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TEXT BOOK

OF

VETERINARY MEDICINE

BY

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PREFACE.

DURING a long experience in teaching veterinary medicine and surgery in Cornell University, the author felt the urgent need of a compend on the subject, written from the American standpoint and having special reference to the American live stock industry. This led to the production of the Farmer's Veterinary Adviser, which has been well received, and has, up to the present, passed through ten editions in the United States, besides the unauthorized editions published in Canada and Great Britain.

In entering upon a larger field as Director of the New York State Veterinary College, and professor of medicine and sanitary science, he aims at producing a work which will meet the needs of the American student and practitioner. The special phases of animal pathology in America, the diseases peculiar to our soil, and the parasites that prevail here, but are unknown in Europe, demand consideration from the American point of view. The special features of our breeding, grazing and feeding industries, and of the dairy, over our great extent of territory, and the varying influence of soil, water, climate, altitude and traffic, the scope and limitations of our interstate traffic, and our special relation to the old world in the matter of meat products, combine with other conditions in demanding a somewhat different treatment of the subject from that which we find in European publications. Then, too, the recent extraordinary advances in the field of bacteriology and sanitary science, which have virtually revolutionized modern medicine, and are an earnest of still greater advances in the near future, demand a work which shall, as far as possible, set forth the present advanced status, and thus lay a solid foundation to intelligently follow, if not to lead, in the imminent advance. As a contribution to this, the present volume, the first of a series, is offered to students, practitioners and scientists by their friend,

THE AUTHOR.

New York State Veterinary College,
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October, 1896.

VETERINARY MEDICINE.

OBJECTS AND METHODS OF STUDY.

Pathology—general—special. Morbid anatomy. Pathological chemistry; Disease. Health. Death—Somatic—partial—necrosis. Syncope. Apucea. Asphyxia. Coma. Death from old age.

The principles and practice of Veterinary Medicine should embrace all that is known of the causes, nature, symptoms, prevention and cure of disease in domestic animals. Incidentally it includes diagnosis and prognosis.

Pathology is the science which tells of the causes, and nature of disease, and the functional and structural changes by which it is characterized. In modern usage the term pathology is understood to refer to the intimate nature of disease, but this necessarily involves an enquiry into its sources and the predispositions to its occurrence; its phenomena whether in changes of function or structure; and its results in the form of perverted function, structural changes, degenerations, dependent disorder, etc. The field of pathology is further divided into general pathology and special pathology.

General Pathology treats of disease processes in their generic form, and as they appear in many different diseases. Thus inflammation and fever are the prominent phenomena in a great many different diseases which differ in their seats, their causes, manifestations and results. Inflammation and fever are therefore subjects of general pathology. Similarly all forms of degeneration—fatty, fibrous, calcic, amyloid, etc., are disease processes found in many different organs and under very varied conditions and they are accordingly included in general pathology. Hypertrophy and atrophy are also possible in every organic tissue irrespective of kind or seat, they belong therefore to this particular field.

Special Pathology on the contrary is confined to a particular disease and not only elucidates the causes, phenomena and results of such disease, but seeks to do this in such a way as to differentiate this malady from all others however closely related to it. Thus inflammation of a bone is known under the general name of osteitis, this may be due to a great variety of different causes, and each would have its own special pathology. The osteitis of simple mechanical injury is essentially different from the osteitis of rheumatism, of purulent infection, of tuberculosis, of actinomycosis. So with the inflammations of every other tissue. Each may suffer from a variety of inflammations, springing from different causes, attended with characteristically unlike tissue changes and tending to different issues, and every one of these forms has therefore its own special pathology.

General Pathology may be said to deal with typical disease processes to a large extent irrespective of the individual disease in which they may appear, while **Special Pathology** deals with the morbid phenomena which distinguishes the individual malady from all other diseases however closely allied to it.

Pathological (Morbid) Anatomy deals with structural changes, the cause, the accompaniment or result of disease. These morbid changes are microscopic or macroscopic. Both constitute morbid anatomy, but the microscopic alterations come under the special name of morbid (pathological) histology.

Pathological Chemistry is that branch of pathology which treats of chemical changes produced by disease in the blood, lymph, tissues, secretions and excretions. It demands a previous knowledge of the condition of these tissues and fluids in health, in the particular genus of animal and under the same dietary and environment. Physiological Chemistry is therefore an essential prerequisite to pathological chemistry, just as anatomy, physiology and histology are indispensable to the appreciation of pathology and morbid anatomy.

Disease is an injurious deviation from the normal function or structure. The morbid process resulting in disease is usually in the nature of a modification of the normal or physiogical condition, so that it is often difficult to set the exact limits of health and disease. What is a purely physiological process under given conditions, would be distinctly pathological under others. The

free kidney secretion of cold weather and the profuse perspiration of a hot season are both purely physiological and in the main balance each other. Each under its special environment fulfills an essential work in eliminating from the system toxic materials which would prove hurtful if retained, and thus each is not only physiological but beneficial. If, however, they occurred, not in this mutually compensatory manner, but simultaneously in this profusion, they might well be dreaded as morbid conditions. Again if either were to occur apart from its normal causative environment, if for example the polyuria appeared in hot weather and the perspiration in cold, the phenomenon might fairly be called pathological. In any case if the excessive secretion induced a lowering of the general tone of health the process would be essentially a morbid one. In pronouncing therefore upon a morbid process one must take fully into account the corresponding physiological process, the attendant conditions, and whether the result is injurious or otherwise.

The same is true of structural changes. What under given conditions would be essentially a morbid structure, might under other conditions be a simple adaptation to an unwonted environment, and a means of protection from injuries that would otherwise accrue. Excessive growth of cuticular tissue in the epithelioma, wart or corn is injurious and essentially pathological, while the callus on the camel's knee or the workman's palm is purely protective and physiological. The local development of a mass of fatty tissue in the average man or beast is a disease, but the tendency to the uniform deposition of fat in the connective tissue of the improved breeds of meat producing animals, is the happy culmination of a long continued and skillful selection and regimen, without which the live stock industry of today would be a grievous failure. To constitute disease, therefore, modified function must be permanent, and not simply a compensating increase, decrease, or other change, and it must be in some way injurious to the animal economy. Similarly to constitute disease modified structure must be other than a simple protection or beneficial change, it must not be a simple evolution in the nature of accommodation to the environment but it must be a cause of injury to function or a distinct deformity.

Health may be said to be the harmonious exercise and mutual

balance of all the bodily functions, and any interference with such mutual exercise or balance may be said to constitute disease. But as health passes into disease by insensible gradations, there is of necessity an extensive borderland which often cannot be allotted to one condition or the other, but which must often be left a disputed territory.

Again certain animal constitutions are innately strong and robust, while others are weak and feeble, yet the delicacy of the latter cannot be set down as actual disease, and by maintaining a due balance between the functions, a fair measure of health and even long life may be secured.

Death as the result of disease may be either *partial* or *somatic*.

Partial or **local death** may be *molecular* as in **ulceration**, or it may affect an organ or part of an organ, as in **necrosis**, **sphacelus**, or **sloughing**. **Somatic death** is a loss of vitality of the entire body and is manifested by a complete cessation of the bodily functions, including that of nutrition. Usually the arrest begins with one of the great vital processes, in advance of the others, and thus in different cases, we have *death beginning at the heart, at the lungs, and at the brain.*

Death from syncope or fainting, begins at the heart, which loses its irritability or contractility, or is seized with a tonic spasm. If there has been lack of contractility, the heart is found after death in a flabby, soft condition, and quite frequently filled with blood. If heart-spasm, it is contracted, firm, and empty or nearly so. Syncope may result from severe nervous shock (emotional), from the electric current, from insolation, or from heart sedatives like chloroform, or nicotine. It may, however be but the culmination of a gradually advancing debility, from exhausting diseases, from fatty degeneration of the cardiac muscles, or from starvation, or anæmia. Again the exhaustion coming from profuse hæmorrhage, or from violent over-exertion is a cause of fatal syncope.

In **death beginning at the lungs** (apnœa, asphyxia, or suffocation), the blood failing to receive oxygen and to give up its carbon dioxide is unable to maintain the various functions of the body and the arrest of the other vital processes speedily follows. The arrest of the respiratory process may occur from nervous shock, but more commonly it results from choking, strangula-

tion, drowning, or the action of irrespirable gases. In diseases of the heart and lungs it is liable to occur from the obstruction of the pulmonary circulation and from the depression of the respiratory nervous centres. After death the lungs are found gorged with dark red—almost black—blood, which likewise distends the right heart and systemic veins, and all mucous and serous membranes have a dark red, congested aspect. When breathing has been arrested by mechanical violence there are, first, active contractions of the respiratory muscles, but no loss of consciousness; then as the brain becomes charged with venous blood, consciousness and volition are lost and convulsive movements ensue. Later still there is no respiratory effort nor convulsions, but the heart continues to beat for two or three minutes longer.

In death beginning at the brain (Coma) the sensory functions fail first, as evidenced by drowsiness, stupor, or complete insensibility, while the movement of heart and lungs are still temporarily continued. Pressure on the brain by a fractured bone or blood clot, or in cases of violent congestion or the rapid growth of tumors, usually operates in this way. It may also result from the direct action of certain poisons, like opium, belladonna, or chloroform, or the ptomaines or toxins of bacteria. Causes acting on the brain may, however, lead to death by syncope or asphyxia when the nerve centres presiding over circulation or respiration are the first to feel the full effects of the pressure or poison.

Death from old age, with a gradual failure of the natural processes of nutrition and tissue-growth, and the occurrence of atrophy and various degenerations of the organs is not a common occurrence in domesticated animals, so that it may be dismissed without further notice.

Actual somatic death is marked by the cessation of breathing and pulse, the dilated pupils and semi-closed eyelids, the coldness and pallor of the visible mucous membranes and skin, and the clenching of the jaws with slight protrusion of the tongue. Yet these symptoms may be present in syncope and it may even be impossible to detect the beats of the heart, though the subject still lives. Pressure of the finger on a white portion of the skin or on a mucous membrane may give a further indication. If the indentation made by the finger is slowly effaced and if the blood again slightly reddens the part the presumption is against death.

Even this is not infallible, since by pressure of gas in the internal cavities or deeper blood vessels, the blood may be forced back into the surface capilliaries giving an appearance of circulation, after actual death. On the other hand any exudation or œdema will retain the imprint of the finger even in life. The general relaxation of the muscles and their lack of response to electric stimulation, and the setting in of cadaveric rigidity, and later still of putrefaction give more conclusive evidence of dissolution.

ETIOLOGY: CAUSES OF DISEASE.

Causes—simple—complicated: Proximate; Remote: Predisposing—race, genus, family, heredity, individual, environment, food, age, sex, temperament, idiosyncrasy, debility, plethora, interdependence of organs, embolism, mechanical influence. Exciting causes, intrinsic, extrinsic, inherent, acquired, heredity, dentition, heat, cold, atmospheric conditions, electricity, moisture, dryness, dust, darkness, light, soil, food, water, inaction, overexertion, mechanical causes, poisons,—mineral—vegetable—animal, microbes, contagious, infectious, epizootic, enzootic, sporadic, panzootic, zymotic, mediate contagion, bacterial poisons.

The causes of disease are simple or complicated, and in the latter case a single factor may be altogether harmless unless associated with another which also may have been innocuous alone. For example: the infecting germ of glanders (Bacillus Mallei) is harmless to the ox which lacks the predisposition to the disease:—feeding buckwheat is harmless to the dark-skinned animal, but is injurious to the white-skinned, if exposed to sunshine:—the chicken can bear with impunity exposure to cold or to the bacillus anthracis, but it cannot endure these two etiological factors combined. It follows that one cannot predict the same result from the same cause in every case. Yet with all concurrent conditions the same the result will follow with mathematical certainty. This will serve to illustrate the value of thoroughness in etiological knowledge, as the basis of a sound pathology.

Etiology is primarily divided into **proximate** and **remote**. *Remote* causes are again divided into **predisposing** and **exciting**.

Predisposing Causes are such as induce a condition of the system or of a particular organ or group of organs which renders them specially susceptible to a disease. This may be a characteristic of the *race* or *genus* of animal, thus the genus bovis alone suffers from lung plague, the genus equus from dourine, and ruminants from Rinderpest. It may be a *family trait*, (*hereditary*) hence we see certain families of both men and cattle cut off by tuberculosis, while other adjacent ones largely escape. It may be an *individual peculiarity*, thus some subjects have a congenital insusceptibility to a given disease, from which others of the same family suffer, and one who has passed through a self-limit-

ing disease like measles, cowpox or anthrax is rarely attacked a second time. Again predisposition may be due to *environment* as when we find herds in damp and exposed localities obnoxious to rheumatism, and horses in dark mines exposed to specific ophthalmia. It may be the result of *food* as when the flesh-fed fox or rat resists anthrax and the farina-fed one falls a ready victim. *Age* may predispose, early youth being remarkably susceptible to parasitism and bacteridian infection, and old age to fractures and degenerations. *Sex* is inevitably a cause of limitation of disease as the females and males can only suffer from disease of their respective sexual organs. Again of diseases common to both sexes certain nervous and digestive disorders are common in connection with gestation, and certain calculous diseases in connection with the long and narrow urethra of the male. *Temperament* has a marked influence, thus the sanguinous or nervous race-horse or hound shows a marked predisposition to diseases of the heart, lungs and brain, and to a sthenic type of inflammation and fever, while the heavy lymphatic draught-horse has a proclivity to diseases of the lymphatics and skin. *Idiosyncrasy* is closely allied to temperament, but the condition may be less manifest, and the peculiarity is only recognized by the results, as when a man is poisoned by sound fish or raspberries. *Debility* whether from deficiency or poor quality of food, on the one hand, or from overwork, filth, dampness or disease on the other must be looked upon as strongly predisposing to certain diseases, such as tuberculosis and glanders. *Plethora* which charges the blood and tissues in a different way with effete organic products, lays the system especially open to certain diseases like black quarter in young cattle, and parturition fever in cows. *Disease of one organ* often predisposes another organ through interdependence of function, as when torpid or congested liver leads to portal and intestinal congestion, diseased teeth to digestive disorder, imperfect hæmatosis to kidney trouble ; in other cases blood clots or bacteria from one pathological centre may be arrested in the blood vessels of a distant organ and start new foci of disease (embolism, metastasis) ; in still other cases the impairment of the healthy function in one organ acts injuriously on another, as when emphysema or other disease of the lungs forces the blood back upon the heart causing dilation with atrophy of the walls.

Previous disease in a tissue leaves for a time an impairment of structure and function which may become the essential predisposing cause of the effective operation of a morbific factor. *Mechanical* action on a part may predispose to disease, as for example, by reducing its circulation and nutrition and thereby directly impairing its power of resistance to other inimical agencies. Not infrequently a pus microbe lies deep in the cuticle or even in the tissues without harm, until there occurs a bruise, or a bony fracture when it at once develops a focus of purulent infection (abcess).

Exciting Causes are the immediate causes of particular diseases. Like the predisposing causes they may be *intrinsic* or *extrinsic*, and the first may be **inherent** or **acquired**.

Among **inherent** causes are certain of those already named as predisposing causes, but which have come to be forcible enough to develop disease without the intervention of any other observable factor. Thus a *hereditary* monstrosity (redundancy or defect), will appear in successive generations without any apparent additional cause. The appearance of white calves in herds of black cattle, after the whitewashing of their stables shows a similar hereditary operation though the result is not in this case pathological. The birth of blind foals from blind sires or dams, or of foals with distorted feet from mares suffering from severe chronic foot lameness are true pathological sequences, in which the exciting cause is hereditary and operates during intrauterine life. *Dentition*, as an attendant on early life is often a directly exciting cause, from direct injury by entangled or retained teeth that should have been shed, by fever aroused by the active local changes, or imperfect mastication or insalivation leading to consequent indigestion; in puppies and kittens convulsions are not uncommon as a result.

Extrinsic Causes are such as operate through the environment. *Heat*, if excessive and prolonged, relaxes and exhausts the system and exerts a direct influence on the process of sanguification so that it may become the direct cause of a variety of diseases. As the result of extensive burns, dangerous congestions of internal organs are liable to occur, and even the prolonged heat of summer often superinduces hepatic and gastric disorder, diarrhœa and dysentery. Fat cattle in uncovered cars or yards

under a hot sun and with no breeze suffer extensively from insolation, the temperature of their bodies rising to 110° to 112° Fah. and even higher. *Cold* is equally potent. With a temperature below zero Fah., the iron bit will freeze the buccal mucous membrane, and cause extensive erosion of the mouth. The cold of salted snow or ice will freeze the feet, causing sloughing of the skin above and around the coronet and shedding of the hoofs, and predisposing to fatal septic infection of the wounds. On the system at large, cold causes retrocession of the blood upon the internal organs, and endangers the occurrence of acute disease in any structure which is already debilitated or otherwise susceptible. The nervous effect of the chilling of the skin is often the unbalancing factor which sways the scale in favor of disease, which the system was able to resist until this disturbing element was introduced. The sudden chill from passing out of the warm barn into the frosty air, from plunging into icy water, from standing in cold rain or sleet, from standing in a draught of cold air especially when perspiring, is a fruitful source of many diseases. In the cow, lying with the udder on a cold stone may be the starting point of mammitis. The effect of sudden chill is well exemplified in the great prevalence of diseases of the respiratory organs at the change of the seasons in spring and autumn when the vicissitudes of temperature are greatest, and the system unprepared by habit, to bear the sudden change. Again it must be noted that exposure to cold has a tendency to cause disintegration and solution of the red blood globules, and that certain animals are especially susceptible to this influence. *The condition of the atmosphere* is often a direct cause of disease as when charged with offensive or irritating gases, the result of decomposition of organic matter, with sewer or cesspool emanations, with deleterious gases from chemical works, telluric sources, or fires. A low state of health, a local irritation in some part of the air passages, or even a speedy asphyxia may be the outcome of such atmospheric conditions. Again the presence of solid particles of a more or less irritant, septic or infecting kind prove the starting point of various diseases. The stone cutters' phthisis, and the sand granule ophthalmia are familiar examples of the irritant, which often acts through the dust of the highway. Of the infecting particles we have the germs of cattle lung plague, of infective ophthalmias,

and of tuberculosis carried with the dry dust and inhaled. Of toxic agents borne on the atmosphere we see the compounds of arsenic, mercury and lead. Moisture and dryness of the air induce respectively a lymphatic constitution and low tone of health, and a nervous constitution and a tendency to neurosis, ophthalmias, and skin diseases. The pressure of the atmosphere has a profound effect on animal health as seen in the extreme troubles of the heart and circulation in the diving bell, and the respiratory, hæmorrhagic and brain affections of high attitudes. A low barometer is attended by nervous disorders (neuralgia) (S. Weir Mitchell). Surgical operations do best with a high or rising barometer (Adinell, Hewson). The electrical tension of the atmosphere shows familiarly, in man, in the feeling of heaviness, dullness and malaise that precedes the bursting of a thunderstorm and the relief that follows its termination. To this influence many of the domestic animals are incomparably more sensitive than man, as witnessed in the disposition of swine to hide in their pens or under litter on the approach of the storm, the nervous disturbance even to abortion of certain ewes which are heavy in lamb, and the great discomfort and even piteous cries of some domestic felines on such occasions. The greatest electric tension is seen in the drier climates, where the air, robbed of its moisture, proves a poor conductor and equalizer, and the positive and negative electricity get stored up separately in air, cloud and soil. The presence of ozone in the air, as a habitual concomitant of electric discharges, has been supposed to be a disturbing influence, since it is distinctly irritating to the mucous membrane when present in excess, but such excess apart from its artificial production is highly improbable. As habitually met with it is antiseptic and health giving. *Darkness* always deteriorates the general health, producing bloodlessness and pallor. *Light* is usually invigorating, yet bright sunshine falling upon the eyes from a window in front of the stall, or in the open air when the face is turned up by an overdraw check rein, or reflected from white dust and, above all, from snow, will often induce inflammation and blindness. *Soils* are often potent etiological factors. Dense, damp, cold, undrained soils, are habitually covered by a stratum of cold air, saturated with moisture, which greatly lowers the vital stamina. Damp clays, and waterlogged soils of various kinds, rich in

organic debris, are the natural homes of various pathogenic microbes, such as those of ague, anthrax, milk sickness, actinomycosis and yellow fever. Well drained sandy or gravelly soils are usually healthy, unless they contain a great excess of decomposing organic matter. Again soils with an excess of alkaline or other mineral matter may prove deleterious, and those on the magnesian limestone often harbor the poison of goitre, and cretinism, and favor the occurrence of urinary calculus. *Faulty food and feeding* in the domestic animals are chargeable with many diseases. Stock often fall off in condition, in the hands of one feeder, when the same food given with regularity and judgment by a more careful feeder would keep them in the highest health. Hay and grain which is musty and filled with cryptogams and their products, are common causes of disorder of the stomach, the kidneys, the nervous system or of general nutrition. Smut and ergot at certain stages of their growth or grown under given conditions cause nervous disorders, abortions, and gangrene of the extremity. A long list of vegetable poisons may mix with fodders, and animal poisons with the food of the Carnivora. A number of standard fodders may be poisonous at certain stages of growth, as partially ripened perennial rye grass, millet, Hungarian grass, vetches, etc. *Water and deprivation of water* are fertile causes of illness. Ruminants cannot chew the cud when deprived of water, hence impaction of the first and even of the third stomach with fermentations, tympany and other disorders. Horses suffer more from a full drink of water after a feed of grain, the unchanged albuminoids being carried on into the intestines, and both gastric and intestinal indigestion induced. Sheep suffer fatal fermentations after drinking the alkaline water of the Plains; cattle have diarrhœa and dysentery from selenitions, or from stagnant and putrid water; and the water from the dolomite is the usual channel of the goitre poison. Certain germs like the plasmodia of malaria, and comma bacillus have their natural home in impure water, and others like anthrax bacillus survive in the mud and silt at the bottom of wells, ponds, and rivers and enter the system in the water. *Compulsory rest* in a stall often induces torpor of liver and bowels, general muscular debility, and fatty degeneration especially of the liver and heart. A few months of the swill feed, hot atmosphere and absolute rest

in a distillery stable usually ruins cattle for stock purposes. *Overexertion* on the other hand is prolific of illness. Exhaustion of the muscles, congestion, inflammation, cramps, congested lungs, heart failure or rupture, apoplexies and other hæmorrhages are among the resultant maladies. Auto-poisoning is another result shown in equine hæmoglobinæmia, and the fever of leucomaines. The excessive development of sarcolactic acid from muscular work may render an insusceptible animal susceptible to the anthrax bacillus. *Mechanical causes* would include overexertion, in the production of strains, fractures, and other injuries. They would also include impaction by foreign bodies, calculi, and ingesta, friction of folds of skin or by harness and other objects and pressure which leads to absorption and atrophy. To these must be added *poisons of vegetable, mineral and animal origin* and the *microörganisms* which act as injurious ferments within the animal body. These will be treated more fully later on. Of the microörganisms it may be said here, that they are almost certainly the cause of all transmissible diseases. These diseases are variously named on the basis of different ideas. They are **enthetic**, that is implanted as a seed is planted in the ground to grow and multiply. They are **zymotic** or fermentative because the essential cause multiplies and is propagated like a ferment. They are **contagious** because propagated by contact mediate or intermediate. They are **infectious** when transmitted, not alone by contact but through the atmosphere. They are **epizootic** because they tend to attack animals generally or a given genus or family of animals generally when these are exposed to the infection. They are **enzootic** when confined to the animals in a given locality, the soil or conditions of which are favorable to the preservation of the germ in pathogenic potency, or to the production of a special susceptibility in the animal system. They are **sporadic** when each case occurs without any casual relation to another. They are called **panzootic** when they attack all animals without apparent preference. The term *panzootic* is also used to describe those recrudescences of a disease or cycles of exalted pathogenesis which are observed in contagious diseases, which frequently last for years and again give place to a period of benignancy. Such cycles, of malignancy and benignancy, may be due to modified environment acting either on the disease-germ or the animal system, or on both simultaneously.

The terms **enthetic**, **zymotic**, and **contagious** best express modern views of the nature of these maladies. The term **infectious** when used to express a gaseous or otherwise intangible (unorganized) body, or influence transmitted through the air, necessarily excludes the particulate, living, self-propogating germ upon which the transmissibility of the disease depends. A chemical, electrical, or other body or influence generated outside the animal body, cannot well be conceived of as reproducing itself within the animal body but must act like any other ectogenous poison, according to the size of the dose and the frequency of its exhibition. This might create an *enzootic* disease but would lack all the qualities of a contagious affection since it could not spread from a victim when taken elsewhere and turned among animals which would prove equally susceptible if placed within the infecting area. Suppose on the other hand we apply the term *infectious* to diseases in which the levity of the particulate living germ allows of its being inhaled into the body of the susceptible animal, the case becomes one of simple *mediate contagion*, the air acting as the intermediate bearer.

The term **zymotic** conveys a clear idea of the method of increase of the disease germ in the body by the ordinary process of generation. The old doctrine of fermentation by a continuous change, due to contact with dead fermenting matter, as an inflammable body continues to burn by contact with the incandescent portion, has been definitely disproved by the investigations of Pasteur and others, and today we must recognize that every fermentation is the result of the propogation and vital activity of living organisms. This does not ignore that the chemical products or enzymes which are constructed by the vital activity of the microbes, will dissolve or transform organic matter, but in the absence of the microbe no such enzyme can reproduce nor multiply itself and its action must therefore be exactly limited by its amount. The living germ itself is therefore the one effective factor, by which the contagious disease may be maintained and propagated. In its turn the living germ can only come from a pre-existing living germ. To the scientist of today the doctrine of spontaneous generation is a thing of the past and the aphorism *omnis ovum ex ovo* is dominant. The argument drawn from the saccharizing of starch in the germinating seed by the operation of

diastase is inapplicable, as the diastase is produced by the living cells of the germinal part of the seed, which are thus the counterpart of the disease germ. No such glycogenic action occurs in the seed that has been boiled or otherwise robbed of its vitality. So with the arguments drawn from the ptyaline of the saliva, the pepsin of the gastric juice, and the trypsin of the pancreatic juice; each of these is the product of the living cells of the gland by which it is secreted, and cannot increase its own substance in the absence of these cells. Like the enzyme of the bacteridian ferment, these gland products can break down or digest certain organic matters, but in all alike, the only source of the chemical solvent is the living bacterium or gland cell from which the particular product is derived. The toxins of a virulent liquid, after the sterilization of the latter may still produce most of the lesions and morbid phenomena of the disease, but, although death were to ensue, the body of the victim would not be infecting to other susceptible animals. The parallel between the functions of the secreting animal cells and the disease germs may thus be put in tabular form:

Living Source.	Chemical Product.	Result.
Salivary gland cells	= Ptyaline	= Starch changed to Sugar.
Peptic gland cells	= Pepsin	= Albuminoids changed to peptones in acid solutions.
Pancreatic gland cells	= Trypsin	= Albuminoids changed to peptones in alkaline solutions.
Disease germ	= Toxin: Enzyme	= Morbid phenomena.
Disease germ	= Contagious disease.	

Further consideration of pathogenic microörganisms will be found in connection with contagious diseases.

MEDICAL DIAGNOSIS.

Means of diagnosis. Usual health of the subject. History of the attack. Objective symptoms, inter.dependent disease, fever, diseases that may be confounded, subsidiary disease, diagnostic signs, organ involved, pathological test injections, course of disease, sporadic or zymotic, result of treatment.

Diagnosis is the determination of the seat and nature of a given disease and its distinction from other morbid conditions. Its importance to the practitioner cannot be overestimated as it occupies the pivotal position between causes, nature, morbid phenomena, and symptoms on the one hand, and prognosis, prevention, and treatment on the other. Unless the conclusions are sound as to causes, nature, lesions, and symptoms, there can be no certain diagnosis, and without a correct diagnosis, prognosis, prevention, and treatment can have no intelligent or scientific basis. The practitioner who finds a dropsical condition and who is satisfied to pronounce it dropsy and institute treatment is abusing his trust. He must find whether this dropsy results from disease of the kidneys, heart, blood-vessels, lymphatics, liver, lungs, bowels, or the structures in which it is shown ; whether it is due to parasites or imperfect sanguification or to other morbid conditions, before he dare prescribe treatment and predict results. So in every other affection ; the failure to make a correct diagnosis opens to the practitioner many doors of error, and he is happy indeed if he can escape the injuring of his patient.

In seeking a sound diagnosis we must attend to the following among other indications :

1st. The habitual state of health of the subject. The genus, breed, age, environment, habits, (pet dog, watch dog, hound, sheep-dog, ox, bull, cow, milch cow, sheep in the field or housed, pig in pen or at large, diet, regimen, water, race horse, draught horse, work, exposure, etc.) as well as the personal equation of temperament, idiosyncrasy, heredity, etc., must all be carefully considered.

2d. The history of the present illness as to its apparent cause, mode of invasion, duration and progress.

3d. The objective symptoms by which it is manifested. All that can be ascertained in the way of symptomatology, local and

general, the probable existence of interdependent disease, and all actual structural lesions and disorders of function should be thoroughly investigated. As supplementary to the more prominent objective symptoms any fever or other constitutional disorder must be sought for; a mental list must be made of the diseases which resemble this one, and these must be excluded one by one by careful attention to the differential symptoms; other diseases which are probably subsidiary to this, should be similarly investigated and excluded; any really diagnostic sign of the suspected disease must be carefully established and the diagnosis finally placed on a solid foundation. The discovery of a constitutional (febrile) disease to which a distinctive name can be given is by no means the end of the diagnosis; the structural lesions of the disease may be largely localized in an unimportant organ where they may remain circumscribed without compromising life, or they may be seated in a vital organ which will render the disease grave to the last degree or necessarily fatal. For example: Anthrax of a dense, dry part of the skin may be a mild local disease; anthrax of an internal organ is usually fatal. Every local complication therefore, should be as carefully diagnosed as the connected constitutional disorder.

But diagnosis cannot always be certain. In the early stages of certain fevers two forms may be as yet indistinguishable and a day or two may be required to develop differential symptoms. In some occult forms of disease all differential symptoms may fail us. A method of diagnosis which has hitherto been applied only to tuberculosis and glanders is manifestly capable of much wider application, to diseases attended with a febrile reaction. This consists in a hypodermic injection of a minimum dose of the sterilized and filtered products of the culture of the disease germ, which produces no effect on the healthy system but causes febrile reaction or local inflammation, or both, in the diseased. This will be treated more fully under the respective diseases.

In connection with such a method, but above all when no such resort has been had, the obscure case should be seen frequently, the course, duration, and termination of the disease should be noted, also its tendency—sporadic or epizootic, and finally the result of treatment. This last resort may often secure diagnosis and cure at once as when a course of iodine cures an obscure actinomycosis.

SYMPTOMATOLOGY. SEMEIOLOGY.

Definition. Symptom. Sign. Constitutional symptoms—local, objective, subjective, direct—idiopathic, indirect—symptomatic, premonitory. Anamnesis. Position. Movements. Decubitus. Acute. Chronic. Fever. Sthenic. Asthenic. State of limbs, muzzle, nose, snout, palmar-pad, hoof, bill, digits, mouth. Thermometry. Normal temperature, in doors, in field, at work, in hot season, in nervous subject, in thirst, in youth—age, starvation, plethora, cold, sleep, rest, stimulants, suppressed perspiration, eliminants, antipyretics. Fever temperature, morning, evening, transient elevation, persistent rise, sudden fall—collapse, crisis. Fatal elevation. Rise during defervescence. Pulse. Respiration. Skin, staring coat, pallor, coldness, dryness, harshness, mellowness, pliancy, hide-bound, yolk, clapped wool, scurfy, lesions, itchiness, tenderness, loss of hair, emphysema, anasarca, sweat, sebum. Expression, life, dullness, paralysis, dropsy, jaundiced, eye, discolorations, photophobia, amaurosis, pinched face. Nasal mucosa, red, violet, etc., nodules, polypi, osseous disease, pentastoma, œstrus, discharge from teeth—sinuses—actimomycosis—tumors. State of the bowels, kidneys, nervous system.

The usual basis of diagnosis must be a clear and intelligent observation of the symptoms of disease. A *symptom* is an appreciable evidence of disease. A *symptom* however may indicate illness, without affording the means of diagnosis, while the term *sign* is often used for a pathognomonic symptom—one by which the disease can be identified. Used in this sense a *sign* may be said to be a diagnostic symptom.

1. **Constitutional Symptoms** are such as affect the entire system, like a rise of body temperature, or a shivering fit.

2. **Local Symptoms** are confined to a definite area as redness, tenderness, swelling, ulceration.

3. **Objective Symptoms** include all that can be recognized by the senses of the observer. These alone are available in dealing with the lower animals.

4. **Subjective Symptoms** can only be felt by the patient himself, as pain, giddiness, cold, heat, blindness, numbness. Such symptoms are therefore only obtainable from the human patient who can tell how he feels. In the lower animals they can only be matter of inference, thus pain may be inferred from lameness or wincing on pressure, and giddiness from unsteady

gait. The fact that the veterinarian is restricted to objective symptoms renders his task a specially difficult one, yet this has its compensation, as this very restriction tends to train the observant practitioner to greater skill.

5. **Direct Symptoms** (**idiopathic**) are those which are connected with the seat of disease, as the redness, exudation, and swelling of inflammation.

6. **Indirect** (**sympathetic, dependent**) **Symptoms** are observable at a distance from the actual disease :—as when headache attends on dyspepsia, or lameness in the right shoulder upon disease of the liver.

7. **Premonitory** or **precursory symptoms** precede the diagnostic symptoms of some diseases, thus dullness and languor often heralds an approaching fever, and the strangles of young horses is often preceded by a general unthrifty appearance, poor appetite and indisposition to exertion.

In observing symptoms as in other things, some have far greater natural ability than others, but in all a careful training will do much to develop and improve the power and habit. A most important thing in such habits is the strict maintenance of a system, not to be followed as a cast iron rule but to be constantly kept in mind and strictly carried out except when sound judgement and experience show it to be unnecessary.

Anamnesis. As a rule the first thing to be learned about a patient is his history, and personal and hereditary characteristics. What are his general health, temperament, previous attacks, hereditary predisposition, environment? Is the site of the building, its condition as regards soil, springs, drainage, structure, ventilation, light, cleanliness such as would favor any particular disorder or class of disorder? Is the patient in high, low, or moderate condition, robust or debilitated, alert and lively or dull and stupid? Have other animals suffered recently, or at a corresponding season, or under similar conditions in apparently the same manner? How long has the patient suffered, were there any premonitory indications of illness, what were the first symptoms, and what symptoms have followed up to the present? Has there been any change of food, water or management that might throw light on the cause? Has there been any change of weather or unwonted exposure to cold, storm, overwork, com-

pulsory abstinence or enforced retention of some secretion? If a female is she pregnant?

Having exhausted this method, using such lines of inquiry as promise good results in the particular case, the veterinarian is prepared to bring his own powers of observation to bear more directly.

Position and **movements** will often furnish valuable data. The *horse* which lies on his ribs, stands obstinately in chest diseases, or whenever there is much interference with breathing. The ruminants and carnivora on the other hand which lie on their smooth or padded sternum, can breath with comfort in this position and only stand up persistently in the worst cases. The habit of standing day and night is also characteristic of anchylosed back or loins in the solipede. Roached back may be natural, or the result of overwork and slight sprains or injuries of the loins, of anchylosis, of intestinal or renal inflammation, or of certain injuries to the limbs. The extension of the head on the neck may suggest sore throat, chest disease, tumors around the throat, abscess (fistula) of the pole, sprain or spasm of the extensors of the neck, disease of the axoido-atloid joint, tetanus, or cervical rheumatism. Dropping of head and neck might suggest paresis, mechanical injury to the levator muscles or cervical ligament, extreme debility, or prostration from a profoundly depressing fever or poisoning. Inability or indisposition to back, might indicate sprain or fracture of the back, anchylosis, laceration of the sublumbar muscles, paresis, cerebral or spinal inflammation, softening or other lesion, tetanus, laminitis, dislocated patella and certain other affections. Swaying or unsteadiness in walking or turning would similarly suggest sprain or fracture of the back, paresis and other nervous and locomotor injuries. The solipede with peritonitis arches the back and draws the hind feet forward under the belly, with impacted colon or obstruction to urination he will often stretch with fore limbs advanced and hind limbs retracted. The mode of decubitus may be significant. With peritonitis, enteritis, metritis or acute nephritis or hepatitis the solipede lies down slowly and with caution: with spasmodic colic he throws himself down as if reckless of possible injury. Lying well up on the costal cartilages and side of the breast bone suggests a slight affection of the air passages; lying on the side,

disease of other parts. Rolling on the back may indicate simple intestinal spasm, but also blocking by intussusception, impaction, volvulus or otherwise. Sitting on the haunches may suggest a similar trouble or it may imply ruptured stomach or diaphragm. The dog may sit on his haunches in health, or with dyspnœa in acute affections of the respiratory organs, asthma and heart affections. Decubitus on the belly with hind legs extended backward, may imply paraplegia, or acute inflammation of the abdominal organs. Lying with the nose in the flank or turning the head toward the flank, though a normal position of rest, often indicates abdominal suffering. Turning of the head to one side may, however, suggest injury, spasm or rheumatism of the cervical muscles, or disease on the corresponding side of the brain. Animals, at liberty, lie more frequently on the side on which the heaviest internal organs are lodged, thus ruminants, pigs, and dogs rest on the right (the side of the liver) though in cattle with a heavily loaded rumen the condition may be reversed. Decubitus on the abdomen, with the limbs extended and abducted implies profound nervous disorder or shock.

Habitual decubitus often indicates severe suffering in legs or feet. Resting one limb more than another implies injury to that limb. Standing with the pastern of one limb more upright than the others has the same meaning. Extension of one fore foot in advance of its fellow with flexion of the pastern and fetlock denotes suffering in the posterior part of the foot or in the flexors. Flexion of carpus and fetlock without advance of the foot probably bespeaks injury to shoulder or elbow. Inability to bear weight on the fore limb, without knuckling at the knee, should call for examination of the olecranon and joints especially the elbow. Inability to extend the carpus should lead to investigation of the flexor muscles and tendons, the joints and the heel. Movement of the hind limb without flexure of the tarsus would suggest injury to that joint, the stifle or the flexor metatarsi tendon. Inability to extend stifle and hock, should demand examination of the tendo-Achillus and olecranon, of the triceps extensor cruris and of its nerves.

Atrophy of a muscle or group would require scrutiny of its tendons and its nerve and blood supply.

More precise indications of injury of the locomotor system must be found under surgery.

After posture, the general or constitutional disorder may claim attention. Is the illness acute or chronic? Is fever present? Has the animal had a rigor? Does the coat stare in patches (along the spine) or generally? Is there perspiration? Is there full, clear, somewhat congested eye (sthenic) or drooping lids over a dull brownish sclerotic (asthenic). Are the lower parts of the limbs and other extremities cold, and the roots of the horns or ears hot? Is there significant heat and dryness of the muzzle (ox), nose (dog), snout (pig), palmar-pad (carnivora), hoof (solidungala, bisulcates), bill and digits (birds)? Has the mouth the hot burning feeling of fever? Finally is the temperature as indicated by the thermometer abnormally high? To estimate this with any degree of certainty one must be well acquainted with the normal temperature.

Normal temperature. As taken indoors under ordinary conditions, the normal temperature taken in the rectum may be: fowl, 107°—110° F.; swine, 103°—106° F.; goat and sheep, 103°—104° F.; ox, 100°—102° F.; dog, 99°—100° F.; horse, 99°—99.6° F. Ranging in the fields, at work, or on forcing or stimulating feeding, it may be 1° higher than when at rest indoors. A whole herd may be raised 2° by a three miles drive in warm weather. In our summer heats a rise of 1° is common. In nervous animals any change in management may raise the temperature, for example, 1° to 2° after failure to water at the usual time, or from retaining the milk in the udder when the milker had been changed. Young animals are normally .5° to 1° warmer than old ones though more sensitive to the action of cold. Half starved animals, when put on abundant and nutritious food may have a rise of 1° or more. Females in heat, in advanced pregnancy and at the time of parturition are usually 1° to 3° above the natural temperature. Among the agencies lowering temperature are: cold, (1° to 2°); sleep, (1° to 2°); rest; starvation; alcoholic and other circulatory stimulants which fill the cutaneous capillaries and thereby cool the whole mass of blood; suppression of insensible perspiration (retention of waste matters) as by varnishing the skin which lowers the temperature to 25°; purgatives and diuretics (1°); certain drugs like antipyrin, acetanilid, etc., which act on the heat producing centres and retard metabolic changes.

