly aseptic catheter.

In case of blood clots in the bladder, wash out with a boiled normal salt solution.

ACUTE CROUPOUS CYSTITIS.

This has been found to follow the use of cantharides and other irritant diuretics, and to follow on certain specific diseases. Its nature is that of catarrhal inflammation, but with a fibrinous product or false membrane formed more or less extensively on the inflamed mucosa.

Symptoms are essentially those of catarrhal cystitis from which it is distinguished by the presence in the urine of flakes of the fibrinous membrane.

Treatment is essentially the same as in the catarrhal form, to which may be added the injection of a solution of 4 grains scale pepsin to the ounce of sterilized water. The boric acid solution may be of the strength of 20 per cent. Irrigate two or three times a day.

CHRONIC CATARRHAL CYSTITIS.

This may begin as such or it may continue after an acute attack. It has been noticed in horse, ox, and dog.

It may be associated with calculi, gravel, papilloma, and bacterial invasion especially by the colon bacillus.

Lesions. The mucosa and muscular coat are thickened, corrugated, puckered and contracted so that the bladder will not contain more than a few ounces of urine. The surface of the mucosa is discolored, mottled and variegated, slaty blue, brown, red, purple, or even black, with ulcers, encrustations of triple phosphate, and fungoid elevations. In dogs especially, the prostate is often enlarged.

Symptoms. Frequent urination accomplished with pain, groaning, or whining and it may be with sudden arrest. There may be incontinence, the urine dribbling almost continuously from the penis or vulva and in the latter case trickling down the thighs. The presence of pus and mucus tends to mat the hairs, and a strong urinous and ammoniacal odor is emitted.

Palpation of the prepubian region often, and of the vagina or rectum always causes pain and wincing. Temperature is normal.

Urine is albuminous in ratio to the amount of pus, or above that, and is then suggestive of kidney disease and likely to be complicated by casts.

Complicating lesions of the womb, vagina, prostate, and kidney are to be carefully looked for, also cystic papilloma.

Prognosis. Recovery though not uncommon is too often but partial and it is usually desirable to fatten the animal.

Treatment. Rest, moderate laxative diet, pure drinking water ad libitum, warmth, antiseptic irrigation.

CYSTITIS IN THE OX.

Special Symptoms. Beside general disorder there is a disposition to decubitus, but with frequent rising to urinate though the bladder is not filled to repletion. Then the urine is passed in a slow stream by abdominal contraction, and without pulsating contractions of the urethra at the ischium which are so marked in calculus. Cystitis is greatly aggravated by overdistension, and if the bladder is paralyzed is very liable to go on to rupture.

Galtier considers enzootic hæmaturia as essentially a hæmorrhagic cystitis, due to marshy soils, disordered liver, often distomatosis, and irritation of the urinary organs by the poisons which the liver was helpless to destroy or eliminate.

The *treatment* of cystitis in cattle does not differ materially from that of the horse.

The hæmorrhagic form demands prevention by drainage, cultivation and the use of phosphates to the soil.

CYSTITIS IN DOGS.

The *special symptoms* in dogs are uneasiness and frequent changes of place. The patient passes urine often in small quantity, and with whines or cries. He walks slowly and stiffly with the back arched, and compression of the abdomen and especially of the prepubian region is painful to a marked degree. The tense elastic bladder may often be distinctly felt through the abdominal walls. The inflamed bladder is liable to paresis and paralysis with great overdistension, and aggravation of the general symptoms, the eyes sunken, and dullness, stupor and coma betraying uræmic poisoning. Some claim rupture of the bladder as is so common in the ox.

In the main, *treatment* is as for the horse. Rest, warm bath, or fomentations, catheterism with aseptic catheter, draw urine through hypodermic nozzle in prepubian region. Antiseptics: boric or salicylic acid by the mouth and bladder. Laxatives, and

plenty of water are important. Free access to open air where the animal can urinate, is very essential. In chronic cases, buchu, copaiba, balsams, or piperazine may be employed. Mustard blister. Electricity. Small doses of belladonna to give tone to the bladder.

ATONY AND PARALYSIS OF THE BLADDER.

Causes. This comes usually from troubles of innervation. Paraplegia, dorsal and lumbar fractures with injury to the spinal cord, brain lesions, hæmoglobinuria with effusion pressing on the cystic plexus, overdistension of the viscus, from cervical spasm, urethral stricture or calculus or parasite (strongylus gigas), acute or chronic cystitis. In dogs it may come from obstruction by enlarged prostate. Polypus blocking the cervix and chronic disease of the walls of the organ are additional causes.

Symptoms. More or less complete retention of the urine. The bladder cannot be completely emptied except by powerful contractions of the abdominal muscles. Habitually it may escape in drops, or in jets at intervals during exercise. Palpation will show overdistended bladder as a tense, elastic mass. But as overdistension may occur without paralysis, no case can be certainly diagnosed without catheterization to show that the urethra is free.

When the paralysis affects the cervix, the urine escapes continually and trickles down the insides of the hips in mares, or from the sheath in males.

Diagnosis demands catheterism and rectal examination.

Complication. Cystitis by retention. Infection by catheter.

Treatment. Corresponds to causes. These corrected, use aseptic catheter often. After extreme distension empty partially, or inject a few ounces of borax or boric acid solution. Thus avoid collapse and inflammation, and secure antisepsis. Give tone by a course of strychnia, (ergot, belladonna), mustard blister; turpentine in small doses. Better electricity, 1 pole in bladder, 1 on pubic symphysis. Apply for 5 minutes.

TUMORS OF THE BLADDER.

Recorded cases of sarcoma (Mauri), carcinoma (Cadeac, Hink, Friedberger), and tuberculosis can be adduced. Papilloma is perhaps as frequent as any of the above. The author has treated two cases of papilloma in mares complicated with multiple small calculi and gravel. *Treatment* is exclusively surgical and in the mare with the widely dilatable urethra this is sometimes possible through that channel. In the same animals *diagnosis* may be accomplished by introducing the finger into the urethra and bladder. In other females and males, vaginal or rectal palpation must be resorted to.

VESICAL PARASITES.

Eustrongylus gigas. Dog, horse, man.
Trichosoma plicata. Dog.
Œstrus Hæmorrhoidalis. Mare.

SPASM OF NECK OF BLADDER.

Spasm of the cervix vesicæ has been doubted, save as the result of local inflammation, yet it not uncommonly takes place in horses and other animals in connection with irritation attendant on the retention of urine during work, or in dogs during a period passed indoors. Trouble and suffering continues, with ineffectual efforts to micturate, but practically complete relief is secured by catheterization or by a spontaneous abundant discharge. The neurosis which leads to it is produced or aggravated at times by enlarged prostate, or lesions in the urethra. Intense fear may cause it.

Treatment. Remove cause. Give antispasmodics, valerian, musk, bromides, chloral hydrate, opium, stramonium, hyoscyamus, codeine, etc., may be given as injections or suppositories. For the horse spread the litter and soothe by whistle or song. Or use the catheter and correct any local irritation.

RUPTURE AND LACERATION OF THE BLADDER.

This occurs most commonly in oxen from obstruction of the urethra by a calculus. Similar obstruction in the horse causes most acute symptoms, calling for immediate relief, and rupture is a comparatively rare occurrence. Peuch mentions a case resulting from a fall during an attack of colic, and with a full bladder. It has happened during lithotrity, or lithotomy, and even during parturition, Perforation by parasites has been noted and in one case by an osseous tumor of the pubic symphysis. In horses a fatal result is prompt, in cattle from 6 to 48 days.

Treatment surgical.

EVERSION OF THE BLADDER.

This is really invagination into the female urethra and bladder. It has only been seen in mares, and then by reason of the extreme dilatability of the urethra. A pyriform, red, perhaps rugose tumor shows between the lips of the vulva, during straining. It is covered by mucosa, and on its upper surface near to its neck are two small orifices from which urine oozes or comes in jets during active expulsive efforts. It soon becomes mucopurulent on the surface, and even excoriated. Urine escaping continuously trickles down the thighs with much fœtor. It occurs especially during violent expulsive efforts as in parturition or constipation.

Treatment essentially surgical consists in uniform compression to expel blood and exudate followed by the pushing of the fundus through the bladder and urethra. The more recent the case, the easier is the process. Presseeq claims to have cured an obstinately recurring case, by cauterizing the urethra up to the cervix vesicæ with a round iron rod an inch in diameter. The resulting loss of substance, with the neoplasia and constriction effectually prevented renewed eversion even during parturition. Other veterinarians have successfully excised the bladder, but this entailed incontinence and constant offensive soaking of the thighs with urine.

HERNIA OF THE BLADDER.

This is commonly seen in the mammalian female in connection with rupture of the floor of the vagina during dystokia. It has also been observed without such lesion in both male and female dogs and horses, the bladder forming a cystocele of the vagina, or bulging between the anus and the ischium.

Diagnosis is confirmed by careful palpation through the rectum. The folding of the bladder backward obstructs the exit of urine.

Treatment, essentially surgical, might include replacing of the organ and suturing of the wound, or, in the absence of a wound, evacuation of the bladder by a hypodermic needle, and replacing by palpation through the vagina or rectum. Sometimes suture of the vulva is desirable.

ANOMALIES OF THE BLADDER.

Persistent urachus. Seen in the new-born and mainly in males. Antiseptic closure is essential after having ascertained that the urethra is pervious.

Imperforate cervix vesicæ. A case reported by Lapotre, in a calf, had no cervix, and the ureters were blocked by pea-shaped nodules.

Recto-vesical fistula. In a calf 13 days old the rectum opened into the bladder and the fæces and urine escaped by a pervious urachus. (Kaufmann and Blanc).

URETHRAL ANOMALIES.

Impreforations. In the new born male, foal, lamb, etc. Usually at the outer end, and it may be for some distance back. In one case the sheath was firmly adherent on the wall of the abdomen, thus shutting off all exit of urine. If the canal is absent only at

the orifice or for a short distance, the urethra beyond this can be felt full of liquid and fluctuating. The patient being properly fixed a fine trochar is pushed from the end of the penis into the blind end of the urethra, which will be ascertained by the overcoming of resistance. The trochar is now withdrawn and the urine flows through the cannula. A catheter or sound is now tied in the passage to maintain it pervious until cicatrization shall have taken place.

Hypospadias. Short urethra opening backward on the lower surface of the penis. Considered irremediable.

Epispadias. Urethra opening on the upper surface of the penis. Much more rare.

ACUTE URETHRITIS. CATARRH OF THE URETHRA.

This occurs in all genera of domestic animals, and may be either acute or chronic. It is most common in the entire males, not only because of infections sustained in copulation, but because frequent erection exposes the opening of the urethra to injury and inflammation, and to the entrance of pathogenic germs.

Symptoms. Pruritus of the penis, and difficulty and pain in urination and straining are frequent, but a single small jet may be all that is passed at a time. The papilla on the end of the penis is red and angry and somewhat swollen. Later a few drops of muco-purulent fluid may be pressed from the orifice. In the bull, dog and boar this oozes from the retracted penis into the sheath, so that a collection is found in that canal, and the mucosa becomes infected causing a balanitis.

The infection may be conveyed from male to female and *vice versa*. Dr. Horand of Lyons even found that the muco-purulent discharge of gonorrhœa in man caused an urethral catarrh in the dog, which however did not persist for any great length of time.

Diagnosis is based on frequent and painful urination in jets, tenderness of the urethra under palpation or catheterization, redness and swelling round the urethral orifice, and the oozing of pus. In the absence of any external injury one should always ascertain if cystitis is present.

Prognosis is favorable under appropriate treatment. Spontaneous recovery will usually occur early.

Treatment. Dilute the urine. Give pure water to drink at will, or flaxseed gruel, or gum or barley water. Alkaline carbonates. In the early stage foment and use injection of potassium permanganate (2 grs. to 1 oz.). Later may be used more astringent agents (boric acid 1 : 100, zinc sulphate 1 : 100, lead acetate 1 : 100, potass chlorate 3 : 100. In the presence of great pain cocaine muriate 2 : 100). There is danger of stricture from the stronger astringents or caustics in the early stages before suppuration. To complete the cure give copiaba, buchu, resin, or essence of turpentine.

WOUNDS OF THE URETHRA.

Actual wounds occur in surgical operations, or accidentally as by shafts, poles, forks, hooks, bites, etc., or from calculus or a catheter forced into a false route. An arrested or slowly moving calculus has been known to induce several perforating ulcers causing infiltration of urine and infecting germs into the connective tissue. This determines rapidly increasing œdematous fluctuating tumors. Gangrene and septic intoxication are common results, especially in cattle.

Longitudinal wounds keep more open and heal more readily than transverse wounds, probably because the circular muscular fibres in contracting, pull the edges apart and counteract stricture, the breach being filled up by granulations. The perineal wound in lithotomy will heal thus in 20 days, while that made in amputation of the penis is exceedingly liable to circular contraction and stricture or occlusion.

Contusions of the perineum, may cause lacerations of the urethra and hæmorrhages, with bloody discharge or sanguineous swelling.

Treatment. Will vary. Calculi must be diagnosed and removed. Breach of the walls of the urethra may necessitate frequent catheterization or, better, the wearing of a catheter. Escape of urine into the connective tissue should be met by a counter opening in the skin to drain the part and allow free antisepsis. Similar resorts are required for urinary infiltration, accompanied by antiseptic injection subcutem. Abscesses must be located, punctured with trochar and cannula, evacuated and injected antiseptically.

FOREIGN BODIES IN THE URETHRA.

Apart from calculi, may be found straws, glumes, chaff and catheters and even stones and small bodies which must have been introduced deliberately. An irritation corresponding to the offending mass and its seat, ensues, and must be treated by soothing and anti-phlogistic measures while the offending body must be found and completely extracted.

STRICTURE OF THE URETHRA.

This may be suspected when in spite of much straining the urine is habitually passed in a very fine stream, which has become finer and finer for a length of time, without complete arrest as in calculus. The introduction of a catheter will confirm the diagnosis and show the exact seat of the stricture.

It is determined by irritation caused by calculus, urethritis, ulcer, wounds, etc., which tend to the formation of a cicatrix encircling and narrowing the canal. One efficient cause is the injection of strong astringent or slight caustic solutions in the early stages of urethritis.

Treatment is by dilation, by bougies pointed and gradually thickening, or simply by an elastic staff which at first passes with some force and is replaced by a larger one as the urethra stretches under daily use.

Catheterization. In connection with diseases of the bladder and urethra the passing of the catheter is a most important operation which requires considerable skill on the part of the operator. A short statement of the method to be adopted for each of the domestic animals will therefore be in place.

Catheterization in the male Soliped. The catheter, a hollow, gum elastic tube, must be proportioned to the size of the animal, but for the average adult horse about 3½ feet in length and ⅛ inch in thickness. To give it the requisite solidity and resistance it is usually furnished with a stilet of whalebone or cane.

The operation is performed with the animal standing, in quiet

animals without any restraint, but in the more sensitive or restive, with one fore foot held up; or with both hind feet in hobbles furnished with ropes passing between the fore limbs and tied over the neck in front of the withers; or finally with a twitch on the nose.

The rectum is emptied, and with the oiled or soapy hand the penis is found and slowly withdrawn from the sheath by steady traction. This is usually easy, though in certain cases, with a short penis and specially strong retractors, it will seriously tax the operator's skill and address. In a specially obstinate case a hypodermic injection of morphine may be resorted to.

If the horse is down, as in paraplegia or hæmoglobinæmia, he may lie on his right side while the operator stoops over him from the loins; or his feet may be drawn together by hobbles, and the subject turned on his back, the operator placing himself as before on the left side.

The catheter must have been previously cleansed and disinfected outside and in. A mercuric chloride solution 1 : 2000, or boric acid 1 : 50 or permanganate of potash 1 : 50 may be employed. Then it must be smeared, preferably with vaseline but, in case of necessity, with sweet oil, glycerine, borated lard, or even castile soap.

The penis being withdrawn from the sheath, the catheter containing its stilet is introduced into the urethra and pushed on slowly and carefully until its point can be felt over the ischiatic arch. The stilet is now drawn out a few inches and the point of the catheter is bent forward over the ischium by the finger. The stilet is further drawn out and the catheter can easily be pressed on into the bladder. If any difficulty is experienced it may be guided by the hand introduced into the rectum.

In one extraordinary case, I found that the catheter entered a dilated seminal vesicle and failed to evacuate the bladder. This untoward occurrence must be rectified by the aid of the hand in the rectum. Usually the penetration of the bladder is signalized by the overcoming of resistance, and when the stilet is withdrawn the urine flows in a steady stream. If it fails to flow, a slight compression of the fundus of the bladder by the hand engaged in the rectum will start the stream.

The catheter should be withdrawn slowly and carefully.

Catheterization in the Bovine Male. Most veterinarians suppose that this is impossible, owing to the narrowness of the sheath interfering with the extraction of the penis, and the S shaped curve in the penis preventing the introduction of the catheter. Both obstacles can, however, be overcome in many cases. The bull may be tempted to protrude the penis by the presentation of a cow in heat, or in bull or ox the bulging anterior part of the organ may be protruded by careful manipulation through the sheath. Then the free extension of the penis can be made to efface the S shaped curve. The catheter must be small, not much over a line in caliber, and a metal stilet is employed. The animal may have to be placed under restraint, and the same antiseptic precautions are demanded as in the horse.

Catheterization in the Ram and Wether. These must be dealt with like the bull, the only additional difficulty being in the vermiform appendix. This is small and sinuous but the longitudinal opening on its lower surface is favorable to the introduction of the catheter.

Catheterization in the Dog. The fact that the urethra traverses the groove on the lower aspect of the bone of the penis, is held to prove an obstacle to the catheter, yet the introduction of the latter is in no sense difficult. Small or moderately sized dogs, may be held upright, the body resting on the rump and the pelvis inclined forward, which will favor the spontaneous protrusion of the penis. Or it may be pressed out by manipulation through the sheath. The catheter $\frac{2}{3}$ ds. to 1 line in diameter may be $1\frac{1}{4}$ to 2 feet in length according to the size of the animal. It should be used aseptic.

Catheterization of the Mare. Nothing can be easier than this operation in the mare. The shortness and dilatability of the urethra, and the accessibility of its external orifice in the center of the floor of the vulva, 4 or 5 inches in front of the lower commissure, favors the introduction of the catheter. The latter may be a foot in length, perfectly straight and it may be constructed of silver or some other metal, which may be readily boiled and rendered aseptic. In the absence of a catheter the germ free nozzle of a rectal syringe may be used, or two fingers may be passed through the urethra and parted from each other so as to allow the exit of the urine.

Catheterization of the Cow and Heifer. The operation is often very difficult in the cow, by reason of the small size and undilatability of the urethra, and by the presence of two blind ducts (canals of Goërtner) above and to the two sides of the urethral opening. The thin rigid upper margin of the orifice projects down over it in a valvular manner so that the catheter will almost always find its way into one of the blind sacs. By introducing the tip of the index finger beneath the valvular fold and into the opening of the canal, the catheter may be directed beneath it and into the bladder. An apparatus consisting of a series of ribs of spring wire arranged in the form of a funnel and converging at one end to a point has been devised to insert into the urethral orifice, and guide the catheter which is passed through it.

Catheterization of the Bitch. The operation is rendered difficult by the narrowness of the passage, and the puckers and folds of the vaginal mucosa which serve to hide the urethral orifice. A small catheter like that used on the male is used or a short metallic catheter may be substituted. By directing this forward exactly in the median line of the floor of the vulva, with gentle pressure downward it may be made to enter the urethra. In case of special difficulty a bivalve speculum may be resorted to, to efface the mucous folds and reveal the orifice.

ACUTE PROSTATITIS AND HYPERÆMIA OF THE PROSTATE.

Causes: In dogs, house life, overfeeding, compulsory retention of urine, and fæces, constipation, proctitis (rectitis), piles, calculi, strangury, uric acid, urethral ulceration or stricture, rude catheterization, chill, generative excitement to excess, secondary abscess or infection. Forms. Lesions: follicular, interstitial, circumscribed and diffuse suppuration, Symptoms: frequent straining urination, rectal palpation, incontinence of urine, costiveness, tender perineum, dullness, recumbency, fever, pus in urine, collapse of swelling, fistula. Diagnosis. Prognosis: grave. Treatment: laxatives, mercurials, salines, leeches, acid laxative, non-stimulating, camphor, bromides, ergot, witch hazel, opium, belladonna, enemata of cold water, or ice suppository, catheterization, perineal incision. anti-septics.

Causes. This is most commonly seen in dogs, in which it may

depend on house life with overfeeding on stimulating, spiced, albuminous food, compulsory restraint of urination and defecation in obedience to the demands of cleanliness, distended bladder, and rectum, constipation, proctitis, piles, and other sources of local irritation. In all animals its origin is favored by the formation or arrest of calculi in the prostate, the pelvic urethra or even the bladder ; by drug strangury from cantharides or other irritant diuretic ; by excess of urea, uric acid or other irritant in the urine ; by infection extending from the urethra or bladder ; by ulceration or stricture of the urethra ; by rude or incautious catheterization, or injection ; by exposure to cold ; and by local infection in pyæmia and other general zymotic disorders. Most of these conditions conduce to local excitement and hyperæmia, which from adjacent organs, are sympathetically transferred to the prostate. The same is true of frequent, and intense generative excitement which according to Lafosse and Cadiot is a common cause of prostatitis in stud horses. Again the abscess of strangles may become localized in the prostate, or the nodule of glanders, or the tubercle of tuberculosis (cattle, pigs, dogs). Cadiot suggests that in animals, divested of the tail, external injuries to the perineum may extend by continuity to the prostate, as happens to man from horseback or bicycle riding. He adduces no cases however. The habit of masturbation acquired by certain males may also be adduced theoretically as both cause and consequence of prostatitis but future observation must show how frequently this really operates.

Forms. Lesions. According to the nature of the lesions the affection has been divided into different forms 1st, **Follicular** or **Parenchymatous** ; 2d, **Diffuse** or **Interstitial** ; 3d, **Circumscribed Prostatic Abscess** ; 4th, **Multiple Miliary Abscesses.**

Follicular Prostatitis implicates primarily the follicles and gland ducts and finally the entire gland tissue. It is usually associated with and doubtless often proves an extension from an adjacent infective urethritis, and tends, in persistent cases, to go on to interstitial inflammation and abscess, or hypertrophy. This is characterized by more or less swelling of the prostate, with increased vascularity of its mucosa and the oozing from its openings and gland ducts under pressure, of a thick, yellow, gelatinoid fluid containing pus and granular epithelial cells and sometimes striæ of blood.

Diffuse (Interstitial) Prostatitis shows, in addition to the general swelling and muco-purulent discharge, a considerable exudate into the interstitial tissue, with increased tension and resistance of its substance. It is associated during life with more fever and constitutional disturbance than the simple catarrhal or follicular form.

In **Circumscribed Prostatic Abscess** we find, in addition to the general hyperæmia and swelling, a much more prominent local swelling, the seat of intense inflammation, at first firm and resistant and later softer and fluctuating in the centre, which is filled with pus. This may have its origin in the follicular form, the pus becoming shut up in a follicle and gradually increasing until it bursts into the urethra, the bladder, the rectum, the peritoneum, or pelvic fascia and perineum. In other cases it becomes complicated by pyæmia and secondary abscesses.

Miliary Abscesses may be comparatively few in number or generally diffused through the prostate, and are often the result of a pre-existing general infection.

Symptoms. As the disease usually begins as a local infection the first symptoms are, as a rule, unattended by fever, which, however, appears in two or three days as the local lesions increase. The urine may be passed frequently in small amounts, or there may be frequent straining without passage of urine, the pressure of the swollen prostate, with or without spasm of the sphincter vesicæ, causing retention. It is no uncommon thing to find the last urine passed of a milky or glairy character and, coagula moulded in the prostatic canals may at times be found. The presence of spermatic crystals, fusiform, with very pointed extremities, and precipitated on the addition of ammonium phosphate, is characteristic of prostatic fluid, (Fürbringer). In other cases there is incontinence, the urine dribbling away involuntarily as the animal walks, and especially if anything occurs to excite him. Micturition may be painless or attended by acute suffering, which causes a sudden arrest of the flow. Defecation is attended with difficulty and more or less pain, and obstinate constipation is likely to set in. The animal is dull, spiritless and seeks to lie undisturbed. Pressure on the perineum is painful and exercise aggravates the symptoms. Rectal examination by the hand or finger according to the size of the animal, reveals

the enlarged, tender prostate lying on the cervix vesicæ. This swelling may be unilateral but most commonly it is bilateral or general. When fever sets in with a temperature of 102°–104°, thirst, anorexia and weakness or stiffness in the hind parts may be noticed. In case of abscess, the urine may be perfectly clear until it bursts into the urethra or bladder when there is an abundant flow of pus, and rectal examination shows that the swelling and tension have notably diminished. Should it burst into the rectum, the pus shows in the fæces. Reinemann records a case in a bull with dysuria, œdema of the sheath and a swelling like the fist in the perineum, containing pus, and which communicated with the prostate and urethra. With the rupture of the abscess there is a marked amelioration of the symptoms.

Not infrequently the affection subsides into the chronic form and the abscess, having a restricted channel for evacuation, remains as a suppurating cavity.

Diagnosis. The enlargement and tenderness of the prostate as felt on rectal examination is pathognomonic. If the body of the urine is clear there is further corroboration, as in cystitis it is more or less turbid and flocculent or even bloodstained. Micturition is likely to be much more frequent in cystitis than in prostatitis. Catheterization is much more painful when the catheter passes the prostate in prostatitis than in cystitis.

Prognosis is always grave. Some cases recover completely, while others run on to a fatal termination, and still others merge into the chronic form. Cases that are complicated by abscess are always to be dreaded, as chronic suppuration, or pelvic or peritoneal infection, or pyæmia, or septic poisoning is liable to supervene. As the disease is more common in the old so it is liable to prove more severe and redoubtable.

Treatment. In acute cases active derivation toward the bowels is desirable. A mercurial purge (calomel —horse or bull 1 to 2 drs ; dog, 2 to 5 grs.) may be followed a few hours later by salines (sodium sulphate - horse or bull 1 lb. ; dog 1 oz.). Sodium sulphate or magnesium sulphate may also be given with glycerine as an enema. Great benefit may often be obtained from the application of leeches on the perineum or around the anus. The diet must be restricted and non-stimulating, mainly of amylaceous materials, and with the water, flaxseed tea may be liberally

mixed. When the suffering is severe it may be met by camphor, camphor bromide, ergot, hamamelis, gelsemium or potassium bromide, given by the mouth or rectum. With violent strangury, opium, belladonna or hyoscyamus may be used. Some cases may be relieved by the use of enemata of cold water or pieces of ice in the rectum. In retention of urine, careful catheterization is imperative, the hand or finger in the rectum being employed to guide the point of the catheter under the prostate.

In case of abscess an opening by the side of the anus is preferable to one by the urinary passages or rectum, and will obviate the danger of rupture into the peritoneum. A pasty or fluctuating swelling in the perineum should be incised until the pus flows. A tense elastic fluctuating prostate may be transfixed by a cannula and trochar from the side of the anus, guided by the hand or finger in the rectum. When the pus has been evacuated a drainage tube may be inserted through the cannula and left in place when the latter is removed so as to allow free drainage at all times and frequent antiseptic injections. Punctures and even incisions have been made from the rectum, but they make badly infected wounds, and a rupture into the urethra, determines infection on that side, without any possibility of any effective antiseptic injection or perfect drainage. As injections may be used permanganate of potash 1 : 10000, or boric acid, a saturated solution. Poisonous agents must be eschewed or used with the greatest circumspection.

CHRONIC PROSTATITIS.

Causes. Follows acute. Same causes less potent. Lesions: as in acute, or sclerosis, and abscess. Symptoms: delayed urination, last glairy or purulent, constipation, defecation followed by urethral discharge, little genital ardor, rectal palpation, tenderness of prostate to hand or catheter, atony of hind limbs. Treatment: open air life, idle, milk or succulent diet, saline laxatives or enemata, avoid generative excitement, castrate, check masturbation, iodine, camphor, antiseptic irrigations.

Causes. The acute disease often subsides leaving an indolent chronic inflammation of the organ. Apart from this, the causes

are essentially those of the acute, but acting with lessened force or on a less susceptible system. Thus indoor life and overfeeding, with constipation and urine of a high density, calculus, irritant diuretics, the frequent incautious use of the catheter, infection from the catheter or otherwise, intense and frequent generative excitement, and exposure to cold are all occasional factors. Old age is a common concurrent cause.

Lesions. As in the acute form these indicate three successive, independent or concurrent forms, follicular, interstitial and suppurative.

With the distinctively **follicular** form the gland is usually enlarged and of a deep red color, but soft and friable, and when compressed exudes from its follicles and gland ducts a whitish muco-purulent glairy liquid. With the **interstitial** changes, which are often an advance on the follicular, the organ may be enlarged or shrunken, but the connective tissue has undergone a thickening and sclerosis which renders the mass firm and resistant, and which may have extended to the tissues in the immediate vicinity. In the **suppurative** form or stage, foci of suppuration are found throughout the gland substance, bulging out on its surface and even encroaching on surrounding tissues.

Symptoms. These are by no means obtrusive. There may be some delay in the discharge when the animal attempts to urinate, and the last drops of the urine, white and purulent or glairy, may be passed with evident pain. There is a tendency to constipation with painful straining to defecate. Compression of the prostate during defecation presses out its muco-purulent contents so that there is a greater urethral discharge following this act than at other times. This is also more abundant from the compression of the abdomen when the animal is lying down. This discharge is easily distinguished from semen by the absence or almost complete absence of spermatozoa and *the abundance* of spermatic crystals, precipitated by ammonia phosphate. In the earlier stages there may be undue generative excitement, erections, and even seminal discharge, with or without the movements of masturbation but in advanced cases genital ardor is usually defective or there may be practical impotence. Conclusive evidence is obtained by rectal exploration, when the enlarged, or irregularly shaped and tender prostate can be easily

recognized. If a sterile catheter is passed the pain caused as it touches the prostate is significant.

In the dog the affection may last for years, and tends to advancing atony of the hind limbs. A temporary arrest of the affection is often misleading, though the urine may be clear and normally discharged, yet manipulation may show a gradually advancing abscess, and when this bursts, usually into the urethra, all the symptoms become aggravated and cystitis, urethritis and general infection are to be dreaded.

Treatment. This is far from satisfactory yet in certain purely follicular or catarrhal cases it may prove successful. An open air life, without exertion, and a milk and farina diet are desirable, yet any tendency to costiveness must be obviated by saline laxatives and enemata. The avoidance of generative excitement must be secured, not only by restraining stud animals from service, but by keeping them well apart from all females of the same species. Even castration may be sometimes resorted to with advantage. Stallions given to masturbation must be restrained by net or otherwise. Any disease of the rectum, anus, urethra or bladder should be corrected, and undue exposure to cold prevented. Lafosse advises to slaughter butcher animals for food. Hertwig recommends iodine ointment on the anus and perineum of affected dogs. It must be borne in mind that the affection is maintained by infective microbes yet it is difficult to reach and deal with these thoroughly and effectively.

As an anaphrodisiac may be given camphor, or camphor bromide, ergot or potassium bromide, along with the mild stimulating antiseptic eucalyptol or copaiba. But the irrigation of the urethra, bladder and as far as possible the prostate with such antiseptic solutions as potassium permanganate (1 : 10000) or silver nitrate (0.5 to 1 : 100) or zinc chloride (1 : 100) is desirable. These should be injected into the urethra so as to reach the bladder, the contents of which they will render antiseptic and thus protect the organ against the transported microbes of the prostate. In man iodoform, europhen, and ichthyol are made into a bougie with gum, palm butter or other soluble liquifiable agent and inserted in the urethra as far as the prostatic part. Similar agents are used as suppositories or enemata. Hertwig's iodine ointment on the perineum may be advantageously replaced by sinapisms.

HYPERTROPHY OF THE PROSTATE.

In old dogs. Causes : age, overfeeding on albuminoids, rectal impaction or irritation, calculus, cystitis, urethritis, productive inflammation, trophic derangement when function declines. Lesions : hypertrophy general or partial, hard or soft, condensed or with sacs of pus, red or pale. Infective cystitis. Calculi. Symptoms : straining before urine comes, small or weak stream, sudden check, last part purulent or mucous, incontinence, triple phosphate, ammoniacal odor, crystals and dark color imply calculus. Diagnosis : by rectal exploration, and catheterization. Treatment : palliative, moderate, farinaceous, laxative diet, warmth, correct contiguous troubles, iodine, castration, extirpation of prostate.

This has been seen almost exclusively in old dogs, among the domestic animals.

Causes. Age and good living, more particularly on highly albuminous food, may be adduced as the most prominent. Perhaps even more important are continued irritation in adjacent organs such as the rectum, bladder and urethra. It is the old, pampered dog that above all suffers from atonic, overloaded rectum, proctitis, piles, calculus, cystitis, and stricture, and the constant local pelvic congestion caused by one or other of these tends to a hyperplasia of the prostate. Again atheroma which is especially a disease of the aged is regarded as a cause of both cystitis and prostatic hypertrophy. Chronic inflammation in the prostate has been claimed as a factor, but contested on the ground that inflammation never increases normal growth though it may cause degeneration. The exudate of inflammation, tends, however, on its temporary arrest, to undergo organization, and such organization inclines to assume the structure which is normally built up by the adjacent trophic cells. The products of inflammation may, therefore, well contribute to hypertrophy, and above all to the increase of the simpler tissue represented by the fibrous framework of the gland. The congestion attendant on excessive venery has also been incriminated, and this too has been denied on the ground that the hypertrophy is not found in the young animals and men in which the generative ardor is greatest and most frequently aroused and gratified. Thompson's idea is that the prostate, like the ovaries and womb, is especially prone to

morbid growths and developments at the time when in advancing age, the normal generative functions are undergoing a rapid decline. The two conditions may well be recognized without considering them as mutually excluding each other as causative factors.

Lesions. The enlargement is usually general, but it may predominate in the right, left or median lobe, the latter as a rule exercising greater compression of the urethra so that this is often marked in the worst cases. The hyperplasia may feel firm and resistant or it may be more or less soft from sacs of muco-purulent fluid imprisoned by the obstruction of the outlet canals. On the surface and on section the general appearance of the gland is pale, bloodless and uniformly solid. This comes from the great hypertrophy of the fibro-muscular stroma which has in many cases compressed the parenchymatous or secreting structure so as to cause its atrophy. The presence of calculi (mainly phosphatic) in the follicles is not uncommon.

The complication of infective cystitis is frequent, the congestion, redness, ecchymosis, maculation, puckering and thickening of the mucosa, the granular degeneration and desquamation of the epithelium, the exposure of a raw vascular surface, the discoloration of the urine by mucus, pus and blood, and the formation of ammonia and other products of decomposition, becoming marked phenomena. Vesical calculus is not uncommon, the slowness of the exit current of the urine retarded by the enlarged prostate, tending to prevent its impaction in the orifice and thus minimizing one of the most prominent symptoms.

Symptoms. Among the earliest symptoms, is some modification in the act of micturition. Straining a few seconds before urine comes, retention, incontinence and dribbling, discharge in a small or weak stream, and sudden arrest of the flow and the last few drops may contain muco-pus showing abundance of spermatic crystals, on the addition of ammonia phosphate. Impaction of the rectum tends to occur sooner or later, the animal making little effort to unload the viscus, and the overdistended organ becoming more and more atonic, congested and catarrhal and reacting injuriously on the urinary organs. Incontinence may be especially marked during sleep, the sphincter being sufficiently controlled by volition during waking hours. Retention

may be at first temporary from excitement and later more continuous by reason of the greater compression of the neck by the enlarged and indurated prostate. With the advance of the disease the urine shows abundance of triple phosphates, and becomes ammoniacal and fœtid. A dark or bloody color of the discharge and the presence of crystals suggest calculus.

An accurate *diagnosis* can only be had by rectal examination. The great enlargement of the prostate, in the absence of heat and tenderness is characteristic. Enlargement is usually uniform, though it may be concentrated on the right, left or central lobe. The passage of the catheter may be obstructed, but is not specially painful at the prostatic region as in prostatitis. From **vesical calculus** it is distinguished by the fixity of the swelling on the neck of the bladder as contrasted with the mobility of the stone inside that half-filled organ. From **stricture** it is differentiated by the fact that the obstruction offered to the catheter and the swelling of the prostate exactly correspond in position, that the stream is lessened in force rather than simply reduced in size, and that the history of the case shows no antecedent cause for stricture.

Treatment. This has been considered as mainly palliative. Special care of the general health and above all of the diet which should be moderate, farinaceous and laxative, protection against cold and wet, the correcting of any coexisting trouble of the urinary or generative organs, and the removal from all sources of generative excitement are important elements. Occasional small doses of Epsom or Glauber salts in draught or enema obviate rectal hyperæmia. Ergot, potassium iodide internally, and iodine or mercurial ointment to the perineum have had little good effect. Möller claims to have secured improvement from the injection into the prostate at intervals of fourteen days, of a solution of two parts each of tincture of iodine and iodide of potassium, and sixty parts of distilled water. A small hypodermic syringe is used and the injection is made through the rectum directly into the substance of the prostate. Glass has adopted the recent surgical alternative of castration, with the result of marked relief from the active symptoms in a number of cases, but with a more rapid advance through emaciation and marasmus to death in three or four weeks in others. We would suggest a careful antiseptic

castration in such cases, to obviate any added trouble from absorbed toxins or sepsis.

For the human subject, Lydston strongly advocates removal of the enlarged prostate by surgical means in strong, vigorous subjects, with healthy bladder and kidneys. The difficulty of such an operation in the dog is greatly enhanced by the relatively greater length of the pubio-ischiatic symphysis, and the lessened diameter of the pelvic cavity. Yet with the comparative immunity of the dog from suppuration, and the hopelessness of the case without such radical measure, and with the rigid application of an antiseptic technic, the operation would appear to be fully justified. It would be contra-indicated in all advanced cases, in which the prostate was the seat of active suppuration with discharge into the urethra, in cases complicated by urethritis, cystitis or nephritis, in cases in which there is marked prostration from sepsis or absorbed toxins, and generally in old, worn out and cachectic animals.

Short of this, in cases complicated by cystitis, antiseptics by the stomach and as injections into the bladder are desirable. Eucalyptol in doses of ten minims four times a day, or beta naphthol, guaiacol, or phenol have been used in man. As injections mercuric chloride 1 : 20,000; boric acid, saturated solution; or carbolic acid ·5 : 100 (Lydston) may be used warm several times a day.

TUBERCULOSIS OF THE PROSTATE.

This is a common seat of tubercle in generalized tuberculosis in cattle, and may give rise to the same urinary troubles as chronic prostatitis or hypertrophy of the organ. In a remarkable case recorded by Frauenholz the tuberculous prostate of an ox weighed 10½ lbs. and had contracted adhesions to surrounding pelvic organs. Section of the mass showed numerous centres of extensive caseous degeneration. In such cases the generalized tuberculosis is the important fact and the prostatic disease is only an unusually intractable complication. If less generalized, the implication of the testicle or epididymus is strongly suggestive, and examination of the urine may detect the tubercle bacillus, or the tuberculin test may develop the characteristic febrile reaction.

CANCER OF THE PROSTATE.

Lafosse records as colloid cancer a case of diseased prostate in an ox, in which the mass approximated to the size of the human head, and was made up of numerous cavities the largest not over 1½ inch in diameter, and all intercommunicating, and containing a gluey, or gelatinoid liquid with numerous small round cells and a few multinucleated giant cells. No evidence is given of the implication of even the adjoining lymph glands, so that the case was probably only an enlarged cystic prostate.

Fournier records a case in a three year old horse, which on necropsy showed a ruptured bladder, general peritonitis, and an enlarged prostate, involving Cowper's glands. Nocard identified its cancerous nature by microscopic examination. Yet there is not a word of the implication of adjacent lymph glands.

Goubaux says prostatic cancer is common in dogs.

PROSTATIC CYSTS.

These are not at all uncommon as a complication of hypertrophy of the prostate, the ducts having become obstructed and the follicles indefinitely distended. The case described by Lafosse as cancer of the prostate of a bull is strongly suggestive of such retention cysts.

CALCULUS OF THE PROSTATE.

Two forms of calculi have been found in the prostate in domestic animals: 1st, small, round, angular or branched bodies made up in concentric layers and formed of organic nitrogenous bodies: and 2nd, genuine calculi of calcium phosphate or ammonia magnesian phosphate. These may cause pressure on the parenchymatous tissue and atrophy, but in the lower animals they are seldom the direct cause of prominent morbid symptoms.

They must, however, be recognized as one of the causes of chronic irritation that contribute to prostatic inflammation and hypertrophy.

DISEASES OF THE FEMALE GENERATIVE ORGANS.

MALPOSITION OF OVARY AND WOMB. HERNIA OF THE OVARIES.

Inguinal or crural hernia of ovary or womb. Bitch. Long uterine horns, loose broad ligaments ; Sow ; Ewe ; Cow. Other openings. Symptoms : not marked : strangulation : inflammation : abscess. Gravid hernial uterus. Treatment : reduction : surgical means : Cæsarian section.

The most common displacement of the ovary in the lower animals is through the inguinal or crural arch. It is most frequent in the bitch doubtless for the reason that the horns of the womb are long, and widely separated from each other, and in any case of inguinal or crural hernia, or of undue dilatation of the openings they are liable to pass through. A relaxation of the broad ligaments is another condition of such displacement. The laxity of these ligaments in the normal condition in the sow favors ovarian hernia, and Dupont found the ovaries in the perineal region in five sows examined. Laux found the condition in ewes, and Müller in cows, one ovary lying on each side of the mammæ. With relaxation of the uterine ligaments the hernia might occur in any female mammal, and not only through the orifices named, but through any normal or adventitious opening in the abdominal walls.

Symptoms. In the bitch the hernia is often overlooked although an examination of the inguinal region will reveal the presence of a small nodule and vermiform body which may usually be returned into the abdominal cavity. In some cases it becomes strangulated by the gradual contraction of the neck of the hernial sac, followed by swelling, heat and tenderness of the hernial mass, which may go on to abscess formation. In exceptional cases impregnation occurs with the womb in this position and the steady nodular increase of the mass, and finally the automatic movements of the contained fœtuses become very characteristic.

Treatment consists in passing the womb and ovary back into the abdomen, and if adherent or incarcerated, in exposing and

releasing, and if necessary extirpating them. In case of advanced pregnancy with the gravid womb on the inner side of the thigh, a modified Cæsarian operation is required without the attendant danger of laying the peritoneal cavity directly open. Inflammation and abscess must be treated on general principles.

UNDEVELOPED OVARIES. ABSENCE OF OVARIES.

The absence of ovaries has been often noticed in twin heifers, and most commonly associated with deficiency or absence of the womb, and even of the anterior part of the vagina. The condition is especially common, though not constant as some have supposed, when the other twin was a male. Such females are known as *free martins* and fail to breed. Even when the ovaries are present in such twins they remain undeveloped, and are no larger than a bean or hazel nut. These usually have a firm, fibrous structure, and though there may be interspaces filled with a transparent fluid, no true Graafian follicles are formed. In birds, the left ovary only is developed and physiologically active. The absence of ovary has been noted also in the ewe, and less frequently in the mare and other species, and appears to be more common in twins than in single pregnancy. In cattle only has the influence of the male on the female twin of the same pregnancy been specially noted.

It has been noted that females with ovaries undeveloped, tend to show many male characters, in head, horns, and neck in cattle, in plumage in birds, and in voice in both.

ATROPHY OF OVARIES.

This is observed as a physiological result of having passed the breeding age, and may also take place from disease and degeneration of the organ. The fibrous stroma is usually increased and the cell elements reduced, yet in some cases, a cystic development occurs, giving the appearance of hypertrophy, while the ovarian parenchyma has actually been diminished.

SUPERNUMERARY OVARIES.

Extra ovaries have been found in different cases in the human female, the additional organ being furnished with a fallopian tube, and in some instances an extra uterine horn. No facts are at hand concerning the lower animals, but the occurrence of gestation and the birth of a single puppy a year after careful castration, would seem to suggest that the condition occurs in the bitch. There is no embryological reason why the lower animals should not at times show this deviation from the normal.

IRRITABLE OVARY. NEURALGIA OF THE OVARY.

This has been noticed most commonly in the mare, which from a quiet docile animal, has become very ticklish, especially in the region of the flank, kicking on the slightest touch, or even when approached and showing an amount of nervous apprehension, that may render her useless, for work. The ovaries are usually found to be enlarged, diseased and very sensitive. Œstrum may be in some cases constant and excessive and in others entirely suspended. Such cases are difficult or dangerous to shoe. In one case recorded by Thierry, handling of the flank promptly induced an epileptic attack. Cows with nympho-mania (bullers) are often victims of this condition. The only remedy is castration, which is best performed by the vagina. The shorter the period of the irritability the more perfect is the cure. In some old standing cases the vicious habit may have become so fixed, that it is continued in spite of the operation.

HÆMORRHAGE ON THE OVARY.

Mare and Cow: genital excitement, mechanical injury, diseased ovary in unimpregnated, ovulation with bleeding, falls, slings. Lesions: old degenerations, productive inflammation, varicosities, aneurisms, torpid vessels, blood staining, clots, follicular or not, ruptures into peritoneum, amount, microbes usually absent. Symptoms: obscure, arched, stiff loins, colics, recumbency, large tender ovary. Shivering, fever, anorexia, anæmia, surface coldness, unsteadiness, blood from vulva, liquid in abdomen fluctuates. Treatment: cold, ice, snow, on loins, cold acid drinks, tannin, iron chloride, matico, gelatine, subcutem atropin, ergotin, viburnum, derivatives, castration.

This has been seen in the mare and cow especially in connection with genetic excitement and mechanical injuries, and more especially pre-existing disease of the ovary. Trasbot notes that it has always been in the absence of pregnancy, a fact which we can easily explain on the ground that most active diseases of the ovary render the animal barren. Gestation like castration, calms the genetic instincts, and prevents the recurrence of œstrum with its vascular excitement, general and ovarian, which characterizes the unimpregnated condition. The normal rupture of the Graafian follicle and escape of the ovum is attended by some effusion of blood which passes through a series of changes preparatory to absorption. A more extensive bleeding, at the time of œstrum or otherwise, into a follicle or intrafollicular, and with or without rupture of the albugenic tunic constitutes the morbid hæmorrhage. Among mechanical causes may be named violent exertion, falls, and suspension in slings.

Lesions. Some cases in mares and cows show old standing lesions, to which the extravasation is secondary: a productive inflammation of the ovarian stroma; varicosity of the ovarian veins; aneurism of the utero-ovarian artery: the presence of emboli or thrombi. In the area of the effusion there is a general turgescence of the vessels, and blood staining of the stroma. Or there are distinct blood clots in the follicles or between them, a few lines or an inch in diameter, buried in the depth of the organ, or standing out in rounded swellings on its surface, and sometimes with a rupture two or three inches in length, and the

escape of blood into the peritoneal cavity. This may be sufficient merely to stain the peritoneal fluid, or it may amount to one or two bucketfuls as in cases recorded by Barrow and Palat. In the absence of rupture the effused blood may completely surround the ovary, or may accumulate in one or several of its distended follicles. The effused blood is rarely septic, being usually free from microbes, and it may remain fluid in the peritoneum, or coagulate in the ovary. The enlarged follicle may contain a dark red fluid, in which floats a solid clot, varying in color from dark red to light yellow, according to age.

Symptoms. These vary greatly with the extent of the lesion, and are always somewhat obscure. With slight interstitial or intra-follicular effusion, there may be only some general disorder, with, it may be, arching and stiffness of the loins, colicy pains, a desire for recumbency, and enlargement and tenderness of the ovary on rectal examination. In more severe cases as noticed by Cordonnier, Saucour and Palat in mares, by Laponsée in the ass, and Renault in the cow, there were shivering, hyperthermia, respiratory and cardiac acceleration, congested mucosæ, dull colicy pains, and anorexia, followed by indications of anæmia, small, weak, rapid pulse, pale mucosæ, coldness of ears and legs (in cows, of muzzle and horns), violent heart action, indisposition or inability to rise, unsteadiness on the limbs when up, and in some cases the escape of blood from the vulva. By rectal examination the enlarged, tender, doughy ovary may be characteristic and the fluctuation of liquid in the peritoneal cavity, which may also be recognized by manipulation of the flank.

Treatment This should be directed toward checking the hæmorrhage : Cold water or ice, on loins or flank : injections of cold water : cold water or acids or astringents by the mouth : tannic acid : iron chloride : matico : gelatine : atropine, ergotin. By way of quieting ovarian excitement, viburnum prunifolium or opium may be tried. Sterilized solutions of gelatine may be given subcutem. Mustard or ammonia may be applied to limbs or flank. In case of survival, castration will be indicated.

INFLAMMATION OF THE OVARIES. OÖPHORITIS. PERIOÖPHORITIS.

Mares, cows, sows, etc. Causes : traumas, œstrum, parturition, leucorrhœa, pus infection, strangles, dourine, glanders, abortion, tuberculosis, chill, poisons. Lesions : Ovary enlarged unequally, red, congested, exudate, extravasation, fibroid, caseated, purulent, abscess single or multiple, indurations, cretefactions, cysts, blocking of Fallopian tube, adhesions. Symptoms : mare : genital erethism, soiling of vulva and tail, colics, tender loins and mammæ : fever, dullness, emaciation, decubitus, paraplegia, swollen tender ovary : cow : bellows, paws. Sterility, anæmia, pyæmia. Treatment : Cold to croup, mustard, anodynes to vagina, calmatives antiseptics. Castration.

This has been frequently seen in mares, cows and sows, but it may occur in any of the female mammals or even in birds.

Causes. The condition has been ascribed to blows on the flanks, pressure on the abdomen and the congestion of the ovary which attends on frequent œstrum in the absence of the physiological quiet which comes from conception. In a large proportion of the cases, however, the attack has followed on parturition, abortion, a preëxisting leucorrhœa or metritis, or a suppurating process in some other part of the body. These cases therefore, must be looked upon as secondary and infective, the microbes having been transferred from the womb, along the Fallopian tubes, or through lymph vessels, or peritoneal cavity, or finally through the circulating blood. In mares strangles, abortion, leucorrhœa, dourine and glanders, and in cows and sows abortion, metritis, leucorrhœa, and tuberculosis, may prove the starting point of the infection.

Sudden chills when heated, perspiring or exhausted and especially exposure in inclement weather just before or after parturition, have been regarded as effective causes, and doubtless these lower vitality and power of resistance, but back of these we must look for infection coming from the parturient womb.

Bivort records an extensive epizootic of oöphoritis in sows kept on waste ground which had been used for herding swine years before. He attributes the trouble to poisonous plants, without,

however, attempting to identify them, and the probability is even more strongly in favor of infection left over from the former herds.

Lesions. The inflamed ovary is swollen slightly, or to a great size, in mare or cow like the fist or even an infants' head. The swelling, however, is unequal throughout, and the surface may bulge in rounded masses at different points. In the early stages the organ is firm, elastic, red and on the cut surface bleeding, with here and there a distended follicle with bloody or gelatinoid liquid contents. The exudate into the fibrous stroma may become coagulated, and later may be organized into fibrous substance giving a hard resistant sensation to the finger (sclerosis). In some cases this may become partly cartilaginous. In other cases the distended follicles may have their contents coagulated and transformed into a caseous mass, while much of the stroma has become liquefied and absorbed. When suppuration has set in, the gland is softened at this point, the parenchyma giving way before the pus. The pus may be in multiple sacs, as if formed in the Graafian vesicles, or it may be in one undivided abscess. In the ovary of a cow, Eléouet counted no less than sixty-three separate abscesses. In cases complicated by ovarian glanders, tuberculosis or actinomycosis, the gross, microscopic, and mycotic characters of the lesions will afford the means of diagnosis.

In chronic forms indurations, cretefactions, cystic degenerations, caseations, and sclerosis may be met with.

Lesions in adjacent structures are common, such as thickening and stenosis of the Fallopian tube; congestion, thickening and puckering of the mucosa in the adjacent part of the womb; peritonitis; adhesions of the ovary to the abdominal walls or to an adjacent organ.

Symptoms. Mare. In many cases the early phenomena are those of excessive genital erethism: the animal is restless, feverish, whinnies to attract other horses, snuffs the males on their approach, contracts the vulvar muscles constantly, exposing the congested mucosa and clitoris, and ejecting a glairy liquid which soils the tail, hips, thighs and hocks. She strains frequently, passing small jets of high colored turbid urine, and rubs the tail and hips against available objects, twisting and breaking the hair

and abrading and excoriating the surface. The croup may be alternately drooped and raised and the tail switched. These phenomena are not abated by copulation, nor by time, like ordinary heats, but will last for one or more weeks when a new set of symptoms set in. Meanwhile dull colicy pains cause restless movements, arched back, frequent moving from place to place, crouching by partial bending of the limbs, twisting of the hind parts from side to side. The loins are tender to pressure, and the middle of the flank to pressure or percussion. The mammary glands are usually hot, swollen and tender. The genital erethism may last from four to seven days. Then it subsides, with coincident improvement of the general symptoms and a recovery ensues. Relapses are to be expected sooner or later.

In fatal cases the erethism subsides, but fever, dullness and emaciation continue, the case becomes aggravated at intervals, weakness and exhaustion increase, decubitus may become constant or paralysis ensue. The patient dies in marasmus in one to three months.

In some the genital erethism is absent from the first. There is dullness, prostration, anorexia, fever, hurried breathing, small rapid pulse, colicy pains, tender abdomen, difficult defecation, coated dung, a glairy (perhaps reddish or fœtid) discharge from the vulva, hot, tumid tender mammæ, arched and sensitive loins, and stiffness of the hind limbs.

In all cases alike a rectal examination detects the ovary swollen and exceedingly tender.

Cow. The same general symptoms appear with characteristic modifications. Restlessness, bellowing, pawing, inappetence, arched, tender loins, swollen vulva with discharge, shiny and perhaps fœtid but without contractions, abdomen pendent and flanks hollow and tender, udder turgid, hot and painful, movements of the hind limbs stiff, halting, straddling. There is greater tendency to salacious movements of the croup. The diagnostic feature is palpation of the ovary through the rectum.

In *chronic cases* more or less of the above symptoms are shown in a greatly mitigated form, but oftentimes there are long intervals of apparent health. Palpation through the rectum is the final test in this as in the more acute cases.

Prognosis. This is very uncertain. Unless complete recovery takes place in a few weeks, the inevitable consequence is sterility, or death from hæmorrhage, peritonitis, pyæmia, or marasmus.

Treatment. In acute cases Trasbot strongly urges bleeding in the larger races and leeching of the flanks in the smaller. Mustard plasters to the loins and abdomen, and cold or damp applications to the croup are in order. Vaginal and rectal injections of mucilaginous liquids, containing anodynes and antiseptics are indicated. Opium, belladonna, hyoscyamus, chloral, borax, acetate of aluminium may serve as examples. If needful to quiet the excitement, morphia, atropia or hyoscyamin may be given subcutem. Or the anodynes may be administered by the mouth. As a last resort, and by far the most radical treatment, castration may be performed. With small ovaries this is best done through the vagina in the larger animals, while with large and adherent ones the flank operation is imperative. If the peritoneum is involved, careful antisepsis of the cavity is desirable. In case of adhesions the operation may be risky, but if successful it will obviate secondary infections and establish a permanent cure. Complications must be treated according to their nature.

OVARIAN CYSTS.

Mare, cow, ewe, sow, bitch, hen. Forms. Histogenesis. Dilated vesicles, egg tubes, blood obstruction. Lesions: Ovary large, smooth, lobulated, vascular, size, connective tissue, epithelium, liquid contents. Abscess. Symptoms: impaired portal circulation, muco-enteritis, piles, intestinal torpor, impaction, constriction, obstruction, congestions, inflammation. Urinary disorder. Strangulation. Sterility. Abortion. Dystokia. Indigestion. Anorexia. Colic. Genital erethism. Straining. Altered Urine. Peritonitis. Septic infection. Collapse. Rectal palpation, enlarged, sensitive ovary. *Treatment:* Castration. Tapping cyst. Rupturing cyst by compression.

These have been met with in all races of domestic animals, mare, cow, ewe, sow, bitch and hen. They vary greatly in their characters, being unilocular, multilocular, rounded or lobulated, serous, albuminous, colloid or hæmorrhagic, strictly ovarian or parovarian (in broad ligaments), in one ovary or in both.

Histogenesis. The source of these cysts has been much debated. Many have held with Spencer Wells that they have their origin in dilated Graafian vesicles, and the discovery of an ovum in the contents, by Rokitansky and Ritchie showed at least that this follicle had formed part of the cyst. On the other hand Foster, Rivolta, Klebs, Malassez and others, constantly failed to find ova or other distinct elements of the Graäfian follicles, but did find epithelial elements, and note that the cysts are at an early stage connected with the surface of the ovary like the egg tubes. These embryonic tubules of Pflueger are therefore held to be the starting point for the cysts, which because of their mixed epithelial as well as liquid contents, seem allied to adenoma. From observations on the ovarian cysts of the lower animals Galtier, attaches great importance to vascular obstructions. Obstruction by pressure or otherwise led to hæmorrhages and transudation of blood, and the cavities formed in this way became the seats of epithelial growth, and liquid effusion. The blood remained for a time as distinct clots, and was later indicated by the pigmentation of the walls of the cyst.

Lesions. The enlarged ovary may be uniformly rounded and smooth, or it may be marked by irregular bosses, giving it a lobulated appearance. It is very vascular, and is often covered by a thickening of peritoneum. When multiple they are usually closely adherent and may even be included one within another. The individual cysts may be of the most varied sizes. The cystic ovary has at times reached enormous dimensions : in the mare 46 lbs. (Bouley, Rivolta, Thiernesse): in the cow 250 lbs. (Reynolds, Meyer): in the ewe 7 lbs. (Willis): in the sow 7 lbs. (Reyer): in the bitch 15 lbs. (Bovett). The walls of the cyst are formed of connective tissue more or less perfectly organized, arranged it may be in several superposed layers (Galtier) and lined or not by epithelial cells (cylindroid, nucleated, or of various forms). They may be reddened by hæmorrhages or pigmented from former blood extravasations. The liquid contents may be clear and watery, white, straw yellow, or of a deeper yellow, brown or red. Among other constituents there are alkaline chlorides and sulphates, albumen in solution or flakes, mucin, fibrine, fatty granules and cholesterine crystals. In some instances they contain pus cells (chronic abscess).

Symptoms. Small, tardily growing cysts may cause no appreciable symptoms. The larger ones or those that increase rapidly are liable to cause disorders of circulation, innervation and digestion. The mere pressure of a considerable cystic ovary may interfere with portal circulation so as to entail muco-enteritis, rectal congestion, piles, or intestinal torpor or impaction. Adhesions of the diseased ovary to adjacent intestinal viscera, tend to produce constrictions, obstructions and local congestions or inflammation. In adhesions to the womb or bladder, ureter or kidney, the symptoms will indicate disorder of these respective parts. The weight of the enlarged ovary causing extension of its ligamentous connections will allow of its winding around a loop of intestine and producing strangulation. In those unusual cases in which pregnancy occurs it may interfere with its completion, causing abortion or, failing in this, with parturition, by becoming imbedded in the pelvis. In the line of innervation, disorder is especially common in the digestive organs, anorexia, nausea, impaired rumination, and colicy pains resulting. Again, in many subjects the genesic instinct is stimulated, the patient is more or less constantly in heat, cows become *bullers*, and mares *switchers*, they cannot be impregnated, and under the continuous excitement undergo rapid emaciation. There is often urinary disturbance, frequent straining with the passage of a small quantity only of turbid or glairy liquid, colored, it may be, by blood, or fœtid. The colics are liable to be dull and slight, the patient moving uneasily, switching the tail, moving the weight from one hind foot to the other, pawing, looking at the flank, but seldom lying down or rolling. In other cases, with adhesions, impactions, obstructions, and congestions, all the violent motions of the most intense spasmodic colic may be shown. Where there has been rupture of the obstructed bowel, these symptoms may merge into those of peritonitis, septic infection, or collapse. When with these symptoms of intestinal disorder, there are tender loins and flank, abdominal plenitude and tension, genital excitement, frequent straining to pass urine, the discharge of a glairy or fœtid liquid, and when all these symptoms have increased slowly for weeks or months in a female, the ovaries may be suspected and a rectal examination should be made. Usually the outline of the womb can be made out with the enlarged and irregularly shaped

ovary anteriorly and adherent to it through one of the broad ligaments; it may be sensitive to touch, tense, or even fluctuating. Difficulty may be encountered when the enlarged ovary is so great as to fill the whole region, or when adherent to or wound round the rectum, thus hindering the advance of the hand or the movement of the gut, or when it has become pediculated and displaced to a distant part of the abdomen. Even the obstructed and distended intestine, may prevent a satisfactory diagnosis. Yet in the great majority of cases rectal examination gives conclusive results.

Treatment. Medicinal measures are useless: surgical alone are of any avail. Castration is the natural resort, and in all recent cases, uncomplicated by adhesions, is to be preferred. In the large females it may often be performed through the vagina, but if the ovary is very large the flank operation becomes imperative. Sometimes the evacuation through a cannula of the contents of one or more large cysts will so reduce the mass as to allow of the safer vaginal operation.

A less radical measure is the evacuation of the cyst with cannula and trochar and the injection of tincture of iodine. With a hand in the rectum the ovary may be held against the abdominal wall to facilitate the operation. The results, however, are not satisfactory, for, although re-accumulation of the liquid is delayed, it is not entirely prevented. Moreover, when the cysts are multiple, the punctures also must be numerous, or remain ineffective. Nor is the operation unattended by danger as deaths often occur from resulting inflammation, infection, or iodine poisoning.

Zanniger, in 1860, introduced the method of rupturing the cyst without incision, and met with considerable success. With the hand in the rectum the cystic ovary is pressed against the wall of the pelvis or abdomen, until the attenuated wall of the cyst gives way, the fluid is left in the abdominal cavity, to be absorbed and many animals will afterward become pregnant. In a large proportion of cases in which the symptoms are marked, the walls of the cyst are sufficiently attenuated to allow of rupture by pressure, and, if the escaping contents are free from infecting microbes, no immediate harm comes to the peritoneum. It should be avoided in case of abscess, following perhaps on a shivering fit and constitutional febrile reaction, and when there

is a fœtid discharge from the vulva, suggesting microbian infection likely to dangerously infect the serosa. In appropriate cases it is a resort of very great value, in restoring to use animals that are especially valuable for their progeny and which become utterly useless when rendered barren. According to different observers an average of 70 per cent. can be restored to usefulness in this way. Friedberger and Fröhner claim 90 per cent. Some febrile reaction may be noted for twenty-four hours, demanding rest, restricted, cooling, laxative food and sometimes laxatives and anodynes.

DERMOID CYSTS OF THE OVARY. PILOUS CYSTS.

Closed cutaneous sacs, with hair and sebum. Causes: enclosure of dermoid tissue in embryo: aborted ovum: virgin gestation. Symptoms. Treatment: Castration.

These are much less common than are simple cysts. They are closed sacs, lined by a tissue essentially representing skin, and containing sebaceous matter and hairs, some growing from the dermoid surface, and others detached and formed into a loose mass.

Causes. These cysts have been attributed to the enclosure, in the forming embryo, of the formative elements of dermoid tissue, which may or may not remain latent and inactive until maturity, or until the ovary becomes physiologically active.

Another theory is that an impregnated ovum has remained imperfect, developing only the elements of the skin, instead of the whole fœtal body. Many cases cannot by any possibility be included under this head, seeing that the cyst is found at much too early an age, and its bearer has never had sexual intercourse.

Another doctrine is that the dermoid cyst is derived from the normal plastic or formative powers of the ovary, and the product becomes suggestive of parthenogenesis or virgin gestation. The fact that these cysts are not confined to the production of skin and hair, but at times form bone, teeth, nervous and other tissues as well, corroborates this view. On the other hand we must bear in mind that dermoid cysts are much more common in other tissues than

they are in the ovaries. Thus they are common in the subcutaneous connective tissue and between the muscles.

The *symptoms* do not differ essentially from those of simple cysts and *treatment* is mainly by castration. As the escape of the contents into the peritoneal cavity is especially provocative of infection, the greatest care must be taken to extract the mass whole, or to use the most thorough antiseptic precautions.

SOLID OVARIAN TUMORS.

These are much more rare than cystic tumors. They seldom maintain the character of perfect solidity, for whether fibrous, sarcomatous, melanotic, cretaceous, myomatous, cancerous, epithelial, tubercular, glanderous, or actinomycotic, they are usually associated with cysts to a greater or less extent. Not only are they liable to stimulate the formation of cysts, but the special heteroplasia may become engrafted on the walls of pre-existing cysts, as well as on normal tissues.

The *symptoms* of the solid tumors are in the main, those of the cystic form, and *treatment* resolves itself into extirpation by castration. Its success will vary according to the nature of the tumor, sarcoma, melanoma and carcinoma being especially liable to recur in the same or in distant situations, and the same is true of the colonizing with infectious germs (glanders, tuberculosis, actinomycosis) which are presumably already present in other parts of the body. Castration has, however, this recommendation, it secures the removal of the entire diseased organ, and if the morbid process or infection is confined to that only, it holds out the best prospect of recovery.

INFLAMMATION OF THE FALLOPIAN TUBES. SALPINGITIS.

This condition is met with in the female mammals of all species and mainly as the result of an infection extending from diseased womb or ovary. The results are degeneration of the

epithelium, exudation into the mucosa with thickening, stenosis of the tubes, the formation of cysts along the line of the canal, with pink or straw colored contents, including fibrine, leucocytes, epithelium and granular debris. As in oöphoritis there may be blood extravasations and clots and abscess. In the cow they are at times calcified and create a suspicion of tuberculosis.

The *symptoms* are essentially those of metritis or ovaritis, and as these are usually more prominent the attendant salpingitis is generally overlooked during life. Careful rectal examination may detect the enlarged, tender or sacculated tubes. *Treatment* may be laxative, diuretic, derivative, and antiseptic toward the womb. Ablation of the ovaries, tubes and even the womb is often required.

DISEASES OF THE OVIDUCT IN BIRDS.

Imperforate tube near cloaca. Polypus: snare and twist off. Egg impaction: from atony, inflammation, stricture, congenital smallness, exhaustion, large eggs, thick end first, broken egg. Symptoms: mopes alone, feathers erect, wing and tail drooping, large, solid swelling around anus and abdomen. Rupture into abdomen. Treatment: oil cloaca and oviduct, manipulate, turn, break egg and scoop out, incise and extract, antiseptic oils. Excision of ovary.

Imperforate oviduct usually occurs in the lower part of its course, the tube being connected with the cloaca by a short, fibrous cord. In the case of a very valuable bird it may be incised and the walls of the duct may be brought down and fixed to those of the cloaca.

Polypi of the oviduct may seriously impede laying, and start obstruction and impaction. The seat of the tumor having been ascertained, it may be seized and twisted off by a snare. An elastic wire is passed through a small metallic tube so that a loop protrudes large enough to pass over the polypus. When fixed around the pedicle, it is tightened, and the tissues twisted through.

Egg Impaction in the Oviduct. From weakness or lack of tone in the bird, by inflammation and loss of contractile power in the oviduct, by stricture of the duct as a sequel of inflamma-

tion or abrasion, by congenital narrowing, by weakening of the oviduct through constant laying, by excessive size of the egg, by double yolked eggs, by presentation of a large egg with its thick end first, or by an egg with broken shell, the oviduct may be rendered incapable of passing the egg on and out, and as others continue to press down from above an excessive and dangerous impaction ensues. The bird refuses food, mopes around with ruffled feathers and drooping head, wings and tail. The region of the anus and in front of it hangs downward and feels firm and solid, and the oiled finger introduced into the cloaca comes in contact with the impacted mass. The bird strains violently but ineffectually and rubs its anus on the ground. The swelling goes on steadily and rapidly increasing, and the bird becomes more prostrate and hopeless. Sometimes the overdistended and congested oviduct gives way and the eggs escape into the abdomen. Reul has counted as many as 24 eggs that had thus escaped into the abdominal cavity. Or without rupture of the oviduct, the soft eggs pack together into a solid, dry yolk-like mass, the watery parts having been pressed out or absorbed. In bad cases this may weigh 1½ lb. in the hen (Weber). In the way of *treatment* the cloaca and oviduct should be thoroughly lubricated with a bland oil, which might be injected with a syringe, so as to pass it, if possible, around the impacted egg or mass. By careful manipulation the egg may now be brought away. If the thick end is presented it is sometimes possible to turn it so that the thin end will come first. Should all fail the egg may be broken and its contents together with the other impacted matter may be disloged with a looped wire or small spoon. The oviduct should be lubricated for some time with a bland antiseptic oil (olive oil and boric or salicylic acid). In obstinate cases the abdomen and oviduct may be laid open and both evacuated of any egg matter that may be present. After suitable antisepsis the wounds in the oviduct and abdominal walls are to be sutured. If there appears to be danger of the further early descent of eggs into the weakened oviduct the ovary may be removed.

Eversion of the Oviduct. This appears at times as a result of the intromission of the penis (ducks) being shown immediately after copulation as a pink, lax membrane one or more inches long, dragging from the anus. In other cases it appears to result from

the paresis that occurs in old birds from prolonged laying, or from inflammation and impactions. It may appear abruptly or gradually, and after a few hours becomes the seat of exudation, swelling and redness, forming a pyriform mass. In some cases it is carried out around an egg which does not glide through its canal and may be felt through its walls, and through its terminal opening. A partial eversion may take place as an invagination into the cloaca, without showing externally. When an egg is impacted, or when the protuded organ is inflamed and swollen, violent straining continues, which tends to aggravate the condition, and the bird gets rapidly exhausted, resting on its breast, later upon its back, and dying in convulsions.

In slight cases following copulation, the vermicular movement of the duct, of the cloaca and anus may serve to secure speedy spontaneous reduction. In the partial cases, of eversion into the cloaca, the free local use of oil, may secure the passage of the presenting egg and the return of the oviduct. If necessary the egg may be broken and its shell thoroughly extracted. This last method is imperative when the egg enclosed in the oviduct has already passed through the anus. The oviduct should then be cleansed in tepid water, and laudanum, oiled and returned.

Inflammation of the oviduct is a common condition resulting from debility, from impaction of an egg or of egg-material, from scratching with the shell of a broken egg, and from microbian invasion. The frequent passage of large eggs is an accessory cause, and the egg becomes an important factor in the maintenance and aggravation of the inflammation. The mucosa becomes red, dry, infiltrated, thickened and friable, and the muscular coat increasingly paretic. The egg, becoming impacted, and subjected to constant pressure in the vain efforts at expulsion, hinders circulation and nutrition, and favors necrotic and ulcerative processes, and too often the fragile membranous walls yield, and the mass drops into the abdominal cavity. Short of this, the exudate at a particular point, the main seat of inflammation, contracting in undergoing organization, forms a distinct stricture, which renders the further laying of fully formed eggs difficult or impossible, and further impaction, inflammation and rupture may follow. Sometimes the irritation causes undue peristalsis in the anterior and less actively inflamed part of the tube, and the eggs

are laid prematurely without albumen or without shell, yet with much effort and suffering. Or the bowels become irritable and a profuse diarrhœa sets in, hastening the exhaustion of the patient.

Treatment should be applied early. A cooling diet of vegetables or slops, the careful removal of all irritating contents from the oviduct, and its frequent injection with bland oils medicated with mild anti-septics (boric or salicylic acid, or potassium permanganate) will usually serve a good purpose.

HYDROMETRA AND PYOMETRA.

Cause: chronic metritis, tumors, microbian infection. Symptoms: ill-health, low condition, vulvar swelling or discharge, swelling and fluctuation of womb. Rectal exploration. Treatment: evacuate liquid, disinfect womb and passages, creolin, iodine.

As a rule these conditions belong to obstetrics and would not come under the scope of this volume, but when in chronic cases, with closure of the neck of the womb, the liquids accumulate and distend the uterus, they may deserve mention in a medical work.

The *cause* is usually a chronic metritis, originating it may be at the time of a now distant parturition, or associated with tumors or microbian invasions of the womb. In the deadly cases that follow upon parturition and abortion streptococcus is usually present, in the more chronic forms the staphylococcus or other pus microbe.

The *symptoms* are those of general ill-health, low condition, pallor of the visible mucosæ, sometimes swelling of the vulva with discharge, serous or purulent, lessened milk yield, enlargement of the abdomen with fluctuation felt in the right flank, or still better with the hand in the rectum. Rectal exploration will further detect the distended uterus connected with the vagina behind and dividing in front into two horns.

Treatment consists in the evacuation of the liquid through a catheter or cannula introduced through the os, or through the vaginal wall immediately above, followed by a systematic disin-

fection. By placing the patient upon her back, gravitation of the liquid is more marked, but when this is not convenient it may be done with the animal standing. The flow may be favored by raising the abdomen with a sheet held by two assistants. The womb may be thoroughly cleansed by a normal salt solution which has been boiled, and then daily injected with a creolin solution (1 : 100). In hydrometra an iodine lotion may be used. A course of tonics is often indicated.

UTERINE TUMORS.

These are somewhat rare in the domestic animals, yet they have been met with in the form of cysts, fibroma, fibro-myoma, sarcoma, and carcinoma. In a number of cases the nature of the tumor has not been clearly made out. Mangot saw a mare with two pediculated uterine tumors having an aggregate weight of 12 lbs. These were expelled with much straining and suffering. LaMaitre and Rodet record other cases. Stockfleth describes multiple pediculated fibroid tumors in the womb of the cow. Cysts have been especially seen in the cow and bitch, and carcinoma in the bitch.

Symptoms. These may for a length of time be overlooked, though breeding animals usually fail to conceive. Then a slimy, mucopurulent, serous, bloody or fœtid discharge may escape habitually from the vulva, smearing the tail and hips and collecting on the floor. If the os is sufficiently patent to admit the hand vaginal and uterine exploration will detect the tumor. In other cases it may be felt by rectal examination.

Treatment is essentially surgical and will consist in dilatation of the os, and the removal of the tumor by twisting, ecraseur, or curette, and with careful antiseptic precautions before and after. In malignant tumors in the uterine walls it may be expedient to remove the entire organ.

UTERINE TUBERCLE.

This has been seen especially in sterile cows, the subjects of nymphomania, and it may be associated with a muco-purulent or bloody discharge from the vulva, nodular swelling on the uterine horns, perhaps also on the broad ligaments, one or both ovaries, and the mesentery, to be recognized by rectal examination. The presence of tuberculosis in the lungs or throat, and the response to the tuberculin test will confirm the diagnosis. As a rule it is not desirable to institute treatment.