Temperature in disease. Comparative temperatures should

be taken at the same hours on successive days, bearing in mind that the morning temperature is usually slightly lower and the evening one slightly higher. Where possible both morning and evening temperature should be taken. With elevated temperature, repeat sooner to see that it is not transient. A transient rise of 1° to 2° is unimportant. A permanent rise of 2° or 3° indicates fever. A sudden additional rise of several degrees in the progress of fever is grave. A persistence of the high evening temperature to morning shows aggravation. A persistence of the low morning temperature to the evening bespeaks improvement. A sudden extreme fall to much below the normal (4° or 5°) indicates collapse. This is usually attended with other symptoms of extreme prostration and sinking. A sudden considerable fall to near the normal, without untoward attendant symptoms, may indicate a *crisis* and a more or less speedy improvement may be hoped for. This sudden fall often attends the period of eruption of certain exanthemata, as cowpox, horsepox, sheeppox, aphthous epizootic, etc. A sudden extensive fall of temperature may result from some transient accidental cause, as a prolonged deep sleep, a hemorrhage, the relief of constipation, or of enuresis. A sudden rise may supervene on such suppressed function or other cause of nervous irritation or on toxin poisoning, but it does not persist more than twelve or twenty-four hours after the cessation of the morbific cause.

A rise of 10° or 12° above the normal standard is usually promptly fatal.

A continued high temperature indicates persistent disease, and a considerable rise during defervescence implies a relapse and in the absence of any error in diet or nursing is grave.

Pulse. Before the introduction of the clinical thermometer, the indications furnished by the pulse were held to be of the highest value. Though largely superseded by the usually more reliable thermometer, yet they should not be discarded, but employed as symptoms corroborative of the thermometric indications. In many cases the pulse will furnish criteria, when in the absence of fever, the heat of the body will tell of nothing amiss. This is especially true of diseases of the heart, the large blood vessels, and of the nervous system, and in cases of poisoning. For special indications furnished by the pulse, see diseases of the heart.

Respirations. The morbid activity or inactivity of the respiration, its modified rythm, the pathological significance of the altered breathing sounds and of the superadded sounds, the indications furnished by percussion, palpation, mensuration, succussion, sneezing, snorting, yawning, cough, moan, grunt, stertor, discharge, etc., afford material of inestimable value to the diagnostician. See under diseases of the chest.

Skin Symptoms. The erection of the hair of carnivora in rage or fear implies a profound nervous disturbance, and a similar erection (staring coat) in the larger herbivora especially, implies a corresponding nervous disorder, due however to a different cause. The pallor and coldness of (white) skin and extremities the retrocession of blood toward the internal organs, the contraction of the involuntary muscles of the hair bulbs, the sense of cold, and the actual shivering all come from the fundamental nervous disorder. The loss of lustre and gloss in the hair and the dryness, rigidity and mobility (mellowness) of the skin imply lack of nutrition. The mellow feeling of the skin under the pressure of the finger, soft and yielding by reason of the lax connection tissue and fatty layer in the thrifty animal, is in marked contrast with the dry, hard, tough, unyielding hide firmly adherent to the parts beneath (hidebound), which denotes the unhealthy or unthrifty animal, or from the thin, attenuated, mobile, bloodless skin of the debilitated subject, the victim of lung, liver, or intestinal worms. In sheep in parallel conditions there is a lack of *yolk* in the wool, which is dry, lustreless and brittle and often flattened (clapped) on the skin. In fowls ruffling of the plumage indicates the nervous disorder and chill. The skin may be scurfy in conditions of low health or in connection with the presence of vegetable or animal parasites. Ringworm has excessive scurf, and tends usually to a circular form, and to complete shedding of the hair from the spots. The hairs split up before dropping. In acariasis there may be scurf, scab, abrasion and sore of many kinds, but the outline is not necessarily circular, nor strictly limited, isolated hairs remain even on the bare patches, and itching is extreme as shown by the movement of the body and especially of the lips or foot when the part is scratched.

The hair may be freely shed during convalescence from debilitating diseases, a condition that must not be confounded with

the yearly shedding of the winter coat and the moulting of birds, which is a perfectly normal process. Yet even the spring shedding and the growth of the new coat makes a great drain on the system, and must always be taken into account as a probable cause of derangement of health.

The lesions of the skin in the different cutaneous affections must be remanded to the special chapter on skin diseases. The following however may be named as having a general bearing.

Emphysema may be due to a local wound, (elbow, trachea, rib); it may indicate black quarter, or it may occur sub-cutaneously in cattle without marked impairment of health.

Anasarca, from diseased blood, heart, liver or kidneys is denoted by swellings, often painless, or a general infiltration which pits on pressure. It often shows primarily in the lower parts of the hind limbs. Warty looking elevations must be carefully discriminated, having in mind primarily papilloma, tubercle (grapes), actinomycosis, condyloma (in dogs), cancer, melanosis. The secretions of the skin (sweat, sebum) may be suppressed, or in excess, producing at times a special odor, as in thrush and canker of the horse, cowpox and sheep-pox, and rheumatism. Before death the cadaveric odor may be marked, and attracts crowds of flies to the victim.

Facies. The countenance may be expressive. Between the bright, full, clear, prominent eye of health, and the dull, sunken, lifeless, semiclosed eye of serious disease the contrast is extreme. The drooping lids (ptosis) may be paralytic and even unilateral, in which case drooping ear, and flaccid lips and alæ nasi complete the picture. With paralyzed lips there is usually drivelling of saliva, and dropping of half chewed morsels in the manger and stall. The eye may show dropsical lids in kidney or liver disease and in anæmic conditions like distomatosis in sheep. It may show the upper lid bent at an angle in recurrent ophthalmia of solipedes. The mucosa may be red in ophthalmia, yellow in jaundice, dusky brown in Southern cattle fever, anthrax, cerebral meningitis, and other fevers attended with destruction of red globules and liberation of their hæmatin. The pupils may be all but closed in internal ophthalmias, or widely dilated and irresponsive to light in amaurosis. The iris may lack its normal lustre or may be distorted or torn in various ways from adhesions. Opacities of the cornea, lens, or vitreous may be recognized.

The facial muscles may be flaccid and devoid of expression in palsy, and prostrating diseases; they may be firm, giving the bright, intelligent look of health; or they may be painfully drawn in the agonized expression of spasmodic colic or enteritis.

Nasal Mucosa. The pituita is bright red in sthenic fevers, simple acute coryza, strangles, laryngitis and inflammation of the larger bronchia. It assumes a violet hue in capillary bronchitis, pulmonary congestion, glanders, and petechial fever. Petechiæ appear in the last named affection, and in a number of bacteridian diseases, such as anthrax, swine plague, hog-cholera, the red fever of swine etc.; a yellow tinge in shown in jaundice. Millet like or pealike nodules, or elevated patches, and ulcers show in glanders and may be felt by the fingers. In cattle hard millet-like nodules appear in a chronic coryza with hypertrophy of the mucosa. The orifice of the lachrymo-nasal duct, seen in the horse on the floor of the chamber at the friction of the mucosa with the skin of the false nostril and in ass and mule on the outer ala near the upper commissure, is sometimes plugged with inspissated mucus. Among other lesions of the nasal chamber may be named polypi—soft and calcareous,—thickening and obstruction in purpura hæmorrhagica, osteoporosis and hypertrophy of bone, and parasites—pentastoma denticulata (in the horse and dog), and the larva of the oestrus (in sheep and buffalo). Disease of the upper molars and abscess of the fronto-maxillary sinus may be manifested by swelling beneath and on the inner side of the eye, fœtid discharge from the nose, and obstruction of the air current. Dullness on percussion will show the filling of the sinuses. These conditions must be carefully differentiated from actinomycosis, sarcoma and other morbid growths in the same situation.

Costiveness with fœtor and lack of the normal color in the stools may suggest *liver torpor* or inflammation, while fatty stools may suggest pancreatic disease. The uneasy movements of colic, should lead to a careful investigation of the chylo-poietic organs (see digestive organs). Weakness of the hind parts, tenderness of the loins, and altered condition of the urinary discharge should demand a close enquiry into the state of the *kidneys* and *bladder*. Satyriasis or nymphomania would suggest disease of the *generative organs* or the nerve centres that preside over them. The same is true of impotence, sterility and abortion.

In eruptions on the skin (erythema, eczema, pustule, squama) a cause may be found in the local action of heat, friction, or other direct irritant, but in the absence of any such manifest cause, an enquiry should be made into the functions of sanguification, digestion, urination and the action of the liver. It may further suggest parasitism (ring worm, phthiriasis, fleas, acariasis, verminous disease, etc.)

Symptoms of *nervous disorder* are too numerous to be here traced to local lesions. Motor paralysis of one limb may, however, suggest injury to its motor nerves, to the same side of the spinal cord, or of the opposite half of the cerebrum. Paraplegia almost always indicates injury to the cord. Senory paralysis of one side may depend on disease of the opposite corpus striatum. The animal moves in a circle when a tumor (coenurus in sheep) exists in the roof of the lateral ventricle presumably pressing on the ganglia on its floor. An animal rolls on its axis when there is a lesion of the median cerebral peduncles, of the supero-external portion of these peduncles, of the posterior part of the encephalon, or of different parts of the hemisphere. Amaurosis suggests disease of the corpora quadrigeminia. Loss of coördination of muscular movement usually implies some lesion of the cerebellum. Vertigo may imply disease of the encephalon (congestion, anæmia, inflammation, dropsy, hæmorrhage, tumor, abscess); it may be disease of the internal ear; it may be digestive disorder connected often with cryptogamic poisoning; it may be heart disease with obstruction of the jugular veins; it may be parasites in the nasal sinuses; or it may be disease of the eye. Coma occurs in most congestions and pressures on the encephalon, and like vertigo in poisoning by alcohol, solanine, monoxide of carbon, etc. In acting on any ganglionic centre the agent may, according to its degree, operate positively or negatively, producing spasms, or paralysis as the case may be. As in the case of other visceral affections the specific diseases must be referred to for particular symptoms.

For the more precise points in diagnosis, including chemical, physical, electrical and instrumental methods, etc., the reader is referred to the special diseases.

PROGNOSIS.

Definition. Demands on the veterinarian, the question of economy. Basis of Prognosis. Cause of the illness, internal or external, vital or non-vital organ, enzootic, fatigue, infection, in one or two symmetrical vital organs, regular or irregular in its course, persistence, relapse, complications, effect of treatment, appetite, temperature, pulse, breathing, youth, age, debility, previous disease, breeding, climate, season.

Prognosis is a more complicated question for the veterinarian than for the physician. The latter must pronounce on the malady, whether it is likely to follow a regular or irregular course, whether it will last short or long, whether it will be curable or incurable, and if curable whether recovery would be complete or partial. For the veterinarian there is in addition the question of economy. The veterinary patient is, as a rule, of value, only if he can be rendered sound, and a partial recovery may be even worse than a fatal result, since the subject remains as a ruinous charge on his owner. The veterinarian must pronounce on the prompt and perfect curability of the case, on the outlay that will be requisite for treatment, on the depreciation which will be entailed on the patient, and whether, in certain lesions that do not harm the carcase, it would not be more judicious to butcher the subject. The physician is expected to do the best he can for life and health, and even a very imperfect recovery brings him a mead of gratitude. The veterinarian on the other hand must be an expert not only on disease, but on animal values, and if his treatment, however skillful it may be, results only in the prolonging of the life of an useless animal, the owner may charge him with imposing upon him an unnecessary outlay. The soundest judgment and highest skill are often necessary to secure the interests of an employer in such circumstances. In certain cases the recommendation to destroy is of much more value to the employer than the most skillful, and partially effective, curative treatment. On this basis, the reputation of a skillful man may be securely built. He can deceive no one if his prediction of recovery is not justified, while if he advises destruction and the patient recovers, he is at once discredited.

To give a sound prognosis the practitioner must have a thorough knowledge of pathology, he must have acute powers of observation, and he must be quick to appreciate every point that makes for or against the patient in the particular case.

The *causes* of the trouble must be carefully considered. Are they transient or permanent? Are they removable or irremovable? Are they external or internal? As a rule an internal cause is the more redoubtable. Some lesions are necessarily fatal, as a needle penetrating the heart or an attack of rabies or milk sickness. Is the cause an enzootic one? If so can the patient be removed from the locality? Is it a fatigue fever or an infectious one? Is it a simple inflammation or an infecting one? The latter are usually much more grave. In case of contagious disease, can its propogation be prevented? Is it of a fatal or non-fatal type? Is it situated in a tissue favorable to a fatal extension, (anthrax in lung) or in one unfavorable (anthrax in the tip of the tail)? Disease in a single vital organ like the heart is necessarily much more grave than in one of a symmetrical pair (kidneys, lungs) one of which can carry on the functions. The regular progress of the disease and especially an uninterrupted improvement, following on a critical perspiration or urination, is a good prognostic sign, whereas unevenness of temperature, pulse and respiration, with temporary aggravations of the general symptoms, should demand a less hopeful prognosis. The persistence of the malady is also an unfavorable indication. A relapse after partial recovery is a serious indication unless due to some obvious and easily removable cause, and unless the former convalescent condition is speedily restored on its removal. A complication is a serious indication whether it consists in an embolism, or new centre of the same disease, or the supervention of a second disease upon the first. The system has just so much more to contend with and the very supervention of the second focus or malady argues a special susceptibility, debility, or lessened power of resistance.

The prompt success or entire insuccess of treatment proves valuable.

The preservation of appetite, the slow, uniform descent of the temperature, and the improvement of pulse and breathing are among the most valuable indications.

Something may be deduced from the condition of the patient. If very young or old, debilitated by over-work, bad or insufficient food, previous disease, or any other cause, the prognosis is less hopeful, as it is also as a rule, during gestation, in the parturient state, or if abortion ensues. A hereditary predisposition to the malady in question is equally unfortunate.

Climate may be an important factor. Thus liver diseases are far more to be dreaded in a damp tropical or semi-tropical region, and rheumatism and catarrhal affections in winter and in cold northern localities. Acclimatization should also be considered. The bovine animal, raised on the Gulf Coast is likely to make a good recovery from Southern Cattle Fever while the northern beast would almost certainly die.

All in all the question of prognosis cannot always be judiciously decided at a first visit, and for the sake of his own reputation, it is well that the practitioner should give only a qualified opinion at first until he can certify himself as to the probable outcome of the disease.

PROPHYLAXIS. PROPHYLACTICS. PREVENTION.

A test of public sentiment. Soil. Water. Exposure. Buildings. Local hygiene. Breeding. Diet. Work. Harness. Ventilation.

With advancing knowledge of veterinary medicine the subject of prophilaxis is steadily assuming a more important place, and especially in the classes of enzootic and epizootic diseases. Indeed for the fatal infectious diseases of animals one can fairly estimate the medical intelligence of the people by the extent to which therapeutic treatment is still allowed. With economy as the great central object of veterinary medicine, the problematical recovery of the few can never balance the assured preservation of the many. But this subject belongs to contagious diseases to which the reader is referred.

In enzootic affections, improvements in soil, water, exposure, buildings, and other local unhygienic conditions, are the final ends to be sought, according to the particular nature of the prevailing disease.

So in sporadic diseases the correction of faults in breeding, hygiene, diet, water, work, harness, exposure, buildings, ventilation, etc., are called for in different cases as will be noted under the individual diseases.

THERAPEUTICS. TREATMENT.

Definition. Mechanical and Medicinal Therapeutics. Adaptation to each case of disease.

The ultimate object of all medicine is to prevent disease or when it cannot be prevented, to cure. The term therapeutics covers all measures applied with curative object. Therapeutics are naturally divided into **Mechanical and Medicinal.** To mechanical therapeutics pertains the whole domain of surgery. Medicinal therapeutics has to do especially with internal medicine. Each of them, however, encroaches more or less on the other. Modern surgery is essentially aseptic or antiseptic, and antisepsis is secured by medicinal agents. In medicine when cups are applied we adopt an essentially mechanical treatment. Both methods then must remain open to physician and surgeon. Another and no less important branch of treatment which is open to physician and surgeon alike is diet and general hygiene. The same care must be given to the use of these in the treatment of disease as in its prevention, and in many cases a judicious use of these may almost entirely obviate the necessity for medicine.

It would be useless to enter here into the subject of therapeutics. Suffice it to say that the choice of a system and of individual agents must be determined by the particular conditions of the case, its cause, and nature, the strength, vigor, and genus of the patient, the organ involved, the extent and stage of the disease, the existence of a relapse, or complication, and all other circumstances that would affect the action of the remedy. Specific statements must be made with the several diseases.

HYPERÆMIA. CONGESTION.

Definition. Forms, active—arterial, passive—mechanical—venous. Determination of blood. Causes of active congestion. Vaso-motor nerves. Lesion of spinal cord; or of sympathetic nerve. Reflex irritation. Central cause. Physiological hyperæmia. Medicinal hyperæmia. Bacteridian (toxic) hyperæmia. Arterial obstruction. Thrombus, tumor. Cold, chill. Removal of pressure. Cardiac hypertrophy. Symptoms, bright red color, swelling, dropsy, migration of cells. Rise of local temperature. Tenderness. Altered function. Causes of passive congestion. Obstructions in the lungs, heart, veins. Diminished force of circulation from age, debility, arterial disease, distance from the heart, decubitus, vaso-motor disorder. Gravitation—hypostatic congestion. Tumors. Paresis. Symptoms. Cyanosis. Distended veins. Coldness. Transudation—watery. Hæmorrhage. Thrombus. Hyperplasia. Atrophy. Postmortem lesions. Treatment. Remove Cause. Correct injurious gravitation. Correct any fault in blood pressure. Derivation. Constringe or support part. Massage. Electricity. Improve general health.

Definition. An excess of blood in a part. It is distinguished from inflammation by the absence of that tissue reaction, which leads to or constitutes the special phenomenon of that morbid process.

Hyperæmia is divided into **active** or **arterial** and **passive, mechanical** or **venous.** A capillary form has also been described but usually capillary congestion is seen in both the arterial and venous types.

I. **Active** or **Arterial Determination of blood.** In this form the arteries are dilated under a direct nervous influence. *Causes.* In all the regular functions of the body, the flow of blood is under the direct control of the vaso-motor nerves which proceed from the spinal cord, through the branches of the sympathetic to be distributed with the blood vessels. The hard pulse of pleurisy is due to rigid contraction of the constrictor muscles under the action of the vaso-motor nerves, and the blush of shame is due to their relaxation. Some claim an active dilatation of the arterial muscular coats, others look more simply upon the dilatation as a mere yielding of the coats under the blood pressure, when the constrictor muscles are relaxed. This vaso-motor paresis may be induced: 1st, by any lesion of the spinal cord. 2d, by the

cutting of a sympathetic trunk, that of the abdomen, for example, which leads to active congestion of the abdominal viscera, or the section of the cervical sympathetic which leads to watering of the eye, sweating, congestion, and scabbing on the corresponding half of the face. 3d, by reflex irritation through the sensory nerves, as in congestion through friction, heat or cold to the skin, or that resulting from excessive use of an organ such as the mammary gland. 4th, by causes acting directly through the brain as in emotional blushing or the facial congestion of violent rage.

Physiologically we see the operation of this nervous control in the congestion of the gums during dentition, of the salivary glands during mastication, of the stomach and bowels during digestion, of the womb during gestation, of the mammæ at parturition, and of erectile organs in copulation.

Medicinal agents act in the same way, opium or alcohol producing active dilatation, and belladonna and ergot causing active contraction of the arterial walls.

Bacteridian poisons act in the same way, tuberculin and a number of others causing active dilatation.

The obstruction of one artery by thrombus, tumor, or ligature, causes increased tension in the collateral branches coming off just above and an active congestion in the parts to which these are distributed. While this is directly due to increased local pressure, it is also an instance of the lack of balance between the blood pressure and the resistance of the vascular walls. In this case there is increase of pressure, in the other a diminished resistance.

If there is a superficial anæmia, as from cold or chill, there is of necessity, an internal hyperæmia. This contributes to the production of internal congestions and inflammations, though the seat of election of such inflammation is usually determined by the nervous sympathy between the part chilled and the deeper organ affected.

Another cause of congestion is the lessening of pressure by the parts surrounding the vessel. Thus in cupping, there is prompt cutaneous congestion, and a similar result occurs in pericardium, pleura, or peritoneum on the withdrawal of the liquid of hydropericardium, hydro-thorax or ascites.

Another cause of congestion is found in hypertrophy of the

heart and increased force of the blood flow (blood tension). In such cases those organs become congested in which there is some previous debility or disease of the blood vessels.

Symptoms and results. The symptoms are a bright vermillion redness, tension or swelling, heat and tenderness. Pulsation is stronger in the vessels leading into the part, secretions tend to increase but may give place to a serous effusion or hæmorrhage. The bright redness is attributed to the rapid circulation of the red globules which have not time to give up their oxygen to the tissues. It is sharply circumscribed where the affected arterioles have no free anastomosis with those of neighboring parts, diffuse where anastomosis is abundant, and when on the skin it is liable to rise in knots or buttons as in urticaria. When pressed the redness entirely disappears unlike the redness of inflammation.

The swelling may be due to the simple turgescence of the blood-vessels, but also often to transudation of serum as in and around the cow's udder at parturition. The occasional migration of globules, and their escape through minute lacerations in the vascular walls add alike to color and turgescence.

The elevated temperature, (rising sometimes 3° C.) in the congested area, is attributed to the more active circulation, and Schiff prevented its appearance after section of the cervical sympathetic, by tying the carotid and vertebral arteries on the same side.

The tenderness of the congested parts varies inversely as the looseness of texture and the facility for swelling. It may be scarcely perceptible in the mammary region, and intense under the horn or hoof.

The functions in the congested organ are often seriously interfered with, secretions appearing in excess or entirely altered. When the congestion lasts it may cause hypertrophy, induration or hyperplasia, these are however rather sequels than lesions of the condition. Simple congestion is usually quite transient, and if prolonged, often merges into inflammation.

II. **Passive or Venous Congestion.** In this there is no excess of blood entering the part, but the regular supply is delayed in the veins by some obstruction, and these vessels and, later, the capillaries are gorged with black blood.

Causes. 1st, Mechanical obstruction to the onward flow of blood, as in the case of disease of the lungs hindering the flow of

blood from the right heart ; disease of the right heart allowing a reflux of blood into the veins ; or pressure by tumors or otherwise on the great or small venous trunks. If in the heart or lungs the whole systemic venous system becomes the seat of passive congestion ; if in a single venous trunk then only the parts the venous radicles of which are tributary to this. We find examples of this in phlebitis, in compression by the swellings of strangles, in the result of a bandage or ligature tied round a limb at some distance from its extremity, and in the compression of the iliac veins by a gravid womb.

2d. Diminished force of the blood current in the veins, as from old age or great debility and especially from weakness of the heart's action. Also from disease of the arterial coats which impairs their tonicity. The force being too weak to force the blood actively through the capillaries and veins, it becomes unduly charged with carbon dioxide and other products of tissue waste, so that nutrition suffers and the walls of the capillaries lose their vital force. This condition is aggravated in the hind limbs by the distance from the heart, and the dependent position, and in decubitus by the compression of the vessels of the limbs. Also by injuries to the vaso-motor nerve supply as œdema appeared in the hind limb after tying of the femoral vein in animals the abdominal sympathetic of which had been cut, but not in animals in which this nerve was left in its normal condition (Ranvier).

3d. Gravitation in weak states of the circulation must be looked upon as a cause of venous congestion. This is seen in the examples of hypostatic congestion and œdema seen in the lungs and other internal organs in low conditions and in the advanced stages of debilitating diseases, and in certain cases of stocking of the limbs in horses.

4th. Valvular insufficiency of the left heart and tumors or aneurisms interfering with circulation through the aorta, cause passive congestion of the pulmonary veins and œdema of the lung.

5th. Tumors and diseases of the liver determine passive congestion of the portal system and ascites.

6th. Passive congestion is very liable to take place in an organ the functions of which are impaired as in a paralyzed part. In this the hyperæmia may start in the capillaries and extend to the veins or even to the arteries.

Symptoms and results. If on a mucous membrane or white skin the color becomes dark red, or violet (cyanotic) with evident distension of the capillaries and veins, the latter of which may stand out as knots or cords, there is an appearance of swelling or enlargement and sometimes coldness of the part. Soon the watery part of the blood transudes in excess, constituting dropsy, with increased swelling and pitting on pressure. On the mucous surfaces it determines an abundant serous secretion. The color is deepened by the escape from the vessels of red globules as well as white. The transudation contains little albumen and only exceptionally fibrine. In connection with the marked deoxidation and high carbonisation of the blood, the nutrition of the part is largely arrested together with the functions, secretory, motor or otherwise. The imperfectly nourished vessels may give way, leading to hæmorrhage, or nutrition may be definitely arrested producing moist grangrene or ulceration. Sometimes a thrombus is formed in a congested vein. The changes in the affected organs depend much on the degree and duration of the hyperæmia. If slight and lasting it causes permanent induration and thickening, from connective tissue hyperplasia as frequently seen in the hind limbs of the horse. In case of blood transudations the altered coloring matter gives the various shades of gray, brown or black. If long continued the organ may shrink and atrophy occur from defective nutrition and contraction of the fibrous hyperplasia.

In making post mortem examinations mistakes may be made through the occurrence of changes after death. Thus a hyperæmia which was quite considerable during life may virtually disappear through the contraction of the arterial and capillary coats forcing the blood on into the veins. A minute point of extravasation here and there may be the only macroscopic lesion left. Again a marked venous and capillary hyperæmia in a dependent part of the body or of an organ may be entirely due to hypostatic conditions, the blood having settled into the lowest part of the vessels since the death of the animal. To avoid this source of error one must always carefully note the position of the carcass after death. Under other circumstances the superficial veins and capillaries may fill up with blood through the occurrence of decomposition and the evolution of gases in the internal cavities, which empty the splanchnic and parietal vessels by compression.

Treatment. The general principles of treatment may be stated thus: 1st. Remove the cause of the hyperæmia if possible, especially any mechanical cause; 2d. Secure the influence of gravitation in favor of the return of blood to the heart; though not so available in animals as in man, it is of great value in congestions of the head, ears, tail, and to a less extent of other parts; 3d. Correct any fault of blood-pressure, excess or deficiency, which may act so as to cause active or passive hyperæmia; 4th. Establish derivation by cupping, leeches, fomentations, pediluvia, sinapisms, etc.; 5th. Apply cold, astringents, bandages, to empty the hyperæmic vessels, or kneading, rubbing, or electricity, to hasten the flow of blood; 6th. To improve the quality of the blood and general health, in plethora by low diet, purgatives and diuretics, in anæmic or debilitated conditions by iron, bitters, nourishing food, fresh air, sunshine and exercise.

It is especially important to check passive congestion in febrile diseases, and mechanical congestion at an early stage of its progress (Roberts).

INFLAMMATION. PLOGOSIS. PHLEGMASIA.

Definitions. Relations to active hyperæmia. Redness. Heat. Pain. Swelling. Forms: in vascular tissues: in non-vascular. Changes in tissue elements. Death of cells. Cloudy swelling. Granular degeneration. Cell proliferation. Karyokinesis. Embryonic cells. Amœboid functions. Migration of leucocytes. Red cells escaping. Changes in innervation. Vaso-motor disorders. Fever. Changes in circulation. Contraction of capillaries, dilatation, rapid flow, tardy flow, stasis, oscillations, thrombus, collecting of white globules in periphery of current, migration of leucocytes, blood plates, and red globules, massing of red globules, exudation, softening of the capillary walls, nutrient artery more rigid and transmits more blood, heart contracts more forcibly, increase of fibrine, ncrease of waste products. Buffy coat, physiological causes. Microbes. Ptomaines. Toxins. Chemiotoxis. Phagocytosis. Polynuclear and mononuclear leucocytes. Exudates, unlike dropsies. Mucous exudate. Serous exudate. Fibrinous exudate. Blood exudations. Croupous exudation. Chyliform exudate. Results and Products. Resolution. Deletescence. Metastasis. New formations. Suppuration. Pus microbes. Pus. Healing by 1st intention. Healing by 2nd intention, granulation. Granule corpuscles. Interstitial neoplasia. Degenerations in lymph. Fatty degeneration, melanotic. Softening. Ulceration. Gangrene.

Inflammation has been variously defined as "perverted nutrition," as a "protective reaction of the organism against irritant agents" and in other terms that express at once too much and too little, without actually defining the morbid process. Older definitions dealt with the manifest disorders of circulation, of innervation or of tissue change too often exalting the importance of one set of changes at the expense of another and thus giving in the main a one sided view of the morbid process.

Some modern bacteriologists are inclined to refuse the title to any morbid process that is not caused by the presence of microbes or their toxic products. To them the changes occurring in an aseptic wound or in a simple fracture in process of healing are purely reparatory and partake no more of the nature of inflammation than do the developmental changes in the growing embryo. While to a large extent true, this exclusive view implies exceptions, since if the chemical poisons derived from the bacteria can develope inflammation, the same must be admitted as possible for chemical irritants drawn from other sources.

As a matter of fact inflammation occurring as it does in very different tissues (vascular and non-vascular, fibrous, cellular, parenchymatous, etc.), and in connection with a great variety of irritants, must be held to include a large group of morbid processes bearing to each other a strong family relationship and resemblance, and yet differing in many important details. Each irritant (heat, cold, electricity, chemical irritant, incised, punctured, lacerated or contused wound, rupture, fracture, foreign body, parasite, microbe, toxin, etc.) has its own special character and mode of irritation; each tissue has its own special method of succumbing or reacting and its own amount of blood supply; and each system and organ has its own native or acquired power of resistance and reaction.

Inflammation agrees with active hyperæmia in the tendency to dilation of the vessels and an increased flow of blood to the part or if the irritated part is non-vascular like the cornea or articular cartilage, then to the parts adjacent. It differs, however, in the more active cell proliferation, and in the nature of the liquid transudation which is richer in albumen, fibrine, cells and phosphates. Abstractly the inflamed part retains very active vital processes, trophic and exudative, but these are largely changed from the normal and are, it is claimed, perverted, yet they preside over the processes of cell growth and decay, the removal of injured or useless tissue, and later, over the building up of new material, and repair of loss. Active hyperæmia on the other hand is mainly a circulatory disorder, and when it advances so as to determine changes in the cells and tissues it is held to have merged into inflammation.

The term inflammation (from inflammo, I set on fire) is suggestive of the local heat of the inflamed part, just as fever (febris) indicates an elevation of the temperature of the body at large. Celsius enumerated the features of rubor, calor, dolor and tumor (redness, heat, pain and swelling) which have come down to our own time as at least suggestive of inflammation. But any diagnosis, based on these alone, would be today woefully inadequate. Redness occurs in the transient blush, heat in the febrile state, though no inflammation can be recognized, pain is present in neuralgic and other nervous affections, and swelling in dropsy and tumor. On the other hand redness is entirely absent, for a time,

after the outset of inflammation in nonvascular tissues (cornea, articular cartilage), the heat of the inflamed part may be actually lowered when there is much exudation around the capillary vessels and lessened flow of blood, pain may be absent in some circumscribed inflammations of the lungs, and swelling is not at first visible in the inflamed cornea or compact bony tissue. These phenomena which are so common in inflammation and, in general so characteristic of it, cannot therefore be accepted as infallible evidence of its existence, nor can their absence be held as absolutely implying its nonexistence.

Forms of Inflammation. This morbid process might be divided almost indefinitely according to the organ invaded, the cause, and type, yet it will be more convenient to deal with it generically and notice **inflammation in nonvascular and vascular tissues** respectively, and the different types of **granular degeneration, exudative inflammation** and **croupous inflammation.** It will be requisite further to notice an **acute** and a **chronic** type.

By dealing first with the changes in the anatomical elements of the tissues and in the innervation, we shall virtually cover the phenomena observed in nonvascular tissues, and later the changes in connection with the circulatory system will give the additional characteristics of inflammation in vascular tissues.

CHANGES IN THE TISSUE ELEMENTS.

Death of cells and tissue By the application of an irritant (acid, heat, etc.,) a certain thickness of tissue with its enclosed cells is killed, and a thin layer of necrosis is usually produced. This does not constitute inflammation, but it acts as a foreign body, often septic, in producing inflammation in the parts adjacent.

Cloudy Swelling, Granular Degeneration. This may occur in the inflamed area surrounding the necrosed tissue in the seat of a burn or other injury, it is exceedingly common in the cells of inflamed parenchymatous tissue (liver, kidney), in the muscle of the heart, in the gastro-intestinal mucosa, in febrile affections and in poisoning with arsenic, phosphorus, or mineral acids. The gross appearance of the tissue is that of swelling, with a dull

grayish color and a loss of its normal translucency. The cells of the affected organs are seen under the microscope to be filled with small albuminous granules which may be so abundant as to completely conceal the cell structure. The granules are insoluble in ether, but disappear under acetic acid. This condition of the cells is often associated with the exudative forms of inflammation.

Cell Proliferation and Change. In the nonvascular organ attacked by inflammation the multiplication of tissue cells and their resumption of amœboid movements is a constant phenomenon. Virchow insisted on the fundamental relation of the cell to the morbid process, and Goodsir and Redfern showed the rapid increase of the cells of articular cartilage in attacks of arthritis. There is first a sensible increase of the nucleus of the cartilage cell which shows a more extended and deeper staining in carmine or aniline; then by a special method of division (karyokinesis) the cell and nucleus divide in two; by a similar process these divide in four and so on in regular order. Meanwhile the cartilaginous substance becomes softened and finally dissolves and disappears, leaving in the place a mass of closely aggregated cells.

In the nonvascular transparent cornea, the membrane of Descemet, the epithelium of serous membranes and in the epidermis a similar cell multiplication occurs, also in the lateral cartilages of the horse's foot.

To follow the indirect cell division by karyokinesis, we must note the cell as a semi-solid mass, formed of protoplasm and nucleus, each having as its framework a network of exceedingly fine inter-crossing filaments, much finer in the nucleus than in the cell protoplasm. The nuclear filaments stain with hæmatoxylon and safranin and are called *chromatin* threads. The intervening non-staining material is *achromatine*. The nucleus has a membraneous envelope in two layers, of which the inner only stains. When about to divide two poles are formed in the cell protoplasm opposite to each other and near the nucleus the filaments concentrating to the poles. The chromatin threads in the nucleus thicken, become convoluted, split and multiply, and draw into their substance the chromatin layer of the envelope. Next the chromatin threads form long loops directed toward an achromatine centre or pole like a star, and this is followed by the progressive division of the star-shaped mass into two equal parts.

Finally they separate, together with the cell protoplasm, forming two daughter cells.

This cell proliferation under the action of an irritant is common to the vegetable kingdom in which galls, and tumors are formed in this way. It is a remarkable feature of these multiplying cells that they not only lose their power of developing the tissue in which they formerly lay, and have all their vital powers devoted to proliferation, but they acquire the amœboid power of their ancestors, the embryonic cells, which they further resemble in size. Indeed these cells are freely spoken of as embryonal cells, and the tissue formed by their massing together as embryonal tissue, and there is a widespread impression that they revert entirely to the form and characters of the embryonic cell. In some respects, however, they are unlike. The modified tissue cell of inflammation presents a nucleus of horseshoe outline, or after division of the nuclei they together retain this semi-circular outline; it has the power of actively digesting the adjacent tissues as the embryonic cells do not, and again it does not possess the power of differentiation into widely different tissues as does the early embryonic cell. It may be called a reversion, in the direction of the embryonic cell, however, since it reacquires a number of its functions.

Migration of white blood cells. This is another, and in vascular tissues the main source of the great cell accumulation in the inflamed tissue. This process was observed by Waller in 1846, but was given its true importance through the later observations of Colnheim. The migration takes place through the walls of the capillaries and veins only, and the migrating cells are largely of the poly-nuclear variety of leucocytes. These remaining adherent to the inner wall of the blood vessel may be seen to have a small portion of their substance projected through the wall and appearing as a small buttonlike projection on the outer side. This gradually increases, while the remaining portion of the cell on the inner side of the wall correspondingly decreases until the whole cell is lodged in the tissue outside the vascular wall. The time occupied in passing through is very varied. It may be wholly accomplished in half a minute, and again hours may be required for the complete passage of a single leucocyte. The explanation of this migration has been sought in the supposed

existence of stigmata (openings) in the vascular walls (Arnold), in the effect of the blood pressure within the inflamed vessels, in softening of the vascular walls and, in the contractility of the leucocyte which is strongly attracted by the pressure of certain bacteria and other irritants (chemiotaxis). The migrated leucocyte assumes in the tissues the same habit as the altered tissue nucleus. It multiplies rapidly, assists in the solution and removal of the inflamed tissue, contests the ground with infective microbes (phagocytosis), and subserves the purpose of assisting in building up new tissue, or of degenerations.

Red Cells. The red blood globules follow the active current in the centre of the blood vessel, yet a few of these also become adherent to the softened walls and pass through them (diapedesis). When stasis of blood takes place in the vessels, they become packed more closely with red globules which then pass outward into the tissues in much larger numbers.

Changes in innervation. As shown under hyperæmia the vaso-motor system of nerves exerts a potent influence on the circulation and is largely instrumental in bringing about circulatory disorders. The increase in the number and force of the contractions of the heart, and the rigid contraction of the walls of the arteries proceeding to an inflamed part, are distinctly the result of a reflex nervous action. The implication of the second eye when one has been violently inflamed from a mechanical injury is another example of this kind. The loss of power of the vaso-motor nerves is however even more characteristic. Experimentally the cutting of the cervical sympathetic or crushing of the superior cervical ganglion causes congestion and finally inflammation of the structures on that side of the head; the crushing of the semilunar ganglion similarly affects the abdominal viscera; and the cutting of the pelvic plexus, the structures of the hind leg. The contraction and dilatation of the inflamed capillaries is largely a nervous phenomenon. A certain number of irritants, like warm water, mustard, or ammonia cause contraction followed by dilatation of the capillaries, while others like dilute mineral acids, alkalies, chloroform, or sodium chloride and sugar in concentrated solution produce dilatation at once. Some poisons act variously on different parts, eucalyptol causing dilatation of the arteries and contraction of the veins, while corrosive sublimate causes contraction of the arteries and dilatation of the veins.

So with certain microbian toxins. Introduced into the general circulation they produce active congestion or inflammation in the seat of colonization of the microbe from which they were derived, as witnessed in the use of tuberculine or mallein. Finally the chill and febrile reaction which attends on extensive inflammation is essentially a nervous phenomena in its inception and progress.

Changes in the circulation. The usual changes in the bloodvessels of the inflamed part may be thus succinctly stated: 1. Contraction of the capillary vessels of the affected part and hastening of the current of blood through them. 2. The succeeding dilatation of the capillaries and the slowing of the blood stream, which still flows uniformly throughout the diseased tissue. 3. The flow of blood becomes irregular, at points tardy, and at others oscillating or even recoiling between the pulse beats when it has been forced into a vessel already blocked by coagulum. 4. In the still pervious vessels the red blood globules occupy the centre of the vessel where the current is rapid, while the white globules roll slowly along the inner surface of the walls where the current is slow and become adherent to the walls and stationary, while the general current rolls on. This is a direct abstraction of the white globules from the circulating blood and greatly favors the coagulation of the blood in the capillaries. The blood plates equally collect in the periphery of the vessel and escape. 5. The adherent white globules migrate in large numbers through the capillary and venous walls into the tissues. The red globules migrate to a less extent at first. 6. Small coagula form in the affected capillaries, forming minute red points which cannot be pressed out by the finger. 7. The red globules in the area of stagnation back of these capillary emboli adhere to each other by their flat surfaces and form rolls which pack into the vessel and are enveloped in a fibrinous clot. 8. The liquid part of the blood rapidly exudes into the tissues leaving the red globules relatively much more abundant in the liquid which remains inside the vessel. 9. The walls of the capillaries become softened and allow a readier transudation of liquor sanguinous, and escape of the globules through the walls of the vessels. 10. The arteries leading to the inflamed part have their muscular coats more rigid and unyielding and transmit much more blood than the corresponding artery leading to the healthy part. 11. The heart is equally

roused to more rapid and often more forcible contractions, which modify the pulse both in number and rythm. 12. The circulating blood is found to have received a great increase in the fibrine formers, the fibrine in the shed blood amounting to 6, 8, or 10 parts per 1000 in place of 3 parts as is normal. The contraction of this causes a depression on the surface of the clot. 13. The red globules become viscous and adhere together by their flat surfaces to form rolls, which precipitate much more rapidly than single globules and leave the coagulated blood with a straw-colored upper stratum (buffy coat). 14. Increase of waste products, urea, uric acid, hippuric acid, etc.