IMPERFORATE HYMEN.

Cases of this kind have been described in mare and cow, preventing copulation and conception, and leading to a distension of the vagina, with a glairy fluid, which obstructed defecation, irritated the bladder and caused violent but fruitless straining, under which the mass would project from the vulva. The centre of the swelling may be penetrated with a trochar or bistuory and enlarged by incisions in several directions. Relief is prompt and lasting.

VAGINITIS. LEUCORRHŒA.

Inflammation of the vaginal mucosa is usually a concomitant of metritis and like that follows parturition. It will however occur independently from direct injury or infection or from the presence of neoplasms. In dourine and horsepox, vaginitis is a common symptom, to mare, cow and bitch infection is conveyed by coition. Dieckerhoff quotes old chronic cases, also acute ones which extended to the peritoneum and proved fatal in a few days. The common symptom of muco-purulent discharge having a heavy or fœtid odor is together with the discharge from the womb known by the common name of leucorrhœa. The frequent irrigation of the whole passage with antiseptic solutions is usually successful in putting a stop to the affection, unless in case of constitutional infection, or the presence of some neoplasm.

TUMORS OF THE VAGINA.

The vagina is the seat of different forms of neoplasms in the various domestic animals. Thus cystoma, lipoma, adenoma, fibroma, fibro-myoma, sarcoma and epithelioma have been noted. They are essentially surgical and to be dealt with as such. They are mainly important in this connection as inducing a leucorrhœa, which in the absence of careful examination might be mistaken for that of uterine or vaginal infective inflammation.

PARTURITION FEVER (COLLAPSE). MILK FEVER. PARTURIENT APOPLEXY. CALVING FEVER. PARTURITION PARESIS.

Definition. Predisposing causes: genus, breed, great milking capacity, heredity, mature age, vigor, high-feeding, powerful digestion and assimilation, sudden plethora, drying up of milk, parturition, easy delivery, warm season, chills, idiosyncrasy, cardiac hypertrophy, contraction of womb, emotional excitement: Supposed causes: absorption of toxins from womb, colostrum, Schmidt treatment its significance, microbian infection and intoxication, effect of change of stable. Microbiology. Nature: Theories of nervous explosion, vaso-motor cerebral anæmia from exaggerated excitability of the uterine nerves, or from dilatation of the portal system and womb, metro-peritonitis, cerebral anæmia from congestion of the rete mirabile, etc., palsy of the ganglionic nerves, plethora, intra-cranial arterial tension, narcotic poisons from leucocytic or microbian source. Lesions: variable, cerebral and spinal congestion, pulmonary congestion, collapse, septic inhalation, bronchitis, dessication of ingesta in omasum and large intestine, black thick blood, yellowish gelatinoid exudates in cranium and spinal canal and under spine, glycosuria. Symptoms: time, post parturient, plethoric subject, sudden onset, comatose and violent forms, discomfort, restless movements, inappetence, moaning, mental dullness, unsteady walk, muscular weakness, compulsory recumbency, retained urine and fæces, drowsiness, somnolence, unconsciousness, stertor, venous pulse, tympany, sudden recovery, complete, with paralysis, fatal cases, violence, tossing head, trembling, cramps, convulsions, temperature. Mortality. Prevention: bleeding in plethoric, heavy milkers, purging, low diet, exercise, comfort, milking, blisters, sucking by calf, disinfection, iodine solution

in udder. Treatment: in early stages bleeding, purgatives, peristalsis stimulants, antiseptics, injections, stimulants, rubefacients or cold sponging, elevation of the head, udder massage, milking, iodine injection of the mammæ.

Definition. A nervous disorder which develops suddenly in plethoric cows, heavy milkers, after calving, and is characterized by loss of senses, of consciousness and of muscular control, by hypothermia or hyperthermia, convulsions, coma, and melliturıa.

Causes. While one cannot speak positively as to the essential cause of this disease, certain conditions are so constant and prominent that they must be given a high value as *pre-disposing causes.*

Genus and Breed. Milking Capacity. This is essentially a disease of cows, probably largely because of all domestic animals, cows only have been long and systematically bred to secure the greatest power of digestion and assimilation and the highest yield of milk. It is the disease not only of cows, but of milking breeds, and preëminently of individuals that give the most abundant dairy product. It is rare or unknown in scrub or common herds, while common and fatal in the best milking breeds, in advancing ratio about as follows: short horn, red polled, Normand, Swiss, Ayrshire, Flemish, Dutch, Alderney, Jersey, Guernsey, and Holstein. Heredity may be claimed, as the special pre-disposing qualities are hereditary.

Age has a marked influence, but this is subsidiary to the milking qualities. The disease rarely attacks a cow after the first or second calving when the system is as yet immature, and the milk yield has not reached its maximum: nor one that is past its prime and already failing in vital energy and milking qualities. The following table is from statistics compiled from veterinary records in Denmark and Bavaria:

Age, yrs—	3	4	5	6	7	8	9	10	11	12	13 and over.
Cases —	8	21	65	160	171	202	117	124	44	70	78

It will be noted that it is in the period of the most vigorous, mature life, from the 6th to the 10th year inclusive that the great majority suffer. In a judiciously managed dairy it is the best cows that are carried at these ages, and although the very best are kept on into old age they show a steadily decreasing number of cases as they begin to fail. The disease is all but unknown in primipara.

High Feeding. Heavy and rich feeding prior to calving and immediately after, is a most prominent cause of the affection. This is so well known to owners of milking breeds, that they usually hold to the principle that the cow that is a heavy milker, should be all but starved for a fortnight before calving and for a week after. In herds where this rule is acted on the disease is rare and may be altogether unknown, and when it is neglected the malady is often very destructive.

Plethora. High Condition. Heavy feeding and high condition usually go together, and the majority of the victims are fat or in good flesh, yet a certain number are actually thin. The predisposing condition is plethora rather than fat or flesh, and this may be present in the comparative absence of flesh. The cow that is from a stock famed as heavy milkers, does not tend to lay on flesh, but, on succulent diet especially, the greater part of the nutritive matter assimilated goes to the production of milk, and she remains thin in flesh no matter how heavily she may be fed. Many such cows never go dry, but give a liberal yield of milk up to the day of calving, and if measures are taken to dry them up, it is done at the expense of a sudden plethora, as the milk giving system does not at once accommodate itself to the laying up of fat and flesh.

The *drying up of the milk secretion* sometime before calving in a cow which is normally a heavy milker is therefore a potent factor.

Parturition is an almost indispensable factor as the disease occurs one to seven days after that act, and only in rare and somewhat doubtful cases before it.

Easy Delivery with little nervous outlay or loss of blood, and no exhaustion is a special feature. The attack almost never occurs after a difficult parturition with considerable loss of blood and much nervous exhaustion. This should to a large extent exclude such alleged factors as *shock* or *wearing out of nervous energy*. The nervous prostration which figures so prominently in the disease, seems to be less the result of wear and tear, than of the supply of an excess of blood, which is either over-enriched, or charged with some injurious toxic matter. At the same time there is a manifest susceptibility at the parturient period which is not present at other times, and the plethora or toxin takes occasion to operate when this predisposition renders

such an attack possible. *The Warm Summer Season* has been claimed to induce a greater number of cases, and doubtless exposure to continuous heat, tends to prostrate the nervous system and predispose to congestion, this fails to take into account the still more important element of the rich spring and early summer pastures, where the already plethoric animal is left to feed without stint, or the tempting red clover, alfalfa and other fodder crops, rich in albuminoids, which are fed liberally in a succulent condition.

Chills in cold winter weather have been similarly invoked as driving the blood from the surface to collect in internal organs, including the brain. That chills do act in this way cannot be denied, but there is no demonstration that any number of cases have been materially affected by cold.

Idiosyncrasy. Constitutional Predisposition. This must be allowed, inasmuch as that it covers all those individual conditions, functional and structural, which belong to the heavy milker, or the animal with extraordinary powers of digestion and assimilation. The same shows in the predisposition to a second attack of an animal which has survived a first one. The structural changes in the nerve centres, which occur in the primary attack, leave traces, which render these parts more susceptible at the next calving. In my own experience the violence of the disease is liable to increase with successive attacks, so that a second or third cannot be hoped to be as mild as was the former one.

Cardiac Hypertrophy. Cagny draws attention to the fact that in man and beast alike the heart undergoes hypertrophy during gestation and, above all, during its later stages. In improved breeds of cattle, and especially in milking breeds, a great development of the whole circulatory system is seen, and a large heart is a constant feature of this. This implies an increased force of cardiac systole, an increased blood tension in the arteries and capillaries, a condition which tells with special force on the soft tissues of the brain, as the violent abdominal compression in the expulsive efforts of parturition, tends to drive the blood from the great vascular viscera situated back of the diaphragm.

Parturition and the subsequent *contraction of the womb and expulsion of the great mass of blood*, must be accorded a prominent place among causative factors. The disease is almost restricted

to the first week after parturition, and its gravity is greater the more it is related to the parturient act. Cases occurring in the first three days are usually fatal. The gravid uterus contains a very large amount of circulating blood, and when the womb contracts, the greater part of this is suddenly thrown upon the general circulation, already plethoric to an undue extent. As yet the mammæ are congested and there is no free depletion through that channel, so that there is a marked temporary plethora and vascular tension, before the system can establish free elimination and, as it were, strike a healthy balance. In this period of transient plethora there lies a source of great danger to the general system and, more particularly, to the brain.

Emotional Excitement connected with the removal of the calf is urged by Günther, Jaumain, Félizet and others as a prominent cause. This, however, must be rare, at the most; the disease does not attack the primipara that should be most susceptible to this influence, but the mature animal, at her third calving or later when she is already well accustomed to this treatment; it supervenes so quickly on parturition in many cases, that there was no opportunity for such emotion; it occurs also in cows, the calves of which have remained with them or have received no attention from them.

Absorption of Toxic Matters. The theory of a poisoning of the nerve centres is indicated in the familiar name of *milk fever*, suggesting an absorption, or poisonous condition of the milk. Lafosse charged the trouble on the *uterine milk* secreted in the cotyledons and re-absorbed in quantity. Abadie and Kaiser attributed it to the products of gastro-intestinal ferments, which acted on the nerve centres like a deadly organic alkaloid. Hartenstein incriminated the products of muscular contraction in the womb and systemic muscles during parturition. Ehrhardt invoked a similar auto-intoxication, going on before parturition and only reaching its climax in connection with that act.

Allemani and Gratia attribute the disease to the absorption of the first milk (colostrum), and there are several considerations that strongly favor this hypothesis. The disease sets in always in connection with the parturient development and congestion of the udder and the secretion of the first milk. In exceptional cases it may even appear just before parturition. Even upon the calf

the colostrum operates as an irritant and purgative. Is it wonderful that, in the parturient cow, with a high state of plethora, a highly susceptible state of the nervous system, and the various concurrent conditions already referred to above, a direct poisoning of the nerve centres should appear? It is worthy of notice that the absorption from the mammæ takes place without any metabolic change, such as occurs in the stomach and liver in the case of materials digested. It is to be presumed that the hypothetical mammary poison is delivered in the brain in its pristine condition and possessed of its full force.

The doctrine is corroborated even more strongly by the successful results of treatment by the injection of a solution of potassium iodide into the udder. The iodide solution may presumably act in one or more of several ways. It is unquestionably an antiseptic, and would tend to arrest or control microbian growth and activity, thus preventing the further formation of toxins. It has a potent deobstruent action on glandular tissue, tending not only to dry up the milk, but to hold in check the leucocytic function of producing dangerous leucomaines. There is reason to believe that with regard to some poisonous ptomaines iodine acts as a direct antidote, probably uniting with these and forming new and comparatively harmless compounds. It manifestly acts in this way in the case of cryptogamic diuresis, and in cerebral congestions arising from spoilt fodder. The iodide tends further to act as a calmative to the nerve centres, and as a diuretic, serving to eliminate the poison that may be present in the blood.

Microbian Infection or Intoxication. The doctrine has been advanced that the disease is either a microbian infection of the nerve centres or a process of poisoning by the absorbed toxins of microbes. Of the two hypothesis the latter is the more acceptable, in view of the fact, that cows in a condition of coma will sometimes recover with extraordinary rapidity. This is more likely to occur in connection with the elimination or exhaustion of a transient narcotic poison, than with a deadly microbe colonized in the brain. This hypothesis is in full accord with the acknowledged success of the iodide injections; with the observation of Bissange, which I can endorse, that certain villages and hamlets habitually furnish cases of parturition fever, while neighboring ones, with the same breeds and apparently the same

management escape ; and with the observations of Russell and Wortley Axe, that the malady will sometimes be suddenly prevented in a herd, by the simple expedient of having the cows moved to a new and previously unoccupied stable, for calving and the first nine days thereafter.

In support of the doctrine of a microbian origin is recalled the fact that the disease almost invariably follows parturition, which opened the way for the introduction of bacteria by the genital passages. This is somewhat invalidated by the fact that it follows the easy parturition, in which there was no chance for the introduction of germs on hands or instruments, and does not follow dystokia in which, without question, germs have been planted abundantly in the interior of the womb. Undue weight should not be given to this objection, as the essential accessory conditions of plethora, etc., are usually largely modified in cases of dystokia.

The *microbiology* of the affection leaves much to be desired. Coureur and Pottiez and later Van de Velde found a streptococcus in the blood. Trinchera, Nocard, and Cozette found the common pus cocci (staphylococcus pyogenes aureus, citreus and albus) a streptococcus and a colon bacillus in the liquid squeezed from the cotyledons, and in the liquid debris on the uterine mucosa. These microbes were not found in other organs. They grew readily in artificial cultures, but we lack the final proof of a successful inoculation on a susceptible parturient subject. The whole subject is therefore still a plausible theory.

We are not however limited to the womb as the only possible field of a pathogenic microbian growth. The frequent presence of microbes in the sphincter of the teat, in the galactophorous sinus, and in the milk ducts inside the mammæ is absolutely proved. Guillebeau found on the mucosa in cases of mammitis three forms of bacillus, to which he attributed the disease. In the New York State Veterinary College we have found mammitis usually associated with a streptococcus in the milk. In one cow in the University herd which gave abundance of good milk, and rarely showed any sign of congestion, streptococcus was constantly present. In cows producing "gassy" curd, V. A. Moore and A. R. Ward found in the milk a bacillus which morphologically and in cultures resembled the colon bacillus (evidently

one of the colon group). In the milk and mammary gland tissue got from other (slaughtered) cows, a micrococcus growing in yellow or buff-colored colonies predominated. (Moore and Ward). That the colon bacillus, so constant in the intestines and manure, is not always found in the milk ducts, would show that in its normal condition it is not adapted to this habitat, but when a variety appears that is so fitted, it appears to be able to maintain its place indefinitely.

With such facts before us, we must allow the possibility of poisoning by toxins of bacteria in the udder, or by compounds formed by the synthesis of such toxins and the leucomaines of the expanding udder, or by the union of the udder toxins with those from the womb. The whole subject of microbian and leucocytic causation of parturient fever is still hypothetical, yet enough is known to show the high probability of such source, and to demand a thorough investigation which will place the subject on a substantial and assured basis.

Nature. Theories of the nature of this disease are numerous and varied, and are largely based upon some restricted or one-sided view of phenomena and lesions. Contamine considers it as the reaction of the surplus of nerve force, which was not used up in the easy parturition. The theory is somewhat fantastic as an explanation of the rapidly developing asthenia and paralysis. Billings explains the cerebral anæmia as due to vaso-constriction of the nervous capillaries produced by the exaggerated excitability of the uterine nerves. But with the easy parturition, and delivery, and the moderate contraction of the womb, without violence or spasm, the theory seems rather insubstantial. Trasbot looks on the affection as a congestion of the myelon, apparently shutting his eyes to the far more prominent encephalic symptoms. Haubner considers it as a cerebral anæmia induced by the vaso-dilatation in the portal system and abdominal viscera generally, the result in its turn of the vacuity of the abdomen, from the expulsion of the fœtus and its connections. But the womb is often found contracted and comparatively exsanguine, the plethoric condition of the cow, suddenly increased by the great mass of blood from the uterine vessels, maintains a marked general blood tension, and finally, the closed box of the cranium cannot have its blood so completely drained from it as can a part

outside such a cavity. Stockfleth attributed the malady to a metro-peritonitis, and the absorption of the morbid products and poisoning, but neither a metritis nor peritonitis is a common accompaniment of the affection.

Franck who accounts for the asthenia by an anæmic condition of the brain, explains the anæmia by a pre-existing congestion and œdema of the rete mirabile at the base of the brain. He claims that sows which have also a rete mirabile in this situation sometimes suffer from parturient fever. He fails to adduce cases in the sheep and goat which also have retia mirabilia. The pregnant sheep may die of an asthenic affection, but usually before parturition. Franck's theory is plausibly based on the anatomical and physiological conditions, for the elaborate network of vessels at the base of the brain, undergoes great distention under increased arterial tension, and with the serous effusion, compresses the brain and drives out its blood.

Palsy of the ganglionic system has been invoked, with succeeding congestion of the myelon and encephalon (Barlow, Kohne, Carsten Harms, etc.). Explanation is made that the supposed excess of nervous force fails of distribution through a lack of conductility of the nerves, and the nerve centres suffer. Binz has even found the spinal roots of the sympathetic surrounded by a thick gelatinoid exudate. The theory is, however, essentially speculative and fails to explain the origin of the disease or its connection with the recognized conditions of its occurrence.

Plethora with Arterial tension and all conditions contributing to this, as already set forth under causes must be allowed a prominent place in considering the nature of the disease. The blood globules in my experience are somewhat smaller than normal, implying the density of the plasma, and implying a direct influence on trophic and metabolic processes. Under these influences the congestion of the encephalic circulation, and notably of the rete mirabile, and a serous effusion, tend first to prostrate the nerve force, and second to render the other intracranial structures anæmic.

The direct action of a *narcotic poison*, leucocytic or microbian, though as yet a hypothesis merely, has much in its favor, on considerations drawn from the observed immunity in particular

buildings, the sudden prostration, the promptitude of certain recoveries and the favorable results of the iodine mammary injections. The presence of sugar in the urine, most abundantly in the worst cases, implies a profound disorder in glycogenic centres (medulla, liver), and primarily no doubt in the bulb.

Lesions. These are exceedingly variable in successive cases. Congestion and effusion in the meninges, cerebral or spinal, in the rete mirabile and choroid plexus have been often noticed, and exceptionally clots of extravasated blood. In certain cases congestion and pink discoloration of portions of the brain substance (cerebral convolutions, bulb, ganglia) with marked puncta vasculosa, are found, while in others the greater part or the whole of the encephalon is anæmic. The puncta in such cases, large and dark, on the surface of the section, promptly enlarge until they may form distinct drops.

In the *lungs* areas of collapse, and of dark red congestion and infiltration are common, mostly as the result of the entrance of alimentary or medicinal matters into the bronchia owing to palsy of the pharynx. Such materials can be found in the bronchial tubes.

The *third stomach* and the *large intestine* may be impacted, the contents more or less baked and glossy on the surface, and coincident congestions of the mucosa are not uncommon. In some instances, however, the contents are soft and pultaceous and the absence of mucous congestions is remarkable.

The *womb* rarely shows characters differing from the condition which is normal to the first few days after parturition.

The *blackness and thickness of the blood* has been noted by practically all observers. This is partly the result of its density, but doubtless also of the undetermined toxins which are operative in the disease.

Yellowish *gelatinoid exudates* have been found in the subdorsal and sublumbar regions, as well as the cranium and spinal canal.

Glucose appears to be constantly present in the urine, and in excess in the more violent and fatal cases: from 1.19 grm. per litre in slight cases to 41.8 grms. in a fatal one (Nocard). Albumen may be present, though probably only when local inflammation has supervened.

Symptoms. The conditions of the attack should be noted.

This is a disease of the first six days after parturition, rarely seen in the second week, and never after the fourteenth day. It is very exceptional before parturition, yet Müller quotes 47 cases in 1107 births. The breed, condition, milking qualities, plethora, feeding, etc., of the patient are, as already noted important data in diagnosis. The onset is sudden without premonitory symptoms.

Two very distinct types are met with, the *comatose* and *violent or spasmodic*, which, however, merge into each other by insensible gradations, and may follow each other.

From twelve to seventy hours after an easy parturition there suddenly appear signs of discomfort. Feeding and rumination cease, the calf is neglected, there may be plaintive moaning, the eyes seem dull and clouded, the eyelids drooped, the conjunctiva red, the pulse normal for parturition, sometimes extra strong, the breathing excited often with moans or grunts. The senses are dulled, the walk is unsteady, the feet being abducted and planted like clumps, or the legs sway, perhaps cross each other, remain semi-bent, and soon give way leaving the animal prostrate, resting on the sternum and abdomen, or later on the ribs, with head extended. Attempts may still be made to rise, but this is rarely accomplished unless when improvement sets in. This is the condition in which the patient is usually found, being the first to be noticed by the owner. The bowels are torpid, the urine retained in the bladder, and the animal may remain thus in a drowsy condition, without changing from the sterno-ventral decubitus, or dropping the head on the ground until improvement sets in. The head rests on the shoulder or upper flank. If held outward or forward the upper border of the neck has an S shaped outline.

More commonly the somnolence increases, passing into a complete torpor and insensibility, the eye may be touched without causing winking, pricking or other injury causes no further response, the patient turns upon its side, with its head extended on the ground. She may lie in this condition with no sign of vital activity save pulsation and breathing, and the latter is liable to be slow and stertorous by reason of the paralysis of soft palate and larynx. The jugulars usually show a venous pulse. Fermentations in the inactive paunch cause the evolution of gas with tympany, which still further obstructs the

breathing, and reacts injuriously on the nerve centres. The normal eructations from the rumen may continue, with liquids and floating solids, and in the paralytic state of the throat these too often pass in part into the bronchia, causing septic bronchitis and pneumonia. The same is liable to follow the administration of liquids, the irritant drugs passing into the larynx, trachea and lungs. The pulse becomes soft, small and finally almost imperceptible. It may be 50, 60 and upward.

In favorable cases, defecation may still occur, or the rectum once emptied may fill again through the continuance of peristalsis, the milk continues to be secreted, and in one to four days, spontaneous defecation and micturition may be resumed, and the patient may get on its limbs and commence feeding. There is usually at first a little weakness of the limbs, but this is transient and health is restored in a very short time. The suddenness of the improvement is often as marked as of the attack. The patient is left prostrate and insensible, without giving any response when the eyeball is touched and in two or three hours it is found on its feet, eating, with eyes bright and clear.

Some patients, however, are restored to ordinary sensation, intelligence and appetite, while the hind limbs remain paralytic, or paretic, and the station and gait both weak and uncertain for days or even weeks. In such cases there have been presumably structural changes in the nerve centres, which require time for repair.

In fatal cases, death may occur quietly from apoplexy, cerebral compression, or narcotism, or it may be preceded by a period of marked excitement or disorderly muscular movements. Lifting of the head, throwing it alternately on the shoulder and on the ground, trembling of head, members and body, cramps or jerking of the limbs or of other parts, drawing the hind limbs up against the abdomen, and again extending them, rolling of the eyes, loud, noisy, irregular, embarrassed breathing and a running down pulse are often marked features.

The temperature range is peculiar. At the start there may be some hyperthermia 103° or 104° ; with the advance of the disease it tends to become lower, 98°, 96°, or 94°. When improvement sets in, it rises again promptly to the normal.

Cadeac describes a special form which is ushered in by great

restlessness, bellowing, throwing the head to right and left, grinding the teeth, sucking the tongue, salivation, licking of certain parts of the body, spasms in the neck, back or limbs, and prompt recovery, or lapse into the comatose condition as above described. It proved less fatal than the ordinary comatose type, but seems to depend on similar conditions.

Prognosis. Mortality. The disease is very deadly, the mortality in time past having reached 40, 50 or even 60 per cent., the gravity increasing as the disease set in nearer to parturition. Cases occurring on the first or second day were mostly fatal, those at the end of the first week were hopeful, and those occurring during the second week were very hopeful. With the Schmidt (iodine) treatment the mortality is claimed to be reduced to 16 or 17 per cent.

Prevention. Measures directed toward the lessening of plethora tend to remove one of the most fruitful causes of the disease and though not invariably successful, are yet of great value. The most direct is the *abstraction of blood* in the last fortnight of pregnancy, to the extent of 6 or 8 quarts. This tends to secure a lessening of the blood tension, and blood density, but there is the drawback of a created tendency to a subsequent increase in blood formation to make up the loss. This measure should be reserved for cows that are very plethoric, extra heavy milkers and such as have already suffered from the disease.

Purgatives will measurably secure the same end without the same degree of danger. One to two pounds of Epsom or Glauber salts in the last week of gestation, or at latest when labor pains set in, tend not only to remove solid or impacted masses from the first and third stomachs, and inspissated contents from the large intestines, but to secure a free depletion from the portal system. If not before, this should always be given immediately after parturition to cows in extra high condition, heavy milkers, and that have had a short and easy delivery.

Restriction of food for a week before and as long after parturition is of equal importance. A very limited supply of aqueous, easily digested, and laxative food (roots, sloppy bran mashes, fresh grass, ensilage) will meet the demand.

Exercise in the open air is of great value in giving tone to the muscles, and especially the nervous system, and in stimulating the emunctories and other functions.

In the cold season *protection against cold draughts and chills* must be seen to, and in the hot season the avoidance of an excess of solar heat and above all of the confined impure air of the barns.

At midsummer and later, there is often great danger in the rich clover and alfalfa pasture, or soiling crop, with which the cow will dangerously load her stomach, and the only safe course is to remove predisposed animals and shut them up in a bare yard or box-stall. Under such simple precautions herds that had formerly suffered severely, have had the disease virtually put a stop to.

In individual cases other measures are indicated. When the udder has reached an enormous size and development, and is gorged with milk, days before parturition, it should be systematically milked. The irritation in the gorged gland is quite as likely to induce premature parturition, as is milking, and, at the worst, the result is not so bad as an attack of parturition fever.

Basing his advice on the fact that parturition fever does not follow a case of severe dystokia, Cagny applies sinapisms on the loins, croup and thighs of a fleshy, plethoric, heavy milking, parturient cow. Proof of their efficacy is not obtainable.

Felizet advises leaving the calf with its dam for one week. Kolme doses the cow with nux vomica: Harms, with tartar emetic.

In view of the probability of a bacterial infection the cow should be taken to a clean, pure, well-aired stable a day or two before calving, having been first cleansed from adherent filth, and sponged all over with a 4 per cent. solution of carbolic acid.

To prevent diffusion of infection Bournay recommends antiseptic injection of the womb immediately after calving. Bissauge adds that the stable should be disinfected after every case of parturition fever, the manure carefully removed and the ground scraped and well watered with a disinfectant.

For fleshy, plethoric, predisposed cows, the iodine injection of the udder should be applied immediately after calving. A measure of this kind which is so successful as a curative agent, and which brings such circumstantial evidence of the production of a poison (leucomaine or ptomaine) in the mammary gland, can hardly fail to be even more effective as a prophylactic than as a therapeutic resort.

Treatment. With the state of plethora and congestion about the head in the early stages the question of *bleeding* at once arises. If early enough while there is a full bounding pulse, and as yet no sign of great loss of muscular control it is often very beneficial, as much as 6 quarts or more being withdrawn. It is well however to avoid cording the neck, which must increase the vascular tension in the brain, and to trust rather to digital compression of the vein. The blood should be drawn from a large opening in a full free stream, and may be stopped when the pulse softens. In the more advanced condition, with paralysis and more or less dulling of the senses, or coma, bleeding may be dangerous rather than useful. There is then serious pressure on the brain, with serous effusion, and perhaps blood extravasation, and in any case anæmia, and this latter may be dangerously or even fatally increased by the lessening of the blood pressure, without any compensating advantage in the way of reabsorption of the effusion. In such cases eliminating agents are a safer resort.

Purgatives commend themselves, but with the drawback of a too tardy action. Now however with the peristaltic stimulants given hypodermically this objection is largely obviated. Pilocarpin 1½ gr., and eserine 3 grs. will often secure a noticeable movement of the bowels in the course of fifteen minutes, implying a corresponding motion onward in the bowels more anteriorly, and even of the contents of the gastric cavities. If there is already palsy of the muscles of deglutition, this may be repeated several times at intervals of four or five hours. If however deglutition is still well performed a purgative of one or two pounds Epsom salts, with 10 drops croton oil, and 1 oz. oil of turpentine may be given by the mouth. Should this operate, it will supplement and carry on even more effectively the work of the hypodermic agents, and even lessen the density, plasticity and tension of the blood and act as a potent derivative from the brain.

A compromise may be made by giving aloes 2 ozs., croton oil 20 drops in bolus; or 1 to 2 ozs. sulphate of soda in solution may be injected subcutem.

In any case oil of turpentine or other antiseptic is of great value in the stomach in preventing fermentation and tympany, and thereby obviating a whole series of troubles such as: cerebral disturbance by nervous shock and blood pressure; impaired res-

piration and hæmatosis by pressure on the diaphragm ; and eructations of food to the pharynx and its inhalation or gravitation into the lungs.

It is always well to clear out the rectum by injections, when if there is any indication of pharyngeal paralysis most of the remedies may be given by this channel.

Stimulants (ammonia carbonate, alcohol, anise, fennel, ether, nux, etc.) have been largely employed by the month and may be by the rectum, In the absence of spasms I have relied largely on nux or strychnia.

When the skin chills, some have sought to heat it by enveloping the prosterior half of the body in cotton or wool soaked in turpentine, by applying sinapisms, or by moving over the surface a warming-pan containing red hot charcoal.

More generally *cold* in the shape of cold water, ice or snow has been applied *to the cranium and spine*. Theoretically the anæmic brain might be thought to forbid this, but clinically it often operates well, possibly by inducing a sympathetic contraction of the vessels in and around the nerve centres and thus indirectly favoring the resumption of active circulation and the reabsorption of effusions.

An *elevated position of the head* is no less important. It favors the return of blood from the brain by gravitation, and in this way improves the intracranial circulation, and the resumption of normal function. A halter, or a rope around the horns, may be tied to a beam overhead, or the head may be laid on thick bundles of straw which will keep it up to or above the level of the chest, and in this way not only is gravitation ensured, but the brain is protected against the violent blows and concussions, which come from dashing the head on the ground.

Frequent *rubbing of the udder and drawing of the milk*, is an excellent means of depletion, a removal of a source of irritation, and presumably an extraction of part of the offending poison. It should never be neglected. But of all known methods of treatment the iodine injection furnishes the greatest hope of success.

Injection of the mammæ with Iodine. Iodide of potassium 100 grains (200 grs. in the case of a very large udder) are dissolved in a quart of water which has been boiled for 15 minutes, the solution cooled to 104° F. and injected in equal parts into the

four quarters, which have been just milked out clean. The glands are then manipulated so as to work the solution into all the recesses of the milk tubes and follicles. If the patient does not get on its legs at the end of twelve hours, the glands may be milked out and injected anew. In nearly 2000 cases the recoveries reached an average of nearly 83 per cent. In serious or advanced cases with structural changes of a grave nature, a good result cannot be hoped for. The injection does not forbid the concurrent use of other approved measures.

The injection is easily made with a caoutchouc tube of five feet long fitted to a teat tube at one end and to a funnel at the other. The tube is inserted in the teat, and the funnel at a height of five feet receives the liquid, which readily passes into the teat. When ready to pass the tube from one teat to another, an assistant pinches the caoutchouc tube just below the funnel, until the insertion has been made. Every precaution must be taken against sepsis. The udder, teats and hands, must be washed with soap, and treated with a 3 per cent. solution of lysol. The teat tube and funnel are boiled. The caoutchouc tube is washed and irrigated with a solution of mercuric chloride (1 : 1000), and then with one of boric acid (3 : 100).

DISEASES OF THE EYE.

DESIRABLE FEATURES IN THE EYE.

The eye in the physiognomy. Broad forehead Full eyes. Both eyes alike. Iris smooth, lustrous. Media translucent. Pupil sensitive to light. Convexity median, uniform. Pupil black in ordinary light. Lids open and mobile. Sclera light pink. Tears clear, limpid without overflow. Lids thin, delicate, margins evenly curved. Whole eye responsive to moving objects. Defects: small eye: semi-closed, thick, sluggish lids; convex cornea: sunken eye: projecting eye: weeping eye: blear eye: watch eye: irresponsive iris: dilated pupil: unequal eyes: flat cornea; ovoid cornea.

Much of the expression of the face depends upon the eyes, and in animals as in man it is difficult to find compensations for a forbidding countenance. Perfect, sound, intelligent eyes are always pleasing; imperfect, defective, sunken or lifeless eyes mar the whole expression. The following points may be specially noted:

1st. **Ample breadth between the orbits.** This is of great importance in the horse, in which we seek for intelligence, courage and indomitable energy. This confirmation does not indicate the size of brain, as the cranium is situated higher up, but by placing the eyes well outward, it indicates a wider range of vision, and usually implies large, clear eyes, and since interdependent parts tend to correspond in development and quality, this commanding vision bespeaks a large, active brain, intelligence, docility and activity.

2d. **Full, prominent eyes.** This may be excessive, either through primary conformation or disease. Abnormal convexity of the cornea implies myopia. But within normal limits the prominent eye suggests good health, condition and vigor, with ample cushions of fat under the bulb and a sound, well-developed condition of the eyeball and its muscles.

3d. **Both eyes equal in all respects.** Any variation in size, shape, color, fullness, clearness or in any other respect is at

best unsightly, and implies not only defect but often disease as well.

4th. **The iris should be lustrous, uniform in color and even in surface.** Whether dark brown as in the horse, or yellow as in the dog, it should be brilliant. Any part that lacks lustre, being lighter brown, or yellow and dull like a dead leaf, usually indicates previous disease and a tendency to further trouble. Albinos and those in which the pigment is congenitally absent in patches must be considered as exceptions, yet, even in them, the peculiarity cannot be held to add to the beauty.

5th. **All the Media (Cornea, aqueous humor, lens and vitreous) must be perfectly clear and translucent.** The slightest cloudiness or opacity in any of these is a serious blemish and usually indicates disease, past or present.

6th. **The pupil should promptly and freely respond to light and darkness by contraction and expansion.** Absence or tardiness of movement indicates impaired vision, from disease of the eye, its nerves, or their nerve centres.

7th. **Each cornea should have a median convexity, uniform in all directions implying the absence of myopia, preshyopia and astigmatism.** Any deviation from this will interfere with the perfection of sight, and endanger shying and other troubles.

8th. **Under ordinary light the pupil should appear black throughout.** In the larger animals such dilation of the pupil as to expose the tapetum lucidum under such circumstances implies impaired vision (amblyopia, amaurosis), inflammation of the iris or undue intraocular pressure. A white color or spot shows cataract.

9th. **The lids must be open and mobile without excessive dilation.** Tardily moving or semi-closed lids, distorted by scar or angle, everted or inverted, are unattractive and usually imply disease in the eye, nerves or brain.

10th. **The unpigmented portion of the sclera should be light pink.** The dark red of congestion and the pallor of anæmia are equally objectionable.

11th. **The tears must be clear, limpid and confined within the lower lid.** Any milkiness, flocculency or overflow is indicative of disease.

12th. **The eyelids must be thin, delicate, evenly and uniformly curved along the borders, and fringed by an abundance of strong, prominent and well directed lashes.** Puffiness or swelling betrays inflammation, dropsy, anæmia, parasitism or other disorder, angularity of the upper lid an internal ophthalmia, and depilation or wrong direction of the lashes, local disease.

13th. **The eye should respond instantly, by movement, to new objects and noises, without showing undue irritability or restlessness.** The intelligent apprehension of the objects will introduce an aspect of calmness and docility.

DEFECTS, BLEMISHES AND ABNORMALITIES OF THE HORSE'S EYE.

Some of these may be present in the absence of actual disease, and yet prove so objectionable that they disqualify the animal for any use, in which style or æsthetic appearance is demanded. Among such sources of disqualification may be noted :

1st. **The small eye.** One or both eyes may appear small because of internal pain and retraction within their sockets, or from actual atrophy or contraction of the eyeball, the result of deep seated disease, or the organ may be congenitally small, and deep seated in the orbit, and the thick tardy eyelids may have a narrow opening through which they can only be partially seen. This last condition usually implies a dull lymphatic constitution, low breeding and a lack of intelligence, docility and vigor.

2d. **The semi-closed eye with thick, coarse, sluggish lids.** In this case the bulb may be not unduly small, yet as it is not freely exposed it conveys the same general expression to the observer. Like the small eye it indicates low breeding, lack of intelligence or docility and often stubbornness or even vice.

3d. **The convex eye.** In this the transparent cornea describes the arc of an unduly small circle, suggesting a conical form and projecting unduly beyond the margins of the lids. It implies imperfect vision, myopia, and, it is alleged, low breeding and lack of alertness.

4th. **The sunken eye.** This has been already referred to under **the small eye.** The eyelids are usually flaccid, the upper being drawn in by its levator so as to form an angle, and the edges of the orbit are somewhat prominent. It is seen in old, worn out animals, which have lost the pads of fat in the depth of the orbit, and more commonly in animals that have suffered several attacks of recurrent ophthalmia.

5th. **The projecting eye.** In this case the lids are unduly contracted and the eye protrudes between them so as to show a large amount of sclerotic around the transparent cornea. This may be due to nervous strain and suffering but, however produced it is decidedly unsightly and objectionable.

6th. **The weeping eye.** This is always a condition of disease. It may be due to irritant gases, or solid particles, to inturned cilia, everted lids, conjunctivitis or a variety of other conditions. A careful examination may show whether it is only a transient and remediable fault of a good eye or a permanent and irremediable defect.

7th. **The blear eye.** With swelling and scabbing of the edges of the lids and Meibonian glands, and congestion of the adjacent conjunctiva, there is usually some blurring of the surface of the transparent cornea. The trouble is mostly chronic and constitutes a serious objection.

8th. **The watch eye.** In this, as in the albino, there is a lack of pigment, so that the iris and sclerotic are white or bluish white in part or in whole. Such an eye may be good and durable, but not beautiful nor attractive.

9th. **Blindness of one or both eyes.** In all such cases the pupil remains fixed and immovable, showing no accommodation to light and darkness, and there is a lack of prompt responsiveness on the part of the eye to sounds and objects. In amaurosis, glaucoma and cataract especially, the pupil remains widely open, and alert movements of the ears are employed to make up for the lack of sight. The condition often comes from internal ophthalmia, such as the recurrent form, and is associated with atrophy of the bulb.

10th. **Eyes of unequal size.** This usually implies serious disease in one, not infrequently recurring ophthalmia.

11th. **Too flat corneal surface.** In this case there is a manifest lack of the normal projection, the anterior surface of the cornea describing the arc of a larger circle, the visual rays coming from a distance alone converge on the retina and presbyopia occurs. In this as in myopia and other visual imperfections a horse is liable to stumble and, if nervous, to shy.

12th. **Ovoid Cornea.** In such cases the front of the transparent cornea has an ovoid outline the arc formed by it in one direction being that of a greater circle, than the arc which crosses this at right angles. In consequence of this, the rays impinging on the outer portions of these respective arcs do not converge to the same point on the retina and a blurred and imperfect image results. This astigmatism causes the subject to stumble and, if nervous, to shy.

SYSTEMATIC INSPECTION OF THE EYE.

System in Examination. Eyelids: cilia: lachryneal puncta: mucosa, light pink, brick red, yellow, puffy, dropsical: Ciliary vessels deep, immovable; nictitans; transparent cornea equally smooth, glossy, with clear image at all points: foreign body on cornea: corneal ulcer: opacities in aqueous humor: iris and pupil: corpora nigra: changes in passing from darkness to light: pupillary membrane: adhesions of iris: intraocular pressure: contracted pupil: hole in iris. Oblique focal illumination of cornea, aqueous humor, iris, lens, Purkinje-Sanson images.

In examining animals for soundness and especially the horse or dog, the condition of the eye must be made one of the most important subjects of inquiry, as a disease or defect may render the animal altogether unsuited to the object to which it is destined. As in every other field of diagnosis thoroughness is largely dependent on the adoption of a system which will stand in the way of any flaw being too hastily overlooked. Many of the points to be noted will be decided at a glance, yet this does not obviate the necessity of turning over in the mind, in succession, the different points of inquiry, and directing the necessary attention, however hastily, to each in turn. The following points should be observed:

1st. **Are the eyelids swollen, hypertrophied or faulty in form, position or movements.** Faults as thus indicated may

imply any one of a great variety of disorders which should be followed out to their accurate diagnosis. It may be bruises, lacerations, punctures, parasites, conjunctivitis, keratitis, dropsy, anæmia, hepatic or intestinal parasitism, nephritis, paresis, entropion, ectropion, etc.

2d. **Inspect the cilia as regards form, size and direction.** Absence or wrong direction may imply disease of the Meibonian glands, infective inflammation, demodex or other acarian infesting, or turning in or out in inflammatory conditions.

3d. **See that the lachrymal puncta are open and that there is no overdistension of the sac.** The overflow of tears and the swelling of the caruncle and of the area beneath it will often indicate such trouble. In its turn it may imply inflammation of the duct, and obstruction by the tenacious mucopurulent product, or it may imply merely obstruction of its lower end by a dried scab. This last may be seen in the horse, on the floor of the false nostril at the line of junction of the skin and mucosa, and in the ass, higher up on the inner side of the ala nasi. In exceptional cases it may be desirable to pass a stilet through the canal from the puncta downward or from below upward to determine whether it is pervious.

4th. **Determine the vascularity of the conjunctiva.** When free from pigment as it habitually is in pigs and birds this is easily done, while in animals like the horse, in which the bulbar portion, which covers the sclerotic, is largely pigmented, we can scrutinize only the pigment free parts. **In health** there should be only a few, fine, pink vessels which move with the mucosa when pressed aside on the bulb. **In congestion** the surface may appear brick red, and the vessels are irregular, large, tortuous and are seen to anastomose at frequent intervals. These move on the bulb when pressed. The congestion is usually deepest on the palpebral mucosa and in the *cul de sac*, and may be whitened for an instant by pressure through the eyelid. To expose the conjunctiva the right fore finger and thumb may be pressed on the upper and lower lids respectively of the left eye, and the left finger and thumb for the right, allowing them to slide backward above and below the eyeball. Another method is to sieze the cilia and edge of the upper eyelid between the finger and thumb,

and draw it downward and outward from the bulb, and then deftly invert it over the tip of the finger. In the old the unpigmented conjunctiva may appear yellow from the presence of subconjunctival fat, or this may appear at any age from hepatic disease (distomatosis) or icterus. It is swollen, or dropsical in anæmia, distomatosis, etc.

5th. **Examine the ciliary vessels whether they are congested or not.** These are distinguished from the conjunctival vessels in that they radiate in straight lines outward from the margin of the transparent cornea and do not move on the sclerotic under pressure. They are enlarged and very red in congestion of the ciliary circle and in iritis. In eyes devoid of pigment over the sclerotic, there is usually a circular, narrow, white zone between the congested area and the margin of the transparent cornea.

6th. **Examine the Membrana Nictitans.** See that its free margin is uniformly smooth, even, and thin and that there is no swelling, congestion nor morbid growth on any part of the structure.

7th. **See if the transparent cornea is perfectly and uniformly smooth, transparent and glistening and if it reflects clear, erect images of all objects in front of it.** The image of a round object which shows any irregularity in the curvature of its margin implies a deviation from an uniform curvature of the cornea: the image narrows in the direction of the smaller arc and broadens in the direction of the larger one (see keratoscopy, and corneal astigmatism).

8th. **A foreign body on or in the cornea** may be recognized in a good light, but better and more certainly under focal oblique illumination (see this heading).

9th. **A corneal ulcer** may be similarly recognized. It is made more strikingly manifest by instilling into the lower *cul de sac* a drop of a solution of fluorescin and rubbing it over the eye by moving the eyelids with the finger. This will stain the whole cornea. If now the excess of stain is washed away by a few drops of boric acid, the healthy part of the cornea is cleared up and the ulcer retains a bright yellowish green tint.

10th. **Opacity or Floating objects in the aqueous humor** (flocculi of lymph, pus, pigment, blood, worms) are always to be looked for. They may be detected by placing the eye in a favor-

able light. They may be still more clearly shown under *focal illumination* (see below).

11th. **Changes in the iris and pupil** may also be noticed in a good light. The surface should be dark in the horse, and of the various lighter shades in the smaller animals, but in all alike clear, smooth and polished, without variation of shade in spots or patches and without bulging or irregularity at intervals. Apart from the congenital absence of pigment in whole or in part, which may be found in certain sound eyes, a total or partial change of the dark iris of the horse to a lighter red, brown or yellow shade implies congestion, inflammation, or exudation. The **corpora nigra** in the larger quadrupeds should be unbroken, smooth, rounded, projecting masses outside the free border of the upper portion of the iris. It should show a clear, polished surface like the rest of the iris. **The pupil** should be evenly oval with its long diameter transversely (horse, ruminant), circular (pig, dog, bird), or round with an elliptical outline on contracting and the long diameter vertical (cat). It should contract promptly in light and dilate as quickly in darkness. Place the patient before a window, cover one eye so as to exclude light, then cover the other eye with the hand and quickly withdraw: The pupil should be widely dilated when the hand is withdrawn and should promptly contract, and it should actively widen and narrow alternately until the proper accommodation has been secured. Any failure to show these movements implies a lesion in the brain, optic nerve, or eye which impairs or paralyzes vision, interferes with accommodation or imprisons the iris. In locomotor ataxia the pupil contracts in accommodation to distance, but not in response to light.

12th. **Other causes of pupillary immobility** include: (a) Permanence of a pupillary membrane, which has remained from the fœtal condition and may be recognized by oblique focal illumination and invariability of the pupil: (b) Adhesion of the iris to the capsule of the lens—complete or partial—in the latter case the adherent portion only remains fixed, while the remainder expands and contracts, giving rise to distortions and variations from the smoothly curved outline: (c) Adhesion of the iris to the back of the cornea—complete or partial—and leading to similar distortions: (d) Glaucoma in which intraocular pressure

determines a permanent dilatation of the pupil and depression of the optic disc: (e) The pupil is narrowed in iritis, and is less responsive to atropia or other mydriatic: (f) Lesions of the oculo-motor nerve may paralyze the iris and fix the pupil. The first three and the fifth of these conditions may be recognized by the naked eye, alone, or with the aid of focal illumination, the fourth may require the aid of the ophthalmoscope and the sixth which cannot be reached by such methods, might in exceptional cases be betrayed by other disorders of the oculo-motor nerve (dropping of the upper eyelid, protrusion of the eyeball, squinting outward).

13th. **Coloboma** (*fenestrated iris*), **and lacerated iris** are recognizable by the naked eye in a good light, or by the aid of focal illumination.

14th. **Tension of the eyeball** (*Tonometry*). Elaborate instruments constructed for ascertaining ocular tension are of very little use in the lower animals. The simplest and most practicable method is with the two index fingers placed on the upper lid to press the eyeball downward upon the wall of the orbit using the one finger alternately with the other as if in search of fluctuation. The other fingers rest on the margin of the orbit. All normal eyes have about the same measure of tension and one can use his own eye as a means of comparison. The educated touch is essential. In increased tension, the sense of hardness and resistance, and the indisposition to become indented on pressure is present in the early stages of internal ophthalmias (iritis, choroiditis, retinitis), phlegmon of the eyeball, glaucoma, hydrophthalmos, and tumors of the bulb.

Oblique Focal Illumination.

This is so essential to clear and definite conclusions and is so easily practiced on the domestic animals that every veterinarian should make himself familiar with the method. The method is based on the fact that when two perfectly transparent media touch each other a reflection of luminous rays takes place only at the surface. But in case any opacity exists in any part of the thickness of one of these media, it reflects the rays from its surface no matter what may be its position in the medium. Thus corneal opacities appear as gray blotches and under careful focal illumi-

nation it may be determined whether these are on the conjunctival surface, in the superficial or deeper layers of the cornea or in the the membrane of Descemet. Similarly cloudiness or floating objects in the aqueous, reflect the luminous rays, and so with opacities in the lens or its capsule, or in the vitreous. In the same way the surface of the iris and corpora nigra may be carefully scrutinized. For satisfactory examination of the media, back of the iris, the pupil should be first dilated, by instillation under the lid of a drop or two of a 3 per cent. solution of atropia, and the examination proceeded with twenty minutes later. Homatropin is preferable to atropin as being less persistent in its action, and less liable to produce conjunctivitis. If it fails to produce the requisite dilatation, it may be followed by a drop of a 4 per cent. solution of hydrochloride of cocaine, which will secure a free dilatation, lasting only for one day in place of seven days as with atropin. The cocaine further removes pain and favors the full eversion of the eyelids.

The instruments required for focal illumination are a biconvex lens of 15 to 20 diopters, and a good oil lamp or movable gas jet. The light of the sun is not satisfactory. The examination ought to be conducted in a dark room, or less satisfactorily in semi-darkness. The lamp is held by an assistant at the level of the eye to be examined, either in front or behind, or first one and then the other, so that the rays of light may fall upon the eye obliquely. If the lids are kept closed it may be necessary to expose the cornea by pressing on the lids with the finger and thumb. The light is held 8 or 10 inches from the eye and the lens is interposed between it and the eye and moved nearer and more distant until the clearest illumination has been obtained of the point to be examined. In this way every accessible part of the eye may be examined in turn. The examiner may make his results more satisfactory by observing the illuminated surface through a lens magnifying three or four diameters. It is important to observe that the eye of the operator must be in the direct line of reflection of the pencil of light.

Cornea. By focusing the light in succession over the different parts of the surface of the cornea, all inflammations, vascularities, opacities, ulcers, and cicatrices will be shown and their outlines clearly defined. By illuminating the deeper layers of the cornea

proper, the lesions of keratitis, opacities, ulcers and cicatrices will be shown. To complete the examination of the cornea the light should be focused upon the iris so that it may be reflected back through the cornea. This will reveal the most minute blood-vessels, any cell concretions on Descemet's membrane, or any foreign body in the cornea which may have been overlooked.

Aqueous Humor. Unless the cornea is densely opaque, the anterior chamber can be satisfactorily explored by the oblique focal illumination. The cloudiness or milkiness of iritis or choroiditis furnishes a strong reflection from its free particles of floating matter, its blood and pus globules, and its flocculi of fibrine. The latter have usually a whitish reflection, the blood elements a red (hypohæma), and the pus a yellow (hypopyon). The writhing movements of a filaria scarcely need this mode of diagnosis. Sometimes, and especially in the horse, detached flocculi of black pigment are found floating free in the aqueous and are highly characteristic.

By this illumination one can easily determine the distance of the cornea from the iris and lens (depth of anterior chamber) which is lessened by the forward displacement of iris and lens in undue tension in the vitreous (glaucoma, retinitis, tumors, bladderworms), or of the iris alone, in irido-choroiditis with accumulation of exudate in the posterior chamber of the aqueous. The depth of the anterior chamber may increase in cases of luxation or absence of the lens or softening and atrophy of the vitreous.

The adhesion of the iris to the back of the cornea may be satisfactorily demonstrated by focal illumination.

Iris. The lesions of the iris are exceedingly common in connection with recurring ophthalmia in the horse, and examinations in the intervals between attacks are of the greatest importance. The eye should be examined as already stated, at a window or door, and if available by the aid of a mirror. Any changes in form or color, or luster should be carefully noted, any tension of the eyeball, or angularity of the upper lid, and any slight blue opacity round the margin of the cornea. Then the prompt or tardy response of iris and pupil to light and darkness must be made out. To complete the test the eye should be treated with homatropin for three-quarters of an hour and with cocaine for ten or fifteen minutes, and then subjected to oblique focal illumination.

With partial posterior synechia the rest of the pupil is found dilated while the attached portion extends inward remaining fixed to the capsule of the lens. If the synechia is complete no dilatation whatever has occurred. The edges of the adherent iris extend inward as adherent projections, and any exposed portion of the lens is likely to show black points, the seat of previous adhesions that have been broken up. In such cases the periphery of the iris bulges forward from the accumulation behind it of aqueous humor or inflammatory exudate which cannot escape. The discoloration of the iris as the result of inflammation, stands out more definitely under the fuller illumination.

Crystalline lens. In exploring the crystalline lens or its capsule for opacities (cataracts) oblique focal illumination can be employed to the very best advantage, if the pupil has first been widely dilated by homatropine and cocaine. The light is concentrated on all parts of the anterior capsule in turn, then in succession on the different layers of the lens at all points and finally on the posterior capsule. The striking reflection from any points of opacity whether pigmentary, gray or pearly white is diagnostic, not only of cataract, but of its exact position—anterior or posterior, capsular or lenticular.

Purkinje-Sanson images. If the flame of a candle is passed in front of the eye, at a suitable distance, in a darkened room, and the observer looks into the eye obliquely from the opposite side, he observes three images of the flame, reflected respectively from the front of the cornea, from the anterior surface of the lens and from the back of the lens. The image from the cornea is erect, bright and clearly defined: that from the front of the lens is still erect, but larger and dimmer, because the difference between the index of refraction of the aqueous and lens is very slight: the third image, which is smaller and clearer than the last, is inverted, because the surface of reflection on the back of the lens acts as a concave mirror. The beginner may at first find it difficult to make out the image from the front of the lens but with a little care he can do so, and then by moving the light he should cause each image to pass over all parts of the reflecting surface in turn. Any unevenness or opacity at any point of the reflecting surface, will cause the image reflected from it to become blurred or diffused as it passes over it and thus, not the

existence only, but the exact seat of such opacity is easily demonstrated. Opacities on the cornea cause blurring of the bright, erect image of the flame as it passes over that part: opacities on the anterior capsule of the lens blur the dim, erect image when passed over them: finally, opacities in the body of the lens or on its posterior capsule, blur the small inverted image as it passes over them.

Add to this method the oblique focal illumination and the images of the flame reflected from the three mirror surfaces (cornea, anterior and posterior lens surfaces) are made much clearer and more distinct than in any other way. To do this effectively the convex lens should be held so as to focus the flame in the air nearly in front of the cornea. The Purkinje-Sanson images are made very definite and clear. If the lens is approached nearer to the eye so as to throw the image of the flame within or behind the lens, a gray phosphorescent streak of light is seen in the depth of the pupil. This is due to the laminated structure of the lens as well as to the fact that the lens itself is not perfectly transparent even in its normal condition. The absence of the lens or its dislocation and displacement downward, below the line of vision may be inferred from the absence of this gray luminous reflection under this test.

OPHTHALMOSCOPE.

Principle of ophthalmoscope: Angle of incidence and angle of reflection in same line, light close to one side of the eye, reflected into it by a mirror, having a hole in the centre for eye of observer. Opacities show a dense white in transparent media: if in front of lens move with rolling of eye: if behind in opposite direction. To see fundus must use biconvex lens. Emmetropic eye: myopic: hypermetropic. Static refraction. Mydriatics: Atropine, homatropine, daturine, duboisine, hyoscyamine.

In the healthy eye, the pupil and iris, and in cataract, even the opaque anterior capsule of the lens, can be clearly seen. The reflection of the pupil, however, is dark and no object back of the iris can be observed. The reason of the difference is that the rays of light, entering through the whole cornea, are reflected at the same angle at which they strike the surface of the iris. The

angle of incidence is the same as the angle of reflection. In the hollow fundus of the eye, however, the light entering through the narrow pupil, strikes the fundus at a point which is hidden from the observer, behind the iris, and being reflected by the concave fundus, in exactly the same line along which it entered, it remains invisible. To illuminate the fundus of the eye, for the observer, his line of vision must be made exactly the same as that in which the pencil of light enters the fundus. This is best effected by reflecting the light into the eye by the aid of a small plane or concave mirror having a hole in the center through which the observer looks into the pupil. The concave mirror gives the stronger illumination, but the plane article is more easily manipulated and tends to cause less active contractions of the pupil. This is the simplest form of ophthalmoscope. For careful examination of the fundus of the eye, it is best to have the subject in a dark chamber, with a single large flame of an oil lamp or gas (electric light with an obscure globe may answer). The light is held behind and on the same side as the eye to be examined, at the level of the eye and the perforated mirror and the eye of the observer are kept from 10 to 20 inches in front of the eye and also at the same level. For the horse or ox under favorable conditions in a stall, the light of day coming from a fan-sash over the door may serve the purpose. Nicholas assures us that it may be accomplished even under the shadow of a shed or a tree. In such a case it is better not to have too much glare of light as the reflection from cornea and lens may prevent accurate observation. A somewhat cloudy day may therefore prove advantageous.

In focusing the reflected light on the cornea, and then on the pupil and lens, any opacities in these will be shown as a grayish nebular reflection or a denser white according to their degree of opacity. The opacities in the cornea or aqueous, in front of the axis of vision in the lens move in the same direction and to the same degree as the eye rolls, while opacities on the posterior capsule or in the vitreous, move in a direction opposite to the motions of the eye, and to a degree corresponding to their distance back of the lens. Thus if the eye looks downward such opacities move upward ; if it looks upward they move downward ; if it looks inward they move outward ; and if it looks outward they move inward.

To secure an image of the fundus of the eye, including the entrance of the optic nerve (optic papilla), the tapetum, the pigmentary surface and retina and vessels, accommodation must be made for the normal refraction of the eye of the patient, and even for that of the observer.

In the **emmetropic** (normal) eye, the rays leave the surface of the cornea parallel to each other and it may be possible for the observer to secure a good image on his retina, without the aid of lenses. In the **myopic** (short sighted) eye they assume a convergent course on leaving the cornea, and to secure a satisfactory image a biconcave or plano-concave lense must be interposed between the cornea of the patient and the eye of the observer.

In the **hypermetropic** (long sighted) eye, the rays diverge in leaving the cornea of the patient, and a convex lens must be interposed between this and the eye of the observer, in order that the rays may be focused on the eye of the observer.

To adapt the vision to the different eyes the modern ophthalmoscope is furnished with a series of lenses concave and convex, any one of which can be moved behind the hole in the mirror to suit the demands of the particular case.

To make a satisfactory examination the pupil should be dilated as for oblique focal illumination. A 1 : 200 solution of apomorphia may be instilled into the eye (a drop or two) and in 20 to 25 minutes a satisfactory dilatation will have been secured. The effect of the homatropin will usually have disappeared in twenty-four hours.

Determination of Static Refraction.

This can be best done in the lower animals by determining the strength of the lens required to render clear the image of its fundus. By knowing the refracting power of the lens, we may ascertain what deviation from the normal refraction there is in the eye under observation.

In making this test the mirror of the ophthalmoscope must be brought closely to the eye of the patient—1 to 2 inches.

If in such a case and without the use of any lens a distinct image of the fundus is obtained, and if this is rendered less distinct by interposing the lowest convex lens in front of the eye of the observer, the eye is **emmetropic**.

If the ophthalmoscopic mirror without a lens gives an indistinct vision of the fundus, and if the image is rendered clear by interposing one of the convex lenses, the eye is **hypermetropic**. The strength of the convex lens, + 1, + 2 or + 3, dioptrics will give the measure of the hypermetropia.

If, on the contrary, the ophthalmoscopic mirror gives an indistinct image of the fundus, which is rendered even more indistinct by the interposition of a convex lens, but is cleared up and rendered definite by a concave lens, the patient is **myopic**. The strength of the concave lens used will give the degree of *myopia*, — 1 dioptric, — 2 dioptrics, etc.

The tendency in the horse is constantly to slight long-sightedness, but the deviation is rarely found to be serious either in this direction or in that of astigmatism.

Mydriatics.

Dilation of the pupil by mydriatics (mydriasis dilation of the pupil) is a most important means of diagnosis, and therefore a knowledge of the action of the different mydriatics is essential. The mydriatics in common use not only dilate the pupil, but also paralyze the ciliary body and the power of accommodation in ratio with the strength of the solution employed. This determines an adaptation of the eye to the farthest point of vision and holds it there until the action of the mydriatic passes off and normal power of accommodation is restored. In short it renders the subject long sighted, during its action.

Atropine the alkaloid of atropa belladonna is the most generally available and persistent of the mydriatics, and is in most common use. It is usually employed as sulphate of atropine, though some prefer the nitrate, the salicylate or the borate to obviate the danger of atropinism. This form of poisoning may show in the occurrence of conjunctivitis and in case of one attack the susceptibility to atropine is greatly to be dreaded, so that it should never again be used on the same subject. The real cause of atropinism is uncertain, it has been variously ascribed to too great acidity or alkalinity, or to micro-organisms growing in the solution. Hence the importance of using the antiseptic salts of atropine, and of testing the solution to see that it is exactly neutral before it is applied.

The strength of the solution of atropine is an important consideration. Donders found that 1 : 120 of water produced a full effect, while Jaarsma obtained the full effect in one hour from a drop of a solution of one to twelve hundred of water. The action on carnivora (dogs and cats) is equivalent to that on man, while on the herbivora (rabbit, horse, ox, sheep) it is somewhat less, and on birds very slight indeed. On diseased eyes a large amount may be required, and with senechia (adhesion of the iris to the capsule of the lens) dilatation may be impossible. The full effect may last 24 hours, and accommodation may remain very imperfect for 11 days.

The direct action of atropine on the eye is shown in dilatation of the pupil of the frog after the eye has been detached from all connection with heart or brain, by excision. It acts also in the normal system through reflex nervous action, since, after division of the sympathetic trunk going to the eye, that eye does not dilate so much under atropia as the opposite eye.

Atropine is usually employed by lodging a drop in the pouch of the conjunctiva (inside the lower lid), and from this it makes its way into the aqueous humor, for if that liquid is transferred to the conjunctiva of another animal it causes dilatation. Puncture of the cornea with evacuation of the aqueous humor lessens the action of the atropine. Atropine dilatation is increased by following it with cocaine which causes contraction of the iridian vessels, the antithesis of the dilatation of the vessels which occurs when the cornea is perforated and the pressure of the aqueous humor is removed.

Atropine is one of the most potent poisons and must be used with caution especially in the carnivora and omnivora. The danger lies not alone in the absorption from the conjunctiva, but also from the escape of the liquid through the lachrymo-nasal duct, to the nose and later to the actively absorbing mucosæ of the lungs and stomach.

The symptoms of general poisoning are : rapid pulse, vertigo, weakness of posterior limbs, general prostration and thirst or dryness of the throat.

Homatropine is an oily liquid produced by the action of muriatic acid on the cyanate of atropine. With hydrobromic acid it forms a readily crystallizable salt, the solution of which

acts on the eye like atropine, but more promptly and transiently. One drop of a solution of one to one hundred and twenty, usually gives in twenty minutes, full pupillary dilation and complete paralysis of accommodation which lasts only for twenty-four hours. Add to this that there is little danger of constitutional disturbance and poisoning, and homatropine must be accepted as a more desirable agent than atropine. It is especially to be preferred in cases of senility with shallow anterior chambers, and in glaucoma, in which atropine tends to aggravate the lesion.

Daturine, the alkaloid of datura stramonium is a potent mydriatic, causing pupillary dilatation in a solution of one to one hundred and sixty thousand of water. It appears to be identical with atropine.

Duboisine the alkaloid of duboisia myoporoides is also a potent mydriatic. Jaarsma found that a solution of the sulphate, of one to three thousand, paralyzed accommodation for twenty-four hours. It acts more promptly than atropine but is more poisonous.

Hyoscyamine, the alkaloid of hyoscyamus niger, is also strongly mydriatic. One drop of an one to three hundred solution of the sulphate paralyzed accommodation for from seventy-five to one hundred hours. Risley found it to act more promptly than atropine, and to be less dangerous than duboisine.

WOUNDS OF THE EYELIDS.

Traumas : bites, lacerations, blows, penetrating wounds, gunshot, scratches, kicks. Upper lid or commissure. Reparatory power of eyelid. Danger of distortion. Treatment : sutures, plaster, shellac, collodion, gelatine, Frick's gelatine, birdlime, sterilisation: Quilled and twisted suture. Position in stall. Metallic guard for eye.

Causes. Traumatic injuries of the eyelids are especially common in the horse mainly because of his exposure in connection with the services required of him. In a team he is liable to be bitten by one of his fellows, or the lid may be caught on nails, in turning, or on hooks upon harness, chains or wagons. It is sometimes injured by a blow from a club or whiplash, or

by knocking the head against solid objects that he failed to see on account of the blinds. Again the injury will come from running against prongs of bushes or trees, or of stump fences. Occasionally a blow with the horn of an ox or cow is the cause, but this is much more frequent with the bovine races. Then again gunshot wounds are found in all animals. In sheep the eyelids sometimes suffer from bites of dogs, while in dogs and cats, the teeth and claws are the main causes of injury. These smaller animals also suffer from brutal blows and kicks.

Nature. Wounds of the eyelids almost invariably affect the upper lid, because of its extra size and prominence. Sometimes one commissure or the lower lid is the injured part.

Clean incised wounds are rare, while lacerations with or without contusions are the common experience. The laceration often extends through the free margin of the lid, and then to one side, mostly the outer, in a direction more or less parallel to the tarsus. The result is that the detached flap drops downward exposing a greater or less portion of the bulb covered with blood. The conjunctiva, the cornea, the sclerotic or iris may be implicated in the lesion in different cases, so that such wounds are of the most varied degree of gravity. If, however, the lesion is confined to the lid, and in the absence of absolute detachment of the flap, or severe contusion, a good repair may be confidently hoped for. The vascularity and reparatory powers of the eyelid are unusually great, and the looseness of the skin, connective tissue, mucosa, and even the muscles is such that they do not draw injuriously upon the edges of the wound to disturb the process of cicatrisation. If the two opposing ends of the divided tarsal cartilage are kept in accurate opposition, the elasticity of this structure serves to preserve the even contour of the palpebral margin, and the adhesion or granulation process between the edges of the wound, soon becomes firm enough to prevent further displacement. Even when one half of the eyelid is torn loose, remaining attached only by a narrow portion, reunion without any unsightly distortion is not to be despaired of. In case of a mere vertical laceration on the other hand, the case is very simple and hopeful. Even when a portion of a lid has been completely torn off and lost, the loose textures of the remaining part, often appear to stretch in the process of healing so that a fairly serviceable,

though by no means an æsthetic covering for the eye may remain. This may serve for a common work horse, but the unsightliness would necessarily debar him from use in a carriage or as a saddle horse. The imperfect protection too, exposes the eye to rainstorms, hail and snow, as well as to dust, and greatly predisposes to conjuctivitis.

Treatment. One can trust implicitly to the extraordinary reparatory power of the eyelids, yet so unsightly is any distortion of these parts, that the greatest pains must be taken to obviate loss of substance, or unevenness or puckering in healing. The points to be mainly sought for are the perfect coaptation of the divided edges, and the restraining of the patient from interrupting the healing process and breaking loose the forming adhesions, by rubbing the eye.

Inconsiderable wounds of the skin may be simply stitched together with sterilized catgut. Then the intervals between the stitches may be approximated, dried, and covered with strips of sticking plaster, or with shellac, collodion or gelatine. Frick's gelatine is made by dissolving fine gelatine in a 1 per cent. solution of corrosive sublimate and adding about 10 per cent. of glycerine, perfecting the admixture by the aid of heat. When wanted for use it may be melted by heat and applied on the skin with a camel's hair brush. Bird lime may be used as a substitute. Sterilization must be sought by the use of sublimate lotion 1 : 2000, or boric acid 2 : 100. Formerly the edges were kept in close opposition by the use of quilled sutures, the stitches passed around the quills being inserted at the usual distances while the quills, applied against the edges of the wound kept them smooth and even and obviated puckering. Or, perhaps better, the twisted suture may be employed, the edges being brought together by pins placed close together and a silk thread carried around each in figure of 8, and spirally from pin to pin along the entire length. If one pin comes out it ought to be promptly replaced and the whole left in place until a firm adhesion is established. The points of the pins are cut off short so that there may be no risk of their pricking.

With any method the horse or ox may be turned in his stall so that his tail may be toward the manger and his face outward, and he may be tied by two halters to the two posts, right and left. His

food may be furnished in a sack hung from the ceiling and cut down one side. In this way the animal may be absolutely prevented from rubbing the itching sore against any solid body, and thereby interrupting the healing process. Another method is to apply a hood of stiff material with a metallic guard for the face, having bars extending from above downward and arched outward so that they shall effectually protect the eye in any attempt at rubbing.

DEFICIENCY OF THE EYELIDS. COLOBOMA PALPEBRARUM.

The term coloboma representing merely a hiatus or deficiency is applied to different parts of the eye according as there may be a lack of substance of the part in question :— **Coloboma palpebrarum** (deficiency of the palpebræ or lids), **C. iridis** (perforation of the iris), and **C. choroideæ** (partial absence of the choroid).

Coloboma palpebrarum is usually congenital and takes the form of a vertical notch on the upper lid, separating its two lateral parts into independent flaps. According to the breadth and depth of the notch are the extent of the exposure of the bulbar conjunctiva and the liability to irritation and infection by foreign bodies. The same condition of things will occur traumatically and require identical measures of repair. These consist in paring the edges of the notch and bringing them accurately together with cat gut, silk or quilled suture, the approximation being rendered more perfect by the application of collodion, shellac or gelatine mixture (see wounds of eyelids). The vascularity and extensibility of the tissues of the lids greatly favor a kindly healing. Rubbing of the eye must be guarded against as advised under wounds of the eyelids.

ORGANIC UNION OF THE EYELIDS. ANKYLOBLEPHARON. NARROWED FISSURE BETWEEN THE LIDS. BLEPHAROPHYMOSIS.

Complete closure of the palpebral fissure has been seen as a congenital infirmity in sheep, dogs and cats, while the partial closure has been found in all classes of animals as the result of chronic conjunctivitis and contraction of the exudation in undergoing organization. Narrowing of the fissure gives the appearance of a small eye, so that a progressive diminution is usually supposed to come from a reduction in size of the bulb, though no actual atrophy of that organ has taken place. In drooping of the upper lid (ptosis) too, the fissure is reduced and the illusion of an atrophy of the eyeball is induced. The closure of the fissure may come from *blepharospasm*, as the result of irritants in the eye, or even of nervous disorder.

Treatment. In case of complete closure of the palpebral fissure, the skin is picked up with forceps and an incision is made between the two tarsi into the conjunctival sac. Then with probe pointed scissors, or a grooved director and bistuory the incision is carried between the tarsi to the proper position for the internal and external canthi. During healing the lids should be frequently bathed with a boric acid solution, and an ointment of the same with vaseline should be applied to prevent adhesion.

When the trouble consists in a drawing together of the skin at the outer canthus, the result of inflammation, the adhesions are separated by a horizontal incision leading outward from the line of the angle. The edges of the conjunctiva and skin are then sutured together, so as to prevent further adhesion and the part treated as an ordinary wound. This is known as *canthoplasty*.

Ptosis coming from tumors on the lid, or excess of fat in its substance, or from oculo-motor disease must be treated according to indications. The same remark applies to *spasm* of the orbicular muscle (*blepharospasm*), whether clonic or tonic. In domestic animals the removal of the cause (foreign body, eyelash), will usually succeed.

WIDENED PALPEBRAL FISSURE.

After wounds of the outer canthus the union of the edges may remain imperfect so that the fissure is enlarged and the eye unduly exposed. The case is still worse if the wound has deviated from the horizontal and has involved the orbicular muscle, the divided ends of which continue to draw the edges apart, and cause a constant overflow of tears (epiphora). Enlargement of the bulb or its protrusion by reason of a swelling beneath it may give rise to the same appearance (exophthalmos).

Treatment. Pare the edges of the upper and lower lids at the outer canthus and bring them together by sutures.

LAGOPHTHALMUS. INABILITY TO CLOSE EYELIDS.

This is called *hare-eye* (lagos, hare) from the fact that the hare habitually keeps the eyelids open. It is mostly due to spasm of the levatores palpebræ, or to undue size of the orbicular opening. It may, however, accompany ectropion, exophthalmos, and enlargement or swelling of the eyeball from any cause. Bayer has seen cases in diseases of the trifacial nerve, in neoplasms in the orbit and in buphthalmus.

Cases of the kind are especially liable to irritation, inflammation and ulceration due to foreign bodies falling on the exposed bulb.

The *treatment* is largely that of the attendant condition ectropion, tumor, etc., which may be consulted.

ADHESION OF THE EYELID TO THE BULB. SYMBLEPHARON.

Causes: Conjunctivitis, burns, operation and other wounds. In front of or behind the reflection of the mucosa from eye to lid. Prevention. Treatment: section of adhesion, and vaseline, etc, Two edges of healthy mucosa sutured together over the sore. When fornix is implicated mucosa is transplanted.

This is liable to occur to a greater or less extent, in all animals, in connection with violent conjunctivitis, burns and operation and other wounds. It has been divided into *anterior* and *posterior symblepharon*, the former being an union in front of the normal reflection of the conjunctiva from the lid upon the bulb (fornix), and the latter involving the fornix in the substance of the adhesion. The *anterior* form by anchoring the lid to the eyeball is much more likely to induce blindness, but it has the compensation that the union may be broken up and the parts healed without subsequent reunion. In the posterior form the eye can be better exposed and vision retained, its repair is much more difficult demanding transplantation of skin or mucous membrane on to the sore, and even then the granulation tissue being continuous from bulb to eyelid may so contract in healing as to leave matters no better than before.

These adhesions not only restrict the movements of the lids, preventing their opening and the exposure of the bulb, but they also anchor the bulb itself, and hamper its movements, necessitated for vision. In all cases therefore of wounds, burns, abrasions and ulcers, of the palpebral and bulbar mucosæ it is highly important to take precautions against the formation of such connections. Any forming adhesions must be broken up day by day and the surfaces must be kept apart in the intervals by borated or iodoformed vaseline.

In a small anterior symblepharon the connections may be cut through and subsequent adhesion prevented by the frequent introduction of iodoformed vaseline, and if need be, by the daily separation of the surfaces by a probe. When this fails a plastic operation may be resorted to, the mucosa on the inner side of the

lid being incised in a vertical direction a short distance on each side of the sore and the inner edges accurately stitched together. The raw surface left on the bulb thus comes in contact only with the healthy mucous strips on the eyelid, which have been drawn together over the seat of the former sore, and the two new raw surfaces formed on the lid are well to each side of the sore on the bulb, and are in contact with its healthy mucosa only. Thus no two raw surfaces can come in contact, and adhesion is obviated.

When the fornix is implicated mucous membrane from the mouth, vulva, the bronchia of the rabbit, or the skin of the frog must be transplanted after the requisite incision of the cicatrix has been made.

INFLAMMATION OF THE EYELIDS. BLEPHARITIS.

Phlegmon. Causes: traumas, skin disease. Symptoms: swelling, redness, distortion, infiltration, semi-closed lids, scabs, sloughs, abscess. Tenderness. Itching. In eczema papules, vesicles, weeping eye. Treatment: antiseptic astringent lotions, almond oil, vaseline, zinc oxide, salicylic acid, boric acid, starch, xeroform, pyoktannin; for eczema, mercury oxides, silver nitrate.

Conjunctivitis will be treated later, and under the present head there will be considered only the phlegmon of the outer structures.

Causes. This lesion may come from two distinct causes, traumatism and skin disease. The traumatisms in *horses* and *cattle* are bruises sustained in rolling, especially during colics, in striking the head against posts, poles, shafts and other solid bodies, in enduring blows with horns or clubs, or frictions by the halter or in putting on a collar. *Dogs* suffer especially from blows with clubs and kicks from men or animals. *All* may suffer from wounds of the lids, and from extensions of eczema and other skin diseases.

The *symptoms* consist in swelling, redness, distortion, and often extensive infiltration of the lid, sometimes eversion with exposure of the reddened conjunctiva, usually abrasion, contusion, puncture or laceration, semi-closed eye, the upper eyelid being comparatively immovable (ptosis), and the formation of scabs, sloughs, or abscess. There may be extreme tenderness, or, more com-

mouly, intense itching. Where eczema exists there may be found minute shot like papules at times surmounted by small vesicles and the skin disease is continuous backward upon the face. When abscess forms, the rounded swelling and manifest fluctuation will betray its presence. Usually the eye waters and the side of the cheek is wet and the hairs matted by a whitish coagulated lymph and mucus.

Treatment. In the early stages without scabs, sloughs, or abscess, antiseptic astringent lotions are in place. Weak solutions of zinc sulphate, boric acid and morphine may be kept applied on a light bandage. Or silver nitrate 1 gram to 1 oz. water may be applied daily with a fine brush.

When scabs and crusts have formed they may be softened by the application of almond oil, and then removed. The surface may then be dusted with a bland antiseptic powder such as: zinc oxide 10 parts, salicylic acid 1 part; or boric acid and starch equal parts; or iodoform; or xeroform. Or unctuous applications may be used; zinc oxide 10, salicylic acid 1, vaseline 10; or iodoform 1, vaseline 5. Or a watery application may be used, such as the silver lotion or that of pyoktannin 1 : 1000.

For eczema yellow oxide of mercury 1, to vaseline 10, has an excellent reputation. It may be alternated with pyoktannin.

When abscess has formed it should be excised in a line parallel to the free border and the resulting cavity injected with the silver or the pyoktannin solution.

In all cases the patient must be fastened as for wounds of the lids so that he cannot rub the eye.

For eczema and other skin diseases the special treatment appropriate to the disease should not be omitted.

ŒDEMA OF EYELIDS.

In anthrax, malignant œdema, disease of heart, kidney or liver, distomatosis, trichiniasis, wasp stings, urticaria, petechial fever. Treatment: correct general disorder, remove local irritant, antiseptic astringents.

An œdematous condition of the eyelids with or without inflammatory conditions may be due to local disease or it may be the result of more general disorder. In anthrax districts any of

the herbivora, but especially cattle and sheep, are liable to a diffuse anthrax of the eyelid with a special petechial or brownish condition of the palpebral conjunctiva. Malignant œdema and other local bacteridian affections affect the loose textures of the eyelid in a similar manner, but with extrication of gas and crackling under pressure. Such cases are complicated by local inflammation. When in the absence of inflammation the lids pit on pressure, one should seek for some disease of the heart, kidney or liver, also for indications of similar dropsical effusions in other parts of the body. Distomatosis and, to a less extent, pulmonary and duodenal strongylosis are especially common factors in sheep. In distomatosis (liver rot) a simultaneous dropsy is often present in the intermaxillary space, the chest or the abdomen. The puffiness of the eye is especially marked in the palpebral conjunctiva, and is exposed by everting the eyelid over the tip of the finger. In trichinosis in man and less frequently in swine, dropsy of the eyelid is often present at the end of the first week. Other swellings of the lids partaking more of the nature of inflammation, result from the stings of wasps, hornets and other insects, from urticaria (in horses especially) and from petechial fever in solipeds.

In *treating* such cases the general disorder, if present, must be first attended to, then the removal of any local irritant, and finally the antagonizing of any local inflammation or infection. Astringent and antiseptic lotions are especially called for.

EMPHYSEMA OF THE EYELIDS.

This has been already referred to as occurring in malignant œdema, black quarter and other gas producing infections. It may also come from lacerations made in puncturing the lachrymal sac, and from fracture of the margin of the orbit—the air entering the connective tissue in this case from the cavities of the nasal sinuses. The lid feels puffy and crackles when pressed and apart from a general infection it requires only soothing and antiseptic dressings.

DISEASE OF THE MEIBOMIAN GLANDS. BLEPHARADENITIS. SEBORRHŒA.

This is a blepharitis of the edges of the lids which are swollen, red, and incrusted along their margins with scabs and sebaceous concretions. When this scurf is removed the skin is found to be red, tender and glistening. The glands are the seat of congestion, and produce a modified secretion in excess, which dries into crusts instead of preserving its normal oleaginous consistency. As these glands open into the follicles of the eyelashes, their walls are implicated and shedding of the lashes is a common result. It may be assumed that this affection is often associated with the proliferation of microbes in the glands and gland ducts, while in other forms the presence of acari is the controlling factor. Wilson found the demodex folliculorum in the Meibomian glands of the horse, and Oschatz in those of the sheep.

Treatment. Smear the margins of the lids with vaseline and when the crusts have been thoroughly softened wash them off with Castile soap and warm water. Then dress the margin with the ointment of the yellow oxide of mercury 1, in vaseline 10. If demodex is suspected they may be squeezed out and the lids washed frequently with spirits of wine as a solvent.

HORDEOLUM. STYE. ACNE.

Like acne of the skin in general, this consists in inflammation and suppuration of a hair follicle and sebaceous gland. The whole lid or a large part of it may be swollen, but by stroking it with the finger, a hard, rounded, very tender spot will be detected and as the disease advances this develops a minute collection of pus. A specially wide orifice favors the entrance of the pus microbes, and the onset of the disease. It has been noted in dogs (Fröhner).

For abortive *treatment* Fick recommends dry heat from a pocket handkerchief or a heated teaspoon. If pus is present it must be evacuated, and recurrence guarded against by cleanliness and antiseptics. Use pyoktannin solution (1 : 1000), or mercuric chloride (1 : 5000) or yellow oxide of mercury ointment.

CHALAZION.

This is a pea like tumor growing from the tarsal cartilage, its flattended side toward the mucosa, which is red and angry, and its round surface toward the skin. When manipulated between the fingers it moves with the tarsus. It is usually of slow growth and may continue for years apparently unchanged. Some have thought it tuberculous, but its true nature is uncertain. Warner records the disease in the horse.

Treatment consists in incision and removal of the tumor, curretting of the cavity, and after antiseptic douching, suturing the lips.

TUBERCULOSIS OF THE EYELID.

Described by Jewsejenke in the lower lid of birds, this is manifested by small, hard round knots, covered by bluish red, or yellowish red skin, and when incised showing a characteristic miliary tubercle, with bacilli and sometimes a caseated centre. It is treated by incision, curretting and caustics.

TURNED-IN EYELASH. TRICHIASIS.

Sometimes an eyelash grows inward so as to impinge upon the front of the eyeball, or even to extend between this and the eyelid. The condition exists in *entropion* but *trichiasis* is rather the deviation of one or two cilia by reason of their false direction, individually. It may occur as the result of a pre-existing inflam-

mation affecting the edge of the lid and the follicle, and the offending hair is not only badly directed but small and shrunken as well. On this account it is not always easy to recognize it, and accordingly in cases of conjunctivitis without apparent cause it is well to examine carefully with the aid of oblique focal illumination.

Treatment consists in pulling out the offending hair with ciliary forceps, avoiding bending it lest it break off short and become at once more irritating and more difficult of extraction. In case the hair grows anew in the same direction extract it anew and destroy its root with the electric cautery.

ENTROPION. TURNING IN OF THE EYELID.

In foals, puppies, hounds, with narrow fissure, and conjunctivitis, or tarsitis. Permanent bandaging, orbicularis spasm. Symptoms: disappearance of tarsus and lashes by involution. Treatment: in spasm fix by plaster; suture skin: excise elliptical section of skin and suture edges together. Release cicatrices.

Inversion of the eyelid or a portion of it, with consequent trichiasis, conjunctivitis and lachrymation has been met with congenitally in foals (Aubry, Bourdeau, Hamon) and puppies (Cadiot, Almy). Hounds have especially suffered. In the older animals it is largely determined by abnormally narrow fissure, and by old standing disease of the conjunctiva or tarsus, with cicatricial contraction or adhesion. Persistent bandaging turns in the cilia and contributes to entropion. Finally a persistent spasm of the orbicularis muscle may bring it about.

Symptoms Trichiasis is usually, though not always, present. In any case the tarsus is turned inward so as to press upon the front of the bulb, or even to disappear completely. Thickening and distortion of the lid is a not infrequent condition.

Treatment. In case of simple spasm clip or shave the hairs from the lid corresponding to the lesion, and close to the tarsus attach a strip of plaster. When firmly adherent draw it sufficiently to efface the entropion and attach it to the skin of the face.

This failing, Gaillard's sutures may succeed. With a pair of forceps with looped, transversely elongated blades, pinch up skin and muscle sufficient to correct the entropion, and passing a needle twice through this fold with an interval of 3 m m., tie the suture over a small roll of cotton. The stitches may be removed in two days and the cicatrices may permanently obviate the deformity.

The older plastic operation is more trustworthy: The skin of the affected lid is pinched up to such an extent in length and breadth, as to correct the entropion and is then excised with sharp scissors or bistuory so as to leave a long elliptical sore. The edges of this are then carefully sutured together and the resulting union corrects deformity. In case the entropion is caused by an old standing cicatrix, it may be necessary, first, to make a careful incision along the edge of the lid so as to separate the tarsus and conjunctiva from the cilia and Meibomian ducts, and then to proceed with the plastic operation on the skin.

TURNING OUT OF THE EYELID. ECTROPION.

In large dogs, in old age, debility, conjunctival swelling, cicatrized skin of lids, distortions of lids. Symptoms: exposure of pelpebral mucosa, weeping eyes, conjunctival hypertrophy (chemosis). Treatment: scarify or excise a fold of mucosa, astringent antiseptics, Snellen's suture, Diefenbach's operation, Wharton Jones operation.

This is much more common than entropion, but much less injurious as the tarsi and lashes do not irritate the conjunctiva. It is especially common in large dogs (hounds, mastiff) and usually affects the inner part of the lower lid. Old age and debility contribute materially to the condition, the lack of tone or paresis being an important factor. It may, however, occur in any animal, from conjunctivitis and swelling of the mucosa, from cicatrices or old standing disease of the skin of the eyelids, or from imperfectly healed wounds leaving distortions of the lower lid. It is most frequent in the lower lid, and the slightest pendulous condition, which detaches the tarsus from the bulb, and exposes a narrow zone of the conjunctiva is considered to be an ectropion.

Symptoms. Beside the exposure of the zone of mucosa, there is the overflow of tears, and in old standing and bad cases a hypertrophy of the exposed conjunctiva, which projects as a fleshy-looking mass, and weighs down the lid, with a continual tendency to aggravation.

Treatment. Where the main factor seems to be the infiltration of the mucosa this may be reduced by scarification, or by the complete excision of a fold of the membrane. Use an antiseptic wash (boric acid) and the retraction of healing tends to brace up the lid against the bulb.

Snellen's suture is sometimes employed successfully. A silk thread is armed at each end with a needle, and the needles are passed into the conjunctiva just inside the tarsus and brought out through the skin near the margin of the orbit, where they are tied round a small roll of cotton. Several of these may be inserted side by side so as to extend the whole length of the ectropion and they should be drawn tight enough to correct the deformity. If left some days they will usually determine cicatrices which will overcome the deformity.

The most common operation (Dieffenbach's) is the excision of a triangular portion of skin from just outside the lower lid and having its base or upper side running horizontally outward from the outer canthus. Then pare the margin of the lower lid for a distance equal to the base of the triangle. Then bring together and suture the skin forming the right and left sides of the triangle, and the raw edge of the lid to the skin that formed the base of the triangle. In this way the triangular sore formed by the operation is completely covered and the margin of the lower lid is shortened so as to brace it up against the bulb.

In case of cicatricial ectropion the Wharton-Jones operation is to be adopted. A V-shaped incision is made in the skin of the lower lid commencing just beneath the tarsus and carried down so that the two lines of incision meet well down beneath the cicatrix. The triangular flap of skin thus made, is detached by a bistnory from the cicatricial tissue beneath, and allowed to shrink upward toward the tarsus. Finally the two edges are sewed together from the angle upward, as far as may be necessary to allow the proper application of the tarsus against the bulb, and the remainder of these edges are sutured to those of the triangular flap.

TUMORS OF THE EYELIDS.

Warts. The most common tumors of the eyelids in horses, cattle, and dogs are warts. These are most simply disposed of by seizing them with rat-tooth forceps and clipping them off with sharp scissors curved on the flat. Any bleeding may be checked by a pencil of silver nitrate.

Sarcoma, melanoma, and **epithelioma** are common in solipeds, especially in the gray and white. They usually form a cauliflower-like mass red and angry and bleed easily. They may occupy any part of the lid, the skin, the dark tarsal margin, the connective tissue or the mucosa, and not unfrequently they involve the eyeball, and the surrounding tissues, even the bones of the orbit.

Treatment. These may be excised like warts taking care to remove every vestige of disease. In these cases I have usually found it necessary to remove the entire bulb.

FRACTURE OF THE ORBIT.

Nature and Causes. The usual seat of fracture is the orbital process of the frontal bone, yet any portion of the orbital margin may suffer, and even the inner wall or floor of the orbit may be broken by a penetrating instrument. Horses and polled cattle and sheep are especially exposed to the injury, while in horned stock the region is in a measure protected. Carnivora, which have no bony orbital process, are less liable but may still sustain fractures of the remaining parts. Horses and polled ruminants suffer mainly from beating the head on the ground or other solid body in the paroxysms of colic and enteritis, or in nervous affections; horned stock suffer from concussions in fighting and direct blows by the horns. All animals suffer from blows with clubs, kicks and other mechanical injuries.

Symptoms. With (and less frequently without) a skin wound, there may be indication of depression, or mobility of the de-

tached segment, or its sharp edge may be felt, through the skin, or by the sterilized finger introduced into the orbit. In case of a penetrating or stab wound, which cannot be followed by the finger, it may be followed by an aseptic probe and any fracture recognized. The conjunctival sac must be first thoroughly washed out with an antiseptic lotion, as the introduction of any septic germs into the osseous wound, is likely to cause a dangerous infection or abscess.

Treatment. Simple, slight fractures with blunt instruments are treated by rest and cooling, disinfectant lotions. If foreign bodies or detached particles of bone are found in the wound they should be extracted. Shot that are difficult to find, may be left, as they are often aseptic and tend to become encapsuled. Should they cause abscess they will usually be found in the pus sac and may then be removed. Displaced bones may often be replaced by the finger in the orbit. Sometimes they can be best reached by trephining the frontal or maxillary sinus and introducing a lever through the cavity (Hendrickx). If the sinus has been involved it must be opened in any case. Cadiot advises bandages impregnated with black pitch to fix the bones in certain cases. Antiseptic washes (sublimate 1 : 5000) and antiseptic cotton packing are demanded for all wounds.

BRUISES AND WOUNDS OF THE ORBIT.

These may come from the same *causes* as fractures and though less violent may occasion inflammation which involves the eye or even the brain with fatal results. Thus in horses it has been a cause of infective inflammation, with a fatal extension (Robellet); in cattle a similar inflammation has extended to the cerebral meninges and caused death (Leblanc), and in dogs an advance to the eyeball threatens its destruction (Möller). Short of this necrosis is not uncommon (Rey).

Treatment. This does not differ materially from that demanded by penetrating wounds with fracture. A perfect cleansing and antisepsis of the wound is the first demand. A solution of boric acid (4 per cent.) or of mercuric chloride (1 : 5000)

liberally applied, and maintained thereafter on soft pledgets of surgical cotton, will often have the best results. All foreign bodies must be carefully removed, lacerated flaps and shreds may require suturing, dead portions excision, and finally abscesses or excessive exudate may require the lance, but cooling, antiseptic lotions and an elevated position of the head, are among the most prominent resorts.

RETRO-BULBAR ABSCESS.

Schindelka has observed this in the horse, in connection with petechial fever. If connected with meningeal abscess it will be necessarily fatal. In favorable cases evacuate the pus as soon as detected and dress with pledgets of cotton saturated with a mercuric chloride solution (1 : 2000) or other antiseptic.

PERIOSTITIS OF THE ORBIT.

This may be shown by the firm swelling of the bone and, in case a wound has been formed, by the contact of the probe with the denuded, hard, rough bone. When thus exposed or necrosed on the surface, or when an exostosis has formed, the bone may be laid open and scraped down to the healthy tissue, and then dressed with antiseptic pledgets.

TUMORS OF THE ORBIT.

These may be of different kinds, as **sarcoma, encephaloid, osteoma** and **actinomycosis.** They demand thorough surgical treatment, except perhaps in the case of the latter, which may recover under iodide of potassium. Emmerich records an extensive sarcoma of the orbit in a cow, weighing six pounds and extending into the nasal sinuses, and chambers, and implicating the cerebral meninges. Möller records cases of sarcoma and car-

cinoma of the orbit in horses and dogs, and Leblanc in cattle. Melano-sarcoma is not uncommon in the orbits of gray horses which are changing to white.

Exotoses are common around the orbits of cattle.

If such growths do not show on the surface they cause a more or less unsightly protrusion of the eyeball, owing to the presence of the neoplasm in the depth of the orbit, and the removal of the bulb becomes a necessity.

DISEASE OF THE LACHRYMAL GLAND AND DUCTS. DACRYO-ADENITIS.

Even in man these parts are remarkably free from disease, while in the lower animals, we have literally no record of such conditions. Inflammation of the gland (dacryo-adenitis) would be manifested by a sensitive swelling under the outer part of the orbital process, and upper eyelid and by lachrymation, and obstruction of the gland duct and by a tense transparent rounded swelling inside the lid. A fistula is possible from a penetrating wound of the lid in the same situation. In both of the latter conditions an opening made through the palpebral conjunctiva will allow the discharge of the tears in the proper place, and healing of any external wound may be hastened by suture or plaster.

OBSTRUCTION OF THE LACHRYMAL PUNCTA. ATRESIA. INFLAMMATION.

Congenital atresia of these puncta has been recorded in foals, by Hollmann and obstruction as the result of inflammation, by Lafosse, Verjaus and Tyvaert, and of the entrance of the seeds of bromus by Stockfleth.

Apart from congenital atresia and impaction of foreign bodies the *symptoms* are those of conjunctivitis, with escape of tears over the face (epiphora). Injection of aseptic water into the

lower puncta and its escape by the upper, and by the nasal orifice, will determine the patency or otherwise of the various channels.

Treatment consists in astringent collyria to check the inflammation, in the removal of any foreign body, in the dilation or slitting of the lachrymal canaliculi, and in case of complete atresia, in incising the lachrymal sac. Slitting of the canaliculi is accomplished by a small probe pointed bistuory (canaliculus knife). The lid is drawn away from the carnucle, and the probe point inserted at first downward, then inward and backward, and when it is well inside the sac the handle is brought to the vertical and the walls of the duct slit open.

In case of atresia Leblanc recommends to seize the inner canthus with rat tooth forceps so as to include the structures about the sac and to plunge the bistuory directly into the sac. Then by the aid of a whalebone staff he passes three silk threads through the duct and fixes them in place by attaching them to a copper ring at each end. This is retained in place and moved daily until the passage has been definitely healed and its permanency assured.

WOUND AND FISTULA OF THE LACHRYMAL SAC.

The lachrymal sac, which receives the tears from the canaliculi, is situated in the infundibulum at the upper end of the lachrymal canal and is in great measure protected against external injuries by the prominent orbital edge of the lachrymal bone. Yet violent blows with or without fracture, sometimes lead to rupture of the mucous walls and the formation of a fistula. Wounds made with penetrating bodies, more or less pointed are also liable to involve the sac. The fistulous orifice may be through the skin at the inner canthus or through the mucosa by the side of the carnucle. The cutaneous opening may be a minute orifice from which tears and muco-purulent matter escapes, to mat together the hairs on the side of the face. Sometimes there is a reddish elevation, the size of a pin head, and in fistula through the mucosa this is the rule, and the orifice is elevated so that the

tears flow out over the face. For the symptoms of the attendant catarrh of the sac see below. In infected cases with obstruction of the lachrymo-nasal duct, it has been known to extend to the bone and even to open into the sinuses, or tooth follicles. (Gerard, Leblanc).

The condition is found in horses, cattle and dogs.

Treatment. In fistula resulting from simple traumatism, nothing more may be requisite than rest and soothing astringent applications. Sutures are sometimes resorted to but are liable to cause itching and do more harm than good. It is above all important to keep the lachrymo-nasal duct patent, and for this purpose a lead or silver stilet, or a thick cat gut suture may be worn in the canal until healing has ensued.

CATARRH OF THE LACHRYMAL SAC. DACRYO-CYSTITIS.

Connected above through the canaliculi with the conjunctiva, and below through the lachrymal duct with the nasal chamber this cavity is liable to be more or less implicated in all cases of nasal catarrh and conjunctivitis, (strangles, canine distemper, influenza). If the lachrymal duct is obstructed so that the tears accumulate in the sac, the tendency to catarrh is further enhanced by the distension and weakening of its walls, and by the propagation of bacteria which have entered with the tears, and find in them a favorable and abundant culture medium; the diameter of the sack in the horse being about $^2/_3$rds. of an inch. The presence of foreign bodies is another cause.

Lesions. Symptoms. Swelling at the inner canthus, which raises the carnucle above the normal level, and the escape of tears over the lower lid are the most prominent symptoms. If the swelling is pressed it subsides, the contents, clear or purulent, escaping through the lachrymal duct, to the nose, or through the puncta and accumulating in the inner canthus or flowing over the cheek. The hair beneath the inner canthus is matted together, or drops off leaving bare patches. Wolff found in one

case, a distension of the sac to over two inches in breadth, and 1 2/3 inches long. To the swelling there is soon added conjunctival inflammation, closure of the puncta by swelling and the escape of all tears over the face. Suppuration supervenes in the sac, and in the larger animals the pent up pus often makes its way outward, causing destructive ulceration of the walls of the canaliculi and puncta, or of the walls of the sac, the skin, or even the subjacent bone. In this way fistula results. Caries of the bone and penetration of the molar alveoli may ensue. (Girard, Leblanc).

Treatment. The first object must be to secure a free drainage into the nose. The evacuation of the sac by compression having been accomplished, an astringent solution may be injected through the nasal opening of the lachrymal duct. If the canal is pervious the sac will be re-filled and will swell out as before. The injection may be 0.5 per cent. sulphate of zinc, 1 per cent. acetate of lead, 0 3 per cent. nitrate of silver, 1 per cent. tannic acid, 2 per cent. boric acid, or 0.02 per cent. corrosive sublimate. Cocaine may be added in the proportion of 5 per cent. The injection may be repeated thrice a day at first, then twice, and finally once as the catarrh subsides.

If the injection fails to reach the sac, thoroughly sterilized, flexible probes may be used, increasing the size as they can be passed without too great pressure.

Or the puncta and canaliculi may be injected as in the human subject, the conjunctiva having been first anæsthetized by cocaine, or general ether or chloroform anæsthesia having been induced. The slitting of the puncta and canaliculi may be resorted to, as spoken of under atresia.

The frequent passage of a sound is usually resorted to, and a stilet may even be worn, but there is always danger of resulting thickening and narrowing of the duct, and, if healing can be secured without this measure, it is to be preferred.

STENOSIS OF THE LACHRYMO-NASAL DUCT.

Obliteration of the lachrymal duct may occur from stricture of the canal, the result of wounds or other irritants: from pressure by the inflamed mucosa in nasal catarrh or strangles: from polypus or osseous tumor in the nose: from actinomycosis or other disease of the bones.

The one manifest *symptom* is the escape of the tears on to the face. To complete the diagnosis, injection of one punctum will cause distension of the lachrymal sac.

Treatment. This may be attempted by bougies. In the *horse* a small sound, metallic or whalebone bougie, thoroughly sterilized and smeared with aseptic vaseline, or oil, is inserted from the nasal opening and carefully passed on into the sac. In the *dog* the nasal opening cannot be reached and the bougie must be passed by the puncta and lachrymal sac. To secure the requisite dilation, it is usually necessary to probe the passage daily, using a larger probe when the first passes easily, until the canal has been sufficiently dilated.

A second resort is to distend the canal by a liquid injection thrown into the nasal opening. This will succeed when the obstruction is only caused by concretions in the canal.

A somewhat similar resort is the insufflation of the duct by means of a finely pointed tube inserted from below into the nasal orifice of the duct.

Still another method is to make a new opening for the escape of the tears into the nose. When the stenosis is at or near the nasal opening of the duct, an artificial opening is easily made and usually satisfactory. Under anæsthesia, a sterilized silver probe is passed through the upper punctum, the sac and canal. When it meets definite obstruction its position is ascertained inside the nose, and an incision is made so as to allow its escape. The constant escape of tears tends to prevent it from closing up again, but it is well to examine into this until it has thoroughly healed. A silk thread worn in the duct and held in place by a copper or aluminum ring on each end may be resorted to.

Attempts have been made to establish a new outlet by boring through the lachrymal bone into the nose, but without a permanent success. It has also been advised to obliterate the lachrymal ducts and sac, on the one hand and to excise the lachyrmal gland on the other, but the proposed cure is worse than the disease.

DISEASE OF THE LACHYRMAL CARUNCLE.

The caruncle is inflamed in conjunctivitis. When this inflammation leads to hypertrophy it is known as *encanthis*. This is a common condition in *dogs* and the caruncle may increase to the size of a pea or acorn, and by compressing the canaliculi it leads to a profuse overflow of tears on the cheek. At first there is the acute congestion of conjunctivitis, but later there may be induration and pallor.

The *treatment* of this condition consists in astringent and sedative collyria in the early inflammatory stages, and later in the ablation of the hypertrophied mass. The caruncle is seized with a pair of rat-tooth forceps and snipped off with curved scissors, the free bleeding being afterward checked by cold water.

In cases that seem, by reason of excessive vascularity ill adapted to this method, the hypertrophied mass may be tied at its base with a stout silk thread so as to cut off the supply of blood, and cause it to slough off. A collyria of boric acid (4 per cent.) or mercuric chloride (0.02 per cent.) may be used to prevent infection.

Tumors of the Caruncle are met with, such as fibroma (Wörz), Sarcoma and Melanosarcoma. For all alike the complete extirpation of the neoplasm is demanded.

WOUNDS AND INFLAMMATION OF THE MEMBRANA NICTITANS.

Like other parts of the ocular apparatus, the third eyelid and gland of Harder are subject to accidental injuries of various kinds. What is worse, ignorant persons seeing the cartilage and membrane projected over the eye in ophthalmias and tetanus, have mistaken it for a morbid product and deliberately cut it off in part. The condition of the organ may be ascertained by parting the lids with the fingers and pressing gently on the front of the eyeball, when the nictitating membrane will be fully exposed.

If detached portions cannot be restored, but threaten to slough, or cause distortions or unsightly and irritating neoplasms they should be seized with forceps and snipped off with scissors. Otherwise the treatment consists in soothing astringent and anodyne Collyria as in conjunctivitis.

TUMORS AND HYPERTROPHY OF THE MEMBRANA NICTITANS.

Neoplasms of this organ may occur in any quadruped or bird and may be recognized by the swelling of more or less of its substance, by the unevenness of its free margin, or by distinct outgrowths from its surface. They are especially common in dogs and pigs and may be fibrous, epithelial or otherwise. The treatment is purely surgical and in case of a malignant neoplasm should demand the removal of the entire organ.

ADENOMA OF THE GLAND OF HARDER.

Cases in dogs have been recorded by Fröhner and Schimmel, and it might be expected in other carnivora, ruminants, pigs, rabbits and birds. The treatment is by excision with forceps and scissors, and subsequent treatment with an antiseptic zinc lotion.

FOREIGN BODIES IN THE CONJUNCTIVAL SAC.

Frequency; seeds, glumes, awns, dust, sand, wood, metal; exudate; in conjunctival pouch, under nictitans, in puncta. Filaria lachrymalis. Symptoms: closure of lids, epiphora, congestion, inflammation, infection. Treatment: local anæsthesia, forceps, lead pencil, pin's head, collyria.

So common are foreign bodies in the conjunctival sac of the domestic herbivora, that in any case of epiphora, hyperæmia or inflammation of the mucosa, the first care should be given to see that the condition is not caused by the presence of such an irritant. In animals fed from high racks, seeds and glumes of the gramineæ, awns of barley, and dust of various kinds often get into the eye and stick fast. Under other conditions, insects, particles of sand, dust, wood, metal, etc., prove equally injurious by their presence. Awns and chaff are particularly liable to adhere to the mucosa and even to become covered by an exudate, which renders them more firmly adherent. Other objects lodge under the eyelids, or membrana nictitans, or in folds of the mucosa. Their entrance into the lachrymal puncta has already been referred to. The larger and more rounded bodies are likely to be washed off by the excessive flow of tears, assisted by the movements of the nictitating cartilage, but flat glumes, or awns stick too closely to the surface, while the smaller objects become entangled beneath the lids, or hair, or in the folds of the mucous membrane. The filaria lachrymalis may be the cause of trouble.

Symptoms. There is closure or semi-closure of the lids, the escape of a profusion of tears over the cheek, and active congestion or hyperæmia. A careful examination with everted lids, or even with raised nictitans will usually reveal the foreign body. If overlooked or neglected the hyperæmia rapidly advances to active inflammation, with or without an infective complication. Foreign bodies blown into the eye, as a rule carry with them more or less bacteria, and, if these have any tendency to pathogenesis, the irritation of the mucosa easily paves the way for their colonization. Thus, any grade or form of conjunctivitis may supervene upon the introduction of a foreign irritant.

Treatment. Nearly all such bodies are most easily and certainly removed by a pair of fine forceps. It may be necessary to first anæsthetize the eye with a 5 per cent. solution of cocaine.

The clean tip of the finger passed under the lid and nictitating membrane is a safe and effective method. Less effective methods are to pick up the offending body on the point of a lead pencil, or a small, blunt metallic spud, or with a pin's head covered with a clean pocket handkerchief. This may be followed by an antiseptic (boric acid) collyria, with or without cocaine or morphia.

WOUNDS OF THE CONJUNCTIVA.

These occur in all domestic animals, but are especially frequent in dogs and cats from scratching with the claws. In clumsy handling of the eyelids, the mucosa is wounded by ragged and uneven nails. Injuries and stings by insects which are attracted by the reflection from the eye constitute a specially grave lesion, often proportionate to the nature of the poison instilled.

Symptoms. There are usually closure of the eyelids, with exudation and thickening of the conjunctiva especially in the vicinity of the wound, a free flow of tears, mingled it may be with blood, and the visible evidence of the lesion on the exposure of the injured part. If the cornea is implicated, even the pupil is contracted, showing photophobia.

Treatment. Slight noninfected wounds will heal readily under simple astringent collyria, following upon the removal of any cause of mechanical irritation. A solution of corrosive sublimate, 1 : 5000, or of boric acid, 4 per cent. may be used. If photophobia exists ½ per cent. of atropia sulphate or 1 per cent. of cocaine hydrochlorate will usually give relief. Extensive wounds may require sutures, and sloughing tissue may be excised with fine curved scissors. Excessive granulations may be removed in the same way. For stings use a potassium permanganate solution (2 grs. to 1 oz). Violent inflammation may be met by a laxative and by leeching the periorbital region.

BURNS OF THE CONJUNCTIVA.

Burns may occur in all domestic animals from acids, alkalies, quicklime, carbolic acid, boiling liquids, etc. The cornea usually suffers, being the part most exposed. The caustics cause swelling, blanching and finally exfoliation of the epithelium, or even of the superficial layers of the cornea. In burns by hot liquids vesication may be present. If the destruction extends deeply into the cornea there may be escape of the aqueous humor and destruction of vision. If less penetrating, there may still develop vascularity, and permanent opacity by reason of the formation of a cicatrix or a change of structure in the layers of the cornea, or, in dogs especially, adhesion of the cornea to the eyelids (symblepharon). In the early stages there is closure of the eyelids, with swelling, profuse lachrymation, and photophobia.

Treatment. The first object is to remove or neutralize the offending body. Thus sulphuric or other mineral acid would demand a free irrigation with a 1 per cent. solution of carbonate of soda or potash. For alkalies, carbonated water, or a 4 per cent. solution of boric acid may be employed. For lime, Gosselin recommends free irrigation with saccharated water. The first step, however, should be to wipe out the particles of lime with a soft rag soaked in oil.

The pain may be met by a solution of cocaine (1 per cent.), or atropia ½ per cent. In addition, we may irrigate with cold water or apply weak antiseptic collyria, and employ derivation by the bowels or the skin.

SIMPLE ACUTE CATARRHAL CONJUNCTIVITIS.

Causes: irritant gases, smoke, dry air, dust, pollen, microbes, insects, seeds, chaff, awns, sand, dust, quicklime, coal dust, hairs, whips, canes, branches, stubble, fences, nails, claws, teeth, iodine, chlorine, bromine, formalin, turpentine, rain, hail, sleet, glare of sun, ice, snow, white sand or clay, filaria, acari, infectious diseases. Symptoms: closed, lids, epiphora, redness diffuse on lids, ramified on sclera, projected nictitans, milky flocculent discharge, later purulent, matted lashes or tarsi. No photophobia.

Fever variable. Lesions: exudate: cell proliferation: papillæ enlarged: desquamation: swollen lymphoid bodies: hyperplasia: abscess: ulcer. Treatment: remove cause, sterilized syringing, antiseptic and astringent collyria, sublimate, zinc sulphate, lead acetate, pyoktannin, silver nitrate, morphia, atropia, cocaine, vaseline, cupping, leeching.

Under this head may be noted the simple forms of conjunctival inflammation, which result from direct injuries, irritant gases from manure, smoke, very dry air, dust, pollen, and in which no specific nor contagious element is a prominent feature. Strictly speaking, all purulent conjunctivitis are probably infecting, as all may be held to be associated with the presence of pus microbes. As early as 1864, de Graefe said every inflammation of the conjunctiva which "secretes, is inoculable and therefore transmissible," and Warlomont concurs, saying that "conjunctival ophthalmias have one character in common: all can produce a secretory product which, when brought in contact with a sound conjunctiva, is capable of provoking the different affections of this mucosa." The exposure of the conjunctiva of man and beast alike, to the germs borne on the dust or carried in liquids or on solids, renders this structure preëminently susceptibile to infection, while the lachrymal and mucous secretions furnish a favorable culture medium. Fortunately in the lower animal the danger of infection is greatly reduced, since there are comparatively few opportunities for the transference of the germ through water, soap, sponge or towel, to which man is exposed, not only in public lavatories but even in private families. Thus in the animal the single case remains isolated and in a sense sporadic, only because there have not been the means of transferring and inoculating the morbid product.

Causes. The contact of mechanical and chemical irritants of all kinds, insects, seeds of graminæ, chaff, particles of fodders, barley and other awns and spikes, grains of sand, dust, quicklime, coal dust, smoke, hairs, blows with whips, canes, branches, stubble, fences, etc. Also erosions caused by nails, claws, teeth, and by falls must be considered.

Irritant gases and vapors whether from accumulation of manure in the stables, or from chemical products of fires and factories are direct causes. The mere exposure for sometime to the warm, dry air from a furnace is injurious to the mucosa,

and the emanations from certain medicines, iodine, chlorine, bromine, formaline, turpentine are strongly irritant.

Exposure in a cold draught, or in a blast of cold rain or hail, or even to a cold dry wind, in the case of hunting dogs working much in water, are potent factors.

The glare of the sun through a window at the front of a stall, or from a white surface of snow, ice or water or even from white sand, lime or clay is an active cause. The overdraw check exposing the eyes directly to the sun is often injurious. We must include the irritations caused by the filaria lachrymalis and by various lesions already described—trichiasis, entropion, ectropion, stenosis of the lachrymal duct, etc.

In certain animals a distinct predisposition exists, often as the result of a previous attack, and such are readily attacked under slight disturbing causes.

It should not be forgotten that hyperæmia and even inflammation of the conjunctiva, often accompanies different infective diseases—strangles, influenza, contagious pneumonia, rinderpest, canine distemper, etc.

Symptoms. The discharge of tears and the closure more or less complete of the eyelids are among the earliest and most marked symptoms. The general conjunctiva is congested and that on the inner side of the lids especially, so as often to hide the individual vessels, while on the sclerotic portion they usually remain distinct, tortuous and freely movable with the mucosa upon the sclera beneath. The membrane is more or less infiltrated, swollen and opaque. In two or three days this has increased so that the lids are visibly swollen, and completely closed, or the paw is projected over the cornea. The discharge has become milky or flocculent, and mats the cilia together, or holds the eyelids closed. Unless the cornea has been directly injured it usually remains clear, and there is no photophobia nor contraction of the pupil. The lids, however, are tender and nervous animals show active resistance to any attempt to examine the eye. In aggravated cases a free muco-purulent discharge takes place, and the red, swollen mucosa projects between the eye and the tarsus, constituting *chemosis*. In such circumstances the inflammation may extend to the cornea causing opacity and photophobia or to the iris and choroid or other deep-seated parts.

In the milder forms of the affection there may be little or no hyperthermia, while in severe attacks the febrile reaction may be considerable.

Lesions. The non-traumatic cases which are not complicated by varied wounds and injuries, show exudation and cell proliferation in the palpebral conjunctiva and to a less extent in the bulbar. The conjunctival papillæ are swollen and become visible to the naked eye and in aggravated cases stand out like fungous masses. The superficial layer of cylindroid epithelium is found swollen, opaque and sometimes desquamating. The lymphoid bodies which are most abundant in the depth of the conjunctival sac, are infiltrated, swollen, and bulging in reddened masses.

Other lesions in the nature of granular or follicular hyperplasia, abscess and corneal new growth and ulcers may follow, but will be better considered under separate headings.

The tendency of simple uncomplicated conjunctivitis is to recovery which may be completed in a week or ten days. In case of contused wounds, abscess, ulcer, special infection in a lymphatic subject like the ox, the affection is more likely to be prolonged or followed by grave lesions.

Treatment. The first object must be to remove the cause, hence foreign bodies, displaced lids or cilia, irritant gases, excess of light, cold draughts or exposure, etc., must be sought and corrected. For the removal of the foreign bodies, fine forceps or various common articles (hair pin, lead pencil, a pin head, a folded clean handkerchief, or even the clean finger) may be used. In case of wounds especially, they should be first sterilized. Boiled water which has been cooled to luke warm may be used from a sterilized syringe.

Locally antiseptic and astringent collyria (sublimate solution—1 : 5000, zinc sulphate—1 : 1000, lead acetate, alum, tannin, silver nitrate, etc., are valuable and may even be thrown under the eyelids from a syringe with a finely rounded nozzle and many orifices. Even a 2 per cent. solution of silver nitrate may be applied in the conjunctival sac twice daily, or as being less irritating, a solution of pyoktannin (1 : 1000). If the irritation is great a soft rag wet with the solution may be attached to the headstall of the bridle and hung loosely over the eye, care being taken to prevent drying. A few grains of morphia added to the collyria

will be specially soothing. Irrigation of the eyelids or bathing for ten or fifteen minutes at a time with luke warm water, will often greatly relieve. When the eyelids become agglutinated during sleep or prolonged closure, as in the small animals, cleanse with a tepid sublimate solution, and smear with vaseline. In severe cases local depletion may be resorted to. The hair may be shaved off from an area of 2 inches below the orbit, and a cup applied for ten minutes. Scarification may or may not be resorted to. Or a leech may be applied to the same part.

A laxative often proves an excellent derivative especially useful in costive conditions.

CHRONIC CONJUNCTIVITIS.

Sequel of acute. Same causes. Old horses, young foals, damp lands, night dews, frosts, eczema, follicular scabies. Lesions: swelling of lymph bodies in conjunctival pouch, general congestion, chemosis, hypertrophied papillæ, clouded cornea; Symptoms: as in acute form but less intense. Treatment: Remove cause, irritants, damp soils, stables, etc., glare of light, heat, etc., astringent antiseptic lotions, atropia, mercury oxide, counter-irritation, setoning, cupping, leeching.

This is generally recognized by veterinary writers as a sequel of the acute form, though it may begin *de novo*, from slight persistent causes of irritation. Habitual exposure to fierce light, dusty roads, ammoniacal and other gaseous emanations, and at other times to the dust from musty fodders in a high rack, trichiasis, entropion, ectropion, and indeed any continued irritation may start the disease or keep it up. Leblanc says it is especially common in *old horses* and *young foals* pastured on low damp grounds, bottom lands and lake shores, and in worn out work oxen which have been exposed to cold night dews, or frosts. In this respect it resembles the recurring ophthalmia of the horse which prevails especially in lymphatic subjects. In *dogs*, Leclainche has observed it associated with eczema or follicular scabies of the lids.

Lesions. Besides the general congestion of the mucosa, there are liable to be special lesions in the depth of the conjunctival sac at the point where the mucosa passes from the eyelid to the scler-

otic, causing uneven swellings from infiltration in this region. The lymph bodies or follicles in this part are swollen and project as small red or grayish nodules visible when the eyelid is everted, and at other times the whole infiltrated conjunctiva projects between the lids constituting the condition known as **chemosis.** The papillæ conjunctivæ are also hypertrophied, so as to become visible to the naked eye, and ulcers may be present on the mucosa. The cornea is often clouded blue or milky, or it may have become in part vascular and reddish, or even ulcerated. A more or less abundant muco-purulent discharge is always present.

Symptoms. The inflammation is usually moderate in degree, the eyes water without being habitually closed, the hairs are lost from the cheek, which is habitually wet, the lids are swollen, and like the cilia tend to stick together after sleep, and the whole mucosa is visibly infiltrated and congested, but usually especially at given points, as in the *cul de sac*, on the lids or membrana nictitans. Unless the animal has been treated with irritant dressings, there is much less pain on manipulation than in acute conjunctivitis. On everting the lids the follicülitis in the *cul de sac* and the circumscribed swellings become apparent. The membrana nictitans is partly projected over the cornea, and the eyeball usually appears smaller by reason of its retraction within the orbit.

In cases of eczema, or demodectic mange, the lesions of the skin of the eyelids will furnish the key to the trouble.

Treatment. The first object must be to remove the cause which tends to keep up the malady. Foreign bodies, musty hay fed from high racks, dusty roads, excess of light, windows in unsuitable places, damp stalls or pastures, and local parasitism must be corrected. In case of persistent chemosis keeping up the inflammation, and which will not subside under the usual astringent lotions, the hernial mucosa may be excised. (See chemosis.)

The usual astringent lotions may be employed, sulphate of zinc (2 : 100), sulphate of copper (1 : 200), alum (5 : 100), tannin (10 : 100) to which may be added sulphate of atropia. In place of being simply applied to the surface of the lids, or the cornea, it is usually desirable to inject it into all parts of the cul de sac beneath the upper and lower lids and beneath the nictitating

membrane. Alum 4 grs. and boric acid 3 grains, in water 1 oz. make a good combination. As the mucosa becomes accustomed to one agent, we may change for another. Thus in addition to the above, silver nitrate (2 : 100), and lead acetate (2 : 100) are often useful as alternates. In obstinate cases red or yellowish oxide of mercury in vaselin (5 : 100) may be applied inside the lids and on their margins.

Counter-irritation is often desirable, in the horse a blister of cantharides or mercury biniodide to cover a spot as large as a silver dollar above the anterior end of the maxillary spine (zygomatic), or a stout silk thread as a seton inserted in the same place. In the dog the blister may be applied on the temporal region.

Cupping or leeching beneath the eye, or phlebotomy from the angular vein of the eye may prove useful. Great care should be taken to prevent further injury by rubbing.

PURULENT CONJUNCTIVITIS. BLENNORRHŒA.

More purulent and more infective through dust, tongues, rubbing posts, kennels, swill; a class due to different microbes. Prevalence in dogs, swine, horses, sheep, goats, cats. Symptoms: acute conjunctivitis with excess of pus, follicular swelling and enlarged lymph bodies in cul de sac. Diagnosis. Treatment: Astringent and antiseptic lotions, injected often: silver nitrate: pyoktannin.

The forms of conjunctivitis in which there is an abundant production of pus are usually relatively more inoculable and therefore more liable to pass from animal to animal in a casual manner. Infection takes place through the dried up discharges floating as dust, but more directly by means of the tongue when the animals lick each other, and through posts against which they rub the head. Animals smelling or licking the infected genital organs and then the eyes of their fellows may convey it readily. Dogs occupying the same kennel successively, contract the disease (Guilmot). Swine feeding from the same trough and plunging the face into swill up to the eyes are especially subject to infection. In speaking of such infections one must be understood to refer to a group and not to one specific disease, as that will vary

with the particular pus microbe present, and with the virulency of such microbe in the particular case. In keeping with the greater fertility of microbes in the warm season, these affections have been more commonly met with in summer than in winter, and where the animals are kept in filthy surroundings rather than otherwise. This is above all true of swine. Möller records a wide spread epizootic of gonorrhœal ophthalmia in dogs in Berlin and environs in 1883. In different cases, however, he failed to induce disease in the eyes by direct inoculation with the preputial secretion. Heinman equally failed with the gonococcus of man in inoculations on the eyes of rabbits, and dogs. Fröhner, however, succeeded in infecting the eye of the dog by applying the gonorrhœal discharge of man.

Infecting inoculable, purulent ophthalmia has been reported in the horse (Vernast, Sobornow, Blazekowic, Menard, Möller, Leclainche), in sheep (Repiquet), and in goats (Mathieu). Again Blazekowic found in an infectious ophthalmia of horses, dogs and cats a microbe which was like that of malignant oedema.

The *symptoms* are those of conjunctivitis with especially free production of pus, and a tendency to chemosis or to follicular inflammation in the depth of the conjunctival sac, with irregular swellings of the lymph bodies. The pus accummulates in the inner canthus, inside the lids and along their margins, and tends to mat them together. The diagnosis depends on the rapidity and severity of the course of the malady, on the depth of the congestion and on the profuse suppuration.

Treatment. Astringent and antiseptic lotions are especially indicated. Mercuric chloride (1 : 5000), boric acid (2 : 100), creolin (1 : 100), salicylic acid (1 : 1000), silver nitrate (1 : 200). It is not however enough that these should be applied externally; they should be freely injected under the lids at all points so as to act on the deepest portions of the conjunctiva, and this should be repeated once or twice daily. Or they may be applied with a soft brush. In a specially virulent outbreak silver nitrate (2 : 100) or pyoktannin (1 : 1000) solution may be used. Setons and blisters, laxatives and cooling diuretics may be employed as in the severe types of simple conjunctivitis.

INFECTIOUS CONJUNCTIVITIS IN HERBIVORA. ENZOOTIC OPHTHALMIA.

Causes: infection, pollen, soil emanation, winter or summer; cases of extension by infection. Cattle, sheep, goats, ponies. Accessory irritants. Symptoms: severe purulent conjunctivitis. Papillary and follicular hypertrophy, uniform redness, protruding head, opacititis, erosions, ulcers, photophobia, staphyloma. Treatment: rest, darkness, coolness, elevated head, purgative, diuretics, sedatives; locally solutions of pyoktannin, sublimate, silver nitrate, boric or salicylic acid, atropia, puncture.

Causes. This affection which attacks at once or in rapid succession a large portion or the whole of a herd or flock, is by many held to be infectious while others attribute it to irritant pollen or soil emanation. Its origin from vegetation in flower is held to be supported by its greater frequency in summer than in winter, and the few outbreaks seen in winter are attributed to pollen preserved in the hay. But other things being equal, organized germs are preserved, multiplied and diffused to a greater extent in the hot season so that the origin of the disease from a purely microbian source is at least equally plausible. Certain outbreaks indeed show the transmission of the infection in a most unequivocal manner. A cow suffering from the affection was brought into a stable occupied by a herd previously sound, and in a few days the cow standing next her was attacked, and thereafter a number of others in rapid succession. A small number of cattle from the Buffalo Stock Yards, but which had sore eyes on their arrival in Tompkins Co., N. Y., were placed in a sound herd and the disease spread rapidly to the other members of the herd. Similarly in both sheep and cattle the writer has seen the disease prevail in one herd or flock, while the adjacent herd or flock, separated only by a good stone wall and subject to exactly the same condition of soil, water, exposure, vegetation and pollen has entirely escaped. Kayser has seen it introduced into a herd by a bull; Fünfstück saw a herd of 300 head attacked in a few days, and Klink 20 out of a herd of 40 head in 14 days. Trumbower has never seen an animal suffer a second time. This is the common experience and would suggest an acquired immunity, yet the comparative rarity of the disease forbids a positive conclusion, without further experience. Menard saw an outbreak among the ponies in the Jardin d'Acclimatation,

and as many as fourteen weeks later in the same gardens it attacked the bovine animals.

In sustaining the doctrine of infection, however, we must not altogether ignore accessory causes. Like other affections of the eye, this has seemed to appear especially in low, damp lands, bottom lands, deltas, marshy borders of lakes and level prairies, so that a general lack of tone or a lymphatic constitution may be held to predispose. Nor is it necessary to ignore the influence of pollen, dust and other irritants, which though they may not cause the specific disease, yet prepare the way for its attack by reducing the resisting power of the tissues.

Symptoms. These are the phenomena of severe purulent conjunctivitis. Closed eyes; profuse secretion of tears, sometimes mixed with blood, changing in a few days to a thick, purulent, white or yellow secretion, which collects in masses inside the lids, along their margins, in the inner canthus and on the cheeks, gluing together the cilia, lids and hairs. When separated the lids show a mucosa of an uniform deep red, covered with pus, and irregularly swollen according to the amount of infiltration. Papillary and follicular hypertrophy are marked features, and the nictitans projects excessively over the eyeball. In many cases the cornea becomes opaque and in some instances erosions occur which may cause perforation or loss of the eye. In other instances the ulcers heal with the formation of cicatrices, or the weakened portion of the membrane yields under the internal tension and staphyloma supervenes. In such cases the pupils are contracted.

The disease is usually attended by marked hyperthermia, the secretions, including the lacteal, are materially decreased, appetite and rumination are impaired and the animal leaves the flock or herd. The disease affects cattle and sheep, and Menard and Hoffmann add horses and goats.

Treatment. This must be primarily antiseptic, but without neglecting constitutional disorder. Rest in a dark, cool stall, with the head elevated by tying to a high point. Give at once an active purgative 1½ lb. Glauber salts in 4 quarts warm water, and follow up with cooling diuretics and sedatives (saltpeter ½ oz. and tincture of aconite 20 drops, thrice a day). If the temperature runs very high a few doses of acetanilid or phenacetin may be given. Locally use silver or other antiseptic collyrium.

Locally use pyoktannin solution (1 : 1000) or mercuric chloride (1 : 5000) injecting under the lids so as to bring it in contact with the whole diseased surface. A cloth wet with the same solution may be hung over the eye. Boric acid (1 : 100), salicylic acid (1 : 1000), or silver nitrate (1 : 100) may be substituted for the above when they seem to be losing their efficacy.

Ulcers are treated by pyoktannin or silver nitrate solution applied daily with a soft brush. Keratitis will demand atropia. Abnormal tension or staphyloma will demand puncture of the cornea. Improvement may be expected in a few days and recovery in a week or ten days.

VARIOLOUS CONJUNCTIVITIS.

In cow pox, horse pox, and above all sheep pox, the infection sometimes falls on the conjunctiva, giving rise to the formation of the specific eruption on that membrane. The coexistent eruption on other parts of the body (udder, heels, hairy portions of skin) of the specific variolous eruption, furnishes the means of a satisfactory diagnosis. The lesions in the eye develop rapidly to an extreme severity. The lids are swollen and closed, lachrymation abundant, and early mixed with pus, and when exposed the conjunctiva is strongly congested with circumscribed areas of elevation. In cow pox these are circular in form, raised above the surrounding mucosa, having a deep red areola and a paler, flattened center. In *sheep pox* the elevations have the same general character, but are liable to be more numerous and confluent, and tend to permanent opacities, cicatrices, and perforations of the cornea with loss of the eye. Short of perforation, internal inflammations are not uncommon.

Treatment. At the outset this form may be aborted by the application of silver nitrate solution (2 : 100) or sodium hyposulphite (1½ : 100). Otherwise the local treatment is like that for simple conjunctivitis, cooling astringent and sedative lotions, and if need be, derivatives and eliminants.

APHTHOUS CONJUNCTIVITIS. PHLYCTENULAR CONJUNCTIVITIS.

Closely allied to the last, are those cases in which vesicles appear on the conjunctiva. In exceptional cases these are seen during an epizootic of foot and mouth disease, while in other instances they are associated with eczematous eruptions on other parts of the body, particularly in dogs. In the human subject conjunctival vesicles are often associated with tuberculosis, but this has not been recorded of animals so far. The disease usually makes a rapid eruption, with symptoms of extreme inflammation, and its duration is largely determined by the general disease.

Beside the local *treatment* by astringent or antiseptic and sedative collyria it may be desirable to correct the hepatic, digestive or other disorder on which the eczema depends, or to improve the general health by a course of bitters or even of cod-liver oil.

DIPHTHERITIC CONJUNCTIVITIS IN BIRDS.

Hens, ducks, pigeons. Local inflammation, fibrinous exudate, concreting or becoming cheesy, necrotic, sloughy. Beneath the mucosa red, raw; without epithelium, but excess of lymphoid cells, some only muco-purulent, gravity varies. Grave cases inoculable on birds, mice, rats and rabbits. Bacillus diptheriæ avium, morphology and biology. Pathogenesis. Prevention: exclude germ; cleanliness; pure air; pure food; pure water. Quarantine strange fowls, keep flocks apart, seclude manure, segregate sick, disinfect. Treatment: Antiseptics; boric acid; sublimate: ichthyol; silver nitrate. Infection to man.

A disease of the mouth, fauces, nose and eyes, associated with the formation of false membranes, has been long recognized in birds (hens, ducks, pigeons, etc.), and is known to bird-fanciers by the names of roup, and diphtheria. The disease is characterized by the presence of a local inflammation in patches, associated in the early stages, with a free serous discharge, but, later, with the formation of white, or grayish, fibrinous exudate, which may be at first firm and smooth, later soft and cheese like, and still

later in many cases more or less necrotic and sloughing. In Dr. Moore's experience this is more common on the conjunctiva than on the nares or fauces. The interior of the eye was not involved in the inflammation. Beneath the false membrane the mucosa was devoid of epithelium and the underlying tissue was red, raw and angry, bleeding readily when handled, and infiltrated with an excess of lymphoid cells.

The lesions however were by no means constant. In some even of the fatal cases, the exudate was merely muco-purulent, while in others the false membrane was quite abundant.

The gravity of the affection also varies in different outbreaks or in the same outbreak at different times. Sometimes it runs a very rapid and fatal course, while at others it becomes chronic and comparatively dormant and the great majority recover.

In its more virulent form it is readily inoculable on birds, mice, rats, and rabbits, while the milder cases are not easily propagated in this way.

The false membrane contains a variety of saprophytes, and among them the pathogenic bacillus, which may be obtained in pure culture by inoculating it on a rabbit or mouse. Emmerich believed this bacillus to be identical with the Klebs-Löffler bacillus of diphtheria in man, but Löfflller recognizes it as essentially distinct.

The *bacillus diphtheriæ avium* is 0.8 to 1.5μ long, by 0.8 to 1.2μ broad. The ends are oval so that short specimens seem round. In bouillon cultures they form chains or clumps. They stain in aniline dyes, most deeply at the poles. Are not stained by Gram's method. They are non-motile, ærobic, grow in agar, and alkaline bouillon, but render the latter acid in one or two days. Do not grow on peptone gelatine, nor produce gas with sugars. Are killed in 5 minutes at 58° C. (137° F.). Are killed by dryness in 24 hours, by sulphuric acid (0.25 : 100) in 10 minutes, by lime water in 1 minute, and by sulphur fumes.

Pathogenesis. Eight rabbits inoculated subcutaneously with 0.1 cc. of the bouillon culture died in 18 to 36 hours. Of several white and gray mice inoculated only one died. Inoculations of mature hens subcutem and on the nose had no effect. Inoculation of a six weeks old chicken caused death in 4 days with bacilli in the liver and blood (Moore). Inoculation of pigeons pro-

duced the disease (Löffler). It seems doubtful whether the milder forms part with their virulence to birds, when cultivated artificially, or whether a special susceptibility is required in order to render the inoculation effective.

Prevention. Besides the general conditions of good hygiene, cleanliness, pure air, and pure water, the strict exclusion of the germ is the great desideratum. New fowls should not be taken into a flock, when they show any indication of disease in the mouth, throat, nose or eyes, nor when they come from a flock in which such signs of disease can be found. When examination of the flock, from which they are sold, cannot be made, the new fowls should be placed by themselves in quarantine until proved sound. Sound flocks should not be allowed to wander at large and mingle with the birds that are unsound, or open to suspicion. Neither should they be allowed to come in contact with manure from suspected poultry yards. If disease of air passages, mouth or eyes appears, separate at once the diseased fowl, and sprinkle roost, house and yard with dilute sulphuric acid, quicklime, or other disinfectant.

Treatment. Beside separation and disinfection the local use of antiseptics to the surface divested of the false membrane gives the best promise. Boric acid solution (4 : 100) or sublimate solution (1 : 5000) may be applied to the eye. For the nose and mouth somewhat stronger applications may be made.

Prevention of infection to man, The essential difference of the Klebs-Löffler bacillus of diphtheria in man, and the microbe of this affection in fowls does not exclude the necessity of avoiding contagion from birds to man. Among reported cases of such infection are: (1) That at Wesselhausen, where 4 attendants on the fowls contracted the disease from the sick fowls at a time when no other cases existed in the human population: (2) That of Sebdon where 6 persons suffered and 10 fowls fed by a hospital attendant also suffered: (3) That of Tunnis in which diphtheria prevailed in fowls, and soon also in those who fed them producing an extended epidemic: (4) That of Jacksonville, Ill., where a diphtheritic chicken, conveyed the disease, with fatal effect, to a child which fondled it (Moore).

These and other similar instances seem to show that the disease of the bird may become so virulent as to be communicated to

man, and the disease of man so potent as to be transmissible to the bird. Every precaution therefore should be taken to prevent infection passing from one to the other.

FOLLICULAR CONJUNCTIVITIS.

Common in dogs. Violent congestion with enlargement of lymph follicles, or beneath the nictitans, dark red, like millet seed, also over sclera and lids. Enlarged lymph follicles with excess of lymphoid cells. Often chronic. Entropion. Infection. Treatment: astringent and antiseptic lotions: lunar caustic: cocaine and crushing of follicles: pure air: excision of nictitans.

Fröhner has drawn attention to the frequency of this affection in the dog, which according to him attacks 40 per cent. of the race. It is especially liable to begin on the inner surface of the membrana nictitans, and though it may be at first somewhat hidden by the severe congestion, yet when that subsides in part, the swollen follicles can be seen as dark red elevations, the size of a millet seed, when the nictitans has been everted. It may extend over the sclerotic and palpebræ. Microscopically these swellings are found to be enlarged lymph follicles, with an excess of lymphoid cells.

The disease tends to assume a chronic course and may cause entropion and other troublesome lesions. The diagnosis depends on the recognition of the swollen follicles, and especially in the depth of the conjunctival sac.

It is presumably infective yet all Fröhner's inoculation gave negative results.

Treatment. In the earlier stages the common astringent and antiseptic lotions may be used. Sublimate, or boric acid lotions are especially valuable for their antiseptic properties. Should these fail, the eyelid and nictitans may be inverted and the individual follicles touched with a fine pencil of lunar caustic, the resulting smarting being lessened by application of cold water. In case they should still prove obstinate, the conjunctiva may be cocainized and the follicles individually crushed with ciliary forceps. Pure out door air is a prime essential in the treatment.

In extreme cases Fröhner counsels the excision of the membrana nictitans.

NEOPLASMS OF THE CONJUNCTIVA.

Pinquecula : fatty growth : Lipoma : Melanoma : Dermoid tumor in young dogs, calves, sheep. Cocainize the part and excise. Cold water : silver nitrate. Polypus. Pterygium.

A pale fatty looking elevation on the sclerotic at the inner side of the cornea is not unknown in the dog, resembling **pinquecula** of man. It has not been seen to prove harmful and may be safely ignored.

Lipoma has also been observed (Müller) and when troublesome may be removed by excison with scissors.

Melanosis is met with in gray horses in connection with the same disease of the skin of the lids, and usually with generalized pigment tumors. In the latter case surgical interference is useless unless it is to secure a very temporary relief.

Dermoid Tumor of the Conjunctiva. This consists in a cutaneous product, consisting externally of a mass of epidermic cells, beneath which are connective tissue, fat cells, and muscular fibres, glands and growing hairs. It usually extends inward from the outer portion of the sclerotic conjunctiva and may encroach on that of the cornea. It is firmly adherent to the sclera, and sometimes to the cornea by its base and deeper aspect, but the apex is free and more or less projecting. The color is yellow, or more or less blackened by pigment or even reddened by blood. It has been observed, above all, in dogs, Prince reports a case in a calf and Zundel in a sheep. The Cornell Veterinary College clinic has furnished cases in ox and dog. They have, however, nearly always been seen in young animals and are probably congenital.

These are easily removed from the eye anæsthetized by a 4 per cent. solution of chloride of cocaine. The inner projecting end of the tumor is seized by rat-tooth forceps, and carefully snipped off with sharp scissors curved on the flat. Where adherent to the cornea it must be carefully handled, but where attached to the thicker and more resistant sclerotic it can be dealt with more freely. A pencil of silver nitrate may be used to check the bleeding, or that may be effected by cold water freely applied.

Polypus, a small, pale, pediculated tumor of the conjunctiva is described by Lafosse and should be removed by scissors, and bleeding checked by cold, wet applications.

PTERYGIUM.

This name is employed to designate a triangular conjunctival fold broader at its sclerotic end and gradually narrowing to its corneal extremity, with loose, slightly overlapping borders, and firmly fixed to the structures beneath. It is more vascular than the surrounding conjunctiva, and its comparatively large blood-vessels have suggested the veins of an insect's wing—hence its name. The growth may extend from either canthus toward or partly over, the cornea.

Möller and Leclainche claim its existence in dogs, though rarely to such an extent as to demand surgical interference. Dunewald operated on a case in the cow.

Unless growing, it need not be interfered with. It may be dissected up with sissors the narrow end being dragged on by forceps. Another method is to cauterize the narrow end with the electric cautery which leads to material contraction of the entire mass.

XEROSIS CORNEÆ (EPITHELIALIS). DRY KERATITIS.

This is described by Mayer as following distemper in dogs. It seems to begin in the epithelial layer of the conjunctiva, which becomes dry, lustreless, spotted, opaque and fatty so that water runs over it without wetting it. It may extend deeply into the substance of the cornea and lead to the development of a scar. When scraped and examined under the microscope the scrapings are found to consist of epithelium undergoing fatty degeneration and myriads of *xerosis bacilli*. As the disease takes occasion to attack by reason of the debility of the system, the *treatment* is mainly corroborative and tonic, including the arrest of the affection on which the weakness depends. The early application of antiseptics is desirable (iodoform 1, vaseline 10; mercuric chloride 1, vaseline 3000). Warm compresses and a bandage may be tried.

WOUNDS OF THE CORNEA.

Causes: harness, whip, nail, hay, straw, stubble, thistles, spikes, twigs, pine needles, cones, burdocks, stones, gravel, glass, splinters of wood or metal, scratches, stings. Symptoms: closed lids, epiphora, sight of lesion, soon cloudy swelling, opacity. Treatment: antiseptic bandage and lotion, boric acid, sublimate, potassium permanganate, avoid lead or zinc, atropia, cocaine, with perforation, bandage, eserine, excision. For foreign body, antiseptic cotton, spud or curette.

Causes. Corneal wounds are common in working animals by reason of contact with harness, canes, whips, etc., and in the stable from contact with nails or with the hard ends of hay or straw. At pasture the cornea is injured by the ends of long stubble, the sharp points of thistles, the spikes of various thorny plants, and twigs of bushes and trees. The last named factors are especially operative in hunters and horses worked in forests. Punctures with pine needles and cones, and with burdocks, are other common causes. Stones, gravel, pieces of glass, and splinters of wood or metal, produce traumas of the cornea, and, in cats and dogs, scratches and even perforations with the claws are common. In this connection the stings of insects are not to be forgotten.

Symptoms. There is always a prompt and complete closure of the eyelids and a profuse secretion of tears. Then on parting the eyelids with finger and thumb, the lesion of the cornea, its nature and extent should be recognizable. In case of a small, punctured wound, however, as with a smooth thorn or other conical body, the normal elasticity of the corneal tissue may lead to such a perfect coaptation of the divided edges that the lesion may escape even a close scrutiny. If the case is seen early, before time has been allowed for cloudy swelling and opacity the wound is all the more likely to escape observation. In incised, scratched and torn wounds, on the other hand, the seat and nature of the lesion are made out with the greatest ease.

Treatment of a slight wound which is at once recent and free from infection, is by a simple antiseptic bandage and lotion. Boric acid (1 : 100), sublimate solution (1 : 5000) or potassium permanganate solution (1 : 100) may be used. Lead and even

zinc salts, are liable to precipitate in the abraded tissue and cause a lasting opacity. If the pain is severe it may be moderated by the addition of atropia sulphate, or a solution of 1 to 100 water may be instilled into the eye several times daily. Cocaine makes an excellent substitute. In deeper wounds, perforating the cornea and allowing the escape of aqueous humor, there may be prolapse of the iris through the wound. It may be pressed back with a flat sterilized spatula, and retained by bandage and a course of eserine. Should it still escape, it must be seized with forceps, drawn out and snipped off with a sharp pair of sterilized scissors. The greatest care must be taken to avoid infection which may cause panophthalmitis and destruction of the entire eyeball.

FOREIGN BODIES IN THE CORNEA.

In case of penetration of the cornea by thorns, thistles, glass, metal, etc., there usually follows inflammation with a red area around the offending object. If the foreign body is a piece of iron there is a brownish area caused by iron oxide. Focal or transillumination will usually reveal the object. Should both fail, a solution of fluorescin when applied will develop a greenish area around it.

Treatment may be made as advised by Gould by pressing a little antiseptic cotton to the front of the eye, so as to entangle and withdraw the foreign body when the eyeball is rolled. Failing in this we may cocainize the eye and remove the offending object with a small curette or spud. A careful focal illumination of the eye will enable the operator to see and remove the smallest particles without injury. Subsequent treatment is that of wounds.

ACUTE KERATITIS. INFLAMMATION OF THE CORNEA.

Extension from conjunctivitis, wounds, foreign bodies, bites, stings, blows, infections, filaria. Symptoms: eye tender, closed, epiphora, red, pannus, photophobia, congested sclera, opaque or ramified red cornea, or diffuse red, exudation, suppuration, corneal abscess, ulcer, perforation, prolapsus iris, panophthalmia. Focal illumination. Recovery. Permanent

cicatrix or opacity. Lesions : exudates of lymph and leucocytes into corneal layers : embryonic tissue : vascularization : abscess : ulcer : cicatrix : opacity : staphyloma : hypopion : prolapsus iridis : panophthalmia. Treatment : antiseptic astringents, atropine, leeching, derivatives, blister, seton, opacities ; in severe cases antiseptic puncture, sublimate lotion, silver nitrate, potassium permanganate, boric acid, pyoktannin : in perforations antiseptic bandage and eserine, iridectomy : in chronic cases mercury oxide.

Keratitis occurs in all domestic animals as a primary disease, or as an extension from conjunctivitis.

Causes. Extension from acute, enzootic, infectious conjunctivitis in sheep and cattle has been noticed by a great number of observers. Bayer and Lohoff have studied maculated keratitis of the superficial layers in horses. Again it has followed wounds by foreign bodies, spikes of vegetables, particles of iron and glass, blows of whips, or insects, stings, etc. It also occurs in connection with the local action of particular poisons, such as variola (foot and mouth disease), canine distemper, etc., and from the local irritations caused by trichiasis or entropion or by the filariæ lachrymalis (ox) and palpebralis (horse).

Symptoms. The eye is extremely sensitive, and habitually closed, with a profuse flow of tears, and a disposition to resist opening of the lids. When exposed the cornea is seen to be more or less clouded and perhaps reddened by the formation of vessels proceeding from its sclerotic margin. This is known as *pannus*. If the anterior chamber is still visible the pupil is found to be contracted showing photophobia. The congestion is first visible in the sclerotic and in the absence of pigment is most intense near the margin of the cornea. Upon the cornea itself it is preceeded by a deep white opacity, into which the vascularity gradually extends. The whole cornea may finally become of a bright pink hue.

The congestion of the cornea may advance to fibrinous exudation, or the formation of pus between its layers, to molecular degeneration and the formation of ulcer, or even to perforation and escape of the aqueous humor. In this case prolapsus iris, panophthalmia and destruction of the eye are likely to ensue.

Ulcer if not readily seen with unaided vision can be easily recognized by the aid of focal illumination, and abscess can be detected by the presence of a sharply circumscribed centre of in-

tense opacity, white or yellow, and some bulging of the membrane.

The pus may be absorbed, or it may escape by rupture and discharge externally, or into the anterior chamber when the resistance is least in that direction, and when this takes place, a dangerous internal infective inflammation is the result.

In the slighter forms of keratitis the inflammation may come early to a standstill, and recede, tenderness and photophobia pass off, the eyelids may be opened, and the corneal opacities gradually disappear. If any portion of the cornea has become vascular, that portion is liable to remain opaque or even pink.

Lesions. Under the influence of an irritant on the cornea, the vessels in the margin of the sclerotic become actively congested and pour out lymph freely, leucocytes also escape and with the lymph pass through the lymph channels into the substance of the corneal tissue. Here they undergo active fission and increase, and the normal cells of the corneal tissue multiply in like manner, so that in a short time there is an extraordinary production of embryonic cells. Into the embryonic tissue so formed, blood escapes from forming loops of new vessels, and this goes on extending until the whole cornea may have become vascular. Degenerations in the newly formed structure may result in suppuration, (*hypopyonkeratitis*) or molecular decay and ulceration, (*ulcus corneæ*) or organization may take place into the fibrous tissue with contraction and permanent opacity, (*macula*) or a hyperplasia may form in the shape of a staphyloma.

Among the other complications may be named pus in the anterior chamber (hypopion), prolapsus of the iris, iritis and panophthalmia.

Treatment. In the milder form of keratitis, antiseptic astringents with atropia sulphate are often effectual: zinc sulphate, boric acid or alum (1 : 100). Any direct mechanical cause of the irritation must be removed, and the eye rendered as far as possible antiseptic or aseptic. Derivatives also may be of service, and Trasbot especially advises bleeding from the angular vein of the eye but only in the very earliest stages. Cupping, leeching or setons may be employed. Excessive tension may be relieved by puncture of the cornea near its margin. The remaining opacity after the inflammation has subsided may

usually be removed by touching it daily with a camel's hair brush dipped in a solution of silver nitrate (1 : 200).

In the more severe cases antiseptic lotions are even more essential, mercuric chloride (1 : 5000), potassium permanganate (1 : 100), boric acid (1 : 100), silver nitrate (1 : 200). Careful massage is of value.

Ulcers may be touched daily with a solution of silver nitrate (1 : 400), or of pyoktannin (1 : 100).

Perforations must be treated by antiseptic bandage, eserin, and in case of necessity, iridectomy as advised under perforating wounds.

Abscesses of the cornea should be opened with a flamed needle and treated with antiseptic lotions.

Obstinate cases are often benefitted by ointment of yellow oxide of mercury 1, vaseline 10, or by the red oxide of mercury or calomel.

POISONING WITH COTTON SEED OR COTTON SEED MEAL.

Poisons in cotton plant: on man, pig, cow, stock cattle. Symptoms in latter: Nervousness, debility, exhaustion, in-coördination, paresis, dyspnœa, dullness, anorexia, drooping head, trembling, lachrymation, corneal ulcer, opacity, vesiculation; unilateral or bilateral; with rest and change of food recover in five days except eye lesions. Treatment: suspend cotton seed, purge, and treat eye lesions.

The cotton plant develops poisons for various genera of animals. The bark of the root is a favorite abortifacient for woman and may be used for the same purpose in the domestic animals. The seed when fed continuously to swine will destroy life with symptoms of scorbutus, and grave constitutional disorders. Cotton seed meal fed in excess to dairy cows has a bad reputation for inducing garget and mammitis. In stock cattle it has the reputation of producing diarrhœa, running from the eyes, abscess and ulceration of the cornea, staphyloma, hyperthermia (103° to 109° F.), swelled legs, congestion of the liver and spleen, and high colored urine. As described by Dr. F. C. McCurdy, of Kansas City, the southern cattle arrive in poor condition, seem nervous, weak and exhausted, move with an uncer-

tain, staggering gait, and may fall and make convulsive but ineffectual efforts to rise. Dyspnœa, blue mucosæ, and protruded tongue are noticeable in such cases. In the slighter cases, dullness, inappetance, suspended rumination, drooping head, and trembling limbs are characteristic features, and profuse lachrymation is constant. In some eyes there is a small opaque spot around a minute ulcer containing small granules like dust or sand, and situated in the centre of the cornea on the line of approximation of the two eyelids. Larger opaque areas when present were generally confined to the corneal surface, without any areola of distended vessels, and without a vascular zone at the junction of cornea and sclera. In certain cases the whole transparent cornea stood out in the form of a vesicle, so prominently as to interfere with closure of the eyelids. The affection might attack both eyes or only one.

An important feature is that cattle coming from the cars in this condition and left at rest for five days on hay without cotton seed recovered rumination and appetite, and the weakness and nervous excitement or depression disappeared. There remained only the lesions of the eye which progress tardily according to their extent or severity.

The southern origin of the cattle, together with the congested liver and spleen and the high colored urine would have suggested the southern cattle fever, but from the promptitude of the recovery under a change of regimen and the prominence of the lesions of the eye.

The important point in connection with this subject is the prophylaxis by avoidance of the too liberal diet of cotton seed. When the disease has actually set in, the true course is to suspend this aliment, clear the bowels of any that may remain therein, and treat the lesions of the eyes according to their respective conditions.

CHRONIC KERATITIS.