Other changes in the blood are alleged, like lessening of the albumen, as balancing the increase of fibrine, and lipæmia, but the constancy of these in all cases of inflammation is uncertain.

By way of comment and explanation of the above changes in the circulation the following may be advanced: The primary contraction of the capillaries is by no means a necessary condition of inflammation, and contractions and dilatations within certain limits occur in health and as a purely physiological act. The dilatation of the capillaries and the increased flow of blood to the part are related to each other as in part cause and effect, yet both are due to a reflex act from the seat of irritation which inhibits contraction in the capillaries and determines a more rigid contraction in the walls of the arteries running to the part. A rigid inelastic vessel of the same calibre and under the same pressure transmits more liquid than the one with elastic walls. The movement of the white globules to the walls of the vessel depends in part on their levity, light bodies passing into the outer slow moving layer, which is less dense, from the central stream where the force and density are greater. The epithelial cells of the intima undergo cloudy swelling and are often detached, allowing the readier migration of the globules through the openings of the lymphatics and the softened and friable walls. When the capillaries are blocked the pressure necessarily increases on the arterial side, favoring laceration of the friable walls and the escape of minute masses of blood. The formation of the buffy coat is characteristic of the normal equine blood; in inflammation it becomes more abundant. In the other genera a buffy coat apart from inflammation may be shown in: (a) anæmia or oligocythæmia in which

the blood is deficient in red globules; (b) in plethora in which there is an excess of blood solids; (c) in pregnancy in which there is an excess of white and small red globules; (d) in violent exertion or over-excitement, in which the blood has circulated with extraordinary rapidity. The all-sufficiency of the tissue cells in determining inflammation may be deduced from the following experiment. A ligature is tied around a frog's thigh so tightly as to arrest circulation, and the leg amputated above the ligature; mustard is then applied to the web of the foot and a blister rises precisely as though circulation continued.

MICROBES, DIAPEDESIS AND PHAGOCYTOSIS.

The rôle of **microbes** in inflammation is much greater than was formerly supposed. It is now demonstrated that a large class of inflammations are directly caused by the colonization of microbes in the tissue and by the local irritation caused by their ptomaines and toxins. We must also admit the direct action of the latter on the heat producing and vaso-motor nervous centres, as a factor more or less potent in different cases in the causation and maintenance of inflammation. No less important is the relation of the microbe to the migration of the globules and the subsequent results of the inflammation. This influence microbes share with certain chemical agents. Migration may be greatly checked even in inflamed parts by the hypodermic or intravenous injection of sulphate of quinia, eucalyptol, salicylic acid, or iodoform. Some have thought these acted by a chemiotactic attraction, but quinia is otherwise found to repel the leucocytes. Their action on the leucocytes or capillary walls is problematic.

Chemiotaxis is that power by which a microbe or any element attracts or repels the leucocytes. When it attracts the chemiotaxis is said to be positive, when it repels it is negative. Among negative chemiotactic agents are quinia, solutions of sodium chloride (10%), and potassium salts, lactic acid, alcohol (10%), chloroform, glycerine, jequirity, and bile. To some agents, (creatine, creatinine, allantoin, peptone, phlorydzine,) leucocytes are indifferent. To gluten, wheat casein, pea legumin and the great majority of pathogenic microbes, leucocytes are positively attracted. As microbes exercise a great influence in producing local in-

flammation, so they are important factors in procuring an abundant emigration of leucocytes. Some of the most fatal of microbian diseases, like fowl cholera, repel leucocytes, and the benefit of their defensive work is to a large extent lost. The toxins of the chemiotactic microbe filtered from the bacteria exert the same influence as the living bacteria, as shown by Gabritchevski, Massart and Bordet.

But chemiotaxis may be exerted from within the bloodvessel as well as from without. Bouchard, Massart and Bordet have shown that a tube containing a culture of bacillus pyocyanus, introduced beneath the skin of a rabbit attracts in a few hours a great number of leucocytes. But if, immediately after its introduction, ten cubic centimetres of a sterilized culture of the same bacillus are injected into a vein, very few leucocytes enter the tube inserted under the skin. The chemiotaxis seems to operate in this case from within the blood, and the desires of the leucocytes are satisfied without leaving the vessel. It would seem that in such cases the migration and protective work of the leucocytes is best exerted at the outset of the illness and before the toxic products have been poured into the blood in any quantity, whereas in the advanced stages when the blood is charged with ptomaines and toxins migration and phagocytosis would be likely to be limited and ineffective. The same consideration would forbid the use of drugs that check migration in all cases of attacks by microbes for which leucocytes have a positive chemiotaxis.

Phagocytosis is the act by which the leucocytes englobe and dissolve the invading microbe. By its amœboid movement the leucocyte flows around, and envelopes the microbe for which it has a positive chemiotaxis, and then begins the struggle of vitality between the two living germs. If the poison (leucomaine antitoxin,) and digestive ferment (enzyme) of the leucocytes are more deadly to the invading germ, than its ptomaines, toxins and enzymes are to the leucocyte, the white cell comes off the victor, and recovery takes place, but if the converse obtains the triumph is on the side of the microbe. As a rule much depends on the more or less deadly nature of the products of the invading microbe, on the numbers of the germ, the rapidity of its proliferation, and the consequent amount of its toxic products thrown into the system, on the one hand: And on the other the potency of

the chemiotaxis of the leucocyte for the invading germ, the number of white cells that emigrate into the inflamed tissue and engage in the work of phagocytosis, and on whether the particular animal system and its white cells have sustained a previous attack by the same germ and has thereby been educated to produce a greater amount of the defensive proteids (leucomaine, antitoxin, enzyme) than it naturally would (acquired immunity).

Even with an abundant emigration of the leucocytes into the inflamed or invaded tissue, a number, greater or less, are usually destroyed by the bacterial poisons and pass into degeneration or liquefaction, as in the formation of pus, and yet the attacking germ may be overcome, destroyed and devoured by the rapidly increasing survivors. In general terms the migration of the cells is in inverse ratio to the susceptibility of the animal to the microbe or the disease which it causes.

The positive and negative chemiotaxis, which determine phagocytosis or prevent it, may be seen in the action of the leucocytes toward the germs of two diseases, to one of which the animal is susceptible and to the other of which it is not. Thus the leucocytes of the pigeon take in the bacillus anthracis and suffer nothing apparently, whereas the same white cells of the dove are repelled by the bacteria of fowl cholera which are not therefore found in their interior.

The leucocytes that migrate from the bloodvessels are in the main, the most numerous, (the neutrophile or polynuclear) form; the mononuclear leucocytes with horseshoe shaped nucleus also migrate but in much fewer numbers and are as a rule less occupied in phagocytosis. At the same time, these two forms may show each a preference for a particular microbe, the polynuclear cell sometimes devouring one which the mononuclear cell rejects, and the mononuclear cell taking in one which the polynuclear refuses.

The small round white cells (lymphocytes) and the eosinophile leucocytes take no prominent part in phagocytosis.

EXUDATION.

In inflamed vascular tissues one of the most important results is the exudation. This is not, however, a mere transudation of

the liquid parts of the blood, as takes place in dropsy, but it is to a large extent a selective process determined apparently by the condition of the capillary walls, and the nature of the inflammation is stated according to the character of the exudate. The dropsical effusion contains little albumen, fibrine or cell forms, and does not coagulate. The inflammation exudate contains abundance of fibrine, cells and other solids and coagulates spontaneously in contact with inflamed tissue, or when removed from the body, by reason of the transforming leucocytes. Inflammatory exudate usually contains 6 to 8 per cent. of solids whereas the normal canine lymph contains 4 to 6. The exudate varies not only in different inflammations, but in successive stages of the same inflammation. The exudate may be mucous, serous, fibrinous or hæmorrhagic.

Mucous Exudate. In inflammation on a mucous or synovial surface the inflammatory exudation, mingled with the more or less altered secretion of the mucous glands, and the epithelial cells and leucocytes forms a viscid fluid, rich in mucin, and characterizing the *mucous* or *catarrhal inflammation*. The nature of the discharge varies greatly, the serous character predominating at the start of the inflammation, and a thick, opaque creamy or semi-solid muco-purulent material appearing as the disease advances. It contains filaments of precipitated mucin insoluble in acetic acid or alcohol and cells in all stages of change from the exudation leucocyte and mucous cell to the pus corpuscle, the latter being characterized by its bipartite or tripartite nucleus rendered visible by contact with weak acetic acid.

Serous Exudate. This consists of the liquid elements of the blood with only a limited amount of fibrine formers and consequently little tendency to clot firmly. The presence of fibrinogen however serves to distinguish it from the liquid of mechanical dropsy, as does also the greater quantity of cells and nuclei of common salt and phosphates. It is usually straw colored in mass, but is sometimes slightly opalescent by reason of the numbers of cells and floating filaments of fibrine. Serous exudations take place in the early stages of inflammations (as in catarrh) and in inflammations of serous membranes (pleura, peritoneum, joints), in strong, vigorous subjects. They constitute the liquid contents of blisters whether raised by medicinal irritants, chafing,

or heat. They clot under heat and nitric acid with a firmness proportionate to the amount of albumen.

These effusions are dangerous by reason of their interference with the functions of organs by pressure as with the dilatation of the lungs, the movements of the heart, the action of joints, or the integrity of the brain or spinal cord. When the causative disease has subsided they are usually speedily reabsorbed, the cells passing into the lymph vessels, or becoming degenerated, liquefied, and absorbed. Yet serous effusions often remain as permanent accumulations. For the blood staining of serous effusions and their clearing up, see under pleurisy.

Fibrinous Exudate. This is characterized by the amount of fibrinogen and fibro-plastin in its composition and by the comparative absence of leucocytes. It oozes through the vessels and coagulates in the tissues or on the surface of inflamed serous or mucous membranes. The more liquid part separating from the coagulum escapes from the free surface or accumulates in the lower part of the serous cavity. The coagulation is doubtless caused by the fibrine ferment derived from the rapidly proliferating cells and degenerating leucocytes. It usually occurs promptly in or on an inflamed tissue, but in contact with healthy structures only (as in a serous sac) it may remain fluid for an indefinite length of time. This exudate constitutes the false membranes that form on the pleura, pericardium or arachnoid, the coagulum of fibrinous pneumonia, and the plastic lymph on the surface of a granulating wound. It is especially injurious by reason of its enveloping organs (lungs, heart, bowels, iris) and subjecting to permanent compression by reason of its contracting, also by binding them to adjacent structures by false membranes. In coagulating it becomes first fibrillar then granular and finally undergoes molecular degeneration (Cornil and Remvier), or development into new tissue(Paget). When organized it usually takes the form of the adjacent tissue from which its trophic cells are derived. Thus in divided tendons, in serous membranes and in granulating wounds it is fibrous, and between the ends of a broken bone it is osseous. If however, the adjoining tissue is a highly organized one, like nerve or muscle it may be replaced by a simpler (fibrous, osseous).

Fibrinous inflammations are especially found in connection with inflamed fibrous tissues and in strong vigorous subjects.

Blood Exudations. In all inflammations there is some migration of blood globules (red as well as white) but seldom in quantity sufficient to stain the tissues materially. Minute ruptures of the capillary vessels are not uncommon, with punctiform clots in the tissues, but extensive escape of blood is mainly seen in penetrating or contused wounds of the loose, subcutaneous connective tissue, and in infective inflammations (anthrax, Rinderpest, swine plague, petechial fever, malignant catarrh, snakebites) with destruction of blood globules or extreme changes in the walls of the capillaries. Newly formed vessels in friable neoplasm are subject to blood effusions. In acute inflammations of serous membranes the exudate is usually of a dark port wine hue at first. In such cases it may pass in succession through all the stages of dark red, brick red, yellow, reddish, and chocolate color, before becoming milky and finally transparent.

Croupous Exudate. Croupous inflammation usually occurs on or near a mucous surface and is characterized by an exudation consisting mainly of fibrinous material entangling white cells, epithelium, a few pus corpuscles and some form of bacteria. In true diphtheria of children this is the Löffler bacillus, in the pseudodiphtheria, attending on scarlatina, etc., it is streptococcus pyogenus, in the diphtheria of calves it is bacillus diphtheriæ vitulorum, and in that of chickens and pigeons it is the bacillus diphtheriæ columbarum (Löffler). Pseudomembranous inflammations therefore constitute a group agreeing in the nature of the exudate but differing essentially in the cause. This difference in the cause has a most material effect on the course and gravity of the disease. One form like true diphtheria in man not only extends into the tissues, and tends to necrotic changes, but also poisons the nerve centres by the toxic materials absorbed inducing troublesome paralysis, while another like croup of children establishes a violent but essentially superficial disease and when that recovers it leaves no ulterior ill effects elsewhere.

A **Chyliform exudate** has been noted in peritonitis in the dog the milky whiteness being due to fatty granules.

RESULTS AND PRODUCTS OF INFLAMMATION.

As nearly all inflammations have significant exudations it is well to follow these in their subsequent progress through reab-

sorption and removal, development into new tissues, necrosis, suppuration and ulceration.

Resolution. If an inflammation, slight in character and with only a moderate exudation, subsides and is followed by a rapid liquefaction of the cells and fibrinous coagula and a reabsorption of the exudate, so as to leave the part in its primary healthy condition structurally and functionally, it is said to have terminated by "*resolution.*" If this occurs with extraordinary rapidity it is said to have ended by "*delitescence.*" This is not always an unalloyed good, as often in delitescence, coagula and infecting material may be carried on by the circulation, to block the next set of capillaries in its course and set up new centres of inflammation. This is one form of "*metastasis*" though a more definite *metastasis* is in rheumatism where the disease attacks one joint to-day and a distant one to-morrow.

Inflammatory New Formations. Of the growths in lymph there are two principal kinds: first, *the plastic, fibrinous, granular* or *molecular;* and second, *the aplastic* or *corpuscular.* The first form tends to develop into new structure, the second to disintegrate and decay. The tendency to one or other form depends largely on the strength or weakness of the system's health, on the deficiency or excess of corpuscles in the exuded fluid, and on the distance of the latter from living tissues and blood supply. Much also depends on the predisposition of the genus, the tendency to suppuration in lymph being in a descending series from horse, ass, and mule, through ox and sheep, to dog, pig, and finally, the bird, in which latter suppuration is quite exceptional.

Suppuration. In inflammations of a high type, in those occurring on the skin or mucous membranes in which there is an extraordinary increase of nuclei and embryonal cells, and in lymph thrown out in excess at one point, so that its central parts are far from vascular tissue and nourishment, the cell elements undergo a rapid increase and degradation into pus-corpuscles, and its solidified intercellular lymph undergoes granular decay and liquefaction into pus

While the above conditions are favorable to the formation of pus, the process of suppuration must now be recognized as an infective process due to the propogation of bacteria (mainly chain forms—*Streptococcus pyogenes*—cluster groups—*Staphylococcus py-*

ogenes—and rod forms—*Bacillus pyogenes*). These or other bacteria are found in the pus of acute abscesses, and when absent in chronic abscesses are to be considered as having perished since the abscess was recent and active. Inoculation of a rabbit with an excess of the pus of an acute abscess produces general purulent infection (pyæmia) and early death; from a medium dose an abscess is produced; while from a small dose there is no effect whatever. In the latter case the bacteria are overcome and devoured by the abundance of vitally potent white blood-globules and tissue cells. This pus-forming action of these bacteria explains the great difference in results in wounds exposed to the air and those in the interior of the body and far removed from air and its floating bacteria. A broken bone, with no wound in the skin and little injury to parts around the fracture, is readily repaired without any formation of pus, if merely kept still and immovable; whereas a broken bone, continuous with a wound through the skin, always tends to form pus or become otherwise infected, and is extremely dangerous even to life. The tendency of every open sore is to form pus on its surface but this may be arrested and avoided by preventing the access of germs, or by a free use of disinfectants and a covering which shall arrest and filter out the germs. Similarly in an abscess, evacuation followed by the injection of disinfectants, without the formation of any perceptible permanent opening to the outer air, will put a stop to the pus-formation. The subjection of an inflamed part to the control of these pus-forming bacteria is dependent on the lowered vitality and power of resistance of the inflamed tissues, and of the white cells of their circulating blood. Healthy parts can successfully resist them, though they are constantly present in surrounding air and on objects, but in this as in all other cases, of bacterial infection, so soon as the tissue is injured, inflamed and lowered in its power of vital resistance, the pyogenic bacteria assail it successfully. Hence, too, the more abundant exudations of lymph, the centres of which are farthest removed from the healthy tissues and from nourishment, are the most prone to suppuration. That the germs can make their way to such deep-seated exudations in the substance of solid tissues is to be accounted for by their gradual advance through the inflamed and weakened structures from the adjacent skin or mucous membrane, or in some instances by

reason of their presence in small numbers in the blood. It is further noteworthy that those animals in which suppuration does not occur readily are such as have a special power of resistance to some other organic poisons. Thus the hog, which is supposed to be proof against snake-bite, is also, to a large extent, proof against the pus-forming bacteria.

Pus. This is a white, or yellowish-white, creamy-looking product, composed of a clear, transparent fluid, rendered opaque by numerous floating pus-corpuscles. These pus-corpuscles have the same size as the white globules of the blood ($\frac{1}{2500}$ to $\frac{1}{3000}$ inch) and are peculiar in that each shows within it three or more nuclei, which become visible on the addition of a drop of water or acetic acid. Each of the common embryonal cells found in the inflamed tissue usually contains two nuclei, the indication of the active increase by division into two, but when the supply of nutriment is checked the nuclei continue to divide, while the cells remain unchanged, and thus every cell comes to contain several nuclei in addition to fatty granules, and constitute pus-corpuscles.

When pus is formed in a well-maintained system and tissue, the outer layer of the lymph is developed into a fibrous sac inclosing the liquid pus and constituting an *abscess*. In an unhealthy system, or when the inflammation depends on some injurious poison, like that of erysipelas, this sac may not be formed, and the pus, burrowing into and between different organs, destroys the connections and substance—*diffuse suppuration*. When an abscess has formed in soft tissues its investing sac shrinks as it assumes the fibrous character, and the confined pus being incapable of compression, presses the membrane outward on the side in which the surrounding tissues are most loose and least resistant, hence, usually, though not always, in the direction of the skin; the soft tissues become absorbed and removed in the track of the advancing pus; and, finally, the latter reaches a free surface and escapes. Thus, an abscess usually bursts through the skin, but also, at times, through a mucous membrane into the lungs, bowels, etc., or through a serous membrane into chest, abdomen, etc. When an abscess is formed in bone or dense fibrous tissues which press equally on all sides, it may remain imprisoned for months and years after all inflamma-

tion has subsided, constituting an *indolent* or *cold abscess*. When the imprisoned pus is inclosed by thick fibrous or resistant tissues at all points but one, it will make its way along the narrow passage of yielding tissue, but as the resulting outlet is constricted, long, and tortuous, the contents cannot readily escape through it nor the walls of the abscess contract so as to expel the confined pus, and the latter goes on forming and discharging through the narrow outlet for months or years. This is a *fistula* or *sinus*.

Healing by Adhesion or First Intention. When a clean-cut wound has the blood staunched and its lips brought together without exposure to the air (or contact with pyogenic germs), they adhere at once and heal without pus or almost any appreciable formation of new tissue. Here the lymph thrown out on the cut surfaces agglutinates them, and the cells, multiplying, form a thin layer of embryonic tissue which gradually develops into a fibrous structure and repairs the breach without any perceptible scar.

Healing by Second Intention. Granulation. When a wound has caused destruction of tissue, or when a simple incision is left exposed to the air, the breach is filled up by new tissue through the process known as granulation. The superficial layer of lymph thrown out on the raw surface becomes oxidized and degenerates into pus, while the deeper layers become solid, fibrillated, the seat of cell-growth, and are finally transformed into a fibrous structure. New blood-vessels form in loops in the developing lymph and constitute the bright-red granulation-points which cover the raw surface. The fibrous tissue into which the lymph is transformed undergoes gradual contraction in development, and thus, day by day, the edges of the adjacent healthy skin are drawn in, so as to cover the wound more or less perfectly, and a slight scar only is left when healing has been accomplished.

Granule Corpuscles and Masses. This is another degenerative transformation in lymph and, is seen mainly in inflamed glands and brain and lung-tissue. The cells found in the exuded lymph are made up of granules $\frac{1}{10000}$ inch in diameter, and besides these, large, irregularly shaped masses of granules are extended along the capillary blood-vessels. After the lymph has coagulated these granular masses soften and liquefy prelimi-

nary to re-absorption and removal, and the restoration of the tissue to a healthy condition. When in excess this softens and disintegrates the tissues, leading to permanent loss of substance. See *granular* degeneration.

Interstitial Development of Lymph into Tissue. This is equivalent to what takes place in the formation of the sac of the abscess or of granulation-tissue. The liquid lymph in coagulating, becomes fibrillar, and the cells and nuclei of the adjacent tissue, having an abundant supply of blood and nutriment, multiply first as simple, rounded embryonic cells, then deposit around them new tissue, becoming elongated, spindle-shaped, branching, etc., and thus get imbedded in a fibrous material of their own formation. These new formations are usually of a low type of organization, like white fibrous tissue or bone, and hence, although breaches in the higher structures like muscle, nerve, gland, skin, are filled up, it is usually only by the drawing together of the remaining healthy parts by these new formations without the restoration of any of the original tissue which has been destroyed. The cicatrix (scar), alone is made up of new material.

Lymph developing in this way may undergo any degeneration to which normal tissues are subject. Thus it may undergo black pigmentary (*melanotic*) degeneration, it may become impregnated with lime-salts (*calcified*), it may wither up into a hard *gelatiniform* or *horny* mass, or it may undergo *fatty* degeneration.

Fatty degeneration is the most common form, and consists in the excessive deposit of fatty granules, first in the cells which are in excess or badly nourished, and next in the adjacent tissue, the normal elements of which are replaced by fatty granules.

Softening is an almost constant result of inflammation. The exudate infiltrates and separates the tissue elements, destroying their cohesion; the liquefaction of these elements impairs this still further, and the more or less perfect transformation of the tissue into embryonic tissue entails the loss of its rigidity and power of resistance. Thus the inflamed brain-tissue may become a mere pulp, and the inflamed bone may be cut with a knife.

Ulceration is closely allied to softening. On the surface of a sore there is an excessive exudation of lymph, which loosens and disintegrates the layer of lymph that is already in process of development, and also a part of the tissue beneath. The cells in

these parts fail to develop naturally and to build up good tissue; they become fatty, die, and together with the tissue in which they lie, break down and pass off as a pulpy débris. Thus the sore constantly deepens and widens, or at least refuses to contract and heal. It is usually the result of bacterial infection.

Gangrene or **death of a part** is another effect of inflammation. It results usually from the cutting off of the blood-supply through the obstruction of the blood-vessels; by the pressure of excessive exudation in unyielding structures, as in bone, or under the hoof; by implication of the inner coats of the blood-vessels in the inflammation, when the contained blood will clot and obstruct them; or by blocking with the blood-clots that have been formed at a distance and washed on in the blood-current to be arrested when they reach vessels too small to admit them. Like suppuration, gangrene is associated with and often caused by a bacterial growth. The dead mass remains as an irritant, and is slowly separated by the formation around it of embryonal tissue, granulations and pus. A second form is *molecular gangrene*, in which the cells and minute elements of the tissue die, and are cast off, leading to phagedenic (eating, extending) sores, as noted above under Ulceration. When gangrene occurs on an exposed surface, that may be altered from the normal color into shades of yellow, brown, green, red, or black, according to the amount of blood and the stage of decomposition, and may be cut without pain, if the subjacent parts are not pressed upon; it may be soft, may pit on pressure, may crackle under the hand from the evolved gases of decomposition, and may be covered with blisters (*phlyctenæ*) with red, grumous liquid contents (*moist gangrene*); again, it may be white, as after freezing, or it may be dark-colored, dry, and horny, as from ergotism (*dry gangrene*).

FEVER.

Definition. Symptomatic. Idiopathic. Symptoms. Contagion. Incubation. Premonitory symptoms. Chill, rigor. Reaction, hot stage. Defervescence. Crisis. Lysis. Natural temperature. Fever temperature. Retention of water in the system. Production of waste materials. Typhoid condition. High fever, low, hectic. Treatment in vigorous subject, in weak one. Regimen. Solipedes. Ruminants. Carnivora. Drink. Rest. Clothing. Air. General and local bleeding. Cupping. Warm baths, tepid, compresses, derivatives. Cold. Diaphoretics. Laxatives. Diuretics. Sedatives. Alkalies. Antipyretics. Stimulants. Tonic refrigerants. Tonics. In low fever. No depletion. Judicious elimination. Stimulants. Refrigerants. Antiseptics. Diet. Local treatment of inflammation. Cold. Astringents. Antiseptics. Warm applications. Stimulating embrocations. Blisters. Firing. Massage. Suppuration.

Definition. Whether occurring as an accompaniment of inflammation or independently of it, fever is an unnatural elevation of the temperature of the body, the direct result of an excess of destructive chemical change in the blood and tissues, and more remotely of disordered nervous function.

Of all extensive inflammations fever is the constant result and accompaniment, rising as the inflammation rises or extends, and subsiding as the inflammation subsides. It also occurs as a distinct affection, as in all the infectious diseases, as the result of a specific irritating poison in the system, and then is the manifestation of the disease, while a local inflammation may or may not be present as a special secondary feature of the malady or as an accidental complication.

Symptoms of Fever. Fever is marked by certain definite stages, each of which has its own special manifestations. In the cases due to a specific disease-germ, or *contagium*, these are, however, preceded by a period of *latency* or *incubation* in which no symptoms whatever are manifest, but during this time the germ is rapidly multiplying in the system, and it is only when it has gained a certain increase that it disorders the nervous system, wastes the tissues, raises the temperature of the body, and induces the other phenomena of fever. The same may be said to hold in the fever attending on inflammation. The slight and circum-

scribed inflammation is at first productive of no fever, and it is only when it gains a certain extent that the nerves and nutrition are disordered so as to bring about a feverish condition.

Premonitory Symptoms. These usually last but a few hours and are often entirely absent or unnoticed. There is a lack of the customary vigor and spirit, an indisposition to exertion, a loss of clearness and vivacity of the eye, a manifest dullness, with hanging of the head, and frequent shifting of the limbs as if fatigued. Appetite is less sharp and ruminants chew the cud less heartily or persistently.

Cold Stage. These are soon succeeded by the *chill*, *rigor*, or *shivering fit*, in which the hair, especially that along the back, stands erect (staring coat), the skin is cold and adherent to the structures beneath (hidebound), the extremities (legs, tail, ears, horns, nose) are cold, and the frame is agitated with slight tremors, or even a shivering so violent that a wooden floor or building is made to rattle. The back is arched, the legs brought nearer together (crouching), the mouth is cool and clammy, the breathing hurried, the pulse weak, and it may be rapid, but with a hard beat, the bowels costive, and the urine higher colored than natural. The temperature of the interior of the body, taken by a thermometer in the rectum, is already found above the normal, the excessive destruction of tissue having begun, and the blood driven from the cooler surface, and accumulating in the hot interior, at once favors tissue-change and maintains the extra heat thereby produced. In cattle the end of the tail is soft and flaccid from this stage onward. The *cold stage* lasts a few minutes, or one or two days in different cases.

Hot Stage. The hot stage appears as a reaction from the chill, the contraction in the minute vessels of the skin giving place to dilatation, so that the whole surface, including the extremities, becomes hot and burning, but still dry and parched. The burning is especially noticeable in the more vascular parts, like the roots of the horns and ears, the muzzle or snout, the mouth, the hoofs, the bare parts of the paws in carnivora, and the mammæ (udder) in suckling animals. The mucous membranes lining the nose and mouth become hot and red, the breathing freer, but not less rapid, the pulse softer but accelerated, appetite (and rumination) greatly impaired or lost, thirst great, costiveness increased,

urine diminished and of a higher color, the flow of milk greatly impaired or entirely arrested, and the dullness and prostration greatly increased.

The hot stage lasts longer than the cold one, usually persisting until death or convalescence. It may alternate with chills throughout the whole course of the illness, and in the fever of inflammation the interruption of the *hot stage* by a chill usually implies either a considerable extension of the inflammation or the occurence of suppuration.

Defervescence. The decline of the fever may take place by a sudden reduction of the body temperature to the natural standard, or near it, and a sudden and general improvement in the symptoms (crisis), or by a slow improvement from day to day through a more or less tedious convalescence (lysis).

Fever Temperature. A temporary rise of one or two degrees is unimportant, but a permanent rise indicates fever. A rise of ten or twelve degrees is usually fatal. A sudden fall to or below the natural, unless with general improvement in the symptoms indicates *sinking*. A similar fall, with a free secretion (perspiration, urination, relaxed bowels) and general improvement in symptoms, betokens recovery. For normal and febrile temperature see Semeiology.

Retention of water in the fevered system is as significant as the elevated temperature. The patient drinks greedily but all the secretions are arrested or diminished, and liquids go on accumulating in the system. The sudden bursting forth of secretions (especially sweating) implies that the fever has, at least temporarily, given way.

The production of waste matters in the system is necessarily proportionate to the amount of tissue destroyed. This appears in the blood mainly as urea, the organic acid of urine (hippuric in herbivora, uric in carnivora), together with phosphates, sulphates, and chlorides. These thrown off by the urine give it its high density. If not thus thrown off they remain as poisons in the circulation and bring about that prostrate, sunken, debilitated condition which characterizes the advanced stages of all severe and continued fevers—*the typhoid condition*. This is not to be confounded with the specific *typhoid fever,* in which a special fever germ expends itself, mainly on the bowels, and that runs through

a regular course. The *typhoid condition* is that state in which an animal system, already greatly weakened by a severe disease, and perhaps further prostrated by a specific disease-poison, is subjected to a species of poisoning by the retained chemical products of the waste of the tissues.

Types of Fever. These are as characteristic as the types of inflammation, and of the same kind. The *strong* type of fever which attends on an acute inflammation in an otherwise healthy vigorous system, is spoken of as a *high* or *inflammatory* fever. The *weak* type which occurs in a broken down or debilitated system, or in connection with the action of a specific disease germ, or with the saturation of the system by waste chemical products is known as *low*, *typhoid* (better *typhous*), or *adynamic* fever. That form which persists in the utterly debilitated system, where the power of assimulation is practically lost, is known as *hectic*.

TREATMENT OF INFLAMMATION AND FEVER.

Treatment will be guided very largely by the type of the attendant fever. If that is of a high type, with a hard, full, rapid pulse, bright red mucous membranes, a clear eye, and well sustained strength in a strong, vigorous animal, what is known as antiphlogistic (depleting, depressing) treatment is admissible at the outset. But in many cases with a low type of fever, a weak, rapid pulse, pallid, yellow, or livid mucous membranes, a coated tongue, a dull or sunken eye, much depression and prostration, swaying on the limbs in walking, pendant head, ears, eyelids and lips, and varying and irregular temperature of the limbs, etc., such measures are forbidden from the first, and tonics and stimulants are demanded from the outset. Between the two extremes there are many grades, which demand a judiciously adjusted intermediate treatment. The general principles only of each characteristic form of treatment can be here formulated, it being understood that no two cases can be most advantageously treated in precisely the same way, but that according to its special grade each case will demand its own specific management applied according to the skill of the physician.

Regimen. An antiphlogistic diet will consist in a moderate or very sparing amount of non-stimulating food of easy digestion

(wheat bran or oil meal in warm, sloppy mash, carrots, turnips, beets, potatoes, apples, pumpkins; fresh, tender, green grass or in winter a little scalded hay, may be taken as examples). *Ruminants* should have no food necessitating chewing of the cud; thus the roots, etc., should be pulped or boiled, and hay and even grass must be interdicted until rumination is re-established. When food is absolutely refused for days in succession well-boiled gruels of oat-meal, barley-meal, linseed-meal, bran, etc., may be given from a bottle or by injection. *Dogs* and *cats* should have only vegetable mush (unbolted flour, barley, or oat-meal) with just enough beef-juice to tempt the animal to eat a little. Milk with an admixture of oxide of magnesia, or even lime-water is often at once palatable and cooling. *Drink* should be pure water, cool, if kept constantly fresh before the animal, but warmed to something less than tepid if supplied only at long intervals, so that the thirsty patient is not tempted to drink to excess and chill himself. *Rest* in a clean, well-aired building, free from draughts of cold air and with a southern exposure, is desirable, especially in winter. The best temperature is usually sixty degrees to seventy degrees, especially in inflammations in the chest, and extremes of temperature are to be avoided. *Clothing* will depend on the weather. In warm weather it may be often discarded, while in winter it should always be sufficient to obviate the access of *chill* and consequent aggravation of the disease. Whenever the atmosphere can be kept warm only at the expense of impurity it is better to secure the comfort of the patient by the requisite clothing than to subject him to impure air. As the extremities are the first to suffer from cold, loose flannel bandages to the limbs are often imperative.

Remedies. General bleeding, a great resort of our fore-fathers, has been long all but discarded from modern practice. To-day it is rarely resorted to, except to save from an urgent and extreme danger, as in the plethoric cow merging into parturient apoplexy, or the fat and overdriven horse, gasping for breath and life, in general acute congestion of the lungs. There are other cases of extensive acute and dangerous congestions, especially in a strong, vigorous, and plethoric patient, in which general bleeding in beneficial in warding off threatened death; but sound, discriminating judgment is necessary to its safe employment. When resorted to

at all, the blood should be drawn from a large orifice, in a full stream, to secure the desired depressant effect with the smallest loss of blood, and the patient should be kept especially quiet and apart from all excitement which would tend to counteract the sedative action.

Local bleeding is more extensively applicable than *general*, as it usually effects the same purpose without the permanently weakening effect. It acts in two ways, first, by emptying and contracting the vessels in the skin over the inflamed organ, it solicits a sympathetic contraction of the capillary vessels in that organ itself, and thus inaugurates a progress toward recovery; and second, by so much as it draws blood to the surface it diminishes the blood-pressure on the deeper inflamed organ, and affords a better opportunity for the restoration of the healthy circulation and function. Local bleeding may be practiced by simple scarification or leeches, or better, by cupping with or without scarification. To apply leeches, the skin must first be shaved. To cup, it must at least be greased. As a cup, an ordinary large drinking-glass may be used, the air contained in it being driven out by a lighted taper, and then the taper being withdrawn, the mouth of the cup is instantly and accurately applied on the skin and held there, until, as it cools, it draws up the skin within it and clings like a sucker. A number of these may be applied according to the extent of the inflammation, and, if desired, they may be removed, the part scarified, and the cup reapplied. The cupping usually effects more than a mere local attraction of blood; it very commonly causes a free circulation in the whole skin, a generally diffused warmth, and even perspiration. Thus we may secure the derivation of blood from the inflamed part, the cooling of a large mass of blood in the extensive cutaneous circulation, the cooling of the entire system by the return of this blood internally, the elimination of injurious waste matters through the skin, the lowering of the febrile heat and tension, and a better functional activity of all the organs of the body.

Similar good results are obtained from all remedies that induce surface warmth and vascularity and a free secretion from the skin.

Warm baths, for animals to which they can be applied, abstract blood temporarily from the inflamed internal organs, diminish the blood-pressure, and really cool the system, beside securing elimi-

nation from the skin and other secreting surfaces. They may be commenced warm (80° F.) and gradually cooled down to 65° F. after the skin has become freely active. In the larger quadrupeds, in which the warm bath is too often practically impossible, the same revulsion of blood and warmth to the skin may be secured by *rags wrung out of hot (almost scalding) water*, wrapped tightly round the body, covered with two or more dry blankets, and kept tightly applied against the surface by elastic circingles. The legs may be rubbed with straw wisps till warm, and then loosely bandaged, or applications of red pepper, ammonia, or mustard, may be made prior to bandaging. In place of hot water rugs, bags loosely filled with bran, chaff, or other light agent, heated to 110° F., may be applied round the body, or, where it is available, a Turkish or steam bath may be resorted to. These hot cutaneous applications, to produce glow and perspiration, are especially valuable in the chill that heralds a violent inflammation, and if that can be suddenly checked by this means the inflammation will often be warded off, or at least rendered slight and easily controllable. After perspiring for half an hour the patient may be gradually uncovered, rubbed dry, and covered with a dry, warm blanket. If the skin is still glowing, a slight sponging with cool or cold water may beneficially precede the rubbing and drying.

Cold Baths. In cases of very high fever a full cold bath (68° F.) may be employed for fifteen minutes, and repeated as often as the temperature rises. In many cases of parturition fever in cows great benefit accrues from sponging the body with cold water and allowing it to evaporate from the burning skin. In the extreme fever of heat apoplexy (sunstroke), with a temperature of 110° F. and upward, a strong current of cold water from a hose directed on the head and body often gives the best results. In ordinary fevers in large animals the *cold pack* will often serve a good purpose. Wring a blanket out of water (cold or tepid, according to the height of the fever and the strength and power of reaction of the patient), wrap it round the body, cover it with several dry blankets so that no part is exposed, and keep the whole in close contact with the skin by elastic circingles. In fifteen minutes the skin should be glowing and perspiring, and in half an hour the wrappings should be removed, a little at a

time, the parts rubbed dry and covered with a dry woolen blanket. It may be repeated as often as the fever rises.

Diaphoretics. Besides these remedial methods of inducing a revulsion and glow in the skin with perspiration, *medicinal diaphoretics* may be resorted to. Among these may be included *copious drinks and injections of warm water, acetate of ammonia, antimony, ipecacuan,* or *pilocarpin,* or one of the sedatives, *aconite veratum,* or *opium,* etc. Many a threatened acute inflammation has been to a great extent cut short and nipped in the bud—the stage of chill—by warm clothing, active hand-rubbing, and such an apparently unscientific nauseant as tobacco.

When the preliminary stage has passed and the hot stage of the fever has set in, cooling and eliminating agents are especially called for.

Laxatives. In many cases, and especially in those with marked constipation or bowels loaded with indigestible materials, a laxative is beneficial. For the horse, aloes, or, often better, sulphate of soda, and for cattle or sheep, the latter, or Epsom salts, will at once remove an irritant, cool the general system, draw off much blood and nervous energy to the bowels, and secure a considerable depletion and elimination from the intestines. For swine, dogs, and cats castor-oil or salts may be used, and for fowls castor-oil. If the mucous membranes are yellow, the tongue furred, and fæces scanty, hard, and fœtid, a dose of calomel (horse or ox, one drachm; sheep or pig, one scruple; dog, three grains; chicken, one-half grain) with tartar emetic (horse or ox, two drachms; sheep, twenty grains; swine, one-half grain; dog, one-fourth grain; chicken, one-eighth grain) may be given and followed in ten hours by one of the laxatives named above.

Diuretics. In the absence of any manifest disorder of the digestive organs, the laxative may be omitted and refrigerant diuretics resorted to. Acetate of ammonia or potassa, nitre, tartrate of potassa, carbonates of potassa or soda, may be used along with sedatives.

In cases of infectious disease with poisoning by ptomaines and toxins the elimination of these by the bowels and kidneys is of the greatest importance.

Sedatives. Of the sedatives, aconite, bromide of potasium, veratrum, hyoscyamus, or chloral hydrate may be used according to the special indications.

Alkalies. Resolvents. When the organ inflamed is a serous membrane in which dangerous adhesions or other functional disorders are likely to occur from newly formed false membranes, their formation should be counteracted as far as possible by the free use of alkalies (carbonates of soda, potash, or ammonia, nitre, iodide of potassium, muriate of ammonia, etc.), and in the same conditions excessive effusion should be controlled by free action on the kidneys.

Antipyretics. To reduce the febrile temperature and especially, when caused by the ptomaines and toxins of bacterial infection, agents like acetanilid, antipyrin, exalgin, analgene, benzanilid, salicylate of soda, and quinine have been largely employed and will usually lower the temperature several degrees in a few hours. They nearly all depress the vital forces, or hinder reparatory processes, so that their use is to be carefully guarded. Quinine which is less depressing than the others hinders migration of the leucocytes and thus stands in the way of successful phagocytosis. With a dangerously high temperature they may be temporarily admissible, but they should be suspended as soon as possible. In all ordinary cases they are probably better avoided. A judicious use of the cold or tepid bath, or of wet compresses is incomparably safer and more generally applicable.

Stimulants. When the disease results in great prostration or when symptoms of septic or ptomaine poisoning set in stimulants are often required to sustain the flagging heart and circulation. These may be alcoholic, ammoniacal, etherial, camphor, digitalis, etc.