Sequel of trichiasis, entropion, eczema, etc. Age. Symptoms: moderate, lids partly closed, cilia matted together, crusted, cornea clouded, dull, with ramifying vessels. Resolution. Fibroid degeneration, permanent opacity. Treatment: tonic regimen, out-door exercise, iron, bitters, calcium sulphide, astringent antiseptic collyria, atropia, mercury oxide.

This is especially common in dogs in warm latitudes. Trichiasis and entropion are perhaps the most common of the direct causes. Eczema and other skin eruptions affecting the lids are additional causes, while old and debilitated dogs are especially subject to the affection. It is less frequent in horses.

The *symptoms* are much less severe than in the acute form. The lids are usually partially but rarely completely closed, lachrymation may be absent and is never excessive, the secretion usually sticks together the cilia and lids, and always forms crusts on them, the palpebræ are less sensitive than in acute keratitis, the cornea is habitually clouded of a bluish-white color, yet in the main partially transparent and without the disc opacities of the acute type of disease, and the pupil, which is usually visible in a good light or under oblique illumination, may be slightly but is not excessively contracted. The surface of the cornea seems to have lost some of its polish, and in its substance blood vessels can usually be made out.

Under favorable conditions these cases may end in resolution and especially under a change of food and environment. In less fortunate cases they result in a fibroid degeneration of the cornea and deep permanent opacity.

Treatment. It is usually desirable to change the regimen so as to improve the general health, and to allow a fair amount of out-door exercise. In the very old and debilitated the case is rather hopeless. A course of iron or bitters will sometimes have a good effect. In other cases sulphide of calcium $\frac{1}{10}$ grain thrice a day will prove useful. Eczema must be treated secundum artem.

Locally astringent and antiseptic collyria may be used as in the acute form. Atropia, 5 grs. to the oz., is a valuable adjuvant, to be instilled in drops. Ointment of yellow oxide of mercury, a piece like a pin head rubbed inside the lids once or twice a day often acts well. Finally Trasbot strongly commends liquor of Van Swieten.

OPACITY OF THE CORNEA. NEBULA. MACULA. LEUCOMA.

Nebula, macula, leucoma, pigment spots, infiltration, cicatrix, vascular or not, result of lead, silver or cocaine. Treatment: silver nitrate solution in young and vigorous; calomel: iodoform: avoid mercury and iodine at the same time. Tatooing.

As a sequel of inflammation of the cornea, persistent opacities are very common occurrences. These may last only a short time after the subsidence of the inflammation, or they may be persistent and chronic. They are of all degrees of severity from a mere bluish haze to a dense white cloud, or a dark pigment spot.

The term **nebula** is given to the slightest form which appears as a grayish blue but still transparent blue and may be so slight as to pass without recognition except under focal or oblique illumination. It shades off gradually into the adjacent healthy cornea, and is often seen as a marginal zone when the centre of the cornea is clear.

Macula is more marked, requiring no special illumination to detect it, especially when the dark pupil forms a background for the affected area. It is not, however, of a clear white, but of a grayish blue tint.

Leukoma is a dense white spot or patch which reflects all the light falling upon it, and has usually a sharply circumscribed margin.

Pigment spots are usually on the membrane of Descemet and are the result of a previous adhesion of the iris and detachment of a portion of its pigment.

The white opacity may be merely a remnant of inflammatory infiltration or it may be a fibrous cicatrix with or without a remaining minute ulcer. It may be the result of an insoluble deposit of lead or silver in the tissues. Sometimes it will form as the result of the application of cocaine.

Treatment. A case of slight inflammatory infiltration can usually be cleared up by touching it daily with a solution of 2 grs. silver nitrate in an ounce of distilled water. This is especially satisfactory in the young and healthy, in which the

power of repair is greatest. Finely powdered calomel or iodoform applied to the cornea will often prove effective. In case potassium iodide has been given by the mouth, calomel or corrosive sublimate is liable to form mercurous or mercuric iodide and cause ophthalmia. The same is true of iodoform if mercury has been given internally. As a last resort tatooing the spot has been resorted to, to hide the opacity.

ULCER OF THE CORNEA.

Infection of abrasions may cause ulcer. Age. House dogs. Puppies on vegetable food. Exhaustion. Starvation. Improper, insufficient diet. Specific microbes and toxins. Symptoms. Ulcer with peripheral zone of opacity. Photophobia. In marasmus little other local trouble. Diagnosis by oblique focal illumination or fluorescin. Granulation of Descemete's membrane. Escape of aqueous. Keratitis. Panophthalmia. Staphyloma. Prognosis in debilitated, vigorous. Treatment: tonics, fresh air, good food, sunshine, exercise, silver nitrate, mercuric chloride, iodoform, alcohol, chlorine water, boric acid, cocaine, eserine, atropine, warm antiseptic compress, juice of fresh cassava.

Causes. Wounds of the cornea making an infection entrance for pus microbes, are liable to lead to ulceration, and a corresponding destruction of the epithelium and superficial layers by inflammation, may start a similar ulcerative process. Apart from these conditions, ulceration is especially liable to occur in very old dogs, in closely confined house dogs, in puppies raised on an exclusive diet of vegetable food, and in animals worn out by disease, exhaustion, starvation, or improper and insufficient diet. Majendie's dogs fed on sugar, starch and other imperfect diet, suffered in this way. Finally, the local action of certain specific disease poisons, enzootic purulent ophthalmia, canine distemper, dogpox (Trasbot), equine influenza (Schindelka), sheep-pox, and blennorrhœa (Möller), leads to ulceration.

Symptoms. In keratitis there is usually a marked local opacity in the centre of which the breach of the surface may be found. The attendant photophobia with closure of the lids and pupil is strongly suggestive of ulcer. In the specific diseases, the local inflammation, the rapid progress of the lesion and the coexist-

ence of the particular infective disease are characteristic. In cases due to debility and marasmus the disease may appear with little indication of attendant irritation, lachrymation, tenderness, photophobia, or even opacity. At one circumscribed point only is there a grayish cloud, perhaps no more than a thirtieth of an inch in diameter, and slightly projecting. This becomes soft and gelatinous and finally drops off, leaving a shallow excavation or abrasion, surrounded by a narrow grayish zone. This necrobiosis may extend inward and even penetrate the membrane, before the lesion has enlarged to more than a hemp seed in diameter. In other cases lateral extension occurs.

It is always important to recognize the ulcer at an early stage, and this may be done by oblique focal illumination and the use of a magnifying lens. In case of doubt a drop of solution of fluorescin placed on the cornea and at once washed out, will promptly reveal the lesion by the high color given to the tissues which have been denuded.

When perforation has taken place the membrane of Descemet may bulge out of the orifice and undergo granulation, or it may open and allow the escape of the aqueous humor. Active keratitis and even panophthalmia are liable to follow perforation. Again, the escape of aqueous humor tends to the approximation or contact of the iris with the cornea, where it may become adherent and staphyloma may ensue.

Prognosis is unfavorable in debilitated subjects, and when the lesion is extensive and in the line of vision. In slight recent cases in good constitutions it is favorable.

Treatment. Debility must be met by tonics and rich diet, fresh air, sunshine and exercise. Specific diseases must be met according to their nature.

Locally the daily application of silver nitrate lotion (1 : 200) is often very effective, proving an excellent antiseptic, checking the microbian proliferation, and coagulating the albumen in the wound so as to form an antiseptic barrier to further invasion. A mercuric chloride solution (1 : 5000) is an excellent substitute. Iodoform powder though less antiseptic, is especially valuable in favoring the healing process. It is dusted over the cornea, and the upper lid immediately drawn down and held over the cornea for several minutes. If this is neglected the dry powder is re-

moved by the flow of tears, and the movement of the lids and membrana nictitans. Trasbot recommends dilute alcohol (5 : 100). Moller advises chlorine water reduced to one-third the standard strength, or boric acid solution (2 : 100). Bouley found good results from a cocaine solution. Cadiot and Almy get the best results from creolin (.5 to 1 : 1000) 5 or 6 times a day, with eserine.

In all cases great relief can be obtained from a strong atropia lotion (1 : 100). Indolent cases may often be helped by warm antiseptic compresses, which seem to stimulate the circulation and nutrition of the part. The juice exuding from the scraped fresh cassava and concentrated to a syrupy consistency, is strongly antiseptic, and used with atropia or pilocarpin is the best agent known for senile ulcer (Risley).

In perforation use eserine, and antiseptic bandages and in case of prolapsus iris, excise as already advised.

CORNEAL STAPHYLOMA.

Bulging corneal scar with adherent iris: from perforation, escape of aqueous, intraocular pressure, vascularization of cornea. Diagnosis by central cicatrix, vascularisation, pigmentation. Oblique illumination. Treatment: iridectomy, eserine. Suture. Enucleation.

This is a bulging forward of a corneal scar with the iris adherent to its internal surface. It may originate in perforation of the cornea and escape of the aqueous humor, or in intraocular pressure that advances the iris until it comes in contact with the cornea, which becoming adherent and receiving an abnormally large supply of blood or plasma, softens and bulges outward. It may grow out to a great length in some cases, Eck has seen it two inches in the horse, and somewhat smaller in an ox. May records a case affecting both eyes in the dog.

Diagnosis is not usually difficult. The scar in the midst of a granulating projection of the cornea is nearly conclusive, but the recognition of pigmentation of the growth and the adherent iris often revealed by oblique illumination will nearly always show the true nature of the case.

Treatment is by iridectomy and eserin if the disease can be recognized in its earliest stages, but it is rarely satisfactory. Later the choice may be between excision of the staphyloma and coaptation of the edges of the wound by suture, and the enucleation and removal of the eyeball. The last resort is preferable to the continued irritation of the staphyloma by the lids and cilia under the ocular movements.

ECTASIA CORNEÆ. KERATOCONUS. CONICAL CORNEA.

This consists in a thining and protrnsion of the cornea in the form of a blunt cone, without loss of transparency. It has accordingly been called *staphyloma pellucida*. There is a gradual attenuation and distension of the corneal tissue from some unknown cause. It has been seen mainly in the yonng and is manifestly due to a trophic defect. Stockfleth records a case in a foal and Bayer in a cow. No satisfactory treatment has been proposed, but as the trouble usually comes to a standstill without perforation, it can be left to take its course. With rapid increase and manifest tension antiseptic puncture of the cornea or even iridectomy might be tried.

KERATOGLOBUS.

This is a variety of *ectasia* in which the clear, pellucid, protruding cornea is more globular and less conical in outline. It is seen especially with enlargement of the entire eyeball (*buphthalmus*).

TUMORS OF THE CORNEA.

Various tumors may grow from the cornea. Dermoid cysts may implicate the cornea and demand excision. Malignant growths demand extirpation of the eyeball.

WOUNDS OF THE SCLERA.

Covered as it is by the bones of the orbit, and by the palpebræ the sclera is little liable to traumatic lesions. Wounds with swords, needles, nails, splinters of wood, and other sharp pointed bodies are not unknown, however, and penetration by shot is especially common in setters. Rupture from blows of clubs, beams, poles, stumps, etc., are also met with.

The *symptoms* are profuse lachrymation with more or less of blood, and when the eyelids are separated the wound may be discovered and its gravity estimated by protrusion of the vitreous. Slight injuries which are not infected heal readily under the treatment recommended for keratitis. Infecting and penetrating wounds are liable to cause panophthalmitis and destruction of the eye. Foreign bodies, if present, should be removed when possible. Pyoktannin is especially recommended by Stilling.

EPISCLERITIS. INFLAMMATION OF THE SCLERA.

Scleritis in man is described as a manifestation of rheumatism, gout, or tuberculosis. It occurs in animals in connection with traumatic lesions, with iritis, cyclitis and choroiditis and is manifested by more or less congestion, swelling and tenderness of the sclerotic, but is always subordinate in importance and the *treatment* demanded is for the more serious disease.

ECTASIA (BULGING) OF THE SCLERA.

Cases of this kind are adduced by Schleich and Mayer, in dogs, in which there was a corresponding bulging or even an absence (*coloboma*) of the choroid and retina. With a large protrusion of the sclera behind, there was a shrinkage of the front of the globe (*microphthalmus*), so that an atrophy might be suspected. The condition is irremediable.

PROLAPSE OF THE IRIS.

This has been already referred to as a complication of perforating ulcer or wound of the cornea. If it cannot be returned and maintained by a compression bandage and eserin, the only resort is to draw out the prolapsing portion and cut it off with scissors, the eye and instruments having been rendered thoroughly aseptic.

INTERNAL OPHTHALMIA.

Diagnosis of internal ophthalmias difficult. Causes: as in conjunctivitis, extension of conjunctivitis or keratitis to iris, choroid, ciliary circle, retina; Lymphatic constitution, damp soil, air and stable, pit life, dentition, grain feeding, training. Symptoms: ophthalmic symptoms generally, enlarged ciliary vessels in sclera not movable; white zone around corneal margin; iris dull, brownish, sluggish; intraocular tension increased: flocculi in aqueous humor: photophobia: oblique focal illumination: sudden change from darkness to light: synechia: ophthalmoscope. Cyclitis. Diagnosis: from keratitis, recurrent ophthalmia. Lesions: according to chief seat of the disease: inflammation of Descemet's membrane, iris, choroid, ciliary circle, lens, vitreous and retina in variable degree. Opacity of aqueous, lens, capsules, or vitreous. Prognosis: always grave, often vision impaired or lost. Treatment: rest, pure air, apart from strong sunshine, removal of causes, local bleeding or cupping, derivation, purgative, cooling diuretics: locally astringent antiseptic lotions, cocaine, homatropine, blister, undue tension antiseptic puncture, mercury oxide ointment; in rheumatic cases salicin or sodium salicylate.

In the domestic animals it is not always possible to distinguish between inflammations affecting different portions of the inner and middle coats of the eye (iritis, cyclitis, choroiditis, retinitis), so that it is convenient to give in general terms the phenomena and treatment of the class known as *ophthalmia internus*. This is all the more appropriate that inflammation of one of these divisions so frequently extends to the others producing *panophthalmitis*, that the disease in one usually implies an early implication of all.

Causes. Many of the causes of conjunctivitis, when acting with special intensity, or for too long a time, may cause internal ophthalmia. Severe blows, bruises, punctures, lacerations, sand, cinders, dust, lime, foreign bodies inducing traumas, sudden transitions from darkness to bright sunshine, habitual exposure to sunshine or to the reflection from snow, ice or water, through a window in front of the stall, the abuse of the overdraw check rein, the glare of electric light or of lightning flashes, draughts of cold damp air between windows or doors, the beating of cold storms on the eyes and skin, a sudden chill from plunging in water or standing in a cold draught when perspiring, blows with branches, pine cones or needles in the eye, the constant irritation from entropion, trichiases, burdocks or thistles in the forelock, irritant gases, etc., are among the factors which coöperate in setting up the disease. Again diseases of the digestive organs, rheumatism, influenza, canine distemper, brust-seuche, petechial fever, variola, eczema, and aphthous fever may be direct causes. Conjunctivitis and keratitis are liable to merge into irido-choroiditis by extension, and above all when owing to perforation of the cornea a direct channel is opened for the easy entrance of infective, pathogenic microbes. A lymphatic constitution, connected with low breeding, or living in a low, damp, cloudy region, or in dark, damp, impure stables, has a strongly predisposing influence. The period of dentition, connected as it usually is with domestication, stabling, grain feeding, and training is often a potent accessory cause.

Symptoms. With the general phenomena of superficial or external ophthalmia there are some indications which may be called pathognomonic. These may be summarized as follows: in eyes devoid of pigment the *enlarged ciliary vessels run deeply and are not tortuous, nor mobile when rubbed; the scleral redness increases toward the margin of the cornea, but leaves a white zone in front of the penetration of the ciliary vessels; the iris has lost its clear reflection, appearing dull or brownish; the pupil is contracted and sluggish in response to light and darkness, it may be fixed or may show marked unevenness in its margin: the tension of the eye ball is often increased, flocculi of lymph may be seen in the aqueous humor settling into the lower part of the anterior chamber.* This deposit may be white or yellowish or it may even be reddened by ex-

travasated blood especially in traumatic injuries. In traumas, too, the cornea and even the aqueous or vitreous humor may be opaque. In cases resulting from exposure to cold or from internal causes, the media of the eye are at first clear and transparent. The condition of the interior of the eye is usually to be learned by examining the patient as he stands facing the light from a dark back ground. A stable door or window will afford the requisite amount of rays falling from above and from each side upon the interior of the eye. The observer looks indirectly or obliquely and under favorable conditions can see the iris and through the pupil. If the pupil is unduly closed it may often be dilated by instilling a few drops of a 5 per cent. solution of atropia and waiting for fifteen or twenty minutes.

The examination is made more satisfactorily with a candle or other single source of light in a dark chamber. If this light is surrounded by a chimney opaque except at one side which is directed toward the eye, the results are much more satisfactory. Focal illumination with a biconvex lens, or oblique illumination will show a swollen condition of the iris with uneven bulging swellings at different points, and generally a lack of the clear dark surface which marks the healthy iris. It may be yellowish or brownish, rather than dark, or blue, or yellow, but is always duller than normal. The pupil may be contracted or dilated, but is always uneven at the margin according to the degree of congestion of the different portions. It may be quite immovable under the stimulus of light and darkness, and is always sluggish as compared with the healthy condition. To test this reflex action, the one eye may be bandaged, and the other eye covered with the palm of the hand for one or two minutes. When exposed the pupil will be found to be widely dilated, and in the healthy eye it will rapidly contract and dilate alternately until it has reached a condition of adaptation to the intensity of the light when it will remain immovable. With the inflamed iris these contractions and dilatations will be lacking altogether, or they will be sluggish and imperfect in various degrees according to the intensity of the inflammation, the degree of congestion or the tension of the liquid media of the eye. Restricted movement may also be due to adhesion to the cornea, (*synechia anterior*) or to the capsule of the lens (*synechia posterior*).

When viewed with the ophthalmoscope properly focused the choroid may show a lack of its normal lustre and an unevenness due to the formation of small rounded elevations in connection with congestion, or exudation, and patches of yellowish red or whitish discoloration together with lines of the same color following the course of the blood-vessels. It may also reveal dark spots of opacity in the lens (*cataract*) or clouds in the anterior region of the vitreous, the result of exudations. The blood-vessels may appear enlarged and tortuous.

In some cases the exudate may form a false membrane which completely closes the pupil.

A special tenderness around the margin of the cornea is suggestive of *cyclitis*. Internal ophthalmia is usually accompanied by a variable amount of fever.

Diagnosis. From simple keratitis, it is distinguished by the thickening, discoloration and sluggishness of the iris, by the absence, in many cases, of corneal opacity, and of free lachrymation, and in some instances by increased tension of the eyeball.

Recurring ophthalmia, which is usually also an internal inflammation, appears more abruptly and often at first with greater severity, and accompanied by more hyperthermia. There is almost always a bluish white opacity around the margin of the cornea, the eye is retracted in its sheath so as to appear smaller, and the upper lid usually shows a marked angle between its inner and middle thirds in place of the evenly curved arch of the healthy palpebra. It usually appears for the first time in the young and in those that have inherited the susceptibility and have been kept on damp soils, in cloudy districts, or dark buildings.

Lesions. These are necessarily varied according as the inflammation is concentrated on particular parts of the interior of the eye. The secreting membrane of the aqueous humor is nearly always inflamed giving rise to an exudate and a milky opacity of the aqueous humor. The iris is the seat of congestion exudation, thickening, cell proliferation and investment by false membranes. The capsule of the lens is early clouded, may be covered by exudate and is rendered vascular in some cases. The choroid is also the seat of congestion, exudation and discoloration with the covering up at points of its pigmentary layer. The vitreous

and lens finally become the seat of exudation and opacity which is liable to prove permanent.

Prognosis. The internal ophthalmias are always to be dreaded. In other organs exudates may take place and become organized as permanent structures without abolishing the function or rendering the organ physiologically useless, but in the delicate and transparent tissues of the eye, any such permanent product almost infallibly causes opacity and loss, or serious impairment of vision. In the retina the displacement, derangement, or covering up of the cones and rods necessarily interferes with or abolishes sight, the opacity of the cornea, lens, capsule, or vitreous interrupts the rays of light, and the destruction, or coating over of the pigment of the choroid leads to undue reflection and destroys vision. Beside this the destruction or impairment of one part of the eye, changes the refraction and blurs the vision, or interferes with accommodation and destroys the utility of the organ. Unless therefore the disease can be cut short in its early stages and a complete resolution effected it is likely to leave the patient very much deteriorated in value. Fortunately it is only in the most violent cases or in very susceptible animals that the disease in the one eye is transmitted to the other by sympathy and leads to destruction of that eye as well.

In the *treatment* of internal ophthalmia, rest in pure air and moderate warmth, away from a fierce glare of light is imperative. The causes should as far as possible be removed. Next, it is desirable to establish derivation. Leblanc and Trasbot attach great importance to phlebotomy from the jugular on the same side. A more direct local action with less loss of blood may be obtained from opening the angular vein of the eye or applying a leech beneath the lower lid. In most cases a sufficient derivative action can be secured by an active purgative which may be followed by daily doses of cooling diuretics. Locally astringent lotions (lead acetate or zinc sulphate 1 dr. to 1 qt. water; mercuric chloride, 1 : 5000; boric acid, 2 : 100; pyoktannin, 1 : 1000) in combination with cocaine hydrochlorate, homatropin, atropia sulphate, duboisia or hyoscyamin (1 : 1000) would be appropriate. These may be applied over the eye on a soft cloth, and in cases of infective inflammation the more antiseptic agents may be injected under the lids. When the inflammation is very severe the

atropia or other sedative agent may be made of the strength of 1 : 100 and a drop or two placed inside the lids with a dropper every two or three hours.

A blister of biniodide of mercury may be applied to a space the size of a dollar above the anterior end of the zygomatic ridge, or in dogs back of the ear on the side of the neck : or a seton may be passed through the skin in the same situation.

When the eyeball is unduly tense, puncture through the margin of the cornea with a fine aseptic lancet will relieve the tension and in some cases induce a more healthy action. Assiduous antisepsis is needful until the wound has healed.

In other cases benefit can be obtained from the use of an ointment of yellow oxide of mercury 1 part, in vaseline 10 parts, or of iodoform of the same strength. A small portion the size of a grain of wheat is put under the lid, and the latter manipulated with the finger to bring it in contact with all parts of the surface. In case of a rheumatic origin salicin and salicylate of soda are demanded.

SIMPLE IRITIS.

Causes. Symptoms : redness of sclera, in dogs, cats, birds, pigs, with a narrow zone of white next the cornea, red scleral vessels immovable, iris dull gray or brown, uneven, sluggish in response to light, synechia anterior or posterior, lens and capsule clouded or clear, pupillary margin uneven, myosis or midriasis, black cataract. Treatment : rest, dark stall or covering, head elevated, midriatics, cocaine, antiseptic puncture, purgation, leeches, seton, cooling astringent lotions, diuretics, for tension in convalescence iridectomy. In traumatic cases careful antisepsis.

This may come from any one or more of the *causes* of internal ophthalmia above named. The inflammation, however, concentrates itself on the iris so as to overshadow the disease in the adjacent organs.

The more distinctive *symptoms* are the redness of the sclerotic in unpigmented organs (swine, birds, dogs, cats), the redness increasing as it approaches the margin of the cornea but leaving a narrow white zone surrounding the edge. The red vessels on the sclerotic are not moved with the conjunctiva when the lid is moved over the front of the eye. The front of the iris is dull,

grayish or brownish, it is thickened unevenly and very sluggish in response to light and darkness. Not infrequently it is adherent to the back of the cornea (synechia anterior) or to the front of the lenticular capsule (synechia posterior). The lens and its capsule may or may not be clouded, but if the interior of the vitreous can be seen it is found to be clear. The pupil is more or less uneven in outline and sometimes it is torn at its inner edge so as to form shreds and projecting tongues. Myosis (contraction of the pupil) or midriasis (dilatation) may be present. If the latter has been preceded by adhesion a portion of the uvea may remain attached to the lenticular capsule constituting **black cataract**. The lens or its capsule may become opaque, and a fibrinous membrane may form over the pupil.

Treatment. Rest for body and eye are essential. A dark stall, or a thick covering for the eye is desirable. The head should be kept moderately elevated to facilitate the return of blood. The pupil should be kept widely dilated to prevent adhesions to the lens. Sulphate of atropia 5 grs. to the oz. of water should be applied a few drops at a time, thrice a day, or as often as may be necessary to secure dilatation. In case the atropia fails to secure dilatation a 5 per cent. solution of cocaine should be dropped into the eye every three or four minutes for four or five times and then another application of atropia may be tried warm. Should it still fail and should there be indications of extra congestion and swelling of the iris or of excessive tension of the eyeball, relief may be obtained by puncturing the cornea. With the reduction of the tension the iris will often respond to the midriatic. Benefit may also be obtained from an active purgative, or the application of leeches in the vicinity of the eye.

Cooling astringent applications may be kept up over the eye, or warm antiseptic applications will often give great relief.

In obstinate cases the yellow oxide of mercury ointment may be applied as advised for *internal ophthalmia*.

Cooling diuretics may also be of essential advantage.

If, after a fair recovery the bulb remains unduly tense, iridectomy may be resorted to as a prophylactic measure for the future. An incision is made with a lancet close in front of the margin of the cornea, and the iris seized and withdrawn with a pair of fine forceps, and a portion snipped off with fine scissors. The eye

and instruments must be rendered absolutely aseptic by carbolic acid and boiling water, and the antisepsis of the eye must be carefully maintained until the wound is healed. This tends to relieve congestion in the iris and to moderate the secretion in the anterior chamber, so that the former extreme tension does not recur. In making choice of the seat of the iridectomy a selection may be made which will do away with adhesions, or one that will expose a portion of the lens which is still transparent, and which may restore vision when obscured by a cataract.

In traumatic cases there should be extra care in maintaining a thorough antisepsis of the eye as the great danger is that of infective panophthalmitis. The injection of antiseptic liquids under the eyelids, and the covering of the eye with antiseptic cotton wool or with a soft rag wet with an antiseptic lotion are important factors in treatment.

SYMPTOMATIC OR METASTATIC IRITIS.

Complications of infectious diseases, influenza, contagious pneumonia, strangles, tuberculosis, omphalitis. Symptoms: exudation of fibrine and blood, with those of simple iritis. Treatment: as in iritis, plus measures for the specific primary disease. When second eye is threatened enucleation.

Under this head Möller describes those forms of iritis which occur as complications of various infectious diseases. It has long been observed that iritis and other ophthalmias, occurred as complications of the acute infectious diseases of the respiratory organs of the horse formerly known under the general name of "influenza." More recently many veterinarians and others have classed these influenza irites separately under the name of "pink-eye." The same can be said of "contagious pneumonia" (brustsenche) of horses which is distinctly caused by the *diplococcus (streptococcus) pneumoniæ equina*. Attention was called to the iritic complication of this disease in 1881 by Siedamgrotzky and it has been often noticed since. Conjunctivitis is however a more frequent complication of this disease than iritis. In both influenza and contagious pneumonia the iritis often supervenes

when convalescence has apparently set in. Strangles is another affection in which the iris occasionally suffers. Matthieu has described tuberculosis of the iris in cattle, and Möller mentions with some hesitancy cases of iritis which complicated the infection of the navel in new-born animals.

The *symptoms* of symptomatic iritis vary according to the particular infection. In addition to the fibrinous exudate the infections of the respiratory organs are liable to be complicated by blood extravasations. In influenza this may show as deep blotches on the bulbar conjunctiva and in chemosis. In contagious pneumonia Shütz met with iritis of a distinctly hæmorrhagic character.

In Matthieu's cases of tubercle of the iris there was first a slight lachrymation, and soon the iris assumed a grayish tint, and became uneven and unduly approximated to the cornea though it failed to become adherent to it. The swellings of the iris increased and became of a grayish yellow color, and the pupil was usually contracted and varied little in size. Post mortem examination showed the presence of tubercles. The same condition has become familiar in connection with experimental inoculation in the eye. As in ordinary iritis adhesion to the capsule of the lens and cataract are common results.

Apart from the *treatment* of the specific primary disease this type of iritis demands the same treatment as other forms. Strong atropia lotions to prevent or break up adhesions and antiseptic astringents are especially indicated. When implication of the second eye is threatened it may be desirable to remove the first by enucleation. (See Panophthalmitis).

FOREIGN BODIES IN THE IRIS.

These are sometimes fine shot particularly in dogs, and splinters of iron and steel in other animals. Their presence can sometimes be made out by careful focal illumination. If septic they cause violent iritis and panophthalmia. If aseptic they may sometimes cause little trouble. If they can be exactly located, they should be removed at once before the aqueous humor and cornea become

clouded. If the offending body is a piece of iron or steel and can be reached by a magnet introduced through the original wound or through one made with a lancet in the edge of the cornea it may be extracted by this means. If it is shot or other body that is not attracted by a magnet the portion of the iris in which it is entangled may be drawn out with forceps and snipped off with fine scissors. Due antiseptic precaution must be exercised.

COLOBOMA IRIDIS. CONGENITAL APERTURE IN IRIS.

This is a congenital defect in which there is an aperture in the iris. Hering figures the two eyes of a horse in which these appeared in the direction of the outer canthus. Renner records a case in a foal in connection with intra-bulbar enchondroma. Dochtermann and Berlin record that among 64 pigs the result of breeding a boar on his daughters and grand-daughters no less than 36 showed coloboma. Möller figures a dog with the same affection.

The condition is not known to prove hurtful to the affected animal so that it may be wisely let alone.

DOUBLE PUPIL.

Mayer notes a case of congenital double pupil in the horse, a bridge extending across the space from the upper to the lower border and cutting off the outer third of the opening. The present writer has seen a similar condition as the result of union of the corpus nigrum in severe iritis. Section of the bridge is possible, though rarely desirable, seeing that it opens a door to possible infection.

ALBINISM. WATCH-EYE.

The albino is an animal in which there is a complete absence of pigment in the eye. It is usually seen in white races of rats,

rabbits and dogs, and both the iris and choroid reflect a pink tint. It may cause photophobia and some weakness of vision but, in the main, it seems to be harmless to the lower animals. In horses it is occasionally seen as a partial defect, a portion only of the iris and adjacent sclerotic appearing of a brilliant white color. It does not usually seem to impair the vision, so that at the worst, it is only looked on as a blemish. It is needless to attempt a remedy.

PERSISTENT PUPILLARY MEMBRANE.

The persistence of this embryonic membrane has been noticed in the horse (Schindelka), ox (Meyer), rabbit (Mayerhausen), and dog (Möller). It tends to disappear with the growth of the animal and rarely does any perceptible harm.

OCCLUDED PUPIL.

This has been frequently found in horses as a sequel of iritis, and permanent adhesion of the contracted iris to the front of the lens capsule. It is in short, a posterior synechia with closure of the pupil. The lens and its capsule are usually opaque so that there would be no gain in detachment of the iris. If, however, there is reason to conclude that any part of the lens is still transparent, the performance of iridectomy over this portion, would produce a new aperture for the entrance of light.

CYCLITIS.

This is described by Möller as occurring in the domestic animals, but he fails to furnish instances of its diagnosis during life, and it is not likely to be often recognized in the living animal. Beside the usual signs of iritis, there is extreme tenderness to pressure around the anterior border of the sclera, increase

of intra-ocular pressure, followed later by its diminution, and a cloudiness of the anterior portion of the vitreous humor. This last condition can only be detected by a full illumination of the vitreous, and its examination with the ophthalmoscope. It is quite liable to be complicated by suppuration and to go on to panophthalmitis.

The *treatment* does not materially differ from that of iritis, yet atropia must be used with caution as it is liable to increase the suffering. The preparations of mercury have been especially recommended.

CYSTS OF THE IRIS AND CORPORA NIGRA.

Mayer speaks of these lesions in horses, but it is very difficult to diagnose them correctly, even with the aid of the opthalmoscope. The very manifest bulging at the part may be due to excess of pigment, especially in the corpora nigra, and an exploratory puncture would only be warranted when the protrusion became excessive and injurious. One such puncture by Eversbusch led to infection and loss of the eye.

TUBERCULOSIS OF THE IRIS.

This has occurred as the result of inoculation of the aqueous humor in the smaller animals, and as a spontaneous localization of the disease in cattle (Hess, Röder, Fischöder, etc.). In Hess's case, the left eye was shrunken to half the size of the sound right eye, and small caseated tubercles were present in both iris and choroid. There are usually coincident tubercles in other organs, and these with the nodular appearance of the iris swellings, if visible in life, may assist in diagnosis. (See *Symptomatic Iritis*, and *Tuberculosis.*)

CHOROIDITIS.

Causes: as in iritis: traumatic and infective. Exudative. Suppurative. Symptoms: as in iritis: less change in iris and of flocculi in the aqueous humor; opacity of lens and vitreous. Lack lustre choroid under ophthalmoscope, uneven, detached. Suppurative form: early profuse weeping, bleeding, later suppuration, pus oozing from orifice; panophthalmitis. Treatment: as in iritis; atropia; cocaine; astringents; purgatives; diuretics.

Causes. These are largely the same as those of iritis and cyclitis. Blows, traumas, foreign bodies, sand, cinders, dust, lime, fierce light, reflection from snow, water, etc., chills, draughts, storms, irritant gases, and a number of specific diseases like influenza, contagious pneumonia, canine distemper, rheumatism, omphalitis, pyæmia, etc., may be named. It is a common lesion of recurrent ophthalmia in horses, and is not unknown in tuberculous cattle. It is usually more or less involved in iritis, as the iris is in choroiditis. The name given to the disease which involves both, will depend mainly on whether the inflammation predominates in the iris or choroid. Mayer divides it into exudative and suppurative, the latter being a common result of trauma, and likely to issue in panophthalmitis.

Symptoms, These are largely those of iritis. The congestion and redness of the sclerotic around the margin of the cornea, the fact that the enlarged vessels are firm in the sclera and not easily moved as in conjunctival congestion, and a certain partial blindness, without much change in the brilliancy of the iris, or opacity or flocculi of the aqueous humor, would suggest choroiditis. Later some opacity of the lens, or its capsule or of the vitreous humor would be equally significant.

The only certain manifestations would be such as are found on ophthalmoscopic examination. Swelling and unevenness of the inner surface of the choroid, and a loss of luster, a change of its dark surface to light colored spots and patches, (dull-red, yellowish red, grayish green) and of the tapetum lucidum to a dirty grayish green in solipeds. Areas of minute blood clot may also be seen. But these are rarely recognized or indeed skillfully sought during life, and it is mainly to necropsies that we owe most of our diagnosis of choroiditis in the lower animals.

In the *suppurative form* there is early profuse lachrymation more or less tinged with blood, and later oozing of pus from between the lids. The redness and swelling of the conjunctiva and lids are very prominent features, and if the lids can be separated the corneal or scleral orifice may be seen oozing pus. If visible at all the anterior chamber shows yellowish opaque contents, and the symptoms of panophthalmitis supervene.

Treatment of the exudative form is essentially the same as for iritis. Atropia lotions with or without cocaine, also astringents, which may be used warm, and generally purgatives, diuretics, local bleeding, cupping, and counter-irritants are in order. In obstinate cases ointment of the yellow iodide of mercury, and in cases of extra tension puncture of the cornea may be the means of relief. Iridectomy may be advantageous under careful antiseptic precautions. In case of extensive or general suppuration (*panophthalmitis*) enucleation of the eye may be the only resort, and may contribute to save the other eyeball. See Panophthalmitis).

DETACHMENT OF THE CHOROID.

The choroid is detached from the sclera by exudates, blood effusions, or blows with blunt articles. The lesion is especially common in recurrent ophthalmia, choroiditis, and cyclitis. The ophthalmoscope will show the detached portion as a rounded elevation on the otherwise smooth concave surface, with normal or diminished intraocular tension. A *tumor* of the choroid is usually associated with increase of tension. After inflammation has been subdued these cases may be left to rest and time, and will often recover through absorption of the exudate. **Rupture of the choroid** from violence is to be similarly dealt with.

RECURRENT OPHTHALMIA OF SOLIPEDS. PERIODIC OPHTHALMIA. MOONBLINDNESS.

Definition. Causes: wet impermeable soil, clay, river bottoms, deep valleys, inundations, enclosing forests, damp air, lack of sunshine, rank fodders, wet seasons, damp, cold, basement stables, heating constipating fodder (corn, buckwheat, wheat), dentition, training, age of domestication, sale, etc., spring, shedding coat, heredity, debility, ill health, worms, debilitating infectious diseases, over work, insufficient, indigestible food, local irritants. Microbes. Rheumatism. Parasitism. Symptoms: fever variable, lack of vigor, sudden attack, irritation, photophobia, lachrymation, closed lids, contracted pupils, retracted eye, redness, swelling of lids, conjunctiva, haw, sclera, slight corneal opacity, with vascularity, aqueous turbid, flocculent, iris of dull color, sluggish, pupil contracted, hypopion, posterior chamber yellowish green, intraocular tension, crisis seventh to tenth day, convalescence fifteenth day. Recurrence. Obscurity of vision. Eye between attack: blue zone around cornea, eye seems smaller, retracted, prominent haw, angle in upper lid, dull iris, tint lighter, contracted pupil, cataract, alert ears. Lesions: exudates back of cornea, narrowed anterior chamber, sizy aqueous, thickened iris, adhesions of lens capsule, or iris, torn iris, lens opaque, fibroid, calcareous, atrophied, black cataract, vitreous opaque, yellow, black, shrunken, choroid uneven, discolored, detached, retina with exudate, detached, posterior chamber contracted. Prevention: drainage, mature horses for damp lands, liming soil, get fodder from dry locality, don't breed in cloudy regions, good diet and regimen, avoid corn, wheat and buckwheat, Glauber salts, pure dry stable, exclude debilitating diseases and parasites, keep in hard muscular condition, change to dry locality, don't breed from blind stock, legislation. Treatment: remove causes, cure rheumatism or other morbid factor, darkness, antiphlogistics, laxative, diuretic, local bleeding, cupping, blister, seton, locally atropine, cocaine, pyoktannin, sublimate, lead, puncture, tonics, treat corneal opacities. Jurisprudence: return a newly bought horse in 30 days, (France) or more: extend the time if suspected.

Definition. This is an inflammatory affection of the interior of the eye, intimately related to certain constitutions, soils, climates, and systems of management, showing a strong tendency to recur again and again, and usually ending in blindness from cataract or other destructive lesion.

Causes. A wet, impermeable, swampy or undrained soil is a potent cause of this disease. Heavy clays, which absorb and retain moisture, river bottoms and deltas which are frequently

overflowed and constantly wet, hollow basins where no effective drainage has been secured, and the coasts of seas and lakes which scarcely rise above the level of the water and are submerged at intervals, are the especial homes of the affection. In time past the disease was very prevalent in the low districts of France (Reynal), Belgium, Alsace-Loraine (Znndel, Miltenberger,) Germany, Holland (Möller), the English fen country and above all the damp lands of Ireland. Lafosse mentions a whole family of horses in South Western France which were characterized by blindness. Reynal records the terrible devastation which it caused in former times in the government studs at Limousin and Pompadour. It also prevails on the low banks of the Guadalquivir near Seville (Hurtrel d'Arboval), around Ostend, Cassel and Frankfort (Hofgeismar). Wet soils surrounded by forests or hills, which hinder free circulation of air, are especially injurious (Reynal). At Schlestadt, Alsace, at the beginning of this century, Miltenberger found 75 per cent. of the horses of the environs affected, whereas after great drainage works and the removal of all stagnant water Zundel found in 1870 not more than 2 per cent. In many localities in England, Ireland, France, Belgium and Germany the disease has greatly diminished in connection with land drainage and improved methods of culture. Harmon tells how in different parts of Brittany, drainage supplemented by the free use of marl and lime on the soil has caused a striking decrease in the prevalence of the malady. In the department of Ain a ratio of 333 per 1000 was thus reduced to 100 per 1000 (Reynal.) On the contrary in the absence of such drying of the soil the previous high ratio of attacks was maintained. This has been notorious in the damp lands of Northern France and Belgium (Picardy, Artois, Flanders, where it often reaches 40 to 70 per cent. Reynal): Alsace-Loraine, Holland, Hanover, Mecklenberg, North and East Prussia, Lithuania, the low parts of Austria and Hungary and the Danubian Principalities—Moldavia and Walachia.

Reynal further shows that dealers are in the habit of taking young horses, which have so far escaped, or which have suffered but one moderate attack, away from the low damp soils of the low Pyrenees or of the Jura Valley in France to the dry elevated lands of Dauphiny, Provence and Lanquedoc in France, or to the mountainous regions of Catalonia in Spain in the well justified confidence that few of them will suffer a second attack.

As a direct test the French Government sent ten yearling foals from the affected depot at Limousin to the healthy depot at Tarbes, retaining an equal number at home as test animals: it also sent ten yearlings from Tarbes to Limousin, retaining an equal number at Tarbes as test cases. Then the twenty yearlings at Limousin were divided, five of the home bred and five drawn from Tarbes having been sent into a very low wet country at Lariviere, and the rest were sent to a high dry location at Maraval. The result was that but one of the ten yearlings sent from Limousin to Tarbes contracted the disease, while on the damp land at Lariviere one Limousin-bred and four Tarbes-bred colts suffered; and finally on the dry soil at Maraval not a single colt, from either Limousin or Tarbes was attacked.

The other conditions that usually attach to a low, damp soil are important factors. **Damp air and a cloudy, rainy climate** are potent accessory causes. Hence the great prevalence of the disease formerly in Ireland, on the west coast and in the fen country in England, in Belgium, in the Low Pyrenees, in the valleys of the Loire, Jura, Meuse, Moselle, the Guadalquivir, etc., (Reynal). Such an atmosphere relaxes the system, induces a heavy lymphatic temperament, with coarse bones and muscles, an excess of connective tissue, thick hide and hair, and thick, shaggy and often gummy legs. All this implies a low tone of health which will less effectually withstand inimical influences.

The **rank, aqueous fodders** grown on such damp localities have a similar effect. These are more bulky and less nutritive and fail to maintain the highest tone and vigor. The animals must overload the stomach and intestines in order to obtain the requisite amount of nutriment, so that with a large, pendent belly they are still in poor condition. The case is even aggravated when they go on the succulent grasses of early spring, as they continue to gorge and may even make fat, but they lack in muscle and tone and in this condition even the rapid formation of blood seems to favor the attack. Möller records the great prevalence of the disease in Central Germany in 1884, in connection with excessive rainfall, inundations, and spoiled fodder.

Dard records that a low, overflowed meadow in the Soane bottom near Chalons, caused blindness in nearly all horses put upon it.

Bouin quotes a case of a sewage irrigated meadow in Vendée which almost infallibly produced recurrent ophthalmia in the horses fed on its products. On the other hand, oats and hay from a dry, rich soil and well harvested, are the most trustworthy food when the disease is to be dreaded.

Damp night air on wet, impermeable soils is to be especially feared as not only relaxing the system and lowering its power of resistance, but also producing a chill and thereby increasing the susceptibility.

Damp, cold basement stables are concurrent causes acting like the wet soils, the damp air, and the attendant relaxing conditions. Thierry long ago noticed that improved stable hygiene around Strassburg led to a notable decrease of recurrent ophthalmia. In 1807 horses were blind to the extent of 16.5 per cent., in 1821 to 8.5 per cent., and in 1870 Zundel found but 1.79 per cent., and few of the latter from recurrent ophthalmia.

Again the ventilation, lighting and drying of close, low, dark damp stables where the disease had previously prevailed has often practically banished the affection. Hofgeismar states that during the prevalence of the disease in a detachment of the German army in Alsace, out of 84 attacks in 700 horses 52 appeared in February and March, the period of severe weather and close stabling.

Fodders of a heating, or starchy nature, like **Indian corn, buckwheat**, or **wheat** strongly predispose to the disease, probably by inducing costiveness, slight impactions and indigestions, and in bad, frequently recurring cases the writer rarely fails to find that the animal is being fed on corn. Beans, peas, vetches and other leguminous fodders are less injurious and when grown on high, dry soils and fed judiciously they are as a rule harmless. When grown rank, badly harvested, and used to excess they become distinctly hurtful. Any fodder which has been allowed to become musty is to be avoided, since any condition which lowers the general tone of health strongly predisposes to an attack. The same remark applies to irregularities and faults in feeding. The best food and the most abundant supply will fail under such circumstances to maintain the condition, and the horse that is losing condition is becoming increasingly susceptible to this malady.

The period of **dentition** and **training** is the most common occasion for the attack, partly because this is the time when the colt is taken from the pure air and exercise of the field, into the hot, close, impure atmosphere of the stable, partly because he is made to exchange the cooling succulent grass for the stimulating ration of hay and grain, partly because he is subjected to severe constraint and much excitement in the hands of the trainer, and partly because of the irritation of the gums, the jaws and the whole head in connection with the shedding and eruption of the teeth. When injudicious bitting is resorted to, to give a special curve to the neck, the consequent obstruction of the jugulars and capillary congestion in the head is another potent cause. Heavy draught and overdriving have a similar effect. In 53 cases Schmidt found that 3 occurred under one year old, 5 in the 2d year, 10 in the 3d year, 16 in the 4th, 15 in the 5th, and 4 from the 5th to the 9th years.

Influence of Season. Mayer gives a statement of the percentages seen in the various months of the year as follows: January 4.9%, February 4.7%, March 10.1%, April 15.1%, May 13.4%, June 9.9%, July 11.9%, August 6.4%, September 4.9%, October 6.2%, November 6.7%, December 5.2%. The high rate in March, April and May may have a significance in connection with the debility connected with shedding the coat, and the resumption of more active work, when somewhat out of condition, it must also be noted that this is in Germany the season of the most active trade in horses and consequent change of ownership, stabling, feeding, work and management.

Heredity must be accepted as one of the most potent accessory causes. The lymphatic constitution is of course transmitted and with it the special susceptibility. This is notorious in the case of both parents, and is of course more potent if both sire and dam were predisposed, and have themselves suffered. In the latter case the heredity of the lymphatic temperament, and of the impaired organ of vision combine to render transmission more certain. A mare may have born a number of sound foals before suffering from the malady and then have offspring that do contract the disease. So with the stallion. Möller quotes the case of the eastern horse Turk-Mayn-Atty which served for a length of time in Prussian Studs and left a great stock of blind progeny.

Lafosse records the existence of a family of horses in Southern France all of which went blind. The same cause has greatly extended the disease among fast American horses in which the great strain of the track served to intensify the tendency. The writer has seen a colt which was born blind, by a blind dam and got by a sire with diseased eyes, but still held at $300 for service. Mangin, Marimpocy and Hamon record congenital cases from parents with affected eyes.

This hereditary susceptibility is so strong and pernicious that intelligent horsemen everywhere refuse to breed from either horse or mare that has once suffered from recurrent ophthalmia, and at the Government studs in France not only is every unsound stallion rejected but the service of the healthy stallion is refused to any mare which has suffered from disease of the eyes. A consideration for the future of our horses would demand that no stallion shall stand for the public service of mares unless he has been examined and licensed as a sound animal.

Yet, as already stated, heredity is not the one controlling factor, since the offspring of the victims of this disease will often escape when brought up in a high, dry locality.

Reynal records the appearance of the disease in **alternate generations**, the stallion offspring of blind parents remaining sound himself but producing foals which became victims of the disease in large numbers. A partial explanation may be found in the better conditions under which the stud horse was kept, while under less favorable surroundings his offspring developed the disease.

It must be added that every condition which induces **debility** or **ill health** is favorable to the development of the malady. The presence of **worms in the bowels** is a familiar example. Any debilitating disease like **strangles, influenza, contagious pneumonia, indigestion,** etc., and **overwork** or **insufficient or indigestible food** act in the same manner.

Again, **local irritants** may rouse the latent tendency. Street or stable dust, sand, hay seeds, chaff, blows on the eye with whip or other object, wounds, irritant gases, smoke, fierce light, cold draughts, storms, and whatever determines inflammation of the eye may be the occasion of an outbreak of recurrent ophthalmia.

Micro-biology. It will be recognized that none of these causes fully account for the specific and recurrent nature of this affection, and it is felt that something more is wanted to furnish a full and satisfactory explanation of the malady. This explanation is sought in a direct *infection*, but in spite of extended investigations by many observers no specific microbe has been demonstrated as uniformly present in all cases.

Potapenke found in the blood of the affected horses a plasmodium like that of ague. This agrees with the damp regions in which the malady prevails and no less with its intermittent or periodic character.

Vigezzi found in the aqueous and tissues a micrococcus (ophthalmo-coccus) which, cultivated on gelatin agar and inoculated in the anterior chamber or under the conjunctiva, produced an affection which he recognized as recurrent ophthalmia.

Trinchera found in the aqueous of the affected animals a bacillus and cocci. Drawing this aqueous into a sterilized syringe and injecting it into the anterior chamber of sound horses produced in 12 to 48 hours characteristic periodic ophthalmia. This was repeated by Schütz and Schwartznecker.

Robert Koch found in the affected aqueous, cocci, singly or in chains and bacilli with rounded ends. Injection of the latter into a sound horse's eye led to characteristic inflammation and loss of vision. In the cornea of the rabbit it had no effect.

Richter found in the eyes of a foal born with recurrent ophthalmia, of sound parents, diplococci and triplococci.

These observations do not demonstrate the constant presence of one definite microbe, nor that the disease is invariably due to any one particular organism, yet they may be held as strongly suggestive that any one of a variety of microörganisms may prove an exciting etiological factor in a susceptible system, or that, along with the organisms heretofore demonstrated, there exists an essential microbian cause which has up to the present eluded detection.

Other points which give circumstantial support to the microbian theory may be shortly stated:

1. The recrudescence of the disease after its various intermissions, and its preference for low, damp, cloudy localities seem to ally it to the malarial diseases of man.

2. Its appearance in certain predisposed systems, whenever an injury or debility of the eye seems to open the way for the admission of the hypothetical microbe.

3. The increased susceptibility to the malady when the system has been debilitated by disease, overwork, heating food, bad hygiene, or parasitisms, which have undermined the native power of resistance.

4. The prevalence of the affection in given localities has been supposed to imply the preservation and perhaps the multiplication of the germ in such places.

5. The increasing number of victims, year by year, when the same regiment or stud has been kept for a number of years in the same place. The theory is that with the presence of infected horses the hypothetical microbes become increasingly prevalent in the locality and above all in the stables, until even the more resistent subjects tend to succumb under the repeated infections. Thus Zundel says that in 1878, 700 army horses were stationed in Saarsburg, in 1879, 6 were attacked with recurrent ophthalmia, and in 1880, 84, Hofgeismar mentions that a dragoon regiment in Frankfort had 5 horses attacked in 1876, 12 in 1877, 11 in 1878, 14 in 1879, and 42 in 1880. We have, however, no assurance that the excessive rainfall, spoiled food or other unhygienic condition, may not have been a potent factor in the increase.

6. The obvious connection of certain cases of recurrent ophthalmia with a rheumatic condition suggests the probable operation of the same microbian cause.

Up to the present the microbian causation of this malady cannot be taken as proved, yet as a hypothesis it explains satisfactorily many of the observed morbid phenomena. That there is no such rapidly spreading infection, as would warrant us in listing this with animal plagues, is conceded, and that constitutional conditions have a potent influence is allowed, but that, in addition to these, microbian invasion is often a means of precipitating the malady is altogether probable. It may not be necessary that the microbe should in every case be of the same kind, yet the addition of a germ as the last item in the chain of causes is presumptively true.

Parasitism. Willach claims that many cases are directly due to parasites in the eye. In 19 affected eyes he found one young

filaria, a number of rhabditis (?), 1 cysticercus, and a number of distomata. Leider also found round worms in such eyes. Mayer and Dexler examined a number of cases, using the centrifuge on the liquids of the eye, without in any case finding such parasites. It may be assumed that the presence of embryo worms may rouse a latent predisposition into activity, but they cannot be adduced as active causes in the vast majority of cases.

Symptoms. These vary with the severity of the attack. In some cases there is high fever while in others this may be absent, yet a lack of vigor and energy bespeaks a general constitutional disturbance. The attack is sudden with marked local irritation, photophobia and lachrymation. The eyelids are closed some times so firmly as to suggest blepharospasm, and if opened the pupil is seen to be contracted. The affected eye is retracted and appears smaller, the conjunctiva is the seat of diffuse redness and swelling, and there is a bright red peri-corneal injection, occupying the anterior portion of the sclera. The outer zone of the cornea is already the seat of a bluish white opacity, the surface appearing dull and as it were smeared with oil. The centre of the cornea may be opalescent but not so obscure as to prevent examination of the interior of the eye. In a few days the outer margin of the cornea may show vascularity, and the aqueous humor a certain degree of turbidity. The iris if still visible is seen to be swollen and rigid, and to have parted with some of its lustre, assuming a grayish or lighter color owing to congestion and exudation. The pupil is usually contracted and dilates only sluggishly and imperfectly in darkness or under the action of atropia. The iris arches forward more than is normal and may even approximate and adhere to the back of the cornea. Bayer noticed that in a partial albino (watch eye) the iris becomes sulphur yellow. The anterior chamber of the aqueous humor usually shows a grayish yellow sediment which in severe cases may fill one-third or even one-half of its depth. This may be grayish white flocculi of lymph only, or it may be colored with blood or in suppurative cases by pus. In the first day of its appearance this may be diffused through the humor, but from the fifth to the seventh day it precipitates and leaves the iris and pupil open to inspection. The pupil if not already open, may be partially dilated with atropia and then discloses the interior of the

eye of a dark green, or sometimes with much exudate on the choroid, of a more yellowish green. This greenish discoloration appears to depend on opacity of the vitreous, on an exudate between the choroid and retina or on some opacity of the cornea and aqueous. At the same time under a good light some opacity of the lens or its capsule may be detected, or, with direct illumination, of the vitreous as well.

The tension and hardness of the bulb is materially increased in some cases but not at all perceptibly in others.

From the seventh to the tenth day the acute inflammation subsides, the lids and pupils dilate, and the deposit in the anterior chamber is rapidly reabsorbed. It may first assume a dull brownish green or brownish tint. Meanwhile the opacity of the cornea commences to clear up, and any redness or congestion of its margin to diminish or disappear.

With this disappearance of opacities in the cornea, lens and humors, all the symptoms of congestion subside and by the tenth or fifteenth day from the commencement of the attack the eye may have become approximated to its normal condition.

The characteristic of the disease, however, is its tendency to return again and again until the eye is destroyed. From five to seven attacks usually result in blindness, and then the second eye is likely to have a similar experience until both are useless. In some instances the eye which is first attacked may recover and remain well, while the second to suffer is rapidly ruined by a succession of severe attacks. The intervals between the attacks may be thirty, forty or sixty days and upward according to the state of the health, the condition, the food, the regimen, the exposure, and perhaps of other accessory causes.

Reynal claims that some eyes which have retained their normal function after one or two attacks will sometimes gradually lose the power of vision without any new appearance of inflammation. In other cases an eye which has been clear and transparent becomes suddenly filled up with an inflammatory exudation in the anterior chamber which obscures the iris and lens and in a few days vision is permanently lost, yet without conjunctivitis or apparent suffering.

Condition of the eye between attacks. After one, two or more attacks the eye is not restored to its former condition in the

intervals, but continues to exhibit morbid phenomena which betray the previous existence of the disease. The recognition of such persisting lesions is all the more easy that one eye only is usually attacked at first and a comparison between this and the sound eye renders the modifications all the more patent. Even after a first attack there is usually a hazy bluish white zone round the outer margin of the cornea and this becomes more distinct after each successive attack. The faulty eye is distinctly smaller in appearance, at first because it is retracted in its sheath and later in certain cases because of actual atrophy. In proportion to the retraction of the bulb, is the protrusion of the membrana nictitans which covers a greater part of that eye than of its fellow. The upper eyelid in place of forming a continuous and regular arch shows a distinct abrupt bend between its inner and middle thirds caused by the contraction of the levator muscle. The front of the iris has lost something of its normal lustre, and the posterior chamber is liable to show an abnormally light reflection, greenish yellow or yellowish blue. Under direct illumination, lines of opacity may be detected in the aqueous humor, or in the lens, or dark filaments in the vitreous. After several attacks the lens is very distinctly obscure and this increases with each relapse to a white or yellowish white complete opacity. After the first or second attack the pupil may be distinctly contracted, while later in the disease, with advanced cataract it is usually widely dilated. Another feature is the erect, attentive carriage of the ear, to compensate for the waning vision.

Lesions. These are not often seen, as animals do not often die of this disease. Beside the superficial lesions of the conjunctiva and cornea which may be seen during life, exudates have been found on the posterior surface of the cornea, in some cases binding that to the iris. In advanced cases the greatly contracted anterior chamber may contain a little mucilaginous liquid strongly pigmented with debris from the iris, the whole mixed with shreds of exudation. The iris is thickened by congestion and by exudation on its surface and in its substance, and is displaced forward so as to diminish the size of the anterior chamber, and it may have contracted adhesions with the cornea (anterior synechia) or with the lens (posterior synechia). This leads to unevenness in the pupillary margin, where the iris is often torn into shreds.

The crystalline lens is usually opaque, and may have undergone various changes, fibrous, calcareous, or atrophic. The anterior surface of its capsule has often adherent masses of black pigment derived from the urea in previous adhesions.

The vitreous humor and hyaloid membrane are sometimes clear, but usually yellowish or blackish and reduced to one-half their normal bulk by accumulations under the retina. A dense exudate often exists on the lamina cribrosa. The choroid is very uneven showing irregular rounded elevations, and like the iris is the seat of active congestion, exudation and thickening. The exudate on its surface raises and detaches the retina and, as shown by Eversbusch, this may increase so that the retina from the two sides may come together in the centre of the eye, the vitreous having been absorbed and removed. Reynal records instances in which the exudate had become cretified, or as he claimed transformed into true bony tissue. Finally the optic nerve is atrophied, often in advanced cases to half its natural thickness.

Prevention. As treatment is somewhat unsatisfactory there is the greater reason to give attention to measures of prevention.

In view of the great evil of low, damp, overflowed lands, it is important to drain and improve such lands whenever possible, and when this cannot be done, to abandon the breeding of horses upon them, and to buy the animals necessary for agricultural purposes from high, dry, healthy localities and introduce them only after they have passed the age of five years at least.

The improvement of wet lands by liming, so as to lessen the amount of decomposing organic matter, and improve the character of the vegetation has proved very beneficial in different parts of England. The substitution for the products of marshy meadows and wet lands, of those of dry cultivated meadows and lands is important.

Misty cloudy regions in the vicinity of sheets of water, or cold mountain ranges cannot be made wholesome, but they can be abandoned for horse breeding, and devoted to more remunerative uses. Something may also be done by stabling the working horses at night.

An insufficient or debilitating diet, for example, in winter, should be carefully avoided, the more so if it is to be followed by a sudden access to rich grass in the spring.

All forms of spoiled food, damp, musty, or fermented fodders should be withheld, especially in the case of young and growing horses.

Indian corn, wheat and buckwheat should be carefully excluded from the grain ration or if used should be combined with 1 oz. sulphate of soda for each animal daily.

Damp, close, filthy and underground (basement) stables should be avoided, the building on the other hand should be placed on high, porous and well drained ground, and should be clean, moderately well lighted and well ventilated but without draughts. While such special stable hygiene is demanded for all it is doubly demanded for young horses under six years of age.

As every debilitating condition renders the already predisposed animal more open to attack, all causes of ill health should be guarded against, and especially for the young, and in the case of such as are inevitable, every effort should be made to curtail and lessen the evil influence. Food or water which contains the eggs and embryos of intestinal worms, must be avoided and parasites which have already invaded the system must be got rid of as far as possible. Care must be taken to exclude the various infectious diseases, and in case of their introduction, to adopt every measure to mitigate their violence and to prevent debility and weakness. Overwork and irregular feeding and watering must be guarded against.

At the same time moderate work or exercise daily which will develop the highest tone of the muscles, nervous system, digestion, assimilation and other functions is a measure that can never be neglected. Idleness with resulting fatness, softness and weakness of muscle, and lowering of the power of endurance is always an invitation to renewed attacks. Regular invigorating work is essential ; exhausting work is injurious.

Change of locality to a drier soil, clearer, drier atmosphere, and more abundant sunshine, when it can be availed of, is a most important preventive measure. Reynal who made an extended official inquiry into this matter found conclusive evidence of its truth. Young horses removed from the low affected regions of Cantal, Poitou, Brittany and Anjou rarely suffered another attack when taken to the high land of Catalonia, and those moved from the damp lands of Franche Compté, Bresse, Dauphiny,

Provence, Languedoc, Bassigny and Belgium to the dry, calcareous portions of Champaigne also escaped further trouble. In many such cases the eyes already slightly affected would materially and permanently improve.

Finally the influence of heredity is never to be over looked. The ideal system would be to have all stallions professionally examined, and licenses granted to such only as are free from this affection, and to place the owners of such horses under obligation to serve only, mares the eyes of which are sound. This might be enforced as a state or county ordinance. Serious difficulties it is true stand in the way of such a measure. The horse which has an extraordinary record on the track, and to the development of the ophthalmia of which, overwork has doubtless contributed, will be run after by breeders who seek speed at any cost, and it may be questioned whether the State has any right to interfere with the prospective profit which may be expected from the reproduction of the strain of blood. But aside from such fancy products as racers and trotters, this objection has much less force. For carriage, riding and road horses and for the draught and agricultural animal the advantage of sound eyes so greatly over balances all consideration of special values with imperfect eyes, that a statute which will prevent the propagation of such unsoundness is more than justified in every case. The importance of this will be admitted when it is considered that in the great majority of cases, the young animal is attacked after it leaves the hands of the breeder, and therefore a high price is secured for a subject which is almost certainly doomed to become blind in given surroundings. Such a law would work well in every locality. In the low, damp region where the disease prevails habitually, the unprofitable breeding would practically cease, unless a race could be secured which was proof against the infection. The value of such a race could hardly be over-estimated. On the high, dry lands, on the other hand, the natural tendency to immunity would be still further enhanced, as the most susceptible animals which contracted the disease, in even such a healthful district, could be in no sense fit for reproduction, and should therefore be doubly condemned.

In the case of the mare the proprietor is under strong temptation to ignore the sanitary measure under consideration. When

her eyes fail, her value in the public market is greatly depreciated, yet she can yearly produce a foal, which is finely developed and will bring a high price if sold young, before it has been attacked by the disease. Hence the great importance, at least in the case of all horses which are not intended for exclusive use on the race course, that a law shall be strictly enforced which will put an absolute limit to the breeding from horses that have been affected with recurrent ophthalmia.

Blows, dust, sand, smoke, irritant gases, fierce light, and all sources of irritation must be avoided as in other eye diseases.

Treatment. Radical treatment for the disease is far from generally satisfactory. Too often the affected animal is still in the environment which has tended strongly to its development, and it is impossible to secure a satisfactory change. As far as possible, however, every available sanitary measure mentioned under the head of prevention should be enjoined, and largely in proportion to the thoroughness of such measures, and the slightness and recentness of the attack will be the hope of a successful treatment.

In some instances in which there appears to be a **rheumatic** complication, the employment of anti-rheumatic agents have proved of essential value. Powdered colchicum corms may be given twice a day in doses of 1 scruple, combined with salicylate of soda, salicylic acid, or salicin in 2 drachm doses. To these may be added bicarbonate of soda or of potash in drachm doses.

In cases attended with marked fever, hyperthermia and anorexia, **antiphlogistic treatment** may be desirable, but with the primary consideration that it must not be materially depleting, nor calculated to induce debility or atony. A laxative dose of aloes will sometimes benefit, but should be avoided in the absence of manifest fever. Two or three ounces of Glauber salts, twice a day, will effect the same purpose with less danger. Or saltpeter or other cooling diuretic may be given daily. In most cases bitters may be added with advantage.

In severe cases, **rest** is essential until the violence of the inflammation shall have abated, and a **dark stall** or a cloth to obscure the light is equally important.

Trasbot advises bleeding from the jugular, but such a depleting measure finds little support in England or America. **Local**

bleeding from the angular vein of the eye or by leeching or cupping is not open to the same objection.

Counter-irritants are, however, more suitable. A stout silk thread may be inserted above the lower end of the zygomatic ridge and bathed and moved daily to prevent the lodgment of pus. Or a blister of cantharides or biniodide of mercury may be rubbed in on an area as large as a silver dollar in the same situation.

In all cases a strong solution of **atropia sulphate** (2 per cent). may be instilled into the eye once or twice daily. Or a mixture of atropia and cocaine (1 per cent. of each) will give even greater relief. If to these is added 1 per cent. of pyoktannin we get a collyrium which is at once anæsthetic, midriatic and **antiseptic**. This is often of material value. Vigezzi advises a mecuric chloride lotion (1 : 1000) as a collyrium, and for injection in the submucosa.

If the local inflammation runs high an **astringent lotion** may be applied externally on a soft rag hung over the eye and kept constantly wet. Sugar of lead or acetate of zinc may form the basis of such lotion with a little atropia or morphia added.

Puncture of the cornea and **iridectomy** have been strongly advocated on the ground that the disease is identical with glaucoma, but the burden of evidence is decidedly against their use as a regular method of treatment. In case of increased intraocular tension, however, the puncture of the cornea can be very profitably employed, but it should be reserved for such special cases. Theoretically, iridectomy should be advantageous in preventing a relapse, but experience has not fully sustained this. When employed, the most careful disinfection should be secured. Under rational treatment the attack subsides in ten days and the eye may appear to be restored to the normal condition in two weeks. This natural tendency to a temporary recovery has served to give a wide acceptance to the most irrational methods of treatment, which have not in any sense hastened the recovery.

As soon as active inflammation and hyperthermia subside, every attention should be given to prevent a relapse, and to this end all the measures mentioned under prevention and which can be applied to the individual case should be adopted. Among these, moderate exercise or regular work must never be omitted.

A course of **tonics** embracing preparations of iron and bitters, is equally essential, and may be begun as soon as fever and active inflammation subside.

Special lesions, like corneal opacities and ulcers, must be treated as in other affections.

Jurisprudence. The question of the right to return upon the seller a horse attacked with recurrent ophthalmia has been beset with difficulty, mainly because of the intermissions during which, to the ordinary observer, the eyes may appear sound. In France a period of thirty days is allowed in which to return such a horse after purchase. This is, however, too narrow a margin as the second attack may not appear until after two, four or six months. It does, however, provide for the return of the worst cases in which the recurrence is likely to take place at an early date. Another provision is that a suspected horse may be put in pound under veterinary observation for thirty days, in anticipation of a second attack, and if such fails to appear the purchaser is debarred from returning him.

In many cases the symptoms during an attack and between attacks are such as to identify the recurrent inflammation, and the expert can pronounce positively as to the nature of the malady. In other cases there is a degree of uncertainty, and the animal must either be returned on the general plea of diseased eyes, if they can be shown to have been faulty at the time of sale, or otherwise the horse must be put in the hands of a veterinarian and the seller notified of the action, until it can be shown whether it is the recurrent disease or not. If it can be shown that the disease is the recurrent affection the seller is responsible at common law for selling a diseased animal for a sound one. If on the other hand, it is a non-specific ophthalmia, it must be shown that it existed prior to sale, and that a warranty of soundness was given, in order to hold the seller responsible.

PANOPHTHALMITIS.

General suppurative inflammation of eye. Experimental cases. From traumas. Diagnosis; foul wound, violent eye inflammation, yellow purulent appearance, high fever, involves second eye. Treatment: antiseptic, enucleation.

This term has been applied to a general purulent inflammation of the eye resulting from infection with pus germs entering from without through traumatic injuries, or by reason of inflamed tissues, or on the other hand, reaching the eye as a general infection through the blood. It may begin therefore, as conjunctivitis, scleritis, or keratitis, and gradually extend to active infection of the iris, choroid, and ciliary body.

Möller produced an experimental case in a foal by the injection of the staphylo-coccus pyogenes aureus into the anterior chamber. In 24 hours there was violent inflammation: the eyelids were closed, the conjunctiva dark red, and a mass of glairy pus under the eyelid. The cornea was cloudy throughout, though still dimly transparent so that the accumulating pus in the anterior chamber could be seen. The iris was strongly dilated and the eyeball abnormally tense. The second day the bulb was visibly enlarged, the eyelids greatly swollen, the conjunctiva infiltrated so as to cause chemosis, and the cornea completely opaque. The infiltration of the orbit caused the eyeball to protrude from its sheath. A high fever set in and on the fifth day the foal died.

Cases in the lower animals are usually the result of direct infection through some traumatic lesion of the eye.

The special feature of the disease is the rapid and abundant production in all parts of the eye of pus cells until the whole organ has become a bag of pus.

The chief diagnostic symptoms are the presence of a foul wound, the rapid advance of the phlegmonous inflammation of the conjunctiva and lids, the yellowish opacity of the cornea, and, if visible, of the aqueous humor, the prominence of the entire eyeball, the high attendant fever and the early destruction of the

eye. In the domestic animals the sympathetic irritation of the second eye has not been observed so commonly as in man. If the patient survives, the pus makes its way slowly to the surface, and escapes, and the cavity granulates and heals with contraction of the eye into a small nodular mass.

The *treatment* of the condition is essentially antiseptic and should be made preventive if possible, as there is little hope of saving the eye if the suppurative inflammation has been already established. The wound should be treated at the earliest moment with antiseptic lotions, sublimate solution (1 : 5000) or potassium permanganate solution (1 : 100) or pyoktannin (1 : 1000), or creolin (1 : 100). When inflammation has actually set in, these should be used still more assiduously by frequent injection under the lids, or by inserting antiseptic cotton between these and the bulb.

Enucleation. When the eye has become a virtual abscess the quickest and most perfect relief is secured by the complete extirpation of the eyeball. The patient is narcotized by ether or chloroform, and a thread or hook being passed through the cornea, the globe is quickly dissected out by curved scissors. Bleeding may be checked by pressure with cotton wool steeped in tincture of the muriate of iron, and later the wound may be dressed with stupes wet with a mixture in equal parts of standard solution of sulphurous acid, glycerine and water.

GLAUCOMA.

Sea green pupil. Causes: intraocular pressure from serous choroiditis, deranged fifth nerve, increased blood pressure, inflammatory obstruction of sclero-corneal canal, irritation of the ciliary ganglion. Symptoms: excessive tension and firmness of the globe, anterior chamber shallow, iris contracted, sluggish, pupil grayish or yellowish green, cupping of optic disc, pulsations of retinal arteries. Acute inflammatory form, simple form, secondary form. Convexity of pupil with synechia. Traumas. Luxations. Atheromas. Lesions: inflammation of the iris, choroid, ciliary body or cornea, round cell infiltration, cupped optic disc, atrophy of optic nerve, hydrophthalmos. Treatment: massage, puncture of aqueous, iridectomy, eserine, cocaine, antiseptic bandage.

This has been so named from the sea-green color of the pupil. The nature of the disease has been much debated and up to the

present time ophthalmologists are far from being agreed as to its true pathology. All are agreed as to the essential feature of the malady, namely, increased tension of the eye ball, but every case of increased tension of the bulb is not recognized as glaucoma, and the true cause of the persistent and progressive increase of pressure in cases recognized as glaucoma is not absolutely settled.

Causes. The immediate cause of the condition is the increased intraocular pressure, on this all are agreed, but as to the cause of this pressure there is difference of opinion. Gräfe attributed it to a serous choroiditis: Donders to deranged innervation of the 5th cranial nerve which controls secretion; others to increased blood pressure; others to inflammatory contraction at the sclero-corneal border where the principal drainage canal of the aqueous humor lies. The increased blood pressure theory appears to be contradicted by the fact that exalted blood pressure, as in fever, does not tend to glaucoma. The arrest-of-drainage-of-the-aqueous theory seems to be contradicted by the reduction of the anterior chamber while the theory would demand its increase. Priestly-Smith injected the sheep's eye with a pressure of water of 30 centimetres high, but while he caused an increased outflow he failed to induce distinct glaucoma. Möller tied the ophthalmic vein of the horse, but he also failed to produce glaucoma. "By artificial stimulation of the ciliary ganglion in dogs, the internal tension of the eye can be noticeably and permanently increased, and we may therefore assume that when this ganglion is stimulated, the secretion of fluid is increased, and that glaucoma depends upon an analogous process" (Fick). It would seem that necessity demands at the same time an obstruction of the normal drainage through nervous influence or otherwise. Schœn ascribes glaucoma to overexercise of the accommodation, a cause which would hardly be expected to operate in dogs. As bearing on the nervous causation Fick mentions that in man glaucoma is often preceeded and accompanied by trigeminal neuralgia. Again the symptoms of glaucoma often appear in the course of recurrent ophthalmia in the horse.

While it seems impossible to ascribe the disease to any single definite cause there appears to be good reason to accept as factors in different cases, a derangement of trigeminal innervation, an irritation of the ciliary ganglion, and an inflammation

affecting the region of the ciliary circle and the sclero-corneal line.

Symptoms. The tension of the eye ball is excessive as ascertained by pressure of the finger, or by the spring tonometer. If the increase of tension has come on suddenly, the cornea is somewhat cloudy, and less sensitive to the touch. If one eye only is attacked the contrast between the two is very marked and diagnostic. The anterior chamber is diminished in size by the projection forward of the iris and lens. The iris is usually contracted so as to show a dilated pupil reflecting a smoky, grayish green or yellowish green hue. The iris is either irresponsive to light or responds very slowly and imperfectly. With the ophthalmoscope the most marked features are the "cupping" or depression of the optic disc by pressure, and the pulsations in the retinal arteries. These pulsations are especially easily seen at the margin of the depression which represents the seat of the lamina cribrosa and the point of entrance of the optic nerve. They are rendered even more manifest by pressure on the eye. They are due to the prompt emptying of the blood vessels by the intraocular pressure, so that these are only momentarily filled at each cardiac systole.

Ophthalmologists recognize three varieties of glaucoma: **acute inflammatory glaucoma**, **simple glaucoma** without apparent inflammation, and **secondary glaucoma**, the result of another disease.

Acute inflammatory glaucoma is the one condition in which, in the absence of a midriatic, inflammation is associated with dilated pupil. It is liable to occur in a series of attacks, which increase in severity, hence its supposed identity with recurrent ophthalmia in the horse. The entire group of symptoms have, however, been rarely or never seen in the horse. They are distinctly more common in dogs.

Simple glaucoma comes on more slowly, becomes chronic and is to be recognized by the physical symptoms in the absence of inflammation, notably by tension of the bulb, diminution of the anterior chamber and cupping of the optic disc.

Secondary glaucoma is the direct result of some other disease of the eye:

Complete posterior synechia acts by confining the liquid which is secreted, to the posterior chamber whence it finds no ready outlet through the pupil, and causes a marked bulging forward of the iris and tension of the eyeball.