Tonic Refrigerants. Later, when both inflammation and fever have been somewhat reduced, temperature, breathing, and pulse rendered more moderate, eye clearer, and even appetite perhaps slightly improved, the sedatives may give place to refrigerating tonics, such as mineral acids (nitric, muriatic, sulphuric, or phosphoric), in combination with bitters (quassia, cascarilla, calumba, gentian, salicin), without as yet the suspension of refrigerant diuretics. Thus for the horse the following: Recipe: Pharmaceutical nitric acid, two drams; infusion of gentian, ten ounces; nitrate of potassa, two ounces. Dissolve. Give one ounce every six hours.

In Convalescence. When convalescence has fairly set in, the

fever has subsided, and there remains merely some debility with a remnant of the inflammatory exudation to be removed or organized into tissue, or when an abscess has developed and burst, the tonics must be even more freely given, the mineral acids may even give place to preparations of iron or cod-liver oil, and the diet must be made increasingly liberal. But throughout the whole progress of the disease the bowels should be carefully watched. Costiveness may quickly undo all that has been gained, hence any indication of this should be met by laxative food (boiled flaxseed, etc.), or, this failing, by injections or laxatives. Similarly, if a freer action of the kidneys seems to be necessary for elimination of waste matters or to reduce fever, diuretics should be continuously kept up.

Treatment of Adynamic Inflammation and Fever. In treating *low asthenic* or *adynamic inflammation* all depression and depletion is to be carefully avoided. Even *laxatives* must be employed with extreme caution. If absolutely necessary it is best to give them in small (half) doses and supplement their action by liberal injections of hot water. Elimination of waste matter from the blood and system is still to be sought, but it must be by *stimulating diuretics* (sweet spirits of nitre, carbonate, acetate, or muriate of ammonia, digitalis), and direct *stimulants* and *tonics* must be given from the first (ammonia, wine, strong ale, whisky, brandy, ether, gentian, calumba, nux vomica). For the horse the following may serve as an example: Recipe: Sweet spirits of nitre, four ounces; sulphuric ether, two ounces; tincture of gentian, ten ounces; digitalis, one dram. Mix. Dose, two ounces in a pint of cool water four times a day. When there is great debility and prostration ammoniacal and alcoholic stimulants must be given freely, while if the fever heat rises very unduly the cooling diuretics (citrate, tartrate, or acetate of potassa, or nitre, etc.), and even sedatives (bromide of potassium, hydrobromic acid, chloral hydrate, salacin, salicylate of soda), must be resorted to. If there is any indication of a special depressing poison in the system, or of the absorption of septic or other noxious matter from a wound, antiseptics (hydrochloric acid, or salicylic acid, sulphite of soda, quinia, or chlorate of potassa) may be advantageously added to the prescription.

In these cases of asthenic inflammation, as in the advanced and

debilitated stages of sthenic inflammation, the diet should be as good as the patient can digest. Boiled oats, barley, or flaxseed, rich, well-boiled gruels, and beef-tea (even for herbivora,) may frequently be resorted to with advantage.

Local Treatment of Inflammation. In all forms of superficial inflammation the local treatment occupies an important place. The persistent application of *cold* (cold water in a stream, ice-bags, freezing mixtures) will sometimes overcome the tendency to inflammation or arrest it. This is especially sought when a violent inflammation (as in a wounded joint) threatens to destroy an important organ. If adopted, it must be persisted in, as if it is suspended too soon the reaction is likely to make matters worse than ever. *Cold astringent applications* have a similar tendency. Sugar-of-lead, one-half ounce ; laudanum, one ounce ; water, one quart, may be kept applied by means of a linen bandage. The water may often be advantageously replaced by extract of witch-hazel. If the inflamed part is superficial the lotion may be made *antiseptic* (carbolic acid, one dram ; or sulphurous acid solution, five ounces ; water, one quart). *Hot applications, fomentations, poultices* are nearly always appropriate but they should be made antiseptic to prevent bacterial development. When adopted they should like *cold* ones be kept up as continuously as possible. These soothe alike the superficial and deeper parts, the latter through sympathy, producing first a relaxation of vessels and tissues, and later a contraction of the former attended by pallor of the surface. They greatly favor suppuration when that is already inevitable, though in other cases they may obviate it by checking at an early stage the acute inflammatory process on which it depends. Any bland agent that will retain heat and moisture will make an excellent poultice, though flaxseed-meal is the type of a soothing demulcent application. Very slight inflammation may be successfully treated at the outset with a *stimulating embrocation* (alcohol or camphorated spirit), yet in the more violent type of acute inflammation all local excitants tend to aggravate the disease. In these violent forms the activity of the disease should be first abated by local soothing and general sedative measures, and then the part over the inflamed organ may be safely treated with a stimulating liniment or even a blister. In such cases the liniment first acts as a derivative of blood and nervous

energy from the inflamed part, and later and still more beneficially by securing in it a sympathetic healing process, like that set up in the skin. It is further probable that the absorbed albuminoids, which have been modified in the congested part often exercise a decided effect on the inflamed tissue. In raw sores where inflammation has been set up the granulations may become dropsical or excessive, bulging beyond the adjacent skin as *proud flesh*. This should be repressed by touching it gently with some mild caustic (lunar caustic), so as to produce a thin, white film, and the remote cause of the inflammation (often a local irritant) should be sought and removed. In some unhealthy sores tending to excessive granulation, the compound tincture of myrrh and aloes may be applied daily with great benefit. When the granulations become excessive they may be scraped down to the level of the skin and then treated with an antiseptic (iodoform, boric acid, acetanilid, aristol.

Blistering. In subacute and chronic inflammations and in those acute forms in which the violence of the inflammatory action has been already subdued by soothing measures, blisters and other counter-irritants may be employed to counteract the remaining inflammatory action. These act primarily by drawing off blood and nervous energy from the inflamed organ to the skin, and secondarily, by establishing a sympathetic healing process in the diseased part, simultaneously with the work of recovery in the skin, when the blister has spent its action. But if applied above a part which is still violently inflamed, there is apt to be serious aggravation, through this same sympathy with the part suffering under the rising of the blister. In this way great and irreparable injury is often done through the laudations of particular blisters for the cure of given diseases, without any reference to the stage or grade of such disease. The value of a blister depends far more on the time of its application than on the ingredients of which it may be composed.

Firing. This acts in nearly the same manner as a blister, and demands similar caution in its application. It is especially available in subacute and chronic diseases of the joints, bones, and tendons, and may be made more or less severe according to the nature and obstinacy of the disease. It is applied in points or in lines at intervals of one-half to one inch, and penetrating one-

third, one-half, or entirely through the skin. The hotter the iron the less the pain, but the greater the danger of destruction of the intervening skin by the excess of radiating heat. Hence the contact of the heated iron with any one part must be judiciously graduated to the heat of the iron and the delicacy of the skin, and should not exceed the fraction of a second.

Massage, Rubbing. In chronic inflammation and even in some acute forms, with considerable exudation, rubbing or massage is of great value. It hastens the progress of the blood through the veins, tends to restore the normal circulation in the stagnant or partially obstructed capillaries, moves on the exuded liquids in the lymphatic plexus, rendering the absorption more active, and at once prevents the process of disintegration of the tissues and obviates, the necessity for their solution and removal. This may be largely accomplished by the use of the brush or rubber, or by careful manipulation especially in the direction of the veins. If the inflammation is near the surface the use of antiseptic and deobstruent agents will heighten the good effect. Iodoform, iodide of potassium, boric acid may serve as examples.

Suppuration. Abscess. The great variety of the causes and forms of suppuration would forbid any extended notice of its treatment in this place. It seems preferable to refer the reader to the subject of pyæmid and the various surgical and medical diseases in which suppuration takes place.

DISEASES OF THE RESPIRATORY ORGANS.

Importance of diseases of the respiratory organs—in horses and dogs. Proclivity through over-exertion, through extent and delicacy of the mucosa, through changes of temperature, through weather, through air pollution, through kind of diet, through change of latitude, through nervous sympathy, through debilitation of the lung tissue, through suppression of perspiration, through a high dew point, through bacteria and other germs, through youth and change of habits.

These are among the most frequent and grave of all affections of the domestic animals. They are especially important however in the case of animals that depend on the soundness of their wind. In horses and dogs accordingly any permanent injury to the organs of respiration will seriously impair the value, not only because of the diminished usefulness of the affected animal, but also because of the probable deterioration of their progeny. The rapid paces demanded of these animals and the strain to which the respiratory organs are subject are potent causes of respiratory disorder. In all animals, however, the extent of the respiratory surface and its extreme delicacy and tenuity especially predispose it to disease. Hales estimates that the mucous membrane covering all the air sacs and air cells is, in the calf, no less than 250 square feet. As the chest of the horse is at least double that of the calf, and as it contains much less connective tissue, and is made up of minute air cells from $\frac{1}{70}$ — $\frac{1}{200}$ inch in diameter and separated from each other by walls so attenuated that the contained capillary blood-vessels are equally exposed to the air on both sides, in two adjacent air cells, the estimate for the average horse must be considerably above 500 square feet. This membrane, incomparably the most delicate and susceptible in the animal economy, is constantly in contact with the air in all its variable conditions, and is necessarily affected by these variations.

The severe changes of temperature are not without their influence on this sensitive membrane. If these changes are sudden, as for example in our northern states where the temperature will vary from 50° to 70° Fah., in a single day, the danger of injury

becomes imminent, and the lungs require to be strong indeed to resist their effects. Sudden transition from the hot close atmosphere of the barn or stable to the chilling winds of winter is equally hurtful. But it is not alone the transition from warmth to cold that is injurious. The general relaxation attendant on the sudden change from a cold bracing atmosphere to one unduly hot is even more injurious. How frequently do human beings suffer from colds as the result of a close sultry period at once supervening on a clear cold one? How extensively do chest diseases prevail among horses brought from the clear pure atmosphere of the field, and shut up in close, hot stables? Here, no doubt, there is superadded the impurity of the too often infected air, the change of diet, of exercise and of general care yet we find that affections of the air passages are to a great extent in ratio with the heat of the building. Hence their constant presence in dealer's stables where it is thought desirable to keep the horses warm to hasten the improvement in the coat.

The suddenness of the transition is usually a principal cause of injury. Where the climate changes slowly the animal economy becomes habituated to it and resists successfully the injurious influences. Thus when spring merges gradually into summer and autumn into winter, diseases of this kind are far less frequent. But on the other hand a sudden and extreme variation of temperature, whether in the ordinary course of the season or from a wide change of latitude, is notoriously attended with diseases of the air passages. Ayrshire, shorthorn and Jersey cattle, when first imported into the Northern States of America, contract colds, consumption and other chest diseases to a far greater extent than the native races, though their progeny or even they themselves after acclimatization, exhibit powers of resistance nearly equal to the native stock. Sheep that have been shorn in midwinter or early spring often repay the inhumanity of their owners by dying of inflamed lungs. Southdown and Leicester sheep, sent from England to the north of Scotland, demand at first the greatest care to protect them against the increased rigor of the climate. The army veterinary statistics of France show that horses transported from the southern parts of the country to the more northern stations, suffer largely from pulmonary affections. But if the change is effected slowly the requisite powers of resistance are

acquired and the novel conditions of life cease to be injurious. That this varied power of resistance is not confined to the higher animals would appear from the experiments of W. Edwards on cold blooded animals. He subjected them in winter and in summer respectively to a very low temperature and found that whereas in summer their temperature declined 3° to 6° Cent., in winter they had a greater resistance and barely declined $\frac{4}{10}$ths of a degree.

The action of cold on the surface of the body often leads to morbid states of the air passages as the result of nervous sympathy. A beast is subjected to a keen cold wind, is attacked with shivering, and inflammation of the chest supervenes. The result is rendered more certain if the wind is associated with rain and if the animal has been previously in a state of perspiration. A heavy coat of hair, a profuse perspiration, and a cold draught often combines effectively to produce respiratory disease.

It must be added that the chilling debilitates the nuclei of the animal tissues, and lessens their power of resistance to noxious influences. The excess of cold in the freezing of a part, is followed by congestion and even violent inflammation with perhaps sloughing after it has been thawed. The persistence of such tissue debility is familiar to us all in the example of chillblains. A less extreme application of cold affects the tissues and nuclei less powerfully, but none the less surely. The increased liability to disease of the chilled system is strikingly illustrated in the experiment of Pasteur with anthrax. The chicken which had proved refractory to an ordinary dose of anthrax virus, was dipped in water at ordinary temperature until the heat of its body was reduced, and then it fell an easy victim to the anthrax bacillus. In the same way the person who recklessly exposes himself to wet and chill falls a ready victim to intermittent or yellow fever from which he would otherwise have escaped. Debility from another cause, such as bruise or laceration, favors deep-seated invasion by pus cocci, and a resulting abscess, from which the patient would have remained free, but for such traumatism.

But the effect of cold is not confined to the sympathy between the skin and respiratory mucous membrane, nor the revulsion of the blood toward internal organs, nor to the debilitating of the

tissues. The application of cold constringes the vessels and lessens the freedom of the circulation and suppresses the normal cutaneous exhalation. A somewhat similar condition may be induced by prolonged exposure to the rays of a burning sun, the skin becomes hot, dry and rigid, and incompatible with the maintenance of the respiratory function. In either case there is a retention of effete and deleterious matters in the circulation which it was the function of the skin to have eliminated. The danger of such retention may be best exemplified by noting the result of the complete repression of perspiration in the remarkable experiments of Fourcault and Bouley. The former covered dogs and other small animals with an impermeable varnish which induced death after some days or in some cases in a few hours. Bouley shaved three horses and covered the skin with tar. There resulted dullness, torpor, deep, slow breathing, weak and diminishing pulse, muscular tremors, manifest cooling of the body and expired air, and deep violet color of the mucous membranes. They died respectively on the seventh, ninth and tenth days. A fourth horse covered with a layer of strong glue and then with tar perished nine hours after the application. The bodies were like those of animals that had died of suffocation. The mucous membrane of the stomach and bowels was gorged with black blood, the lungs violently congested—dark red and heavy—the air-tubes filled with frothy material, and the lining membrane of the heart had dark spots of blood extravasation. It is no longer then matter for surprise that temporary suppression of the insensible perspiration should be followed by diseases of the chest or abdomen, that extensive burns of the surface of the body should be speedily followed by inflammations of internal organs or that extensive and severe cutaneous inflammations should be associated with internal lesions.

Since the days of Hippocrates it has been universally acknowledged that moist seasons and localities are less salubrious than dry ones. As already observed moisture in a cold atmosphere intensifies its effect. In a hot, close atmosphere it strongly conduces to putrefaction in dead organic matter, and the air becomes loaded as a consequence with noxious gases, and in its lower strata with bacteria in a state of active growth. This condition

is most intense in close, unventilated stables, and manifestly operates in both predisposing to and exciting those diseases of the chest and other parts, so frequent in such places. Winds raise and carry such germs, but also sooner rob them of virulence. (See Zymotic Diseases). Susceptible, young animals, newly housed, usually suffer the most severely from these injurious conditions. Often in their case frequent, extreme and sudden changes, and great atmospheric impurity, are combined with a diet to which they have been hitherto altogether unaccustomed. In young horses there are superadded the exertions—too often extreme—connected with training or work. There are the heats and chills, the soaking perspiration and the frigid winds and rain, the general exhaustion, but particularly the overwork of the respiratory organs, each of itself calculated to superinduce disease. Percivall justly remarks that among young horses, newly stabled and put to work, the prevailing diseases are "catarrh, sore throat, strangles, bronchitis, pneumonia and pleurisy." His tables of the diseases attacking the horses of his own regiment (1st Life Guards), are so instructive that I here reproduce them:

A TABLE (COMPILED FROM EXTRACTS FROM A "REGISTER OF SICK HORSES" LIMITED TO A GIVEN PERIOD) SHOWING THE COMPARATIVE AGES AT WHICH HORSES APPEAR MOST DISPOSED TO CERTAIN ORGANIC DISEASES.

	No. of Patients Under 5 Years.	No. in Their 5th Year.	No. above 5 Years and Under 10.	No. 10 Years and Upwards but under 20.	No. 20 Years and Upwards.	Total.
Disease of the lungs	170	50	20	50	10	300
Disease of the bowels.	10	20	40	70	20	160
Disease of the brain	4	2	5	14	2	27
Disease of the eyes	30	10	70	35	5	150

It will be seen that nearly one-half of the sicknesses, occurring among the horses of the regiment, were chest diseases, and that nearly three-fourths of these were in animals under five years old, or in those newly purchased from the country.

The subjoined table shows the relative prevalence of disease in different months of the year, deduced from the Register above referred to:

	Disease of the Lungs.	Disease of the Bowels.	Disease of the Brain.	Disease of the Eyes.
January	20	12	1	10
February	25	8		9
March	23	11	1	7
April	19	10	6	10
May	13	3	3	9
June	14	16	1	13
July	13	13	3	19
August	11	23	3	17
September	11	5	10	19
October	24	3	3	9
November	19	10	3	9
December	16	9	1	4
Totals	208	123	35	135

In this table the extraordinary prevalence of lung diseases in spring and autumn is very noticeable. There only remains to notice the number of deaths occurring in the same regiment from pulmonary and other diseases.

Deaths from pulmonary disease 77
Deaths from other diseases (Glanders and Farcy and accidents excepted) 57

It is thus seen that though individually less dangerous than many affections of the abdomen, brain, etc., yet by reason of their greater frequency chest diseases induce the greatest mortality among this class of stock.

In treating of the diseases of this class of organs they will be sub-divided according as they affect the *nose*, the *throat*, the *neck*, and the *chest*.

DISEASES OF THE NOSE.

EPISTAXIS. BLEEDING FROM THE NOSE.

Epistaxis as a primary and secondary affection. Causes—mechanical, over exertion, blood pressure, new formations, diseased mucosa, disease of the nasal venous plexus, disease of heart or lungs, in blood diseases, in hæmorrhagic constitution, in bacteridian diseases of the respiratory organs. Symptoms. Often one nostril, blood bright, red, clotted, sneezing, (not retching, acid, nor cough). *Treatment.* Mechanical, astringent, cold, plugging in solipedes and other animals: hæmostatics.

As a primary affection this occurs more frequently in the horse than in any other domestic animal, though as a symptomatic disease it is common in all farm animals.

Causes. The most common causes are mechanical injury of the Schneiderian membrane, violent congestion of this membrane during extraordinary excitement or exertions, as in coughing, in a closely contested race, in a trying hunt, in drawing heavy loads, especially if uphill and with a tight collar. It may coincide with congestion of the brain acting to some extent as a vicarious discharge, or with the formation of new structures as polypus, or cancer, in which, from the looseness and friability of their texture, the vessels readily give way. The softened membrane is equally liable to laceration or rupture during the progress of inflammation and particularly when fibrinous (croupous) exudations are being detached. In all these cases animals of a strong, vigorous constitution and with a full or plethoric habit are most liable to be attacked. Various congestions of the mucosa in diseases of the heart or lungs are additional causes. Disease or injury of the cervical branch of the sympathetic nerve, and varicosity of the pituitary venous plexus must be accepted as occasional causes.

Epistaxis is also met with in states of general weakness and with deteriorated blood, as in anæmia, in the course of various fevers and in those hemorrhagic constitutions in which the altered blood appears to find an easy passage through the debilitated or ruptured coats of the bloodvessels. Thus it is seen in the so-called *purpura hemorrhagica* in the horse, in *small-pox* in sheep,

in anthrax, and in *swine plague* and hog cholera. *Hering* records the case of a number of pigs suffering from a scorbutic affection and which bled profusely from the nose. In bleeders (haemophilia) and in leucocythaemia it is liable to appear.

The ulcerations of the mucous membrane occuring in *glanders* and *chronic catarrh* have proved exciting causes of the hemorrhage. Lastly the intense heats of summer and prolonged exposure to the direct rays of the sun induce a general relaxation and a determination of blood to the surface which rouses to activity the latent tendency.

Symptoms. The bleeding, usually from one nostril only, falls in a succession of drops, (rarely in a stream), collects in clots around the nostril, and bespatters surrounding objects as it is expelled forcibly in sneezing. It is usually of a bright crimson hue or, in fevers or poisoned conditions of the blood, of a dark or blackish color. It is distinguished from pulmonary hemorrhage by the absence of cough and of a frothy condition, and from bleeding from the stomach by the absence of the blackened clots and acid odor which indicate the presence of the gastric juice.

It is usually to be further distinguished from these in all animals, save solipedes, by the absence of blood in the mouth.

Treatment. Nasal hemorrhage often stops spontaneously, but if the discharge is profuse or long continued, and especially in weak or anæmic conditions it must be treated energetically. Care should be taken, however, to ascertain first, whether it is not vicarious of some other and more dangerous condition like cerebral congestion.

The head should be placed in an elevated position by tying it up to the rack, and cold water or ice kept applied over the head and neck. Matico powder may be blown into the affected nostril during inspiration, or a solution of alum (4 drachms to 1 pint of water) or other astringent may be thrown in by means of a syringe. A tablespoonful of peroxide of hydrogen thrown into the nose with an ordinary syringe will give immediate relief. (Gillette.)

Plugging the affected nostril with a pellet of tow covered with matico, tannin, tincture of chloride of iron (1:10 or 20) or other astringent may be employed when other means fail. By means of a cord attached to the plug it may be withdrawn after all dan-

ger is past. In solipedes, if both nostrils must be plugged, wrap the tow around two elastic caoutchouc tubes and introduce these, or in the absence of these perform tracheotomy.

Any tendency to recurrence may be met by the internal administration of gallic acid (horse and cow ½—1 drachm), acetate of lead (horse and cow ½—1 drachm) or, in anæmic conditions, tincture of the perchloride of iron (horse and cow ½ oz.) in water.

RHINITIS. CORYZA. NASAL CATARRH. COLD IN THE HEAD.

Coryza in the horse: Causes, wet, cold after perspiration, damp climate, stable, soil, new buildings, hygroscopic building materials, youth, age, poverty, nervous sympathy, local irritants, iodine, specific disease poisons. Symptoms, dry congestion, watery discharge, muco-purulent discharge, eyes involved, chill, fever, circulatory and breathing disturbance, defecation, urine, glandular swelling. Inflammation of the sinuses, the severe effects. Duration in slight cases, in severe, in sinus complication. Treatment, hygienic, nursing, dietary, steam, sulphur dioxide, febrifuges, insufflation, electricity, solvent, antiseptic, stimulant.

Under this head will be considered simple inflammation of the nasal mucous membrane. This disease might be considered as a mild febrile affection with the local manifestation in the nose, but it is more convenient to treat of it here as a malady of the nasal chambers.

CORYZA IN THE HORSE.

The chief *causes* are exposure to wet and cold and especially when the subject is exhausted and the skin relaxed and covered with perspiration. In these circumstances a piercing wind, a cold drizzling rain, or a draught in the stable is particularly dangerous. Sudden alternations of temperature and especially a change to a warm stable when the general effect is aggravated by the impurity of the atmosphere and the irritant emanations from dung and urine. Damp climates are more injurious than those that are clear, dry, and bracing, and so are equally damp stables whether the moisture is due to the nature of the soil, such as a cold impervious and undrained clay, or of the building which, from its newness, may retain a dangerous amount of moisture in the plaster, or because of the hygroscopic properties of the building materials which draw moisture from the surrounding soil. It mainly attacks young horses after they have passed out of the hands of the breeder or dealer, and have been placed in new conditions of life alike as regards feeding, stabling and work. Old and ill-conditioned animals are more susceptible than the strong

and vigorous, and the changes of the coat in spring and autumn prove strong predisposing causes. Nervous causes are potent in causing engorgement of the erectile tissue covering the turbinated bones, and local irritants, like septic dust, lime, ipecacuanha, pollen of certain plants, smoke, and irritating fogs may precipitate it. Iodine in large doses produces temporary catarrh. The weakness of the mucosa from a previous attack predisposes to a second. Occasionally the disease sweeps over a country, assuming the form of an *epizootic* when it may perhaps be preferably considered as a catarrhal fever, strangles or mild type of *influenza*, *which see*.

Symptoms. In the *milder forms* of *coryza* the symptoms may be almost exclusively local, consisting in redness and dryness of the membrane lining the nose and sneezing, soon followed by the bilateral discharge of a thin transparent watery liquid, succeeded by a turbid flow (epithelial cells in excess) and after two or three days by a thick, white, flocculent, puriform fluid (suppuration diapedesis). With the supervention of the purulent discharge, comes an abatement of the local inflammation and the freer the discharge the greater usually is the relief obtained and the more rapid the recovery. The eyes are usually red and watery and sometimes the eyelids are swollen. This implies the continuity of the inflammation through the lachrymo-nasal duct, and the obstruction to the flow of tears into the nose.

When *constitutional disturbance* exists a rough or staring coat appears as one of the first symptoms, the sneezing is more violent, the nasal mucous membrane is more reddened and swollen, the eyes more dull, sunken and watery, the mouth hot and clammy, the temperature of the body raised, the pulse more frequent and having a sharper beat, the impulse of the heart may often be felt by applying the hand to the chest just behind the left elbow, the appetite is fastidious and the secretions of the bowels and kidneys are diminished, the latter being denser and more highly colored, from the absorption of irritating or infecting matters the glands under the throat are swollen and the swelling of the mucous membrane may be such as to impair breathing and even to threaten suffocation. In severe cases in which the inflammation extends to the nasal sinuses there is heat and tenderness over the forehead and the pain and weight are manifested by the pendent head and the red

sunken, watery eyes and tumefied eyelids. When it extends to the throat, the cough, the difficulty in swallowing and the local tenderness on handling are characteristic.

Course. With the occurrence of suppuration, improvement commences and if the inflammation does not extend beyond the nasal chambers, and if it is not kept up by a repetition or continuance of the cause the disease will have terminated in recovery in eight or ten days. For ulterior consequences in bad cases see *chronic catarrh, conjunctivetis, abscess of the nasal sinuses, laryngitis, stomatitis, staphylitis.*

Treatment. In slight cases the simplest treatment only is required. Place the animal in a dry, airy, loose box, clear of draughts, and with uniform temperature of 55° to 60° Fah., if obtainable. In the cold season blanket warmly, and hand-rub and loosely flannel bandage the legs. Feed on sloppy bran mashes only and add half an ounce to an ounce of powdered nitrate of potash daily. Give fresh water *ad libitum*, solicit the action of the bowels by giving injections of warm water three times a day, and encourage the nasal discharge by causing the patient to inhale steam for half an hour or an hour twice daily. This may be done by giving scalded bran in a nose-bag or by keeping the head over a bucket containing hay with boiling water poured over it, the steam being meanwhile directed by a bag open at both ends one of which is fixed around the animal's nose and the other round the mouth of the bucket. As a local astringent, tonic and antiseptic the fumes of sulphur (burned behind the animal and no more concentrated than can be breathed with comfort) will do much to cut short the attack. It is more soothing if combined with steam. Shut doors and windows, add a few drops of alcohol to some pinches of sulphur and burn on paper laid on a clean shovel or piece of sheet iron, When enough has been used extinguish by covering with a cup or other object. Repeat several times a day. Under this treatment recovery may be completed in three or four days.

In severe cases attended with fever, besides the above a dose of laxative medicine may be given (three or four drachms of aloes), with this precaution, that if the fever is of a low type or the malady epizootic, half the dose only can be safely allowed (2 dr.) on account of the danger of superpurgation. The nostrils must

be more assiduously steamed and linseed tea may advantageously replace fresh water as a beverage. If there is much swelling and tenderness of the glands a poultice should be applied to the throat and between the jaws, and sulphur fumes as advised above, or anodyne astringent insufflation powder may be resorted to. Morphia chlorate two grains, bismuth nitrate, six drachms and finely powdered gum arabic three drachms may be blown into the nostril during inspiration, or the astringent anodyne injection advised below for chronic catarrh may be used. Cocaine spray is often very helpful, or the same agent may be used in the liquid form on cotton wool inserted in the nasal chamber. If this is without effect a weak continuous current of electricity will cause constriction and give prompt relief. It may be repeated every few hours. In the absence of this the emanations from a weak solution of ammonia or from carbonate of ammonia may be used. In cases with excessive and persistent muco-purulent discharge, with presumptive infection from outside sources, or in the young, from the diseased maternal passages, insufflation with calomel, painting with a two grains to the ounce solution of nitrate of silver, or injection with some other germicide may be resorted to.

In case the fever is of a low type, liquor of the acetate of ammonia (4 ozs.), sal-ammoniac (¼ oz.), or even carbonate of ammonia (½ oz.), may be given several times a day, with sweet spirits of nitre (½ oz.) and tincture of gentian (1 oz.) Alcoholic stimulants are often used. Inhalations of iodine and iodide of potassium with ether and chloroform are often successful.

SIMPLE CORYZA IN CATTLE.

Coryza mild in cattle. General treatment. Coryza in sheep from exposure, intemperate seasons, clipping. Acute and chronic. Wholesale treatment in flocks. Coryza in pigs. Coryza in dogs, simple, secondary. Treatment, food, laxative, febrifuge, nauseating, expectorant, antiseptic, gaseous, electric.

This is usually a very simple malady when confined to the nasal chambers, and not of infective origin. When, on the other hand, it attacks the sinuses it becomes a disease of extreme gravity. (See Catarrh of the Sinuses). Symptoms are as seen in the horse, but the discharge may be overlooked because of the animal licking it out with his tongue. Treatment does not essentially differ from that laid down above, and recovery may be expected in seven or eight days. If a laxative is wanted give from one to two pounds Epsom salts.

SIMPLE CORYZA IN SHEEP.

Coryza is usually slight and is manifested by sneezing and running from the nose. It occurs in animals clipped or badly sheltered during the more inclement seasons. In the worst cases the discharge becomes persistent and emaciation ensues so that it is necessary to interfere. Valuable animals may be treated on the same principles as oxen, and in the case of large flocks by shelter in a warm, dry, cleanly and airy place and fumigations of steam and the fumes of burning sulphur repeated daily, together with nourishing diet, such as boiled barley or other grain, and quarter ounce doses of nitre and common salt.

CORYZA IN THE PIG.

Hogs are not very subject to this disease and are easily treated by warm, sloppy food, and as a laxative three or four croton beans, according to size, powdered and given in the aliment.

CORYZA IN DOGS.

Dogs are rarely the subjects of simple coryza, though it is constant in distemper. It sometimes proves troublesome in pup-

pies and old dogs. A laxative (½—1 ounce castor oil) may be followed in strong and very feverish cases by tartar emetic (¼—½ grain) three times a day. Spraying or sponging the nose with a weak solution of chlorate of potass, common salt, or potassium permanganate will greatly relieve. Inhalation from burning sulphur, or from carbonate of ammonia, or both may be used when sponging or spraying is difficult. In inveterate cases, the weak electric current sent through the cheeks, or the insufflation of acetanilid, iodoform or calomel may be tried. As a rule, saltpeter in five grain doses, given in the water, will prove helpful, and in weak conditions wine, tincture of gentian or nux-vomica may be used.

CHRONIC NASAL CATARRH. NASAL GLEET. OZŒNA IN THE HORSE.

Chronic catarrh in horse, simple form, loss of tone, inflammation, nature of discharge, glandular swellings, differentiation from glanders. Treatment, astringent, tonic, stimulant, hygienic, locally astringents, antiseptics, injections.

A chronic discharge from the nose is often seen in the horse as a sequel of *coryza* or *sore throat*, or as an attendant on other affections of the upper air-passages, and the different conditions productive of this symptom may here be noticed.

1ST. SIMPLE NASAL CATARRH. NASAL GLEET. OZŒNA.

In long standing *coryza* the nasal mucous membrane becomes relaxed, fails to acquire its lost tone and continues to pour out a mucopurulent product. This is really a persistence of inflammation of a low type, under the influence of which the membrane secretes pus in place of its normal mucus. The discharge is white, thick, creamy, has little tenacity, and flows uninterruptedly. There may be slight enlargement of the submaxillary glands, and if the case is of long standing and the patient in low condition sores may appear on the mucous membrane. These ulcers are distinguished from those of *glanders* by the absence of the unhealthy angry aspect and excavated borders of the latter, by the absence of the small nodular deposits on the mucosa, by the less viscid nature of the secretion, and by the absence of submaxillary swellings, or if these exist, by their being less nodular, less indurated and less firmly attached to surrounding parts. The coincidence of ulcers and submaxillary swellings is always, however, matter for the gravest suspicion, and such cases should, as a rule, be subjected to the mallein test. (See Glanders and Farcy).

Treatment. In simple nasal catarrh, due alone to the relaxation of the mucous membrane, the internal use of tonics and the local application of astringent solutions to the nose rapidly restore the parts to a healthy state.

Among *stimulants*, cubebs, cayenne pepper and copaiba have a

stimulating and styptic effect on the mucous membrane and each of these has been successfully used in such cases. *Cantharides*, in five-grain doses, have proved even more successful, (Vines, Percivall). *Sulphate of Copper* in drachm doses in mucilage night and morning has proved very efficient (Sewell, Percivall). *Arsenious Acid* has been employed with still better results. The dose, of five grains may be intimately mixed with a scruple of bicarbonate of soda and given daily in food.*

But the most efficient tonic in these cases is arseniate of strychnia. Its good effects may be secured by combining with the above mentioned powders of arsenious acid and bicarbonate of soda, half a drachm of powdered nux vomica for each dose.† These powders will usually be taken in food, and may be continued for a month, or until the discharge ceases.

In all cases the general health must be carefully attended to. Keep the patient in a dry, clean, airy building without draughts of cold air; give moderate exercise in the open air; and good grooming; and allow nutritious food of mildly laxative properties, —as occasional bran mashes and roots in winter and succulent grasses in summer.

Local Applications. These are the most important remedial measures and usually of themselves succeed in reëstablishing a healthy condition.

The agents proving most useful are of an astringent nature and in obstinate cases one may be substituted for another as the last appears to lose its effect. Sulphate of Zinc or Sulphate of Copper in the proportion of half a drachm of either to a quart of water, may be used, or if there is much fœtor, a solution containing a drachm each of carbolic acid and carbonate of potash in a quart of water is to be preferred. In either case the addition of an ounce

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* In giving this agent, any redness or watering of the eyes, or colicy pain should be carefully watched for, and when these premonitory symptoms of poisoning are noticed the medicine should be at once suspended to be commenced a few days later in smaller doses.

† Whenever nux vomica or its alkaloids, strychnia or brucia, are given, increased irritability and nervousness should be carefully watched for and especially any involuntary twitching of the muscles. On their appearance the agent must be suspended and commenced a few days later in half the former doses.

of pure glycerine renders the lotion at once more soothing and more efficient. The solution must be rendered tepid before injecting it, to obviate the irritation attending on the contact of a cold fluid with the delicate membrane of the nose. Among other agents may be named creolin, creosol, creosote, acetate of lead, potassium permanganate, and silver nitrate. Peroxide of hydrogen may be used either as injection or in spray.

The mode of injection is a matter of no small moment. It has been done in some instances by means of a large syringe but the irritation attendant on such a process is an insuperable objection to its use. A better instrument is that introduced by Professor Rey of Lyons. It consists in a tube bent on itself at an angle of 35° so as to form two arms of inequal lengths. The longer fifteen inches in length, one and a half in diameter and widening into a funnel at its free end ; —the shorter about five inches long and tapering towards its free end where its aperture is only two-thirds of an inch across. The instrument is made of block tin or extemporaneously of gutta percha. Over the shorter arm is placed a tightly fitting leather ring four and a half inches in diameter on which is applied some wet tow to adapt it to the nostril and effectually close it. The nose having been drawn in so as to place the head in a vertical position, the short arm of the instrument is introduced into the affected nostril, and the liquid being gently poured into the long arm rises slowly in the nose until it is filled and the liquid flows from the nostril on the opposite side. In introducing the tube care must be taken that it may not irritate the inner wall of the nose on the one hand, nor pass into the blind pouch, known as the *false nostril*, on the other.

The greatest gentleness and tact are requisite in thus injecting the nostrils, though in troublesome animals it is sometimes necessary to resort to blindfolding or even to the application of a twitch on the ear, or finally to strapping the animal (head included) to a smooth firm vertical surface (operating table).

COLLECTION OF PUS IN THE NASAL SINUSES.

Nasal Sinuses, position, orifice, suppuration, symptoms, treatment, tonics, astringents, antiseptics, trephining, significance of the fetor, mode of recovery.

In severe *coryza* the nasal sinuses become implicated as shown by the intensity of the symptoms, the prostration, the hanging head, and the heat and sometimes tenderness between the eyes and immediately beneath them on the side of the upper jaw. These sinuses are large spaces filled with air, situated between the superficial and deep plates of the bones of the face and opening into the nostrils by a narrow orifice in the upper part of the nasal chambers. When pus is largely formed in these it fails to flow out as rapidly as produced, parts with a portion of its liquid elements, increases in consistency and sometimes even undergoes decomposition, so that the discharge from the nostril has a putrid odor.

The most distinctive symptoms of this form of *nasal gleet* are obtained by percussing the sinuses, and in those cases in particular in which the accumulation is confined to one side of the head, the contrast between the two sides is unmistakable. By gently tapping the forehead with the middle finger from one eye to the other the flat solid sound on the diseased side is easily distinguished from the clear drum-like resonance on the healthy one. By tapping on the bone beneath each eye and just above the ridge on the side of the upper jaw, the difference between the two sides will be recognized in the same way. In some old-standing cases increased tenderness and slight bulging of the bones over the affected sinuses are often superadded to the other symptoms. The eye on the affected side is usually retracted so as to seem smaller.

Treatment. In some cases the use of tonics and astringent injections as recommended for the treatment of *ozæna* will prove successful, but more usually it is needful to open and inject the sinuses.

For this the following articles are required: scissors, a knife, forceps, a trephine or circular saw from half to three-fourths of an inch in diameter and a whalebone or metallic prob.

The horse is thrown and made fast with the diseased side of the head uppermost. A point is then selected on a line drawn between the centres of the two eyeballs and an inch to one side the median line of the forehead ; the hair is closely removed with the scissors, and a semi-circular flap of skin over an inch in diameter is dissected from the bone and turned back toward the poll. The trephine is next applied on the bone and a circular portion, having been cut through, is pulled out by the forceps, when the imprisoned pus will commonly ooze from the opening. A second point is chosen just above the lower end of the bony ridge of the upper jaw already referred to and opposite the third molar tooth, counting from before ; the hair is removed as before, a flap of the skin raised upward and backward and the bone trephined to open the second sinuse. The point of election for this orifice is more important than that of the first. If it is too near the eye the lower part of the sinus, which is separated from the upper by an imperforate bony plate, is not opened and may continue to keep up the discharge from the nose. If on the contrary it is made too low down, the lower sinuse only is opened and the upper being imperfectly washed out from the wound in the forehead will keep up the discharge. Either then this plate must be struck with the trephine or it must be afterward perforated to secure a favorable result. The prob introduced by the wound in the forehead should further appear at the lower orifice.

The cavities are to be washed out first with clear tepid water, and thereafter daily with an astringent solution such as that used for injecting the nose. If the discharge does not escape freely by the lower orifice its exit may be facilitated by drawing a tape through the sinuses, from the upper to the lower, and retaining it there by a knot on each end.

Marked fœtor of the wound will usually indicate necrosis at the edge of the wound, and demands the use of bone forceps or chisel to remove the offending bone.

A cure is affected by the restoration of the membrane to its natural state, or in other cases by the filling up and obliteration of the cavity by granulation.

ABSCESS OF THE FALSE NOSTRIL OR TURBINATED BONES.

Structure of turbinated bones : suppuration or abscess, obstructed breathing, treatment, puncture, plugging, injection, trephining.

The turbinated bones are two fragile bony structures attached to the outer wall of each nasal chamber. The posterior half of each bone closes the corresponding nasal sinuse; the anterior half is rolled upon itself as a sheet of paper might be, and is accordingly open along one side. In this latter a collection of pus may result from severe inflammatory action and the resulting discharge may become somewhat chronic. The flow is greatest after the nose has been raised, from the pus having previously gravitated into a sac in the lower end of the bone. The pus may moreover pass backward into the larnyx from the raising of the head and induce a violent fit of coughing. Sometimes the inflammation has extended to the bones covering the nose which are bulging and tender. The thin turbinated bone gives way under the distension, bulges into the nose, and often stops the passage of air through that side. This symptom and the appearance of the swelling cause a close approximation in symptoms to nasal polypus. The facts that it supervened on a severe *coryza*, that it fluctuates on pressure if within reach of the finger, and that pus escapes when it is punctured, exclude the idea of polypus.

Treatment. Puncture of the abscess inside the nose, plugging and daily astringent injections will usually rapidly cure. Gamgee, Jessen and others, recommend trephining of the bone above the nose and washing it out daily, adding that an extensive removal of the bone will correct any existing bulging and deformity.

NASAL DISCHARGE FROM CARIOUS TEETH, ETC.

Ulceration into sinus from caries, loss of molar, overgrown molar. Fœtor, tenderness. Foreign body in the nose.

In cases of a diseased molar tooth in the upper jaw, food getting firmly impacted in the hollow space, irritates the pulp in the fang and the adjacent bone until the progress in ulceration reaches the nasal chamber or sinuse and a nasal discharge is established. If an upper molar tooth is lost the molar formerly opposed to it in the lower jaw grows out and sets up the same train of symptoms. In all cases then in which nasal gleet is associated with much fœtor and with difficulty in eating, a careful examination of the teeth should be made. (See Diseases of the Teeth).

FOREIGN BODY IN THE NOSE.

Professor Gamgee records the destruction of an animal for glanders in which the cause of the discharge was afterwards found to be a physic ball coughed up into the posterior part of the nose and firmly impacted there.

COLLECTIONS OF PUS IN THE GUTTURAL POUCHES.

Structure, position and opening of pouches, inflamed by extension. Symptoms, discharge intermittent with pendent head, swallowing, pasturing, cough, roaring, dyspnœa, inhalation of food, parotid swelling, glandular enlargement. Nature of contents. Treatment, pasturage, blister, tonics, irrigation, puncture, injection.

Though this is commonly a result of severe sore throat or strangles, yet as it causes a chronic discharge from the nose liable to be confounded with those properly due to diseases of the nasal chamber, it is noticed in this place.

The guttural pouches are two mucous sacs peculiar to solid footed animals. They lie side by side above the throat, and in direct contact with the lower surface of the superior bones of the head and the first bone of the neck. They are properly speaking dilatations of the Eustachian tubes which in all animals establish a communication between the pharynx and the middle ear. The opening into the pharynx is at the anterior extremity of the pouch and close to the posterior opening of the nostril, hence the discharge takes place chiefly or exclusively when the head is lowered, since gravitation then favors the escape of the fluid.

Frequently implicated in severe sorethroat the walls of the guttural pouches pour out pus as readily as other mucous membranes in a state of inflammation. As the escape of this product is hindered alike by the narrowness of the orifice and, in the elevated position of the head, by gravitation, it frequently becomes imprisoned and inspissated and proves a permanent source of irritation and discharge. In the early stages the contents are glairy with whitish or yellowish clots ; later they are creamy, caseous or even cretaceous. The mucosa, at first red, congested and tumefied, becomes in chronic cases, hard, thick, puckered and adherent to adjacent structures. It sometimes ulcerates and the contents escape in mass, through the pharynx and nose, or externally behind the angle of the lower jaw. In the last case water swallowed may escape through the opening. More commonly the pus remains pent up, and thickens, and may dry and roll into round or oval pellets from the movements of deglutition. The dis-

charge may be arrested for weeks or months when such masses block the outlet.

Symptoms. The nasal discharge is intermittent or irregular, being often partially or wholly suppressed by keeping the head elevated, and reappearing or becoming profuse when it is lowered. Feeding from the ground, nibbling roots, or pasturing increases the discharge, as the dependent position of the outlet, the jerking and shaking of the head and the movements of deglutition all favor its exit. Swelling of the parotidean region, a flatness instead of resonance on percussion, and the flattening and discharge and sometimes gurgling by manipulation are characteristic. There is cough, roaring during active exertion, sometimes dyspnœa, and, in bad cases, food may be drawn into the bronchia with serious and even fatal results. When the orifice is blocked and the pouch filled with gas the elastic swelling and resilience are characteristic, and pressure may flatten it with a gurgling sound. These symptoms serve to differentiate it from peripharyngeal abscess. The submaxillary lymphatic glands are usually swollen but less than in glanders and not so hard.

Treatment. In mild and recent cases in which the contents of the pouch have not yet become thick and dry, a cure may be effected; *in winter* by feeding the animal from the ground and largely with roots; *and in summer*, by turning out to grass. In either case the matter is allowed to escape almost as soon as formed and the irritated membrane tends to resume its healthy functions. This result will be favored by giving a course of tonics as recommended in *simple ozæna*, and the application of a mild blister to the throat.

Should this fail an operation must be resorted to. Gunther, of Hanover, uses an instrument in the form of a tube a yard long, half an inch in diameter, slightly curved for two inches at one end which is blind, and having an orifice on one side close to this extremity. This tube having been introduced through the chamber of the nose on the affected side and its curved end having been carried into the narrow opening of the Eustachian pouch, tepid water is pumped in and the pouch thoroughly cleaned out. Astringent solutions are then employed. The introduction of the tube is, however, a very difficult operation and one quite impossible to any one who has not the most accurate knowledge of the parts in question.

A second mode of operating is by external incision. For this purpose are wanted scissors, knife, artery forceps, iron probe bent in the form of the letter S, and a tape. The horse having been thrown and fastened and the head extended, the hair is removed from a surface in front of the prominent border of the first bone of the neck, and an incision made between this border and the parotid gland. The incision is made immediately beneath a tendon which may be felt as a flattened cord crossing the border of the bone in its upper third, and it should be carried downward one and a half inches parallel to the margin of the bone. In this preliminary stage the operator has to carefully avoid injury to the parotid gland and the posterior auricular artery and vein. The skin and fascia having been divided the index finger of the left hand is pushed inward and forward until the prominent angle of the large cornu of the hyoid bone is felt, together with the muscle (stylo-hyoid) inserted into this bone above the angle referred to. The next step is important since crossing on the inner side of this muscle and bone at their point of union is the (internal carotid) artery which becomes subsequently enveloped in a fold of the membranous wall of the guttural pouch. The slightest variation in the position of the artery may here prove fatal unless the greatest caution is used. With the knife guarded by the index finger of the right hand the muscle is cut through from behind forward and the pulsation of the artery felt for beneath. Avoiding its position the knife, with its cutting edge turned forward and its point directed toward the horse's nose, is pushed through the walls of the sac. The curved prob is now introduced and carried downward until it is felt beneath the skin just behind the angle of the lower jaw. This may be safely cut down upon with the knife as important parts (vessels and nerves) have been turned aside by its pressure. A tape attached to the prob is now drawn through the pouch and retained by a knot on each end. Tepid water must be injected through the lower orifice daily for three weeks, astringent antiseptic injections thrown in occasionally and the horse fed from the ground. At the end of this period the tape may be removed, and the wounds allowed to heal. During the course of treatment it is always advisable to change the tape several times by cutting the knot off one end of the old one, stitching the new one to it and drawing it through.

Puncture of the pouch at its lower part is a very simple operation when the accumulation of pus is abundant and chronic. The distended pouch gravitates downward largely separating the parotid from the deeper vessels and nerves, and finally fluctuates toward the lower end of the gland. In extreme cases it even opens and discharges. When fluctuation can be felt the sac may be incised with a bistnory or abscess knife and treated like a common sore. Opening with a pointed or olive-shaped cautery has the advantage of checking hæmorrhage and securing more perfect drainage. When there is no fluctuation the incision must be made just beneath the lower border of the parotid, the parotido-auricularis being first cut through, then the gland dissected from the deeper parts when the distended sac can usually be felt and opened. If not felt at once it can easily be reached by a careful dissection upward through the loose subparotidean connective tissue, with the finger nail or handle of the scalpel. A free opening may be made and the wound injected daily with a weak antiseptic solution.

ABSCESS OF THE FALSE NOSTRIL.

In young horses as the result of injury from the bridle or severe *coryza*, a circumscribed swelling sometimes appears on the outer flap of the nostril, at first firm, hot and tender, with a surrounding pasty infiltration, then forming into a tense elastic ovoid mass, the size of a pigeon's or chicken's egg. It may become chronic and remain for an indefinite period comparatively insensible to touch and only slightly interfering with the movements of the nostrils. As soon as the elastic tension betrays the presence of pus it should be evacuated by a free incision made from inside the nostrils and the wound plugged with medicated tow and allowed to heal by granulation.

NEOPLASMS IN THE HORSE'S NOSE.

Nasal fibrous polypus, connection, form, size, bony distortion, obstructed breathing, abrasion, ulceration, sloughing, sub-mucous polypus, structure, degeneration. Symptoms, sneezing, snuffling, discharge, palpation, bony swelling, tenderness. Treatment, forceps, hook, ecraseur, knife, saw. Actinomycosis. Sarcoma, Carcinoma. Consistency, structure, fœtor, glandular swelling. Treatment. Recurrence. Fatty tumors. Bony tumors: cancellated or compact tissue, localized or extended. Cysts. Strongyli. Angioma. Varicosity. Color, obstruction to breathing, hæmorrhage, cicatrization.

These are essentially surgical diseases yet as they induce Chronic Catarrh they may be profitably noticed here.

I. **Fibrous Nasal Polypus.** These are connected to the mucosa by a pedicle or broad base, and vary in size from a pea to a mass which fills the entire nasal chamber, projects from the nostrils and presses outward the septum and facial bones. At times they weigh one or more pounds. They may cause whistling or rattling in breathing, or may completely obstruct the passage of air on the affected side. In time they may cause bulging or even attenuation and perforation of the bony walls, projecting through the hard palate or on the face. Sometimes the surface becomes the seat of granulation, ulceration, or sloughing, causing more or less fœtor. The large polypi make their main growth forward and backward, moulding themselves to the form of the chamber, and displacing the turbinated bones. They commence to grow under the mucous membrane and as they grow and become more loosely attached they carry this as an outer covering and pedicle. When incised they show a structure of interlacing bundles of fibres, with cell elements more or less abundant, according to the rapidity of growth. Gravitz found amyloid degeneration of the walls of the blood vessels and mucous follicles and of the fibres.

Symptoms are difficult breathing, snuffling, a smaller current of air on the affected side, or none, sneezing, a watery, purulent, bloody, or fœtid discharge, and the appearance of the polypus when the nasal chamber is examined in a good light. If beyond reach of vision the polypus may often be felt by the finger. Care must be taken not to mistake the red, angry surface of the

turbinated bones in Catarrh for a polypus. If beyond the reach of the finger, the flat sound on percussion of the nasal and frontal bones on the affected side, and the persistently diminished flow of air may serve for diagnosis. Tenderness shown on percussion is common to this and abscess of the sinuses.

Treatment. The horse having been cast with the diseased side uppermost and the head turned to the light, the tumor is seized with the fingers, the forceps, or hook, and drawn gently outward. The chain of the *ecraseur* may be passed over it and slowly tightened upon the pedicle until it is cut through. This will usually obviate any laceration of the turbinated bones and consequent bleeding. In case of serious hæmorrhage check by cold water, ice, the actual cautery, or by plugging. Polypi with a broad base may be removed with a prob-pointed knife, curved on the flat, and furnished with a long handle. The mass is seized with a vulsella and detachment made by passing the knife with the concave side toward the tumor. In cases where the tumor cannot be seen or reached some have resorted to slitting up the outer wall of the nostril as far as the angle of union of the nasal and maxillary bones, care being taken to make the incision outside the upper end of the cartilage of the ala nasi. If too high to be satisfactorily reached in this way the nasal or frontal bone may be trephined over the body of the tumor as indicated by the flatness on percussion, and the operation performed through the opening thus made.

II. **Actinomycosis.** Though much more common in cattle than horses, yet the occasional occurrence of this in the face of the solipede must not pass unnoticed.

III. **Sarcoma** and **Carcinoma.** These are found growing from the periosteum, or even starting in the cancellated tissue and projecting into the nose, where they give rise to symptoms like those of fibrous polypi. Being much softer in texture and more liable to ulceration and degeneration they are likely to cause a much more offensive discharge. There is also more tendency to the implication of the submaxillary lymphatic glands. The only treatment is surgical and recurrence is always to be feared. (See Diseases of the Orbit.)

IV. **Fatty Tumors** of the nose are described by Röll and Gurlt as existing on the septum and in the sinuses. Being simple, they can be removed with great confidence as to nonrecurrence.

V. **Osseous Tumors of the Nasal walls.** These are described by Röll as osteophytes in the maxillary sinus in chronic catarrh, and by Gamgee as osteomata attached to the outer wall of the nasal chamber, which had to be detached by saw and bone forceps. I have found these latter of a soft porous structure easily detached by the knife, and in other cases dense and requiring, chisel, saw and forceps. In one instance the tumor grew from a dense hypertrophy of the maxillary bone which could not be entirely removed because the molar alveoli were implicated.

VI. **Cysts** named by Röll and others as present in the mucosa of the ethmoid cells in solipedes often contain larva of the strongylus armatus.

VII. **Angioma** may be but an exaggerated development of the abundant venous plexus and erectile tissue on the surface of the turbinated bones. There appears to be at other times an actual increase of the vascular tissue. As might be expected it has no abrupt margin, but gradually shades off into the healthy tissue. The prominent centre has a bluish red or brownish hue. It obstructs breathing, is apt to bleed under violent exertions in draught, or in contested races, and readily ulcerates with a bloody discharge. If it subsides and heals, it is followed by a whitish puckering like the so-called *cicatrix* of glanders.

CATARRH OF THE FRONTAL SINUSES IN CATTLE.

CATARRH FROM TRAUMATISM.

Extent of sinuses in cattle. Causes, blows, unequal teams, locking horns, fracture. Pathology. Congestion, exudation, suppuration, swelling and closure of outlet, prostration, fever, agalactia, septic infection, ulceration, exclusion of oxygen. Symptoms. Crimson hæmorrhage, disturbed breathing, appetite, rumination, position of head and eyelids, percussion and temperature of forehead, fever. Chronic form. Slow progress, emaciation, anorexia, facial expression, hide, discharge, breath heavy or fœtid. Duration. Prognosis. Lesions in sinuses and glands. Treatment. Cold irrigation, icebags, elevation of head, laxatives, diuretics, dehorning, trephining, injections, astringent, antiseptic, blister, tonics.

The gravity of this affection is a consequence of the great extent of the delicate mucous membrane which lines the frontal sinus. This cavity not only occupies the whole forehead from beneath the eyes up to the frontal crest, but extends, in the mature horned animal, into the tapering bony process which forms the basis of support for the horn. The mucosa is rendered all the more extensive by the numerous pillars and septa that pass from the outer bony plate to the inner, giving great strength to the part for purposes of offense and defence. Inflammation of this membrane is usually the result of blows on the horns, and these are much more common among working oxen than dairy cows. The immediate cause is violent contact with the yoke when the head is lowered at pasture, and from blows of a club in the hands of the driver. In countries where the yoke is a broad padded board hung from the horns and resting on the forehead traumatic injuries are much more common. The active and vigorous animal gets the greater part of the work, and the wrench and jar may induce hæmorrhage and catarrh. If the yoke is ill-made or badly fitted the case is worse. The blows sustained by horn or forehead in an ordinary fight, may also be the cause, and a partial or complete fracture of the bony support is especially hurtful when the detached horn is replaced so as to close in the cavity. Blows on the frontal crest are also dangerous.

The *pathology* of the disease consists in an inflammation of the mucosa of the sinus, and the filling of that cavity with blood or,

later, with a muco-purulent fluid, the escape of which is prevented by the closure of the nasal outlet by swelling. This of itself produces violent headache and much nervous disorder as witnessed by the drooping head, closed eyelids, prostration, high fever, anorexia, and in cows suppression of the milk secretion. But there is reason to believe that this is aggravated by the septic germs, which inspired with the air, were already present in the sinus, and which in the comparative absence of oxygen, in a rich culture medium and in contact with injured and debilitated tissues, assume an enhanced pathogenic rôle. This may serve to explain the ulceration of the mucosa of the sinus found in subjects that have suffered for some time. It further explains the notorious fact that the free access of air (oxygen) to the inflamed sinus is one of the most helpful therapeutic measures.

Symptoms of the Acute Form. When the disease is traumatic the first symptom is usually a hæmorrhage from the nose, the blood being of a bright crimson. Respiration is hurried, and appetite diminished, yet rumination may be imperfectly performed. The bleeding may be repeated for days in succession, but the ox is still capable of work. On the fifth or sixth day there is complete anorexia, rumination ceases, the head sinks resting on the manger or soil, the ears droop forward and downward, and may be swollen. The head inclines to the affected side, the corresponding horn is intensely hot, and the eyes are closed. Light percussion of the forehead on the affected side gives pain, and the sound elicited is flat and dull as compared with that from the opposite side. The temperature of the body rises 2° or more, the pulse becomes frequent, full and hard and the impulse of the heart abnormally strong. Costiveness, partially suppressed and high colored urine, and dry hot muzzle betray the fever. Unless relieved the chronic form may supervene.

Symptoms of the chronic form. When this comes on slowly, working oxen get emaciated, lose appetite, have the eyes dull and sunken, and the lids drooping, the coat rough and staring and the skin harsh, dry, and lacking in pliancy, the head is carried low when out of the yoke and, after shaking the head and sneezing, a glairy, slightly fœtid matter escapes from the nostril. The breath is fœtid and appears to be offensive to adjacent cattle.

This may continue for months with no other change than a

more constant nasal discharge, and increasing emaciation and weakness.

Prognosis. This is favorable for the acute disease at the outset. But if no relief is furnished it is liable to go on to a fatal issue. Even the chronic form is curable unless the subject has already become hopelessly weak and debilitated.

In *fatal cases* the sinuses are found to be filled with a glairy fluid and the mucosa thickened and raw or ulcerated. There may be enlargment of the pharyngeal lymphatic glands, and there may be attendant pharyngitis.

Treatment. The patient must have absolute rest and cold water irrigation or icebags applied to the head. The bowels may be opened by a saline, or a diuretic administered. If the head is persistently dropped it may be kept moderately elevated by a halter tied to a higher point. Should there be no relief at the end of twenty-four hours, no time should be lost in securing free admission of air to the cavity. Cruzel advises to saw off the horn at its base, as the one certain method of securing prompt improvement and speedy recovery. If a horn and its bony support have been broken off they should be at once removed and the head turned up to evacuate the accumulated glairy fluid from the sinus. From an apparently hopeless condition a few hours will suffice to restore an appearance of good health. If the horn has not been broken and it is desirable to save it, the bone may be trephined in front of the root of the horn and the liquid evacuated, or less effectively and more painfully the horn may be bored at its root by a large gimlet.

If no hæmorrhage has taken place and if active treatment has been adopted at the outset recovery may be complete in two or three days, but if the disease has been ushered in by a hæmorrhage which recurs several days in succession, amputation of the horn or trephining will be demanded. In chronic cases this should be followed by astringent and antiseptic injections and a blister may be applied to the throat or the side of the neck. In these cases too a course of mineral tonics is desirable.

CHRONIC CATARRH OF CATTLE.

Catarrh, chronic, summer aggravation, thickened, roughened, mucosa, discharge, twigs in nose. Question of parasitism. Treatment, remove causes, antiseptic astringents.

A remarkable form of chronic catarrh with summer aggravation exists in some of the hilly districts of New York but has not received such study as to enable us to state its true nature.

One or two in a large herd will have a loud snuffling breathing, which may subside so as to be entirely overlooked in winter, but reappears when put to pasture in the spring and continues in a marked form throughout the warm weather and until after the animal is returned to winter quarters. There appears to be little or no fever nor constitutional disturbance except what comes from the obstructed breathing, and the yield of milk may be unchanged. The symptoms would indicate a purely local disease. Yet so few are attacked out of a herd that it cannot be actively contagious.

On close examination the nasal chambers are found to be narrowed, there is manifest thickening of the mucosa, and its surface feels rough and uneven, with miliary elevations. There is of course more or less glairy discharge. If the examination is made about midsummer, the finger introduced into the nose will usually detect the ends of twigs that have been introduced into the cavity and broken off. When withdrawn these may prove individually from four to eight inches long, and some force may be required to extract them. In winter these are often absent, having been apparently dropped one by one. The absence of these sources of irritation sufficiently accounts for the manifest improvement during the colder months. In spite however of the winter remissions the disease tends to a steady advance year by year. While nothing definite is known of its pathology, the occurrence of this disease in given localities, its manifestly local nature, and its persistence when once established would suggest enquiry as to the possible existence of parasitism, bacteridian or otherwise.

Until further discovery treatment can only be of a general nature. Removal of the foreign bodies from the nose, pasturage where there is no brush to replace them, soiling when clean pastures cannot be found, and the use of astringent and antiseptic agents by insufflation or injection would be indicated.

MALIGNANT CATARRH OF CATTLE.

Local causes. Debilitation. Polluted air. Poor diet. Symptoms, gastrointestinal, fever, lacrymation, turbid aqueous, photophobia, congestion of mucosæ, generally disturbed circulation, breathing, depression, heat of forehead, buccal petechiæ, epithelial desquamation, abrasions, ulcers, abortion, albuminuria, local swellings, shedding of horns, dropsy, dyspnœa. Lesions, in nasal mucosa, subcutem, cerebral, dark blood. Prognosis. Treatment, antiphlogistic, laxative, diuretic, tonic, locally steam, antiseptic, astringent, trephining.

This disease occurs chiefly in cold damp marshy localities where the vital power is impaired or in cold situations exposed to severe north and east winds. In the wet cold seasons of spring and autumn it is especially prevalent. According to *Rychner* it rarely attacks old cows but prevails among young cows and oxen. In the south of France on the contrary it appears chiefly in the hot season (June and July) and is attributed to suppressed transpiration. It prevails especially however in herds kept in small filthy stables, low in the roof, hot, close and badly aired. (Festal). In New York it appears in cattle on black muck pastures and in Minnesota on the dried up ponds.

Symptoms. Diarrhœa is a common premonitory symptom arising from the irritation of the intestinal canal as it is soon followed by some degree of costiveness, the dung becoming dark colored, firm and scanty. Diarrhœa reappears later. The coat stares or the beast actually shivers; the head is depressed; the roots of the horns and the forehead are hot; the eyes are sunken, swollen and red, suffused with tears, turbid in their anterior chamber (aqueous humor) and intolerant of light; The muzzle dry and hot; the mouth hot but moist with abundant saliva; the mucous membranes of the mouth, nose and vagina have a bluish red color; the pulse is rapid and more or less full or hard; impulse of the heart weak; the breathing is accelerated, the respiratory sound is heightened in intensity and a cough is frequent. Temperature 104° to 107° F. The urine is scanty and high colored. The surface of the body is alternately hot and cold, and after some time a watery fluid begins to distil from the nose.

At the end of twenty-four hours the symptoms are intensified

or altered. The eyelids are more swollen and the flow of tears more profuse; the nasal discharge becomes slimy, and streaked with blood, and accumulations take place in the frontal sinuses as indicated by the increasing heat of the forehead and the dullness on percussion. In the mouth appear dark red spots, from blood extravasation, over which the epithelium sloughs off leaving raw unhealthy sores. The appetite entirely fails; dung and urine are passed painfully and with effort, and abortion frequently takes place in pregnant cows. The urine is albuminous with cell forms, and casts. The limbs appear rigid and it pains the animal to move.

From the fourth to the sixth day the ulceration appears on the mucous membrane of the nose which has often a claret color, and the nasal discharge becomes again more watery and irritating. The muzzle is swollen and a dropsical infiltration appears beneath the jaws which extends along the neck to beneath the thorax and into the limbs. Portions of the nasal mucous membrane now slough off, and similar sloughs are often seen on the skin of different parts of the body; the secreting structures of the horns and hoofs even participating so that these are easily detached or shed. Saliva flows profusely from the lips, a fetid watery diarrhœa succeeds the constipation, the dropsy becomes nearly general and death occurs on the eighth, ninth or tenth day of the illness. Convulsions and symptoms of suffocation may precede death.

In a *post mortem* section the principal lesions are found in the nasal cavities and skin. The areolar tissue in both is the seat of an abundant serous infiltration, which has taken place into the deeper layers of the skin as well, rendering it thick, hard and unyielding. Besides the sloughs and ulcerations on the skin and mucous membranes, false membranes have been met with, on the lining membrane of the mouth and air passages. The ulcers in the nose have in many cases reached the bone, and from the abundant infiltration and softening, the membrane is easily stripped from the walls of this cavity and of the sinuses. The general infiltration appears to have reached the brain, which is described as softened and having an undue amount of liquid in its cavities. The blood contained in the vessels is dark colored and numerous patches of extravasation are visible on the mucous and serous membranes as well as in the interior of organs.

Unless the malady can be controlled in its early stages it usually proves fatal. Patients that recover after it has been well developed at times retain its effects in permanent blindness or palsy of the hind limbs.

Treatment. Early and vigorous antiphlogistic measures are strongly recommended by French and Italian veterinarians. Gelle and Ercolani advocate the most copious bleedings. Festal insists that all other measures are useless when this is neglected. Before adopting free sanguineous depletion the history of his practice was a record of deaths, whereas later his losses were in cases where from a failure to recognize the disease at the outset, from the existence of diarrhœa, from the patient being pregnant or from a fear that the milking properties might be impaired, bleeding was deferred. He pushed the bleeding to the extent of causing acceleration of pulse, quickened breathing and heaving of the flanks, to effect which sixteen pounds had to be abstracted on an average. If this were done early the engorgement of the muzzle had usually greatly diminished if not entirely disappeared in the course of seven or eight hours thereafter. The alleged benefit is probably largely due to elimination.

Less heroic treatment is now generally adopted. An active purgative (one and a half pounds Epsom salts) may be given even though apparently contraindicated by the premonitory diarrhœa, and a further useful derivation may be obtained by applying active friction or even stimulating embrocations to the legs.

Steam with or without sulphur dioxide may be inhaled as for ordinary *coryza* and cold water or ice kept applied to the forehead.

Nitre in ounce doses daily or liquor of the acetate of ammonia in three ounce doses may be given after the purging has ceased. Or drachm doses of hydrochloric acid with bitters may be given thrice a day in at least a pint of water.

Where the nasal discharge persists after the subsidence of the other symptoms the sinuse should be trephined in front of the horn, and tepid water and mild astringent and antiseptic lotions injected until a healthy action has been established. Change to a dry, well drained pasture or building is desirable for both treatment and prevention.

CONTAGIOUS DISEASES OF THE NOSE.

These are omitted here to be treated under that heading.

PARASITIC DISEASES OF THE NOSE.

Among these may be named:

LEECH BITES.

Form of wound, leeches in posterior nares, discharge of blood, mucus, sneezing, snorting, dysphagia, anorexia, unthriftiness, anæmia. Treatment, removal, sodium chloride, tar fumes, ether on sponge.

Though it more commonly attacks the mouth and lips yet the leech (Hirudo Decora) will sometimes fasten itself inside the nose when that is plunged in water. Its bite is to be recognized by its triangular shape. When taken in by the mouth it may fasten itself in the posterior nares where it is difficult to recognize its presence. In the anterior nares it can be readily discovered and removed, but in the posterior nares it may maintain its hold indefinitely. There appear in the nasal discharge streaks or clots of blood which may also show at the corners of the mouth. Sneezing, snorting, and difficulty of deglutition, may draw attention to the trouble, and in protracted cases signs of anæmia, inappetence, unthriftiness and general weakness. The most effective treatment is to remove the leech with the fingers, but as they cannot always be reached in this way, an injection of a strong solution of common salt may be used. Blaise succeeded by burning tar under the nose twice a day, and Louvigny by introducing a staff bearing a sponge soaked in ether.

COCCIDIAN CATARRH IN RABBITS.

Coccidia in rabbit's nose, etc., inflammation, fatality. Treatment, sulphur dioxide.

Zurn describes a contagious catarrh of rabbits caused by the presence in the mucosa of the nose, pharynx, Eustachian tubes and middle ears of myriads of coccidia. They create acute irritation and prove fatal in many cases. Embedded in the mucosa they are difficult to reach with medicinal agents, yet the free parasites may be destroyed by frequent fumigations with sulphurous acid, or by spraying or injecting the nose with its solution.

LARVA OF ŒSTRUS OVIS (GRUB) IN THE NASAL SINUSES OF SHEEP.

Season of attack by fly, hibernation, botfly of sheep, mode of attack, embryo, defensive acts of sheep, habitat of larva, mature larva, its exit, chrysalis in soil, its transformation. Symptoms, sneezing, snuffling, rubbing of nose, lachrymation, unsteadiness, discharge, respiratory digestive and febrile disturbances, emaciation, septic action. Lesions, larva, congestions of mucosa, brain. Treatment, warmth, errhine, parasiticides, mechanical extractors, trephining, injections. Prevention, newly turned furrow, quicklime, tar. Other larva in nose.

Sheep are especially subject to the attacks, in summer and autumn and in warm sunny barns even in winter, of the Œstrus (Cephalemia) Ovis, the larva of which hibernates in the nasal sinuses or turbinated bones.

The **sheep-bot-fly** is only about four lines in length, of a light yellowish or slightly brownish gray hue, hairy, with dull black transverse lines on the upper surface of the thorax, and a lighter color on the abdomen where the black lines are more broken. The transparent, colorless wings extend beyond the body: winglets are long and cover the poisers: abdomen is formed of five rings.

They appear during the whole summer hiding away in walls, stumps and grass, unless when pairing or pursuing the sheep to deposit their young. The mode of attack is difficult to follow on account of the small size, gray color and rapid flight of the fly and fear and shyness of the sheep. It cannot be doubted, however, that they approach and drop on the margin of the nostril, the larva previously hatched from the egg. The old authors describe the deposition of the egg on the margin of the nostril and its prompt hatching by the animal heat, but the observations of Brown, Kelly, Cockrill, Riley and Ormerod abundantly prove that the fly is viviparous. Cockrill obtained no less than 300 live hatched larvæ from one fly caught while pursuing a sheep.

The sheep seek to avoid the fly by resorting to dry dusty roads where they lie with the nose close to the ground, or they stand with the nose close to the soil and between their fore legs. At

other times they will collect in a dense phalanx with their heads directed toward the centre of the mass and held low so that the fly cannot reach them. The moment the fly touches the nose they shake the head, stamp with the feet, and gallop off with the nose close to the ground, looking from side to side to see if the fly pursues and frequently smelling at the grass as if apprehensive of other flies hidden there. If such appear they instantly turn and scamper to other parts of the field or take refuge in a dry dusty place or gravel bank.

The **young larva** when deposited on the nostril speedily makes its way up and takes refuge in the cavities of the turbinated bones and the frontal and maxillary sinuses, where it passes the winter feeding on the mucus and the purulent discharges determined by its presence. When mature it leaves the nose and assumes the chrysalis form in the soil.

The **mature larva** is narrow anteriorly, broad behind: its upper surface is prominent and rounded, lower surface flat, and furnished at the anterior of each ring after the third, with a series of pointed tubercles or spines: the cephalic end bears the buccal organs directed downward, and bearing two great hooks connected with the hard framework of the pharynx and recurved downward, backward and outward; mouth small; antennæ thick and short placed above the buccal organs: the inferior part of the last ring projects beyond the upper portion and is furnished with two nodules with intervening spines: pentagonal patches of stigmata on the last ring: very small anterior stigmata between the first and second rings. The color is white with brown spines, stigmata and transverse striæ. Length seven lines to one inch.

When dropped from the nostril in the course of summer they pass into chrysalis in one or two days; and after a residence of six or eight weeks in the soil emerge as the perfect fly.

Morbid Symptoms Caused by the Larvæ in the Head. Grub in the Head. These bear a close relation to the number of larvæ present. If there are only two or three no trouble may result. If many there is muco-purulent discharge from the nose, sniffling breathing, frequent sneezing and snorting expelling mucus and even blood; shaking of the head; rubbing of the nose on the fore legs or other objects; weeping eyes; and occasional unsteadiness of the gait.

In the worst cases the respiration becomes sighing, wheezing or even snoring; the mouth open; head pendent; appetite fails; a dull, apathetic condition ensues with grinding of the teeth rolling of the eyes, and, rapidly advancing emaciation.

Fatal cases are not uncommon but most frequently the larvæ reaching maturity are dropped and health is promptly re-established. Septic poisoning from decomposition of dead larvæ and debris is a dangerous complication.

Lesions. These consist in the presence of the larvæ in the sinuses, with violently congested, purple ulcerated mucous membranes and collections of pus. The mere presence of the grub is not conclusive as the majority of the sheep harbor two or three from October to June.

Treatment. It is advised to place the sheep in a warm building to encourage the parasites to come out of their recesses and then introduce some agent to destroy them or to induce their expulsion by sneezing. The value of the hot building is probably hypothetical unless the larvæ are approaching maturity. The following agents are used:—moderately strong solutions of salt, vinegar, carbolic acid, creosote (1 part to 100 parts of water), or carbonate of ammonia, lime water, snuff, or even such irritants as quick lime, oil of turpentine or hellebore. These last must be used with caution as they are liable to induce fatal inflammation of the air passages though no larva is present.

By passing a feather up the nostril twisting it round and then withdrawing it some grubs can usually be withdrawn and there is no harm in first dipping the feather in some of the milder agents mentioned above. But the larvæ in the sinuses can never be reached in this way.

In dangerous cases it is best to trephine the outer plate of bone covering the frontal sinuse and wash out freely with tepid water, lime water, or benzine. The operation may be performed close in front of the root of the horn if there is one, or to the inner side of the lower part of the eye if there is not. A semi-circular flap of skin is to be turned upward and backward sufficiently large to allow the use of a trephine $\frac{1}{3}$ inch in diameter, which is to be used as for *Cœnurus Cerebralis*. The opening being made the sinuse is to be syringed freely for some time until the parasites

come from the nose in the stream of liquid. The wound heals very promptly. In the absence of a trephine use a gimlet.

Prevention. Some turn up a furrow in the pasturage, in which the sheep may burrow their noses and evade the fly, others lay down quicklime in covered boxes which has the further advantage of inducing sneezing and favoring expulsion of the entering parasites. But perhaps the best plan is to procure a log and bore a number of holes in it with a two inch augur; place salt in the holes and smear their margins with tar, and renew it often. The sheep then takes a protective dressing with every lick of salt.

Œstrus Purpureus (Brauer) is a species which infest the nose, etc., of the Syrian sheep, and **Cephalemia Maculata** (Wedl) one which infests the nasal chambers of the Egyptian buffalo and camel.

NASAL CATARRH IN DOG AND HORSE FROM LINGUATALA (PENTASTOMA) TAENIOIDES. RHINARIA TAENIOIDES.

Form. Family. Habitat when mature, and immature. Development. Symptoms, sneezing, discharge, irritability, ill-temper, shaking the head, rubbing nose, parasites in discharge. Treatment, injections, trephining. Prevention.

This parasite has a worm-like body, but is closely allied to the mites and belongs to the Arachnida. It differs from the mite in having but four short limbs retractile and protractile and furnished with sharp claws. The body is thickest toward the anterior end and prolonged and narrow posteriorly ; marked by about 90 rings ; head rounded off abruptly, mouth broadly open, with a horny lip ; integument with numerous openings or stigmata (respiratory) ; *male* 7 lines long by a line broad in its anterior part genital orifice on the front part of the abdomen in the median line ; *female* 3 to 4 inches long, by 3 or 4 lines broad anteriorly : genital opening at the end of the tail. *Reproduction* oviparous.

Habitat. Nasal chambers and sinuses of the dog, wolf, goat, and horse.

Pentastoma Denticulata. The young partially developed *P. Taenioides.* Has all the rings except the two first, garnished with fine sharp recurved spines ; legs more slender with accessory booklets ; length 2 lines ; breadth $\frac{1}{2}$ line.

Habitat. Cysts in the lungs, liver, mesenteric glands, etc., of the hare, porpoise, goat, sheep and other mammals, not excepting man.

Development. Leuckart found that the adult Pentastomata copulate in the nasal chambers, as many as half a million of eggs being fertilized in a single female ; that these eggs are discharged with the nasal mucus and falling on vegetables are taken in by herbivora ; their shells are digested and destroyed in the stomach, and the liberated embryos perforate the intestinal walls and encyst themselves in various organs. The encysted embryo varies from $\frac{1}{500}$ to $\frac{1}{40}$ inch in length, is rounded and blunt anteriorly but very thin posteriorly with the tail slightly curved toward the ventral aspect. It is several months before the feet, cutaneous spine and generative organs are developed, and during this period it

undergoes several moultings. Finally it leaves its cyst and may live free in the cavities in the body of its host, and if it does not escape from the body it finally constructs a new cyst and then dies. If the host is eaten by a carnivorous animal the liberated pentastomata reach the nose either from the lips or pharynx and in a few months more acquire their complete development. They must reach the nose of the horse by their presence in the food or water.

Symptoms Caused by the Pentastoma. No morbid symptoms have been traced to the young encysted condition of the parasite. Yet it would not be surprising if their presence in large numbers in the mesentric glands and liver should give rise to troubles of assimilation, sanguification, biliary secretion and the like. Frerichs says they are more common in the human liver in Germany than echinococcus, but adds that they have no clinical importance.

In their mature condition however they cause considerable irritation and nasal discharge when present in large numbers. In dogs there is running from the nose the discharge containing an abundance of the ova, restless, fretful habits, sometimes a morbid readiness to bite, frequent shaking of the head and rubbing of the face.

The *treatment* would be to trephine the sinuses and inject lotions impregnated with creosote, carbolic acid or naphtha. From the danger to man of becoming infested it is important to ascertain the true nature of any nasal discharge of the dog especially in countries like Germany and Egypt in which this parasite is common.

Prevention. Deny raw offal of herbivora to dogs.

AFFECTIONS OF THE THROAT.

Sore throat, Angina, Cynanche—is a generic name applied to a series of inflammatory affections of the various structures about the throat. If the *larynx* is specially inflamed the disease is known as *laryngitis*, if the *pharynx*, as pharyngitis, if there are exudations forming false membranes it is *croupous* or *diphtheritic*, or if associated with some general febrile affection, it takes its name accordingly, *influenza, strangles, distemper*, or *scarlatina*, as the case may be.

LARYNGITIS IN THE HORSE.

ANGINA LARYNGITIS, CYNANCHE LARYNGEA, ETC.

Causes, mechanical, cold, irritants, extension, diet, close stables, infectious disease. Symptoms of acute form, head extended, throat swollen, tender larynx, cough, in early stage, after exudation, wheezing in inspiration, dysphagia, fever, œdema glottidis, spasms, dyspnœa, successive discharges. Lesions, tumefaction, softening, friability, redness (ramified or not), erosions of mucosa, œdema. Course, duration, sequelæ, cough, roaring. Subacute form, chronic form, in old debilitated animals, in those reined too tightly, in those which perspire with difficulty or bear heavy coats. Symptoms, local, in breathing, cough, effect of cold air, or water or of dust. Sequelæ, ossified cartilages, roaring, emphysema, bronchiectasis. Treatment, hygienic, soothing, sheepskin, compress, poultice, mustard, sulphur dioxide, laxative, neutral salts, expectorant, sedative, derivative, tracheotomy, with trochar and cannula, with scalpel, tracheotomy tubes. Insufflation, injection. In chronic laryngitis, electuaries, mustard, derivatives, astringents, caustics, tonic inhalations.

Causes. These are the ordinary causes of chest diseases. As special causes may be noted severe compression of the larynx as in roughly and repeatedly *coughing* an animal; the sudden contact of piercing cold air, of irritant gases, powders or liquids with the membrane, and the rapid, forcible and continuous current of condensed air through the glottis during severe exertion.

Among the general causes the most fruitful are the high feeding, hot, close stables, heats and chills, and other circumstances attendant on domestication. Laryngitis may be an extension from coryza, pharyngitis, bronchitis or pneumonia. It may further be but a local manifestation of influenza or strangles, contagious pneumonia, etc. The disease is *acute*, *subacute*, or *chronic*.

Symptoms of acute form. All acquainted with horses can recognize the general symptoms of sore throat. The nose is elevated and protruded to avoid compression of the larynx; it is carried stiffly for the same reason. There is some swelling around the throat or beneath the root of the ears. If the cartilages of the larynx are compressed between the finger and thumb, or if pressure is made in the median line below upon the connecting

crico-thyroid membrane the patient instantly coughs and throws up the head to avoid a repetition of the suffering. This tenderness of the larynx to touch is peculiar to laryngitis and serves to distinguish it from pharyngitis. The cough is at first very hard and painful and only gives way to a soft mucous type when a free mucous exudation puts an end to the tense, thickened and dry state of the mucous membrane. The inspiratory act is accompanied by a whistling or deep bass sound, particularly after the slightest exertion. This may be heard at times during expiration as well, though not invariably so. Sometimes the animal drops the food from his mouth after mastication, because of the pain attendant on swallowing, but this is really a symptom of coexisting pharyngitis, and its absence implies the nonexistence of that complication. There is usually a slight pasty swelling between the branches of the lower jaw.

There are besides the general symptoms of fever more or less marked, such as increased temperature, accelerated pulse, red injected eyes and nose, slightly hastened breathing, the expiration being effected by a double lifting of the flank as in broken wind, etc.

In two or three days in favorable cases exudation takes place from the mucous membrane, the cough becomes softer and less frequent, the local tenderness decreases and the general symptoms subside.