Slighter anterior synechia in the form of cicatricial adhesions between iris and cornea in the sclero-corneal margin, acts by blocking the principal drainage canal of the aqueous humor, which lies in this angle.

Traumatic injuries implicating the capsule of the lens and admitting the aqueous humor freely to the lens substance determines softening, swelling and so much irritation as to increase the secretion largely and determine intra-ocular tension.

The same may result from luxation of the lens and irritation of the ciliary circle by dragging.

Other causes are disease (atheroma) of the retinal vessels and the growth of tumors in the interior of the eye.

Lesions. These are very varied. Inflammation of the iris, ciliary body and choroid and even of the cornea is not uncommon. The iris and ciliary body show round cell infiltration, as may also the choroid. In the ciliary body this is likely to be especially abundant along the drainage canal (canal of Schlemm) thereby reducing its calibre. Leber and Fuchs found drops of liquid in the epithelium and cornea. One of the most significant lesions in man is the cupping or depression of the lamina cribrosa of a depth in ratio with the force of the intra-ocular pressure, and inflammation or atrophy of the optic nerve, back of the eye. Möller, however, has never been able to find actual cupping of the optic papilla in animals, but instead thereof a general distension of the outer coats of the eye, a hydrophthalmos. In view of the fact that these coats have the same structure and nearly the same relative thickness as in man, this throws considerable doubt on the supposed identity of glaucoma in man and cases which have been considered such in the lower animals. The comparative absence of pulsations in the retinal arteries in animals adds to the uncertainty.

Treatment. Although such cases lack some of the diagnostic symptoms of glaucoma in man, yet they agree with that in the increase of the intra-ocular pressure, and demand similar measures for relief. Some reduction of the tension can be secured by

careful massage over the eyeball so as to favor the progress of the lymph out of the bulb. A prompt but rather transient relief can be obtained from evacuation of the aqueous humor by puncture with a lancet close to and parallel with the margin of the cornea. The most effective treatment is, however, by iridectomy. On account of the great power of the muscles in the lower animals it is usually desirable to anæsthetize the patient and then fixing the eyeball with a pair of rat-tooth forceps, an incision is made close in front of the upper border of the cornea, and the lancet slowly withdrawn. A pair of iridectomy forceps are then introduced and the iris seized and drawn out through the wound, and a portion snipped off with a pair of fine scissors. The iris is then pushed back into the anterior chamber, and a drop of eserin solution placed in the eye. The parts and instruments must be rendered thoroughly aseptic before the operation, and the eye cocainized both before and after. The eye should be kept covered for some time with a cloth wet with a solution of mercuric chloride (1 : 5000) or other antiseptic.

Appropriate treatment may be employed in case of co-existent inflammation, or to improve the general health.

EXOPHTHALMOS.

This consists in an increase of the media of the eye so as to cause an excessive increase in size, and an unsightly bulging outward from the orbit and between the lids. It may be said to be a more exaggerated enlargement of the eye ball, than has been already noticed under glaucoma.

It has been seen in nearly all classes of domestic animals. Everhardt and Möller reports it in horses, Hausmann, Pradal, etc., in cattle, LaNotte, in lambs, Cöster, Trasbot, etc., in dogs, and Trasbot, in birds (chickens and parrots). It has been found congenitally in lambs and at a few days old in foals, especially when weak and puny; in older animals it appears to be most frequent in the anæmic or starved animal, in the lymphatic, or, as in man, in the goitrous.

The manifest projection outward of the eye may occur as a nervous phenomenon, without intra-ocular pressure, and without abscess, neoplasm, or inflammation in the depth of the orbit. In a case of tuberculosis in a three year old cow, I have found this condition, with normal tension of the eyeball, but with acute tubercular meningitis of the pia, surrounding the pons and crura cerebri, a grayish exudate with lighter miliary centres, and a considerable clot of extravasated blood.

Symptoms. Mostly without any febrile reaction, there is a manifest enlargement and bulging of the globe of the eye, so that it stands out between the lids which can no longer cover it. The cornea, aqueous, lens, and vitreous are not usually opaque, but show only a pale, blue, opalescent tint. The pupil is often widely dilated so as to show clearly the interior of the eye. Vertiginous symptoms have been observed in the cow (Pradal), the supposed result of intra-ocular pressure.

Treatment has had little effect when it stops short of puncture of the cornea or iridectomy.

HYDROPHTHALMOS OR BUPHTHALMOS CONGENITUS.

This has been applied to a congenital enlargement of the eyes from internal distension in children. The cases in lambs and foals quoted in the last article were evidently of this nature. They are charged on intra-ocular pressure acting on the delicate tissues of the embryo or unborn animal. There is not necessarily cupping of the optic disc so that persistent tension after birth cannot be insisted on.

Cases occurring in older animals, may be forms of secondary glaucoma though classed under hydrophthalmos by Mayer and others.

Treatment when demanded is along the same lines as in glaucoma.

CATARACT. OPACITY OF THE LENS OR ITS CAPSULE.

Definition. Forms: lenticular, capsular, cortical, nuclear, polar, black, diabetic, traumatic, immature, mature, senile. Causes: impaired nutrition of lens, inflammation of iris, choroid, ciliary body, retina; recurrent ophthalmia. Proliferation of cells. Increased density, chemical changes, degenerations. Sugar, sodium chloride, naphthalin. Rachitis. Senile. Blood pigment. Symptoms: shrunken bulb, opalescent zone around cornea, angle on upper lid, shying, extra ear activity, high stepping, better sight in twilight, homatropia, examination facing the light, Purkinje's images, ophthalmoscopic examination. Prognosis hopeless. Treatment: phosphureted oil, massage, operation in horses, discission, under antiseptic precautions, extraction under careful antisepsis, suction.

Definition. Any pathological change in the lens or its capsule diminishing its transparency.

Varieties. The opacity may be situated either in the lens (**lenticular**) or in its capsule (**capsular**). Again, it may be in the outer part (**cortical**) or in the central part (**nuclear**) of the lens. If the opacity is on the capsule in front of the lens it is **anterior capsular**; if on the portion behind the lens it is **posterior capsular.** If the opacity is caused by black iris pigment adherent to the capsule it has been called **black cataract.** If the lenticular cataract is small and round, it is **polar**, and it may be **anterior** or **posterior polar** according as it is situated near the front or back of the lens. **Diabetic cataract** is one associated with mellituria. A **traumatic cataract** is one resulting from a wound of the lens which admits the aqueous humor and causes softening, swelling and finally solution of the substance of the lens. The **immature** or **unripe cataract** is one in which the lens is not yet wholly involved and indurated; the **mature** or **ripe**, when such consolidation has extended throughout. **Senile Cataract** is seen in old horses, dogs, cats, birds and very exceptionally in cows. This usually attacks both eyes at once. A degeneration takes place in the fibres of the lens, which are invaded by sclerosis beginning at the centre of the organ.

Causes. In domestic animals cataracts are commonly the result of impairment of the nutrition of the lens in connection with

inflammation of the iris, choroid, ciliary body, retina, or hyaloid membrane, and above all, in solipeds, in recurrent ophthalmia. It may be assumed that a transparent tissue composed of cells can only maintain its translucency so long as the most perfect equilibrium is maintained as regards the mutual relation of the cells, the pressure of its interstitial plasma, and the chemical composition of both plasma and cell structures. The slightest deviation in any direction will impair or abolish the transparency of the tissue. In inflammation this occurs in various ways, through the increased cell multiplication and the change in the nature of the cells, through the increased exudation and the alteration of the solid parts as regards compression and relative position, and through chemical changes in the exudate which contains more salts, fibrinogenous material, etc., than the normal plasma. The same is true of all the post inflammatory degenerative processes that take place in the lens.

The formation of cataract from chemical alteration in the fluids is familiar in diabetic subjects,—man or beast (Altenhof). It can be produced experimentally in frogs by injecting sugar, common salt or any other readily diffusible saline solution under the skin (Kunde). Rabbits that are fed naphthalin develop cataract which radiates in lines and streaks from the pole towards the periphery and in the cortical portion of the lens. Perhaps the lamellar cataract of rachitic patients is also to be attributed to the lack of earthy salts in the plasma of the lens.

Senile cataract may be hypothetically attributed to impaired nutrition, degeneration in the lens or its capsule, or less commonly to disease of the blood vessels of the eye, or gradual changes in the plasma. It occurs in horse, ox or dog at ten years old and upward.

Anterior Capsular Cataract sometimes results from the deposition of **blood pigment on the capsule** in cases of extravasation into the anterior chamber. This is closely allied to the black or spurious cataract which consists in the adhesion of the uveal pigment to the capsule, and its detachment from the iris.

Kunde who caused cataract in frogs by injection, subcutem or ingestion of concentrated solutions of sugar or salt, attributes the result to the sudden abstraction of water from the crystalline lens. Even the cell multiplication in inflammatory cases, he

holds to favor this, since the new cells having little vitality are especially subject to granular and other deposits and degenerations, with loss of water or of transparency.

Symptoms and diagnosis. The examiner should apprehend cataract after internal ophthalmia. Much more so, if there is apparent diminution of the bulb, an opalescent zone around the outer border of the cornea, or a marked angle in the curvature of the upper eyelid, as usually occurs in recurrent ophthalmia. When a horse suddenly acquires a habit of shying, of starting back or to one side when approached, when confronted with strange or unexpected objects, or with deep shadows like those from electric lights, he is to be suspected. When he carries his ears in an unusually alert manner, turning one forward and the other back, when he steps higher than before to avoid unseen objects, suspicion should attach to him. If he sees better in twilight than in the full sunlight, central cataract may be feared, while the periphery which is exposed by the dilatation of the pupil in semi-darkness is still clear. In all examinations for soundness, the greatest care should be taken to exclude the possibility of overlooking an existing cataract.

In the very early stages, while internal inflammation and photophobia are still present, the pupil may be contracted so that lesion can easily escape notice. Any contraction of the pupil therefore disproportionately to the light, should demand a careful examination with the pupil dilated in darkness or by the action of atropia or homatropin. In the more advanced cases with no persistent inflammation and an advanced opacity of the lens, sensitiveness to light is greatly lessened, the pupil is dilated and the cataract is easily detected.

In cases approximating to the condition last named it is usually only necessary to place the animal in a sombre or dark building, with his head facing the light at an open door, or window and best with full sunlight. Let this fall full upon the eye, and let the observer view the pupil diagonally from each side when any opacity may be detected.

When the pupil is too narrow, several drops of a one per cent. solution of atropia sulphate may be dropped within the lower lid and left for ten minutes until the pupil is widely dilated. Then the examination may be made as above, or still better

the animal may be taken into a dark chamber and examined by one of the following methods:

A light, preferably a candle, is placed in front of the eye and moved from side to side, upward and downward, so as to bring its images over all parts of the cornea and lens. In the normal eye there are reflected three images of the light, one large, clear, and upright from the anterior surface of the cornea, one, much smaller but still upright, from the anterior capsule of the lens, and one, small and inverted, from the posterior surface of the lens and capsule. Any opacity in the lens or on its posterior capsule, will cause the posterior (inverted) image to become indistinct, and as it were a diffuse white blur, as it passes over that spot. The other small (erect) image may be even clearer than normal in passing over the opaque area because of the mirror-like reflecting action of the white cloud behind it. The movement of the light so as to pass its image over all parts of its surface in succession will certainly reveal the existence and seat of the cataract, by the blurring of the inverted image of the flame.

Another method is by oblique illumination, the patient's head being turned away from the light and the interior of the eye being lighted up by reflection from a mirror. If the pupil has been sufficiently dilated all parts of the lens can be scrutinized in this way and the slightest opacity detected by the grayish or whitish haze.

If there is still doubt as to the nature of such appearances, it may be set at rest by illuminating the depth of the eye with the ophthalmoscope when the opacities will appear as dark areas in the general red ground. (See Systematic Examination of the Eye.)

The *prognosis* of cataract is almost invariably hopeless. I have seen newly formed opacities of the capsule clear up in a day or two, and such recovery in very slight traumatic injury and superficial exudation is recognized as possible, but a slowly forming cataract is usually there to stay. Those that clear are presumably only exudates on the capsule and not true cataracts.

Treatment. While exudates on the capsule may disappear under a course of purgatives and diuretics, practically nothing is to be expected from medical treatment in true cataract. The instillation of phosphorated oil (1 – 2 per cent.) daily into the con-

junctival sac as formerly recommended, may be helpful in some of the superficially opaque membranes, but for formed cataract it has proved useless. Massage with, or without ointments can temporarily lessen ocular tension and reduce the liquids in the zonula of Zinn, and canal of Schlemm, but it is only in very exceptional recent cases, in which it has given permanent benefit, and even these were probably spurious cataracts.

The question then is essentially whether we should operate or not. In the horse the objections to operation are almost conclusive in all cases. The eye in which the lens has been depressed or extracted can never see objects clearly without the aid of biconvex glasses, and it is impossible to fit these to the animal. The horse that is blind can go to pasture or be driven in harness with safety, but the one that sees all objects distorted or blurred is liable to become a shyer endangering the life of his rider or driver. The greater number of cataracts in horses come from recurrent ophthalmia and are associated with opacity of the vitreous, detachment of the retina, exudates in the choroid, degeneration of the optic nerve, or other lesion which of itself would destroy vision. Almost the only object of removal of the lens in such cases would be to make an unsound horse pass for a sound one. Even this is usually unattainable because the thickened capsule remains as a dense white cloud or the opacity of the vitreous shines through the pupil. In dogs the cataract is usually associated with fewer complications, and the resulting imperfect vision is not a source of danger to man. Extraction of the opaque lens may in this case appeal so strongly to the sense of comfort of the owner that the operation may become permissible or desirable.

In man the operation may have to be delayed for a considerable time because of the unripeness of the cataract. The center of the lens may be firm and opaque while the outer layers are so soft that they would be likely to be retained in the capsule and would not only produce persistent opacity, but would be a continual threat of destruction of the eye by active inflammation. The ripeness is ascertained by careful scrutiny of the shadow of the iris during illumination of the interior of the eye. If ripe, the dark shadow of the iris approximates closely to the margin of the iris itself, whereas if the outer

portion is unripe there is a clear zone of greater or less depth between the margin of the iris and the shadow reflected by the opaque portion of the lens.

In the lower animals the question is less important as we do not aim at securing perfect vision, and the danger of inflammation is therefore the main consideration. Escaping this, the aqueous humor may be expected to dissolve and remove the greater part of the still adherent lens substance, and the unsightliness of the dense white cataract is largely done away with.

Discission. Tearing of the capsule so as to admit the aqueous humor to the lens may be admissible in the **young** with **soft cataract.** The liquid causes gradual swelling up, solution and absorption of the lens so that in the course of a week or two the whole may be removed. It is not, however, unattended by danger, as the rapid swelling of the lens will sometimes determine an inflammation which will lead to complete destruction of the eye. The eye is first thoroughly washed with aseptic cotton and a sublimate solution (1 : 1000), and is then rendered anæsthetic by cocaine (5 to 10 per cent. solution) or in the large animals general anæsthesia is produced by ether or chloroform. The eyelids are held apart by the lid speculum, the nictitans held if necessary by forceps, and the bulb steadied by seizing it with hooked forceps. A cataract needle is passed through the cornea close to its border, and carried through the pupil, previously dilated with atropia, so as to tear an opening in the anterior capsule about two-thirds the diameter of the lens. If the toughness of the capsule threatens to endanger the ciliary body by dragging upon it, two needles or fine hooks may be introduced through opposite borders of the cornea (inner and outer) and the capsule may be torn without throwing any strain on surrounding parts. The pupil must thereafter be kept dilated by atropia to obviate adhesion of the iris to the wound and the eye must be kept in comparative darkness and aseptic. If active inflammation sets in, cold, astringent or iced dressings may be called for, while if the swelling of the lens is threatening it should be at once extracted. If the eye becomes unduly tense, puncture of the cornea is indicated, and the relief of tension will sometimes start a tardy solution into renewed activity.

Linear extraction of the lens. The animal and the eye having been prepared anæsthetically antiseptically, and midriatically as for discission, the lids are fixed with a speculum, the nictitans and the bulb with forceps, a Gräfe cataract knife is introduced through the inner side of the cornea, close to its margin and with its point parallel to the front of the iris. The handle is then raised and the cornea detached from the sclera by a series of gentle sawing motions until it has reached a point parallel to the outer margin of the cornea. If the pupil is insufficiently dilated, the iris should now be seized by forceps drawn out through the corneal wound and snipped off by scissors curved on the flat. Then the cystotome (hooked knife) is introduced with its back turned downward and carried to the further side of the capsule and close to the iris, its cutting point is turned backward and inserted in the capsule, and drawn across from side to side to make an orifice large enough for the escape of the lens. It is then given a quarter revolution so as to turn the point of the knife downward and is withdrawn from the wound back first. The lower part of the sclera and cornea is now gently pressed with a lens spoon so as to dislodge the lens from the capsule and deliver it through the corneal wound. Counter pressure may be made on the sclera at the upper part of the eye ball. The cornea is now gently stroked with cotton dipped in sublimate solution to pass all blood from the anterior chamber, and render the parts antiseptic. The iris is carefully replaced inside the cornea and any obstinately protruding parts must be excised. The eye is now covered with cotton steeped in a sublimate solution (1 : 1000) and bandaged without undue pressure, and the animal tied to two sides of the stall so that it is impossible to rub the eye.

It is well to dress the eye on the second day, and if adhesion of the wound is complete it may be left without bandage at the end of a week or a fortnight.

Success depends mainly on the avoidance of infection. Therefore every indication of local or general infection should forbid the operation. Any existing infectious disease or any local eczema, conjunctivitis or disease of the lachrymal apparatus should be cured and the region thoroughly disinfected before proceeding. The head should have a good soapy wash, followed by a sponging with sublimate solution (1 : 1000), the conjunctiva

carefully washed out with the same and a bandage damp with it applied over the eye. This bandage is only removed on the operating table. Cloths dampened with the solution are laid on the face around the eye, the eye is cocainized with a 5 per cent. solution applied at intervals of one minute and when quite insensible the operation is commenced. The greatest care must be taken to render the hands of operator and assistants and all instruments thoroughly aseptic. The instruments are taken from a 4 per cent. solution of carbolic acid and placed in water (which has just been boiled) until needed, and to wipe the eye or make any application, sterilized cotton only is used and never touched to the eye more than once. A sublimate bandage is placed over the eye and worn for ten days or a fortnight. Then if the corneal incision is healed and pale it may be left off. The pupil should be kept dilated by a few drops of atropia daily for this length of time.

Any occurrence of iritis or choroiditis usually indicates infection and must be treated on general principles, but with especial reference to disinfection, and if this cannot be secured the eye will be almost inevitably lost.

In case of renewed opacity through thickening of the capsule an aperture must be torn in that membrane by the same method as in discission. This is commonly known as needling. It must be conducted under the same antiseptic precautions as in extraction.

Suction. This is only applicable to a lens which has become fluid as well as opaque. It consists in withdrawing the liquid lens through a hollow needle.

DISLOCATION OF THE LENS.

Congenital; acquired; traumatic, softening of suspensory ligament: hinge motion, lens drops behind iris, protrudes through pupil, cataract. Apparent increased depth of anterior chamber, tremulous iris, projecting edge of lens like black ring. Treatment: extraction.

Dislocation of the lens may be congenital, or acquired. In the latter case it is explained by a traumatism or a liquefaction of the

vitreous and coincident softening of the suspensory ligament. In either case, if the ligament is torn through in more than one-half its circumference, the lens will hang by the remainder and move on it as on a hinge so as to change its location in the different positions of the head. This is especially so where the vitreous has become abnormally fluid as there is then no resistance to the free backward movement of the lens. The writer has seen the eye of a cow affected in this way, so that the cataractous lens advances to the pupil and recedes from it as the eye is moved. In other cases the dislocated lens, being attached below only, drops down and virtually disappears behind the lower part of the iris. In still other cases it becomes wedged into the pupil, or protrudes into the anterior chamber and lies in front of the pupil and iris. The semi-detached lens sooner or later becomes opaque. A cataract with contraction of the newly formed tissue on the capsule and undue tension on the suspensory ligament may, however, precede the dislocation which is then precipitated by some shock, as a blow, fall, sneeze, cough or emesis.

The condition leads to an apparent increase in the depth of the anterior chamber, and tremulous movement of the iris, and if illuminated the impacted condition of the lens or its changes of position can be detected. If its edge is exposed it appears as a black ring.

Treatment is useless, unless it be extraction of the lens, or iridectomy in suitable cases.

PERSISTENT ARTERIA HYALOIDEA.

In the embryo this artery occupies the central canal of the vitreous and extends from the optic papilla to the posterior pole of the lens. At times it persists after birth and even to mature life and is then detected as a gray or dark thread on ophthalmoscopic examination. Berlin records a case in an old horse and others in young cats, and Möller other cases in dogs. Treatment is manifestly hopeless.

OPACITY OF THE VITREOUS.

From inflammation of the ciliary body, choroid or retina. Pupil copperas green with white points, lines or patches, move in opposite direction from the eye, liquefied vitreous, crystals of cholesterine, scintillance, opacity. Treatment.

Opacities visible with the ophthalmoscope usually come from effusion of blood into the vitreous, or inflammation of the retina, choroid, or ciliary body. Blood thrown into the vitreous will usually leave some permanent turbidity. In choroiditis or retinitis the ophthalmoscope may reveal the changes in these structures. A turbidity appearing in the anterior portion of the vitreous, without any apparent cause, is probably due to cyclitis which cannot be seen with the ophthalmoscope, but may cause special tenderness around the sclero-corneal zone. It is common in recurrent ophthalmia of the horse and in irido-choroiditis in animals generally, and may be a distinct feature of glaucoma. The general reflection of the pupil is copperas green, but gray or white points, lines, bars or patches may often be recognized. These being back of the lens and axis of the eye, move in the opposite direction from the line of vision, thus if the eye looks upward they descend, if it looks downward they ascend, if it turns to the right, they turn to the left and vice versa. Tested by a burning candle the three reflections remain clear and distinct unless lens or cornea are affected.

Not infrequently the vitreous is found abnormally fluid, and among its solid particles in affected horses have been found crystals of cholesterin (Jacobi) and tyrosin (Möller). In the illuminated vitreous such crystals may be seen to reflect the light like a shower of sparks (synchysis scintillans). Opacity of the vitreous has been seen in cases of amaurosis in horses (Hertwig) and glaucoma in lambs (Prinz).

Treatment is rarely satisfactory, though in some recent cases, and in the absence of any other irremovable lesion, reabsorption of a slight exudate may take place, in connection with the use of mild saline laxatives and diuretics.

RETINITIS.

With choroiditis and cyclitis, albuminuria, nephritis, mellituria, leukæmia, petechial fever, contagious pneumonia. Photophobia, contracted pupil, redness around optic disc, enlarged retinal vessels, white and gray spots and radiating lines, exudates, clots, fatty degeneration. Treatment: correct primary disease.

Retinitis is usually an accompaniment of choroiditis and cyclitis, but it also occurs as a complication in a number of constitutional maladies, such as albuminuria, nephritis, mellituria, leukæmia, petechial fever, contagious pneumonia, etc. Fröhner records a case in a leukæmic horse, Peters in one suffering from petechial fever, Schindelka in cases of contagious pneumonia in the horse, and Eversbusch in recurrent ophthalmia of the horse, and under other conditions in dogs.

The attack is accompanied at the outset with much photophobia and contraction of the pupil. When this is dilated and the fundus of the eye examined with the ophthalmoscope, the retina is seen to be reddened for some distance around the optic disc and the blood-vessels are materially enlarged. Later, white or gray spots and lines are seen in and around the disc, tending to assume a radiating direction, and the retina at large, on careful examination may have a distinctly striped appearance. Brownish, reddish or light colored exudates and hæmorrhages may be made out in certain cases between retina and hyaloid, or between the retina and choroid. Fatty degeneration of the fibrous tissue is common.

Treatment must be first that for the primary disease of which the retinitis is a complication, and the result will depend on how amenable that affection is to therapeutic measures. In advanced albuminuria or mellituria, the retinitis, which is usually double, is hopeless, while in contagious pneumonia, petechial fever, leukæmia and other less fatal affections, retinitis in its initial stages may recover. In cases of advanced disease with serious structural changes in the retina, recovery cannot be looked for.

DETACHMENT OF THE RETINA.

This may occur in any case of retinitis or choroiditis. It is especially common in recurrent ophthalmia in horses. It may also occur through the dragging by contracting inflammatory products in organization. Spontaneous recoveries have taken place through re-absorption, and attempts have been made by the injection of iodide lotion to hasten this, or more safely by rest and diaphoresis. Puncture and aspiration have also been tried with most varied results. As a rule in the lower animals the treatment of the inflammation, with rest, a pressure bandage, and pilocarpin will be indicated.

RETINAL HEMORRHAGE.

This occurs in inflammations of the retina or choroid, also in degenerations of the vascular walls and as a result of traumas, or poisons. Schindelka quotes a case in a dog from ptomaines, Eversbusch, one from scurvy in the dog, Appenroth, one in a calf from a blow with a cow's horn, and others in cats and horses from traumas. It is present to a slight extent in all inflammations of the retina. With the ophthalmoscope the recent lesion appears as a bluish red blotch on a bright red ground, and the older lesion of a brownish red. Rest and a pressure bandage may be employed after subsidence of the inflammation.

TUMORS OF THE RETINA.

Eversbusch found subretinal cysts in a 24 year old horse, Merkel and others have found similar bodies in old dogs and cattle. Sarcomata and melanosarcoma are found on the surface of the choroid in gray horses, implicating the retina. The only remedy is enucleation of the bulb.

INFLAMMATION OF THE OPTIC NERVE. PAPILLITIS.

Choked disc. Neuro-retinitis. Papillo-retinitis. Nerve swelling blocks veins. Causes: lead, tumors in brain bilateral, in orbit unilateral, influenza, petechial fever. Symptoms: redness of optic disc, retinal arteries contracted, veins gorged, papilla swollen (woolly), vitreous opacity. Treatment.

This is also known as *choked disc* from the obstruction of the return of blood by the veins of the retina which pass out at the cribriform lamina. The swelling of the nerve ending and papilla compress the veins, causing stagnation and venous congestion in the retina. When the retina is thus involved the term neuro-retinitis or papillo-retinitis is often applied.

Causes. The condition has been traced to lead poisoning, brain or orbital tumors, and various infectious diseases. Except in orbital tumor the disease is usually bilateral. In horses Schindelka has seen the condition in influenza and Peters in petechial fever, heart failure and dyspnœa. In a necropsy on a horse the writer found complete cataract, atrophy of the bulb to less than two-thirds the size of its fellow, thickening and furrowing of the sclera, the presence of an extensive old blood clot in the seat of the vitreous, cupping of the disc, retro-ocular neuritis with exudate inside the sheath, and a considerable reddish brown tumor of the choroid plexus on the same side. The trouble was attributed to the blow of a rope on the eye a considerable time before. The blood in the vitreous retained its dark hue and showed no evidence of microbian invasion.

Symptoms. To the phenomena of retinitis there is added violent injection of the capillary plexus of the papilla. The arteries of the retina are contracted while the veins are dilated and tortuous. The papilla is swollen and is said to have a woolly appearance. The vitreous may be opaque by reason of exudation or blood extravasation into its substance. Retro-ocular neuritis is usually present, and brain tumor in certain cases. Möller calls attention to the fact that with atrophy of the bulb the elliptical outline of the papilla gradually lessens and it becomes more nearly round. In dogs, too, the form of the papilla is also changed and the same author has found it distinctly triangular. The

pupil may be at first strongly contracted and later freely dilated.

Treatment is usually of little avail and does not differ materially from that adopted in retinitis. In tumor of the orbit pressing on the optic nerve it may sometimes be reached through the temporal fossa, but usually in all such cases enucleation of the eyeball is demanded.

RETRO-BULBAR OPTIC NEURITIS.

Definition. Neurites descendens and ascendens. Extension to bulb, other eye, or brain. Causes: traumas, meningitis, infectious diseases, toxins, lead, tobacco, etc. Symptoms: partial blindness: congestion of papilla, exudates in adjacent retina. Treatment: eliminate poisons, lead, toxins, correct precedent disease, saline laxatives, diuretics, potassium iodide, cupping, local bleeding, counter-irritants. Hygiene.

This is inflammation of the optic nerve commencing behind the eyeball and only involving the papilla secondarily. Extension to the papilla has been called *neuritis descendens* in contra-distinction to *neuritis ascendens* in which the inflammation extends along the nerve toward the brain. There is always a tendency to extension, it may be to the papilla and retina, it may be to the chiasma and opposite eye, and it may be inward toward the ganglia and choroid plexus.

Causes. Apart from traumatic causes, retro-bulbar neuritis in the domestic animals has been mainly seen in connection with constitutional or infectious diseases; in the horse with petechial fever (Peters), contagious pneumonia (Schindelka), meningitis (Peters, Straub) and parotitis (Möller). It has also followed meningitis in cattle (Nebelen). In man it has been traced to lead, tobacco, alcohol and other poisons.

Symptoms. At first the patient may seem partially blind without apparent cause. Later ophthalmoscopic examination reveals congestion and swelling of the papilla, and congestion (especially venous and capillary) of the retinal vessels. The discolorations in streaks and spots, from exudates and degenerations follow as noticed under papillitis. It is difficult to distinguish between this and papillitis during life.

Treatment should be first directed toward the elimination of any poisons, such as lead or tobacco, which may act as a causative factor. Saline laxatives and in chronic cases potassium iodide would be indicated. Every attention should be given to the maintainence of good general health, and in acute attacks, local bleeding and counter-irritation may be resorted to.

ATROPHY. PARALYSIS OF THE OPTIC NERVE. AMBLYOPIA. AMAUROSIS.

Definition. Horses, dogs, cattle, sheep. Amblyopia, Amaurosis. Causes: retinal paresis from brilliant light, of cerebral ganglia, snow blindness, poisons, lead, opium, atropine, lolium, tobacco, carbon bisulphide, cryptogams, mushrooms, "loco" plants, carbon monoxide, illuminating gas, quinine, heat apoplexy, cerebral concussion, brain parasites, exudates, pressure, degeneration, gastric or intestinal disorder, hæmorrhage, anæmia, diarrhœa, diuresis, excessive milk secretion, gestation, embolism. Symptoms: constant excessive dilatation of pupil, no response to light, brilliant reflection from retina and choroid, blue or yellow, (glass eyes); bilateral, cerebral, tract or chiasma; unilateral, bulbar or nervous; face without expression, ears alert, head to one side, nose elevated, nostrils dilated, ox and dog smell ground, slow pace, high stepping, does not dodge a feint to strike. Lesions: inflammatory exudate and retinal atrophy, congested, "woolly" papilla, retro-bulbar congestion or atrophy of nerve, thickening of neurilemma, tumors of orbit or foramen optica, cœnurus, cysts, abscesses, cholesteatomata, psammomata, hydrocephalus, meningitis, pineal hypertrophy, cranial fractures or tumors, embolisms or aneurisms, hypertrophy of dura, apoplexy. Treatment: remove causes, laxatives, diuretics, for lead, sulphuric acid and potassium iodide, darkness, extract cœnurus, elevate depressed fractures, ice, cold snow, to head, blisters, setons, electricity, strychnia, remove tumors.

Blindness without adequate, corresponding, visible changes in the structure of the eye, has long been recognized in the lower animals, having been observed in horses, dogs, cattle and sheep. When partial it is known as amblyopia, and when complete as amaurosis or blindness. The term amaurosis is, however, being gradually eliminated from ophthalmological nomenclature in favor of that of atrophy.

Causes. These may be factors which act directly on the retina, as the intense light of the sun, of an electric, lime or

magnesium light, of the reflection from snow, ice, water, sand, or white dust. These exhaust the power of vision by over-stimulating it, but whether the blindness is due to fatigue of the retina or of the cerebral ganglia which preside over sight, it is difficult to decide. This form is much more likely to occur in horses which spend most of their time in darkness, as in unlighted stables or mines, when they are suddenly brought into the sunlight. Snow blindness, however, from prolonged exposure, is common in animals as in man, and the case of the staghound reported by Johan, is by no means an isolated one.

Much more common are factors which act primarily on the nerve centres. Poisoning with lead, poppy, belladonna (sheep and goats, Gerlach), lolium temulentum (Kliiver) tobacco, carbon bisulphide, cryptogams (musty fodder), mushrooms, astragalus mollissimus, etc., abolishes vision more or less perfectly. Common illuminating gas and carbon monoxide have shown this effect on dogs and chickens. Quinia subcutem has induced the condition in dogs (Becker and Eversbusch). More or less complete amaurosis is noticed in connection with heat apoplexy in horses and fat cattle.

Cerebral concussion from blows on the head, knocking the head against a post or wall, or from violent falls induce blindness which is to be attributed rather to lesion of the brain. The same remark applies to cases that appear during the progress of brain disease, parasitic or otherwise, to those that occur in connection with overloading of the stomach or intestines, and from gestation (Riss).

In cases appearing after severe depletion, like profuse hæmorrhage, diarrhœa, or diuresis, or excessive secretion of milk (bitch) the anæmia of both eye and brain may be taken into account.

When amaurosis develops during some inflammatory or infectious disease, as in parturition fever in cows, the immediate cause may sometimes be found in embolism of the retinal, or cerebral arteries.

Symptoms. The most striking feature of amaurosis is the constant excessive dilatation of the pupil. This is the same in light and darkness, and in fully developed cases the animal may be taken from perfect darkness to the full glare of sunlight or

electric light without the slightest contraction of the pupil. The pupillary reflex to light is entirely lost. In the slighter cases (amblyopia) there remains some slight response of the iris to light but always far short of the normal. The widely dilated pupil admits a flood of light, and in the absence of cataract, lights up the chamber of the vitreous to an unusual degree. The blue or yellowish reflection of the tapetum is very striking, and led to the obsolete names of gutta serena and glass eyes. Sometimes the disease is unilateral and at others bilateral. In case it is unilateral there is a strong presumption that the causative lesion is in the affected eye or in the optic nerve in front of the chiasma. In the bilateral cases it is more likely to depend on disease of the brain or some more distant organ.

Certain important indications are to be drawn from the expression of the face, the position of the head and ears and the mode of locomotion. In all blind animals the face is without expression. The dilated pupils give no suggestion of mind looking through them, they have on the contrary an uniform stony stare. There is no sense of fire or life, but the face is like a dull sculptured mass. The immobile face finds a sharp contrast in the alertness of the ears, which may be carried one forward and another back, but fixed and yet ready to turn to catch every sound. In the horse the head is usually carried a little to one side for the same reason, and with the nose elevated (*star-gazer*) and nostrils dilated. The ox inclines to carry the head low, while the dog keeps his nose near the ground to guide himself by the sense of smell. All blind animals are disposed to move slowly and carefully, and the horse acquires a habit of *high stepping* to avoid stumbling.

A common and important test where both eyes are equally affected and cannot be contrasted with each other, is to strike the lower part of the face with the hand and immediately threaten to repeat the blow. If the animal can see, it will seek to jerk the head out of the way as soon as the hand is raised for the second blow, while if blind there will be no such movement provided the motion of the hand has not been such as to induce a current of air on the face.

Lesions. Blindness, or the general symptoms of amblyopia or amaurosis, may attend on the atrophy of the retinal fibres in con-

nection with inflammatory exudation or blood extravasation on, in, or beneath, the retina. In other cases the lesion is in the papilla which is inflamed and swollen with the woolly aspect characteristic of choked disc. In others there is congestion of the optic nerve behind the bulb, with exudation into its substance or beneath its sheath, or the nerve has already undergone extensive atrophy with thickening of its neurilemma. In other cases there is atrophy of the arteria centralis retinæ, or tumors of the orbit or optic foramen pressing upon the nerve. Cases of this kind are reported in the larger animals by Leblanc and Tscheulin, and in dogs and ducks by Hilbert.

Much more frequently the determining lesions are situated in the brain or its meninges. In sheep it is very common from the development of cœnurus cerebralis over the optic lobes. Kühnert found a cyst with delicate walls in the brain of a horse affected in this way. Amaurosis is occasionally seen in connection with the cerebral abscesses which form in complicated cases of strangles or in pyæmia, also in cases of cholesteatoma of the choroid plexus. Other conditions of its occurrence are hydrocephalus, meningitis, hypertrophy of the pineal gland, fracture of the cranium, tumors of the cranium or dura mater, embolism or aneurism of the cerebral arteries, hypertrophy and induration of the dura mater (ox, Leblanc), or sanguineous apoplexy. In a certain number of cases there may be no lesion of brain or eye, or only a congestion of the former in connection with lead poisoning, rye grass poisoning or other gastric disorder, or of gestation.

Treatment. Success will depend on the sympathetic nature of the condition or on the transient and removable character of its causative factor. When the condition is but a symptom of overloaded stomach or a transient poisoning by vegetable or mineral agents a direct recovery may be expected to follow their disuse and elimination from the system. This may be hastened by the exhibition of laxatives and diuretics, and in the case of lead by sulphuric acid followed by iodide of potassium. In cases of snow blindness it is only necessary as a rule to place the animal in the shade until the over-stimulation shall have subsided. In all these cases the attack has come on abruptly and without any local symptom of ocular hyperæmia and this with the preserva-

tion of the general senses can always be looked on as a favorable indication.

In cases that come on slowly and which are accompanied by symptoms of vascular disturbance or structural changes in the eye, or brain, treatment is likely to be less successful. Nevertheless cœnurus may be removed by surgical means, depressed fractures may be elevated, and acute cerebral and meningeal congestions may be met by appropriate measures. If the cerebral congestion is acute, free bleeding from the jugular with a strong purgative and the application of ice or cold water to the head may prove useful. Apparent benefit has also followed the use of blisters on the face or back of the ear, of setons, and later of a weak electric current and strychnia. Tumors also may be advantageously removed.

But in cases marked by destruction of the retina or papilla, by congestion or atrophy of the optic nerve, by destructive disease of the optic foramen, or of the brain or its meninges, treatment is futile.

ANOPHTHALMOS. ATROPHY OF THE EYEBALL. PHTHISIS BULBI. MICROPHTHALMOS.

In some cases the eye is congenitally absent (*Anophthalmos*). In others it is abnormally small. One such case came under the notice of the author in which the eyeball was represented by a small black sphere about half an inch in diameter moved by the ocular muscles. The dam of the filly, born with this defect, had, during the pregnancy, a burdock entangled in the forelock and causing a violent ophthalmia which was supposed to have lasted for months. In other cases there is a fistula opening from the vitreous behind.

Cases of wasting and atrophy of the eye follow on exudates into the vitreous and their subsequent contraction, or on suppuration and granulation as noted under *internal ophthalmia*, *recurrent ophthalmia*, and *panophthalmia*. The condition may also result from atrophy or degeneration of the optic nerve or of its cerebral ganglia (thalamus, corpora quadrigemini, geniculata, etc). These conditions are irremediable.

LUXATIO BULBI. DISLOCATION OF THE GLOBE OF THE EYE.

Definition. Dog, anatomical factors. Symptoms: protrusion of bulb through palpebral orifice, orbicular spasm, vessel, muscle, nerve stretching or tearing. Sphacelus. Panophthalmia. Fracture of orbit. Treatment: early reduction, antisepsis, astringents, scarify sclera, cold, astringents, puncture aqueous with hypodermic needle, enlarge palpebral opening, suture and compress, remove foreign bodies and injurious fragments of tissues, enucleation.

Definition. Displacement of the globe of the eye out of the orbit and through the eyelids.

Causes. Among domestic animals the condition is most frequently seen in the dog, which is predisposed by reason of the normal prominence of its eye, the width of the aperture between the lids and the absence of the orbital process of the frontal bone. Blows upon the region and the insertion of pointed bodies, (teeth, horns, etc.), which can act as levers using the margin of the orbit as a fulcrum are especially liable to cause the lesion. Dog fights are the most common occasions. Other animals may also suffer but not at all frequently.

Symptoms and lesions. In the simplest form the bulb is displaced forward out of the orbit and through the palpebræ which latter contract spasmodically behind it and effectually prevent a spontaneous reduction. The optic nerve, muscles, and vessels are unduly stretched and the circulation in the bulb is seriously impaired, so that even in the least complicated cases any undue delay in reducing the dislocation will lead to serious and destructive changes in the eye. Sphacelus of the globe is not uncommon under such conditions.

In the more complicated cases, the conjunctiva, palpebræ, nictitans, muscles, nerves, etc., may be more or less lacerated and the globe itself may be seriously damaged either by internal lesion or by an external trauma. In all these cases there is most imminent danger of general infective inflammation of the eye, of panophthalmia, and even of secondary general infection of the system. Fracture of the bones of the orbit may also be looked for.

Treatment. When dislocation is uncomplicated and recent, say of a few hours standing only, it may be reduced and a favorable issue secured. The bulb should be first washed with water which has been sterilized by boiling or rendered antiseptic with sublimate (1 : 5000), and can usually be pressed back by steady uniform pressure. The insertion under one lid of a small spatula bent at the end or the one limb of a lid speculum may assist in difficult cases. When replaced the parts may be again washed with antiseptic solution and covered by a bandage wet with an astringent collyria.

When the condition has been neglected for a day or more the bulb is congested and swollen so that its return is rendered much more difficult, and its subsequent retention may require much care and ingenuity. The reduction of the turgid globe may be assisted by opening the veins and arteries on the sclera, by astringent applications, by massage, and in obstinate cases by evacuation of a portion of the aqueous humor, by the aid of a fine aseptic needle. Finally the palpebral opening may be enlarged by incising the outer canthus with a probe pointed bistuory. When the eye has been replaced in its socket this must be closed by suture. For the retention of the eye in such cases a bandage may suffice, or this failing, the lids may be held together by strips of adhesive plaster, or by collodion. In very difficult cases Lafosse and Trasbot recommend sutures through the skin 1½ to 2 inches from the palpebral borders and the whole covered with a bandage impregnated with an antiseptic and astringent collyrium.

It is not requisite to keep the bandage in position for over four or five days as the swelling of the eyelids and other adjacent structures effectually prevents any tendency to repetition of the luxation, and the eye may be treated like an ordinary traumatic lesion.

At the outset, and later if need be, any foreign body in the orbit should be removed and any detatched pieces of bone which cannot be retained firmly in position, and which are liable to prevent healing or to determine infection of the wound.

In the worst cases and in those that have been neglected until gangrene or panophthalmitis threatens, the removal of the eyeball may be the only resort. The animal may be anæsthetized

by chloroform or ether, or locally by cocaine. The conjunctiva covering the sclera is then pinched up with forceps and cut through with scissors, this is continued all around the globe. Then the recti muscles, the superior and inferior oblique muscles, the retractor and finally the optic nerve are cut through with a pair of scissors curved on the flat. The divided ends of the muscles are now sutured together around the nerve which has been cut shorter, and the cavity irrigated by a cold antiseptic solution. Bleeding vessels may be twisted through with forceps if the flow is not readily checked by cold irrigation. Or a pledget of cotton dipped in tincture of muriate of iron may be loosely inserted (firm pressure would be unnecessarily painful). As a subsequent dressing, standard sulphurous acid solution, glycerine and water in equal proportions, or other antiseptic dressings may be applied.

ARTIFICIAL EYE.

These are largely in use in the human being, and have been employed in the lower animals in different cases, especially in the horse, with excellent effect. The advantages may be summed up in this, that they do away with the unsightly appearance of an empty orbit with the edges of the lids turned into the dark aperture, enhance the value by restoring the face to nearly the natural appearance, and prevent the lodgment of dust and insects in the cavity.

The artificial eye may be made to appear more natural if made of glass, yet when made of horn or still better of hard rubber, colored like the normal iris and pupil, it has the advantage of greater lightness. It must be perfectly smooth so as to cause no discomfort, and should never be introduced so long as there is any irritation in the stump or conjunctiva. It may be slipped in like a button, first beneath the deeper upper lid, and then beneath the lower, and should be worn only while at work and so long as it causes no irritation nor purulent discharge. On the return of the animal to the stable, the artificial eye is taken out, washed and placed in clean pure water. The orbit should be sponged out with a weak collyrium (boric acid 1 : 100).

In man, excentration is sometimes substituted for enucleation, the cornea is removed together with the lens, vitreous, choroid and retina, leaving only the sclera which contracts into a dense scar tissue with the muscles attached. Or an artificial vitreous of glass or unoxidizable metal is introduced around which the sclera is allowed to heal. This introduces an additional element of danger over the formation of a simple sclerotic stump, but, when successful, it affords a better support to the artificial eye, turning it freely in harmony with its fellow and giving it a more natural aspect.

STRABISMUS. SQUINTING. LACK OF MUSCULAR BALANCE.

Causes : paralysis of eye muscles, bulb rolls from affected muscle, spasm of eye muscles, bulb rolls toward affected muscle, convergent squint most common. Hold head still and move object in front of eyes, imperfect movement toward paretic muscle or away from the spastic one. Ptosis. Overfatigue. Debility. Nerve or brain lesion. Dislocation of bulb. Treatment : treat any transient etiological factor, cerebral congestion, parasitisms, debility, anæmia. Tenotomy of rectus : advance of paretic rectus.

Strabismus may be due to a variety of causes, among others to the following :

Paralysis of one of the ocular muscles. When the eyes are turned in the direction away from the affected muscle the muscle is deficient in power. It may be the external rectus (abducent nerve) producing convergent squint. It may be of the superior oblique muscle (4th or pathetic nerve) causing a faulty movement of the eye downward and inward or a slightly convergent squint. Divergent squint commonly indicates paralysis of upper, lower and inner recti, and the inferior oblique (3d or oculo motor nerve): this is usually associated with ptosis or drooping of the upper lid, the levator of which is supplied by the same nerve. The existence of squint is usually so marked that no special method of examination is required. If otherwise, however, the animal's head may be held still and some object which will attract his attention is moved before the eyes, outward and inward, when the affected eye moving in the direction of the

paralytic muscle will lag visibly behind its fellow. These conditions are usually due to lesions in the respective nerves or their cerebral ganglia.

Spasmodic or Spastic Squint is the exact antithesis of the above, the eye turning toward the muscle which is the seat of spasm. It may be seen in certain cases of rabies and is always due to disorder of the central nervous ganglia.

In some cases squinting is associated with over fatigue, or debility, and then usually partakes of the paralytic character.

In the lower animals convergent strabismus has been most frequently observed. Brouwer records a case in the horse and Koch a congenital one in the cow. Zschokke reports a case in the cow connected with an angioma at the base of the brain. Other forms are noted by Peters, Barrier, Bayer and others. Stockfleth quotes a case in the dog following prolapsus bulbi and doubtless connected with injury to ocular muscles or nerves, sustained in the accident.

Treatment will vary with the ascertained cause. As a rule cases that depend on structural changes in the brain are hopeless. Those that depend on temporary congestion or other transient disorder of that organ may recover when that has been overcome. In cases in which debility is a prominent feature, tonics, moderate exercise in the open air and general hygienic care are demanded. The final resort in bad cases is tenotomy of the rectus on the side toward which the eye turns. In man when this is found to be insufficient the opposing weak or paretic muscle is also advanced. The tendon close to its sclerotic attachment is laid bare by incision, and a silk or catgut thread is passed through each border, upper and lower. The tendon is now cut through with scissors on the corneal side of the sutures and, by means of their needles, the latter are passed through the conjunctiva and capsule of Tenon, from within outward and close to the margin of the cornea. The sutures are now tied somewhat tighter than is absolutely necessary to properly balance the eye so as to allow some room for relaxation in healing. We are not aware that this measure of advancement has been employed in the domestic animals.

NYSTAGMUS. OSCILLATORY MOVEMENT OF THE EYE.

This consists in spasmodic involuntary oscillation of the eyeball in a horizontal, lateral, oblique or rotary direction. In animals it has been seen in connection with poisoning and brain diseases. Johne has observed it in horses in cerebro-spinal meningitis, Wenderhold in epilepsy, and Möller in chloroform anæsthesia. Möller has further seen it in puppies with congenital microphthalmos, and Siedamgrotzky in swine which had been poisoned by herring brine.

Slight cases of functional disturbance may improve under good hygiene, open air life and tonics, cases due to poisons may recover spontaneously when such poisons have been eliminated, but those which depend on structural disorder of the brain are beyond remedy.

DISEASES OF THE SKIN.

Ultimate skin lesions in man and animals similar. Masked by thick cuticle, pigment, hair, fur, feathers. White, hairless skin. Lesions and deranged functions: Maculæ, erythema, papules, nodules, blisters, blebs, pustules, boils, carbuncles, scales, crusts, sitfasts, horny growths, erosions, abrasions, chaps, fissures, ulcers, excrescences, cicatrices, neuroses, morbid secretions, changes in glands, hairs, in derma. Scleroderma. Elephantiasis. Vegetable and animal parasites.

In cutaneous diseases in man and animals the actual lesions are largely of the same nature, yet in the animal covered with hair, fur or feathers, and with the cuticle deeply pigmented, the diagnosis of the different affections becomes much more difficult. On white-skinned animals and on parts with little or no hair, the identification of the different forms is usually possible. The following list may serve to indicate the nature of the different lesions, but these must not be accepted as indicating distinct diseases, as two or more of these forms often co-exist or succeed each other in the same affection :—

1st. **Maculæ: Spots: Discolorations.** Examples: Black, melanotic spots in skins of white horses: white spots in dourine, after pustules, etc.: ecchymosis after contusions, stings, insect bites, etc.: petechial spots in anthrax, rouget, hog cholera, rinderpest, canine distemper, swine plague, scurvy, etc.

2d. **Erythema: Rash: Flush.** Congestive redness usually disappearing under pressure. *Physiological* in blush or glow of exercise, *pathological* from insolation, friction, deranged innervation, etc.

3d. **Papulæ: Papules: Pimples.** Small, red, hard, conical elevations, not forming blister nor pustule. Due to exudation and the accumulation of leucocytes at given points, having a local or general cause, (psoriasis, intertrigo, etc.).

4th. **Tuberculæ: Nodules.** Larger but still circumscribed

thickening of the entire skin from exudation and cell growth, from ½ inch to 2 or more inches in diameter and sometimes becoming confluent. Examples: Urticaria (surfeit) in solipeds, and cattle; petechial fever, farcy, etc. Sometimes chronic.

5th. **Vesiculæ: Blisters.** Rounded or conical elevations the size of a millet seed to a pea, and having a small liquid exudation under the cuticle in the centre. In inflammations of the papillary layer, of a sufficiently acute type the tendency is to the formation of vesicles. These lesions are, therefore, often present in very different forms of skin disease from those due to simple thermic irritation, to constitutional diseases like eczema, or contagious ones like sheep-pox. May merge into pustules or other advanced lesions.

6th. **Phlyctenæ: Bullæ: Blebs.** In these the individual lesion is larger than in vesicles. They are of any size from a pea upward. The most striking example is in cantharides, blisters, scalds and burns, but in other cases it depends on a constitutional condition or a specially exudative dermatitis.

7th. **Pustulæ: Pustules.** These differ from vesicles in that the central exudate becomes the seat of suppuration and a limited collection of pus, at first central, though later involving, it may be the whole area of the exudate. It is often merely an advanced stage of the papule or vesicle. We find examples in the different forms of variola, in lesions caused by tartar emetic or croton oil, and in several forms of dermatitis. It is essentially microbian.

8th. **Furunculus: Furuncle: Boil.** Inflammatory nodosity of the derma, resulting in a necrotic central core and suppuration. Is bacteridian and common on the coronet and lower parts of the limbs in solipeds.

9th. **Carbunculus: Carbuncle.** An inflammatory nodosity or cluster of nodes of much greater extent, tending to necrotic change and sloughing over a much more extending area. Microbian.

10th. **Squama: Scales: Dandruff.** Exudation products and cells dessicate and exfoliate as bran-like scales or thicker coherent laminæ. Examples are found in psoriasis, pityriasis, eczema, variola, rinderpest, etc.

11th. **Crustæ: Crusts: Scabs.** Hard, solidified masses of epidermis, blood, pus and serous exudate.

12th. **Callositas: Callosity.** Abnormal thickening of the epidermis, as a physiological protective cell growth. Examples: pads on the knees of camels, cows and even horses from kneeling on a hard, uneven surface.

13th. **Sitfasts: Necrotic Callosities.** Combination of dried up exudate of horny consistency, and a thickened, fibroid and partially necrotic portion of the subjacent derma with little or no disposition to spontaneous detachment.

14th. **Cornu Cutaneum: Keracele.: Horny Growth.** Abnormal horny growth from keratogenous tissue, or from the derma in its vicinity or at some other point of the skin.

15th. **Erosions: Abrasions.** Lesions of the cuticle exposing the true skin, and the result of itching, scratching, friction, biting or other mechanical or thermic injury.

16. **Rimæ: Cracks: Chaps.** These are linear breaches often confined to the epidermis in the bends of joints, under congestion and suppression of sebaceous secretion, in elephantiasis, dropsy, petechial fever, etc. Unless they have ulcerated they may heal without cicatrix.

17th. **Crevasses: Fissures.** These are chaps, which extend into the derma, giving rise to destruction of tissue and leaving a cicatrix on healing. Examples are found in the hollow of the pastern, behind the knee (Mallenders), in front of the hock (Sallanders), in the swellings of petechial fever, malignant catarrh, stocked legs, grease, etc.

18th. **Ulcus: Ulcer.** A sore that extends by the continual molecular breaking down of the forming granulations and of the adjacent and subjacent diseased tissue.

19th. **Excrescences: Hyperplasiæ: Phymata: Dermatomata.** These may include over luxuriant granulations which rise above the level of the skin and become organized into projecting fibro-cellular, raw or scabby masses: tumors of all kinds —warty, papillomatous, horny, epidermic, cancerous, sarcomatous, pigmentary, angeiomatous, tuberculosis, etc.

20th. **Cicatrices: Scars.** These are puckered, raised or sometimes depressed, lines or areas of condensed connective tissue with a covering of epidermis, taking the place of the normal dermis and epidermis and their appendages, which have been destroyed. They result from traumatic, ulcerous, or atrophic destruction of the skin.

21st. **Neurosis.** These may be exemplified by the intense itching of the skin without appreciable structural change. So in cutaneous anæsthesia and hyperæthesia.

22d. **Modified Secretions.** These include absence of perspiration—anidrosis, excessive perspiration—hyperidrosis, suppressed sebaceous secretion—asteatosis, excessive sebaceous secretion—steatorrhea or seborrhea, fœtid sweat—bromidrosis, colored sweat—chromidrosis, *urinous sweat*—uridrosis.

23d. **Structural alterations in glands and ducts.** Cystic ducts—hydrocystoma, blocked ducts—acne, inflamed glands—hidrosadenitis.

24th. **Abnormal conditions of the hair.** This embraces baldness, hypotrichosis, alopecia, excessive growth of hair, hypertrichosis, white patches, canities, nodular hairs, piedra, brittle hair, fragilitas crinium, filted hair, plica, trichoma.

25th. **Scleroderma.** Hard, leathery, thickened skin. Examples in old boars on shoulders, and in other animals.

26th. **Elephantiasis Pachydermia.** Enormous hypertrophy of the skin, with usually distention of the lymph plexuses and vessels (lymphangiectasis: see Vol. I).

26th. **Vegetable parasites.** Trichophyton, achorion, microsporon, actinomyces, etc. (See parasites).

27th. **Animal parasites.** Lice, fleas, diptera, trombidium, Acari, ixodes, cimex, filaria, coccidia, etc. (See parasites).

GENERAL CAUSES OF SKIN DISEASES.

External. Internal. Traumas, abrasions, excoriations, lacerations, contusions, compression, radiating heat, scalding, incandescent objects, solar heat, chemical caustics, cold, freezing, kicks, bites, tusk horn or claw wounds, stings, venoms, envenomed bites, road dust, sweat, excretions, sebum, mineral and vegetable poisons, essential oils, fungi. Hyperæmia, exudation, depilation, sudation, moulting, climatic changes, unwholesome or irritating foods, ptomaines, alimentary fermentations, hepatic, renal or blood disorders, altered innervation, youth, age, temperament, heredity. Experimental nervous cases.

These may be **external** or **internal** or both.

External Causes. Some affections of the skin are due to

external causes exclusively, while in others the local cause of irritation is accessory but no less important in maintaining the trouble. Among the more prominent external factors may be named: traumatisms, abrasions, excoriations, lacerations, contusions, compression, radiating heat, boiling water, hot or incandescent solids or liquids, solar heat, chemical caustics and irritants, cold, freezing, injuries by harness, kicks, lacerations with teeth, tusks, horns or claws, stings, bites, (leeches, snakes, etc.), venoms, (snake, toad, etc.), road dust and sweat, liquid fæces or urine, excess of sebum in sheath or vulva, mineral poisons (mercurial, iodides, bromides, arseniates, caustic alkalies, caustic salts, etc.), vegetable poisons (croton, bryonia alba and dioica, heracleum or cow parsnip, polygala or milk wort, cyclamen or sow bread, polygonum hydropiper, mustard, œnanthe, cicuta, hypericum perforatum and androsaemum, rhus toxicodendron, radicans and venenata, capsicum, pepper, radish, Indian syringa, anemone nemorosa and patens, ranunculus acris, scelerata, flammula, mericatus and bulbosus, cytisus, euphorbium and the essential oils of turpentine, origanum, lavender, etc.), fungi of musty food, ergot, etc.

Internal Causes. Among these are all conditions that induce stasis in the capillaries or lymph vessels, active hyperæmia, exudation, depilation, profuse perspiration, shedding the coat, exposure to cold, chill, etc., sudden access of warm weather, poor and insufficient or rich, stimulating food, cotton seed meal, indian corn, buckwheat, purple clover, animal food (in dogs), spiced food, food spoilt by wet and cryptogams, indigestions, gastric and intestinal fermentations, hepatic disorders, renal disorders with imperfect elimination, blood disorders, and nervous disorders which entail vaso-motor changes. Early age predisposes to some affections (variola, warts); old age to others (eczema,). A nervous temperament in horses favors the drier eruptions (pityriasis), a lymphatic temperament the exudative (grease, canker, moist eczema). A hot, moist season favors most skin affections (eczema, acariasis, etc.), dry insolation others (erythema) and cold still others (chillblaines, frost bite, chaps, etc.). Some eruptions are at first summer troubles, disappearing on the advent of cold weather, yet in time the predisposition increases, or the inflamed skin becomes less resistant

and the disease becomes permanent. A marked predisposition in certain animals, appears to inhere in the constitution and proves hereditary in the family (Blain, Lafosse, Cadeac). Nervous causes have not been satisfactorily traced in the lower animals, yet the dermatitis of the face and neck caused by the experimental lesion of the cervical sympathetic or its connecting ganglia shows clearly enough how any portion must be affected by disorder of its trophic or vaso-motor nerves. Charcot found that experimental lesions causing inflammation of the spinal cord, led to trophic changes and finally gangrene of a corresponding part of the skin. Babesiu and Israï injected oil of mustard in one side of the cord, in three dogs, and found in several days a vesicular eruption and atrophy of the skin on the same side, a result they supposed of the myelitis.

DIAGNOSIS OF SKIN DISEASES.

Diagnosis. Clip or shave skin. Examine in warmth : skin and mucosæ, where uninjured by rubbing, moisture, dryness, color, odor, discharge. Soapy wash. Exudation into skin : pliancy : rigidity : eruption : tenderness, itching, history, association, feeding, watering, exposure, housing, harnessing, driving. Coincident disease. Prognosis. Microbian dermatosis, parasitic dermatosis, external irritants, ingested irritants, toxic systemic products, constitution, renal disease, movement of joints, harness.

The thick hairy covering of animals, and the vicious energy with which they often rub, scratch and bite themselves, thus turning simple into extensive and severe lesions, interfere seriously with a satisfactory diagnosis. The following precautions are usually demanded :

1st. Clip the animal close to the skin to allow of careful examination. In some affections, this may be dispensed with, but as a rule it should be followed. What appears to be a circumscribed eruption may be shown to be general, or at least extensively diffused over different regions. Or what was shown only by scurf or scab may be seen in its earlier and more characteristic stage as erythema, papule or vesicle. It may even be desirable to shave the affected part, care being taken, not to slice off the characteristic papules, etc.

2d. Make the examination in a clear day in full sunshine if possible. In dark, cloudy weather, and in dimly lighted stables it is impossible to identify the different lesions. Artificial light is very unsatisfactory. Warmth, as in sunshine, or in a warm day or room, increases any itching and the cutaneous circulation and congestion, and renders more lively and active the animal parasites that may be present. These may be found in the surface scrapings taken in warmth, and not at all if taken in cold. A hand lens will assist in the discovery of the larger parasites, while for the smaller ones the microscope must be employed.

3d. Examine carefully all parts of the skin and even the visible mucosæ, estimating whether any lesions of the latter indicate extension from the skin, by proximity, or a general constitutional affection. Scrutinize particularly such parts as have not been abraded by mechanical injury—those which show the primary character of the lesion. Is the affected portion of the skin dry or moist? Some eruptions like impetigo or grease are always moist, others like pityriasis or dry eczema are habitually dry apart from mechanical injuries. Ascertain the color, odor and consistency of any discharge. It may be a limpid or reddish serum in grease, honey like in impetigo, oily in farcy, greasy in swine-plague. The odor is fœtid in grease, canker and thrush of the frog, cheesy in variola, and mousy in favus.

4th. To learn the true nature of the eruption a warm, soapy wash may be essential to remove scurf, scab, and other encrustations.

5th. Note the depth and extent of the skin lesions, the thickening of the skin, its pliancy or rigidity, its adhesions to subjacent parts or free movement upon them, whether it is contracted into folds or ridges, the degree of congestion, the nature of the eruption, uniform congestive redness, papule, vesicle, pustule, squama, sore, ulcer, nodule, slough, etc. Are the individual lesions isolated or confluent?

6th. The presence of itching and its degree are important data. Pruritus is always excessive in ordinary acariasis, marked in eczema, phthiriasis, and some neuroses, and very slight in a number of skin affections (pityriasis, ringworm, grease, thrush, contagious acne). The abrasions and sores caused by rubbing,

scratching, etc., will usually give a key to the degree of pruritus, and handling the part will render the condition evident.

7th. The history of the case is always important. Is it chronic or acute? Continuous or intermittent? Associated with any special conditions of proximity to other diseased animals, to special feeding, watering, exposure, housing, harnessing, driving, which might account for it? Did there coincide with its eruption any indigestion, gastric or intestinal, or any hepatic, urinary or nervous disorder on which it might be dependent?

Prognosis. This is subordinate to the nature, causes, course, duration and complications of the disease.

Microbian dermatosis (variola, aphthous fever, rouget) usually follows a rapid course and recovery is perfect with some measure of immunity.

Parasitic dermatosis (acariasis, phthiriasis,) is liable to have serious secondary results (infection to man or animals, loss of wool, tender skin), and to run a chronic course.

Maladies from external irritants (chafing, caustics, traumas, vegetable, or animal irritants), do not tend to chronicity and are often promptly curable.

Maladies due to ingested irritants (urticaria, distillery waste eruptions), also tend to recovery when the source of irritation is cut off.

Maladies due to toxic products of the system will be obstinate or incurable, in ratio with the incurability of the causative factor. Those due to the absorbed products of a simple indigestion, will tend to terminate with the removal of the cause, while those dependent on chronic and perhaps irremediable disease of the digestive organs, liver, or kidneys will be correspondingly inveterate or incurable.

Maladies due to a constitutional vice, in sanguification, nutrition, innervation, etc., are likely to be irremovable or only temporarily curable.

Burns and some other skin diseases are liable to become complicated by renal embolisms, albuminuria, indigestions, etc., which may render the skin affection inveterate or incurable.

Dermatitis on the folds of articulations or on the seats of harness, are sustained by the local irritation, and may necessitate long rest, or abstention from work requiring the use of such harness.

All dermatites are liable to show special features of inveteracy, or amenability to treatment according to surrounding conditions—hygienic or otherwise.

GENERAL PRINCIPLES OF TREATMENT OF SKIN DISEASES.

General and local. Diet : wholesome diet following laxative : restricted, generous, vegetable. Rest, avoiding congestion, perspiration, friction, stretching, pressure. Cleanliness. Diuretics. Purgatives. Tonics. Alteratives. Sulphur. Antimony. Phosphorus. Calcium sulphide. Pilocarpin. Baths, tepid, warm, soapy, alterative. Emollients, simple, medicated. Drying powders. Protective films. Stimulating and antiseptic applications. Parasiticides. Caustics. Counter-irritants. Bandages.

These must be general and local, and the first hygienic, dietitic and medicinal.

The **diet** is especially important in eruptions due to poisons such as green food, distillery refuse, silage, roots, ergoted or smutty food, musty fodder, irritant plants in hay or grain feed, buckwheat, etc. In many cases a change to sound fodder and a laxative to clear the alimentary canal of the irritant, may be all that is required. In cases where the feeding has been parsimonious, a judiciously gradual change to a generous diet may be required. Again when the feed has been unduly rich, or spiced as in the patent food for stock or the table leavings for dogs, a plainer, simpler and less exciting diet will be called for. Indigestions, urinary and hepatic disorders due to diet may be often corrected by a more judicious ration.

Rest is a most important element in horses and hunting dogs. When pressure of the harness keeps up the irritation, or when active movement reopens cracks in the tense rigid congested skin of the heel, carpus or tarsus of the horse, the parts must be kept quiescent. When on the other hand chaps and fissures are caused and maintained by *stocking*, the patient may do much better with exercise. In skin congestions which are aggravated by work and increased cutaneous circulation, rest is imperative.

Cleanliness is no less imperative. Many cases are started

and maintained by filth on the skin and in the air of the stable and hence sponging, currying, brushing, rubbing, are directly therapeutic. Yet care must be taken to avoid irritation where the skin is tender. In the sensitive heels of the horse congestion, chaps, and *stocking* are often determined by washing in ice cold water and leaving to dry uncovered, in a draught of air, or by washing with common laundry soap having alkali in excess. Even tar soap will sometimes keep up the trouble in a specially sensitive skin. Apart from such exceptional conditions, thorough grooming is commendable, not only in cleaning the skin, but in improving its circulation and nutrition.

Diuretics are often beneficial in eliminating from the system the irritant products generated from disorders in sanguification, digestion, urinary secretion and hepatic function, as well as those that are derived from the cutaneous disorder. They tend further to reduce any existing fever, and to cool and relieve the burning integument. The alkaline diuretics are often very useful.

Purgatives act in a similar way and are especially indicated in cases due to ingested irritants, and in such as depend on morbid products of gastro-intestinal or hepatic disorder. In many acute attacks these may be said to be almost specific in their action as in urticaria, and in the eruptions due to distillery products or green food.

Tonics are often called for to correct dyspepsias, to improve the general health and vigor, the sanguification and nutrition in weak and debilitated conditions. Iron, cod liver oil, bitters, quinia, quassia, calumba, gentian, nux, are often of value in such cases.

Alteratives. Arsenic may be said to act as a tonic with a special tendency toward the skin where it affects the epidermis and epidermic products and is applicable to many subacute and chronic disorders, as psoriasis, acne, dry eczema, and pemphigus. It has been further supposed to be most useful in superficial lesions, and in those due to a neurotic origin, from the known operation of arsenic on the nerves. It is little suited to acute skin diseases, and though often valuable is not to be trusted as universally applicable.

Sulphur is often useful as a laxative, but also as a stimulant to the cutaneous secretions when these are impaired.

Antimony is similarly a cutaneous stimulant and is sometimes useful in chronic inactive conditions.

Phosphorus has been found useful in obstinate cases and probably acts on the nerve centres in improving nutrition of the integument.

Calcium sulphide is sometimes useful with free secretion from the diseased surface, but its action is somewhat uncertain.

Pilocarpin operates by securing free secretion from the skin as well as from the various mucosæ, and seems to benefit by elimination, as well as by modifying the cutaneous functions and nutrition.

Local Applications. Baths may be placed foremost among these. Cleanliness is a prime necessity in treating skin disease. Tepid or warm water is especially required in acute disease in sensitive skins. In chronic cases with accumulation of scabs a soap wash following a 24 hours inunction with oil or lard may be demanded, but as a rule castile or other non-caustic soap should be used. In certain cases the baths may be advantageously medicated, as with calcium sulphide, potassium sulphide, salt, alum, tannic acid, tar, creolin, lysol, cresol, chloro-naphtholeum, arsenic, mercury, etc. The water alone is, however, of great value in soothing and moderating inflammation, softening and dissolving scabs and epidermis, and relieving the dryness and rigidity.

Emollients are used for the same end as calmatives, and relaxing and protective agents. Fatty bodies occupy a front rank, the bland vegetable and animal oils being not only soothing but nutritive (cod, lard, olive, cotton, almond, linseed, rape, pea nut, lanolin, neats foot and goose oil). Care should be taken that these are pure and in no sense rancid. Vaseline or petrolatum are free from the risk of rancidity, yet it should be free from contamination unless a stimulating action is wanted. Glycerine often used as an emollient has the disadvantage of drawing water from the surface and of actually irritating some sensitive skins. Glycerol made with glycerine and starch is more soothing. Glycogelatine made with glycerine 5, gelatine 3, and water 9, is very emollient and protective. This can be made the basis of astringent, sedative and antiseptic preparations by adding zinc oxide, lead acetate, chrysarobin, salicyclic acid, tannin, sulphur, oil of birch

or of tar, etc. An excellent emollient paste is compounded of zinc oxide and vaseline one-half ounce of each, salicylic acid, ten grains. Oleate of lead is an excellent sedative application in irritation or pruritus.

Drying powders are found in starch, talc, magnesia, zinc oxide, lycopodium, bismuth oxide, boric acid, iodoform, aristol, salicylic acid, tannin, and, above all, magnesia carbonate. A slight addition of morphia sulphate will render them analgesic. Tar in zinc oxide or bismuth will secure antiseptic and stimulating qualities.

Protective films for irritable surfaces may be had from collodion, or from a solution of gutta percha in chloroform 1 : 10 (traumaticin).

Stimulating and antiseptic applications are found in tar or oil of tar in suitable excipient and of a strength suited to the case, oil of white birch, oil of lavender, oil of cade, oil of cashew nut, oil of juniper, oil of hemlock, Canada balsam, balsam of Tolu or Peru, creolin, lysol, cresyl, creosote, carbolic acid, chloro-naphtholeum, etc. Ichthyol, of great value in chronic affections, may be used in oil or vaseline (5 : 100), or in the form of Nuna's varnish : Ichthyol 40, starch 40, concentrated albumen solution 1 to 1½, and water 20. Add the water to the starch, then rub in the ichthyol and finally the albumen. Resorcin is a useful stimulant, alterative, and antipruritic (1 : 30 alcohol and oil).

As **antiseptics** and **parasiticides**, in addition to the above, are alpha- and beta-naphthol, iodized phenol, chloral camphor (rub together till they form a clear fluid), phenol camphor (add camphor gradually to the melted phenol crystals), mercuric chloride, cupric sulphate and silver nitrate. Potash (green) soap, medicated or not with tar or other agent, is of great use in many chronic affections. The phenol combinations are all more or less anæsthetic, and therefore sedatives and antipruritic. Quassia, Stavesacre, tobacco, etc., are of great use in parasitisms though not antiseptic. Sulphur fills both indications, and is a bland generally applicable agent.

Caustics (silver nitrate, antimony chloride, electric or thermo-cautery) are useful in luxuriant granulations, hyperplasias, and often in excessive secretion, or on infected surfaces.

Counter-irritation over the vaso-motor centres, is often of value, when the distribution of the eruption coincides with that of particular nerves, and indicates a nervous element in the causation.

Bandages of various kinds may be demanded to afford support in threatened dropsical effusion or excessive granulation or hyperplasia, to protect the surface against outside infection, to confine volatile applications to the affected part, and to prevent injury from biting, licking, scratching, or rubbing.

For the same reason it may be desirable to employ a muzzle, beads on the neck, tying to two opposite rings by short halters, hobbles, or other means of restraint.

ERYTHEMA. ERYTHEMATOUS DERMATITIS.

Definition, congestion, heat, redness, tenderness without eruption. Sheep: swine: dogs: white horses and cattle. Causes: slight irritants. Symptoms: congestion momentarily effaced by pressure, may go on to a distinct irruption.

Definition. Congestion of the papillary and adjacent layers of the skin with heat, redness, and tenderness, or a diffuse superficial inflammation with some superadded swelling.

Genera susceptible. The affection is seen in sheep, swine, dogs, and in white horses and cattle or on white parts of the skin. It is not readily recognized on pigmented parts.

Causes. The action of any slight irritant: pressure, friction, brushing, currying, blows, vesicants, rubefacients, stings, parasitism, radiant heat, intense sunshine, cold (reaction), storm, plunging in cold streams when heated, feeding on stimulating agents, notably buckwheat.

Symptoms. On white skins there is a uniformly diffused redness, without papule or other eruption, and the color may be momentarily effaced leaving a perfectly white spot, made by the pressure of the finger. The affected part is warm, tender, and it may be, itchy. It may be but the first step of a distinct eruption of another kind, such as variola, vesicles, papules, pustules, but then the affection takes a different name. It has been named according to its seat, cause and nature as follows:

INTERTRIGO. INTERTRIGO OF CHAFING.

Causes: friction, inside thighs, side of scrotum or mammæ, inner side of elbow, between digits on clipped heels, under harness. Dried sebum, sweat, dust, clay. Pricks with stubble. Symptoms: lameness on starting, abduction, straddling, knuckling, steps on toe. Treatment: abate cause, cleanse, rest, dusting powders, zinc, lycopodium, magnesia, starch, bismuth, alum, lead, morphia, surgeon's cotton. Carron oil, vaseline, antiseptics.

This occurs where the folds of skin come in contact and rub on each other as between the thighs or beside the scrotum, sheath or mammæ in fat horses and other animals, on inner side of the elbow, between the digits in ruminants, on clipped heels in horses, and under harness. Irritant perspiration and sebaceous matter dried on, and mixed with more or less gritty or septic road dust contribute to it. Drying of clay and mud in the cleft of the frog, or in the interdigital space of ruminants is a common cause, also pricking with stubble.

Symptoms. These are most marked when the animal has been standing with the raw surfaces partially dried and adherent. After moving for some time, and when the surface has been moistened by the exudate there may be little lameness. Until then he moves hesitatingly and stiffly, with the legs abducted or in case of the hind ones straddling. With intertrigo of the pastern or frog, the horse stands on the toe or with the fetlock knuckled forward, and avoids as far as possible a full extension. The same is true of cattle with interdigital intertrigo.