If otherwise the symptoms may become more intense, and breathing may get loud and difficult in connection with thickening and rigidity of the mucous membrane, or a serous exudation into it and beneath it (œdema glottidis) which by closing the glottis renders breathing almost impossible. The same distressing symptoms may arise from spasm of the larynx excited by the inflammatory action. As arising from thickening or infiltration of the membrane these symptoms may come on comparatively slowly, but in the case of spasm they appear suddenly and have periods of intermission, reappearing on succeeding days and usually at the earlier part of the night. In such circumstances the loud, noisy breathing is heard at a considerable distance, the horse stands obstinately still, the fore feet apart, his elbows turned out to allow a firm action of the chest, the flanks working laboriously, the head low, the nose protruded, the nostrils widely

dilated, the mouth open, the eyes standing out from their sockets red and wild looking, and the face constrained and pinched, the whole expression being that of intense agony from impending suffocation.

Lesions. In cases where death has supervened, perhaps in connection with another disease, the laryngeal mucosa, especially on and above the glottis, is soft, tumid, friable, with ramified or uniform redness and petechial spots. The epithelial layer may be softened, disintegrated and shed, leaving pointed or larger erosions, which are, however, usually superficial. In case of œdema glottidis the mucosa and submucosa are thickened by an abundant exudate which may extend to the connective tissue outside the larynx as well. In aggravated cases there may be dark red or brownish red discolorations of the mucosa.

Course, Duration. Fortunately these aggravated forms of the disease are rare and unless the patient perishes during such an attack or the inflammation extends down toward the chest, laryngitis rarely proves fatal. Its duration is from twelve to fifteen days. Its extension to the lungs may be suspected when the extreme tenderness of the throat subsides without any corresponding improvement in the health. Examination of the chest will then rarely fail to detect the presence of disease.

But although sore throat is rarely fatal its effects are not unimportant nor trivial. It occasionally merges into a chronic form, with a hacking cough, tenderness to pressure and an increased liability to other diseases of the air passages. More frequently it is followed by wasting and fatty degeneration of the laryngeal muscles and the horse becomes a confirmed *roarer*. In all cases it leaves a greater susceptibility to a second attack.

Sub-acute Laryngitis. This form has been chiefly seen in young animals and up to eight years old. At the outset its symptoms are moderate but as it is usually associated with serous effusions in and around the mucous membrane the symptoms above mentioned as indicating imminent danger of suffocation may suddenly appear and life can only be preserved by opening the trachea.

Chronic Laryngitis. This may follow the acute form or it may come on independently and by slow degrees. It may accompany nasal catarrh, or chronic bronchitis. Old animals which have had heavy draught work and repeated attacks of sore

throat, are frequent subjects of it, and as Fergusson has pointed out it is most prevalent among horses whose throats have been compressed by the inconsiderate use of the bearing rein. Reynal has observed it often in horses that are sweated with difficulty, and in those which remain long wet from the length and thickness of their winter coats.

Symptoms. When acute laryngitis passes into the chronic form all the symptoms subside except a slight nasal discharge, the cough, tenderness of the larnyx, and *roaring*. The cough is dry, short, and hacking, rarely soft, and is heard mainly when the animal feeds, when he leaves the hot stable for the cold air, and after drinking cold water. During exercise, it is equally excited, the cough becoming harder, and the horse extending his head and neck as if to disengage some body from its throat. The subject may in nearly all other respects maintain the appearance of vigorous health.

Course, etc. This disease is liable to prove obstinate and if of old standing, often incurable. Unless checked, the continued congestion and irritation of the larynx, the frequent, hacking cough, and the consequent violent distension of the lungs bring about extensive and irreparable structural changes. Among these may be mentioned ossification of the cartilages of the larnyx; paralysis of the left laryngeal nerve with wasting of the muscles to which it is distributed, and *roaring*; dilatation of the bronchial tubes, and permanent distension and rupture of the air cells (emphysema, broken wind, heaves).

Treatment. Acute form. Unless in the very mildest cases unaccompanied by fever, repose is essential. If available, a roomy, clean, dry, and airy loose box should be allowed, care being taken to avoid draughts of cold air and to secure a soft equable temperature neither too hot nor too cold. Blankets should be used and even flannel bandages applied loosely to the legs if the weather is cold, or, if there is any tendency to chills and shivering. The nostrils must be steamed as directed for *coryza*. A piece of sheepskin with the wool turned in may be tied around the throat and up to the ears. In very acute cases a linseed meal poultice or wet pack may be applied to the throat, while in the milder forms, a mustard poultice or a lotion of Spanish flies or other stimulant may be employed. Unless the malady has an epizootic type, with

prostration and a weak, rapid pulse, the bowels may be opened by a laxative (3 or 4 drachms, aloes), and the water or gruel the animal daily drinks should contain ½ to 1 ounce nitre. As an expectorant the patient may take salammoniac 1 oz. daily in the drinking water, or this agent may be evaporated from a clean chafing dish every two hours and inhaled. Or he may take carbonate of potash or soda, or iodide of potassium. If the cough is troublesome, half a drachm of Dover's powder may be given thrice a day or 1 grain chloride of apomorphine every hour. Bromide of potassium or sodium may also be resorted to. Inhalations or spray of sulphurous acid, or vapor of oil of turpentine and insufflations of calomel may benefit as local applications. The diet must be confined to sloppy bran mashes, cut roots, or boiled barley, or oats. Hay should be withheld in the more acute cases until improvement appears. Under treatment such as the above and even without the medicinal part of it, the great majority of cases will do well.

In cases attended with high fever with strong full pulse and bright red nasal membrane, the purgatives and diuretics are especially called for, and the former should have their action encouraged by frequent hot water injections. Twenty drop doses of the tincture of aconite repeated four times a day, or ten drops every three hours, will be further useful.

When the symptoms are of such a type as portend the access of paroxysms of threatened suffocation, bleeding has been strongly recommended, but unless resorted to in the first twenty-four or forty-eight hours is rarely admissible. Also in weakened constitutions and when the fever is of a low type, with small, weak pulse and general dullness and prostration, the temporary relief obtainable from blood-letting will not often counterbalance the danger of increasing weakness, and the loss of recuperative power. In all such cases the application of a strong mustard poultice for several hours in succession, until an abundant effusion has taken place, into the skin and beneath it, has often the best effect by virtue of its depletive and derivative action. Active friction of the limbs to improve their circulation and increase their temperature is also useful.

Tracheotomy. When suffocation becomes imminent not a moment must be lost in performing *tracheotomy*. This operation

is always available in threatened suffocation from obstruction to the passage of air in the nostrils and throat.

Different methods of opening the windpipe have been resorted to. One is by means of a cannula and trochar at least three-fourths of an inch in diameter and about five inches long and with two large oval orifices in the middle of the cannula, and on opposite sides. This is made to transfix the windpipe with its investing skin and muscles from side to side in the middle of the neck, care being taken to pass it in the interval between two adjacent cartilaginous rings. The trochar is now withdrawn and the orifice in the cannula corresponding to the interior of the windpipe, the animal is enabled to breath freely through the tube. The cannula has only then to be secured in its place by a tape carried round the neck.

The more common plan is by introducing a tube through a circular opening made in the trachea. For this operation are needed, sissors, knife with a thin narrow blade, needle and thread, and tracheotomy tube. The common tube is about an inch in diameter, four to five inches long bent upon itself so as to fit into the trachea, and furnished with a flat shield to slits in which cords may be attached to fix it in its place. A second variety is only long enough to reach into the windpipe. It is provided with a flattened shield externally and from its inner extremity projects downwards at right angles a plate curved so as to adapt it to the form of the interior of the trachea. There is an additional plate to fit into the upper part of the tube, provided with two lips projecting from it at right angles; the outer lip is screwed to the shield after the tube has been introduced into the wind pipe and the inner lip is thus fixed inside the ring of the trachea, immediately above the opening and effectually prevents any displacement of the tube.

In operating the animal is kept standing with the head as nearly as possible in the natural position. The hair is removed from the skin beneath the windpipe between the middle and upper thirds of the neck. The skin having been rendered tense, (without displacement) by the fingers and thumb of the left hand, an incision is made in the median line from above downwards, for about two inches and is carried through the muscles so as to expose two rings of the trachea. The needle and thread are passed through

the membrane connecting the two rings, and with the knife a semicircular piece of cartilage is cut from each of the two adjacent rings. The thread in the connecting membrane prevents them from being drawn in by the rush of air. It only remains to introduce the tube and fix it in position.

Not only does tracheotomy obviate immediate danger of suffocation, but by removing the source of irritation in the continuous and forcible rush of air through the narrowed and inflamed tube, and in securing for the blood a freer aeration and a purer constitution it often induces a rapid change for the better in the character of the inflammatory action. The wound may be daily cleansed and dressed with sodium hyposulphite.

Some veterinarians following the example of Bretonneau and Trousseau have treated sore throat from the first by what is called the abortive treatment. For this purpose a long whalebone prob with a pledget of tow firmly attached to its end and covered with powdered alum is introduced through the mouth into the pharynx and larynx even. Violent paroxysms of coughing are induced, but cures are affected in from two to five days. Under Delafond's treatment calves and foals recovered in twenty-four hours. A more modern method is to inject a solution by means of a hypodermic syringe inserted between the upper rings of the trachea.

Milder treatment such as the inhalation for an hour several times a day, of the fumes of burning sulphur and water vapor will be found generally successful. The air should be impregnated with sulphur fumes only so far as can be breathed without inducing coughing on the part of the patient. Such measures should not divert attention from the necessity for general care, a control of diet, clothing, air, the state of the bowels, nor from local external applications to the throat.

Treatment of Chronic Laryngitis. The patient should have a loose airy box with an equable temperature. The avoidance of work and exposure must be sought for the time. Green food, cut roots, boiled grain, or bran mashes, with little or no hay, or other dry food, must be given. The bowels must be regulated. An electuary compound of linseed meal, molasses, and a drachm of belladonna extract to every tablespoonful of the mixture, may be given to the extent of a tablespoonful smeared on the inner side of the cheek twice daily. A mustard poultice to the throat has

often a good effect. Light firing over the larynx is sometimes beneficial.

If secretion is defective and cough hard and dry chloride of ammonium, carbonates or bicarbonates of soda, potash or ammonium or borax, in solution or in gaseous form, may be given, the various bitters being at the same time drawn upon as tonics. If secretion is excessive, with a loose gurgling cough, astringents are indicated like ferric sulphate or chloride, (½ dr.), or they may be applied as spray: alum or iron alum five grains to the ounce, zinc sulphate or sulphocarbolate two grains to the ounce, silver nitrate one-half grain to the ounce. These may be introduced through the nose with the head elevated, or in small genera through the fauces. Tar, oil of tupentine, creosote, carbolic acid or eucalyptol may be inhaled from hot water.

PHARYNGO—LARYNGITIS IN CATTLE.

Susceptibility. Causes, symptoms, cough, salivation, wheezing, lachrymation, muzzle dry, tender throat, dysphagia, disturbed innervation and circulation, hyperthermia. Duration. Abscess. Treatment, laxative, local treatment, lancing

Cattle are less subject to sore throat than horses. The skin appears less sensitive to the influences of cold and heat. The ox is not subjected to the same severe exertions. It is rarely seen to sweat, the moisture passing off from the surface as insensible perspiration only. The disease, however, recognizes the same causes as in the horse, though these are manifestly less injurious.

Symptoms. The disease usually affects at once the larynx and pharynx so that the symptoms are somewhat modified. In the simplest form there is only a small, hacking cough, a flow of saliva from the mouth and some loss of appetite but no fever. In more acute cases the breathing is loud and wheezing, the cough, soft and rattling, is followed by a free discharge of mucous from the mouth, the nostrils and eyes are red, the muzzle dry, the pulse accelerated and full, the throat tender to the touch, and swallowing difficult, part of the food and drink being rejected through the nose. If the larynx is chiefly involved the loud

noise in breathing is the predominant symptom and sometimes almost the only one.

Course, etc. The cough and other symptoms are usually moderated with the access of the abundant secretion on the second or third day, and recovery is perfect on the eighth to the fifteenth. If abscess results, to which there is a far greater liability than in the horse, it may not burst till the twentieth day and the case is correspondingly protracted. This should be carefully distinguished from the deposits of tubercle which take place around the throat in cattle. In rare cases the disease becomes chronic.

Treatment does not differ from that advised for the horse except in the greater safety of purgatives which must in this case be saline (Epsom or glauber salts one to two pounds), and in the greater ease with which local treatment can be applied owing to the shortness of the soft palate. When abscess forms it must be encouraged by poulticing and opened with the knife or lancet as soon as it points.

LARYNGITIS IN SHEEP.

Infrequency. Causes, damp lands, storms, close buildings, clipping. Symptoms, cough, sneezing, discharge, snuffling, oral breathing, tender throat. Treatment, ventilation, warm water vapor, sulphur dioxide, salines.

Sore throat is fortunately even more rare than in the larger ruminants. It occurs chiefly where this animal, constituted to feed on the dainty grasses of the dry mountain side, is kept on cold, marshy ground and exposed to frequent cold, wet blasts. Sheep suffer also from hot, close, filthy buildings in winter, and from unseasonable clipping.

The *symptoms* are frequent coughing and sneezing, running from the nose, working of the jaws, and breathing through the open mouth as being easier than through the plugged nostrils. The larynx is tender and may be swollen.

Treatment is usually confined to ventilation and cleansing of the fold, frequent fumigations with water vapor from the spout of a boiling kettle, and with sulphur fumes, and giving tepid farinaceous gruels or mashes containing sulphate of soda in the daily proportion of two pounds to each hundred head of sheep. Sal ammoniac may be given in food or drinking water.

LARYNGITIS IN PIG.

Frequency. Causes, wet, cold pens, exposure, withholding liquids. Symptoms, prostration, dullness, cough, fever, swollen throat and neck, dyspnœa, dark mucosa, sloughing of epithelium and epidermis, general petechiæ, fœtid breath, great prostration. Lesions, gangrenous patches on pharynx and fauces, ulcers, infiltrations. Treatment, hygienic, dietetic, emetic, laxative, poultice, bandages, locally, astringent, antiseptic, caustic, tonic.

Sore throat is common in some localities when pigs live in herds.

Causes. Chiefly faulty hygiene. Exposed, cold and wet piggeries, cold blasts for which the pig has an extraordinary aversion, and the deprivation of liquids in warm, dry seasons are frequent causes.

Symptoms. These have been described by M. Pradal, who divides the disease into three stages, evidently dealing with an infectious malady. The *first stage* is marked by loss of appetite, dullness, slow, listless movements, a tendency to hide under the litter; low, hoarse grunt and cough, the last aggravated by moving the animal; pain in swallowing; red, sunken eye, and constipation. If there is no improvement on the second or third day it merges into the *second stage*. This is characterized by a still hoarser grunt, painful, hard hacking cough, difficult breathing, especially in the sunshine, and a rapidly increasing swelling of the throat, soon extending to both ears and as far down as the breast bone. This engorgement feels soft and pasty though firm, tender lumps may be felt, indicating the approaching formation of abscess. It is so abuudant that suffocation may ensue in the course of forty-eight hours. If the progress of the swelling is not arrested it soon passes into the *third* or *gangrenous stage*. The breathing is more hurried; the mouth open, the protruded tongue of a bluish black color, the cough followed by a continuous rattle, the head unsteady, swallowing impossible, and the swelling extends to the side of the face and beneath the chest. The swollen surface is cold and livid; the bristles easily detached; it is bedewed by a serosity which exudes from it, and portions of the dead skin tend to detach themselves. The mouth and throat

participate in the gangrene, the breath, saliva and nasal discharge is fetid, and the epidermis peels off. The snout, ears and skin generally assume a bluish black hue, the prostration is extreme, the creature lying constantly on its side; the pain ceases and in one or two days death ensues, preceded by a state of comparative calmness.

On opening the throat after death the mucous membrane is engorged and thickened, bears various hues of black, blue, livid and green, and breaks down into a pulpy mass under slight pressure. The surrounding (pharyngeal) muscles even are implicated in this change. In the earlier stages there is only engorgement with blood of the tonsils and the mucous membrane of the pharynx and larynx; serous infiltration of the surrounding parts, and often the presence of inspissated mucus resembling false membranes or of ulcers on the surface.

Treatment. In the earlier stages, hygienic measures alone may suffice to check. A warm, dry, comfortable piggery, emollient and astringent drinks, such as sheep's head broth, oatmeal and other gruels acidulated with vinegar or buttermilk, an emetic (six grains of tartar emetic); a dose of physic (four croton beans powdered and given in the food, or from two to three ounces of castor oil), and if the patient will permit it a flannel bandage or piece of sheepskin round the throat. If the symptoms are more threatening it is recommended to bleed from the ears and tail; to apply a linseed meal poultice round the throat to hasten the formation of abscess, or in the absence of such indications to employ a mustard poultice made with spirits of turpentine, or rugs wrung out of boiling water, to the same part. Local astringent and caustic applications to the throat are the most promising, applied by means of a whalebone prob as recommended for other animals, the mouth being held open by a noose round the upper jaw. Sodium sulphite, silver nitrate, potassium permanganate, hydrochloric acid diluted, and tincture of iodine, may be employed.

When the gangrenous stage has been reached all treatment is useless.

LARYNGITIS IN THE DOG.

Sore throat is chiefly seen in pampered pets and in hunting dogs, as the greyhound. In the latter class it is the result of chills, a cold ducking when heated, cold damp kennels, etc. It is sometimes almost the only manifestation of distemper.

Symptoms. Dullness, impaired appetite, a slight cough becoming more frequent and paroxysmal. These paroxysms give rise to accelerated and panting breathing, and if severe, to the ejection of a glairy yellow (bilious) material from the stomach. There is also running from the nose and frequent sneezing. The dull muffled bark has led to the supposition of *rabies* but it has no resemblance whatever to the characteristic cry of *rabies* while beginning like an impulsive bark merges into an agonized and baffled howl. Accelerated pulse, elevated temperature, reddened fauces and swollen tonsils are marked symptoms.

Sometimes a short, dry cough obstinately remains after the disease has apparently subsided.

Treatment. Attend to general comfort, steam the nostrils, give a laxative if costive, and follow with iodide of potassium or sal-ammoniac (5 grains) repeated thrice daily. Apply a mustard poultice to the throat.

PHARYNGITIS. SORE THROAT.

Causes. Symptoms, larynx insensible, tender parotid and sub-maxillary swelling, cough mucous, difficult swallowing with gurgling, liquids returned by nose, buccal mucosa hot and red, salivation, chronic cases. Treatment, medicated drinks and electuaries.

ANGINA PHARYNGEA. CYNANCHE PHARYNGEA.

Inflammation of the pharynx owes its existence to the same *causes* as *Laryngitis.*

Symptoms. The general symptoms being like those of *Laryngitis,* the specific and distinguishing ones only will be here noticed. The larynx is not tender to the touch nor is cough

thereby excited. The glands beneath the root of the ears (parotids) are swollen and tender and cough is induced by handling them. The intermaxillary glands are enlarged. The cough is loose and followed by the ejection of glairy materials by the mouth and nose. Food and drink are swallowed with difficulty and effort, dry grain or hay is often refused, or dropped from the mouth, after it has been chewed, to avoid the pain of swallowing. Deglutition is accompanied by a gurgling sound caused by the abundant secretion in the pharynx. In swallowing liquids a portion is often rejected by the nose. The mouth is hot, red at its back part, and filled with fetid saliva which often drivels from between the lips in the coarser breeds of horses. The fever varies according to the intensity of the inflammation. This disease is rarely serious, and improvement is manifested, by a free discharge from the nose of a white opaque color, by the ability to swallow without pain, and the better appetite and general appearance. Collections of pus in the *Guttural pouches* may result from pharyngitis. See *Chronic Nasal Catarrh*.

Reynal has seen chronic cases of this disease due to: 1st, fracture of the large branch of the hyoid bone; 2nd, Laceration or ulceration of the soft palate; and 3rd, an abscess of the pharyngeal mucous membrane.

The *treatment* does not differ materially from that of laryngitis except in the greater value of soft food, mucilaginous and acidulated drink and of electuaries which act on the throat as they dissolve. Subjoined is a formula:

Recipe: Extract Belladona, four drams; potassium iodide, one ounce; sodium hyposulphitis, three ounces; mellis, five ounces. Mix. A desert spoonful to be smeared on the inside of the cheek thrice daily.

CROUP.

Croup : croak. Acute laryngitis, with spasms and perhaps pseudo-membranes. Relation to diphtheria. Causes, low, damp localities, exposure, youth, form of inflammation. Symptoms, onset sudden, crowing inspiration, hard cough paroxysmal, fever, larynx sensitive, dyspnœa in paroxysms or intermittent. Complications. Duration. Lesions, intense congestion, false membranes on larynx, fibrinous. Treatment, fomentations, ether, chloroform, chloral hydrate, laxative saline, sedative, derivative, surgical, water vapor, calomel, caustic, stimulants, tonics. Croup in sheep and horse.

CROUPOUS LARYNGITIS. PSEUDO-MEMBRANOUS LARYNGITIS.

Name and Definition. The word *croup* by which this disease is known over the whole of Europe and a great part of America is, essentially *Scotch*, and is familiarly used in the Lowlands of Scotland to signify—*to croak*. The disease consists in an acute inflammation or high vascular irritation of the larynx, associated with spasms of its muscles and commonly though not invariably with a firm layer of exudation on the surface of the mucous membrane. In some cases undoubtedly croup is but a form of the contagious pseudo-membranous affection diphtheria, the germs of which grown on a surface freely swept by continuous currents of pure air, retain too much of an ærobic habit to penetrate deeply into the tissues. (See Authors, "Malignancy mitigated by Oxygen," Medical Record, 1881, p. 673). It does not follow, however, that croup is always due to even a weakened germ. So far as yet appears it may develop independently of any particular pathogenic germ, from some violent local irritant in a predisposed subject. Croup therefore may be treated here as a presumably noninfectious disease. Being a very rare disease in horses its manifestation in ruminants will first be noticed.

CROUP IN THE OX.

Causes. These are not well understood. Low, damp situations would seem most liable, especially if the animals are much exposed at night. So far indeed as can be observed it arises from the same causes as *laryngitis*. Age affects its development. Croup is

mostly seen in animals between six months and a year old, and rarely in those over five or six years of age. The specific cause of the formation of false membranes and of spasms of the laryngeal muscles is a mystery, but to these the susceptible constitution and tissues of young animals appear to predispose. No mere grade of inflammation from the slightest hyperæmia to the highest type of inflammatory action is of itself sufficient to arouse the special phenomena. All of these are seen everywhere but *croup* may be said to be confined to certain localities and ages.

Symptoms. Unless it supervenes on a pre-existant attack of catarrh, croup is usually as sudden in its outset in the lower animals as in man. An extremely hard *croupy* cough, or loud, crowing, difficult breathing, loudest in inspiration, is usually the first symptom and appears to seize the animal in an instant and without the slightest premonition. This is closely followed by intense fever, full, hard pulse, 80 to 100 and upward per minute, increase of bodily temperature sometimes to 107.5° F., costiveness and high colored scanty urine. The throat is excessively tender, the slightest touch giving rise to violent paroxysms of coughing, during which the eyes redden and protrude from their sockets, the veins of the skin are gorged, the tongue, dry and livid, is protruded and small portions of the contents of the stomach and white shreds of false membrane are occasionally brought up. Sometimes in the intervals of coughing as well the mouth is constantly open and the tongue protruded and partly covered by a frothy but tenacious mucus. Suffocation appears imminent in many cases and the beast may perish suddenly in this way. On the other hand the threatening symptoms may be present only at certain periods of the day and may be moderated remarkably at others, especially at early morning. If complicated by any chest affection the symptoms are more urgent and the issue more commonly fatal. If associated with a low type of fever, a small, weak pulse, and much prostration, as it tends to be if it continues several days without relief, it has a more fatal tendency. The same may be said of its occurrence epizootically.

Duration. Croup will often run its course and prove fatal in twenty-four to forty-eight hours. Improvement is manifested by the cough becoming less convulsive and painful, by the expulsion through the mouth of shreds of false membrane, and by return of spirits and appetite.

Postmortem Appearances. If the animal has died suffocated, the lungs and right side of the heart will be gorged with blood ; if in a stupor (coma), attendant on brain poisoning with venous blood, the veins will be specially engorged. The mucous membrane of the larynx has a more vivid arborescent redness than in ordinary laryngitis but the special feature is the presence of false membranes. These layers of exuded material are almost confined to the air passages. They may extend to the soft palate and nose in an upward direction and to the trachea and bronchial tubes in a downward, but they rarely exist in the mouth, pharnyx, or gullet like the false membranes of diphtheria.

Characters of the false membranes. These are gray or yellowish white, though they may be reddened in patches or streaks. They vary in consistency from that of glairy mucus to a firm layer as of dense fibrine, and become more adherent as they are of older standing. Sometimes they are partially detached, the free end of the shreds floating in the larynx. The deep or attached surface presents redness in points, in streaks, or as ramifications very visible if the membrane is held up between the eye and the light. They vary in thickness from half to a line. Delafond has found these membranes in the lower animals to be mostly formed of fibrine, with a little albumen, and traces of alkaline and earthy salts.

Treatment. This must be prompt and energetic. Wet cloths as hot as the hands can bear, wrapped around the throat and neck, and replaced as they cool, will usually arrest the spasm. If this fails ether or chloroform by inhalation or chloral hydrate by injection may be employed with caution. The action of the bowels must be secured by salines (sulphate of soda ½ to 1 lb) or oil (linseed oil ½ to 1 pint) and injections of warm water. Sulphate of soda should be thereafter given in half ounce doses twice daily, or nitrate or acetate of potass may be substituted. They are advantageously given in linseed decoction and may be combined with laudanum, (½ ounce), belladonna, or other agent to check the spasms.

A blister (mustard poultice) should be applied at first either to the throat or breast, the windpipe being left untouched lest tracheotomy should be required. Similar applications to the legs are useful.

If suffocation appears imminent *tracheotomy* should be at once performed (see under Laryngitis). This operation has been depreciated because of the late period at which it has been employed, when the patient was already past all hope, but the resulting wound in the neck is more than counterbalanced by the greater freedom of breathing and the better aeration of the blood which tends to obviate the justly dreaded low fever. It often leads to a rapid diminution of the spasms and laryngeal irritation.

Agents applied directly to the inflamed mucous membrane are often requisite. The air of the building should be rather warm, equable and moistened by water vapor, if that can be conveniently done. Calomel or alum powder may be frequently introduced into the larynx by means of a whalebone prob and sponge as spoken of under laryngitis, or a solution of nitrate of silver (10 grains to the ounce of water) may be applied several times a day. These not only hasten the removal of false membranes but counteract their production. They produce violent and convulsive coughing at first and have to be used carefully. Delafond blew in such agents through an opening made in the windpipe. They may be injected with a hyperdermic syringe. In prostrate conditions it may be necessary to resort to stimulants (wine whey, carbonate of ammonia) and tonics (gentian, Peruvian bark).

CROUP IN SHEEP.

According to Roche Lubin croup is sometimes observed in spring in lambs and hogs. The common cause is "the shutting up of the animals for the whole twenty-four hours in a hot confined place, the floor of which is covered by a fine dust, and the air loaded with the same, owing to the jostling of the sheep together, the effects being intensified by the weight of the fleeces."

The disease is manifested by constant working of the jaws, extreme tension of the neck, abundant salivation, respiration hurried and whistling, extreme pain and threatened suffocation when the slightest pressure is made on the throat, and refusal of all food liquid or solid. The weak, hacking, convulsive cough is associated with the discharge of a whitish glairy mucus by the nose until the third or fourth day when false membranes may be expected.

Treatment is like that for the ox, medicine being given in about one-fifth of the doses.

CROUP IN THE HORSE.

The rare cases of *croup* in foals and young horses appear due to the same general causes as in ruminants. M. Riss records two cases, and Bonley one from breathing smoke when the straw of the stable had taken fire. The suddeness of the attack, the spasmodic symptoms and the duration of the disease and the treatment do not differ materially from those given for the ox.

PHARYNGEAL AND LARYNGEAL POLYPI.

Pediculated tumors. Dyspnœa through change of position, operation by ecraseur, snare, or cricoid incision.

Tumors of varied structure developing in or beneath the mucosa of pharynx or larynx often become slowly detached until they hang by a loose pedicle, and having much latitude of movement they may at times slip between the arytenoid cartilages or even into the glottis producing the most urgent or even fatal dyspnœa. Pediculated tumors in the posterior nares lead to the same accident. In one case of multiple small tumors on the pharyngeal mucosa of the horse, the largest and loosest, attached to the front of the epiglottis, was occasionally displaced into the larynx threatening instant asphyxia. One such attack supervened on the opening of a suppurating guttural pouch by the writer, necessitating prompt tracheotomy. A time was set for the removal of the polypus, but the tracheotomy tube having been accidentally displaced during the preceding night the patient died of suffocation. Dick mentions a polypus growing from the interior of the larynx and causing loud *roaring*. Such tumors may be removed by operating with the *ecraseur* through the mouth, or by a snare passed through a long narrow tube and used to seize and twist through the pedicle. If the polypus grows from the laryngeal walls, it may be best reached by incision through the cricoid cartilage and crico-thyroid membrane as in the operation for *roaring*.

DYSPNŒA LARYNGEA. ROARING. HEMIPLEGIA LARYNGEA.

Generic name for common symptom. Low and high notes. Grunter, roarer, whistler, piper, highblower. Pace or effort develops. Causes: of temporary roaring, inflammations, abscess, phlebitis, choking, dropsy, petechial fever, phlegmons along vagus. Causes of inveterate roaring, paresis of left recurrent laryngeal nerve, fatty degeneration of left arytenoid muscles, fracture of facial bones, polypi in air passages, chronic thickening of mucosa, foreign bodies in passages, tumors of lymph glands, abscess of guttural pouches, pseudo membrane, laryngeal ulceration, ossification, distortion, fracture of cartilages, action of forcible inspiration, leading on left side, deep origin of reccurrent nerve, effect of chest diseases and violent heart action, examples of morbid conditions impairing innervation. Lesions in muscles, and nerves. Facial palsy, poison (chick vetch, winter vetch, lead, fungi, moulds). Intermittent roaring. Hereditary roaring. Symptoms, grunting when coughed or threatened, heavy draft, galop, noise, larnygeal tremor, cold as a complication, roaring with expiration, lesions. Treatment, its use. Prevention, avoid breeding roarers, bearing reins, chick vetch, lead. Tonic medication, caustic to mucosa, firing, setons, iodine, pad nostrils, tracheotomy, arytenectomy, electricity.

This is the name of a symptom rather than a disease. It implies a sound made in breathing in connection with some contraction of the air passages. The term is however usually reserved for those conditions in which the sound results, from chronic disease or malformation, the noise attendant on laryngitis and other acute diseases being rarely spoken of as *roaring*. In neither case does the noise indicate more than that there is some impediment to the ingress and egress of air through larynx or trachea.

The pitch of the note varies exceedingly with the causes that produce it and with the hurried nature of the breathing. There have thus arisen the epithets of *grunter*, *roarer*, *whistler*, *high-blower*, *piper*, *trumpeter*, *wheezer*, *etc.* The most common distinctions are those of *roaring* and *whistling*. The *roarer* produces a loud deep basso sound in inspiration, the larynx or windpipe being only slightly narrowed while the *whistler* or *piper* produces a shrill blowing or sibilant noise because of the greater constriction of the passage. The term *grunter* is derived from the facts that a *roarer* usually makes a grunting noise when struck or threatened with a cane, and that when the upper cartilages of the

windpipe are pinched between the finger and the thumb the resulting cough is prolonged and somewhat like a *grunt*. A *wheezer* is usually suffering from spasmodic contraction of the bronchial tubes, from broken wind or from chronic bronchitis. A *high-blower* should never be spoken of in the same connection, as the noise is made from a playful flapping of the false nostrils or soft palate and disappears when the animal is put to the top of his speed. It is from confounding *highblowing* with *roaring* that *Eclipse* and other brilliant performers on the English turf have been erroneously pronounced *roarers*.

The noise produced by the *roarer* is not heard while he stands quiet, nor many cases even during a short trot or gallop. Such horses are in consequence often sold at the hammer and the purchaser is grievously disappointed to find that what he thought a sound horse is absolutely useless for the purpose for which he designed it.

Causes. Before noticing the symptoms of roaring a consideration of the causes will be useful to enable the reader the better to understand the signs by which the different forms are manifested.

Causes of temporary roaring. Whatever impedes the current of air causes roaring. Hence inflammatory diseases of the nose, throat, windpipe or bronchial tubes; abscess of one or the other of these parts; inflammation of a jugular vein and pressure on the trachea or vagus nerve by the resulting swelling; choking; the swelling in the neck consequent on the cutting open of the gullet for the relief of choking; thickening of the nostrils from dropsy, loss of a jugular vein, purpura hemorrhagica etc.; and swellings pressing on the vagus nerve, and which may be situated at the base of the brain, in the neck or in the anterior part of the chest. Also temporary infiltration of the laryngeal mucosa.

Causes of inveterate roaring. The one great cause of roaring and that which sustains the disease in nineteen cases out of every twenty is paralysis of the left *recurrent nerve* of the larynx and wasting of several of the arytenoid muscles on that side. It may be well, however, first to notice the less frequent causes and wind up with this more common one. 1st, Fracture with distortion of the nasal bones and narrowing of the nasal passages (Gamgee). 2nd, Polypi and other tumors of the nose, pharynx,

larynx, windpipe or bronchi. 3rd, Chronic thickening of the nasal mucous membrane, the result of inflammation. 4th, The presence of foreign bodies in the nose, as for example balls coughed up from the pharynx. 5th, Hering records a case resulting from the closure of the posterior opening of one nasal chamber by a membrane. 6th, Cancerous or melanotic deposits in the lymphatic glands above and to each side of the pharynx and larynx. 7th, Distension of the guttural pouches by inspissated pus. 8th, Chronic thickening of the mucous membrane of the larynx consequent on inflammation. 9th, The formation of a projecting fold of the mucous membrane or of a new production (false membrane) in the windpipe as the result of inflammation. Such false membranes have been known to become detached at their median part and remain attached at their two extremities thus constituting a band stretching from one side of the windpipe to the other. 10th, Ulceration of the membrane of the larynx particularly on the projecting folds circumscribing the glottis. 11th, Ossification of the laryngeal cartilages and loss of their elasticity. 12th, Distortion of the cartilages of the larynx, most commonly from unduly tight reining and pulling the nose in toward the chest. In such cases the cartilages of the larynx and those of the windpipe adjoining being compressed slide within each other, and the enclosed edge projecting within the air tube materially diminishes its calibre. 13th, Fracture of one or more rings of the trachea. This usually results from blows, as in running the neck against the back of a cart or wagon. The cartilaginous rings are usually broken at their median part in front and being retained together by the investing elastic tissue which enables the pieces to move on each other as by a hinge, and being approximated by the contraction of the trachealis muscle above, the ring is flattened from side to side and the channel for the passage of air correspondingly decreased. This flattening can be easily felt by the hand in the living horse. 14th, A peculiar congenital distortion of the trachea caused by the curling in of one end of each cartilage of the windpipe and the straightening out of the other. This occasionally proceeds so far that the gullet is lodged in the interspace overlapped and hidden by the free ends of the cartilages, the diameter of the windpipe being proportionately diminished. Distortions and fractures are usually overlooked by veterinarians but

from the frequency with which the author has met with them in his dissection he is convinced that they deserve greater attention than has been awarded them. 15th, Percivall records a case of inveterate *roaring* in which, even tracheotomy having failed to cure, the patient was destroyed and the lungs found to be extensively consolidated, many of the air tubes having been so compressed as to be almost impervious. I have known a case of roaring from the presence of a pedunculated tumor in the lower end of the windpipe where it divides to enter the lungs, and the same result may ensue from the partial obstruction of the bronchial tubes by masses of tenacious mucus in chronic bronchitis.

16th. The immediate cause of *roaring* in the immense majority of cases is the **paralysis and fatty degeneration of certain muscles on the left side of the larynx.** The muscles supplied with motive power by the **left recurrent laryngeal nerve** (Crico-arytenoideus posticus, Crico-arytenoideus lateralis, thyro-arytenoideus, and the left half of the arytenoideus) are those constantly and exclusively affected, while those supplied by the *superior laryngeal nerve* (Crico-thyroideus, hyo-thyroideus and hyo-epiglottideus) remain unchanged. The left recurrent nerve is also wasted and considerably attenuated as compared with that on the opposite side. The *modus operandi* of this paralysis and wasting in the production of *roaring* is beautifully seen when the upper part of the windpipe is laid open so as to expose the interior of the larynx in laryngectomy. The triangular opening of the glottis is seen fairly dilated while the muscles are relaxed in the act of expiration. As soon, however, as inspiration commences the left arytenoid cartilage slides completely into the passage, its lower border projecting so much to the right that it forms a prominent crest extending beyond the median line and in some cases closely approaching the right wall of the larynx, the superior elastic and free border of the same cartilage meanwhile gets drawn inward by the suction power of the air so as to block up the passage still more. The closure of the glottis being thus seen to be largely controlled by the current of inspired air, it becomes evident that any increase in the force of the current will aggravate it and a decrease will lessen the extent of the closing and alleviate the distress of breathing. This fact furnishes a means of palliating the symptoms. (See treatment.) It ex-

plains moreover why roaring should not be heard in quiet breathing and why it should increase in force and in pitch as the respiration becomes more and more hurried. It further accounts for the noise being heard only during the act of inspiration, the outward rush of the air in the expiratory act being of itself sufficient to carry this valvular cartilage out of the passage and secure a free and unimpeded current.

The paralysis and wasting of the left recurrent nerve and muscles are in their turn due to very varied morbid states.

It may commence in the larynx from distortion of its cartilages and inflammation of the mucosa, in which case the wasting of the nerve is probably a result of its prolonged inactivity. This mode of origin is strongly insisted on by Percivall, and no doubt occasionally arises. Under this explanation, however, it is difficult satisfactorily to account for its almost invariable occurrence on the left side. The mere fact that the horse is habitually approached on this side and more commonly turned toward it is a most insufficient reason.

Even if admitted it utterly fails to explain the immunity of the muscles supplied by the superior laryngeal nerve.

The fact that a horse has usually a hard and soft side of the mouth and carries the head slightly to the latter is no better explanation, as the tender side is not always the left.

More commonly the disease arises at some other point near the origin or in the course of the nerve, and the changes in the larynx follow as the consequence of deficiency or entire absence of motor innervation. Many cases can be cited in which such an origin was unquestionable, and on the hypothesis that this is the true and constant history of the development of the malady, its regular occurrence in the left side, and the absence of all signs of wasting in the muscles supplied by the superior laryngeal nerve are alike perfectly explainable.

Let it be noted that the vagus nerves (right and left) of which the recurrent laryngeal are branches, originate from the base of the brain, pass down the neck beneath the jugular vein in company with the carotid artery ; that on entering the chest the right vagus nerve gives off its recurrent branch which proceeds at once up the neck along the course of its parent trunk till its reaches the larynx, to the muscles on the right side of which it is dis-

tributed ; that the left vagus nerve on the other hand proceeds backward in company with the left innominate artery as far as the base of the heart, where on about the level of the space between the sixth and seventh ribs it gives off the left recurrent nerve ; that this left recurrent nerve closely applied at its point of origin to the great parent arteries turns round the posterior aorta enclosing it in a loop, and gaining the lower end of the windpipe follows its course to the larynx. It will thus be understood how many chest diseases may implicate the left recurrent nerve, and from which the right, which extends no deeper than between the two first ribs, may be completely exempt. The frequent supervention of roaring as a sequel of chest diseases receives in this an ample explanation. Its connection with pleurisy becomes especially probable, as the nerve lies in contact with the surface of the pleura alike in its descending and ascending course within the chest.

Finally the loop encircling the posterior aorta exposes the nerve to constant stretching and shocks from the heart's action during violent exertions and in excited states of the circulation generally. Vaerst and Sussdorf show that the nerve is habitually flattened between the posterior aorta and trachea, the effect being worst when the heart's action is excited.

It remains to notice a few instances in which dissection established the connection of interference with the nerve at some part of its course and the existence of roaring.

(a.) Godine found in a roarer a tumor about the size of a chicken's egg, pressing on the commencement of the pulmonary artery. He attributed the roaring to the impaired circulation of blood in the lungs by the pressure on the artery. Considering that the tumor must have been precisely in the situation of the left recurrent nerve at its point of origin, it becomes much more probable that the symptom resulted from pressure on this nerve.

(b.) The elder Bouley found in one case a considerable engorgement of the group of lymphatic glands in the anterior part of the chest and through the centre of which the left recurrent nerve passed.