The affected part is hot, perhaps swollen, red, damp and exceedingly tender. Under renewed work, lameness disappears, but becomes worse on standing, and an extensive exudation may occur subcutaneously. If this becomes infected it may result in severe and even destructive lesions, but it usually remains simple and proves readily responsive to soothing and protective treatment. It is most amenable to treatment in dogs and meat producing animals of which no work is required. On the contrary the latter when travelled long distances on foot may suffer severely.

Treatment. First abate the cause. With castile soap wash

from the affected part the dried accumulations of sweat, sebum, dust and other matters, remove clay from frog, interdigital space or heels. Rest until the congestion and tenderness subside. Drying applications in the form of dusting powders are usually best: zinc oxide, lycopodium, magnesia oxide, may be dusted in freely after the affected part has been thoroughly exposed and dried. In the absence of these use gloss starch, corn starch, farina or white bismuth. To one or other of these may be added a little alum, lead acetate or morphia. The addition of a layer of surgeons' cotton is useful, if in a place where it will be retained. Of liquid applications the veterinarians white lotion (zinc sulphate and lead acetate, of each 1 oz. water 1 qt). is one of the best, being at once sedative and astringent. It may be applied on cotton. Astringent preparations with glycerine are useful but glycerine has the serious drawback of attracting moisture and increasing the secretion when drying is desirable. Carron oil (equal parts of lime water and linseed oil) is most effective and free from this objection. Vaseline alone or medicated with zinc oxide, lead acetate, alum or tannic acid may be resorted to when dusting powders fail. Morphia and camphor have been added when itching is violent.

In intertrigo of the frog or interdigital space the danger of infection from the floor or road is so great that the demand for antisepsis must overcome other considerations. Calomel freely applied to the surface, previously cleansed and dried, is most successful. It may be bound in place by a pledget of cotton and bandage. In other cases alum 5 parts, copper sulphate 1 part, or carbolic acid and tar may be bound to the part.

ERYTHEMA CALORICUM: SOLAR ERYTHEMA.

WHITE FACE AND FOOT DISEASE IN HORSES.

England to Africa, on white skins, especially of face. Symptoms: local congestion, swelling, itching, desquamation, during extreme heat: in worst cases fever, dullness, inappetance, costiveness, diarrhœa, head constantly moving, rubbing, vesicles, pustules, excoriations, cracks, fissures, septic ulcers in nose and mouth, submaxillary and pharyngeal swellings, recovery with dermal thickening and tenderness. Diagnosed from petechial fever by the itching, and the absence of petechiæ. Prevention: breed solid dark colors, avoid white skinned in hot climates or work in shade, away from hot winds; sunshade; avoid friction and wetting in sunshine. Treatment: shade, astringents, cool irrigation, vaseline, zinc ointment, lamp black, antiseptics. Tie so as to prevent rubbing.

This has been observed in different latitudes from the coo climate of Great Britain, to the burning suns of Africa, though it reaches its highest intensity in the hotter regions. The parts to suffer are those that are devoid of pigment, as the white star, snip or blaze on the face, the white feet or legs, the white areas in the piebald, and the whole surface in the albino. The white face, however, suffers more than the white legs, apparently because of its more constant exposure, the absence of shadow from the trunk, and the delicacy of the skin and fineness and thinness of the hair.

Symptoms. In slight cases there may be no constitutional disorder, only redness, itching, swelling and subsequent desquamation of epidermis on the white portion of the skin, which may prove persistent so long as hot weather lasts and recover on the advent of cold.

The more violent cases seen in warmer climes, may be heralded by febrile reaction, dullness, prostration, inappetance, constipation and sometimes diarrhœa. The head may be kept in constant movement, the itchy white parts being rubbed on any object within reach, and the limbs are stretched, the front ones forward and the hind backward, with a tendency to rub them with the nose or foot. The skin at first red, becomes later swollen, covered with vesicles which pass into pustules, burst

and discharge. Meanwhile the subcutaneous connective tissue is infiltrated and gravitates toward the lowest parts, causing extensive submaxillary swellings and stocking of the limbs. In its worst forms it may go on to necrosis and sloughing, but more commonly the affected part becomes raw, excoriated, cracked and fissured. Sometimes the pituita or even the buccal membrane becomes involved, with muco-purulent discharge. In other cases the absorption of septic products causes inflammation and swelling of the submaxillary or pharyngeal lymph glands. Under favorable conditions, the secretions dry, the sores heal and the exudate is in great part absorbed, but there usually remains some thickening of the affected parts and a diminished vitality of the skin, which renders it morbidly sensitive to sources of irritation.

Severe cases might be mistaken for petechial fever, but there is much more pruritus, and there is an absence of the petechiæ, on the mucosæ, and of a tendency to the extension of the disease far beyond the patches of white.

Treatment. Prevention. This malady should be warded off by breeding or selecting for warm, sunny climes, animals of a solid color, and discarding all with white patches. Animals bred in a cooler climate should not be suddenly transferred to a hot one. When the animal with white face or feet is found in the hot sunny climate, it should be devoted as far as possible to work in the shade (indoors or in mines), or its white patches should be protected against the full unmitigated rays of the sun, and the hot winds. Sun shades are useful or in their absence leafy branches fastened to the bridle so as to protect the face. It is further important to avoid the friction of harness on the susceptible parts, or wetting of them when in the full glare of sunshine. Another obvious precaution is to keep the white patches well covered with lamp black.

When attacked the animal must be placed under cover and eruption treated with cooling astringents, constant irrigation with cool water, or lotions with acetate of lead, tannic acid, alum or sulphate of zinc. When the skin is dry and rigid it may be treated with vaseline, alone or with zinc oxide, lamp black or any one of the astringents above named. Open sores may be treated like ordinary wounds, tense engorgements may

be drained by punctures followed by antiseptic dressings, and abscesses may be opened and evacuated.

During the treatment the patient should be tied short to two sides of the stall, and other measures taken to prevent him from rubbing or otherwise injuring the affected parts.

BUCKWHEAT ERYTHEMA : FAGOPYRISM. WHITE SKIN DISEASE.

Form of white face disease, with irritating ingesta, buckwheat, etc., occurs from dried products, no insects ; growing potatoes ; sunshine ; idiosyncrasy. Symptoms : as in white face disease : in winter itching and rubbing : in summer may go on to nervous symptoms. Treatment : stop feeding buckwheat. Give laxative and diuretics. Local treatment as in white face disease.

This may be held to be but a form of the last named affection, in which, however, certain irritating ingesta (buckwheat, maize, wheat), are essential factors in addition to the white skin and strong sunshine. It is seen only on white skins or the white portions of parti-colored skins, while the blacks, browns and other colors usually escape. Black breeds of hogs (Essex) escape under the same feeding and exposure, as do solid colored horses of the darker shades. Of the different food factors, buckwheat (Polygonum fagopyrum, persicare, etc.), is the most to be feared, and the poison seems to be inherent in all the products (green vegetable, dry seeds, bran and straw) and is not destroyed by cooking. Buckwheat cakes sometime produce erythema in man. This excludes the idea of the transfer of a living cryptogam to the skin, though not the theory of pathogenic products of the fungi. The invoking of bee stings and the bites of insects, which are strongly attracted to the buckwheat, is untenable because the affection occurs from the dried seeds, bran and straw, and has been known to break out weeks after the buckwheat was withdraw from the ration.

In addition to buckwheat, maize and even wheat when liberally fed have been known to cause erythema. Hemminger records a similar outbreak in horses working among growing potatoes.

In addition to the food, clear sunshine is essential and an individual idiosyncrasy. All animals, though equally exposed are not equally attacked.

Symptoms. These do not differ materially from those of the white face and foot disease already described. There are intense redness and tumefaction of the white skin or the white portions, showing prominently in the delicate parts (ears, eyelids, lips), with violent itching, rubbing and sometimes vesicles with yellowish contents, followed by sores and scabs. In the winter season there may be itching and rubbing only. In summer it may become erysipelatoid and extend to the mucosæ, of the respiratory and digestive organs, with hyperthermia, nervous excitement, vertigo, turning in a circle and even spasms and convulsions.

Treatment. This is essentially the same as for the white face and foot disease with this additional that the buckwheat or other offensive ingredient must be withdrawn from the ration, and what remains in the bowels must be expelled by saline or other laxative. Cooling diuretics must follow to eliminate irritant matters that may have been absorbed.

ERYTHEMA FROM IRRITANTS.

Irritant agents of all kinds, even if caustic or pustulant in concentrated forms, may be so diluted as to cause simple erythema. Among chemical agents we have ammonia, lye, caustic acids and salts, iodine, carbolic acid, the various tar products, kerosene, aniline, chloroform, ether, formaline, etc. Of thermic irritants may be named solar heat, fire heat, steam, hot water, hot air, and congealation (chillblains, freezing) the latter being especially common in the digital region when salt has been applied to snow or ice. Vegetable irritants include mustard, capsicum, moulds, euphorbium, rhus, milkwort, the volatile oils, etc. Among animal irritants must be named cantharides, mylabris, potato beetle, insect stings, venom of the irritated toad, liquid discharges from the kidneys or bowels, and discharges from open sores. Even the escape of tears upon the face will cause erythema.

The *symptoms* in all such cases will vary with the potency of the factor and the susceptibility of the skin. Beside the common symptoms the color and odor of the skin will often yield valuable diagnostic indications. There is the white of muriatic acid, carbolic acid or zinc chloride, the black of silver salts or sulphuric acid, the yellow of iodine, chromium compounds and nitric acid, the well known odors of ammonia, iodine, chlorine, carbolic acid, tar, essential oils, chloroform, ether, formaline, kerosene, mustard, etc.

Treatment is that of simple erythema after one has applied the appropriate antidote to the particular irritant. For the alkalies and carbolic acid use vinegar, for the acids oxide or carbonate of magnesia, chalk or lime water, for iodine and chlorine weak alkaline solutions, for rhus, ammonia or other alkali, or potassium permanganate, and for insect stings and animal venoms, alkalies or permanganates. The caustic salts may demand milk, white of egg, or some other albuminous solution. When no chemical antidote is available, the first step is to wash off the offending agent, and then treat the erythema.

DERMATITIS.

Under this head may be included a class of inflamations of the skin, due mainly or entirely to external causes, not essentially parasitic, and attended by distinct eruptions (papular, vesicular, pustular, etc). Many of these are but an advance beyond the simple erythematous inflammation, and coincide with it, the one condition being present at one part of the diseased area, and the more advanced and severe lesions at another part. Bearing in mind the frequency of this connection, it will be unnecessary to dwell at length on this class of affections.

Dermatitis Traumatica. Dermatitis from Mechanical Injury. Besides the veritant factors already named as causing traumatic erythema, we must here recognize contusions, overreaching, treads, interfering, bruising and chafing by harness, girths, circingles, hobbles, ropes, traces, twists, and blows with whip, club, chain or rope. Here the source of injury must be re-

moved, by attention to shoeing, harness, etc., and the lesions treated according to their gravity as simple wounds.

Dermatitis Venenata: Dermatitis Calorica. These, like the corresponding erythema, come from contact with irritant plants or animals or their products, from excessive heat or cold, and may show any grade of eruption or even gangrene and sloughing. Apart from the washing off of the irritant or venomous matter and the application of alkalies or permanganates in the case of the latter, these must be treated like ordinary sores. In burns, especial value attaches to solutions of sodium bicarbonate, and preparations that will exclude the air. Vesicles may be emptied by puncture and the part covered with cotton soaked in carbolated sweet oil and lime water, or carron oil (linseed oil and lime water) may be freely applied, or a resorcin solution (2:100 water), or a thick varnish of linseed oil and litharge, equal parts, with 5 per cent. of salicylic acid, melted and painted over the surface. A layer of surgeon's cotton covered by a rubber bandage where admissible, will complete the dressing. In the process of healing, skin grafting may be required. Thin slices of cuticle are placed in the center of the granulating surface, or at intervals and carefully bound in place.

When a part has been frozen the usual method is to recover circulation slowly by rubbing with cold water or snow. The parts are then treated by astringent and antiseptic dressings. If the skin sloughs use antiseptics until it separates, and then treat like an ordinary sore.

Chillblains may be treated with a mixture of sweet oil, 5 ozs., oil of turpentine ½ oz., Aqua Ammonia ½ oz., oil of peppermint 1 dr., or powdered camphor 10 grs., Peru balsam 20 drops, linseed oil 2 ozs. Nourishing food and a course of iron should be given.

Dermatitis Medicamentosa. Medicines given by the mouth sometimes cause inveterate skin eruptions. Among these are arsenic, belladonna, bromides, iodides, mercurials, salicylates, tansy, turpentine, tar, and the carminative seeds and oils. The latter are chargeable with many eruptions in live stock fed on patent foods. In cattle treated with iodides for actinomycosis, an universal eruption and desquamation is a common condition.

In all such cases the drug must be withheld, the bowels cleared out by a purgative and the elimination of any remaining irritant products favored by gentle diuretics.

ECZEMA. A BOILING OUT. A PUSTULE.

General method of eruption. Successive advancing lesions. Definition. Causes: usual factors and special susceptibility.

This term, standing for what boils out, has long been applied to vesicular eruptions on the skin, but inasmuch as the inflammation rarely stops short with vesiculation, but usually in part at least goes on to more advanced lesions, it must be held to include in many cases erythema, papules, vesicles, pustules, crusts, desquamations and erosions. All of these may co-exist or succeed each other in the same subject, so that considerable latitude must be allowed to the name to cover all parts and stages of the same attack. Dermatologists have defined eczema as a non-infectious inflammation of the skin with multiform manifestations, but recent observations would indicate that it may at times, at least, be contagious, and micro-cocci have been found in the serum of the vesicles, while the very occurrence of pus must virtually imply the existence of a bacterial infection. Doubtless different diseases pass under this name in the different genera and species, and even in the same variety of animals, yet until we learn to discriminate sharply the one from the other, it is convenient to consider the whole as a kindred clinical group, if not a pathological entity.

Definition. An acute or more frequently, a chronic inflammation of the skin and sometimes of the mucosæ, characterized by itching, erythema, papules, vesicles, serous or sero-purulent exudation with squama or crusts and loss of hair, and usually largely due to an internal cause. The exudative condition has suggested a catarrh of the skin.

Causes. These are the usual causes of skin disease, local and general, together with a special susceptibility, under which, what are ordinary irritants produce this characteristic disease. Many local irritants can produce eczema, but again it is often the case

that these factors will operate on a given susceptible subject while on another they are without much effect. This susceptibility is called a "dartrous diathesis" by the French writers, while most English and American writers are willing rather to find the hidden cause or causes in the disorder of internal organs (digestive, hepatic, urinary, generative, hæmatic, trophic, infective, plethoric, atonic).

ACUTE ECZEMA IN SOLIPEDS. DORSAL ASPECT.

Head, neck, shoulder, back, under girths, breeching, crupper. Summer. Moulting. Heavy coat. Thin skin. Youth. Symptoms: erect hair, papular groups, hot, thick rigid skin, itching, abrasion, ulceration, encrusting, pustules, white spot and hair. Treatment: laxative, cooling diet, cleanliness, pure air, shade, rest, alkalies, locally vaseline, astringents, dusting powder, anodynes, tar water, creolin, etc.

This shows itself especially on the head, the sides of the neck, under the collar, or saddle, the circingle or crupper, the breeching or general surface. In these cases the profuse secretion of sweat, and the friction of the harness is a marked local factor in its production. It often shows a preference for the summer season, the period of shedding the coat, the heavy coated animal, the animal with white, thin or delicate skin. Youth also predisposes.

Symptoms. There is usually erection or roughness of the hair, and the formation at such points of minute papules like small peas collected in groups. The skin may feel hot, thickened, lacking in pliancy, not to be pinched up in folds, the panniculus is contracted and manipulation shows tenderness. Soon the papules flatten and desicate and more or less violent itching sets in. The patient rubs or scratches himself, causing deep red congestion of the surface or even abrasion, or ulceration. Apart from abrasion the skin becomes covered with crusts or even scales which agglutinate tufts of hairs and dry up and desquamate.

In other cases the eruption advances from the condition of papules to that of vesicles and even of pustules, though finally drying up with the same pruritus as in the papular form.

In either case the affected parts are more or less depilated, red if on unpigmented skin, grayish and scabby or scurfy if on the darker. At times, after recovery, the patch remains devoid of pigment and hairs growing from it are white.

Treatment. It is usually desirable to clear out the prima viæ by aloes or Glauber salts, to resort to a carefully regulated, non-heating diet, to clean the skin of all concretions from sweat or otherwise, to give pure air and shade and to protect the animal from active exertion, profuse sweating and friction by harness or otherwise. In the early stages benefit will often come from the use of alkalies, especially sodium bicarbonate. Locally an inunction with vaseline to soften crusts, and the subsequent removal of these with tepid water, may be followed by some soothing or astringent application, always bearing in mind that what is soothing to one skin is irritant to another. Dusting powders (starch, lycopodion, magnesium carbonate, oxide of zinc, calamine, bismuth) will often do good; soothing lotions or liniments (lead acetate with laudanum, lime water and olive oil; sodium bicarbonate in well boiled gruel of oatmeal or marsh mallow; zinc oxide or sulphate in water or glycerine or as ointment in vaseline, etc, etc). In chronic stages with much squama and pruritus tar water or ointment; a lotion of tar and alcohol; creolin lotion; chloral lotion; or other stimulant application may be used.

CHRONIC ECZEMA OF THE HEAD IN SOLIPEDS.*

Affects face, eyelids, cheeks. Symptoms: papules, vesicles, dry, rigid skin, scurf, glistening, shedding hairs. Treatment: as in eczema; antiseptics.

The cheeks and forehead are the most liable to suffer in this affection, yet the eyelids and the parts below the inner canthus may participate in connection with the escape of tears and the disease of the lachrymal sac or ducts. It has been seen in the young when strangles had merged into skin eruption, but also in the aged and independently of that affection.

*Acute eczema of the heels. See chapped heels and grease.

Symptoms. Following strangles the papules or vesicular eruption may have passed leaving the skin thick, rigid, dry and scurfy. The pigment may be increased and the hairs are usually shed in connection with atrophy of their follicles and rubbing of the itching surface, so that the cuticle is smooth, glabrous and even glistening. In implication of the lachrymal apparatus, there is shedding of hairs beneath the eye or the wet matted condition of those that remain.

Treatment. In strangles use a lotion of silver nitrate or sodium hyposulphite to destroy the local infection. In other cases treat as for ordinary eczema.

CHRONIC MOIST ECZEMA (IMPETIGO) AT THE MANE AND TAIL.

Fleshy neck, thick mane and tail, lymphatic constitution, profuse perspiration, lack of cleanliness, alkaline soaps, plethora, foul stable, pus microbes. Symptoms: itching or tenderness, shedding hair, thinning of mane and tail, skin thickened, ridges and folds, tenderness, moisture, crusts raise hairs from follicles, fetor, sores and ulcers, matted hairs. Treatment: remove general and local causes, cleanse, cool, pure stable, clip, reduce grain; cooling, laxative food, soothing or stimulating applications, zinc oxide, talc, olive oil and diachylon plaster, iodoform, silver nitrate, oil of cade, or of white birch, sulphur iodide, Canada balsam and sulphur, green soap, dusting powders, ointments.

This condition is especially common in horses with a profusion of long hairs in the mane and tail, and in the heavy draught animal with a thick, fleshy neck. In such the skin is very sensitive, and when profuse perspiration soaks the skin, or concretes and decomposes about the roots of the hairs, the local irritation necessary to the production of the eruption is present. A lack of careful grooming is therefore a common cause, yet soap left in washing the mane or tail may be no less injurious. Plethora has its influence in many cases, and the ammoniacal fumes from a wooden stable saturated with excretions are not to be ignored. Finally in cases accompanied by pustular eruption, the pus microbes must be recognized as factors.

Symptoms. There may be marked itching or extreme tenderness of the part affected or in the absence of both there may

occur a gradual shedding of the long hairs, so that an increasing thinness of the mane and tail (rat tail) becomes apparent. The skin covering the affected parts is thickened, inflamed and thrown into ridges and folds, one rubbing against another. The surface feels moist or is covered by crusts formed by the condensation of the moist exudate, and embracing the hairs and drawing them out of their follicles. Beneath the concretions the skin is soaked in the tenacious fœtid liquid discharge. The hair follicles become atrophied in connection with the evulsion of the hairs, or under congestion the hairs stand rigidly erect, and bristly or curly. As the freer secretion abates, the exudate become more purely scally or encrusted, but the skin remains thickened and thrown into folds. Under the inveterate rubbing or gnawing the skin is often extensively abraded and large open sores are formed which are indolent and slow to heal. That matting together of the hairs which has been known as *plica Polonica* is often the result of the disease of the hair follicles and the accumulation of scabs which takes place in this disease, rather than to a special infection like gregarina (coccidiosis).

Treatment. The first consideration must be to remove all general and local causes of eczema, insure perfect cleanliness and good grooming in any case in which these may been lacking, purify the air of the stable if that has been foul, procure a cool environment when that has been too hot, clip the patient if habitually soaked with perspiration by reason of a heavy coat, suspend or moderate the work if that has been too exacting, withhold a heating grain ration (corn, buckwheat, barley, wheat, peas, beans), and furnish cooling, laxative, easily digested food. In the cases before us the acute, irritable stage has usually passed, so that the more stimulating applications may be safely used, yet in many old standing cases a fresh eruption may have taken place, which would demand for a time the most soothing applications only. Apart from such cases the more stimulating dressings are applied at once.

The affected surface is exposed by clipping or shaving off the long hairs, thus at once removing a source of heat and irritation and allowing of the direct and thorough application of the dressing. Among the astringent and stimulant applications oxide of zinc ointment and benzoated oxide of zinc are among the sim-

plest and least likely to irritate, but the stronger applications can usually be borne. The Lassar paste consists of two parts each of finely powdered talc and zinc oxide, four parts of vaseline and three per cent. of salicylic acid. Oxide of bismuth may substitute the zinc oxide. Three parts of olive oil and four of diachylon plaster melted together and stirred until cool, makes another mildly astringent and sedative application. Iodoform 1 dr. to an ounce of vaseline is an excellent agent. A mixture of iodoform and tannin is used as a dusting powder by Friedberger and Fröhner: or silver nitrate solution (6:100) may be used. Tar ointment (1:8) with a little subcarbonate of potassium added makes an excellent application. Oil of cade and oil of white birch may be used in the same way, the latter being the most desirable as a rule. Ammonia chloride of mercury as an ointment (1:10), often acts well and the black wash, formed by the decomposition of calomel with potash is often serviceable. Iodide of sulphur and vaseline (1:10) is often an excellent resort. An ointment of equal parts of Canada balsam and sulphur or iodide of sulphur in four parts of vaseline is often effective. Other valuable preparations are ointments (10%) of ichthyol, naphthol, chrysarobin or pyrogallol. Hebra's *last resort* of green soap is never to be forgotten, the affected skin being thickly smeared with the soap which is left to dry on, and is repeated and rubbed in, for several days in succession. It may seem at first to aggravate the disease by reason of the solution and removal of the covering of the vesicles or pustules and the exposure of a pink sensitive surface, but day by day this improves and the skin becomes smooth and more natural. After a few days of this treatment, it may, if necessary, be followed by astringent or stimulant dressings, or the varied medicaments may be incorporated with the soap so as to form one dressing to be applied from the first. When a healthy action has been once established, all that is required further may be cleanliness, with the use of bland dusting powders or ointments to establish the cure.

CHRONIC ECZEMA OF THE CARPUS AND TARSUS; MALANDERS: SALLENDERS.

Eruption in bends of carpus and tarsus and downward: Causes: lymphatic temperament, constitutional predisposition, deranged internal organs, excessive secretions, modified, congested skin, friction between dermal folds. Symptoms: stiffness, heat, thickening and redness, vesicles or oozing, crusts, erect hairs, shedding hair, squamæ, cracks, abrasions, fissures, subcutaneous engorgement, lymphangiectasis. Treatment: Cleanse, get pure air, regular exercise, non-stimulating food, avoid cold water, mud, slush, caustic soap, lime, sharp sand, foul organic matter. Massage. Light bandages. Bland ointments. Dusting powders. Rest. Iodoform. Starch. Zinc oxide. Boric acid. Magnesia. Bismuth. Lycopodion. Lead. Tannin. Pyoktannin. Stimulating ointments. Green soap. Arsenic.

The bends of the carpus and tarsus in heavy, lymphatic, coarse skinned horses are especially subject to eczema followed by a dense scabby eruption, which in the old farrier's nomenclature was known as malanders in the fore limb and sallenders in the hind. It is not always confined to the joints but may extend down the limb, especially on the back, where the hair is coarser and the skin thicker, as far as the fetlock or even to the hoof.

In the matter of *causation* much depends on the general constitutional state which tends to eczema, and on the torpor or derangement of some of the internal organs the functions of which are interdependent with those of the skin. Something too must be attributed to the freer secretions of these parts in coarse bred horses, to the accumulation of such secretions and of extraneous irritants under the long hair, to the sluggishness of the circulation in the limbs which has to overcome the force of gravitation, and to friction between the thick folds of skin in flexion, and stretching in extension. Swelling of the lower limbs is at once a cause and an effect of the disease.

Symptoms. At the outset the animal may be seen to move rather stiffly, and the skin is found to be hot, thickened and if white reddened. Soon a close observation may detect the eruption of vesicles, or simply an oozing of a yellowish or bloody serum which concretes around the hairs forming an encrusted cov-

ering for the part, holding the hairs erect and bristly, and even lifting them out of their follicles. Cracks also appear in the depth of the fold, leading to a more abundant exudate, and the disease may extend around the whole surface of the limb.

In the more acute cases this may be followed by more or less depilation, dessication and recovery, but too often the condition becomes chronic, the thickened, encrusted or squamous skin continues to exude, crack and cover itself with crusts, under which the decomposing liquids macerate and irritate the exposed cuticle, and engorgment of the whole limb with hyperplasia of the connective tissue and lymphatic plexus and vessels is the result. This hyperplasia of the skin and connective tissue (elephantiasis) is also a common result of lymphangitis.

Treatment. As in other skin affections attention must first be given to removal of the causes. Ensure cleanliness, pure air, regular exercise, non-stimulating food, the avoidance of cold water, melting snow, soapy washes and all other sources of irritation. Deep mud, especially if charged with lime, sharp sand, decomposing organic matter or other irritant, is particularly offensive.

Hand rubbing (massage) of the limbs and evenly applied light bandages are often of the greatest value in dispersing or obviating swelling.

The slighter attacks may be met at the outset by bland ointments or dusting powders and rest from all but necessary exercise. Dressing with iodoform may bring about a recovery in a few days. Starch and oxide of zinc, boric acid, magnesia carbonate, bismuth or lycopodion may give good service. Lotions of lead acetate, tannin, iron sulphate, alum, potassium permanganate or pyoktannin may be used as in other forms of eczema. In obstinate cases green soap followed by stimulating ointments or liniments, tar, oil of white birch, Canada balsam, turpentine and glycerine, oil of cade, etc., will often serve an excellent purpose. In these advanced cases an alterative such as arsenic may be employed.

ECZEMA OF ALIMENTARY ORIGIN IN CATTLE.

STARVATION MANGE. STALK DISEASE. MALT ECZEMA. POTATO ECZEMA.

In low condition : erythema, hæmorrhagic extravasations, or vesicles on tail, lips, fore legs, udder. Trombidium holosericeum. Malt or potato eczema : marc eczema on legs and body. Causes : feeding on marc only, skins, green potatoes, fermenting. Attack in ratio with marc eaten. Worst on new stock, and feeding cattle. Calves have diarrhœa, children eruption. Bean trefoil and milk sickness act similarly. Solanin. Unaffected by boiling. Season. Field. Chlorophyl. Narcosis absent. Is brain adaptable? Other ingredients inoperative. Eczema ceases with change of food : is not inoculable. Symptoms : fever, costiveness, inappetence, red mucosæ, weeping, stringy salivation, debility, emaciation, black diarrhœa. May lie with extended head, grinding teeth, tympany, lethargy, coma. Pig and dog vomit. Abortion. Redness, swelling, stiffness on pasterns : may extend to whole body : exudations : thick crusts : erect or shed hairs : rigid thickened, folded, cracked skin, buccal mucosa may suffer : abscess, sloughs. Mortality slight and up to 20 per cent. Lesions : congestions of small intestine, brain and muscle. Treatment : stop or lessen the marc adding grain : turn to pasture : locally bathe, cold or tepid : lead lotions : dusting powders : tannin : blue stone : creolin : cresol : tar or birch oil : carbolic acid.

The skin of cattle seems to suffer more than that of other animals in connection with the ingestion of poisons. In starved or very low conditioned animals, eruptions are met with which may be in the form of a simple erythema, a hæmorrhagic extravasation in spots, or an eruption on the end of the tail in the form of epidermic concretions or pustules (impetigo). Among the vineyards it is common to find an eruption with papules and vesicles on the lips, fore legs and udder of cows which were fed on the succulent young shoots and leaves of the grape vine. In cases of this disease, Railliet and Moreau have found a great number of the silky trombidium larvæ (harvest bug), and accordingly attribute the affection exclusively to their attacks. The growth of the vine on the warmest and sunniest exposures, the most favorable to the propagation of this acarus, gives much support to this conclusion.

Malt or Potato Eczema. On the continent of Europe where

potatoes are largely used for distillation and the production of starch, herds of cattle are fed often almost exclusively on the refuse or marc, and in such herds an eczematous eruption of the legs and exceptionally of the body is a familiar occurrence.

Causes. The disease has been definitely traced to an exclusive dietary on potato marc, and still more so to the skins, to tubers rendered green by exposure to the sun, and to the distillery potato refuse which has undergone fermentation. Thus 80 litres of the pulp daily without dry food will determine a violent attack in the animal consuming it, while the animal consuming 40 litres has it much milder (Friedberger and Fröhner). It attacks animals living in the best conditions of cleanliness and pure air, and the essentially toxic quality of the cause may be deduced from the fact that newly bought animals, which are not yet habituated to it suffer the most, that fattening cattle are the common victims, while work oxen which perspire more freely and milch cows escape, yet calves fed upon their milk may suffer from diarrhœa and infants from a cutaneous eruption (Johne). The poison it is to be inferred is eliminated in the milk. Similar examples of the protecting of the milch animal by elimination of the poison through the milk are found in bean trefoil (cytisus) which poisons the milk while proving harmless to the goat which yields it, and the poison of milk sickness which is deadly to cattle which are not giving milk, and harmless to the milch cow, yet deadly to those that consume her milk.

The exact nature of the poison is as yet uncertain, and as solanin is the only toxic principle so far discovered in potato, this has been held tentatively to be the essential cause. The amount of solanin in young and germinated potatoes has been given by Cornevin as follows :

	Germinated tubers	Young tubers.
The entire tuber contains	0.21	0.16
The central fleshy part	0.16	0.12
The parings and pickings	0.24	0.18

The toxic strength of the marc is not impaired by boiling, cooking or other culinary treatment, and the same is true of solanin. The toxicity is greatest after the potato has been subjected to germination, or when it has become green by exposure to the sun, and in these conditions the solanin is increased. The

toxicity of the marc is higher in certain years, and in the product of certain fields, than in others, and this is in keeping with the effect of environment in modifying the products of a plant. The increased production of chlorophyl under the action of sunlight is associated with a material increase of the amount of solanin. Until therefore another toxic product can be shown to be the essential cause of this affection the solanin must be charged with this result. This conclusion would be more inviolable if the animals attacked showed other symptoms of solanin poisoning such as narcotism, vertigo, stupor and paralysis, and the absence of these may perhaps be due to the gradual advance of the toxic action, and the progressive immunizing of the animal system. The brain may be able to accommodate itself more readily than the skin.

The other constituents of the potato or of the marc fail to produce the eruption under other conditions: the alcohol in brewers and distiller's grains, the acetic, lactic and butyric acids in the refuse of starch, beet sugar and canning factories, the potash in turnips and other roots, the yeast ferment in brewers' grains. The acarus of foot mange (symbiotis bovis) is rarely present in the affected animal though the eruption in the same situation would strongly suggest its presence and lead to a search for it. Moreover the eczema appears at once in a large number of animals, affecting a large area without evidence of slow and steady progression and disappears with equal rapidity in many cases when the diet is changed. Finally the eczema has not been successfully propagated by inoculation which conveys mange infallibly from animal to animal.

Symptoms. The disease is associated with slight fever, costiveness, impaired appetite, hyperæmia of the mucosæ, epiphora, viscous salivation, muscular weakness, and finally emaciation and black diarrhœa. The gravity of these symptoms varies, being greater when the animals have eaten the leaves and stems, the raw potatoes in their skins, the young shoots and parings, or green potatoes which have been sunned. The animals may lie most of their time stretching themselves out with head extended on the ground, they may grind the teeth, may have pulse small and rapid, tympany, lethargy, coma and even paraplegia but these severe symptoms are exceptional and almost altogether

confined to the cattle of distilleries which receive an exclusive diet of potato marc. In the pig and dog vomiting has been noticed (Cornevin). Pregnant animals may abort.

The local symptoms begin with redness and swelling of the skin around the pasterns, especially of the hind limbs, stiffness and a disposition to lie most of the time; then small flattened vesicles appear, isolated or confluent, which bursting, form extended, raw patches the abundant exudations of which concrete into thick crusts. The hairs stand erect and are abnormally thick at their roots. The eruption may extend to the whole limb, the scrotum, mamma, tail and body at large, so that in severe cases it is practically universal. The skin becomes thick, rigid, hide bound, wrinkled and folded with intervening cracks. As a rule, however, the eruption is confined to the limbs, scrotum, mammæ and tail. In some extensive and persistent cases the buccal mucosa suffers, particularly on the pad on the upper jaw, which shows extensive and irregular ulcers with purulent centre and swollen, congested margin. Abscesses may develop in the skin and subcutem and sloughing of the integument is not unknown.

Mortality is slight as a change of food is usually made and a recovery ensues in a few weeks. Yet Baranski noted 20 per cent. of deaths in Galicia, mostly in old, worn out animals which had been stabled for a length of time.

Lesions. On examination, *post mortem*, there are found hyperæmia and inflammation of the small intestine, some congestion of the cerebral meninges, and a red, bloody condition of the muscular system.

Treatment. The toxic provender must be stopped, or reduced to 20 or 30 litres of pulp daily, supplemented by sound wholesome dry fodder. Marker claims that 70 quarts daily of the potato marc may be given if combined with a fair ration of Indian corn. Turning out doors to pastures usually effects a speedy cure.

Local treatment is rarely demanded but when the irritation is great it may be soothed by bathing with cold or tepid water, lead lotion, glycerine and lead lotion, or by the application of ointments of lead, tar, oil of cade or birch, or carbolic acid. Dusting powders of zinc oxide, starch, lycopodium, boric and

tannic acids may also be employed. Decoction of oak bark or solution of blue stone is often used, also creolin or cresol one part, to alcohol five parts.

It is rarely necessary to use other than the cooling and astringent lotions, yet the persistence of irritable sores, ulcers and crusts must be treated as in other chronic skin affections.

MOIST ECZEMA OF THE PASTERNS IN THE OX.

Causes: hot season, foul stables, streptococcus. Symptoms: sudden attack, red, swollen, warm, tender pastern, vesicles, crusts, scabs, lameness, foot rested on toe, cracks, fissures, interdigital foot rot, shedding hoof, scaly chronic form. Treatment: clean stables and yards, cleanse feet, lead lotion or zinc, phenol, iron or copper. Tar water, tar, creolin, creosote, iodol.

This affection is comparable to the simpler forms of grease or digital eczema in horses.

Causes. It occurs especially in the hot mid-summer season in cattle kept in filthy stables, where the feet and pasterns are kept filthy and the air charged with irritant ammoniacal fumes. A streptococcus is usually met with and may be found in pure cultures in resulting abscesses.

Symptoms. The attack is sudden, the skin around the pastern becoming red, warm, swollen and tender, with the formation of vesicles, isolated or confluent, which rupture and discharge a serous exudate that dries up into crusts and scabs. Lameness is a marked symptom and in bad cases the swelling and pain are such that the foot may be habitually raised from the ground and rested only on the toe. The swollen skin is thrown into folds which rub on each other, and breaks open into cracks from which exudes a serous fluid that macerates and irritates the skin, the heel pad and the interdigital space, so as to determine interdigital foot rot. This may lead to inflammation inside the hoof with shedding of the horny mass, or it may subside into a chronic form with an abundant squamous product.

Treatment should be mainly prophylactic in the direction of cleanliness and abundant litter in the stables, and the avoidance

of pools of liquid manure and of septic mud puddles in the yards and roads.

In the early stages of the affection the pasterns and interdigital spaces should be thoroughly cleansed and covered with a bandage with a weak solution of acetate of lead, or of sulphate of zinc, or carbolic acid, or sulphate of iron or copper. In the more advanced stages tar water or crude tar will serve a good purpose, or watery or alcoholic solutions of creolin, creosote, oil of tar, carbolic acid or iodol. When the horn has been separated from the quick, it is usually best to pare away all such, to bevel the edges so as to make them less rigid and more pliant and to dress with tar water and later to cover with undiluted tar and bandage.

MOIST ECZEMA OF THE TAIL, NECK, CHINE AND DEWLAP OF CATTLE.

Definition. Causes: in work oxen, winter, foul stables; dairy cows on spoiled fodder or maize, wheat, buckwheat, cotton seed, etc. Contagion. Symptoms: skin hot, thick, tender, exuding, matted hair, vesicles, itching, excoriation, ulceration, bleeding, sloughing. Treatment: Soothing. Cleanliness. Pure air. Tepid sponging. Dusting powder. Clip or shave. Calomel with care. Phenol. Creolin. Silver or copper salts. Tannic or boric acid.

This is an acute eczematous eruption of cattle beginning as a congestion and swelling of the skin and advancing to an exudation or secretion which bedews the surface with a sticky discharge, and concretes into scabs and crusts.

Causes. The disease has been mainly seen in work oxen during winter, when kept in close, foul stables and not properly groomed. It is also seen in dairy cows and may be attributed to the indigestion and gastric disorders which come from the ingestion of spoiled fodders, or from a too stimulating diet, such as Indian corn, wheat, buckwheat, barley, cotton seed, and the seeds of the leguminosæ. Lafosse looked upon it as contagious, but Cadeac denies both this and its alimentary origin.

Symptoms. The attack is severe, the skin becoming swollen, hot and tender, especially at the base of the tail, on the neck, chine and forehead. Soon the turgid, congested skin exudes a

somewhat glutinous serous product, which mats the hairs into tufts and exposes the intervening red, excoriated skin, with here and there vesicles singly or in groups. Itching is usually intense and the animal licks, rubs and scratches the affected surface unmercifully. The resulting excoriations and sores add greatly to the severity of the troubles, including ulceration, bleeding and even sloughing.

Treatment. Prophylaxis should be the first consideration, and in the acute stages of the disease, its arrest by soothing applications. Cleanliness, pure air, and tepid sponging, to be followed by a dusting powder of boric or salicylic acid, or a lotion of acetate of lead or sulphate of zinc may serve a good purpose. If the case proves obstinate, the hair may be clipped or shaved to allow of the more direct and thorough application of the dressings. Cadeac especially recommends an ointment of calomel (1 : 10) but this must not be applied over an extended surface, nor must it be recklessly repeated owing to the dangerous susceptibility of the bovine race to mercurialism.

Lotions and ointments of carbolic acid are of great value in moderating the intense pruritus, and a combination of this with lead acetate will often prove quite effective. Lotions, liniments or ointments of tar, oil of cade, creosote, or creolin. When ulcers are present they may be treated by solutions of silver nitrate (2 : 100) or cupric sulphate (2 : 100) or powdered iodoform. When the exudate is excessive, astringent dusting powders often serve a good purpose ; tannic acid and boric acid, with starch or lycopodion.

CHRONIC ECZEMA IN CATTLE.

Summer disease. Depilation. Scaly. Itchy at first. Lesions of bones, red zones representing successive attacks. Alterative tonics indicated.

Megnin records the case of an ox which on three successive springs had a miliary vesicular eruption on the loins and upper walls of the abdomen, which persisted until the advent of cold weather in the fall. The vesicles were followed by an exudate which concreted in solid crusts, enveloping the roots of the hairs which were lifted from the follicles and failed to be renewed, so

that the animal entered on the winter with an appearance of alopecia. The denuded surface was red, shining and covered with a dense covering of lamelliform epidermic scales. In the early stage of the eruption there was moderate pruritus, but when the scaly stage was reached it was neither tender nor itchy to any marked degree. Tar ointments had no effect in stimulating the growth of the hair, and the skin remained bald until the next attack. The second and third years the eruption extended farther, invading not only the trunk, but the legs, and passing through the same successive stages.

The animal was butchered and the shafts of the bones were found to be abnormally red, and showed three concentric rings of deeper brown, manifestly representing the three acute attacks and resembling the concentric rings formed in growing bones when the young animals are fed on madder.

The manifest disorder of nutrition in this chronic skin disease, is an argument for the *treatment* by alterative tonics, such as arsenic, as well as for the employment of tonics and corroborants in general. In such cases the presumption is that local treatment would be useless or nearly so until the general disorder could be repaired.

ECZEMA IN SHEEP.

In anæmias squamous eruptions. In cold rainy weather moist eczema. Salving. Thin wooled. Congestion, swelling, papules, vesicles, scabs, depilation. Recovery with dry weather. Prevention: fold in rainy weather, covers. Pruriginous eczema. Fagopyrism.

The skin of the sheep is so densely covered by wool and so lubricated with its own secretion, that it is little liable to non-parasitic dermatitis, or such as exist are to a large extent overlooked. In internal parasitisms (distomatosis, strongyliasis, etc.), the wool becomes flattened ("clapped") and the skin the seat of a dry (squamous) eczema with scaly accumulations around the roots of the wool.

Moist Eczema, the "*rain rot*" of the Germans is seen in low conditioned sheep which have been left out in the heavy cold rains, and is attributed to the direct entrance of the rain by

the dorsal shed of the wool. In salving sheep it is a great point with the shepherds to avoid opening the way for such entrance, by shedding only at a short distance on each side of the spine, and never directly in the center. Thin wooled sheep are also specially liable to the disease. When the rain enters so as to soak the skin and deeper layers of the wool, it softens and macerates the skin, introduces microbes and favors decomposition and in various ways incites to dermatitis. The skin becomes red and swollen with an eruption of papules and vesicles, and an exudation which concretes in scabs around the wool, which under the constant, accretions from below lifts the wool from its follicles, leaving bare scurfy, or vesicular patches. This appears in different parts of the body beginning in the region of the vertebræ (back, loins, croup), and extending on the shoulders, neck, sides of the chest and abdomen. The disease is rarely inveterate and generally subsides spontaneously on the return of the dry weather. Still it may cause considerable loss of wool and hence it may be desirable to fold the flocks during cold rainy seasons, or, if they must be run at pasture, to cover the back of each with a piece of sacking.

OTHER CUTANEOUS ERUPTIONS IN SHEEP.

A *dry* and a *moist eczema* have been noticed in the sheep, (*pruriginous eczema : impetiginous eczema*) and a moist eczema of the pastern comparable to grease in the horse. *Fagopyrism* also occurs.

ECZEMA IN SWINE.

Secondary skin lesions, macule, vesicles, seborrhœa, crusts. Impetigo of young : cold weather : exposure : filth : spoiled or improper food. Symptoms : eyelids, etc., show itchy, red, swelling, pustules, scabs, erosions, may affect nose or mouth Duration 20 days. Hot weather aggravates. Treatment : cleanse : soapy washes : emollient ointments, astringent lotions, saline laxative, diuretics.

Disease of the skin is by no means uncommon in swine, but it occurs mostly as a manifestation of an acute general malady. Thus in the different specific diseases, caused by microbes,

maculæ in the form of blood extravasations, punctiform or in extended patches are constant phenomena. In some cases this is complicated by a vesicular eruption, or by a seborrhœa and by a dense accumulation of black crust on the surface.

Impetigo of Pigs. Benion and Cadeac describe this as a sporadic affection of young pigs especially, which has been attributed to cold stormy weather, lack of shelter, filthy pens, spoiled food and insufficient nourishment.

Symptoms. The skin of the eyelids and other parts of the body presents itchy, red, hot and swollen patches, which gradually pass into a pustular eruption. The pustules no larger than a millet seed, burst in forty-eight hours, and discharge a yellowish or purulent liquid which concretes around the eyelashes or bristles, and glue the eyelids together. The crusts may increase so as to cover the affected part of the skin by a dense scabby covering which is firmly adherent and when detached leaves a bleeding surface. It may extend to the different mucosæ of the eye, nose or mouth. The disease runs a course of twenty days or less being retarded by the extremes of temperature. During the heats of summer the attendant pruritus is very great and annoying. During convalescence the scabs and crusts gradually detach themselves and drop off leaving the healthy skin covered at first by a somewhat delicate epidermis.

Treatment is confined to cleanliness, soapy washes, emollient ointments and astringent lotions (lead acetate, sulphuric or hydrochloric acid) but no premature detachment of scabs is permissible. Saline laxatives and diuretics are often called for.

VESICULAR IRRUPTION IN PIGS. PITCHY AFFECTION. SEBORRHŒA.

This also affects the young and is characterized by the successive appearance of vesicles, pustules and scabs or crusts. Friedberger and Fröhner associate it with debility from youth, disease or neglect, from articular rheumatism, rachitism, hog cholera, etc., but also as a result of lying on manure, and the accumulation of sebaceous matter and filth of all kinds on the skin.

Symptoms. Among the symptoms of general disorder are dullness, inappetence, prostration and slight fever. There is red eruption with vesicles and even pustules on the early rupture of which the discharge concretes into a black pitchy layer. It may be at first most marked on the ventral aspect of the body, but usually extends to the whole integument.

Treatment. Where it is not dependent on some grave internal disorder, this commonly yields to soapy washes, generous food and a clean pen.

GRANULAR ERUPTION IN SWINE.

Zschokke describes a disease of this kind affecting the ears, back and croup, and caused by a micrococcus in the epidermis and papillary layer of the derma. It appears in the form of patches, often of the size of the palm, showing bluish gray papules which dry up without forming pustules. It runs a chronic course and produces little or no itching.

Treatment would consist in absolute cleanliness, soapy or alkaline washes, and the free use of solutions of the hyposulphites, sulphites, or other antiseptics which are neither irritant nor poisonous.

URTICARIA is met with in swine as already noticed.

SCLERODERMA occurs in boars especially in the region of the shoulders and back.

ACUTE ECZEMA OF THE DOG.

Prevalence and forms. Red Mange. Causes: constitutional, hereditary, races most susceptible, short-haired, delicate skins, 1st and 2d years, flesh fed, overfed, spiced food, secondary to internal disorders, heat, cold, dust, irritants. Symptoms: blush inside elbow, thigh, belly, heat, tenderness, itchiness, scratching, vesicles, abrasions, sores, skin thickens, wrinkles, moistens. Diagnosis: from demodex, distemper, mange. Treatment: change diet, restrict in quantity, from flesh, or stimulating food, one meal daily, laxative, bitters; locally, cleanse skin, antipruritic non-poisonous dusting powders or lotions, starch, magnesia, bismuth; with muzzle, phenol, lead, thymol, thiol, later creolin, oil of cade, lysol, etc. Acute general eczema. Causes as in red mange. Symptoms: Common on head, ears, back, rump, eyelids, lips, scrotum, arms, digits, crusts and depilation, large vesicles, bleeding digits. Treatment.

In none of our domestic animals is this condition so common as in the dog, and of all skin affections of this animal this is the most frequent. As in other animals it may show itself in all forms or grades from simple erythema, through the papular, vesicular, pustular and scurfy or scabby, and all of them may often be seen at the same time in one animal. Yet special names have been given to different forms and localizations and it seems convenient to retain some of these for every day use.

Eczema Rubrum. Red Mange. This form is familiar to dog fanciers as one of the acute types of this disease.

Causes. Among these are recognized a constitutional predisposition, so that the disease appears in successive generations in the same family, without apparent reason for charging the trouble on any particular feeding or management. While not confined to any race or group of races it has been noticed especially in greyhounds, setters, pointers, fox hounds, harriers, bulldogs, St. Germains and braque hounds. It is especially common in dogs in their first and second years, and those that are nervous and lively, with a delicate and naturally dry skin. Again, the dog fed largely on flesh, and above all the house dog fed thrice a day or oftener on highly spiced animal food from the table, or on cakes, rich in fat, is a frequent victim. There is besides that tendency to irritation of the skin which comes from hereditary peculiarities and idiosyncrasy, from diseases of the stomach, intestines, liver or kidneys, from faults in sanguification, nutrition and secretion, agencies that disturb the circulation in the skin, like excessive heat or cold, irritant dust, dessicated perspiration or sebum, overheating and subsequent plunging in cold water. These acting locally may serve to precipitate that which was otherwise imminent from a generally acting cause.

Symptoms. There is first erythema, usually on the inner side of the elbow, or thigh, with redness, heat and tenderness, which soon extend to the belly, breast and inter-maxillary region, but it confines itself as a rule to the ventral aspect of the body where the hair is sparse and delicate, and the skin thin and sensitive. The symptoms are more marked in white haired dogs. The tender skin is more or less (usually intensely) itchy, causing violent scratching with the development of minute vesicles and even open sores. The skin may become moist, thickened and wrinkled.

but is rarely encrusted to any degree. Spontaneous recovery may take place under a change of diet (restricted or vegetable), or an outdoor life in summer with liberal exercise, or the disease may last indefinitely so long as the etiological conditions are unchanged.

Diagnosis. The affection is easily distinguished from demodectic acariasis which attacks a different part of the body, namely, the head, the eyelids, the feet, and the back, whereas, this form of eczema confines itself to the ventral aspect of the trunk. From the eruption of distemper it is diagnosed by the absence of the hyperthermia and catarrhal symptoms of that disease, and by the very small size of the vesicles ; those of distemper are broad, flattened and often have dark colored contents. From acariasis it is differentiated by its confinement to the ventral aspect, in place of attacking the head, ears, neck and back, by the less severe and incessant itching, and above all by the absence of the acarus, and the element of contagion.

Treatment. A change of diet is a prime consideration. It may be in the direction of simple restriction, but usually also in the avoidance of meats that are highly peppered or spiced. A change to vegetable food,—biscuit or mush and milk, is of great importance, but in some animals a little fresh plainly cooked steak or raw lean meat may be essential. In other cases a little beef juice or gravy well skimmed of fat may tempt the patient to eat mush. In the same way it may be necessary to temporize in the matter of meals. Some dogs can be safely put on one meal a day, while for others accustomed to frequent feeding it may be needful to give two and restrict the amount. For the overfed or dyspeptic animal a laxative, at the outset, serves to remove irritating and fermenting ingesta, and to place the stomach and liver, and indirectly, the skin in a better condition for recovery. Any persistent indigestion should be treated in the ordinary way.

Locally it may be requisite to first clean the surface by sponging with tepid water, to be followed by soothing and antipruritic agents, due care being taken to avoid such as when licked will poison the patient. Starch powder, magnesium carbonate, and bismuth oxide may be used without apprehension. The same is true of limewater and to some extent of zinc oxide. When we advance to others we must take the precaution to use a close wire

muzzle, to prevent the ingestion of the agent. Carbolic acid lotion (1–2 : 100) acts as a local anæsthetic, and often materially lessens both licking and scratching. Lead acetate or thymol or both (1 : 100) have a similar action. Thiol 20, glycerine 50, water 50, often acts as well. When the acute symptoms have subsided the more stimulating agents may be employed : Creoline (2 : 100) ; oil of cade 1, vaseline 5 : Canada balsam 1, vaseline 5; zinc ointment, or lead acetate ointment.

OTHER ACUTE ECZEMAS IN DOGS.

Apart from eczema rubrum, the acute forms have been designated according to their seat and the nature of the attendant eruption.

Acute General Eczema. This may be often traced to various causes of irritation local or general : overfeeding, over-stimulating or spiced food, digestive, hepatic, or urinary disorders, irritant dust or inspissated secretions on the skin, hot seasons, over exertion, cold baths when heated, skin parasites and scratching.

Symptoms. The whole skin, or a portion thereof is the seat of pruritus, causing active scratching and on separating the hairs on the affected parts there is found redness, congestion, and swelling with the formation of papules or vesicles, abraded, or moist surfaces, and scales or crusts. These patches are common on the *back*, the *head*, *ears*, *rump*, (**Caudal eczema**), the *palpabræ*, the *lips* (*eczema labialis*), the *interdigital space* (**interdigital eczema**) the *scrotum*, or the *anus*.

Sometimes the formation of crusts and the loss of hairs is to be noted, sometimes the eruption of large vesicles which burst and discharge a honey like fluid (**impetiginous eczema**), sometimes blood escapes from the irritated surface and concretes in dark crusts. The vesication and moist exudation is especially common about the head, ears, eyelids, and rump, while bleeding is especially seen around the claws and in the interdigital spaces in connection with running on rough ground, snow or stubble. The impetiginous form often bears a strong resemblance to vesicles caused by a burn with hot water. The treatment of these differ-

ent forms does not differ materially from that of eczema rubrum, being first dietetic and hygienic, then soothing, and finally stimulating.

CHRONIC ECZEMA IN THE DOG.

Follows acute. Same general causes. Symptoms: skin thickens with papules, vesicles or pustules, scurf, crusts, depilation, surface glossy, abraded, scratched, raw, rough, fœtid, itching, emaciation, exhaustion. Chronic eczema of the back. Fat, old, gluttons. Symptoms: circumscribed patches on back, loins, quarters, tail, intense itching, skin thickened, cracked, raw, encrusted, black, folded, rigid, fœtid, hair broken, erect, shedding. Very inveterate. Chronic eczema of elbow and hock. Causes: friction on summits of prominent bones, filth, infection, predisposition. Symptoms: red, thickened, bare, indurated, calloused skin, cracks, sores, discharge. Inveterate. Chronic dry eczema of head, ears, neck and limbs. Circumscribed area, slow progress, thick, rigid, folded skin, hairless, dry, scaly, moderate itching. Treatment: Fresh eruption like acute form. For old chronic form, stimulating astringents, silver, mercury, copper, boric acid, tannic acid, iodoform: for dry and scaly, ointments of oil of cade, tar, green soap, zinc, cresol, lysol, chloronaphtholeum, sulphur, sulphur iodide, ichthyol, salicylic acid, chrysarobin, naphthalin, naphthol, resorcin.

While acute eczema may recover permanently under hygienic measures alone, yet any case is subject to relapse and the new eruptions may succeed each other so persistently that the affection becomes essentially chronic. Like the acute, chronic eczema may be general or local and be named accordingly.

The same general *causes* as produce acute eczema are operative in maintaining the disease indefinitely. Faults in diet, overfeeding, unhealthy kennels, foul air and surroundings, hot weather, licking and scratching are among the common causes.

Symptoms. Under the continued inflammation the skin becomes thick (on the back it may be double or treble its normal thickness), it has a general angry congested appearance, papules, vesicles and pustules coexist or succeed each other and as these dry up, scales and crusts accumulate. The hair drops off over extensive patches, leaving a somewhat shining skin. What hair remains is largely twisted or broken by rubbing and

scratching. Hypertrophy of the papillary layer is not uncommon giving a rough uneven aspect and feeling to the skin. A common feature is an offensive odor from the affected skin, and which may betray the persistence of the disease when it has been supposed that all eruption has been overcome. While not prepared to follow Cadeac in making this a diagnostic symptom from other skin diseases, yet as an evidence that an eczema is not yet entirely healed it serves a very useful purpose. In old-standing cases the continued irritation, the unintermitting itching, the absorption or circulation of morbid products, and the constant nervous excitement may lead to emaciation, exhaustion and death.

Chronic Eczema of the Back in Dogs. Rodent Eczema is a disease of fat, old, voracious dogs. It appears in circumscribed spots and patches on the back, loins, croup or tail and is marked by inveterate itching, congestion and thickening of the skin, cracking of its surface, bristling, breaking and shedding of the hair, exudation from the surface and its dessication in the form of crusts. These crusts may be black from contamination with dust or blood, and the affected surface is more or less fœtid. The skin may be puckered into irregular folds, thick and inelastic. Not infrequently the malady may remain dormant for some time, only to break out again and again with renewed energy. It is very obstinate and intractable.

Chronic Eczema of the Elbow and Hock in Dogs. This attacks the summit of the olecranon or calcis and is manifestly connected with compression and friction on these parts when lying down, and perhaps with foul and irritating matters on the ground. This need not be looked on as the sole cause but only as the occasion for the localization of a predisposition which was already present in the general system. The skin becomes red, thickened and indurated, the epidermis undergoing hypertrophy to form a callus, in which a few cracks and sores may form, giving rise to a discharge which encrusts the surface and adds to the thickness and induration. The affection is very inveterate.

Chronic Dry Eczema of Head, Ears, Neck and Limbs in Dogs. The dry eczema of the head, neck and limbs is characterized by its slow progress and its restriction in the majority of cases to one or more of these parts. The small

affected patches, have some thickening and folding of the skin, which is usually dry, scaly and largely divested of hair. Itching is moderate only, and the hairs are shed less rapidly than in the encrusted forms.

Treatment. When there has been a fresh irruption it may be be requisite to treat chronic eczema, for a time, after the manner of the acute, so as to avoid any tendency to aggravation of the already existing irritation. A careful regulation of the diet is as essential in the chronic forms as in the acute and in the inveterate types, especially those of a squamous character, alteratives like arsenic are often of value. In the acute stage or during a recrudescence the mild dusting powders (starch, zinc oxide, lycopodium, magnesia bicarbonate, bismuth oxide, thiol) may be applied, or bland unguents (zinc, benzoated zinc, lead, vaseline, glycerine, spermaceti and almond oil, paraffin, wax), or sedative lotions (lead, opiate, thymol, thiol, carbolic acid).

In the more advanced and moist forms astringents and stimulants may be adopted: silver nitrate (2 : 100), applied with soft cotton, mercuric chloride (1 : 1000), or black wash (calomel 1 : lime water 60) care being taken to use a close wire muzzle to prevent licking. Copper sulphate (1 : 100) is at times useful, and boric acid, and tannin may be tried. Iodoform 1 part and tannic acid 5 has a good effect in many cases.

For the dry and scaly forms, and indeed for many of the others, as well, the more stimulating ointments and liniments are called for. Cadeac recommends oil of cade, tinctures of cantharides, or a tar liniment made with alcohol, as a supersedent to produce an active inflammation and displace the unhealthy eczematous one. The agent is rubbed upon the skin and the resulting scabs are left for a week when it is washed off with tepid water and the skin is found healthy or greatly improved. As a rule a second dressing of the tar is then applied. Müller strongly recommends Hebra's treatment with green soap and alcohol (2 : 1) to be rubbed on the affected surface and washed off the following day when all scales and crusts will come off with little trouble. He follows with zinc oxide or lotions of mercuric chloride or silver nitrate. Friedberger and Fröhner use cresol 2 parts, green soap 2 parts, alcohol 1 part; also creosote in alcohol (1 : 10) or in paraffin (1 : 10). Zuill looks upon sulphur iodide as virtually

a specific: sulphur iodide 1 part, sublimed sulphur 7 parts, cod liver oil 7 parts. This is applied once and repeated at the end of ten days, if necessary. Application is made to the whole skin healthy and diseased alike, and rarely requires to be repeated.

Ichthyol is commended by Müller in cases which show great cutaneous thickening with cracks and fissures. It may be made with water (1 : 5) or in glycerine or lanolin of the same strength. Müller combines it with lime water and olive oil and applies it daily.

Other agents in use are salicylic acid in olive oil (1 : 3): chrysarobin in paraffin ointment (1 : 4): naphthalin or naphthol (1 : 10): resorcin in water (2 : 100)

LICHEN. HEAT PAPULES. PRICKLY HEAT.

Horses, cattle, sheep and dogs suffer in hot season or hot stables. Nervous temperament. Delicate skin. Over-driving. Heating foods. Cold water when heated. Unwholesome food. Indigestion. Chronic affections of stomach, liver, kidneys, etc. Symptoms: Clusters of small papules on neck, back, croup, or thighs, crest, tail, exudate concretes, lifts hairs from follicles, depilation in round spots, or patches, abrasions, ulceration, corrugated skin. Diagnosis; sudden eruption, its isolation, subsidence on the coming of cold weather, and reappearance with the hot, intense itching. Treatment: As in eczema. Protect against friction, give shade, and spray with cold water.

Under this name has been described a papular eruption occuring in horses, cattle, sheep and dogs in the hot season, but also occasionally, in winter, in hot, confined stables.

It is seen especially on the neck, back, croup and thighs, is common in fine bred horses with delicate skins, and nervous temperament, and is pre-eminently a disease of hot weather. Over-driving, heating food, a drink of cold water when heated or indigestion connected with unsuitable food may be the occasion of its irruption or tend to perpetuate it. In the same way different chronic affections of the stomach, liver, kidneys or other organs may be causative factors.

Symptoms. The affection usually begins with a few minute papules, isolated or in clusters, which dry up into scales or crusts.

These are mostly situated at the roots of the mane or tail or on the sides of the neck, withers or trunk, and as a rule produce a pruritus, resembling that of scabies in its intensity. When the exudate agglutinates a tuft of hair, enclosing it in a dense crust, the hairs may be lifted from their follicles and thus small, round spots of depilation appear. If recovery ensues and new hair starts, it differs in color from the old and gives a dappled appearance to the skin. In many cases, however, the points of eruption and encrustation become confluent and an extensive area of bareness, with more or less abrasion, and even ulceration may be formed.

Megnin mentions two cases and the author can adduce another in which the eruption appeared in vertical lines, so that the skin of the trunk was raised in a series of elevated lines or ridges, running transversely to the body, like the stripes of a zebra. In the author's case the skin seemed to be thrown into a series of folds to the production of which the cutaneous muscle evidently took part. The itching was doubtless the immediate cause.

Diagnosis is based largely on the suddenness of the eruption; on its limitation to a given area instead of spreading from the primary seat of invasion as in acariasis; on the fact that it is usually confined to a single animal and has not spread with the use of the same brush, comb and rubber; and on the absence of acari and vegetable parasites from the affected parts. The absence of chicken roosts or manure is another valuable indication.

Prognosis. Appearing in spring or early summer, the disease is liable to persist until the advent of cold weather in fall, and even after a winter's intermission there is a strong tendency to its re-appearance on the following spring or summer. The intolerable itching interferes seriously with docility and steadiness in harness, and the loss of hair renders the subject very unsightly, and as a family or driving horse practically useless.

Treatment. As in cases of eczema the general and special causes should be corrected by hygienic and general medicinal measures, laxatives, diuretics, antacids, tonics, and in the advanced stages, alteratives coming in as important factors. (See under acute eczema). Great care should be taken to prevent irritation by pressure of the harness, and shade and daily cold spraying may be availed of.

PITYRIASIS : SQUAMOUS SKIN DISEASE : HORSE.

Dry, scaly, or powdery affection. Causes : Fine, thin, dry skin with little hair, race, Arab, Barb, racer, trotter, nervous temperament, age, dry summer heat, dry winter cold, foul skin, caustic soaps, ingestion of salt, iodides, bromides, etc., derangement of internal organs bacteria or cryptogams. Symptoms : scurfy patches, general or circumscribed, where little hair is, where harness rubs, depilation of ears, crest, tail, shoulder, back. Diagnosis, from eczema by lack of pruritus, of rapid extension, of thickening of the skin, from acariasis by absence of acarus. Treatment : correct disorder of stomach, liver, or kidneys : green, succulent or nutritive food ; alkalies ; arsenic ; tonics ; locally potash soaps, ointments of tar, birch oil, creolin, creosote, naphthalin, lysol, mercury, iodine, salicylic acid, zinc oxide.

This is a skin disease characterized by excessive production of epidermic scales, and depilation without any attendant elevation of the skin. The desquamation may be of fine scales like wheat bran, or of a fine dust like flour.

Causes. The disease is especially characteristic of animals in which the skin is naturally fine, thin and dry and covered sparsely with hair. It is therefore more common in the Arabian, Barb, English racer, American trotter and other breeds of a nervous organization than in the heavier draught breeds. Old horses in which the skin is drier and the hair thinner are more subject to it than the young. Again it has been especially noticed in the heats of summer with thin coat and a withering action of radiant heat on the skin, and less frequently in winter when the blood is driven from the surface by cold. Much also depends at times on the lack of grooming, on the accumulation of dust and dried up secretions about the roots of the hair, and on washing with caustic irritant soaps especially in long-haired regions. It has even been claimed that the ingestion of salt, potassium iodide, or bromide, etc., contributes to the affection. There is undoubtedly a certain individual predisposition to the disease, shown as already stated in certain breeds, but also inherent in particular families and even animals, and associated not only with the character of the skin, but also probably with variations in the activities and products of various internal organs. In man pityriasis versicolor

is associated with a specific fungus, and in the horse Megnin has described cases in which the surface of the skin and especially the hair follicles show a mass of epidermic cells mingled with mycelium and an abundance of spores.

Symptoms. The scurfy product and depilation may be found in patches scattered indiscriminately over the body (generalized), or confined to particular regions (circumscribed) as to the head, ear, crest, tail, or the parts that receive the friction of the harness. It may commence as a dry, rigid, state of the skin under the headstall with loss of hair and the excess of dandruff. From this or from another point the extension takes place slowly and with comparatively little irritation or itching. The hair is pulled out with great ease, and from its spontaneous evulsion, more or less baldness appears progressing slowly from the original centres of the disease. It may leave the whole crest divested of the mane, or the tail of its hairs (rat tail), or the ears may become bare and scurfy. Again the parts subject to friction like the back of the ears, the crest, in front of the shoulder, or the seat of the saddle may be the main seats of depilation and baldness.

It is to be distinguished from dry eczema mainly by its tendency to spread over a larger area in place of confining itself to circumscribed patches, and more particularly by the absence of the marked thickness and rigidity of the skin which characterize eczema. From acariasis it is distinguished by the lack of the intense itching, of the tendency to more or less moist exudation and above all by the absence of the acari.

Treatment. It is well to correct any disorder of any of the internal organs, notably of the stomach, liver or kidneys, and to encourage a free circulation in and secretion from the skin. To fill the latter indication green food, ensilage, roots, sloppy mashes of bran, oilcake and the like may be given. Also bicarbonates of soda or potash or other alkaline diuretics, and in certain obstinate cases a course of arsenic. The alkalies tend to eliminate offensive and irritant matters and to lessen the irritation in the skin. A course of tonics is often valuable.

Locally Cadeac recommends potash soaps rubbed well into the affected parts. If this should fail some of the stimulant ointments as of tar, oil of tar, oil of white birch, oil of cade, creoline, creosote, lysol, naphthalin, may be tried. Megnin strongly

recommends a combination of ointment of biniodide of mercury, 1 part, to mercurial ointment 3 parts. Others advocate salicylic acid (10 to 20 %) mixed with Lassar paste which is compounded of 1 part each of zinc oxide and starch in 4 parts vaseline.

PITYRIASIS IN CATTLE.

On neck and dewlap; Causes: anæmia, debility, spoiled food, starvation, constitutional predisposition. Symptoms: shedding hair and scales without skin thickening, or itching. Treatment: green soap, tar, creolin, lysol, naphthalin, etc. Alkaline lotions: generally nutritive, succulent food, bitters, iron, arsenic, etc.

This is noticed especially on the neck and dewlap in connection with anæmia, low condition, unsuitable, innutritious and spoiled fodder and a constitutional predisposition. It has the same general characters as in the horse, an excessive production of dandruff or dry scales without any marked change in the thickness of the skin or in its circulation. Treatment consists in the application of green soap, pure or medicated, with tar, creolin, lysol, or other empyreumatic product. Lotions of carbonate or bicarbonate of potash are often effective. Any disorder of digestion, or of the urinary or hepatic functions, or of general nutrition should be corrected, and in most cases, a course of bitters, with iron and arsenic is desirable. A good, in-door hygiene or a run on succulent grass in the open air may be resorted to with benefit.

PITYRIASIS IN THE DOG AND CAT.

Head, neck and back of overfed, old house dogs. Symptoms: floury dandruff, with little itching or redness, on limited areas; in cats over the whole back, where stroking causes electric development, the collecting of the hair in tufts, and insufferable irritation. Hair constantly shedding without necessarily bare patches. Treatment: simpler, restricted diet, correct internal disorders, laxatives, arsenic, locally solutions of alkalies, borax, potassium sulphide, sulphur iodide, baths.

In dogs this affection attacks especially the head, neck and back of pet and house dogs gorged with dainties, and particularly in

those that are already becoming aged. The affected parts are covered with a floury or branlike product lying upon a dry surface usually devoid of irritation or congestion, though it may be distinctly congested and reddened, and even the seat of pruritus. The affection is usually confined to limited areas, more or less destitute of hair, and without showing a disposition to active extension. In the cat, however, it may affect the whole dorsal aspect of the body, being associated with extreme electrical susceptibility, so that on being stroked the hair at once collects in tufts, crackles, and in the darkness sparkles, and the animal at first fawning on the hand, will fly at and scratch it after a few strokes. The scaly product is excessive and drops off abundantly when handled, without, however, leaving thin or bare patches.

Treatment is mainly in the line of a simpler and more natural diet, the avoidance of sugar and cake, the correction of disorders of the digestion, or of the hepatic or urinary functions, the exhibition of an occasional laxative, and of alteratives, especially Fowler's solution.

Locally, alkaline lotions, carbonate or bicarbonate of soda or potash, borax, sulphide of potassium and iodide of sulphur are often useful. A moderately strong solution of common salt with glycerine in water is an useful alternate, and a warm saline or bran bath may soften the skin and modify its nutrition.

CONTAGIOUS PUSTULAR DERMATITIS IN THE HORSE. ACNE.

History. Cause : bacillus. Symptoms ; incubation 6 to 15 days, skin tenderness, heat, swelling like peas, hazel nuts, vesicles, pustules, exudation, concretions among hairs, depilation, healing in 15 days. Leaves white spots with lighter hair. Extension by grooming : general eruption : subcutaneous swelling, sloughs, delayed healing. Lymphangitis. Diagnosis : from chaps and bruises, from horse pox, from impetiginous eczema, from urticaria, from farcy. Prevention, quarantine new horses, separate diseased, disinfect skins of the unaffected, disinfect stables and harness. Treatment : soapy wash : germicide lotions.

This has been largely described as an imported disease thus on the European continent it is the English variola, and in England

the Canadian contagious pustular affection. Yet the first authentic account dates back to 1841-2 when Goux found it attacking an entire squadron of the French army in a fortnight. Axe described it in England in imported Canadian horses in 1877, and Weber observed it in the same year on the continent, where it was attributed to imported English horses. In 1883 it was noted by Schindelka, in 1884 Siedamgrotzky inoculated it from the horse on two rabbits and two Guinea pigs, and to horse and goat. The rodents developed a "malignant œdema" at the point of inoculation and died in six days. Grawitz and Dieckerhoff cultivated the bacillus on ox or horse serum and found it 2μ in length, dividing by segmentation into round or ovoid refractive spores, which may remain connected as diplococci or short chains and which color deeply in fuchsin. It grows most rapidly at a temperature of 37° C., growth ceases at 17° C., and it is destroyed in half an hour at 80° to 90° C. Preserved, dry, it remained virulent for four weeks and produced the characteristic eruption when rubbed on the skin of the horse, ox, dog, sheep or rabbit. It proved fatal to all rodents, including white mice. The microbe is found abundantly in the pus and crusts and is easily shown when these are treated with potash. It produces no putrid fermentation.

Symptoms. When inoculated it had an incubation of six to fifteen days followed in mild cases by swelling heat and tenderness of the skin with collection of the hair in erect tufts. Next day there are rounded elevations like peas or hazel nuts, discrete or confluent on the swollen patches. These nodules, at first firm and resistant soon become soft in the center, forming vesicles and finally pustules, which burst in five or six hours and exude an abundant liquid which concretes in a thick amber colored mass. The hairs in the center of the resulting raw surface are easily detached leaving bare spots the size of a dime, with often times a slough attached in the center. When this is finally eliminated the surface gradually cicatrices and recovery may be complete in fifteen days. The skin remains long dappled from the partial discoloration of the epidermis in the seat of the pustules. The malady is local and hyperthermia is rarely seen. The submaxillary and pharyngeal lymph glands are usually swollen and indurated, but this disappears speedily after the subsidence of the eruption.

In certain cases the extent of the primary eruption is greater from the first, or it extends through reinfection by combs, brushes and rubbers used in grooming or by friction by the harness, the affected skin is hot, painful, congested and thickened throughout its entire substance, the pustules are much more numerous, often confluent, and may even implicate the subcutaneous connective tissue. The crusts formed on the sores may acquire a breadth of 1 inch to 1½ inch. Considerable abscesses may be formed and the lymph glands communicating with the affected part are hot and swollen. Even after the opening and discharge of the abscess, the base of the sore remains indurated and indolent, and centres of softening and caseation may appear so that healing is delayed for one or two months or more. In such cases extensive cicatrices remain after recovery. Lymphangitis is a common accompaniment with even abscess of the lymphatic glands.

Diagnosis. From chafing and bruising by the harness, this is easily recognized by its appearing also on other parts than those covered by the harness, by the development of the characteristic pustules, by its following a regular cycle of eruption and subsidence covering a definite period of usually 15 days, and by the indisposition to maintain itself indefinitely under the friction of the harness.

From *horsepox* it is distinguished by the habitual avoidance of the common seats of election of that disease (heels, lips, nostrils, buccal and nasal mucosæ, lips of the vulva), by the absence of hyperthermia, and by the comparative absence of the remarkable amber-like concretions which characterize horse-pox in the lower limb.

From *impetiginous eczema* it is diagnosed by its contagious and inoculable properties, by the absence of the early falling of the hair from the circumscribed rounded nodules, and by the absence or moderate character of the pruritus which is usually intense in the eczema.

The eruption of *urticaria* appears much more suddenly, shows no tendency to form vesicles nor pustules, is not inoculable, and subsides often as suddenly as it appeared when the irritant food materials have been expelled from the alimentary canal.

From acute *farcy* it is distinguished by the moderate degree of the implication of the lymph vessels and glands, by the white

creamy nature of the contents of the pustules, as compared with the glairy, oily nature of the farcy discharge, by the absence of coincident nasal ulcers, submaxillary nodular swellings or other lesions of glanders, by its short course and tendency to spontaneous early recovery, and by the absence of reaction under the mallein test.

In all cases the known prevalence of the contagious pustular dermatitis in the locality, or the introduction of strange horses which exhibit sequelæ of the lesions will assist greatly in the diagnosis.

Prevention. If animals are introduced from an infected or unknown locality they should be kept apart from others for two weeks. In a stable where it has already appeared the diseased and healthy should be carefully separated and the skins of those as yet unaffected may be washed with a solution of mercuric chloride (1 : 1000) or creolin (1 : 100). The walls of the stable should be whitewashed, and all stable utensils disinfected in boiling water or one of the above named antiseptics. The harness demands particular attention

Treatment. This is essentially germicide. After a soapy wash, any one of the usual disinfectants may be used : aluminum acetate, (1 : 15), mercuric chloride (1 : 1000), carbolic acid (1 : 50), creolin (1 : 50), copper sulphate (1 : 50), etc. Lead acetate 2 parts, alum 1 part and water 50 parts, has been found to be effective.

PEMPHIGUS IN HORSE, OX, PIG AND DOG.

On rare occasions the horse or ox is attacked with a skin eruption, attended with the formation of bullæ or blisters, from the size of a hazelnut to a hen's egg, or larger. It is sometimes shown sporadically and at others appears at once in a large number of animals in the same herd. The *causes* are obscure, yet the enzootic appearance of the affection is suggestive of a common factor entering probably by the food. Loiset and Seaman have recorded enzootic outbreaks in cattle and Dieckerhoff in the horse.

Symptoms are cutaneous congestion with the formation of swellings like a walnut, but exceptionally as large as the fist, on

the head, neck and thorax, which in 2 to 4 days form a large central vesicle, with yellowish serous contents. Cases in the ox (Loiset, Seaman) had a similar eruption on the loins, quarters and hind limbs, some of the swellings attaining the size of a hen's egg, and with similar contents. Later these ruptured, crusted over and healed, with, for a time, a smooth glistening surface. Winkler records cases in swine and Schneidemühl in dogs, but the condition is rare in both animals.

Treatment. To a nutritious, non-stimulating and easily digestible diet, may be added a course of arsenic and, in low condition, of bitters. Locally dusting powders of zinc oxide, boric acid, starch and lysol. Should the exudate form these into hard cakes, they may be replaced by carbolized oil or, better, a 5 per cent. mixture of ichthyol in vaseline.

CRACKED HEELS IN HORSES. SCRATCHES.

Special susceptibility and exposure of posterior pastern region. Divisions. Causes: local irritants, decomposing manure, chill water, slush, mud, pools of liquid manure, septic irritation, stones, sand, lime in mud, salted snow or ice, washing heels, caustic soaps, stubble, clipped or singed hair, stocking of limbs, lymphangitis, sprains, arthritis, anæmia, cardiac, urinary or hepatic disease, parasites, heavy bedding, constitutional predisposition. Symptoms: redness, heat, tenderness, swollen, erect hairs, lameness, knuckling, or exudate, crusts, scabs, abrasions, chaps, fissures, ulcers, loss of pliancy, engorgment of limbs, fetid secretion. Prognosis according to cause. Treatment: remove causes, give rest, cleanse limb and stable, astringent antiseptic lotions, sulphurous acid, carbolic acid, creolin, lysol, pyoktannin, chrysophanic acid, moderate laxative food, diuretics, arsenic, bandaging, hand rubbing, exercise.

The affections of the heel or posterior part of the pastern in horses are largely modified by the anatomical character of the skin in this region, and the special exposure to inimical agents, so that it is convenient to consider them under special headings, even though the eruption may be of the same kind with that seen in other parts. The dermatitis of this region, which are not primarily contagious may be conveniently divided into 1st, such as are unattended with free secretion, and 2d, those that implicate the sebaceous glands and are marked by an offensive discharge. Cracked heels belong to the former category.

The *causes* are extremely varied, consisting in the application of irritants of many kinds, to the susceptible skin in a system too often already predisposed to skin disease.

Standing on reeking dungheaps, or on heating manure in filthly stalls subjects the heels, and especially the hind ones, to ammonia and other irritating fumes, and when taken out to the cold air, chill water and mud, the sensitive parts suffer. Again in the farm yard and even in neglected stalls the hind feet are immersed in pools of liquid manure, the ferments and toxic matters of which dry on the skin, attack the surface and determine septic congestions and inflammations. On country roads where there is no pretense of pavements, or macadam, the mud in spring and fall is a source of great irritation on certain soils which contain small flat stones, pebbles or sand, or in which lime or decomposing manure is a prominent feature. Standing in snow or slush, especially if chilled by salting, produces partial or complete congelation with the result of chillblains or even more active and destructive inflammation or sloughing. The habit of washing the heels and allowing them to dry spontaneously in the stall is only less injurious by the chill induced. This is still further aggravated by the use of caustic soaps on the already tender skin. The lighter breeds of horses, devoid of long hair on the pasterns, though less subject to the *greasy* secretion, are even more exposed to chills and direct injuries, and suffer readily and often persistently from erythema and cracks. In many cases trouble comes from the ends of stubble and other vegetables acting on the skin. A common fault is the close clipping and even singeing of the hair in the hollow of the heel. The stiff, bristly ends of the hairs on one fold of the pastern continually prick the skin of the adjacent fold when the animal is in motion and not only is this irritating to the healthy skin, but it becomes incomparably more so when that is congested and tender. Even in summer the deep dust on unpaved roads, mixing with the normal secretions of the heel, rolls into semi-solid masses between the folds and proves the more irritating, the greater the admixture of sand or solid bodies. A common cause is the stocking of the limbs, with the attendant congestion, distension and debility of the skin. This may be due in its turn to a great variety of proximate or remote causes, lymphangitis, sprains,

arthritis, osteitis, anæmia, cardiac, urinary or hepatic disorder, parasitisms, etc., so that accessory causes must often be widely sought. Even an excess of straw around the hind limbs will cause stocking in some animals which escape on bare pavement. Finally we must take into account that constitutional predisposition in some animals that makes them liable to inveterate skin diseases under the slightest causes.

Symptoms. In the *milder forms* there may appear a redness, with heat, tenderness and swelling in the hollow of the pastern, the hairs stand stiffly erect, and the surface may be perfectly dry. The affected limb has the pastern more upright than the others and the fetlock starts slightly forward. In a nervous, sensitive horse the skin is so tender and rigid, that the animal can hardly be persuaded to use the limb, and goes dead lame for a considerable distance until it has become more pliant.

With some aggravation of the condition the skin is felt to be somewhat rough and uneven by reason of the encrustations of epidermis, dried secretions and dust over its surface, which may convey to the finger a slightly oily sensation. In many cases these epidermic and exudation products form scabby elevations, and a chronic condition of this kind may persist indefinitely, constituting what is known in America as *scratches*. This will vary by reason of the detachment of these concretions with the formation of abrasions and sores of various sizes, which may heal, or extend by coalescence, chapping, or ulceration.

In other cases, even at an early stage, the formation of chaps or cracks is a marked feature. At times this may seem to be the result of over distension in the inflamed superficial layers of the skin which have lost their natural pliancy and cohesion. They will, sometimes, form under slight exercise, but not when at rest. They may simply extend through the epidermis, exposing the papillary layer, or in bad cases one or more fissures may extend through the integument and expose the tendons beneath. They may extend forward on the sides of the pastern or upward over the back of the fetlock and metatarsus.

In all cases, when the local inflammation is acute, some swelling of the limbs appears, and this keeps pace with the character and extent of the trouble. With extensive chaps or fissures it becomes extreme, extending up toward the hocks and attended by great

pain and stiffness. The sores become the seat of active suppuration, with it may be considerable destruction of tissue. Even in the milder forms there may often be seen a fœtid muco-purulent secretion in the depth of the folds of the pastern, and in the worst cases this extends to the whole surface after the manner of *grease*.

Prognosis. The milder uncomplicated cases recover readily and perfectly under rest and judicious treatment ; the more advanced cases are liable to leave swelled legs with susceptiblity to a relapse, and in cases associated with a constitutional diathesis or chronic internal disease, recovery may become problematical and uncertain.

Treatment. In all cases the cause must be done away with, whether filthy stalls, reeking dunghills, septic pools, work in irritating road-mud, or melting snow, washing the heels with caustic soaps, drying them in cold draughts, pricking with stubble or clipped hairs, and all the causes of stocking of the limbs. If heels are washed, use pure tepid water, and, if, necessary, the best Castile soap, and rub them dry at once. If this cannot be done bandage them rather than leave them in a cold draught.

Give rest in a clean stall and thoroughly clean the affected heel, then wrap in a bandage wet with an acetate of lead or sulphate of zinc lotion (1 : 50), or apply benzoated oxide of zinc, or cream of glycerine and salicylic acid.

When chaps have formed they will often promptly heal under standard solution of sulphurous acid 1, glycerine 1, and water 1. This is applied on soft cotton and covered by a rubber bandage to confine the acid. The sulphurous acid solution should be recently prepared, since it will prove injurious if it has oxidized into sulphuric acid. To one or other of these preparations the addition of a little carbolic acid, creolin, pyoktannin, or lysol will often prove useful. When the cracks have healed, zinc ointment, chrysarobin ointment, chrysophanic acid 1, vaseline 15, or other soothing and antiseptic agent may be employed till all inflammation has subsided, and the animal must not be returned to work until the skin has been restored to its former healthy and elastic condition.

It may be desirable to greatly restrict the grain during treatment and even to giving cooling laxatives or diuretics. With a constitutional diathesis arsenic or other alterative may be tried,

and any internal disease must be attended to. For stocking, use careful bandaging, hand-rubbing and exercise.

With the formation of the deeper fissures the same antiseptic agents may be employed, or salol, iodoform, glutol, aristol, or some tincture of iodine, or iodide of starch may be used. A weak solution of copper sulphate has often an excellent effect. The measures advised below for *grease* will usually apply in this condition.

SEBORRHŒA OF THE DIGITAL REGION: DIGITAL IMPETIGO, GREASE: STREPTOCOCCIC DERMATITIS IN HORSES.

A sequel of erythema or cracked heels. Causes: constitutional predisposition in lymphatic draught horses, rare in ass and mule, anatomical conditions, wet damp regions, digestive disorder, over-feeding and lack of exercise, diseases of liver or kidney, change to stable life, cold water, slush, mud, salted snow, steaming manure, urine in mares, infection, streptococcus pyogenes. Symptoms: swelling, heat, and tenderness of pastern hollow, itching, hairs erect, unctuous exudate, vesicles, excoriations, discharge opaque, grayish, sticky, fœtid, chaps, knuckling, resting on toe, kicking: in severe cases discharge purulent, more opaque, sloughs, excessive granulations, "grapes," extensions forward, upward, downward, canker, quittor, sand crack, etc. Lesions: first, congestion of derma, hair follicles full, hairs loose, connective tissue infiltrated, or thickened, ligaments and bones involved, grapes in superposed clusters pediculated. Diagnosis: from horse-pox. Treatment: remove causes, secure cleanliness, laxative, diuretics, moderate grain ration, or tonic regimen; locally, soothing antiphlogistic, antiseptic treatment, lead, zinc, phenol, creolin, lysol; when advanced, antiseptic dusting powders, calomel, salicylic acid, iodine, zinc oxide, salol, or solutions, zinc chloride, tar. Value of changes. For "grapes" actual cautery, excision, ligature.

This may develop as an advanced condition of the erythema or cracked heels already described. Yet it is so distinctive in its habit of profuse secretion, the eruption of vesicles or pustules and the abundant, fœtid sebaceous discharge that it deserves a special consideration.

Causes. Something depends on constitutional predisposition. This is preëminently a disease of the heavy, lymphatic, draught

horse, being rare in racers and trotters, with fine sinewy limbs, no long hair on the fetlock, delicate skins, and less abundant sebaceous glands. It is almost, though not quite, unknown in the spare limbs of ass and mule, and though claimed by Reynal as attacking cattle its occurrence is equally rare in them. Much of this may be attributed to conformation. The limb of the draught horse is so much thicker and coarser, with a great excess of connective tissue and lymph plexus which become readily gorged in idleness, inducing stocking, congestion and debility of the whole limb. This same condition operates as a powerful predisposition to lymphangitis. Again the great length and profusion of the long hairs, entails the necessary compliment of an excessive development of the sebaceous glands which become over-stimulated by congestion, and afford a much more open and favorable infection atrium for the pus microbes. These structural conditions are much more marked in the draught horses of wet regions as in Ireland, the western counties of Great Britian, Belgium, Holland, and the Atlantic provinces of France, and in these the affection is remarkably prevalent. In our Eastern States and on the Plains, where the progeny of imported draught horses lose their digital hair, the malady is comparatively rare. A similar immunity has long been noticed in the horses of Spain and Africa. Disturbances of the digestion in heavily fed horses, subjected to transient confinement in the stall, and diseases of the liver and kidneys, must be recognized as further predisposing causes. The age of five and six when many horses change hands, and are subjected to extreme changes of stabling, feed and work, has furnished the greatest number of cases.

External causes we find in all those conditions already enumerated which favor chapped heels. Wet, mud, gritty masses, irritant fumes of manure, cold, heat, filth are potent factors. In connection with these are the pus and septic microbes that are always present in stables, farm yards, manure, street dust, etc. No one of these can be adduced as the constant and exclusive cause, and it is inevitable that a complex infection should be present, yet the propagation and persistence of the disease may often be connected with the streptococcus pyogenes.

As emphasizing the importance of such external irritants and infections, it should be noted that the disease bears an appreciable

relation to the filth and wet of the stable and farm yard, and to the absence of cleanliness in dealing with the feet, and that the extension of good pavement and protection from road mud have invariably lessened its prevalence. The irritant action of the urine renders mares more susceptible in the hind limbs than horses.

Symptoms. The disease may appear as a swelling, heat and tenderness of the hollow back of the pastern, involving the fetlock and lower part of the metatarsus or metacarpus, and this may last for one or two weeks, the engorgement lessening or disappearing during exercise and reappearing when at rest in the stall. The local tenderness is great as manifested by the prompt and excessive lifting of the leg when the heel is touched, as well as by the lameness when first moved, which subsides with further exercise. Itching may be shown by kicking the floor, or by a disposition to rub the pastern. The hairs of the affected part are rigidly erect, and a slightly moist, soapy sensation is felt on the skin. Close examination may detect the presence of small vesicles with as yet limpid contents, but the greater part of the liquid product is traceable to the openings of the hairs and gland ducts. This is followed by small excoriations taking the place of the ruptured vesicles, and the discharge becomes more profuse, opaque, white or grayish white, sticky, and fœtid. It covers the entire affected surface, mats together the hair in tufts and forms a thicker grayish border. The hairs are loosened in their follicles and easily pulled out. The erosions become complicated by chaps, and the swelling increases around the pastern and above the fetlock. When at rest in the stall the foot may be rested on the toe only, or held suspended and occasionally kicked backward as if to dislodge the cause of irritation, yet if moved the patient may gradually get over the greater part of the lameness, and the swelling partially subside.

In severe, protracted cases the discharge becomes essentially purulent, but often with a darker, greenish, reddish or blackish tinge, and portions of the skin may slough, leaving deep intractable sores. Still more commonly the raw surfaces become the seat of hypertrophied granulations, which grow out to form raw, red fungous like, pediculated neoplasms familiarly known as *grapes.* Between these the spaces are filled with tufts of hairs and the condensed discharges, in process of active septic change,

and giving off a most repulsive odor. Like the preceding eruption these *grapes* may extend around the front and sides of the pastern, and upward beyond the fetlock, but especially behind.

This advanced condition shows no tendency to spontaneous recovery and the connective tissue and lymphatic plexus becoming involved, the leg often swells to enormous dimensions, from six to twelve inches in diameter at the fetlock. It may last indefinitely until the patient is worn out, or it may extend to other organs by contiguity or embolism. Canker of the frog and sole, fistula (quittor), sand crack and seedy toe may be named as complications, also septicæmia or pyæmia with abscesses in the lungs, liver, brain or bowels.

Lesions. In the first stage there is mainly the congestion of the skin extending into the large and numerous hair follicles of the pastern. If pressed, a transparent serum bedews the surface, and if sectioned the follicles around the hair bulb are seen to be distended by a similar product. The hairs are easily pulled out. The subcutaneous connective tissue is filled with a yellowish serosity and at intervals may be seen a red point of vascular stagnation or blocking. Later these products are more abundant and those on the now swollen and excoriated surface are distinctly fœtid. The infiltrated lymph plexuses in the connective tissue are more distended, their walls thickened and consolidated, and the rigid skin is thus firmly bound to the structures beneath. A careful examination shows the presence of subepidermic vesicles of various sizes. The congestion may extend deep enough to involve the periosteum of the digital bones and the ligaments of the joints. The *grapes* are each attached by a pedicle from which branch out cauliflower-like, fine papillary processes, that aggregate into a solid cluster. They are very vascular and grow out cluster above cluster until they reach large dimensions.

Diagnosis from Horse Pox. Since the days of Jenner the claim has been constantly made that *grease* and *horse pox* were one and the same. Horse pox is however to be distinguished by its transient course, its inoculability, its incubation of three days, its abundant exudate concreting on the hairs of the pastern as a yellow mass suggestive of crystalline structure, by the red pit in the skin in which this mass is imbedded, by the spontaneous

recovery in about 15 days, and by the immunity on a subsequent inoculation. It is communicable to cattle and to man, producing the characteristic large umbilicated vesicle and scab.

Treatment. The first consideration is to remove the causes of local irritation and infection, give a clean sweet stall, with dry floor, and allow no contact with putrid liquids, mud, cold water, melting snow or other irritant. If exercise is needful to obviate *stocking* of the legs give it on dry clean ground.

If inflammation runs high with fever and costiveness a laxative will be valuable and it may be well to follow this in some cases with cooling diuretics. When the animal has been on a heavy grain ration this should be largely cut down in keeping with enforced idleness, or restricted work. If on the other hand condition is low, and the discharge profuse a more generous ration may be desirable.

Local treatment is essentially soothing and antiphlogistic, and in view of the infection should be antiseptic. *White lotion* (acetate of lead and sulphate of zinc of each ½ oz., water 1 quart) has been long used with fair success, for although lead sulphate is thrown down, it is in part freed again through contact with the exudate. It will be materially improved by the addition of 1 dr. carbolic acid, creolin, lysol or chloronaphtholeum or by some other antiseptic. Lead acetate alone with an antiseptic is an excellent substitute. In mild cases the surface may be wet with the lotion several times a day; while in severe ones the lotion may be applied on a bandage kept constantly wet. When secretion is well established it may be better to use dry applications, as calomel; salicyclic acid 10 parts, with iodine 10 parts; calomel and lamp black; carbolated oxide of zinc or burned alum, salol, etc. After washing and drying the skin, dust this freely even into the deepest wrinkles and cover with cotton and bandage. Dress twice daily. For very fœtid cases, Robertson recommends zinc chloride 1 oz. in 1 qt. water with the addition of glycerine and phenol, and again a saturated solution of copper sulphate with carbolized glycerine. For profuse secretion after the subsidence of active inflammation Renal highly recommends wood tar with 5 to 10 drops of sulphuric acid to the ounce. Under these circumstances a powder of gloss starch 5 ozs. with iodine ½ oz. may be employed twice daily. Or again, 1 oz. each of carbolic

acid, tincture of iodine and glycerine may be employed. When one agent seems to be losing effect, it is well to change for another and never to neglect the regular dressing, until full recovery has been secured.

In case of *grapes* the actual cautery is the most efficient measure. Heat a blacksmith's fire shovel to a bright red and use this to cut through the pedicles, a cool shovel being kept constantly beneath it and in contact with the pedicles, so as to protect the adjacent skin from injury by the radiated heat. The lower shovel must be dipped in cold water at very frequent intervals to cool it and prevent cauterization of the skin between the pedicles. This not only removes the diseased and infected masses, but leaves the stumps of the pedicles aseptic. Another method is to cut off the "grapes" and staunch the blood with the actual cautery at a dull red heat. Still another is to tie the pedicle of each excrescence separately so as to cut off circulation and secure sloughing. This is, however, a long, tiresome process, and entails prolonged contact with much infecting dead tissue. After either method the parts must be dressed with antiseptics, and dealt with generally like cases in which the excrescences had not formed.

CUTANEOUS HEMORRHAGE: BLOODY SWEAT: HÆMATIDROSIS. HÆMATOPEDESIS.

Forms of cutaneous hæmorrhage ; in specific diseases ; in parasitism ; in insect bites ; in congestions of sweat glands ; in deranged innervation ; in hæmophilia, Section of sympathetic. Salt on sciatic. Hysteria. Sclerosis of cord. Inflammation. Symptoms : drops, crusts. Hæmorrhagic nodules. Treatment : styptics, cold, ice, snow, tannin, matico, iron chloride, alum, gelatine, atropine, ergot, lead acetate, quinia. Gravitation.

The escape of the blood by the skin is seen in a variety of morbid conditions, due it may be to profound changes in the blood and capillary walls, as in petechial fever, anthrax, scorbutus, septicæmia, swine erysipelas, etc., in which this is only a subsidiary phenomenon of a general disorder :—to the presence of parasites (Filaria hæmorrhagica,) in the skin :—to insect bites :—to violent congestions implicating the sweat glands (bloody sweat):—or to deranged innervation of the part as in cases of trauma of the sympathetic or sciatic nerve, or disease of the nerve centres. It may further be a manifestation of hæmophilia in which any slight lesion becomes the occasion of persistent hæmorrhage.

Cases that appear in the course of specific contagious diseases and those dependent on filaria will be considered under these headings, and we may confine our attention here to the forms of sweating and oozing of blood from independent causes. German writers draw attention to its frequency in eastern horses, attributing it to the great development of the vascular system especially of the skin, but its comparative infrequency in the English racer and American trotter would throw doubt on this doctrine. It may be questioned whether the frequency of the disease in Oriental horses is not to be ascribed rather to filariasis. This idea is not contradicted by the especial prevalence of the

bleeding in summer when the filaria is most active, but when also the skin is the most vascular and its tissues most relaxed.

Of nervous hæmorrhages we have the experimental examples of Bouchard and Simon from section of the sympathetic nerve in animals, also those of Glen and Mathiew from irritation of the sciatic in dogs with common salt. In man the nervous causation has been seen in hysteria, under profound nervous shock, in sclerosis of the cord, and even as the result of auto-suggestion. This influence is constantly operative in violent inflammations in which diapedesis and minute hæmorrhages into the affected tissues are marked phenomena, and under such a cause the gland ducts especially are the seat of transudation. When the skin is abraded, cracked, or blistered it occurs also on the surface of the exposed derma.

Symptoms. With active local congestion or inflammation the blood usually oozes in drops from the surface, and drying concretes into dark red crusts. In other instances, however, it drops from the surface, or even flows, producing anæmia and even death. Into such cases hæmophilia presumably enters. Hæmorrhagic swellings like wheat kernels or beans also form in the skin.

Treatment. Apart from the contagious and parasitic diseases, and scurvy, the general treatment will be styptic. Cold water, ice, snow, a stream from a hose, solutions of tannin, matico, iron chloride or sulphate, alum or gelatine may be employed. Internally the iron salts, gelatine, atropine, ergot, lead acetate, or quinia may be given. In hæmophilia the gelatine especially should be tried both locally and generally. When it is possible, as in the case of the head, gravitation should be availed of. Elsewhere a compress bandage may be used.

ULCERATION. GANGRENE. BED-SORES.

Causes: inflammation, exudation, obstructed circulation, lesions in trophic nerve centres, sclerosis, toxins, ergot, caustics, freezing, gangrene, microbes, cryptogams, spoiled fodder, white skins, buckwheat, insolation. Symptoms: inflammation, molecular disintegration, dry sloughs. Treatment: camphorated spirit or vaseline, antiseptics, phenol, salicylic acid, iodoform, iodine, creolin, lysol, tar, detach sloughs.

In all cases in which the skin is violently inflamed, and particularly when the seat of an abundant exudation or infiltration which blocks circulation and retards nutrition, the tissues are especially liable to death, molecular or by sloughing, and formation of bed-sores. As a general cause lesions of the trophic centres in the medulla and cord must be accepted as a cause of the imperfect nutrition and lack of vitality. This is seen in sclerosis of the cord, but may appear as the result of poisoning of the myelon as well as the gangrenous tissues by absorbed toxins. Again a common cause of circumscribed cutaneous gangrene is the capillary contraction and obstruction of ergotism. This usually involves all the tissues, soft and hard, at the distal end of a member or organ, causing the separation of all at one common level, but in less severe forms the skin only sloughs, in the form of round or irregular masses, usually around the coronet, and the resulting sores heal up under an appropriate diet. Cauterization and freezing may be a further occasion of gangrene. Finally, the local operation of the microbes of gangrene, determines both ulceration and sloughing. Cryptogams on spoiled fodders (trefoil, lupins, vetches, rusty gramineæ) are also charged with developing gangrene.

White skins or white patches on the skin are especially liable to suffer as in cases of fagopyrism and "grease". The action of the solar rays in summer must therefore be accepted as a concurrent cause.

Symptoms. The first symptoms are usually those of cutaneous congestion or inflammation. Redness, swelling, pitting on pressure, or tension, are accompanied or followed by vesicles, chaps

or erosions. The margins of the sores become thick and irregular, often undermined, and they gradually increase by breaking down of tissue in their depth or on their margin. In other cases patches of skin dry or wither up, either in superficial layer or throughout its entire thickness, and these dried extra vascular sloughs are gradually detached by granulation beneath. The surrounding tumefaction is always extensive and the sores may expose the deep seated structures—tendons, ligaments, fascia, bones, joints—causing widespread destruction.

Treatment. If the disease is due to capillary occlusion of nervous origin, compresses with camphorated spirit, followed by camphorated vaseline may be of advantage. If otherwise, antiseptics will be in order : carbolated vaseline, salicylic acid cream, iodoformed vaseline, a weak iodine ointment, creolin, or lysol in water, tar water. When the dead tissues are partially separated the detachment may be hastened with knife or scissors and the sores treated like a septic sore.

CUTANEOUS HYPERTROPHY. ELEPHANTIASIS. PACHYDERMIA.

Chronic thickening of skin and lymph plexuses, horse hind limb after eczema, grease, glanders, ox neck and head, knees, shoulder. Calcification. Treatment : laxatives, diuretics, exercise, elastic bandage, friction, astringents, iodine.

Chronic thickening of the skin is most commonly seen in horses as a sequel of lymphangitis in the hind limb, the engorgment of the lymph plexus and thickening of its walls being associated with a general productive inflammation and thickening of the derma until the fetlock may be thirty inches or more in diameter. It may follow eczema, grease or chronic glanders. In cattle a productive dermatitis in the region of the head and neck, has led in the experience of the author to a similar distention of the lymph vessels and morbid thickening of the skin. The pads and calluses which form on the knees of the camel and on other parts subject to friction, furnish examples of hypertrophy of another kind. Again the thick dense cutaneous plates on the

shoulders of the old boar may serve to illustrate a physiological hypertrophy. The writer has seen thickening of the skin in the seat of an incision made in spaying the pig and the deposition of earthy salts so as to form a distinct calcification.

Treatment is very unsatisfactory, yet something may be done by laxatives, diuretics, regular exercise, an evenly applied elastic bandage when in the stable, massage and the use of astringent and dilute iodine ointments. It is much more important to prevent the lesion by cutting short the morbid condition on which it depends. When developed, attention is usually given to prevent its advancement and to utilize the animal at slow work.

ICHTHYOSIS. FISH-SKIN DISEASE.

This consists in a scaly formation of the epidermis which is also formed in excess, and is supposed to be dependent on disordered trophic innervation. In new born calves suffering in this way Van Stettin found an excess of phosphate of lime in the epidermis. The calves usually die in a few days.

FURUNCULUS. BOIL.

Definition. pustule with necrotic core. Digital region. Microbes. Symptoms: Prevention: antisepsis. Treatment: phenol, iodine, alcohol, boric or salicylic acid, iron, bitters, calcium sulphide, sulphur, sodium sulphite.

A deep seated inflammation of the derma resulting in suppuration with the formation of a central adherent necrotic slough or core.

Boils are not uncommon on the digital region of horses in winter; or where the parts are exposed to street mud containing an abundance of decomposing organic matter. They are unquestionably due to pyogenic microbes, and have been largely associated with staphylococcus pyogenes aureus. As they often come out in successive crops, it may be assumed that the second focus is infected from the first, or that there is a special susceptibility

in the particular animal system, or that both these factors contribute to the result. Debility, and traumas contribute to bring about the infection.

Symtoms. A nodular, hot, and very painful swelling, implicating the substance of the true skin, and surrounded by a hot, swollen zone, progresses to suppuration in the centre, yet when it bursts, or is opened, a *core* or small mass of necrotic, tough, fibrous tissue is found to be firmly adherent in the center of the bottom of the sore.

Prevention. Must be sought in sustaining the general health and in preserving the greatest cleanliness of stables and skin. Washing with a weak antiseptic solution when returning from muddy streets may be of use.

Treatment. When developing, the application of carbolic acid in crystal or on the end of a glass rod may often relieve the pain and destroy the microbes. Next day the part may be painted with tincture of iodine. If already opened the phenol may still be applied and followed after five minutes by alcohol to check the caustic action. Or boric or salicylic acid may be applied. To counteract the constitutonal tendency, iron, bitters and other tonics and antiseptics, calcium sulphide, sulphur and sodium sulphite may be given.

HYPERPLASIA OF THE SKIN.

Productive inflammations and tumors of the skin are common in domestic animals but as they are essentially surgical lesions, they will be better considered under that head. We may name papiloma, warts, fibroma, melanoma, epithelioma, sarcoma. adenoma, carcinoma, dermoid cysts. Parasitic and microbian diseases of the skin will be found under their respective headings.

ALOPECIA CONGENITA. CONGENITAL BALDNESS.

Cases of this kind have been met with in foals and calves, which were born entirely bald or with only a few thin delicate hairs scattered over the surface. In a calf observed by the author, and which lived for several months, the body was almost absolutely bald, and the mouth remained edentulous, a coincidence which has been observed in other cases. The teeth belong to the same class of embryonic tissues as the epidermis, and a failure in the development of the one is likely to entail a corresponding failure of the other. Schneidemühl observed that the few hairs present in such cases were especially delicate and brittle.

ALOPECIA. POST PARTEM. ALOPECIA AREATA.

Normal shedding. Shedding out of time; laminitis, dropsy, exudative dermatitis, acariasis, ringworm, traumas, folliculitis. Debility, excessive lactation, starvation, petechial fever, spoiled fodder. Without apparent cause, alopecia areata, neurosis, micrococci, disease of derma. Symptoms: general disorder has general shedding. Local disorder extends from a centre. In horse with increased pigmentation. Treatment: correct general causes, use hair stimulants, cantharides, kerosene, tar, pilocarpin, mercuric chloride, cresol, iodine, balsam of Peru, silver nitrate. Arsenic.

Acquired baldness is recorded in horse, cattle, sheep and dog.

Causes. The simple shedding of hair occurs physiologically in animals with the change of season, and if anything interferes with the growth of the new hair a transient baldness may ensue. If such shedding occurs from any cause at the wrong season, before the new hair has started, the baldness may be accentuated. Thus shedding may occur in some forms of indigestion, in laminitis, in dropsical swelling of the limbs or ventral aspect of the body, in dermatitis with an exudation which concretes around the hairs and raises them out of their follicles, in mange, in demodectic acariasis, in circinate ringworm, in traumas as on

the elbows of dogs, etc., from lying on them, and in inflammation of the hair follicles from a variety of infections. It has been charged on general debility in excessive lactation, in gestation, and in starvation, on poisons in the blood as in petechial fever, and on musty or spoiled fodders in bad seasons, or from low damp lands.

When in the absence of such appreciable causes it commences at one or more points and gradually extends, and persists, it constitutes alopecia areata. This has been attributed to a disorder of the cutaneous nerves (tropho-neurosis), but the progressive advance of the disease, without limitation to areas representing the distribution of given cutaneous nerves, and the complete absence of other derangement of nerve function, throw doubt on this conclusion. Another doctrine attributes it to a microbe, but though micrococci and other organisms have been found, they have not been proved to be constant nor to be absolutely causative of the disease. Still another theory holds that it is a disease of the derma and not of the hair at all, the evulsion of the hair following the implication of the tissues around the follicles.

Symptoms. The baldness dependent on a general disorder occurs at once over an extended area. That of ringworm, acariasis, and of the specific alopecia areata, advances gradually and often slowly from a given point, until it may include a large area. Röll has seen it extend from a few points to nearly the whole body of the horse in a single year. In this, as in other cases in horse and dog, the baldness was followed by a considerable increase of the pigmentation of the skin.

Treatment. In cases that occur as the result of other diseases, the rational treatment is to deal with these diseases, and then to stimulate the growth of hair by some one or other of the known stimulants (dilute tincture of cantharides, kerosene, tar water, solution of pilocarpin hydrochlorate). In the more specific form, no treatment has been very successful, yet the best results on the whole appear to have come from local germicide applications. Mercuric chloride in alcohol and water (1 ; 500); cresol 1, alcohol 20 ; tincture of iodine reduced to half its strength by addition of alcohol ; balsam of Peru 1, alcohol 5; nitrate of silver 1, alcohol 15, serve as examples.

As general treatment arsenic has been employed, but with no very encouraging results.

TRICHORRHEXIS NODOSA. NODULAR SWELLING AND SPLITTING OF HAIR.

Debility, ringworm, nodular hair. Hair bursts with brush like end. Cases in horse like singed hair. Causes: infection probable, disturbed innervation, dry air, impaired nutrition. Treatment: shave, oil, vaseline, petroleum, cantharides, sulphur, tar, favor shedding coat.

In a variety of conditions the hairs split up and break, leading to bristling or baldness. In debilitated conditions, when the hair is badly nourished, in ringworm when it is invaded by a cryptogam, and in the various nodular or ringed conditions of the hair this brittleness and splitting appears. The term *Trichorrhexis* (rexis a bursting forth) has been applied to one of these affections, in which the hair swells and bursts into a number of filaments giving it a broom-like termination. Trofimo describes this as affecting two-thirds of the horses of an artillery brigade, and showing upon the back, croup, mane and tail in isolated patches. Megnin, Trasbot and Roy have observed similar cases. The affected hair a short distance from the skin swells into a grayish white nodule and breaks across, leaving a tuft of fine filaments. The patches look to the naked eye as if singed, but when the hair is magnified the difference is easily made out, as there may be several nodular enlargements on the same hair, and the brush-like spread of filaments at the broken end is entirely unlike the solid node on the end of a singed hair.

The *causes* of the affection have not been demonstrated, though indications point to an infection which gradually extends from the first point of attack. This extension is not limited to the area presided over by particular nerves, and there is no other indication of disordered innervation. Trasbot has seen the disease transmitted from horse to horse by the use in common of combs, brushes and rubbers, and in other cases experimentally by rubbing the scurf from a diseased animal on the skin of a healthy one. From observations on the human subject Montgomery looks on it as a result of extreme dryness and brittleness of the hair, and charges dry climates and seasons, and an abuse

of soap in washing as conducive causes. Debility and impaired nutrition of the hair may operate in the same way. No specific microbe has been constantly found in these cases and until such factor can be proved, it may be held that among the etiological agents impaired nutrition and dry air are important.

In the way of *treatment* shaving of the affected parts and oily or vaseline applications may be tried, together with such slight cutaneous stimulants as petroleum, weak tincture of cantharides, sulphur, tar ointment, etc. Roy noticed that cases that had lasted all winter recovered after shedding of the coat. It might be assumed that the evulsion of the infected hair, and the vascular stimulus necessary to the new growth contributed to the result.

CONSTITUTIONAL DISEASES.

RHEUMATISM.

Definition. Past views. Causes: heredity, age, cold, damp, cold climates, seasons, exposure, buildings, cellars, night chills, weather vicissitudes, valleys, wading, checked perspiration, lactic acid, metabolic products, acid phosphate of soda, vegetable acids, neuropathic causation, infection, microbes, injuries, over work. Lesions: in joints, synovia, serosa, articular cartilage, fibro-cartilage, articular lamella, bone, eburnation, ligaments, joints affected in horse and ox, blood changes, albumen, fibrine, blood globules, pericardium, endocarditis, valvular disease, myocarditis, embolisms in lungs, pleura, nervous lesions, digestive system.

Definition. A constitutional, inflammatory affection, probably toxic, tending to localization in the joints, muscles, tendons, fascia, skin, heart and serous membranes and with a marked disposition to shift from place to place.

The word is derived from *rhein* (ῥεῖν) to flow, and was originally employed to indicate that an acrid humor, generated in the brain or elsewhere escaped mainly by the nose and eyes as a catarrh. The idea naturally followed that the retention of this humor caused inflammation in the joints, muscles, heart or elsewhere. The connection of these various conditions with exposure to cold, led to the association of the name with the various internal inflammations in which chilling appeared to have been a factor, until it was difficult to limit it by any definite line. Finally infectious diseases implicating the joints or muscles (influenza, contagious pneumonia, omphalitis, gonorrhœal rheumatism), and diseases of metabolism (gout and possibly rheumatoid arthritis) have added to the general confusion.

General Causes. *Heredity.* This has been more definitely traced in man than in the lower animals, the line of family descent being more easily followed in man. Children of rheu-

matic parents are more obnoxious to the disease in the ratio of 5 to 1. In the absence of data for animals, we may say that it is probable that the influence of heredity will hold good for live stock in this matter. Whether the disease results from faulty metabolism or from microbian invasion or toxins, the hereditary defensive powers of the animal cell are likely to be an important factor.

Age. Genuine rheumatism is not common at a very early age, but in animals as in man, attacks especially those in the prime of life, accustomed to vigorous exercise, free perspiration and succeeding chills. There is as yet no direct evidence of an exhaustion of the susceptibility of early maturity and of any consequent immunity of the old.

Cold and damp. The association of rheumatism with cold and damp has always been so notorious that no argument is needed to enforce the truth. It is pre-eminently the disease of cold climates and cold situations exposed to the north and east in Europe, or to the north and west in North America on the Atlantic slope. It is very little prevalent in the tropics. Buildings and yards that lack sunshine and exposed storm-swept hillsides show cases most numerously. Dogs kept in cold, damp sunless cellars often suffer. Cold, damp or frosty nights start or aggravate cases in animals left out in the field, while warm sunshine usually brings immediate improvement. A sudden change of weather to cold and wet is the signal for aggravation of the existing disease, though this may have set in before the change of weather has been fully developed. Late autumn and early spring with their sudden changes, their sleet and rain are especially hurtful and particularly to chronic cases. The cold, raw ocean winds on the Pacific coast contrasting with the warm sunshine is a marked contributing cause. The currents of cold air that draw up through shaded valleys, gullies and ravines expose their denizens to attacks. Rheumatism is more rare among well housed and carefully treated family horses, and less so in draught, dray and other hard working horses that are made to cross streams and stand with feet and legs in cold water while the body is drenched with perspiration. Dogs suffer in the same way while hunting. Haycock attributes many attacks to wash-

ing the legs with ice cold water when the animal has just returned to the stable heated and perspiring. If then left undried and unbandaged in a cold draught these conditions are still farther aggravated. This was formerly attributed to the suppression of perspiration, but it occurs in an animal which has just perspired freely and is now in no special need of further elimination through the skin. It appears to be due rather to the action of the cold on the nerve endings, the susceptibility of which has been enhanced by the free secretion and general relaxation.

But potent as cold is in precipitating or aggravating an attack, it cannot be looked on as the sole or essential cause of rheumatism. The great majority exposed to the cold escape. The animal which has stood in the stall, or shed, is less likely to be attacked than the one at work in which the heat production has been more active. Indeed an immunity has been claimed for the Artic regions, provided the subject is not exerted so as to cause perspiration. One might readily conclude that this apparent immunity, depended on the absence of an essential germ, as is also claimed for catarrhs, yet Nansen after his prolonged bath in the frigid waters seems to have had an attack of rheumatism. Cold appears to be one of the most potent accessory causes, but evidently not in itself the essential cause.

Cold undoubtedly affects profoundly the metabolism of the body and especially when the vital powers have been reduced by severe exertion, fatigue and perspiration. The same applies to many other affections especially such as are associated with an infecting element, which takes occasion of the debility caused by the cold to establish itself in the tissues.

Lactic acid and other metabolic products. Prout long ago claimed that rheumatism was caused by an excess of lactic acid in the system, and this was supported by the fact that an exclusive diet of buttermilk given in diabetes, is likely to produce acute articular rheumatism. Again the production of rhachitis in the young can be traced in certain cases to excess of this acid. The strongly acid odor of the sweat in certain rheumatic attacks is thought to support this theory. The excess of lactic acid is variously explained by the overwork of the muscles of which it is the normal product, and by the imperfect oxidation of the

muscular tissue and its product into lactic acid ($C_3H_4O(OH)_2$), instead of carbonic acid ($CO(OH)_2$). But in spite of the perfect theory, there is the fact that as a rule no special increase of lactic acid can be found in blood or urine in acute rheumatism and the improbability that an excess of this acid caused by sudden excessive muscular waste could be kept up during a long attack of acute much less of chronic rheumatism. Again the comparative immunity of sucklings in which there is the greatest opportunity for the production of lactic acid, would imply that that alone cannot be accepted, as the one essential cause of the affection. That its excess in the system will aggravate rheumatism, or even produce it under favorable circumstances must be freely acknowledged; also that acidity of the saliva is a marked feature of rheumatism.

The acid phosphate of soda may be assigned a somewhat similar rôle. In strongly predisposed subjects the ingestion of citric or other organic acid will sometimes precipitate rheumatic articular pains.

Still other products, the result of imperfect oxidation or metabolism must be allowed a place as probable factors in rheumatism. The occurrence of gout in connection with the excess of uric acid in the system is strongly suggestive of this, and the frequency of muscular rheumatism in pampered, overfed dogs with diseased livers and abnormal hepatic products, seems to give further support. Even in man, the subject of acute rheumatism, often leads a luxurious life and suffers from inactive or disordered liver, while in man and animals alike, a low grade of health and imperfect functional activity of important organs, are often precursors and accompaniments of acute rheumatism.

Neuropathic theory. The doctrine of a neurotic cause originated by Dr. Mitchell, of Philadelphia, has considerable basis in theory. The primary chill tends to nervous derangement, which may readily affect the overworked or already diseased and debilitated joints. Similar peripheric disturbance of nutrition occurs in locomotor ataxia in which the central nervous lesion is very evident. There is a strong disposition in rheumatism to show a bilateral symmetry, which points directly to a central nervous control. The frequent violence of the pain, disproportionate to the moderate structural changes, points in the same direction,

and the free perspiration present in some severe attacks, points alike to its origin in cutaneous chill and to derangement of the centres presiding over perspiration. It may be added that the development of rheumatic symptoms in the advanced stages of infectious diseases, when the toxins are accummulating in the system, suggests that they are the direct result of a toxic action on the nerve centres. In man the influence of severe nervous shock in developing and maintaining rheumatism is recognized.

Theory of infection. This has been advocated by Cornil and Babes and Friedberger and Fröhner. The former quote the frequent presence of microörganisms and above all of micrococci in the liquids of the affected joints, serosæ and valvular exudates and ulcers, and even in the blood, and the occurrence of rheumatism in the course of various septic affections. The latter quote Auer and others as to the frequent supervention of articular rheumatism on the retention of the afterbirth in the cow. The weak point in these theories is the multiplicity and variety of the microbes found in the exudates. Either we must accept the rheumatic lesions as an occasional result of many microbes, which habitually act differently on the system, or we must look upon them as mere accessory causes or accidental complications. It might even be, that the invasion of these microbes are made possible by the inflammation and debility of the tissues, without being directly chargeable with the rheumatic process. Even then there remains the possibility that a specific microbe is present, which by reason of its infinitesimal size, or other physical property, has as yet escaped recognition. If such specific microbe is present, it manifestly requires a very special predisposition, since it is not seen to advance from one individual to another unless such favoring conditions are present. The symptoms and lesions of rheumatism are not incompatible with the idea of such an essential, specific germ, but as yet no such germ has been satisfactorily demonstrated as present in all cases.

Local injuries. Rheumatism seems to attack by preference parts that have already been debilitated by disease, a disposition which is also true of distinctly microbian infections. A pre-existing sprain, blow, bruise or an inflammation arising from any other cause seems to practically invite the localization of the morbid process at that point, and thus what was at first a purely traumatic

lesion becomes the seat of active and perhaps permanent rheumatism. Unusual overwork and fatigue of given joints and muscles induce a similar pre-disposition, and habitual overexertion, sprain, injury or inflammation affecting repeatedly the rheumatic organ tends to fix the process in chronic form.

Articular lesions. These tend to concentrate as a form of inflammation in the synovial membranes, but usually implicate all the constituent structures of the joint, capsular and binding ligaments, cartilage and fibro-cartilage, articular lamella and osseous tissue. The synovial membrane may show only slight hyperæmia, or in severe cases it may be congested, red, thickened or even extensively infiltrated with a serogelatinoid liquid. These lesions are most marked around the line of attachment on the articular surface and in the synovial fringes. The synovia is usually in excess, distending the capsule and is whitish, opaque, flocculent or more or less deeply colored with red. It contains flakes of fibrine, leucocytes, albumen and it may be red blood globules. Pus cells are usually absent unless in distinctly infective cases. Coagula and false membranes floating from or adherent to the solid tissues, may be present in considerable amount and if these become organized they tend to lay the foundation for future stiffening and rigidity. In and beneath the serosa, cell proliferation may go on actively, especially in the synovial fringes. The synovia is usually neutral or slightly alkaline, though in rare instances it has proved to be acid.

The inflammation of the synovial membrane of the joint often extends to those of the adjacent tendons, implicating at the same time the tendons and their fibrous sheaths. Softening and rupture of the tendons have been noted by different observers (perforans, Oger; gastrocnemii, Trasbot: suspensory ligament, Ollivier).

The articular cartilages and the fibro-cartilages may be the seat of congestion, with ramified or uniform redness, and areas of swelling, softening, absorption, erosion and ulceration, the ulcers varying in size from a millet seed upward. The nuclei increase in size and the cartilage cells multiply. In chronic forms calcification is not uncommon.

The articular lamella and subjacent bone may show inflammation with increased vascularity, softening and even ulceration.

In the chronic forms on the parts denuded of cartilage, the surface of the bone may show the clear, polished condition known as eburnation.

The binding ligaments may show congestion, thickening, exudation, softening, with increased rigidity, or in some cases relaxation. Calcification is not uncommon in chronic cases. The soft parts around the joints are often extensively infiltrated and swollen.

In *horses* these lesions are specially common in the fetlock, but occur also in the knee, elbow, shoulder, hips, stifle and hock. In *cattle* they tend to attack the same parts with a preference perhaps for the hocks and fetlocks.

Changes in the blood. The blood becomes profoundly altered, the albumen reduced, the fibrinogenous elements increased (5 to 10 parts per 1000 instead of three), the red blood globules disappear (in man 2,850,000 per cubic millimeter instead of 4,500,000), the hæmoglobin and oxidizing power of the blood are reduced by about 50 per cent., the leucocytes are relatively increased, and coagulation takes place with unusual firmness, a cupped surface and an excess of buffy coat. No excess of urea, uric acid, nor lactic acid, has been found, nor has acidity been found save in very rare cases. In rare and severe cases petechiæ have appeared on the skin and mucosa.

Lesions of the heart. The implication of the fibrous structures of the pericadium and heart and especially of the valvular structures is a common lesion, and to be dreaded more than all others. In all animals this tendency to cardiac lesion is well marked, but especially in solipeds in which the great demands made on the heart during rapid paces, heavy draught, jumping, etc., severely strain the cardiac mechanism. In dogs there is not only the violent exertion and high blood pressure, but also the great irritability of the nervous mechanism presiding over the heart and the tendency to irregularity and intermissions in the rhythm and palpitations even when the organ is sound. There is every reason to conclude with Trasbot, Magnin, Heu and Laurent that in many cases the heart is primarily attacked, and that this heralds the articular rheumatism. In other cases undoubtedly the cardiac affection is secondary, following the articular attack.

Endocarditis is the most frequent, being found in a large proportion of fatal cases, and thickening of the valves, wart-like exudates and coagula are especially common. The clots may fill nearly the entire ventricular cavity, or at times the auricular, and show a preference for the left side, probably because of the more vigorous systole and the higher blood tension. The clots as a rule are firmly adherent to the diseased valve. Ulceration of the valve is rare in rheumatism. Other parts of the ventricular endocardium may be involved, becoming red, congested, rough or thickened, with adherent blood clots.

Pericarditis is less common though it may exist on either or both the cardiac and visceral folds. It is shown by vascularization, thickening, fibrinous exudate, and serous effusion. Haycock found in a horse a quart of reddish serous exudate with floating fibrous shreds and false membranes. Pus has been found in exceptional cases manifestly indicating a complex infection. Like endocarditis it may precede, follow, or coincide with an articular attack (Leblanc, Cadeac).

Myocarditis is usually seen as a complication and extension of rheumatic inflammation of the visceral pericardium, or of the ventricular endocardium. The muscular tissue may appear parboiled and friable, and shows granular or fatty degeneration.

Pulmonic and pleuritic lesions. Embolism of the lungs and pleura may occur from the transference of clots from the right heart, yet the sequence is much more commonly an articular rheumatism following infective disease of the lungs. Cadeac suggests that the impaired nutrition in pneumonic and pleuritic animals predisposes to the rheumatic arthritis, and again that the microbes of the infectious chest affection, colonizing the joints and other synovial sacs, determine the rheumatism. This last theory has the weakness of identifying influenza and contagious pneumonia with articular rheumatism, and is negatived by the experience that these two pulmonary affections never develop *de novo* from simple rheumatism. The rheumatism which follows influenza and contagious pneumonia therefore must either be considered as a pseudo-rheumatism, or a rheumatism occurring only concurrently and accidently with the pulmonary affection. Apart from this, pleurisy or even pneumonia occurs as a simple extension from a rheumatic pericarditis.

The occurrence of the rheumatoid affection as a complication of influenza and contagious pneumonia in the horse, usually appears not earlier than 15 days after the outset of the pulmonary affection and may be delayed, according to Palat, for 102 days. Palat who had excellent opportunities for observation in army horses found that about one in ten was the ratio in which the rheumatic affection followed these pulmonary diseases.

The pulmonary lesions in these affections are essentially those of uncomplicated influenza, or contagious pneumonia.

Cerebro-Spinal Lesions. Nervous disorders are occasionally seen in rheumatism in man (dullness, prostration, delirium, coma, spasms) and traced in different cases to hyperthermia, congestion, exudation, embolism and toxins. In a few cases in the horse, cerebral complications have been observed. Olivier saw a horse with lachrymation, closed eyelids and hot, tender forehead, which showed at the necropsy articular inflammation, and sanguineous effusion in the cranium, encephalon, frontal and maxillary sinuses and ethmoid cells. Jacob records two cases (mare and horse) in which rheumatism was complicated by meningo-encephalic congestion but without necropsy as both recovered.

Digestive system. In man rheumatism has been exceptionally preceded by pharyngitis, dysphagia, and diarrhœa. In the horse Haycock has seen concurrent congestion of the pharyngeal mucosa, Olivier congestion of the stomach and intestine, and Jacob diarrhœa and abdominal pain, Leblanc and Palat record cases of peritonitis accompanying articular rheumatism in the horse.

SYMPTOMS OF ACUTE ARTICULAR RHEUMATISM IN THE HORSE.

Chill, hyperthermia, lameness in fetlock, hock, knee, shoulder, stifle, hip, elbow, tendons, sheaths, bursæ, bilateral, shifting, swelling, heat, tenderness, decubitus, joint semiflexed, pain on extension or flexion, signs of cardiac disease. Course: rapid to recovery, improvements and relapses, metastasis, debility.

The attack is usually sudden and marked by a slight chill, shivering or staring coat with hyperthermia and lameness. The temperature

may vary from normal to 107° or 108° F. in severe attacks. In many cases the fever and lameness appear simultaneously, the former being in ratio with the extent and severity of the latter, but not infrequently the elevation of temperature precedes the articular symptoms, and then it is to be considered as concurrent with the internal lesions—cardiac, pericardial or otherwise. In other cases the articular lesions and lameness precede by several days the appearance of the fever. So far as we know the fever never antedates the occurrence of some local lesion, external or internal.

The joints affected are very varied. The fetlock is the most frequently attacked, but some of the other larger joints, the hock, knee, shoulder, stifle, hip and elbow are often involved or exclusively affected. The adjacent tendons and their synovial sheaths are very often implicated; the attack is very prone to show a bilateral symmetry, the same joints (right and left) on the corresponding fore or hind limbs, suffering at once, or, as in the case of the fetlocks, all four are simultaneously attacked. A joint that is weak by reason of previous injury or disease is especially liable to suffer, and is then less likely than joints that had been previously healthy to undergo speedy improvement. When the symptoms wander from joint to joint or from joint to muscle, or fascia or tendon, the disease in its earlier seat seeming to undergo almost complete resolution, while it advances with great intensity in the newly affected joint or part, the occurrence is highly significant. These transitions often take place with great rapidity. Thus the centre of suffering may shift from one joint to another in an hour, (Magnin, Cadeac), or from a limb to the loins in a quarter of an hour (Lewis). The fact that the inflammation remains fixed in one or several joints, is not, however, proof of the absence of rheumatism. A joint with a primary weakness or injury may remain the seat of disease through even a chronic rheumatism.

The affected joint is usually swollen, hot and tender the tenderness being as a rule greatest where the capsular ligament is pressed upon. These symptoms are very evident in joints that approach the surface, and obscure in such as are thickly covered by muscle (shoulder, hip). The swelling is soft, or tense and elastic (especially over the synovial membrane), or œdematous

and pitting on pressure. On white skins sparsely covered by hair there is marked redness and congestion, the veins standing out prominently and the arteries above the joint pulsating strongly. When the tendinous sheaths are involved, they stand out as elastic lines following the course of the tendons, and with more or less pasty swelling adjacent.

In some cases, however, the swelling may be entirely absent, and the trouble is to be located only by the local tenderness and pain during motion.

Small, hard, pea-like, subcutaneous nodosities were first noticed by Floriep, in 1843, in rheumatism in man, and have been met with in different cases in the horse. Rodet fils met with great numbers of these nodules crepitating under pressure, in a horse that had suffered for months from chronic rheumatism.

Acute pain on moving the affected joint or tendon is a most characteristic symptom. The horse goes dead lame, walks on three legs, or with great stiffness, and avoids as far as possible all flexion of the joint. If left alone the animal stands stock still, never moving from the place, or in the worst cases lies down and refuses to rise. If compelled to walk his suffering is shown by hastened breathing, dilated nostrils, anxious, pinched countenance and low plaintive neighing. The affected joint is held semiflexed, to relieve the tension, the pastern is habitually more upright, and if the foot is lifted and the affected joint bent or extended, the animal winces, or resists, and tries to draw away the limb and groans. The movements of the affected joint in walking or under manipulation, are often attended by cracking which may be both felt and heard. It has been variously attributed to lack of synovial lubrication (Cadeac), and ulceration of the articular cartilage (Lafosse), yet it may occur from the constrained position assumed, as in the case of a man attempting to walk noiselessly on tip toe, and in other cases from the extension of false membranes, or of rigid or contracted binding ligaments.

Rheumatism of the fetlock and sesamoid pulley, as the most common form in solipeds, demands a special notice. The swelling of one fetlock, of the two fore, of the two hind or of all four at once, extends beyond the limits of both joint and sesamoid pulley and may form a general engorgement or *stocking* which serves to hide the synovial distension. Pressure however shows that while

all is tender, the extreme tenderness is referable to the joint, the synovial sheath of the flexors, to the flexors or suspensory ligaments. As the general swelling subsides the rounded or ovoid synovial distensions become more patent. The swelling and tenderness may extend to the knee in which case the synovial membrane of the carpal arch is especially distended and tender from the carpus down, or in the hind limb the synovial membrane of the tarsal arch is distended showing in this case on the inner and outer sides in front of the calcis, and not infrequently implicating the summit of that bone as a capped hock.

In rheumatism of the shoulder the coraco-radial tendon and pulley may be involved, causing a diffuse swelling on the point of the shoulder. If the hip is the seat of disease the median gluteal may suffer, causing an indefinite swelling over the joint. If the stifle is affected the patellar capsule suffers and not infrequently the tendon and pulley of the flexor metatarsi are involved.

The most important internal complications, pericarditis and endocarditis, are manifested by their usual symptoms, sharp, variable, irregular, unequal or intermittent pulse, blowing murmur usually with the first heart-sound, oppressed breathing, and it may be muffled heart sounds, or dropsies.

Course. This is exceedingly uncertain. Some cases make a rapid progress to complete convalescence ; others make partial improvements interrupted by relapses ; others have the morbid process subside in great measure in one joint or organ only to reappear in full force in another ; others leave complications on the part of the heart especially and are rendered permanently useless. Even should the heart escape, the health often suffers so much in connection with the destruction of the red globules, the malnutrition, and the local disease, swellings and distortions of the joints that a perfect recovery seems distant and problematical.

SYMPTOMS OF CHRONIC ARTICULAR RHEUMATISM IN THE HORSE.

Larger joints, muscles, heart, false membranes, indurations, thickenings, calcifications, remittent, weather changes, cold, damp beds, winds, open windows or doors, draughts, cold sponging, clipping. Diagnosis : lameness variable, shifting, electric and meteoric storms.

Chronic rheumatism may be a sequel of the acute, or it may occur from the same causes acting with lessened force, or on a less susceptible animal. It tends to attack the larger joints especially, though it may implicate the muscles as well. Coincident affection of the heart is less common than in the acute, and when it does arise seems to advance slowly. It is liable to cause permanent distensions of the affected joint capsules, as well as false membranes, articular abrasions, degenerations and ulcerations and less frequently bony enlargements and calcifications, the latter implicating the soft tissues in the vicinity.

The attendant lameness is liable to be remittent or intermittent, subsiding in warm buildings and during genial, clear sunny weather, and relapsing in connection with cold, raw nights and mornings, exposure in the dew or rain, and before and during great changes of weather. Cold, damp beds, chilling draughts between open doors or windows, washing with iced water, sudden intense cooling of the body after perspiration, clipping during cold weather, any cause of sudden rigor, when followed by stiffness, lameness and articular swelling, serves to identify the latter as rheumatic. Even the warmth induced by judicious exercise, may cause improvement, so that a horse, starting out stiff or lame, may drive out of it after going a mile or two. The formation of subcutaneous nodules, though rare, appears to be more frequent than in acute rheumatism.

Diagnosis is to be based largely on the variability of the lameness at different times, its propensity to shift from place to place, its manifest association with exposure to cold, and with the immanence of electric storms or change in the barometric pressure, and its improvement under genial weather, warmth and comfort.

SYMPTOMS OF MUSCULAR RHEUMATISM IN HORSES.

Under usual causes, muscles tender, stiffness, groaning, loins, quarter, shoulder, neck, chest.

Developed under conditions similar to those causing rheumatism of the joints, rheumatism of the muscles tends to attack those of a particular region, and to continue in these throughout the attack rather than change to others. The affected muscles are very tender to the touch, but usually show no swelling nor heat. The muscles are relaxed and tend to atrophy, fever is little marked, there is comparatively little tendency to the implication of the heart, and the suffering and stiffness vary with the variation of the weather, or with electric or barometric changes. When generalized, however, fever may supervene, and the joints may be implicated (Thompson).

When the *loins* are affected they become extremely tender to the touch, and the horse shows great stiffness, and groans when made to walk and above all when turned or backed. He does not, however, show the unsteadiness in gait and tendency to stagger that is shown in sprain of the loins, and there is no history of a slip, fall or injury, but an unmistakable connection with cold, exposure, change of weather, or overfeeding on grain.

When the *gluteal muscles* are attacked there is intense lameness, and dragging of the hind limb, with an acute sensitiveness of the skin of the region, which characterizes neither disease of the hip nor of the trochanterean bursa.

When the *scapulo-humeral muscles* are the seat of disease, there is a marked stiffness, shortness of step, drooping of the head, and great tenderness of the skin and muscles to manipulation or the use of the currycomb. Like the other cases named it occurs suddenly, without evidence of accident, but bearing a relation to cold or other change of the weather, and is better or worse as the weather is more or less genial.

When the *cervical muscles* are affected (torticolis), the same features are noted, the absence of traumatic cause, the presence of a meteorological one, or at least of cold or wet, the respons-

iveness of the disease to the state of the weather, and to revulsive agents applied to the part. The neck may be held rigidly in one position, to one side or elevated so that there is great difficulty in getting the nose to the ground.

The *costal muscles* are less frequently attacked (pleurodynia), but the same general principles guide in diagnosis.

Seidamgrotzky alleges the constant existence of acidity of the urine in muscular rheumatism. This may be attributed to the active trophic changes going on in the muscles.

SYMPTOMS OF ACUTE ARTICULAR RHEUMATISM IN CATTLE.

Sudden onset, hyperthermia, chill, fever, acid saliva, decubitus, does not stretch on rising, lameness, joints involved, metastasis, variability, morning and noon, suppuration, walking on toe, secondary articular rheumatism. Course : muscular symptoms, cardiac, pleuritic, digestive. Chronic. Muscular rheumatism : of back, loins, shoulder, quarter, neck. Changes in blood and nutrition.

There is a sudden attack with constitutional disorder, chill, staring coat, cold horns and ears, dry muzzle, impaired appetite and rumination, acid saliva, constipation, thirst, hurried breathing, hard accelerated pulse and more or less hyperthermia. Then there may come reaction with surface heat and glow. The patient inclines to lie and when raised fails to stretch the back or the hind limbs, stands with arched back, and walks stiffly and with more or less lameness. The joints attacked may be determined by local strain, compression on concussion, hence the frequency of lesions of the knees and fetlocks. Yet any of the great joints of the limbs may suffer,—hip, stifle, hock, shoulder or elbow—or several may be affected at once. The disease may extend from one joint to another, may improve in one or more, only to suffer a relapse, and may oscillate better and worse according to the state of the weather or the exposure to cold or warmth. Often almost helpless in the early morning, the patient improves greatly in the heat of the sun.

The affected joint is swollen, distended with liquid, hot and tender with considerable infiltration of the surrounding tissues, in-

cluding the tendons and their synoveal sheaths. Suppuration is much more common than in the same affection of the horse appearing to be due to a complex infection with pus microbes. In walking in severe cases the foot of the affected limb is planted with great care and caution mainly on the toe and there appears to be exquisite suffering when weight is thrown on it, so that the fetlock and knee may knuckle over and the patient comes to the ground. Great infiltrations, fibroid, and other hyperplasias and even calcifications are not uncommon.

Cadeac describes as secondary articular rheumatism, those infective inflammations of the joints that follow on parturition, abortion, omphalitis, enteritis, etc., but it is manifest that these are special disorders due to the presence of the microbes of specific diseases or their toxins and should be described with these rather than with rheumatism.

The *course* of acute rheumatism in the ox is very uncertain. Mild cases may recover in a few days. In others the lesions become extensive, great hyperplasia and induration occur around the joint and permanent stiffness and even anchylosis may supervene. The occurrence of temporary improvements and relapses is a common feature. The extension of the disease to other joints, tendinous sheaths, muscles and even internal organs is to be dreaded. Extreme tenderness of the back and loins when handled or pinched, with groaning is a marked feature especially in cold and damp times or in early morning. Cardiac complications show themselves by shortness of breath, palpitations, hard intermittent, irregular or unequal pulse, blowing murmur with the first heart sound, and other signs of circulatory trouble. Pleuritic, pulmonic and abdominal complications are also to be looked for. The costiveness by which acute rheumatism is ushered in, becomes complicated by congestion of stomach and intestine, and impaction of the first and third stomachs, great dullness, anorexia and even nervous disorder. Colic and even diarrhœa are occasional consequences.

Many cases subside into a *chronic form* which shows a variable condition, better and worse, according to the condition of the weather, the exposure to cold and damp, and even the changes of diet. This may last throughout life.

SYMPTOMS OF MUSCULAR RHEUMATISM IN CATTLE.

This may set in with the same abruptness as articular rheumatism, the animal in the morning after a wet, dewy or frigid night showing general stiffness and lameness with extreme sensitiveness of the skin and muscles along the back and loins. The animal moves slowly and stiffly, grunting perhaps at each step and shows inappetence, fever, dry muzzle and costiveness. This is essentially *rachialgia* or *lumbago*. Pandiculation on rising is entirely omitted.

Not infrequently the *muscles of the shoulder* are mainly affected and become exceedingly tender to manipulation. The patient seeks to remain recumbent and when raised will get up on his hind parts and remain thus for some time resting on the knees before he can be made to get up in front.

When the *muscles of the croup* are attacked the mode of getting up is reversed, the animal rising first on its fore feet and remaining for a time sitting on its haunches or resting on the hocks before it gets on the hind.

If the *muscles of the neck* are involved there is the same stiffness, soreness, tenderness and twisting to one side or rigid elevation of the neck as seen in the horse in similar circumstances.

In any case there is a tendency to extension or shifting from one part to another, and notably to the implication of the tendons, synovial sheaths and joints. This is especially the case in the acute type, while chronic rheumatism may remain long confined to the groups of muscles which are first attacked. In the acute forms too there is the greatest liability to internal complications not only cardiac, but according to Cruzel abdominal and thoracic as well.

A fatal result is rare, but the impairment of appetite and digestion, the constant and often severe suffering, the destruction of the red globules, and the malnutrition, and increased and perverted metabolism as shown in the pallor of the visible mucous membranes, the steady loss of condition and advancing emaciation, the rigid, dry, scurfy, hidebound skin, tends to wear out the subject or render it unprofitable. In the chronic form it may last for months.

SYMPTOMS OF RHEUMATISM IN SHEEP.

Articular rheumatism seems to be very rare in mature sheep, while it has been recorded in lambs. Muscular rheumatism on the other hand has been seen in connection with untimely shearing, exposure to cold storms and cold, damp folds. The back and loins, are tender to the touch, or in other cases the neck or hind quarter, the limbs are carried straight and rigid, the animal moves slowly and stiffly, falls behind the flock, and is found alone, unthrifty and emaciated. It usually terminates in recovery though it may cause chronic disease and distortion of the affected joints or it may even prove fatal. The usual tendency of the morbid process to shift from joint to joint or to muscles, is here again characteristic.

SYMPTOMS OF ARTICULAR RHEUMATISM IN SWINE.

Climatic influences. Rheumatoid. Joints attacked. Muscles. Decubitus. Stiff, rigid, steps on toes, grunts, swelling, heat, tenderness, chaps, cracks, suppurations, inappetence, emaciation, metastasis, cardiac disorder. Duration ; course. Chronic form. Muscular form. Diagnosis from trichinosis. Connection with arthritis. Metastasis. Remissions.

The pig which shows an extreme sensitiveness to climatic vicissitudes and cold winds, fleeing instantly to his lair on their advent, is yet protected by his subcutaneous fat, so that he is not a frequent victim of simple rheumatism. Leblanc attributes it to unwholesome pens. Chaussade to too rapid fattening (overfeeding). Rheumatoid attacks are very common at the onset of hog cholera, swine plague and other infectious diseases, when they are probably but local manifestations of the general infection.

The lesions are mainly concentrated in the stifle, hock, knee and fetlock. In some cases the dorsal and lumbar muscles suffer and there is arching of the back with great tenderness on manipulation. In other cases the muscles of the quarter or shoulder are involved as shown by their stiffness and extreme sensibility to touch.

The pig is found down, indisposed to rise, and when up, stands drawn together with limbs rigid and feet resting on the toes. He will often point one toe to the ground repeatedly, before resting on the foot, or shift the weight uneasily from foot to foot. If moved he grunts plaintively and if handled squeals.

The affected joints may be surrounded by hot tender swellings or they may be nearly normal in outline, but they are always very sensitive to pressure and above all to flexion and extension, and the skin is usually hyperæmic and red. There may be engorgments of the lymphatics on the inner side of the limbs, and chaps and cracks in the flexures of the joints. Suppurations may follow (Graignard) suggesting a complex infection.

There is little appetite and though the disease becomes subacute or chronic there is a steady loss of condition or at least a failure to thrive.

Benion's reference to a coincident or sequent inflammation of the respiratory or digestive organs and Spinola's similar reference to pleurisy are strongly suggestive of swine plague and hog cholera. Any manifest disposition to shift from one part to another and any concurrent disorder of the heart, other than simple palpitation is strongly confirmatory of rheumatism.

The disease tends to recovery in from four to twenty days, or to pass into the *chronic form*. In this state the symptoms are materially mitigated. Fever is absent, but the appetite, digestion and assimilation are poor, the animal remains stunted, emaciated or unthrifty, there is a disposition to lie most of the time under the litter, and when up it moves stiffly with short steps, semi-flexed joints and upright digits. Sometimes the joints are permanently swollen and rigid by reason of thickening and shortening of the binding ligaments, by the organization of false membranes or by anchylosis.

Muscular Rheumatism in Swine. This appears to be rarely seen as an independent disease, but appears at times to coincide with the arthritic form. In such cases the back is arched and very sore to the touch or to pressure. It must be distinguished from the muscular soreness of trichinosis which occurs in infested localities, after trichinous food or water, is preceded by digestive disorder and diarrhœa, and by the passage of

the nearly microscopic worms in the stools, and is independent of arthritis.

Muscular rheumatism leads to atrophy of the muscles, especially those of the quarters, and this may resemble, somewhat, partial paraplegia from disease of the spinal cord. Its connection with arthritis, its tendency to shift from place to place, to undergo ameliorations and relapses, and its exquisite tenderness, serve to distinguish it from paralysis.

SYMPTOMS OF ARTICULAR RHEUMATISM IN THE DOG.

Articular rheumatism rare. Femoro-tibial joints, bilateral, remissions. Exudation, swelling of joint; muscular atrophy, weakness, swaying, staggering, falling, paresis. Chronic, muscular rheumatism common, back, loins, neck, general, stiff, painful movement, decubitus, muscles tender, yelps, stiff neck, wry-neck. Masseteric. Painful defecation and urination. Metastasis. Cardiac symptoms. Pleurodynia. Digestive troubles. Emaciation, weakness, atony, paraplegia. Diagnosis from strongylus, stephanurus, and cysticercus.

This affection seems to be rare in the dog. What is known as rheumatism in this animal, consists in an inflammation with hyperplasia around the articular ends of the long bones, the new material being partly fibrous and partly calcified. It shows a special predilection for the femoro-tibial and confines itself mainly to the inner side of the head of the tibia. Here the swelling may reach the size of a walnut. The whole head of the tibia and lower end of the femur are however often involved, entailing a general enlargement of the joint. It follows the general rule of rheumatism in usually attacking both stifle joints at once, and also in alternate ameliorations and relapses. Less frequently other joints are affected. In all such cases the joints become overdistended and swollen, partly by synovia, and partly by surrounding exudate, the muscles of the quarter and thigh become atonic, soft and flaccid, and are steadily atrophied. The dog shows a lack of strength in the hind parts, swaying, staggering or even falling, and advancing to a marked paresis. The malady follows a chronic course, lasting for months, a year, or more.

SYMPTOMS OF MUSCULAR RHEUMATISM IN THE DOG.

Muscular rheumatism is common in dogs. It is most common and most marked in the back and loins, though the neck may suffer, or the disease may be generalized. It is painful to move and the subject seeks to be as much as possible undisturbed. He walks stiffly and slowly, carrying the limbs with as little movement of the joints as possible, and in bad cases yelps occasionally from sudden pain. He can no longer be tempted to go up or down stairs or to make any special effort. When touched on the back or loins he will wince, cry out, or even snap at the offender. In some cases the pain is so acute that even a feint to touch the back will draw out a yelp. If the neck is affected it may be held so stiffly that the dog can barely reach the ground to find his food, or if unilateral the head is turned to one side. Even the muscles of the jaws may be affected, causing prehension and mastication to be difficult and imperfect. Defecation and urination are also interfered with and the straining may draw forth plaintive cries.

The rapid shifting of the morbid process from one group of muscles to another is often very striking, and if one pronounces on the exact seat of the disease, it is liable to be speedily rendered inexact by a sudden change of place. There is further a great disposition to the implication of the heart and especially the valves. This is shown by irregularity and inequality of the pulse by intermissions and palpitations, by a blowing murmur with the first heart sound and by oppressed breathing.

Short, shallow breathing is also caused when the intercostal muscles are attacked (pleurodynia). Various digestive troubles are also common, to which the difficult defecation and impacted rectum largely contribute.

Emaciation makes more or less progress, and the muscles of the hind parts especially become weak and atonic until marked paresis or actual paraplegia sets in, and the hind limbs are extended backward and dragged helplessly. In fat, sluggish, overfed and pampered animals the lack of control of the hind limbs may come on at an early stage. Stiffness due to strongylus gigas in the kidney or stephanurus or cysticercus cellulosa in the lumbar muscles must not be mistaken for rheumatism.

PREVENTION AND TREATMENT OF RHEUMATISM.

Prevention. Avoid known causes, untimely clipping, exposed buildings, over-fatigue, chills, cold rains, dews and frosts, disorders of liver and bowels, sweets, spiced food, over-feeding, constipation, torpid liver, injuries to joints or tendons. Treatment: warm stall and clothing, laxative food in moderation, purgatives, aloes, castor oil, jalap, saline enemas, colchicum, alkalies, trimethylamine, acetate of ammonia, salicylic acid, salicylates, salicine, oil of gaultheria, salicine for debilitated. Large doses hourly or every two hours. Less effective in chronic cases. Heart failure. Salol. Salophen. Phenocoll. Antipyrin. Acetanilid. Pilocarpin. Tartar emetic. Dover's powder. Ammonium acetate. Chamomile. Boneset. Hot baths and packs, with cold on head. Hand-rubbing. Hot drinks. Nauseants. Hot iron. Anodyne liniments. Rubefacients. Blisters. Salicylate of methyl and other salicylates. Quinine and potassium iodide. Tincture of muriate of iron. In chronic cases, tonics, cod liver oil, arsenic, guaiacum, potassium iodide. Essential oils. Electricity.

Prevention. This consists in the avoidance of all known causes of the disease and must vary to some extent for different genera of animals. The avoidance of cold and exposure, of clipping at unsuitable seasons, of exposed sites for buildings (north and northwest exposures, narrow valleys and ravines), of over-fatigue, of perspiration and subsequent chilling, of cold rains, dews and frosts, of inactivity, or habitual overloading of the liver and bowels, and of local injuries of joints or tendons. House dogs especially should be protected from sweets, spiced food, frequent feeding, constipation and torpid liver.

Treatment. One of the most important considerations is a warm stall or building, or warm clothing including loose woolen bandages on the legs, in the horse. Laxative food is called for.

In acute cases and especially in fat pampered dogs, and in all cases associated with torpid or disordered liver, a preliminary laxative will be of great service, and others should be given later as demanded. The horse may have aloes or salines, and saline enemata may be given to all animals when called for throughout the progress of the disease. Pigs may take 1 or 2 drops croton oil, or like dogs they may be given castor oil or jalap. Torpid liver and constipation must always be carefully guarded against.