(c.) Fergusson of Dublin dissected a roarer in which he found besides some tumors of the lymphatic glands in the pelvic and sub-lumbar regions, an indurated and enlarged gland about four

inches behind the anterior opening of the thorax. The recurrent nerve between this and the larnyx was wasted so that its fibres could scarcely be recognized, the laryngeal muscles on that side were atrophied, and degenerated, and the glottis distorted and partly closed. Fergusson has in his description made the mistake of writing the *right* for the *left*; it is evident that the *right recurrent nerve* could not possibly pass through a tumor in the situation described.

(d.) Gamgee furnishes a drawing of an immense tumor filling up the anterior part of the chest, pressing on the vagi and recurrent nerves and causing roaring.

(e.) The Clinique of the Alfort Veterinary School furnishes the following among other cases of roaring consequent on inflammation of the jugular vein. A well-bred and very fast English thoroughbred had been used for two years by his owner who was a hard rider. In June, 1857, he was bled as a preventive (saignée de precaution), supurative phlebitis was induced and was only cured at the end of six weeks. When again put to work he proved a *roarer* and was still affected when seen six months later.

In connection with this it may be noted that the swelling in connection with the inflammation of the vein extends easily to the sub-jacent vagus and recurrent nerves, leading to their inflammation, functional inactivity and atrophy. Bleeding is usually done on the left side of the neck so that the paralysis and wasting would still be on the same side. Happily with a more humane system of treatment, accidents of this kind are less frequent than formerly. Glöckner furnishes a case which followed thrombosis of the carotid.

(f) Reynal reports several cases in which *roaring* had occurred as a sequel of inflammations and abscess about the throat, and in which infiltrations or gray or yellow indurations had taken place in the areolar tissue around the vagus nerve. As nothing is more common than to find *roaring* resulting from severe sore throat, parotitis, etc., this may explain its occurrence.

Mandl first carefully examined the paralyzed muscles which present to the naked eye a flattened and wasted appearance in marked contrast to the full well-rounded forms or those on the opposite side. They differ no less in color. In place of the deep red of the healthy muscles those on the diseased side are of a yellowish

same or even in a worse condition, the larynx meanwhile unnaturally compressed between his narrow jaws and the nerve compressed or the larynx distorted.

It must be added, however, that like some other acquired distortions or alterations roaring may repeat itself in the progeny. Goodwin mentions an instance of it on the female side through three successive generations of thoroughbreds. Of transmission on the side of the male the following instance is noteworthy: M. Liphaert, an extensive proprietor in Livonia, bought a first-class English thoroughbred stallion. His progeny were healthy until he became a roarer at ten years old. All his foals, got after this date, followed the sire in becoming roarers, and, it is important to observe, almost all at the age of ten years.

Symptoms. These, of course, are manifest enough while the animal is sufficiently excited to give rise to the noise. Certain indications may be obtained even while the animal stands in the stable. If *cough* is excited by pinching the upper rings of the windpipe it is prolonged into a groan. If suddenly threatened with a cane the abrupt inspiration which results is attended by a *grunt.* The absence of these symptoms is not, however, sufficient to establish the non-existence of roaring. The horse must be galloped or put to heavy draught to fully test the breathing organs. Galloping up a steep hill is perhaps the best test. A gallop over a recently ploughed field is about equally good. Soft pasture land or an unpaved road is preferable to Macadam or pavement. Galloping in a riding school on the soft tan is an excellent measure as the sound is confined and the animal is always within earshot of the examiner. The person examining should either ride the horse himself or have a disinterested party, in no way connected with either buyer or seller, to mount him. If the rider is in the interest of the seller he may contrive to slacken the pace before he reaches the examiner, or by irritating the horse may make it difficult to approach him immediately on his being pulled up. If in the interest of the buyer he may succeed, by the use of a powerful bit, in drawing the horse's nose in to the chest, or by compressing the larynx with a tight throat latch he may produce noise in breathing when the animal is suddenly brought to a stand. Unless the course is up a steep hill or over a ploughed field the horse should be galloped for from five

to ten minutes; he should be then made to pass close to the examiner at full speed, and finally brought up suddenly by his side and without any previous slacking of his pace. The ear should be at once placed close to the nostrils, when the slightest abnormal sound accompanying the inspiratory act will be at once recognized.

Draught horses are sufficiently tested by driving them in a heavy vehicle or one with the wheels dragged. By walking alongside or keeping the ear near to the nostrils any harsh sound additional to the normal blowing noise of hurried breathing is easily noted.

The finger placed on the larynx detects the strong vibratory tremor, and Friedberger notes that the left arytenoid is much more easily displaced than the right, increasing the stridor.

If the horse is, at the time of examination, the subject of a cold, sore throat, or other acute disease of the air passages no importance is to be attached to any noise made in breathing, but he cannot be pronounced a *sound* horse until, this malady having passed off, it is found on careful examination that no such sequel has been left.

Among the most puzzling cases are those in which the roaring occurs with periods of intermission. If the horse has been fed for a short time on vetches this may account for its temporary access, and unless the same feeding is again allowed a recurrence is not to be looked for. If due to the occasional displacement of a pedunculated tumor of the nose or pharynx and its interference with the action of the larynx its existence may be recognized by careful examination, diminished current of air through one nostril, etc. But there remain some *rare* cases in which there are no such appreciable causes, and yet the horse would be pronounced *sound* or *unsound* as examined at certain intervals. On this subject more information is desirable.

The following varieties of roaring will be distinguished from that of paralysis by the occurrence of the sound in both acts of breathing (expiration and inspiration):—distortions, tumors or foreign bodies in the nose:—tumors about the throat, in the windpipe or bronchi:—distortion of the windpipe, from tight reining, fracture or congenital deformity:—and the presence of a false membrane stretching across the windpipe.

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Examination by manipulation, auscultation and percussion along the whole length of the air passages alike during rest and after exercise, may enable one in unusual cases to recognize the structural changes that give rise to roaring.

Treatment. This has long been considered as hopeless, yet preservative and palliative measures are usually accessible, whilst even cures can be effected in certain conditions.

Preventive treatment. First may be noticed the rejection for breeding purposes of all animals possessing those conformations of head, neck and chest already referred to as conducing to disease of the air passages or distortion of the larynx or windpipe. Equally ought all roarers to be set aside unless the exciting cause is accidental such as fractures of the nasal bones, of the trachea, the existence of polypi, etc. Stallions that make a harsh noise in breathing from an accumulation of fat about the throat are not necessarily objectionable.

The employment of the bearing rein so as to compress and distort the larynx is to be avoided. If bearing reins are used in horses having short thick necks and badly set on heads and especially with intermaxillary narrowness they should be passed through rings in the cheek piece of the bridle or between the ears and over the forehead (overdraw check) so that while the head is elevated the nose may be projected forward after the Russian fashion of equitation. This measure has indeed appeared to cure several cases of roaring. I have met with fewer roarers in the same number of horses in America than in England, and this I attribute to the better mode of using the bearing rein on this side of the Atlantic.

The Chick Vetch (Lathyrus Cicera) should be excluded from the fodder of horses or used in small proportion only. In man it is found to be injurious when it forms a twelfth part of the bread used and gives rise to paralysis if it amounts to a third (Aitken).

Paliative and Curative treatment. Medicinal treatment will prove useless in the great majority of cases: as for example in paralysis and degeneration of the muscles, in ossifications, fractures, or distortions of the cartilages, etc., etc. Nevertheless where there is merely thickening of the membrane of the larynx alterative and tonic treatment may be successful especially if associated with iodine ointment or active blisters applied to the throat. A

case is reported by Dupuy in which a course of arsenic cured. In these cases as well as in those due to ulceration of the membrane the application of caustic by means of a staff and sponge as advised in laryngitis may prove beneficial. In some cases of this kind the application of the firing iron to the region of the larynx has an excellent effect. Setons have proved useful in some cases.

In cases due to tumors or enlarged glands pressing on the air passages the internal use of iodine and other alteratives and diuretics, and the local applications of iodine, or mercurial ointments or of blisters have been successful. Failing in this the tumors may be removed with the knife when accessible.

If by auscultation the existence and position of a band of lymph can be made out, tracheotomy may be performed and the band excised. Percivall with reason doubts the possibility of the diagnosis.

In cases due to distortion of the larynx from tight reining the bearing reins should be dispensed with or rearranged so as to encourage protrusion of the nose, and the horse should be bitted to the side chains or straps in the stall several hours daily so that the head shall be elevated and the nose protruded.

When *roaring* depends on paralysis of the laryngeal muscles, a mode of palliation may be adopted as practised by the London omnibus and cab men. A strap is fixed round the nose supported by a strap passing down the middle of the face and the cheek piece of the bridle on each side and buckled beneath the chin. On the inner side of this strap where it passes over the false nostrils is attached on each side a semiovoid pad which presses on the flap of the nostril and regulates the entrance of air. The principle on which it acts will be understood when we consider that the paralyzed cartilage is drawn into the passage by the rush of air and that the closure of the channel is more complete and the roaring more marked in proportion to the force of the current. The pads by lessening and regulating the rush of air into the lungs thus leave the passage in reality more open and largely obviate the difficulty of breathing and the noise.

In extreme cases with the structural lesion in the head, throat, or upper two-thirds of the neck relief may be secured by tracheotomy.

A more radical operation is that introduced by Günther for the

excision of the left arytenoid cartilage. As improved by Möller and others this consists in an incision through the cricoid cartilage and crico-thyroid membrane (or even the first rings of the trachea) and the complete extirpation of the left arytenoid cartilage and left vocal cord. The manipulations belong to surgery. The result is satisfactory in removing the violent dyspnœa in hurried breathing and in very favorable cases in obviating noise altogether. More commonly some stridor remains but not enough to interfere with pace or with heavy draft. From my personal experience in performing the operation, I would recommend it in all cases in which the obstruction is so great as to interfere with the use of the horse on the track, or road, or for heavy draught. But in slight cases, in which the disease causes little or no inconvenience beside the noise, I would advise some less radical measure.

Another obvious line of treatment is by the use of electricity locally and of strychnine internally. A weak current kept up for fifteen minutes may be sent from the positive pole in the left jugular furrow to the negative pole over the left side of the larnyx. Strychnia in the dose of two grains may be given daily in the food or in half that amount hypodermically over the left side of the larnyx. This would be useful only in the early stages with little or no fatty degeneration of the muscles.

ŒDEMA GLOTTIDIS

Diseases Complicated by this. Seat, Abundance. Duration, Sequels, Symptoms, sudden dyspnœa, swelling of throat, pits on pressure, differentiation from croup. Treatment, cold, ice, astringents, scarification, tracheotomy.

This is usually a complication of acute laryngitis, but it may be a manifestation of other forms of local disease—tuberculosis, glanders, purpura hæmorrhagica, pseudo-membranous inflammation,—or it may be a result of a more distant affection, like diseae of the heart, lungs, or kidneys. As a complication of local inflammation it consists in an excessive serous exudation into the submucosa, around the base of the epiglottis and extending to the whole larnyx and pharnyx. It may thicken the parts by half an inch, causing complete closure of the glottis. In favorable cases it may subside as rapidly as it rose, while in others it may result in ulceration or abscess. The infiltration has usually a clear watery aspect, but is sometimes a dull red. When incised an abundance of serum escapes mixed in certain cases with pus.

Symptoms. In the course of one of the above named affections there comes on suddenly extreme dyspnœa, with stertorous breathing, a suffocative cough, and intense anxiety. The stridor is first with inspiration and later with expiration as well. The eyes are bloodshot and protruding, the pulse small and rapid, the movements uncertain, and the skin moist with sweat. There is manifest swelling of the throat and manipulation leaves the imprint of the finger.

When symptomatic of some distant affection it is at once slower in its result and more persistent.

The local pasty swelling and the absence of any false membrane suffice usually to distinguish it from croup which it so closely resembles in the suddenness of its onset, and the violence of its manifestations.

The less urgent cases may be treated by application of cold water or ice to the throat, and the injection of solutions of chloride of iron or alum into the fauces. Or the throat may be painted with tincture of iodine and rubbed with the palm to favor distribution and absorption of the exudate. In dogs the mouth may be opened widely and the dropsical membrane pricked at intervals to drain off the liquid. In the most acute cases the prompt adoption of tracheotomy is the only means of saving life.

LARYNGEAL HYPERÆSTHESIA. CONVULSIVE COUGH.

Convulsive cough with visible lesions—without. Excitants, cold air, or water, rough or dusty food, irritant agents inhaled or swallowed. Treatment, hygienic, nerve sedative, expectorant, tonics, Muriate of ammonia, Sulphur dioxide, silver solution, ferric chloride, alum, derivatives, elimination, aromatic, dietetic.

The chronic or paroxysmal cough may often be traced to the presence of tumor, ulcer, local inflammation, or parasite, but in some instances no local trouble is recognizable, the general health remains good, and yet the throat is abnormally sensitive and a cough or fit of coughing may be roused by passing into the cold air from a warm stable, by cold water in drinking, by inhaling irritant gas, by the passage of rough or fibrous food, or by handling the larnyx. There is undoubtedly a hyperæsthesia of the larnyx and the horse and dog as being more exposed to severe demands on the physical and nervous systems are especially liable to suffer.

Treatment must be adapted to the conditions. Over-work, damp unhealthy buildings, and all appreciable health depressing causes must be corrected, and a course of iron and nux vomica may be tried. Borax, bromide of potassium, and extract of hyoscyamus, made into an electuary with molasses or honey may be smeared upon the molars four or five times a day. In obstinate cases the inhalation of the fumes of burning salammoniac or sulphur, or the direct application to the larnyx of dilute solutions of silver nitrate, ferric chloride, or alum may benefit. The throat may be blistered by cantharides or mustard. Care should be taken to keep the functions of bowels and kidneys normally active, to protect the patient against cold and damp, and to give nutritive but non-stimulating and easily digested food, as for the horse, bran mashes, roots, grass or scalded hay, and for the dog pulped flesh, soup and mush. Sometimes benefit can be obtained from the vegetable aromatics and stimulants as eucalyptol, tar, turpentine, balsams of Tolu and Peru, tincture of anise, fennel, etc.

INFECTIOUS DISEASES OF THE THROAT.

Infectious throat diseases. Parasites, Leeches. Œstrus larva. Chronic sore throat. Mechanical removal.

Infections are in many respects the most serious affections of this region but their consideration must be sought under strangles, distemper, diphtheria, anthrax, actinomycosis, tuberculosis, glanders, etc.

PARASITES OF THE THROAT.

Leeches. These taken in with the water will sometimes fasten themselves on the walls of the pharnyx or even on the lips of the larnyx, producing cough, sore throat, difficulty of swallowing, bleeding from the nose (or mouth), or dyspnœa. They are to be removed as recommended above under parasites of the nasal chambers.

Œstrus Larva.. Bots. In horses and mules the larva of the œstrus sometimes attaches itself to the mucous membrane of the pharynx or even of the larynx producing chronic irritation, cough and even dyspnœa. A chronic sore throat with nasal discharge, occurring in autumn or winter, in the absence of fever or constitutional disorder may be found to depend on these parasites and to recover when these have been removed by the hand.

GUTTUROMYCOSIS OF SOLIPEDES.

Aspergillus. Complications, ulceration, lesions of adjoining parts, food in lungs, hepatization, gangrene. Treatment, by incision, sulphur dioxide, iodine.

Rivolta and Bassi have found in the guttural pouches of horses and a mule, an advancing ulceration of the mucosa partially covered with crusts composed largely of the mycelium, conidia and spores of Aspergillus or a closely allied fungus. In the mule the ulcer had opened into the carotid artery causing a profuse epistaxis. In the three horses there was dysphagia, and the food, descending to the lungs, had caused pulmonary hepatization and gangrene. The description of the ulcers led Raillet to infer the existence of glanders and that the presence of the aspergillus was accidental, rather than a causative factor. In parallel cases the opening of the guttural pouch and injection with sulphurous acid solution or dilute solution of iodine would be appropriate treatment.

DISEASES OF THE CHEST.

Cough, its artificial production, precautions, character in different animals. Cough in disease, strong, full, ringing, weak, short, broken, abortive, dry, raspiug, croupy, small, husky, soft, humid, rattling, mucous, paroxysmal, sympathetic, wheezing, roaring, whistling, grunt, moan. Expectoration, nasal in horse, also buccal in other animals. Morbid expectoration, watery, viscid, cloudy, flocculent, purulent, rusty, cretaceous, parasitic, fœtid, varicolored, microbic. Expired air, warm, cool, vegetable odor, acid, fœtid, heavy. Respiration, number in health, alteration in disease, rapid, slow, tardy, short, catching, quick, deep, labored. Position, standing, lying. Pleuritic breathing, broken-winded.

Before describing specific diseases, it is needful to consider the methods of physical diagnosis which enable the practitioner to differentiate the diseases of the chest. Some of the following remarks will bear equally on diseases of the nose and throat as well.

COUGH.

The cough so varied in health and in disease deserves careful practical study. It can usually be excited in solipedes, sheep and dogs by pinching the first ring of the windpipe between the thumb and first two fingers. In oxen it is best produced by compressing the anterior part of the larynx. In old cattle it is difficult to produce coughing. In no animal should the attempt be made rudely nor unnecessarily repeated, as it may tend to excite or to aggravate already existing sore throat.

The cough of the healthy *horse* is *sharp, loud* and *ringing*, often repeated two or three times and followed by a snort (clearing of himself). It is weaker in young horses and shorter and drier in the aged.

The usual cough of the *ox* is *weak, dry, slightly husky* and *prolonged*.

That of the *sheep, small, weak* and *dry*.

That of the *dog*, also weak and dry.

A **strong, full, deep, ringing cough** is rarely heard in disease except in slight irritation of the larynx. In such cases the larynx is tender and slight handling or pinching develops the cough.

A **weak cough** wanting in reasonance and heard only at a short distance from the horse, is usually associated with chronic chest diseases and the last stages of acute thoracic inflammations.

A **short, broken** or **abortive cough** is one which appears to be suddenly cut short and suppressed, from the pain it causes. It is seen in the early stages of inflammations of the serous membranes of the chest or abdomen, when the quick rubbing of the dry and inflamed surfaces of these membranes on each other produces exquisite pain. It characterizes especially the *debut* of pleurisy, pleuro-pneumonia and peritonitis. This cough is infrequent for the same reason that it is short.

A **dry, loud, rasping**, or **croupous cough** is peculiar to the early stages of laryngitis, tracheitis and bronchitis, when the membrane is swollen, tense and dry. It is equally met with in diphtheritic and croupous affections implicating the larynx.

A **small, weak, dry, husky cough** without any *rasping* is characteristic of broken wind (heaves) emphysema of the lungs, asthma, or chronic bronchitis.

A **soft, humid** or **rattling cough** exists in the advanced stages of laryngitis, bronchitis and pneumonia when the activity of the inflammation has given way and a free exudation has taken place from the mucous membrane. It is usually accompanied by a discharge, in solipedes from the nose, and in other animals from nose and mouth.

A **soft cough** with a peculiar gurgling in the larynx is sometimes met with in croup.

A **paroxysmal cough** is one repeated five, ten, or twenty times in rapid succession. It is common in chronic bronchitis, early heaves, emphysema, verminous bronchitis and influenza. In such cases it is observed chiefly when the subject is brought out to the cold air, when he takes a drink of cold water, or when he has just had some active exertion, or some dusty or fibrous food.

A **symptomatic cough** is one due to disease in some other organs than the respiratory, and which irritates the air passages through nervous sympathy (reflex action). It is commonly small, short and dry. Inflammation or other disease of the liver, indigestions and intestinal worms are occasional causes of symptomatic cough. In the case of worms it may be loud, clear and ringing.

OTHER MORBID SOUNDS.

Besides cough may be noticed the *wheezing* breathing characteristic of *broken wind*, *chronic bronchitis* and *asthma*, *roaring*, *whistling*, etc., as already described, and the sound between a *moan* and *grunt*, produced in pneumonia especially in the ox.

EXPECTORATION.

This escapes almost exclusively by the nose in horses, because of the length of the soft palate. It may come from the mouth of other animals, especially when they cough. In the ox the discharge from the nose is rarely seen because of his licking it out with his tongue. Rattles (râles) in the larynx, trachea or bronchia, enable us to ascertain the source of such discharges.

The nasal discharge in acute catarrh, laryngitis or bronchitis, is thin, clear, and slightly viscid, becoming thick, whitish and flocculent as the disease advances. It is yellowish, thick, flocculent and intermixed with shreds of false membranes in diphtheria or in the croup of young foals and calves. It is clear, slightly viscid and watery at the onset of bronchitis. At the debut of pneumonia it is often reddish (rusty). It is bright, red, frothy and bloody in haemoptysis. It is scanty, clear, watery, and containing minute white flocculi in pulmonary emphysema (broken wind). It is white, thick, curdy, and devoid of viscidity in chronic bronchitis or when a pulmonary abscess is being emptied. It is grayish, thick and flocculent in advanced pneumonia in the horse.

Cows in the advanced stages of pulmonary tuberculosis expectorate a yellowish, sticky matter containing minute hard masses often cretaceous. Calves and lambs suffering from strongyli in the lungs expel these in little pellets in the midst of a thick white material.

The expectoration is fetid, dark red and grumous in gangrene of the lungs.

In pulmonary tuberculosis and glanders the expectoration usually contains the respective bacilli.

CHARACTER OF THE EXPIRED AIR.

The breath is sensibly warmer in excited breathing, high fever, and acute bronchitis and pneumonia. It is cool in most chronic diseases, in advanced consumption and hydrothorax. Its odor is vegetable and acid in the acute indigestions of cattle, and fetid in many chronic diseases of the air passages attended with destruction of tissue, or the escape of imprisoned pus, but especially fetid in gangrenous sore throat or gangrene of the lung.

MODIFICATION OF THE RESPIRATION.

The number of respirations in a given time may afford valuable indications in the horse but in the other domestic animals variation in number imports little. In the ox for instance, the respirations in health may vary from twelve to eighty per minute, according to the heat of the cowhouse, the plentitude of the abdominal organs and other circumstances. So in the sheep and dog slight causes, quite compatible with health, may cause the breathing to become short, panting and hurried.

The young horse breathes ten to twelve times per minute, the adult animal nine to ten. Any excitement accelerates. A horse walked a few hundred yards had the respirations increased from ten to twenty-eight per minute; after trotting five minutes they numbered fifty-two; after galloping five minutes sixty-five.

Hurried breathing occurring independently of exercise, heat of the atmosphere, or distension of the abdomen, is indicative of fever, especially if associated with rapid pulse and increased heat of the body.

Infrequent respiration appears in certain brain diseases in the intervals between the more violent paroxysms, also in poisoning by opium and other narcotics. **Tardy or slow respirations** differ from those last noticed in the act occupying a longer time. In infrequent breathing the act may be short, though there are few respirations in the minute. This is likewise seen in brain diseases and sometimes in broken wind. In the last case there is **double action of the flank,** each act of expiration being effected by two successive and distinct elevations of the flank.

Quick breathing in which the act occupies only a short time is usually abruptly cut off, the inspiration terminating by a catch or

jerk. It is significant of the early stage of pleurisy, and arises from the desire to avoid the pain attendant on the rubbing together of the inflamed surfaces during deep inspirations. It is further seen in tetanos, peritonitis, pericarditis and pleurodynia.

Deep breathing with great lifting of the flanks and loins is characteristic of water in the chest, and consequent inability to inflate the lungs.

Labored breathing, which is at once hurried, deep, and without intermission, is seen in severe laryngitis, croup, capillary bronchitis, and pneumonia, in all cases alike from the difficulty experienced in introducing into the lungs the requisite amount of air. It is especially marked in double pneumonia, pleuro-pneumonia, complicated with effusion in the chest, and in old standing *broken wind* with dilatation of the right heart.

In all such cases where there is much interference with the æration of blood, whether from obstruction to the circulation of blood or a hindrance to the introduction of air, the horse invariably stands. The fact that he has lain down may be taken as an indication that improvement has taken place. The peculiarity is due to the sharp outline of the horse's sternum inferiorly so that in lying down he is compelled to rest on his side and the whole weight of the body tends to compress the chest. In the ox, sheep, pig and dog, which can rest on the sternum, breathing can be carried on with comparative ease in the recumbent position, and these animals accordingly do not necessarily stand except in very extensive and violent affections of the chest.

The occurrence of a short inspiration suddenly checked and a prolonged expiration characterizes pleurisy, the check to the inspiratory act being because of the pain caused by dilating the thorax.

The double lifting of the flank in expiration:—the act appearing to be performed by two distinct and successive acts is one of the most prominent symptoms of broken wind, but is not peculiar to this disorder. In the horse it exists in chronic bronchitis, dilatation of the right heart, old standing hydrothorax, and diaphragmatic hernia. It is further frequent in the acute diseases of the chest. In oxen it accompanies pulmonary emphysema, pulmonary consumption, dilatation of the heart, foreign bodies in the heart, and dropsy of the pericardium.

If accompanied by clear resonance over the chest, a permanent wheezing noise heard over the ribs, and the small, weak wheezy cough, it indicates emphysema (broken wind). If with strong impulse of the heart against the ribs behind the elbows, venous pulse in the jugulars, and modification of the second sound of the heart, it bespeaks cardiac dilatation or other heart disease. If with paroxysmal cough, white curdy nasal discharge and harsh rasping sounds heard at the lower part of the trachea or along the upper part of the lungs it betrays chronic bronchitis.

RELATIVE POSITION OF THE LUNGS, HEART AND OTHER ORGANS IN THE DIFFERENT DOMESTIC ANIMALS.

Relative positions of thoracic organs. Diaphragm, heart, lung, in horse, ox, sheep, pig, dog. Palpitation.

The *chest* is that portion of the trunk closed in on each side by the ribs, above by the bones of the back, below by the breast bone, and behind by the diaphragm. It forms thus a cone flattened from side to side anteriorly, and with its base, represented by the diaphragm which slopes obliquely from above downward and forward and bulges forward in the centre to a greater or less extent according to the plenitude of the stomach and bowels. It results from this arrangement of the diaphragm that a very thin layer of lung only reaches to the posterior part of the chest, and that beneath this are solid and hollow abdominal organs which modify the results of physical examination.

In the **Horse** the anterior third of the chest is covered laterally by the bulky and muscular shoulders so that it cannot be satisfactorily examined. In the median line of the chest, at a point corresponding to the third, fourth, fifth and sixth intercostal spaces, is lodged the heart. It deviates slightly to the left side below and by virtue of a notch in the lower border of the lung is enabled here to reach the surface and its beats may be felt by the hand laid on the side of the chest just behind the left elbow.

In the **Horse** the diaphragm is attached by its outer border to the last rib, and to the lower ends of all the asternal ribs, and the extremity of the breast bone. A thin layer of lung accordingly extends to between the two last ribs superiorly and down to near the lower end of the asternal ribs. The subjacent abdominal organs are arranged as follows :—*On the left side*, and counting from below, the large intestines (double colon), the stomach and spleen and a portion of the left lobe of the liver :—*on the right side*, below, the large intestines, above, the liver and pancreas. Of these the stomach and intestines frequently contain gases, while the liver by its solidity gives a special solid character to the right posterior part of the chest. The spleen is too deeply situated to affect much the results of a physical examination. The greatest substance of lung is between the upper and middle thirds of the thorax. The anterior third is inaccessible on account of the shoulders, but more than usual may be reached by raising the fore limb and drawing it forcibly forwards. The space between the third and seventh ribs is occupied by the solid mass of the heart, which especially modifies the result of physical examination on the left side where a notch in the lung allows it to approach the surface.

In the **ox** the diaphragm is only attached to the last rib for two or three inches at its upper extremity ; it is fixed to the second last rib as far down as about one-third of its length ; thereafter it is attached in succession to the middle third of the third last, to the lower third of the fourth last, to the lower ends of the next two in succession and to the sternum. The result is that the lungs do not extend so far back relatively to the ribs as they do in the horse. They are virtually absent from the last intercostal space, present only in the upper third of the second last, in the upper two thirds of the third last and reach the lower third only in the space between the ninth and tenth ribs. The paunch alone occupies the space beneath the asternal ribs on the left side, and the liver and the solid mass of the omasum and abomasum that beneath the right. The shoulders in fat improved beef breeds absolutely prevent examination of the anterior third of the chest, though in thin animals and dairy breeds and scrubs more of this may be exposed by raising the fore limb than in the horse. The heart corresponding in position to the third, fourth and fifth intercostal

spaces is more completely covered by lung tissue and does not strike the left side so forcibly as in the horse.

In the **sheep** the lung extends to the last intercostal space, nearly as far as its lower end and the heart is covered on the left side as well as on the right by lung tissue. The shoulder is very moveable and unless in very fat animals allows of an examination of the greater part of the anterior third of the chest.

In the **pig** fat and indocility combine to defeat our purpose in examination of the chest. If these can be obviated it is well to know that the diaphragm is attached to the upper two-thirds of the last rib, and to the next three in front above their lower third.

In the **dog** the diaphragm is attached to the upper two-thirds of the last rib, to the lower third of the next and to the lower ends of the two following and to the breast bone. The shoulders are so mobile and the breast bone so thin that nearly all the chest may be satisfactorily examined. The heart, covered on both sides by lung, lies nearly horizontally on the breast bone, through which its position and bulk may be clearly made out by percussion.

EXAMINATION BY TOUCH.

Pressure by the fingers in the spaces between the ribs corresponding to the pleura will cause flinching and perhaps grunting in pleurisy. The same result will be seen in pleurodynia. In hepatized lung and pleurisy with adhesions there is a diminished sense of the movement felt in the intercostal spaces of the part in health.

PERCUSSION.

Methods. Tissues as good and bad conductors of sound. Immediate, mediate percussion. Bilateral symmetry and divergence. Effect of building, race, etc. Horse, left side, right. Ox, left side, right. Effect of 1st and 3d stomachs, liver, etc. Sheep, diaphragm, heart. Pig, fat, lean, heart. Dog, method. Birds, back, ribs. In disease, increase, decrease, absence of resonance, in large area, in patches. Crack pot sound.

This consists in striking the walls of the chest so as to bring out the resonance of the parts. In proportion as we tap gently with the tip of the finger or strike forcibly with the closed fist will we elicit the sounds from the superficial or the deeper parts of the lung. Hence slight blows only must be used when the lung tissue is thin, to avoid bringing out the resonance from the deeper seated organs, and both must be resorted to when the lung is thick to ascertain its condition at the various depths. Where a moderate force is requisite the four fingers and thumb of the right hand are brought together in a line and the weight of the hand as moved from the wrist is employed to bring out the sound. The ribs being hard convey sound best from the deeper parts, and on them percussion is usually made. Care should be taken not to mistake the lesser resonance conveyed through the soft tissues of the intercostal spaces for an indication of a diseased condition. In proportion too as the ribs are covered with flesh or fat, the resonance will be diminished and a stronger blow will be necessary to bring out the sound from the lungs.

If the blow is made directly on the side of the chest the percussion is called *immediate*; if made upon an elastic solid body (plexiметer) laid on the outside of the chest it is *mediate*. The readiest and perhaps the best plexiметer is the middle finger of the left hand which is to be applied flat upon the side of the chest to receive the blow directed perpendicularly to its surface. In fat or fleshy subjects it should be pressed firmly on the surface so as to compress and condense the soft parts and render them better conductors of sound. Some use flat pieces of ivory, silver, caoutchouc but in employing these the nails of the right hand must be carefully pared, lest by striking the solid body they produce a sound which interferes with the true pulmonary resonance.

In examining the chest the two sides should be compared and if allowance is made for the dulness felt in the lower half immediately behind the left elbow caused by the position of the heart, and the deadness of the sound on the last few ribs on the right side where the liver is situated, any further deviation from a bilateral symmetry of sound is indicative of disease. The general resonance will be decreased by a full stomach which prevents the full inflation of the lungs, and it will be increased if the animal stands on a wooden floor with an empty space below. A short statement of the degrees of resonance over the different parts of the chest in the various races of the domestic animals in a state of health may prove useful.

Horse.—Left side. In the upper third the resonance is full behind the shoulder. It diminishes from the 13th rib backward and from the decreasing thickness of lung the blows should become less and less powerful. In this space forcible striking brings out the drum like resonance of the abdominal organs.

In the middle third the sound over the 5th and 6th ribs is distinct but not full ; it increases to the 11th rib and then decreases to the last.

In the lower third a very slight resonance may be observed over the 4th rib. ; over the 5th, 6th, and 7th, where the heart approaches the surface the sound is dead ; while from this to the 13th rib a slight resonance may be made out.

Right side. The upper third resembles that on left side from the shoulder as far back as the 13th rib behind which anything above the gentlest blows brings out a drum like sound from the large intestine (double colon) especially. This is clear when that is distended with gas.

In the median third the resonance resembles that on the left side. In the lower third it equally corresponds as far as the seventh rib behind, which sound is dull because of the proximity of the liver.

Ox.—Left side. The upper third is clear in sound from the eighth to the tenth ribs, and behind this by gentle tapping to the second last (twelfth). Forcible striking, however, brings out the drumlike sound of the upper sac of the paunch which always contains more or less air.

The middle third has a clear resonance as far as the seventh

rib; this diminishes to the ninth, behind which it is usually replaced by a dullness due to the presence of food in the anterior part of the paunch. By drawing back the limb percussion may be employed over the first and second ribs as well.

In the lower third the first two ribs can be examined and a clear sound should be educed. On the fourth, fifth and sixth ribs there is a full resonance, the heart being here covered by lung tissue, contrary to the condition in the horse. From the seventh the sound becomes duller and the dead sound from the food in the rumen characterizes the lower fourth of the ninth rib.

Right side. From the shoulder the resonance gradually decreases in the upper third to the eleventh rib, beyond which the sounds obtained are only from abdominal organs. In the middle third considerable resonance is met with over the first and second ribs, it is very full and clear over the fifth, sixth and seventh, whence it decreases and is quite lost behind the tenth. In the lower third a clear sound can be elicited over the first, second, fourth, fifth and sixth ribs; this is lessened over the seventh and eighth, and completely lost behind the ninth. Any but the slightest blows over these three last ribs brings out the dull, solid sound from the liver.

A very full paunch greatly increases the anterior convexity of the diaphragm, and compresses the lungs into the anterior part of the chest. If the contents of the rumen are solid the resulting dullness on percussion might be mistakenly supposed to indicate consolidation of the lung. This source of error must be carefully guarded against.

Sheep. Percussion in the sheep differs from that in the ox chiefly in the following particulars: The diaphragm being attached to the last rib as in the horse, the diminishing resonance of the lung may be traced as far back as in that animal. Thus a pulmonary sound can be obtained in the upper third as far as the last intercostal space, in the middle as far as the second last, and in the lower as far as the fourth from the last. Over the lower part of the fifth and sixth ribs on the left side the resonance is remarkably clear owing to the great relative thickness of the anterior lobe of the left lung which here covers the heart.

Pigs. In fat pigs the results are almost negative. In lean animals the middle third on each side gives out a clear resonance

behind the shoulder as far as the seventh rib, from which it diminishes to the second last (thirteenth). The sound is less clear in the upper and lower thirds. On the fifth intercostal space below, and on the left side the sound is dull owing to the exposure of the heart through a slight notch in the lung.

Dog. Percussion is very satisfactory in this animal because of the amplitude of the chest, the thinness of its walls and the small bulk of the abdominal organs. In the upper and middle thirds on both sides alike the sound is clear and full as far back as the seventh rib, whence it decreases to the last. In the lower third a distinct but moderate sound marks the first eight ribs and is equally clear on the right and left sides. The thinness of the lung in its posterior part demands that percussion be effected by the middle finger only, without any movement of the hand. Unless the dog is very fat, good results may be obtained by percussion over the first and second ribs, the shoulder blade and breastbone.

Birds. In these and especially in the webfooted (ducks, geese,) the sternum is so thickly covered by flesh that no result can there be obtained. Beneath the wings, however, and upon the back percussion through the medium of a small coin as a plexímeter and with the middle finger alone, is valuable. Beneath the wing a clear sound may be drawn out over nearly all the ribs and on the back over a less extent (two and a half to four inches, according to size).

PERCUSSION IN DISEASE.

Increase of resonance without any perceptible modification in character is usually partial and depends on the increased distension of the air cells of one lung, or part of a lung, to make up for the loss of a part or a whole lung through hepatization or pressure by false membrane or from water in the chest. If a part of a lung is solid and impervious it gives a dull, dead sound, contrasting strongly with the increased clearness of the remainder. So with water in the chest, the clearness of the upper parts contrasts unmistakably with the dullness of the lower. By watching the advance or retirement of these symptoms the solidification of a lung and its process of clearing up, and the effusion

of water in the chest and its removal may be equally traced through all these stages.

If the increased clearness is confined to the upper, lower, or posterior border of one or both lungs, the sound being natural over all other parts, it indicates the existence of emphysema of the lungs, a condition almost constant in broken-winded horses.

If the sound is drumlike over most of the lung it is due either to extensive emphysema or to the presence of air as well as liquid in the cavity of the chest. In the case first noticed there will be the double action of the flank, the weak, dry, husky cough and the wheezing breathing; in the last there will have been the previous attack of pleurisy, and the application of the ear to the chest will detect a splashing sound constant or heard only at intervals or on rising. This should be carefully distinguished from abdominal gurgling.

Diminished resonance, noticed over an entire lung, may be due to congestion or œdema of the lung, to the formation of a thick false membrane over the inner surface of the ribs or to a false membrane enveloping the lung and preventing its due distension. Congestion will be distinguished by the blueness of the mucous membranes and the presence of a crepitant sound heard on auscultation. Pleurisy is known by the tenderness on percussion or on pinching the intercostal spaces, and by the presence in many cases of a friction sound. The sound may be further lessened in cattle by the deposit of tubercle on the inner side of the ribs, or the extensive deposition of miliary tubercle throughout the substance of the lung.

Absence of resonance, the sound brought out by percussion being similar to that obtained by practising it over the muscular masses of the haunch, is always partial. It is due either to hepatization or to water in the chest. Hepatization is distinguished by its rarely affecting the lower thirds of both lungs at once, by the presence of a crepitating râle round the margin of the area of dullness, and by the increased resonance and respiratory murmur over the sound parts of the same and the opposite lung. In water in the chest on the other hand a friction sound and much tenderness precedes the dullness; the tenderness continues and the dullness reaches ths same height on both sides of the chest, in the case of the horse. In the ox, water may exist on one side of the

chest only, but the tenderness on pressure and the absence of any crepitation serve to distinguish the case from pneumonia. In the smaller animals the position of the dulness may be altered by turning the patient on its back as the water always gravitates to the lowest point.

The presence of extensive deposits of tubercle, of cretaceous material in tubercular cows and sheep, and the presence of large cysts in the lung may give rise to dullness over a circumscribed area. Such areas of dullness are usually multiple with sound lung between.

A further modification known as the **cracked pot sound** is sometimes heard in horses and cattle. It may be aptly represented by laying the palms of the two hands together in such a way that they meet all round and leave an interval filled with air right in the centre. The back of the one hand is then struck against the knee when the noise of the air escaping gives the characteristic sound. It occurs in consumption or in the advanced stages of inflamed lungs when a large tubercle or abcess has burst into a bronchial tube and the resulting cavity opens into this tube by a narrow orifice.

AUSCULTATION.

Mediate and immediate auscultation. Methods, quiet, normal chest sounds, tubal, bronchial, vesicular, respiratory, cardiac. Juvenile respiratory murmur. Horse, left side, right. Ox, left side, right. Accidental sounds, rumbling, gurgling, crepitation, friction. Sheep, special features. Goat, force. Pig, Dog, Birds, morbid chest sounds. Increase, general, partial. Decrease, general, partial. Absence. Bronchial sound in excess, in improper place. Cavernous, amphoric, mucous sounds. Râles, sonorous, sibilant, mucous, submucous, crepitant, subcrepitant. Creaking, metallic, tinkling, gurgling, splashing, friction. Timbre of Cough. Palpitation. Mensuration.

This is a term used in medicine to denote the mode of exploring an organ by applying the ear over the region in which it is situated and deducing the healthy or diseased condition by the sounds heard. First employed by Lænnec in human medicine it was quickly availed of for the lower animals by Delafond and Leblanc.

Auscultation is **mediate** or **immediate**. **Immediate Auscultation** is practised by applying the ear directly upon the skin, either bare or covered with a handkerchief. In **Mediate Auscultation** an instrument called a stethoscope is employed to convey the sound from the surface of the body to the ear of examiner. The common stethoscope is formed of soft wood (cedar or ebony) or of gutta percha, is from five to seven inches long and a quarter of an inch in the bore. The end applied on the skin is widened into a funnel three-fourths of an inch across at the mouth; the opposite end is flattened out to apply to the ear, is about two inches in diameter and has a hole in the centre to convey the sound. A flexible stethoscope is also used either with one or two ear pieces and though less convenient in general than the common variety possesses this advantage when the heart is being examined that it conveys the sound without the impulse of that organ.