To cut short the attack much reliance was formerly placed on colchicum which increases the elimination of solids in the urine, and on alkalies, which beside the theoretic antagonism to acidity are at once chologogne and diuretic. The action was somewhat slow but on the whole satisfactory, usually abating the suffering very materially in the course of a few days. To the horse or ox ½ dram doses of colchicum were given daily in combination with 4 to 6 drams of bicarbonate of soda ; pigs of 100 lbs. may take 1 grain of the former to 10 grains of the latter ; a shepherd's dog may take half the amount just named. Trimethylamine proved even more effective than colchicum, and acetate or citrate of ammonia, soda, or potassium was often substituted for the carbonate.

But the modern treatment of rheumatism dates from 1876, when the introduction of salicylic acid and later sodium salicylate, salicine and ol. gaultheriæ gave to such treatment an efficacy previously unknown. Salicylic acid acts very harshly on the gastric mucosa, and with sensitive stomachs is advantageously replaced by sodium salicylate, into which it is transformed in any case in the blood. Salicine which is held to be transformed into salicylic acid in the system, is specially recommended for its bitter and tonic action exercised in the stomach and prior to such transformation. In debilitated subjects, therefore, and in those that suffer from the characteristic rheumatic reduction of the red blood globules it would be somewhat preferable. As a prompt and effective anti-rheumatic agent however it appears to be somewhat less reliable than sodium salicylate or ammonium salicylate. Ol. gaultheriæ may be better borne by the stomach of the dog and pig than the salicylates, the dose being 10 to 15 drops thrice a day.

The secret of success with all of these salicylate compounds, lies in the speedy saturation of the system with the drug, rather than in its moderate and continuous administration. The horse or ox may take ½ oz. repeated every two hours for ten hours if relief is not obtained earlier. The pig may take 20 grains, and the dog 5 to 10 grains at similar intervals. It is not desirable, however, to continue this indefinitely, and therefore when immediate relief has been secured it is well to give the agent but twice or thrice a day, and resort in part to the alkaline treatment. If the salicylates fail to relieve when pushed energetically for ten

hours, there is reason to fear that the case is not one of genuine rheumatism.

The salicylates are less applicable to chronic cases and may be even dangerous when the heart is affected, as they tend to render the heart's action slower and weaker, and thus add to the dangers of hypo-hæmoglobin, and heart failure. A similar caution applies to an excessive use of alkalies and especially of compounds of potassium which depress the heart action.

As substitutes for the salicylates, salol, salophen, phenocoll, antipyrine and acetanilid have been largely resorted to. The first is safe and trustworthy and does not irritate the stomach nor interfere with digestion. It may be given to horse or ox in a dose of 3 drams, thrice a day, to the pig in 10 grain, and to the dog in 5 grain doses.

Hübner had good success with pilocarpine hydrochlorate hypodermically (4 grains for a 7 months colt,) but this was not equally successful in the hands of Siedamgrotzky. Other sudorifics like tartar emetic, Dover's powder, ammonium acetate, hot or spiced drinks (chamomile, boneset,) hot baths, hot air baths, and wet packs have been used successfully and may still be employed in suitable cases The opium is often very helpful in relieving intense suffering, and beside or in place of the Dover's powder internally, morphia may be injected subcutem over the affected region. The main objection to its use is its tendency to lock up the liver and bowels. Liquor of the acetate of ammonia fills at once the rôle of a potent diaphoretic, an antacid, and an eliminant.

In the use of baths and packs it is well to consider the condition of the patient. If the surface is cold with little reaction, and if the attack has supervened on exposure, or chill, persistent hot applications are indicated. Dogs and other small animals should have full hot baths lasting for 15 or 20 minutes, and while in full glow may be quickly sponged with cold water and rubbed dry in blankets, great care being taken to avoid exposure or chill when damp. Or for these and the larger animals as well, a hot air or steam bath may be applied under similar precautions. A cold wet wrapping on the cranium will tend to relieve cerebral congestion during the administration of the hot bath. In horses and cattle surface heat and sudation may be secured by active rubbing with wisps of straw, of both body and limbs, or by covering the

neck and trunk with large bags containing a small amount of chaff, sand or grain hot from an oven. Hot carminative or alcoholic drinks are excellent adjuvants, and even sedatives or nauseants (opium, veratrum, aconite, tobacco). Another resort is to pass a hot smoothing iron an indefinite number of times over the affected region. The part may be finally wrapped in cotton.

In cases where the temperature runs high, on the other hand, and when the surface glows, this dread of chill and reaction may be dismissed. For the small animal a bath starting at 70° F. may be gradually lowered to 60° or 50° F. Or a full pack may be employed, a sheet wrung out of cold water being closely wrapped around the body, and covered at all points with two or more dry woolen blankets, care being taken to avoid the entrance of air and the occurrence of evaporation from the inner, damp layer. This cools the surface and the blood returning inwards, and in fifteen or twenty minutes it should induce free perspiration. It may be kept up twenty to thirty minutes and may be repeated as often as there is a serious rise of temperature. A less energetic method is the mere sponging of the surface with cold water. In all such cases friction is a valuable accessory.

Anodynes and revulsives are often applied to the affected parts with good results. In very acute cases (especially articular), lotions and liniments of salicylic acid or salicylate of soda with laudanum, aconite, or chloral hydrate may be used. In the less violent cases camphorated spirit, soap liniment, or a combination of essential oils (gaultherium, turpentine, cajeput, origanum, peppermint) with aqua ammonia and sweet oil may secure great relief. Mustard or essential oil of mustard in vaseline is an excellent alternate. Finally active cantharidine blisters are usually most effective. These are applied over the affected joints or muscles and if the inflammation shifts to other parts it is followed up until it finally disappears. A concurrent alkaline treatment, and more important still, absolute rest, will serve to protect the heart to some extent, against a metastasis from the exterior. It has been supposed that the beneficial action of the blister is in ratio with the amount of exudate, and hence cantharides has been highly esteemed in this connection. Friedberger and Fröhner have used tincture of iodine and biniodide of mercury.

The local application of anti-rheumatic agents would embrace all the salicylates, oil of wintergreen and guaiacol, the latter mixed with an equal amount of glycerine. Methyl salicylate has been strongly recommended for external use.

Stenel covers the surface with lint smeared with salicylate of methyl ointment, and then applies a plaster bandage over all. This removes muscular spasm, pain and swelling and is rarely required for longer than a few days.

In cases in which salicylates fail, other agents have been resorted to in man and to a lesser extent in the lower animals. Greenhow strongly advocates a combination of quinine and potassium iodide internally, while Russell Reynolds has successfully employed tincture of muriate of iron in large doses repeated every three hours.

In *chronic* cases these would especially commend themselves as calculated to repair the general health and overcome the loss of hæmoglobin. In *chronic rheumatism* a course of tonics is often the best resort, and in dogs especially cod liver oil has benefitted when all else had failed. Arsenic too (3 to 8 drops Fowler's solution thrice daily) has been beneficial in both dogs and pigs. In other cases iodide of iron has been helpful. So also with gum guaiacum given in combination with potassium iodide. In such cases too, treatment by alkalies and salicylates may be called for, and close attention should always be given to secure a free action of the liver, bowels and kidneys. The local treatment recommended for acute rheumatism, (hot baths, frictions with essential oils and above all blisters of mustard or cantharides) is even more applicable to the chronic. A firm bandage over a covering of cotton wool, and a systematic application of electricity will often help. Warmth, a run at grass in a sheltered sunny paddock, moderate exercise and a nutritious and easily digestible diet are important conditions.

GOUT. PODAGRA. ARTHRITIS URICA.

Definition. Affects birds, dogs, perhaps pigs. Causes: excess of nitrogenous food, imperfect oxidation, impaired metabolism and elimination. Susceptibility of birds in confinement. Xanthin bases. Nuclein. Hepatic torpor. Contracted kidney. Affects tissues of little vascularity. Lesions: chalky deposits around joints, and in internal organs. Solubility of biurate of soda in synovia, serum, etc. Symptoms: arthritis, joint tenderness, resting on breast, hard or fluctuating swellings, desquamation, ulceration, chalky urates. Diagnosis: test for biurate. Treatment: less albuminoid diet, eliminating salts, colchicum, piperazin, surgical and antiseptic dressing.

Definition. An arthritis characterized by periodical exacerbations, by the deposit of sodium biurate in and around the joints and at times in other parts of the body, and by more or less constitutional febrile disturbance during the paroxysms.

Animals susceptible. Among the lower animals the disease has been noticed almost exclusively in **birds**, which even normally excrete so much uric acid that the liquid may be semi-solid as found in the cloaca or in the droppings. While this is a constitutional peculiarity in the bird yet it is enhanced in connection with an abundant diet of rich nitrogenous materials, as in forced feeding, and in old animals in which the eliminating action of the kidneys is more or less impaired. Ebstein has shown that gout can be produced in birds by tying the ureters. All domesticated birds, chickens, turkeys, pigeons, ostriches, geese, ducks, Guinea fowl, have been found to suffer. A case of gout has been reported in a **dog**, and Pradal has described it as existing in swine, but the symptoms given are more in accord with articular rheumatism.

Causes. The causes of gout are overfeeding especially on highly concentrated nitrogenous food, acid sweets, and in turn sweet and acid alcoholic drinks, an excess of uric acid in the blood and tissues, imperfect oxidation of albuminoids, impaired metabolism, imperfect elimination of uric acid, and impaired innervation. Probably no single morbid condition is in itself sufficient to induce the disease but a combination of several, unquestionably operate in many cases.

The uric acid theory is favored by the constant presence of this acid in considerable amount in the blood of birds, and by Ebstein's experiment in tying the ureters, but it has to face the fact that young and active birds living in the open air, and hunting for their food do not suffer, that it is usually scanty in the blood of man just before an attack, that Gilman Thompson failed to produce any symptoms of gout by injecting into the blood of animals more uric acid than the amount which they normally excrete in twenty-four hours, that the familiar symptoms of uric acid poisoning are not at all those of gout, and that the excess of uric acid in leucæmia, anæmia and pneumonia produces no such symptoms. In addition to excess of uric acid some other factor is required.

Xanthin bases (Xanthin, hypoxathin, etc.) found in the blood by various observers, are derived from albuminoids, especially nuclein and nuclein bases, including in man caffein and theine, and being closely allied to uric acid are believed to have a nearly similar action.

Various forms of abnormal metabolism are invoked as the cause of uric acid and gout, and Haig and Vaughan hazard the theory that the breaking down of the nuclein is an important factor. This and other metabolisms are attributed to the local action of the uric acid and urates, and again to a fault in innervation. The imperfect action of the liver where the uric acid should be largely resolved into the more soluble urea, and of the kidneys through which it should be promptly excreted must be attributed to a nervous source. Levison incriminates the granular, contracted, inactive kidneys.

Ebstein attaches great importance to impaired nutrition in the affected tissues which undergo necrotic changes that pave the way for the deposition of urates in their substance. This is somewhat sustained by the occurence of the local deposits in tissues in which circulation and nutritive changes are slow, and in older animals in which not only are the osseous tissues more calcic and less vascular, but the articular lamella has been formed by cretefaction of the bone and cartilage. Haig suggests that in the old, the joints are less vascular and less alkaline, and more sensitive to cold. On the other hand those in the greatest vigor of life are more ravenous, digest more actively and are in this sense

more subject to injury from excess of uric acid and allied products. Birds at this age, confined and in process of fattening are thereby exposed. Overfed, obese, lazy, old house dogs are under similar causative conditions.

Lesions. The most prominent lesions in birds are chalky concretions of urates on the articular ends of the bones and in the structures around the joints including even the tendons, with more or less inflammatory exudate and even necrosis, invading the bony tissue and articular cartilage. Abscesses may be present usually outside the bursa. Birds suffer especially in the tarsal, metatarsal and phlangeal joints, but often also in the corresponding joints of the wing, and less frequently in the joints of the trunk, and in the internal organs,—kidneys, liver, lungs, serosæ,—and skin. In these last, miliary chalky concretions and encrustations are found. In Brückmüller's case in the dog the chalky deposits of urates were found mainly on the epiphyses of the ribs, but also on the joints of the limbs.

Uric acid is always abundant in the blood of birds, and Roberts has shown that biurate of soda (the usual form of precipitate) is insoluble in blood serum, synovia and other body fluids when in excess of 1 : 10,000.

Symptoms. In birds the febrile and constitutional symptoms have not been carefully observed so that the objective symptoms in the affected joints have been mainly relied on. There is extreme tenderness marked by standing on one limb, or resting on the breast, and hence moping apart from the flock. When made to rise, the affected limb may be used to steady the body, or even to walk, with a limp, though in bad cases the sound limb only may be used. The affected joints are swollen, soft, hot, extremely tender, pitting on pressure, and later the seat of nodular yellow masses, usually hard, but sometimes fluctuating and in size from a pea to a hazel nut. The superimposed epidermis is thick, dry and scaly, falling off in flakes. At a more advanced stage the concretions may burst through the skin, discharging a buffy, granular, debris containing crystals of urates of ammonium or calcium, or of uric acid. Later still are ulcerous sores, involving the disintegrating urate nodules and the necrotic bones and cartilages. The deposits deflect the bones from their normal direction, causing not only nodular swellings on the toes

but much crookedness and distortion. As in man the disease is essentially chronic and advances slowly, with anæmia, emaciation, debility and at times diarrhœa.

Diagnosis depends largely on the recognition of the excess of urates in the deposits. These appear under the microscope as fine acicular crystals, which in the harder portions have a concentric arrangement. A portion of the concretion may be moistened with a few drops of nitric acid and evaporated to dryness. To one part of the residue is added, by means of a pipette, a drop of aqua ammonia, and to another caustic soda. The ammonia develops a beautiful purple red color, and the soda a blue or purplish blue ring (Murexide test). In tubercular joints, which are common in birds, the caseated nodule is made up of cells and granular debris, with tubercle bacilli, and though cretaceous particles may be present they fail to give the microscopic and color appearances of uric acid.

Treatment. This must be largely preventive. The rich albuminoid feeding and close confinement must be modified especially in the older birds, and eliminating agents must be given in the drinking water. The Carlsbad combination (sodium sulphate 22; potassium sulphate 1; sodium chloride 9; sodium bicarbonate 18) may be used. Powdered colchicum ¼ gr. once or twice daily during an attack, or piperazin ½ gr. twice a day. Locally, abscesses should be opened, and like any sores or ulcers, treated with antiseptics (Salicylate of sodium 75 grs., glycerine 2 ozs.; or piperazin solution 2 : 100).

SCURVY: SCORBUTUS.

Definition. Susceptible animals: pigs, dogs. Causes: unwholesome salt meat, lack of fresh food, vegetables, potassium, bad environment, unvarying diet, lack of free range, putrescent food, foul water, infection; non-recurrence. Lesions: blood black, diffluent, little rigor mortis, excess of sodium, petechiæ and extravasations, red marrow, softened, swollen, bleeding, ulcerating gums. Symptoms: Anorexia, prostration, debility, tardy movements, petechiæ, loss of bristles, ulcers, gum lesions, joint swellings, blood extravasations. Diarrhœa. Prognosis unfavorable. Treatment: correct unwholesome environment and food, wash, rich food partly green or animal, iron, bitters, arsenic, mouth wash (potassium chlorate), for suckling milk, Butcher.

Definition. Scurvy is a subacute or chronic trophic disorder characterized by debility, inanition, anæmia, swelling and bleeding of the gums, gingival ulceration, dropping of the teeth, and petechial or more extensive hæmorrhages and exudations in the skin, serosa, and solid tissues.

Animals susceptible. In past times *man* has suffered extensively in connection with unwholesome food and environment, on long sea voyages, on uninhabited islands, in military campaigns, in besieged cities, in famines, when restricted to one article of of food, etc. Among the lower animals, **pigs** especially suffer, when kept in close, foul quarters and fed a monotonous and insufficient ration. **Dogs** suffer under similar conditions, and probably other animals would if equally badly used.

Causes. Formerly it was attributed to an exclusive diet of salt meat and bread ; to excess of sodium, and deficiency of potassium salts ; to the absence of fresh vegetables ; to tainted food, etc. A broader generalization shows the Eskimo living on a pure meat diet, the Mongolian on rice alone, the Congoese on plantains, and without scurvy. Yet it cannot be denied that these various conditions undermine the general health, and prepare the system for those faulty states of nutrition which are seen in scurvy. In **pigs** the food and environment are usually chiefly at fault, the subjects have been kept closely confined in foul buildings, in a hot, moist atmosphere, and with an uniform diet of maize or other unvarying and insufficient ration. It does not appear when there is a free access to a spacious yard or open field, and when the monotonous diet can be varied by a variety of slugs and other invertebrates. Röll attaches great importance to a putrid condition of the aliments (putrescent swill). Benion has found it mainly in obese swine, the forced feeding and intestinal fermentations manifestly operating as factors. Corrupt drinking water has proved a manifest factor among men living in camps, and pigs above all other animals are subjected to this cause. Benion says it occurs in the advanced stages of measles (cysticercus cellulosa).

It is evident that unwholesome conditions of life such as the, above, contribute strongly to the affection, yet probably no one of these is by itself an effective factor. Its rapid extension among men and animals, that are huddled together in close, filthy

quarters suggests an epizoötic or infective element, and Cornevin, Hess and others attribute the disease in pigs to the germ of erysipelas. Stengel has produced purpuric disease in animals by inoculation of the extravasated blood from human scurvy patients. Müller and Babes found a slender bacillus and streptococci in the tissues of scorbutic gums. The bacillus was present in the mouth of non-scurvy persons. Boruträger found cocci in the spleen. Berthensen alleges that after complete recovery the disease does not attack the same person a second time, which, if confirmed, will go far to establish a bacteridian origin. There is considerable presumption of the existence of a microbian cause, the efficiency of which is dependent on the unhygienic conditions above stated, while these unwholesome conditions are equally non-pathogenic in the absence of the microbe.

Lesions. The blood is black and incoagulable or clots loosely, rigor mortis is slight, changes may be found in the number and character of the white and red blood globules, but are not constant, there is usually an excess of sodium salts and deficiency of potassium ones, and there is marked petechiation of the skin, mucosæ and serosæ. The bone marrow may be abnormally red and the bones fractured at the epiphyses, or carious. The addition of the gum lesions makes the case characteristic. The gums are softened, swollen, red and uneven, with hæmorrhagic discoloration, erosions, necrotic areas and ulcers.

Symptoms. Anorexia or fastidious appetite, prostration, debility and sluggish indifferent movements, are followed by the local lesions on the skin and gums. On the skin appear petechiæ, and extravasations, which often implicate the bristles, so that they may be shed or pulled out with ease, the bulbs appearing dark and bloodstained (bristle rot). These may be followed by necrotic sloughs, and deep ulcers that are slow to heal. The gums are red and swollen, with hæmorrhagic spots, and bleed on the slightest touch. Erosions, sores and ulcers are not uncommon, the tongue is dry and furred, and the mouth exhales a fœtid odor. The teeth may become loose in their sockets. Swelling of the joints, from hæmorrhage or effusion, may be noticed, and lameness or stiffness from muscular or intermuscular extravasation. Blood effusions into the anterior or posterior chamber of the eye have been noticed and paralytic or comatose symptoms from

similar effusions on the brain. In the absence of improvement the patient becomes more and more debilitated and exhausted, and death may be preceded by profuse exhausting diarrhœa.

Prognosis is unfavorable in advanced cases, and when the faulty regimen cannot be corrected.

Treatment. The first consideration is to correct the unwholesome conditions of life, purify the building and its surroundings, and allow a free range on a pasture. Subject each patient to a thorough soapy wash, and if possible allow clean running water in which a bath may be taken at will. Access to green food and invertebrates (slugs, larvæ, etc.) is important, or a varied diet of grain, middlings, bran, roots, fruits, tubers, cabbage, silage, etc., must be furnished. Iron and bitters (nux vomica, gentian,) are useful and sometimes small doses of arsenite of soda solution, or cream of tartar are useful. Acorns or horsechestnuts are recommended. For the mouth a wash of potassium chlorate, soda biborate, or potassium permanganate may be resorted to. Friedberger and Fröhner advise for the dog extract of meat in wine.

In the case of fat pigs it is more profitable to butcher at once, as soon as early symptoms appear.

In pigs or puppies brought up by hand, as in babies, the true course, is to discard milk substitutes and give sweet new milk, preferably of the genus to which the patient belongs. The important elements of cleanliness and outdoor life must not be forgotten.

GOITRE. BRONCHOCELE. ENLARGEMENT OF THE THYROID.

Definition. A non-inflammatory enlargement of the thyroid gland, independent of known microbes or parasitism.

Causes. Goitre is an endemic disorder in man and beast, though it may occur sporadically during or after a *debilitating disease*, or in animals that are overworked or out of condition. As occurring endemically all accessory factors that undermine the general health must be admitted as potent factors, though insufficient of themselves to develop the malady in the absence of

the specific cause. Thus in Europe women suffer more than man, being more *confined indoors* and being less muscular and vigorous. In New York the new born offspring of ewes, kept in close confinement during winter, may be all goitrous, while those of flocks, having a free run through the whole season, escape. Gurlt has seen the same in goats. Apart from debilitating diseases New York horses and cattle develop the greater number of cases in winter, the period of confinement and idleness. House dogs suffer more than hounds.

Poor diet has a similar effect. In Europe where the disease is very prevalent in the underfed peasant population, it is rare among the highly fed domestic animals. Bouley says it is excessively rare in animals even in the localities in which it prevails in man, and though mentioned by Lydtin, Johné, Haubner and others it is not as a common affection. In New York and Pennsylvania on the other hand it is rare in the well-fed human population, and very common in horses, mules, cattle, sheep, swine and dogs. I have known congenital goitre to prove fatal to a new-born dromedary in Central Park, New York. The long, severe winter, close confinement, and impure air, doubtless as much as the spare diet contribute to this prevalence among the animals in New York.

Intestinal worms and other parasitisms must be accepted as secondary factors, the development of goitre often going on simultaneously with the increase of the parasites.

Heredity is claimed as a cause by Möller and others, and doubtless a weak constitution transmitted from parent to offspring, is more susceptible. Apart from this the exposure of both to a common specific cause is the main factor in its production.

Locality. This must be accorded a first position in the causation of goitre, so far at least as it occurs endemically and enzootically. In England it has prevailed, in man, on the limestone hills of Derbyshire, and Gloucester (Cotswold); in Europe it is common in the Alps, Pyrenees, Savoy, Styria, Silesia, in the Black Forest and in the Rhone valley; in Asia it prevails in the Himalayas, the Altai Mountains, the hills of China, and in the Punjaub; in South America it is seen in the valley of the Oronoco; in North America in Saskatchewan, Ontario, Michigan, Ohio, Pennsylvania, New York, Vermont, Virginia and Alabama. A large

number of these localities lie on magnesian limestone or are supplied with water that has percolated through this, so that at one time the excess of magnesia and the lack of iodine were held to be the main causative factors. This contention cannot be sustained in all cases, so that the disposition is, at present, to attribute the disease to some unknown poison. This unknown poison may be present in districts apart from the magnesian limestone, yet the disease is so frequently seen upon this formation that its presence must always be looked upon with suspicion as a probable bearer of the poison, and waters bearing its products are unsuited to the victims of goitre.

Pathological Anatomy. Sometimes the swelling of the gland which appears during catarrh or pharyngitis will subside on recovery. In other cases it remains as a distinct hypertrophy. This is usually an increase of the parenchyma and dilatation of its follicles with an albuminous fluid (*hypertrophic goitre*). This may affect one lateral lobe or both. In other cases the fibrous tissue mainly increases and the gland becomes hard and resistant (*fibrous goitre*). In other cases the individual follicles become distended, and may even break into each other forming a large cavity or several with liquid contents (*cystic goitre*). In other cases there is a great increase of the vascular network of the gland so that blood alone is obtained on puncture (*varicose goitre*). Tumors of all kinds may be found in the gland, thus encysted adenoma, sarcoma and melanoma in horses, carcinoma in old dogs.

Symptoms. In *horses* there may be swelling of one lateral lobe of the gland or of both, reaching individually the size of a hen's egg or the fist, or larger. Cadeac cites cases that weighed 4 lbs. In *dogs*, *cattle*, *sheep* and especially in *swine*, the two lobes are much more closely connected, and the disease affecting both, together with the commissure, the whole may be resolved into one uniform swelling, much larger than in the horse relatively to the size of the animal, often covering the whole front of the neck, and extending into the chest. Cadeac mentions cases in the dog in which the mass weighed 4 lbs.

The smaller swellings appear in the solipeds on the two sides of the larynx, and in other animals more in front. They are mobile, but rise somewhat with the larynx in swallowing, and are

usually covered by loose, movable skin. The consistency of the swelling varies; some are soft, elastic or pitting on pressure, others fluctuate and still others are firm and resistant. Old cases that have become calcified may even feel bony. In dogs it will sometimes pulsate like an aneurism.

Functional secondary troubles are rare in solipeds. In the other animals the goitre may compress the pharynx or gullet causing dysphagia, or the larynx, trachea or recurrent nerves causing more or less wheezing or dyspnœa. Asphyxia is not uncommon in new born sheep, and goats, and Johné and the present writer have seen cases in dromedaries. The soft embryonic tracheal rings had been so compressed from side to side that respiration became impossible. Honert records a case of asphyxia in an adult horse. Cases of *roaring* in adult horses and mules and of asphyxia in adult dogs are also on record. Warz records the obliteration of the jugular in a dog, and Cadeac œdema of the lips and face.

The *course* of goitre is usually slow, extending over years, yet in young dogs it may make a very rapid progress. It will often stand still for a time, and later start a new growth under a fresh access of the cause. Spontaneous disappearance is rare.

Prevention. This is especially important in localities in which goitre is enzootic, and embraces careful attention to the general health, the avoidance of overwork, exhaustion, indoor life, lack of exercise, impure air, faulty feeding, starvation, and water from the goitrous soils. Rain water is preferable.

Treatment. First remove the various causes, and secure the best hygiene. If a change to a non-goitrous district can be had, avail of it.

Among medicinal agents iodine holds the foremost place. It may be given internally as potassium iodide, alone, or along with tincture of iodine, and applied locally as iodine ointment rubbed into the skin, or tincture of iodine painted on the surface.

Of surgical measures the simplest and best is the injection of iodine into the diseased thyroid. The nozzle of a hypodermic syringe is inserted into the gland, preferably into the largest cyst or follicular mass, and the liquid drawn off as fully as possible. It is then injected with the following mixture: compound solution of iodine one part, distilled water two parts. The

amount may vary with the size of the goitre. In cases of moderate size ½ dr. to 1 dr. is suitable. There is usually some resulting inflammation, which may be met by a wet compress around the throat. A second and third injection may be made if necessary, when the effects of the preceding one have passed off. In simple forms it is very successful. For dogs Möller recommends from 5 to 15 drops of undiluted tincture of iodine at an injection. In other cases he used a watery solution of papain (1 : 10) to be left in for 48 hours. The thyroid was then soft and, on incision, discharged its digested parenchyma as a milky fluid, and favorable healing followed.

The removal of the diseased lobe has been successfully accomplished in horses, the reservation of the other lobe, or even of the connecting commissure, being sufficient to prevent the occurrence of tetany. From the extreme vascularity of the organ it is important to ligature the arteries before attempting the removal.

In the other domestic animals in which the commissure is practically obliterated and the two lobes confluent in goitre, the excision of the mass is liable to be followed by tetany, dropsy (myxoedema), stunted development, anæmia or marasmus. If a portion of the gland is left these results do not follow. Grafting of a portion of healthy gland may correct the tetany. The hypertrophy of the gland may sometimes be arrested by ligature of its nutrient arteries, and without the dangers above named. This may be combined with the internal and external use of iodine.

EXOPHTHALMIC GOITRE.

Definition. A complex disorder manifested by hypertrophy of of the thyroid, excessive bulging of the eye balls out of the orbits, cardiac palpitations or tardy heart action and other nervous or trophic disorders.

Cadiot records a case in a horse, in low condition, with painful œdematous swelling of one fore foot, and swellings in other parts of the body, great enlargement of the left lobe of the thyroid, tumultuous heart action with beats 70 to 80 per minute, and strong visible pulsations in the superficial arteries. There was

no leukæmia and no exophthalmia, The patient died on the third day.

Jeswejenko records that of a four year old English thoroughbred which after a race showed anorexia, weakness, thirst, rapid pulse, palpitations, conjunctivitis, enlarged thyroid and after fourteen days exophthalmia with thyroid pulsations. It died in the fourth week, anæmic and exhausted. A second case in a 7 year old bitch recovered in three months under treatment with iodine.

Roder gives the case of a cow with palpitations, abnormally strong pulsations, thyroid hypertrophy and double intense exophthalmia. This persisted for four years.

RACHITIS. RICKETS.

Definition. Lesions and pathology; gastro-intestinal disorder, hepatic, splenic and renal congestion and hypertrophy, lessened blood salts, dilated arteries, hyperæmia of bone, deep red marrow, blue articular cartilage, softening of epiphyseal cartilage and under the periosteum, with hyperplasia, decrease of lime salts, bending of bone, loose periosteum; sclerosis in repaired cases. Causes: appears as if infection, lack of lime salts in food, inconstant, free phosphorus, glycero-phosphoric acid, lactic acid, oxalic, acetic and formic acids, heredity, bad air, crowding, damp soils, cold, confinement, darkness, infection, toxic matters. Symptoms: unthrift, thin neck, arched or hollow back, drooping pelvis, weariness, stiffness, recumbency, limbs not plumb, tender, swollen puffed joints, enlarged epiphyses, bent shafts, or spine, brittleness. Swine fed on potatoes or corn, "snuffles", breaking teeth, diarrhœa, bronchitis, skin eruptions, arthritis. Cattle, epiphyseal swellings, bow legs, crooked back. Dogs, bow legs. Goats. Birds, knotted thickening of bones, flexibility. Fever, colics, indigestion. Lameness shifting, intermittent, relapsing. Paraplegia. Treatment, hygienic, vigorous breeding animals, nutritious rations, rich in earthy salts, well-balanced, from sound land, rich abundant milk without excess of fat, avoid spoiled food, adapt cow's milk to foal or puppy, fresh air, sunshine, damp soils, antacids, lime water, laxatives, bitters, phosphates, bone dust, phosphorus.

Definition. A constitutional disease of young animals, associated with disorders of digestion, nutrition, assimilation, and sanguification, and especially characterized by softening and distortion of the bones.

Lesions and Pathology. Apart from the bones there does not seem to be an absolute constancy in the lesions. There is usually, however, a period of ill-health and faulty nutrition before the lesions in the bones can be recognized. Thus, there may be gastric or intestinal congestion, or catarrh, indigestions, constipation alternating with diarrhœa, enlargement of the liver, spleen and kidney with hyperæmia, and according to V. Jaksch, a diminution of the salts of the blood. Beneke found that the arteries are dilated throughout the entire body, but the heart does not always participate in this distension. The arterial dilatation is very marked in the pulmonary artery, yet the lungs are relatively small. In the bones there is a well marked hyperæmia, most prominent beneath the periosteum, in the cancellated tissue, the line of junction of the epiphysis and diaphysis, and near the articulating surface. The contents of the cancelli are of a deep red, and the color shines through the articular cartilage giving it a bluish tinge. The shaft of the bone does not escape, but like the epiphysis and epiphyseal cartilage may be soft and yielding to pressure, and cut readily with the knife.

At both points the process of growth is increased and its area extended, but it is not completed by the full deposition of earthy salts, and the softening is not confined to the new tissues, but extends into the subjacent bone as well.

The chemical composition of the bone is profoundly altered, the organic basis, at times amounting to 65 per cent., as compared to 33.30 per cent in the normal bone. The softened bone, yielding under the weight of the body, bends out of shape at the epiphyseal cartilage, or even elsewhere, giving rise to bow legs, deviation of the joints inward, or other distortion. The periosteum is red, thickened, the seat of exudate and easily torn from the bone.

The bones are often thickened by new deposit under the periosteum and especially at the junction with the epiphyses. Old cases of distortion, the result of rickets, do not necessarily show a deficiency of earthy salts, as these are restored in case of repair and they may even be found in excess of the normal, increasing the hardness of the bone.

Causes. This disease does not seem to have been recognized in Great Britain until the beginning of the 17th century, the period of

England's early activity on the sea, and the beginning of extensive commerce and manufactures. From that time it has been increasingly and extensively prevalent. Yet it has not been shown to be propagated by any specific germ, nor to have extended in line with the introduction and use of new food products like the potato. It appears to be traceable rather to unwholesome conditions of life and a reduction of the general tone and nutritive vigor.

A deficiency of earthy salts in the food has been a natural and favorite explanation, and the ill-health that is thereby brought about is often an important factor. Yet rachitis occurs independently of such a condition.

In Roloff's experiments, pigs fed on aliment deficient in lime salts, suffered from bone softening, while the control animals on food rich in lime salts remained well. The diseased animals further recovered on a diet rich in lime. Voight, Chossat, Milne-Edwards, Lehman, Bousaingault, Heitzmann, etc., had similar experimental results, and the effects were shown in goats, sheep and dogs, in curvature, shortening, swelling of the costal cartilages and joints and contracted pelvis. Growing pigs have often been found to suffer in this way when placed on an exclusive diet of maize. The great improvement often secured in feeding an excess of calcareous phosphates tends to corroborate the hypothesis. Wagner found that food rich in lime salts, and the administration of small doses of phosphorus, rendered the epiphyses of the growing bones more compact. Kassowitz, on the other hand, found that an excess of phosphorus caused absorption from the bone substance and an irritable inflammation of the osseous tissue. Schneidemühl has seen the disease in calves raised on milk, poor in lime, the product of emaciated cows; in pigs getting only potatoes and swill, and in puppies that were denied bones. It is common in pigs on an exclusive diet of maize. Yet it is most destructive in many breeding studs where the alimentation is rich and generous. It must be admitted that as a concurrent cause, the paucity of lime salts and phosphates is a powerful factor, and that in supplying the bone ash, and improving the nutrition, these often prove of great value. Their privation is, however, not an essential condition of rachitis.

Free phosphorus. Ziegler and Kassowitz emphasize the hy-

peræmia of the cancellated tissue, and Wagner shows that this condition can be induced by excess of phosphorus, but this excess of phosphorus has not been found in the blood in ordinary cases, and is not likely to occur in a great number of young, at the same place and time, irrespective of food, as has been shown in breeding studs in New Jersey, in the South and West. In particular cases excess of phosphorus may operate, but it cannot be looked on as universal or essential.

The presence of *glycero-phosphoric acid* is alleged by Trasbot, but there is no proof of its constancy in rachitis, nor would its presence explain the real cause of the disease.

Lactic acid in the system. Lactic acid, in vitro, dissolves the calcareous salts of the bones. Trasbot alleges that it opposes the precipitation of lime in the form of tribasic phosphate, as found in bone. Siedamgrotzky and Hofmeister found that the salts of the bone were lessened under the administration of lactic acid. Heitzmann and Baginsky showed that by restricting the lime in food and giving lactic acid, by the mouth or subcutem, the lime salts in the bone were lessened relatively to the organic basis. It should be noted that an exclusive diet of buttermilk is liable to cause an attack of arthritic rheumatism. Lactic acid is undoubtedly a coöperative factor in certain cases, but though often found in the diseased bone and urine of rachitic children (Ragsky, Morehead, Simon, Lehmann), it is not shown to be constant.

Oxalic acid. Acetic acid. Formic acid. Beneke found oxalic acid in the urine in many cases of rachitis and attributed to it the removal of the lime salts. Others have made the same charges on acetic and formic acids which are sometimes found in the diseased bone.

It is quite plain that the process of normal ossification is easily disturbed, and that the same agent (lime, phosphorus) will assist or hinder according as it is present in small or large amount, and that certain chemical agents like organic acids may act injuriously even in the presence of an abundance of bone salts.

Heredity. Rickety parents have often rickety offspring, the weak somatic cells, failing in both cases to build healthy, strong tissues, but as a rule also, both have been condemned to live in similar unwholesome surroundings.

Unhygienic Conditions. Schneidemühl notes that in animals

as in man, bad ventilation, close impure air, crowding, damp impervious soils, and cold, are found more or less in places where rachitis prevails. By lowering the general health and tone, these debilitate the tissue cells and impair nutrition and growth.

Confinement has a manifest influence. Rickets prevails in children in the great manufacturing cities, where the exclusion of sunlight and the breathing of impure air rob the system of its vigor. The children of soldiers in India kept in close barracks are largely rickety, while the more poorly fed native children outside escape. Wild beasts in confinement are often rachitic, unlike their fellows of the forest. Colts in confined stables suffer while those in the fields and yards remain healthy. Swine in Sweden in close pens and fed on potatoes alone suffer (Stockfleth).

Darkness usually coincides with confinement and it is noteworthy that deep sea fishes, living in comparative darkness, have usually cartilaginous skeletons.

Infection. Certain facts seem to point to a direct infection, as coöperating with the debilitating conditions above named. The advent of the disease in England about 1700: its frequency in English swine on the European continent (Schneidemühl); and its enzootic prevalence in different parts of America, give seeming support to the doctrine.

Dr. W. L. Williams has seen it appear on an Illinois farm twenty years after reclamation from virgin prairie, prevail for ten years and then disappear. There was a remarkable coincidence of recurrent ophthalmia, and disease of the bones and joints (navicular disease, spavin, splints, ring bones, etc.).

In most of these cases the trouble occurred on low, damp or impervious soils, agreeing with rickets in children, which avoids the Alps or hilly districts, and abounds in valleys or bottom lands.

Symptoms. The colts show a lack of thrift and though there may be no lack of growth or size, they have a rough coat, a poor development of the neck, arching of the back and drooping pelvis. The chest may seem to sink between the scapulæ. They move stiffly with swaying of the limbs, or even staggering and are easily wearied or lacking in endurance. They lie a great portion of their time or even persistently, refusing to rise. When

up they do not hold the limbs plumb, but allow them to deviate one way or another in an unsightly way. There is liable to be swelling of important joints of the limb, (knee, hock, stiffle, fetlock), which are tender to pressure and kept partly flexed. The ends of the ribs are often enlarged. Bending of the long bones (tibia or radius), and deviations of the back or sternum from the straight line are significant. Thickening of the ends of the bones, or in the region of the epiphyseal cartilages are largely diagnostic. The bones are easily fractured. In *swine* fed on potatoes, corn, etc., besides the affections of the limbs, the thickening of the bones and swelling of the joints, especially the hock and pastern, there is enlargement of the nasal and maxillary bones so as to seriously obstruct breathing ("Snuffles"). The teeth suffer and break readily and in the general break down diarrhœa, bronchitis or skin eruption appears and the subject falls into marasmus and perishes. In the necropsy arthritis is commonly found. In *cattle* beside the epiphyseal swellings, the bow logs and joint enlargements, the back becomes crooked, vertically or laterally. The same general symptoms appear in *dogs* in which bow legs are a very prominent feature. Goats suffer badly and mostly remain recumbent.

Birds suffer most, showing knotty thickening of the bones of the legs and wings, and flexibility of the bones generally but above all of the keel of the sternum, which is usually badly distorted from sitting on the perch.

In all alike there are usually a few days of fever, followed by indigestion, colics, anorexia, and a general air of illness. Then appear the lameness, stiffness and swelling of bones and joints. Any joint may suffer, shoulder, elbow, knee, hips, stifle, hock, or fetlock. The lameness may shift as in rheumatism, it may intermit, occurring periodically, or it may advance uninterruptedly to a fatal issue. Paraplegia is common and appears to be due at times to pressure on the spinal nerves by the diseased vertebræ. Before this becomes complete, the animal may walk with the whole digits and metatarsi in contact with the ground, and the softened crumbling calcis may project through the skin forming an unsightly sore which soon becomes septic. The same happens at times to the point of the elbow.

Treatment. The most important, are the hygienic considerations. Reject weak or cachectic animals from breeding, and those

that have been rachitic to a marked degree, as their progeny are likely to show the same weakness. Change the ration giving one that is well balanced and rich in nutritive matters and earthy salts. Clover, alfalfa, and a generous grain diet may serve as an example for herbivora, and a fair allowance of meat and bones for dogs. Food from land that has apparently contributed to the disease in other cases is best avoided. If the land is poor, sandy, or destitute of earthy salts and phosphates, so much the more is it to be suspected and set aside. In the case of sucking animals it should be seen that the milk is rich and abundant, and that it is not too rich in fat, nor otherwise calculated to disagree and induce indigestion. Above all soured or otherwise fermented milk should be withheld, and any buckets or troughs used in feeding should be regularly washed, scalded and disinfected. In case colts, or dogs are being raised on cow's milk it may be requisite to dilute it with one third its volume of barley water, or solution of gum arabic, and to sweeten with sugar. Lime water with each meal is valuable in counteracting acidity, and fermentation, and in furnishing lime which may be absorbed in part.

In prevention and treatment alike, fresh air and sunshine must never be neglected and in warm weather, an outdoor life, night and day is of the greatest value. At the same time cold storms, damp beds, or any condition which may induce chill must be excluded. Close stalls, pens, or kennels must be absolutely forbidden.

Among medicinal agents antacids are often essential on account of the acid condition of the ingesta, lime water will often suffice, but if there is manifest constipation calcined magnesia three times a day on an empty stomach so as to counteract costiveness will often serve a good end. The atony of the bowel may be further met by small doses of strychnia. Other bitters may be used if this has little effect. Small doses of phosphate of soda, or bone dust have been long lauded in the affection, and probably act beneficially as a tonic as well as a food material. Phosphorus in minute doses tends to increase the deposit of earthy salts and consolidate the bones. Large doses induce hyperæmia of the epiphyseal ligament and even favor fracture. A grain of phosphorus daily may be given in olive oil or better in cod liver oil which acts as a valuable tonic. Dieckerhoff recommends the intratracheal injection of the solution of phosphorus in olive oil.

BRAN DISEASE: SHORTS DISEASE: BRAN RACHITIS.

Miller's horses. Bran and middlings as fodder. Torpid bowels, impaction, indigestion, colic, early fatigue and perspiration, stiffness, lameness, epiphyseal swelling, facial bones swell and soften symmetrically, teeth drop, dyspnœa. Ash of bran. Treatment.

A curious form of rickets has been observed, especially in miller's horses as a result of an excessive consumption of bran or middlings. It is characterized by torpor of the bowels, impaction, indigestions, slight colics, early fatigue and profuse perspiration under slight exertion followed by stiffness, lameness, enlargement of the bones in the region of the epiphyseal cartilage (near knee or hock), or of the bones of the face. The superior and inferior maxillary bones are symmetrically enlarged, the teeth are shed, mastication becomes difficult and there may be some dyspnœa and snuffling. This resembles the "snuffles" in pigs on an exclusive diet of Indian corn or potato and Friedberger and Fröhner seek to explain both, by the lack of lime and phosphorus in the food. But wheat bran has 5.1 per cent. of ash, and middlings 2.3 per cent. as compared with wheat flour 1.7 per cent. or oats 2.7 per cent. Putz on the contrary attributes the disease to the excess of phosphorus in the bran acting as the free phosphorus in lucifer match factories in causing necrosis of the jaw. But the phosphorus in bran occurs as phosphate of lime which has no such action on the bone and one must infer that the phosphoric acid is set free by some acid developed perhaps in the intestinal fermentations. This is, however, as yet unproved.

The *treatment* of this affection consists in the suspension of the bran and the expulsion of offensive accummulations and products from the bowels, followed by a course of tonics and the general treatment for rickets.

OSTEOMALACIA (MALAXOS SOFT) : CACHEXIA OSSIFRAGA ; FRAGILITAS OSSIUM : "THE CRIPPLE :" "THE STIFFNESS."

Definition. Disease of the mature. Decalcifying in cancelli and Canals of Havers. Dairy cows. Heavy milkers. Perverted appetite. Limed soils, sandy or limestone. Low, damp, soils rich in organic matter. Cultivation. Watery food. Plethoric. Debilitated. Cold. Change of locality improves. Microbes. Toxins. Lesions : vary with stage, congestion of marrow, excess of cells and fat, osteoclasts, exudates, friability of bone, distortions and fractures in pelvis and elsewhere. Symptoms : low condition, projecting bones, rough coat, perverted appetite, stiffness, decubitus, swaying limbs, inappetence, drying of milk, fever, bed sores, sloughs, sepsis, pus infection, fractures. Duration, 2 to 3 months and upward. Enzootic. Prognosis, varies with enzootic, and stage ; best in recent cases, occurring, sporadically. Treatment : according to cause, rich, generous diet, grain, salt bitters, cod-liver-oil, apomorphia, wholesome pasturage, intensive culture change water, dry up milk, dry stables, pure air, sunshine. Slaughter. Local derivatives.

Definition. A softening and fragility of the bones of adult animals, in connection with solution and removal of the earthy salts.

This is essentially a disease of mature animals and is thus easily distinguished from rachitis, in which the lesions are due to a faulty development of young, growing bone. In osteomalacia, too, the decalcifying proceeds most actively in the walls of the Haversian canals and cancelli, while in rachitis it progresses especially under the periosteum and in and around the epiphyseal cartilage.

The disease is found most commonly in dairy cows, but softening of the bones of mature animals has also been seen in horses and other animals. Dieckerhoff, who quotes cases in mature horses, adduces similar instances in colts under a year old, occurring enzootically, and without the specific lesions of rachitis. Seven out of sixteen broke their femurs in October, 1886, all kept on the same place, in good box stalls, and well cared for. Landois found in bones an abnormal amount of fat, ossein, water and lime salts. Grawitz found no material change in the cancellated tissue.

In one district in Jutland, Stockfleth found an extraordinary number of broken legs as the result of castration of colts, which had not shown the thickening or distortions of rachitis.

Causes. The disease is particularly common in cows which yield a calf every year, and especially in heavy milkers, in which respect it agrees with the osteo-malacia of woman. The heavy demands upon the system for the nourishment of the fœtus and the supply of milk, undoubtedly lay the system open to attack, if they do not directly cause the disease. An early and usually a persistent feature of the malady is a *depraved appetite*, the causes of which may be read up in Vol. II. The statements there made, require some qualification, inasmuch as osteo-malacia is at times found on limestone soils with hard, calcareous water, and on rich, alluvial valley soils abounding in both clay and lime (Sarginson, Leclainche), as well as on barren sands and granite soils deficient in both lime and phosphorus. It may even appear on virgin or mucky soils after liming, which had been free from the trouble up to that time (Thorburn). The decomposition of the abundance of organic matter, hastened by the quick-lime, has evidently been a contributing cause.

The excess of organic matter in the soil seems to be a considerable factor. Both Thorburn and Sarginson mention the "mossy" soils and waters, and in Lanarkshire, Scotland, and Westmoreland, England, where they practised, black muck and peats abound. This is corroborated by the prevalence of the disorder in the damp lowlands of Belgium and Jutland, in the Swiss valleys, on the damp lands of New Jersey and the Carolina seaboard, and generally on damp pastures with rank, watery herbage.

When land has been better cultivated and enriched by manure, the disease has in many cases disappeared. This has been observed in England (Sarginson), Wurtenberg, Switzerland, etc. (Leclainche).

Succulent, watery food (potatoes, turnips), have been quoted as causes, as also rank, watery grasses, deficient in nutritious solids, but such food has invariably come from habitual osteo-malacia soils. On the rich, cultivated soils of the Lothians, Scotland, cattle are fed in large numbers on turnips alone, and osteo-malacia and pica are alike unknown. It is often noticed that the fodder grown on particular (osteo-malacia) soils will cause the

disease when fed elsewhere, so that the inference is that some agent derived from these soils, and which is destroyed or rendered harmless by cultivation, is carried in the food. It cannot be a mere defect of nutritive matter, as this could be counter-balanced by the simple expedient of consuming a larger ration. Leclainche has seen the disease in its worst form in herds which received a rich and varied ration, while it spared adjacent herds that were kept on rather short rations. Even young plethoric animals suffered badly, though having all they would eat of natural fodders (hay) from districts where the disease was unknown, and in addition grain, linseed cake, cooked legumes and bread. In two neighboring stables, where the stock were kept in identical conditions, receiving the same food, in equal quantity, one was decimated by the malady, while the other was spared (Leclainche).

The affection often prevails on the higher lands, which, beside having the poorer soils, are specially exposed to cold storms and frosts, so that chill enters as an accessory condition. In Westmoreland, England, the river Eden divides the affected from the sound lands; the victims are found on the west bank which receives the cold, east winds, and not at all on the east bank where the warm, soft, west winds prevail. Thorburn noticed that the majority of cases start in spring, when the animals, debilitated by the winter's seclusion, are exposed to severe vicissitudes of temperature and driving storms, to the strain of parturition, a fresh, heavy milk yield, and moulting.

The presence of a contagion has been suggested, but if this exists it must be habitually introduced in the food or water rather than transmitted from victim to victim. The healthy will often stand beside the diseased for an indefinite length of time without injury, and in certain recent cases a change to an uncontaminated farm, or an abundant ration drawn from such sound soil, will secure immediate improvement and recovery in a few weeks. In view of such prompt recoveries it would be quite as reasonable to suspect some ptomaine or toxin taken in with the food. The question of a microbe or a microbian poison is as yet a mere hypothesis.

Cows become more susceptible with advancing age, and Deugler alone claims to have seen the disease in calves. This is

unfavorable to the idea of immunity, and rather favors that of debility or cachexia.

Lesions. These are confined to the bones. Decalcification in the Haversian canals and cancelli, reduces the bony tissue to a thin soft plate. Yet the condition is not constant. Grawitz found no special dilatation of the canals or cancelli in colts. Nessler and others found decrease of the lime salts, Bibra and Grouven detected no marked change, and Hoffmann and Begemann found an actual excess of phosphates. Doubtless the specimens selected and the stage of the disease, whether in active progress or during convalescence, may somewhat explain discrepancies. The fat cells increase in the cancelli, with more or less hyperæmia, and even blood staining as the disease advances, the bone cells become less branching, and there may be gelatinoid exudates. The resistance of the bone is diminished, it may be indented with the finger, or scraped off with the nail, or cut with a knife. It breaks under a slight strain, and is easily crushed under the weight of the animal so that fractures and distortions of all kinds are met with. In breeding cows the earliest and most marked lesions are in the pelvic bones, but fractures of the bones of the limbs are common.

Symptoms. Poor condition or even emaciation, with very visible projection of the bones is common. The coat is rough, the skin tense, inelastic and hide bound, appetite variable, sometimes impaired, and nearly always perverted so that the patient will lick the manger continually or pick up and chew all sorts of objects, bones, leather, articles of clothing, pieces of wood or iron, stones, etc. The amount of food consumed may, however, be up to the normal. The most marked feature is the difficulty and stiffness of locomotion. The patient lies most of the time, rises languidly and with difficulty and moves the limbs as if each were a rigid post without joints. The hocks will knock together, and the restricted movements of the joint are often attended by cracking. Yet appetite, temperature and yield of milk may remain normal.

Later appetite and milk secretion fail, temperature rises a degree or two, the animal refuses to rise, remaining down twelve to twenty-four hours at a time, and rising first on the hind parts, and remaining on the knees for a length of time, moaning and

indisposed to exert herself further. Many cases at this stage begin to improve and may get well in five or six weeks. Some will remain down for several weeks and finally get up and recover. With constant decubitus however, the animal falls off greatly, becoming emaciated and weak, the appetite may fail altogether, and the patient is worn out by the persistent fever, nervous exhaustion and poisoning from the numerous bad sores. Abscesses, sloughs and fistulæ are common over the bony prominences.

It is in these last conditions above all that fractures and distortions of the pelvic bones and less frequently of the bones of the legs occur. They occur earlier as well in connection with falls, blows, crowding by their fellows and sudden active movements of various kinds.

The disease may advance for two or three months, and in case of pelvic fractures and distortions, there may be permanent lameness, and dangerous obstruction to parturition, even though the bones should acquire their normal hardness through the deposition of lime salts.

It has been noted that the disease is usually confined to well defined areas, and that even in these it has its periods of abatement and recrudescence so that given years are osteo-malacia years. In the department of l'Aube, France, Leclainche, particularly noted the enzootics of 1865-6, 1870, 1875-6, and 1883-4.

Prognosis. The gravity of the affection varies greatly under different conditions. Some outbreaks are mild while others are very severe, and the prognosis must vary with this gravity. Again at the decline of an enzootic the disease is more benign and less ruinous. A case in its very earliest stage is much more hopeful, than one that is far advanced, with bones extensively softened or even broken, digestion and assimilation badly impaired, and infecting sores and sloughs on different parts of the body. Isolated cases are usually much milder than when the affection has gathered strength enough to determine an enzootic.

Treatment. This will vary with the predominance of the causes, essential or accessory. In some cases the suspension of the injurious food and a rich alimentation on well grown fodders from sound lands will meet every need. Green clover, alfalfa,

and other leguminous products, ground oats, beans, peas, linseed cake, rape cake, cotton seed and vetches may be especially named. Even animal food may be availed of, and cases are recorded in which cows have themselves hunted for snails and frogs and devoured them greedily. The free access to common salt, and a liberal supply of bone meal are helpful. Iron and bitter tonics, (gentian, quinine, salicine, nux, copperas, tincture of iron) and cod liver oil, in pint doses daily, have been found advantageous. Apomorphia has been found especially valuable in correcting the preverted appetite, and stimulating digestion.

Where it is feasible to move the affected herd from the unwholesome pasturage or locality to one in which the aliment is rich and the disease unknown, success usually follows the change.

On poor, uncultivated lands where the disease appears yearly, or at short intervals, intensive culture with heavy manuring, and the heavy feeding of the herd on grain products, linseed cake, etc., will often banish the trouble.

Care should be taken to change the water as well as the food.

Finally every drain upon the system should be lessened or stopped. The milk may be dried up and the animal should not be bred. Sweet, dry buildings, pure air, sunshine and grooming are important auxilliaries.

In severe outbreaks, in high conditioned animals, the owner often consults his interest, in sending the victims to the butcher as soon as the affection shows itself and before time has been allowed for the inevitable emaciation and loss.

Some, on osteo-malacia lands, have profited by changing the entire herd every two years, as they become rapidly worn out under successive attacks.

As local derivatives, oil of turpentine, hot vinegar, tincture of iodine and biniodide of mercury have been employed. Open sores are treated with antiseptic lotions, creolin, lysol, carbolic acid, iodine lotions, iodoform, etc.

RAREFYING OSTEITIS. OSTEOPOROSIS. OSTEOMALACIA OF THE HORSE. BIG HEAD.

Definition. Distinction from rachitis. Process of rarefaction, cell proliferation, congestion, solution of earthy salts and fibrous matrix, osteoclasts, Howship's lacunæ. Dried bone light, spongy, friable, though enlarged in repaired cases, dense, heavy. Face lesions. Dyspnœa. Dropping teeth. Causes: microbian hypothesis, disturbance of bone nutrition, faulty food, lack of bone salts, cellar stables, floor on ground, malaria, cold, damp soils, city life, early life, breed, asses and mules, breeding. Nature. Symptoms: illness, inappetence, lifelessness, early fatigue and perspiration, stiffness, lameness, distortions, stumbling, knuckling, arthritis, tender puffed joints, thickened softened bones, facial swellings, narrowing of intermaxillary space, chisel teeth, difficult mastication, shedding teeth, emaciation, marasmus. Phosphates in urine when disease is active. Relation to exostosis. Prevention and treatment. Hygienic, move from cellar stable, or ground floor, secure air space under floor, ventilation, warmth, sunshine, grain feeding, pasture, change food, rest, salicylates, salicin, salol, phenacetin, blisters, phosphates, bone dust, phosphorus, bitters, iron, barium chloride.

Definition. A form of osteomalacia occurring in the mature as well as in the growing horse, characterized by the absorption of earthy salts from the walls of the cancelli and Haversian canals with excessive production of the organic basis and cell elements and enlargement, softening, lightness and fragility of the bones.

It differs from rachitis essentially in this that while the latter attacks the young growing bone at the chief seats of growth, under the periosteum and in the epiphyseal ligament, and therefore especially on the surface of the true bony tissue, osteoporosis attacks the formed and often the mature bone in its interior, producing attenuation of the walls of its vascular canals and cancellar cavities and increase of their fibro-cellular contents. Both result in enlargement of the bone, but in rachitis this is determined largely by deposition on the surface while in osteoporosis it takes place by expansion from within. The further distinction that rachitis appears enzootically and osteoporosis sporadically applies only to localities in which the latter is

not common. At different points on Long Island, in New Jersey and on the rich soils in the Mississippi valley and on the Atlantic and Gulf coasts, osteoporosis often prevails enzootically and has its recrudescences like rachitis.

The process of rarefaction and softening may be thus stated. In the Haversian canals and narrow spaces there is an active proliferation of cells and increase of vascularity, with a gradual solution and removal, not only of the earthy salts, but also of the fibrous matrix in the walls of these spaces. As in the normal changes in bone, the absorption appears to be effected through the large cells or osteoclasts grouped around the blood vessels. In the osseous tissue, which abuts on the vascular tissue, are to be found excavations, simple or irregular, (Howship's lacunæ), containing granular cells and the larger osteoclasts. In a similar manner bone is softened and absorbed in connection with tubercle, or the pressure of tumors, aneurisms, actinomycosis. In case of recovery, repair takes place by the disposition of new bone, so that the enlarged and rarefied bone may in the end become harder than before. This applies especially to the cancellated bone. On the articular ends of the affected bones, the granulation tissue makes its way into and through the cartilage of incrustation with resulting active disease of the joint.

The condition of the bone is well illustrated in the dried or macerated specimen. Taken from a bad case in the active stage of the disease, it crumbles under pressure and weighs as light as a sponge, whilst from an advanced or recovered case, it is hard and resistant, and weighs as much more than the normal bone as it exceeds it in bulk. The morbid rarefaction usually affects the whole skeleton more or less, yet in perhaps the majority of cases, the change is greatest in the bones of the face, and the resulting distortions are usually symmetrical on the two sides. It may show mainly in the maxilla, which is thickened to twice or even five times its normal thickness, it may show in rounded, general swelling of the nasal and superior maxilliary bones and those around the orbit, or it may involve the turbinated bones the vomer and even the cartilaginous nasal septum. Loosening and evulsion of the molar teeth is common and snuffling breathing may occur as in rachitis affecting the nasal bones of dogs and swine.

Causes. We are still in the dark as to the essential cause of rarefaction of bone. There is a growing tendency to suspect a microbian origin, and many facts are held to point in that direction. It seems to have been unknown in England in the early part of the 19th century, and is not noticed by Blaine, Youatt, Percivall nor other of the early writers. In Varnell's cases the same man had two farms not far apart and equal in soil, drainage and stabling, stocked with horses bred from the same parents with the same kind and amount of feed and work, yet on one farm six cases of osteoporosis occurred, and on the other not a single case. McNeil, in charge of street car stables, found the disease destructively prevalent in the best appointed stables and absent from others in the worst hygienic condition. In a superior stable with 220 horses he had 47 cases in two years, and in a fine stable with 100 horses he had 26 cases in the same length of time. In the poorer stables, the horses bred in the same way on all kinds of soils and with no difference in feeding nor management escaped. It is the common experience in Europe and America that a farm or district, which has been previously free from the disease suddenly has an outbreak in enzootic form, and this will last for a year or two, then remit only to appear with its old force after an interval of some years. Even during the active prevalence of the disease on one or on several adjacent farms, others in the immediate vicinity, and differing in no appreciable way, geologically, hydrostatically, in buildings, food, water, general management nor work, completely escape. Berus, Hoskins and other city veterinarians have noticed, that it was almost the rule, that a fresh horse put in the stall of one that had suffered from osteoporosis soon contracted the disease.

W. L. Williams noticed on two different farms in Central Illinois, on which the disease suddenly appeared, that for years after the comparative subsidence of the affection there was an unusual prevalence of spavins, splints, ring-bones and other diseases of the bones. Meyer has noticed that cases sent from Cincinnati into the country, and that have got well, will succumb to the affection if brought back into the city stables in which they originally contracted it.

All of this points very strongly to one of two things; either a pathogenic germ in the system of the affected animal; or

the presence of a pathogenic microbe in the stable, water, or other part of the environment, the toxic products of which are taken into the animal system.

But as yet no specific pathogenic microbe has been demonstrated so that this doctrine must still be held as a mere plausible hypothesis.

Many veterinarians with long experience in such cases absolutely deny contagion. The hypothetical contagion undoubtedly extends slowly, and uncertainly from animal to animal, probably, like actinomycosis, taking place mainly through the soil, or some outside medium, rather than by direct contact; or a special susceptibility on the part of the individual animal may be necessary to render it effectual.

Accessory Causes can be spoken of more confidently but even of these no one, nor small group, can be advanced as essential. The process of bone-nutrition is readily disturbed by a variety of conditions, and such disturbances may easily become the occasion of weakening the resisting power and mayhap of admitting the hypothetical microbe to get in its pathogenic work.

Faulty food has been a favorite explanation. A lack of lime in the soil and fodder seems, at times, to have had a baneful effect, if only, in lowering the general tone and impairing the nutrition. Yet we see osteoporosis on limestone soils (New York, etc.), and in animals generously fed on grain. The same remarks apply to phosphorus and phosphates. Their deficiency apparently contributes to the production of the disease, and yet under other conditions, their abundance is no barrier to its development. The excess of free phosphorus produces osteitis and it is held by some that an over-abundance of phosphates acts in the same way, It has been sought to incriminate a too nitrogenous diet in some cases, and in others one too rich in fat or carbhydrates. The many cases in Philadelphia and Pennsylvania were mostly in animals that had been well fed and were in good condition when attacked (Marshall).

Special food may be the direct cause, bran diet has been already noted. Hinebauch found an acute osteitis with bone softening and arthritis in horses fed on millet, green, partially matured and ripe. Horses elsewhere have fed on millet, without such results, but not perhaps, in the same environment, nor in

presence of the hypothetical microbe. Millet is not the sole nor common cause of osteoporosis, but there is reason to suspect that it is at times an important accessory cause.

Of all prejudicial conditions none is to be so dreaded as *unwholesome stables*. Of 200 cases reported by Berns, in Brooklyn, almost all were in cellar stables or those with floors laid on the soil. Meyer finds that "most all cases can be traced to an unwholesome atmosphere, or gases arising from vaults, sewers, cellars, filthy streams, or from a hollow space under the floor." Harbaugh says every case was stabled in damp, ill-drained, unventilated and badly lighted buildings. The worst outbreak was in a basement with a damp wall, on one side, and none suffered except those that stood next to this wall. The horses standing on the opposite side, which was on a level with the ground outside, escaped. Removal from a cellar stable to the floor above, put a sudden stop to the appearance of new cases. James, of St. Louis, found 20 successive cases in a stable on a dirt floor, and Jasme, of Charlestown, finds nearly all his many cases on earth floors in malarial regions.

Malaria has been blamed, especially by southern observers, accustomed to see the disease on the warm alluvial seaboard and river bottoms That this environment predisposes to the disease, by undermining the health, is doubtless the case, but in spite of occasional remissions in the symptoms, malarial germs cannot be set down as the constant cause. One of the worst cases I ever saw, with every bone in the body soft, spongy and light, developed at Inglis Green laundry, Edinburgh, where malaria is absolutely unknown, but where the brook received large quantities of chlorine.

Cold is an undoubted factor, though the disease is most prevalent in our warm southern states. Many veterinarians have noticed its coincidence with rheumatism, in which cold is so often the dominating accessory cause. Some have even suspected that it is only a modified type of the rheumatic condition. Hinebauch found his cases of *millet disease* in cold basement barns, or with leaking roofs, so that the floor and bedding were constantly wet. He found that cold always aggravated the disease, and bad air even more so, while salicylates seemed to have a marked curative influence.

Damp soils should be named in this connection. These not only chill the air by evaporation, and condense the cold dews at night, contributing to produce the extremes of hot noon day and cold night temperature, with corresponding disturbances of the bodily health, but they favor the preservation of the infinitesimal forms of life (bacteria, protozoa) and therefore of the hypothetical microbe of the disease.

City life is a most potent cause. Berns tells us that hundreds of horses die yearly in Brooklyn of osteoporosis, and that if sent early to the country a large proportion recover. The same is true of New York City and Philadelphia. Of Cincinnati, Meyer says that he has failed to find a case more than five miles outside the city limits, and that cases sent to the country make a partial or complete recovery. If returned to their former city stables, nearly all contract the disease anew within a year.

Many cases, however, are found in the country and often within a circumscribed area. These indicate, as in the city cases, a localized cause, bacteridian or otherwise.

Early life predisposes, the majority of cases taking place before the sixth or seventh year, yet the disease occurs at all ages up to twenty years and over.

Breed does not seem to make a material difference, and though Shetland ponies have acquired a bad reputation, their propensity to become fat and soft, their too often idle, pampered life, and the confined quarters in which they are frequently kept, account for much of the mortality.

Asses and mules habitually escape, even in the South, where the latter animal is so numerous and often so poorly kept. In Hinbanch's millet disease, the mules suffered more than the horses.

Breeding horses, male and female, have often acute attacks and die early. The drain on the system and confinement seem to act injuriously.

Nature. Until we know the essential cause or causes of osteoporosis, we must be in doubt as to its pathology. We are even debarred from pronouncing authoritatively upon the essential identity or difference of the various forms of softening, or rarefaction of bone. In obedience to the clinical manifestations and structural changes, rachitis, fragilitas ossium and osteoporosis have been separately described, but we cannot positively say that they

are not all due to one essential cause, manifesting itself differently according to the activity of trophic processes in the bones of the victim. In the growing foal the active developmental processes in the epiphyseal ligament and periosteum may determine that the symptoms shall be pre-eminently those of rickets, yet we often see these complicated by the facial and other lesions of osteoporosis. Both may be the result of one etiological factor, or there may be a complex disease resulting from the presence of two. Again in the pregnant cow in which the relaxation of the ischio-pubic symphysis and pelvic ligaments means a profound change in the bone nutrition at this point, the presence of the hypothetic microbe, or other essential factor, may determine a decalcifying and fragility of the pelvic bones generally. Again in the mature male and non-breeding female, in the absence of the disturbing conditions of nutrition just named, the vascular elements in the Haversian canals and cancelli may determine the simple rarefaction and expansion of the bone which characterizes osteoporosis. When present in the bone in any of these conditions, acids doubtless fulfill an important rôle in the decalcifying and softening process, but behind these it may be surmised that there is an unknown cause or causes, which it is for the bacteriologist, chemist or botanist to discover.

Symptoms. These are largely the same as in brain disease. There may be first a period of illness, with poor appetite, lack of spirit and energy, early perspiration and fatigue, or if at large, leaving the herd, soon followed by some stiffness of gait and lameness, which may be intermittent, disappearing under exertion, or shifting from one joint or limb to another after the manner of rheumatism. Sometimes it shows in stiffness of the neck, so that the patient finds difficulty in lowering the head to graze; in others the back and loins are stiff and arched so that the animal has difficulty in rising and turns slowly and painfully; in still other cases the dorsal and lumbar vertebræ are depressed so that the back is hollow. Even before the manifestation of lameness, the affected limb may stand forward at the fetlock or knee, the gait is clumsy and awkward, and the patient may suddenly stumble and fall, showing little or no power of prompt recovery of balance. A horse, and especially a young horse, with this habit of stumbling is always to be suspected. The long bones of the

limbs tend to enlarge or thicken, and this is likely to be more uniform than in rachitis, and not to be confined so much to the epiphysis. The implication of the stifle, hock or other joint, with marked synovial distention, and mobility or dislocation of the patella, is common and may be the earliest manifestation of illness. The bones of the face usually show early changes. The superior maxillary and nasal bones, beneath the zygomatic spine and infra orbital foramen and along the line of the molar alveoli become especially bulging and rounded, the other facial and cranial bones suffering to a lesser degree. In the lower jaw, also, the disease predominates along the region of the molar alveoli, and the loosening of the molars permits them to deviate inward so that the grinding surfaces come perceptibly nearer to the median line, and the outer half of the tooth is rapidly worn while the inner edge projects as a sharp cutting ridge (chisel teeth).

For the same reason, the softening branches of the lower jaw deviate inward, tending to still further destroy the due approximation of the upper with the lower molars, and to diminish the breadth of the intermaxillary space. The great thickening of the rami of the maxilla tends still further to reduce the intermaxillary furrow.

Fractures and detachment of tendons and ligaments are common results of the rarefaction, a portion of the bone often remaining adherent to the tendon.

Cary gives the following statistics of fifteen cases: lame in the fore limbs 11 (mostly shoulder), in hind limbs 11 (mostly hip and stifle), stiff in loins 8, unable to rise without aid 3, had indented ribs 4, had shifting lameness 8, had chronic indigestion 6; mares 5; geldings 8; mules 2; ages were—*one* 3 years, *five* 6 years, *three* 7 years, *one* 8 years, *two* 9 years and *three* 10 years.

When the bones are enlarged their softness and friability may be shown by pricking with a needle, which will often freely penetrate the rarefied bony tissue. It should be first rendered antiseptic by dipping in strong carbolic acid.

From the first the general health fails, there is difficulty in mastication, digestion is more or less impaired, nutrition is imperfect and muscular flabbiness, weakness and wasting advance more or less rapidly. In advanced cases emaciation is often a marked feature.

Examination of the urine gives valuable indications, though the results obtained have been supposed to be contradictory. While rarefaction of the bone is advancing rapidly the urine is charged with an excess of phosphates in ratio with the activity of the morbid process. When on the other hand the disease has come to a standstill and the process of repair in the rarefied bones has begun, the absence of phosphates is no less characteristic. A patient therefore may show greatly enlarged and softened bones with persistent lameness, and yet the urine may show little or no phosphate. The phosphaturia may, therefore, be made the basis of a reasonable prognosis. Excess of phosphates indicates an active pathological process, with an uncertain outcome, while the absence of phosphates indicates an arrest of rarefaction and holds out good hope of recovery.

I have long observed the same in cases of obstinate and intractable spavins, splints, ringbones and other bone diseases. Phosphaturia bespeaks a faulty nutrition of the bone and explains the failure of remedial measures, while lack of phosphates in the urine, or a reduction to the normal amount is likely to become a guarantee of improvement under local treatment. The treatment however, must be first constitutional to correct the condition of malnutrition and then local to correct the osteitis.

Prevention and treatment. As in rachitis hygienic measures give the most uniformly good results. The change of stable is especially demanded from a cellar or basement stable, one with joists laid on the ground or one with an earth floor saturated with urinary and feculent products. In different cases an enzootic has been arrested, coincidently with the removal of the stock to the floor above, and in others with the removal of the filth saturated earth beneath a ground floor, and the laying of a new floor with ample space beneath for the free circulation of air. In the same line would be thorough drainage of the site and to carry off liquid manure to a well ventilated receptacle. Exposure to cold and wet is to be sedulously avoided as greatly favoring the onset of the disease, and hurrying the milder cases into a fatal activity. Free air and sunshine are all important and it is the universal experience that city cases taken early and sent to dry, sunny pastures, mostly recover, or at least undergo marked amelioration. The fact that certain cases originate dur-

ing an open air life does not invalidate this position but merely shows that other pathogenic conditions may be too potent to be overcome by this hygienic one.

A liberal allowance of sound grain is essential to success, even in the case of patients sent to pasture. Those that have recovered or improved at pasture, should be retained in the country and on no account returned to the same city stables in which they contracted the disease. Even in the country a different stable should be secured if possible.

Any food that has manifestly contributed to the disease, should be withheld (bran, millet, musty or fermented food or that drawn from particular fields).

Overwork must be forbidden, and indeed any work at all during the active stage of the disease. The victim should also be withdrawn from breeding, at least until it has fully recovered the normal consistency of its bone.

In cases aggravated by cold or wet, or which show the rheumatic propensity to shift from place to place, sodium salicylate in ounce doses several times a day may appear to benefit, and as a germicide this may be tried on all cases. Salicin, salol, or phenacetin may be used as substitutes. Where the disease has been largely localized, blisters have appeared to be beneficial.

Phosphates and phosphorus have been landed by German veterinarians, but in other hands, and when the morbid process was active they have proved useless, or even hurtful. Bone dust or phosphate of lime or soda may be freely used at any time and appears to act as a general tonic, beside supplying lime and phosphoric acid which may possibly be availed of for bone nutrition. Phosphorus and phosphorated oil in excess always softens the bone and much more so when this process is already excessive. In small doses (gr. $\frac{1}{6}$ to $\frac{1}{2}$) and after the process of rarefaction has ceased, it is valuable in hastening bone consolidation and fitting the patient to return to work.

Bitters, iron and other tonics are valuable in improving the general tone and indirectly the bone nutrition.

Cary had prompt improvement in connection with intravenous injection of barium chloride once a week for four weeks, and ½ oz. doses of sodium salicylate thrice a day. It remains to be seen whether or not this is generally applicable.

OSTEO-MALACIA IN OTHER ANIMALS.

The internal softening and rarefaction of bones in mature animals has been noted in dogs by Pillvax and Röll, in lambs by Haubner, in pigs by Haubner and Anaker, and in goats by different observers. The genuineness of these cases has been questioned by Cadiot and Leclainche, by Virchow and by others, but in the present uncertainty as to the dividing lines between rarefaction, rachitis and other diseases, they deserve notice in this connection.

In *dogs* the lesions are mostly in the young and are largely rachitic, yet the enormous swelling of the facial bones, and especially of the superior maxillary in the comparatively mature animal suggests osteo-malacia. As in rachitis there are usually impaired digestion, unthriftiness, slow, stiff movements and lack of life and vigor.

In goats Virchow believes the disease to be neither rachitic nor osteo-malacia, basing his opinion on the lesions in the bones: "On the maxillary bones of goats there are often found peculiar formations in which the parts that have already assumed osseous structure, have failed to fix the earthy salts. The tumor, which forms a circumscribed swelling on the upper or lower maxillary bone, is soft and easily cut with the scalpel, with at certain points only, a hard resistant material. It is a simple osteoid chondroma, though veterinarians for some reason associate it with rheumatism." Profuse salivation is present.

In *lambs* it is according to Haubner an atrophy with destructive ulceration of the bones of the face, complicated by purulent infiltration of the medullary spaces. "The incisors, and later the molars, fall one by one, because of the changes in the alveoli, the gums swell, become violet, red, and ulcerate, the ulcers extending through the hard palate into the nose, and causing a highly offensive discharge from both nose and mouth.

In *pigs* the disease has been mainly seen in connection with insufficient or unwholesome food, and badly balanced rations, and especially with fermented swill and an exclusive maize diet. The symptoms are shown in the limbs and face, especially ("snuffles"), as noted under rachitis.

Prevention and treatment are to be sought in avoidance of the obvious causes, and in applying the same line of tonic treatment as in the larger animals.

INDEX.