In mediate auscultation the ear should be closely applied to the surface, the right ear being used for the left side and the left ear for the right, but a preference should always be exercised in favor of that in which the sense of hearing is most acute. If a handkerchief is used a single fold only must be applied, otherwise

the two layers may rub on each other and produce distracting sounds. In mediate auscultation the instrument should be held perpendicularly to the surface, accurately applied alike to the skin and the ear, and pressed firmly on the surface to condense the soft structures beneath the skin and render them more conducting. If held by the hand care must be taken to avoid the slightest movement of the fingers on the stethoscope, and long hairs should be prevented from entering the tube as being likely to produce additional sounds.

Among other points the following must be attended to in auscultation. Avoid a position in which the animal can strike you with its hind limbs. If necessary in irritable or ticklish subjects have one fore leg held up. Select a quiet time and place, early morning or night is usually best. Endeavor to protect the patient from the irritation of insects or the examinations may be fruitless. Never auscultate over a contracting muscle; the sound of muscular contraction will prevent a correct result. If the natural sounds are indistinct increase them by exercise. The smaller animals are examined with the greatest facility standing upon a table or held in the upright posture with the body resting on the thighs or on the hind feet only. Birds can be held by the wings which may be raised and drawn inward towards the median line to expose the back and sides of the chest.

HEALTHY CHEST SOUNDS.

In all healthy animals two distinct sounds are heard over the chest:—the **tubal** or **bronchial** sound, and the **vesicular** or **respiratory murmur**. The **bronchial sound** caused by the air sucking through the larger bronchi is best heard by applying the ear to the breast over the lower end of the windpipe or to the upper third of the chest immediately behind the shoulder. The respiratory murmur is clear and full in the middle third of the chest immediately behind the shoulder. It is louder and more prolonged in inspiration than in expiration and in the right lung than the left especially in cattle and sheep in which the former is more capacious. It is louder in young animals than in old, hence the name of **juvenile** respiration applied by Leblanc. In thin animals it is better heard than in fat ones, the chest walls being thinner, firmer, and more conducting. In animals of a nervous tempera-

ment like the English racer it is more distinct than in the Norman, Clydesdale and other heavier breeds. Deep, broad capacious chests emit a stronger sound than such as are shallow, narrow and short. Exercise, fear or any excitement accelerating the respiratory act increases the sound. A full stomach, certain narcotics and other depressing influences lessen it. Other things being equal the sound is lower in cattle and sheep than in other domestic animals.

Horse. The ear pressed strongly upon the breast where the windpipe enters detects a strong blowing sound referable to the lower end of the trachea and the bronchi. In young foals a respiratory murmur is heard when the stethoscope is applied in front of the shoulder, the limb being meanwhile drawn backward. A similar murmur may be heard, but less distinctly over the shoulder blade at this age.

Left Side. *Behind the shoulder* in the *upper third* of the chest the sound is loud and somewhat harsh, the respiratory murmur being here supplemented by the noise of the air rushing through the larger bronchia. From the 13th rib the respiratory sound is alone heard and becomes weaker to the second last (17th).

In the *middle third* the respiratory murmur is moderately clear from the 4th to the 6th rib, it becomes louder and clearer to the 9th from which its force gradually diminishes and is lost over the 16th. In the *lower third* over the 4th, 5th, and 6th ribs the respiratory sound is replaced by the sounds of the heart, each beat being distinctly divided into two sounds, the first dull and prolonged, the second short and quick. The respiratory murmur is heard over the 7th and 8th ribs, is weaker on the 9th and lost over the 10th. In the middle and lower thirds but especially towards the posterior part of the chest, abdominal sounds are often heard. They consist chiefly in gurgling or in a noise like that caused by the air rushing into a bottle which has been turned upon its side when full of water. Such sounds are easily distinguishable from those occurring in a diseased chest as they bear no relation to the rythmical action of breathing.

Right Side. In the *upper and middle thirds* the sounds do not differ from those of the left side. In the *lower third* the respiratory sound is clear from the 4th to the 7th ribs; from this it decreases and is lost at the 10th.

Ox. In very lean cattle the respiratory murmur heard in front of the shoulder and over the scapula is more distinct than in the same region of the horse.

Left Side. In the *upper third* a clear respiratory murmur is heard over the 8th, 9th and 10th ribs but is lost about the 11th. In the *middle third* the vesicular sound is feeble at the lower margin of the region and immediately behind the shoulder because of the proximity of the base of the heart. Towards the upper margin it is loud and harsh being complicated by the *tubal* sound. It is full and clear over the 7th rib whence it decreases in force to be lost at the 11th above and the 10th below. In the *lower third* the double heart beat is alone heard over the lower part of the 4th rib, the respiratory murmur reappears over the 5th and 6th whence it becomes weaker and is lost at the lower and upper margin of the region respectively over the 8th and 9th ribs.

Right Side. The sounds of the *upper third* simply repeat those of the left side. In the *middle third* the chief difference is the greater clearness and strength of the respiratory and tubal sounds immediately behind the shoulder. In the *lower third* a moderately strong respiratory murmur is rendered harsh by a tubal sound due to the proximity of the large bronchus going to the anterior lobe of the right lung. The respiratory murmur continues with diminishing force to be lost over the 8th and 9th ribs.

Accidental but healthy Sounds. These are more loud and frequent in the ox than in the horse. There is the same irregular rumbling and gurgling especially on the posterior parts of the chest. Gurgling as from a full bottle inverted is often clearly heard over the last six ribs on the left side, and appears due to the passage of liquids between the paunch and honey comb bag. An occasional sound as of water falling into an empty barrel is heard in the same region in cases of slight tympany and after saliva has been swallowed. Rumbling sounds are chiefly heard over the last ribs on the right side where the large and small intestines are situated. The superadded sounds in the ox are those of *crepitation* and *friction*. The *crepitation* or fine crackling due to a dryness of the areolar tissue under the skin is frequently present in oxen in average health. A fine *crepitation* is also heard on the left side from the bursting of myriads of minute bubbles of air generated among the contents of the paunch during the

process of digestion. This is especially marked after the animal has fed on green food or potatoes. A loud *friction* or *rubbing* sound, which may be imitated by placing the back of one hand upon the ear and rubbing the palm of the opposite hand upon it, is likewise heard over the left side after eating. It is produced by the movements of the paunch during contraction and not being synchronous with the respiratory acts cannot be confounded with the friction sounds of pleurisy to be hereafter noticed.

Sheep. The diaphragm being attached to the last rib as in the horse the respiratory murmur may be heard to the second last. The shoulders being more movable than in the ox the anterior part of the chest can be more satisfactorily examined. The vesicular murmur is heard along the whole lower third on the left side though the heart sounds are superadded over the 4th, 5th and 6th ribs. Crepitation from the subcutaneous areolar tissue is rarely heard. Otherwise the sounds of the chest and abdomen correspond to those of the ox.

Goat. This animal differs from the sheep mainly in the greater force and clearness of the respiratory murmur.

Pig. It seems ridiculous to speak of auscultating the pig, yet he is sometimes thin enough and quiet enough to permit of one obtaining satisfactory results. Gentle treatment and scratching the back and abdomen will often persuade him to be temporarily quiet and docile. The vesicular murmur is very clear in the middle third of the chest on either side, but diminishes gradually on the last six ribs, and disappears on the second last. It is much less intense in the upper and lower thirds. In the posterior part of the chest rumbling and gurgling abdominal sounds are frequent.

Dog. The respiratory murmur is very clear over the whole chest. It is most intense along the middle third and becomes less clear on the 4 or 5 last intercostal spaces. The mobility of the shoulder permits an examination of nearly the entire chest. The respiratory murmur may be heard over the entire length of the lower third on the left side though the heart's sounds are equally heard over the 4th, 5th and 6th ribs. Rumbling and gurgling abdominal sounds are much less frequent than in herbivora and omnivora.

Birds. The respiratory murmur is loud, clear and almost harsh on the sides of the thorax, beneath the wings, and considerably softer as heard on the back.

MORBID CHEST SOUNDS.

The close study of the healthy chest sounds upon the living animal is an essential prerequisite to the appreciation of the morbid. The abnormal noises are so varied, merge into each other by such imperceptible degrees, and so coexist and complicate each other that they often prove extremely puzzling to the unpractised ear. It is no more necessary that the musician should educate his ear to appreciate the most delicate gradations of musical notes, than that the auscultator should educate his in the sounds of the healthy and diseased chest. Written instructions are of about equal value in the two cases, they prove auxiliaries in the acquisition of knowledge but they can never supersede the practical study of the chest. A mere theoretical knowledge is too often useless in the presence of the patient.

The abnormal chest sounds are either modifications of those existing in health, or superadded sounds which have no counterpart in the healthy chest.

Modifications of healthy sounds. The **vesicular** or **respiratory murmur** may be **increased** or **diminished** in force or it may be entirely **absent**.

Increase of the respiratory murmur, is merely an increase in force without any modification in character and resembles *juvenile* respiration. If increased equally over the entire chest it is **general,** if only in a part it is **partial. General increase of the vesicular murmur** is heard after an animal has been submitted to moderate exertion for ten or fifteen minutes. In animals at rest it is heard in active fevers and in the symptomatic fever which attends acute inflammations.

Partial increase as for example in one lung only, or in circumscribed parts of both lungs, and especially along their superior borders, is indicative of disease of the lungs or the pleuræ. It testifies to the impermeability to air of some other portion of lung, from congestion, splenisation, hepatisation, plugging of a bronchial tube with tenacious mucous, tubercular deposits, tumors, emphysema, or hydrothorax. (See under these names.) The healthy portion of lung in such cases takes on the function of the whole, and the loud breathing is called **supplementary.**

Diminution of the respiratory murmur. like its **increase,**

may be partial or general. **General diminution** is seen in anæmia, in low fevers, in all very prostrate conditions from the mere want of power to dilate the chest; in general emphysema (broken wind, heaves), in general miliary tubercular deposit in the lungs, or in that form in cattle in which the tubercle has been replaced by cretaceous deposits, from the animal's inability to fully dilate the air cells; in enteritis, peritonitis and metritis the chest is more fully dilated because of the pain attendant on that act, and the breathing being short and quick the murmur is correspondingly low. In certain brain diseases with sluggish respiration the sound is equally feeble.

Partial diminution of murmur is more surely indicative of lung disease. It may arise from partial congestion when a supplementary murmur will be observable over other parts of the lungs, and a crepitant râle soon appears in the congested part; from local emphysema in which there is increased resonance in percussing the part; from tubercular or cretaceous deposit, when there will be exaggerated murmur elsewhere, or from bronchitis with blocking up of one or more small bronchial tubes and with louder respiratory sound in other parts.

Absence of respiratory murmur may be due to various causes, all of a diseased nature. Hepatisation of lung may be recognized when this condition is found associated with a crepitating râle around the margin of the silent part, and when percussion shows its solidity and want of resonance. Splenisation is associated with absence of respiratory sound and dullness on percussion, but no surrounding crepitation. Absence of sound in water in the chest is confined to the lower part of the chest, keeps the same level and ratio of increase in front and behind, and in the horse on the two sides, and has been preceded by the characteristic catching breathing and the friction sounds of pleurisy. Large tumors and extensive and circumscribed tubercular deposit will give rise to absence of sound over a limited area and plugging up of one or more bronchial tubes will lead to a similar result. Hepatisation of lung and water in the chest are, however, the common causes of loss of respiratory murmur.

The **bronchial or tubal sound** may be increased in pitch and in harshness in two conditions. 1st. In the early stages of bronchitis when the lining mucous membrane of the air passages is

dry, thickened and inelastic. 2d. When that portion of lung intervening between one of the larger tubes and the surface of the chest is solid (hepatised) and thus proves a better conductor of sound than in the normal condition.

Superadded abnormal sounds. The **bronchial** sounds may be altered in their character so as to become **cavernous, amphoric** or **mucous** (rattling). The **cavernous** sound is usually caused by the presence in the lung of the cavity left after the discharge of an abscess or softened tubercle into a bronchial tube. It is thus preceded by cough and white, creamy discharge from the nose. If the discharge is fetid and grumous there has probably been circumscribed gangrene of the lung. An approximation to the sound may be produced by blowing into a wide-mouthed glass or porcelain vessel. The sound of **amphoric respiration** on the contrary is like that made by blowing into a narrow-necked bottle. It is due to a similar cavity with a small orifice or to the existence of pneumo-thorax communicating by a narrow canal with a bronchial tube. It is rare in the lower animals, but Delafond mentions one case in the horse and two in dogs.

Râles. The remaining morbid sounds are known as *râles*, or rattles. They may either be referable to the bronchial tubes or the lung tissue. They are called dry or humid, according as they convey the idea of air drawn through a dry tube or one containing liquid.

The **dry râles** are due to narrowing of the bronchial tubes from the pressure of adjacent tumors, the thickening of the mucous membrane or the deposition on the surface of layers of tenacious mucus. The greater the narrowing the shriller the sound, and hence the distinction of *bronchial rales* into **sonorous** and **sibilant** (whistling).

The **sonorous râle** has been variously exemplified by the humming of a gnat, the cooing of a wood pigeon or the bass notes of a violin. It commonly bespeaks the onset of bronchitis and testifies to the thickened, dry and rigid character of the tubes, but may give place in as short a time as three hours to a mucous râle from the occurrence of a free secretion. It rarely extends over two or three days. Sometimes when caused by a piece of tenacious mucus obstructing a tube, it is very transient disappearing

at once when the mucus is expelled by coughing. Sometimes it is modified by an occasional clicking sound from the flapping of a shred of semi-solid mucus attached to the walls of a bronchial tube. This disappears when breathing becomes more hurried.

The **sibilant** (whistling) **râle** often acknowledges the same causes as the sonorous, but indicates a narrower closure of the tubes. More frequently it is heard further back on the chest and results from pulmonary emphysema and dilatation of the smaller bronchial tubes (broken wind, heaves). It is then heard chiefly in expiration and coincidently with the second quick lifting of the flank. It is further associated with the double lifting of the flank in expiration with the short, weak, paroxysmal cough and the indigestion characteristic of broken wind. If the whistling noise is so loud as to be heard without applying the ear to the chest it is called wheezing.

A **mucous râle** is caused by air passing through any liquid contained in the bronchial tubes, such as mucus, pus, or blood. It may be imitated by blowing a large number of soap bubbles in a thick lather and noticing them burst simultaneously or successively. It is chiefly observed in bronchitis after the preliminary dry stage of the mucous membrane has passed off and an abundance of mucus has been secreted. The nature of the sound will vary according as it comes from the larger or the smaller tubes or in other words as to whether the bubbles are large or small. That from the smaller tubes is sometimes called a **submucous râle**. Either of these râles may be temporary or permanent as the mucus may be momentarily cleared away by coughing.

The **crepitant râle** is a sound of very fine crackling which has been variously compared to the crackling of salt when put on red hot coals, the noise of a sponge expanding in water and the rubbing of a small lock of hair between the finger and thumb close to the ear. The existence of the *crepitant râle* usually denotes the existence of the early stage of inflammation of the lungs, and the progress of hepatization in such cases may be traced by the advance of the line of crepitation which precedes it. So the progressive absorption of exuded matter in recovery may be equally followed by a line of crepitation gradually decreasing in area until it meets in a point. The observations will be coroborated by the dull sound elicited on percussing the parts. The production of

the sound has been attributed to the passage of air through the thick mucus in the smallest bronchial tubes or more plausibly to the separation of the walls of the air sacs and cells during inspiration, they having been previously adherent by reason of the secretions.

Crepitation is not heard in all pulmonary inflammations. In weak animals with a low type of inflammation tending to gangrene, and in those cases of broncho-pneumonia in which a viscid mucus blocks up the bronchial tubes passing to the affected lobes, it may be altogether absent.

Crepitation may further occur without inflammation. Thus in pulmonary œdema (dropsy of the lung) and capillary hemorrhage in which liquids are effused in the smaller bronchial tubes and air sacs a crepitation is sometimes heard.

A **modified crepitation** (**dry crepitant râle** of Delafond) is usually heard over an emphysematous lung. The noise in this case has been compared to that induced by handling a sheet of paper.

The **subcrepitant râle** is another modification holding a place intermediate between the crepitant and the mucous râles. It has been likened to the sound of a moderate effervescence in beer or other liquid. It is referable to the presence of mucus in the smaller bronchial tubes and indicates bronchitis or broncho-pneumonia.

Still other sounds are heard in diseased conditions of the pleuræ. These are **friction sound**, **creaking**, **metallic tinkling**, and **gurgling** or **splashing**.

A **friction sound** is heard in the early stages of pleurisy and is caused by the dryness of the pleural surfaces from the absence of the halitus or vapor which normally moistens them and the deposition of layers of lymph by which the surfaces are rendered rough and uneven. An approximate sound may be observed by placing the palm of the left hand on the right ear and drawing a finger of the right softly over its back. The sound is quick and jerking, one or a few jerks only being heard with each inspiration as the act is cut short on account of the pain attending the friction. It is rarely heard in expiration. It is chiefly heard at the lowest part of the chest where the lungs have the greatest freedom of movement. The thinness of the walls of the chest above the breast bone in cattle and dogs permits the friction sound to be heard more

distinctly than in the horse. After the lapse of twelve, twenty-four or forty-eight hours the friction sound disappears, the surfaces of the pleuræ being separated by the liquid effusion, but it may reappear when the fluid is absorbed in the process of recovery. Sometimes the friction is further manifested by vibration of the walls of the chest perceptible to the touch.

The **creaking sound**, as from the bending of a piece of strong leather is caused by the movement of a thick and solid false membrane binding the lungs to the side of the chest. This is often confounded with crepitation.

Metallic tinkling is only heard when liquid and gas both exist in the pleural sac and is due to the falling of a drop from the shreds of false membrane above into the fluid contents below. The sound is somewhat like the falling of drops in a closed cask half full of water, or it may be fairly exemplified by placing the palm of the left hand flat on the right ear and striking the back of the hand smartly with the middle finger of the right. The sound is chiefly heard after the patient has changed its position and especially after rising. The explanation of this is that in the recumbent position the liquid changes its place and bathes parts which in standing are surrounded by gaseous products only. Drops accordingly fall into the liquid for some time with diminishing rapidity until they cease altogether. Other explanations of the sound but which less frequently exist are: the ascent of a bubble through the liquid and its bursting on the surface; and the sudden recoil of air from one wall of the plueral cavity to the other as the result of movement or sound generated in the deeper seated solid structures.

A **gurgling** or **splashing** sound is equally indicative of the presence of fluid and gas in the pleural sac. It is almost never heard unless after a sudden movement on the part of the patient causing considerable commotion in the contained liquid. Gurgling sounds transmitted from the abdomen are too often mistaken for this. In small animals with hydro-pneumo-thorax a quick shaking of the patient will develop it.

Auscultation of the Cough is sometimes valuable, though more difficult and less satisfactory in the lower animals than in man, chiefly because of the extensive movement of the ribs in the former. As conveyed through a healthy lung to the ear applied

on the side of the chest, the sound is short, dull and indistinct. When the lung is more solid from hepatisation, pleural exudation or other cause, or when the bronchi are dilated the sound is loud and strong. The extent over which it may be heard thus forcibly agrees with the area of lung in a state of consolidation. When a considerable cavity or canal communicates with a bronchial tube and extends to near the surface of the lung the sound is loud and ringing. The note is specially clear and metallic when such a cavity opens into the bronchus by a narrow orifice; an apt illustration of this noise may be obtained by coughing into a narrow necked vessel.

The results obtained by auscultation should be confirmed by percussion before arriving at any definite conclusion as to the state of the chest. Consolidated lung tissue is a much better conductor of sound than the healthy, and sounds conveyed through this may be heard at a considerable distance from their point of origin. Thus the heart sounds are frequently heard over any part of the right side of the chest, and crepitation and other sounds may be heard in the centre of a hepatized portion. On all such occasions the dull sound elicited on percussion will not fail to correct the fallacy.

PALPATION. TOUCH.

This is chiefly useful in cases of pleurisy. As already noticed the vibration of the chest walls which accompanies the early friction sound is sometimes perceptible by the hand applied on the side of the chest. Pressing firmly in the intercostal spaces at the affected part invariably causes wincing and in cattle grunting. Pinching the back in inflammatory chest diseases in cattle but especially in pleurisy has a similar effect.

MENSURATION.

Measurement of the chest gives less reliable results in the lower animals than in man. A cord four feet long should have one end placed on a definite point on the withers and not removed until both sides have been examined. It should be first carried down to a point in the middle of the breast bone and the distance marked by a knot; a comparison may be made by carrying to the same point over the opposite side. It should next be carried suc-

cessively to the lower end of the 8th rib on the two sides and the difference marked, and lastly from the lower end of the third rib to the lower end of the eighth. These measurements should be made at one stage of the respiratory act, say when the chest is fully dilated, and similar measurements when the chest is collapsed to ascertain any difference in the expansion of the two sides of the chest. In the smaller animals any difference in the expansion of the two sides may be observed by inspection only, the practitioner standing directly behind the animal and watching the movements of the two sides from this standpoint.

A permanent dilatation of one side may be seen in water in the chest confined to one side, and particularly if of some standing. Complete hepatisation of one lung gives a similar result. The intercostal spaces are observed to be wider than usual in such cases, and the movements of the opposite side of the chest are much more extensive than of the affected one.

A collapse with limited movement of one side is an accompaniment of chronic disease of the lung, with wasting of its substance as in cases of tubercular deposit.

DISEASES OF THE LUNGS.

Divisions of lung diseases. Bronchitis, pneumonia, pleurisy, their results, nervous disorders, asthma, hiccough. Œdema. Emphysema. Morbid growths, neoplasms. Infectious and parasitic diseases.

Inflammatory diseases of the respiratory organs situated within the chest may be divided into: inflammation of the air tubes within the substance of the lungs—*bronchitis*:—inflammation of the spongy tissue of the lung—*pneumonia*:—inflammation of the covering of the lungs and lining serous membrane of the chest—*pleurisy*:—and complicated cases in which two or more of these conditions coexist. Beside inflammatory diseases there are the various permanent morbid results of these affections, such as consolidation of lung from exuded products becoming organized; collapse (compression) of lung from organization and contraction of false membranes, thickening or dilatation of bronchial tubes as a result of bronchitis; also nervous affections, such as asthma and hiccough; morbid alterations in the lung tissue independently of inflammation, as pulmonary or pleural œdema and emphysema; specific morbid deposits, as tubercles, glander nodes, cancer, melanosis, etc., and morbid states, due to parasites, as in the verminous affections of cattle, sheep, etc.

BRONCHITIS.

Relation to other maladies of the air-passages. Horse. Causes, susceptibility, heat, cold, sudden changes, thick coat, rebreathed air, on shipboard, in zoological gardens, in close stables, in navies, organic matter in expired air, water vapor in expired air, effect on the air and bacteria. Ingesta in bronchia. Medicinal liquids in bronchia in horses and cattle. Exposed locations. Clipping. Smoke and gaseous irritants. Symptoms, in mild cases, in severe: fever, cough in dry stage, after secretion, anscultatory sounds, percussion, discharge, watery, glairy, frothy, later milky, flocculent, purulent. Convalescence. Capillary and pseudomembranous form. Intensity of symptoms, labored breathing, dyspnœa, violent cough, pinched countenance, dark mucosæ, perspirations, palpitations, asphyxia. Course, duration. Termination, difficult expectoration, blocking of bronchia, pneumonia, bowel susceptibility, skin congestion, laminitis. Chronic condition. Lesions, congestion and contents of bronchia, soft, thick, friable mucosa, absence of vascular ramification, tenacious mucus, false membranes. Collapse, atelectasis, splenisation, emphysema, bronchiectasis. Treatment, in mild cases, in severe, hygienic, steaming, sulphur dioxide, derivatives, guarded laxative, neutral salts, calmatives, expectorants, alkalies, stimulant, oxygen, peroxide of hydrogen, iodide of potassium. Diet. In advanced stages tonics.

Definition. Inflammation of the mucous membrane which lines the bronchia. It is the counterpart of *coryza* and *laryngitis*, being but the inflammation of another portion of the same mucous membrane which lines the whole respiratory track. That portion of this mucous membrane which lines the trachea is rarely or never the exclusive seat of inflammation, so that in case of its being implicated we do not speak of the case as one of *tracheitis* but as *laryngitis* or *bronchitis*, according as the throat or bronchia form the seat of active inflammatory action.

The bronchial mucous membrane is often inflamed in influenza, strangles, contagious pleuro-pneumonia of cattle, distemper in dogs, and parasitic diseases of the lungs, but the following remarks will be confined to the simple inflammatory affection. It appears as an *acute* and a *chronic* affection.

HORSE. ACUTE BRONCHITIS.

This is more frequent in the horse than in other animals, and

especially so in young animals when newly stabled or put in training.

Causes. These are the same as those of *catarrh* and *sore throat.* It is but the continuation of the same mucous membrane which is affected in all alike, and the same atmospheric changes, hot stables, noxious inhalations and exposures to cold and wet will induce this disease rather than the others when the bronchial mucous membrane is more predisposed. Bronchitis often supervenes upon sore throat, by the extension of the inflammation downward into the chest. Chilling of the surface by exposure to cold, drenching rains, is a frequent cause, by reason of the intimate sympathy existing between the skin and the mucous membrane. For the same reason certain conditions of the skin will predispose, thus a long, thick coat which keeps the animal constantly drenched with sweat and the skin relaxed and sensitive. Williams draws attention to the frequency and severity of bronchitis in both horses and cattle conveyed by sea during stormy weather, and especially when the hatches had to be fastened down. Such an experience combines in one the evils of an overheated stall, a sudden transition often to extreme cold, a lowering of the vitality of the whole system by the circulation of non-ærated blood, a systemic poisoning by the retention of the waste organic products that would otherwise have been eliminated, and the special weakening of the lung tissue by congestion of the whole pulmonic circulation.

But the development of bronchitis and broncho-pneumonia is the least fatal result. The statistics of our European cattle traffic are rich in the examples of absolute suffocation of cargoes in transit to Europe. The following from Report of U. S. Treasury Cattle Commission is illustrative:

"Dr. Thayer reports the case of a steamer from Boston to Liverpool, with 400 cattle on board, which encountered a storm and came through it with only one animal surviving. Mr. Toffey, of Jersey City, lost 30 head out of a cargo of 300 by suffocation in 1880. This happened, he informs us, on a calm sea on a southern route with a temperature about 90° F., and the wind astern and light so as just to keep pace with the ship. The air on board the ship became perfectly stagnant, and there was no means of establishing an artificial current. A still more disastrous experience

befell the steamer Thanemore, Captain Sibthorp, of the William Johnson & Co. line. This vessel left Baltimore with 565 cattle on board, of which 228 perished by suffocation before she reached Cape Henry."

Among animals that survive such treatment the susceptibility to lung disease including even the contagious forms like tuberculosis is enormously enhanced.

EFFECTS OF MODERATELY VITIATED AIR.

"When air only moderately vitiated is breathed continuously for a greater length of time the results are still very injurious, and in the front rank of diseases so caused stand pulmonary consumption, and other destructive affections of the lungs. Perhaps no better example of this can be given than that of the monkey houses of the Zoological Gardens of London and Paris. While these houses were small and ill-ventilated the monkeys died in large numbers from pulmonary consumption, but after they had been enlarged and better ventilated the mortality from this cause nearly ceased. (Arnott.)"

"Town dairy cows which are packed in close ill-ventilated buildings and never allowed to go out are very subject to consumption, while horses kept in no better conditions, but spending nearly half their time in the open air, rarely have phthisis. (With lung plague it will be remembered that the out-door exercise and mingling of herds leads to an increase of the mortality.) Horses newly stabled suffer severely from diseases of the lungs. The same holds true of human beings. A long list of careful observers have noticed the essential connection of lack of ventilation and pulmonary consumption. Baudelacque, Carmichael, Arnott, Lepelletier, Allison, Sir James Clark, Toynbee, Guy, Greenhow, Sir Alexander Armstrong, Parkes, and Aitken have especially insisted upon consumption being a sequence of lack of ventilation. Dr. Cormac indeed insists with great force that consumption is originated by rebreathed air.

"The notorious prevalence of consumption in sailors has been directly traced to the impure air in which they sleep, and an extensive outbreak of lung disease (not tubercular), leading to destruction of lung tissue, in the English Mediteranean squadron

in 1860 was clearly traced by Dr. Bryson to the contamination of the air. In a nursery hospital at Dublin with entire neglect of ventilation, 2,944 children died in four years, whereas after the ventilation had been improved only 279 died in the same length of time."

"Parkes (Practical Hygiene) says:

"'But not only phthisis may be reasonably considered to have one of its modes of origin in the breathing of an atmosphere contaminated by respiration, but other lung diseases, bronchitis and pneumonia, appear also to be more common in such circumstances. Both among seamen and civilians working in confined, close rooms, who are otherwise so differently circumstanced, we find an excess of the acute lung affections.'

In this connection, the statement of the air breathed by an ox per hour and that supplied him on board a ship with insufficient ventilation or none may be instructive. The ox takes in with each breath about 5 liters of air. This is at the rate of 50 liters per minute, or 3,000 per hour = 105.9 cubic feet. This amount of air is therefore rendered all but irrespirable by each animal in the course of an hour. And this, be it noted, is by breathing alone, and makes no account of the contamination by perspiration in the overheated hold, and by the emanations from the accumulating excrement."

"On board the steamers we have found the space alloted to each bullock to vary from 150 to 240 cubic feet. On the steamship "Holland," loaded at New York, August 21, 1881, we found the stalls amidships allowed the full space of 240 cubic feet per head. In the bow where there was less height between the decks the space was considerably less. On the lower deck, where 129 cattle were accommodated, the space allowed each was 217.4 cubic feet. The port holes in the upper deck were nine inches in diameter and there was one for each pair of stalls—central and lateral —or for eight oxen. These being well above the water line would be available for ventilation in ordinary weather. The port-holes in the lower deck, similarly arranged, were about two feet above the water line, and consequently not available for ventilation, save in exceptionally calm weather. The temperature on the main deck of this ship (between the outer and main deck), when only half the cattle had been loaded, was in the neighborhood of 90°

although she was lying in the center of the North River with port holes and hatches open, and a fresh breeze blowing from the north."

"On the 'Assyriaan Monarch' the space per head was only 192 cubic feet, but this ship was supplied with a ventilating fan or blower capable of delivering over 50,000 cubic feet of fresh air per hour, so that her ventilation was abundantly provided for. In some smaller ships we found the space per head to exceed little, if at all, 150 cubic feet. In these, accordingly, a single hour without any change of air would threaten the life of every animal on board, and two hours would endanger those for which even the larger space is provided. It is true that such absolute seclusion is rarely required, and that a certain amount of aerial diffusion is always going on through imperfectly closed hatches, companion ways, and ventilators, yet that these are often insufficient has been amply shown by such losses as are reported above, as well as by the bronchitis and tuberculosis which Drs. Whitney, Lyman, and Williams have found in the lungs of American animals arriving in England."

"ORGANIC MATTER IN EXPIRED AIR."

"The decomposing organic matter given off by the lungs and skin is probably the most injurious of the animal excreta, when allowed to act on the system for a length of time. This exhaled organic matter is easily recognized in the air by chemical tests, or by the putrid odor evolved when cotton wool, that has been breathed through, is left to soak in otherwise pure water at a temperature of 70° to 80° Fahrenheit. The experiments of Gavarret and Hammond, in which expired air had its carbonic acid and water vapor removed, leaving only the organic matter, showed that the latter was highly deleterious. Hammond found that a mouse died in forty-five minutes in such an atmosphere. It has also been again and again demonstrated that air containing a given amount of carbonic acid as the result of respiration is far more poisonous than air which contains the same amount of carbonic acid as a product of combustion."

"WATER VAPOR IN EXPIRED AIR."

"The amount of water vapor given off by the lungs varies

greatly according as the air is already more or less saturated with water. As the air in the stalls between decks is always saturated with water vapor, we may take the very lowest estimate for each animal, namely, 60 ounces in 24 hours, which for a cargo of 200 head would amount to over 93 gallons. And this is in addition to the exhalations from the skin and the bowel and kidney excretions. The air between decks is therefore constantly saturated with moisture which condenses and runs down in streams on every solid object. Among the ill effects of this saturation may be noted : "

" First. The saturation of the air with water vapor increases the exhalation of carbon dioxide from the lungs. This effect on the excretion of carbonic acid is usually so great as to counterbalance the tendency of warm air to reduce the production of this acid. This saturation, therefore, with water increases the danger of suffocation by the accumulation of the irrespirable carbon dioxide in the ship, unless the air is being constantly removed. "

" Second. The excess of moisture in the warm atmosphere hastens the decomposition of the organic matter derived from the lungs, skin, and manure. Sir Alexander Armstrong, head of the medical department of the British Navy, says : " There can be no more fertile source of disease among seamen, or, indeed, other persons, than the constant inhalation of a moist atmosphere, whether sleeping or waking ; but particularly is this influence injurious when the moisture exists between a ship's decks, where it may be at the same time more or less impure, and hot or cold, according to circumstances." It has become an aphorism with sanitarians that " a damp ship is an unhealthy ship," and many instances are adduced in which a sufficient renewal of the air between decks, with or without stoves to dry it, has transformed a naval pest-house into a salubrious vessel."

" All such considerations must emphasize the demand for such a constant renewal of air between decks on steamers carrying cattle as shall serve to obviate all those conditions of ill-health, with congestion and inflammation of the lungs, as have proved in the past a serious drawback to our foreign cattle-trade. To accomplish this and at once remove from between decks the excess of carbon dioxide, of decomposing organic matter, and of humidity, and to furnish air approaching in purity and dryness that of the

atmosphere outside, we can conceive of nothing more simple and effective than thorough ventilation by fan or heat extraction, as referred to below." Report of the U. S. Treasury Cattle Commission, 1882.

The above quotations were written with special reference to cattle but the author reproduces them here as in principle applicable to horses as well.

In both horses and cattle treated as above it is common to find ingesta in the bronchia drawn in during the violent paroxysms of coughing. Here we have a direct mechanical irritant and a means of septic infection, highly calculated to induce unhealthy broncho-pneumonia. Williams quotes the case of a horse in which vomition was caused by an over dose of aconite, and a portion of the food entered the bronchi.

In this connection must be named the introduction into the bronchia of liquids forcibly administered to horses and cattle. In the horse the length of the soft palate enables him to hold liquids in the mouth during his pleasure, and among the expedients adopted to coerce him are the very dangerous ones of holding the nostrils and of pouring the liquid through the nose. When the nostrils are held the urgent demand for air leads to attempts to breathe through the mouth, and, whether he succeeds in this or not, the usual result is the drawing of a portion of the liquid into the lungs. When it is poured through the nose the animal cannot protect himself except by rapid gulping, and as he must breathe, a portion of the liquid is usually drawn into the lungs. Any irritant taken in this way will develop bronchitis, and some bland agents like melted lard are almost equally injurious. Cattle having a short palate can scarcely resist swallowing liquids that are poured into the mouth, but a cough with the succeeding quick inspiration will almost certainly draw a portion into the bronchia. To return to the influence of cold, exposed situations which receive the full force of cold winds, those from the north and west on the Atlantic slope are specially conducive to bronchitis. Exposure of newly clipped animals to stand without protection in winter or early spring, has the same tendency. Finally the inhalation of smoke or of heated and irritant gases and vapors, as in a burning building, is an effective factor.

Symptoms. In its *mildest form* bronchitis is a transient illness

with some dullness, impaired appetite, hot, dry mouth, redness of the visible mucous membranes, a moderately strong, resonant cough, attended with slight pain, slight rise of temperature, accelerated breathing and pulse, and mucous discharge from the nose. Such an attack passes over in a few days and without any medicinal treatment if ordinary precautions are taken to avoid a repetition of its causes.

In *severe cases* the symptoms are more intense from the first. Besides the dullness and inappetence, hot, dry mouth, generally increased temperature of the body (102° to 104° F.), accelerated and labored breathing, and other manifestations of fever, there are more specific symptoms. The cough is dry, hard, painful, often paroxysmal, and appears as if it came from the very depth of the chest. A strong, harsh, bronchial sound is heard over the lower end of the trachea and the upper border of the middle third of the chest just behind the shoulder. Percussion detects no change from the natural resonance of the chest, nor auscultation any crepitating sound. Pressure in the intercostal spaces causes no suffering. The expired air feels hot. The pulse though accelerated is moderately soft and sometimes even weak, a condition which marks inflammations of mucous membranes as contrasted with those of the serous. The mucous membrane of the nose has a dark red hue, especially when the inflammation extends to the smaller ramifications of the bronchial tubes so as to impair the æration of the blood. In the same state there is excessive dullness and prostration because of the supply of partially venous blood to the brain. The head is held low, the nose often supported upon the manger, and the eyelids are semi-closed and injected.

From the second to the fourth day a free exudation takes place from the surface of the mucous membrane, and the symptoms are materially changed. The cough becomes more frequent but softer, looser, and attended with a rattle from the air passing through the abundant mucous secretion. The cooing or tubal sound heard at the lower end of the windpipe and behind the shoulder has now given place to a *mucous râle*. A nasal discharge appears at first watery, thin, of a whitish, glairy froth, but soon becoming more opaque, white, milky and flocculent and having little tendency to stick to the nostrils. This is often ex-

pelled with sneezing and accompanied by movement of the jaws. With the access of free secretion there is a great mitigation of the fever and the other distressing symptoms, and, if no relapse nor complication supervenes, recovery may be complete in a fortnight or three weeks from the onset.

From this time all the febrile symptoms decline and disappear, appetite and liveliness return, the discharge rapidly diminishes and finally disappears, when the patient may be said to have completely recovered.

Capillary and pseudo-membranous bronchitis are described by Reynal as occurring in young horses recently brought into the army and subjected to the hot and close stables in some of the French barracks. It began as ordinary bronchitis, which in place of tending to recovery, propagated itself to the most minute bronchial ramifications, and was frequently complicated by the formation of false membranes. The signs of its accession are an extreme intensity of the general symptoms, the rapid, labored, difficult breathing, accompanied by convulsive action of the pectoral and abdominal muscles; the frequent, painful, suffocating and abortive cough, which violently shakes the whole body; the extended head, open mouth, distorted nostrils, reddish brown protruding eyes; the pinched, haggard features, and the frothy mucous, nasal discharge striated with blood, and later interspersed with shreds of false membrane similar to those existing in croup. In connection with these are the symptoms of extreme oppression, partial sweats, tumultuous action of the heart and small, weak, rapid pulse. Death resulted from suffocation during a paroxysm of coughing.

Course. Duration. Termination. Bronchitis is not usually fatal, except in very young or old or worn out animals, or unless it assumes the *capillary* form or is complicated by pneumonia, pulmonary abscess or by metastasis to the bowels or feet. In the mildest cases health is re-established in three or four days, and in the severe, about the twelfth, fifteenth or twentieth day. In old and debilitated animals in which pure bronchitis proves fatal, the abundant effusion into the bronchial tubes, the influence of gravitation retaining this in the smaller tubes, the palsy of the cilia which normally carry it outward, and the want of power to expectorate by coughing, usually bring about suffocation. This

is favored by the nonœrated state of the blood, which rapidly prostrates the already weakened nervous centres. The supervention of pneumonia will be marked by a new class of symptoms, especially labored breathing, dullness on percussion and crepitation on auscultation. The susceptibility of the bowels is so great in some cases of bronchitis, particularly in those associated with a low fever, that superpurgation, enteritis and death may result from the smallest dose of laxative medicine,—the author has seen a fatal result from the administration of two drachms of aloes in a case of this kind. In such circumstances the skin usually participates in an equal degree, and though the superpurgation be checked an extension of the disease to the feet may still prove fatal or induce such changes of structure as to leave the animal practically worthless. In old animals or after repeated severe attacks of bronchitis it may merge into the *chronic* form. *Thick wind* is a frequent sequel of severe cases from thickening or dilatation of the bronchial tubes, from collapse of the lung or from emphysema.

Post-mortem appearances. In the bodies of animals that have died of bronchitis the air-passages within the lungs are filled with a white or greenish yellow mucus. If this is washed from the tubes by a stream of water, the mucous membrane is often found to be injected, studded more or less profusely with red points or with branching red lines, and with petechia, and the mucous membrane is softened, sometimes thickened and friable. When, however, the bronchitis has been attended by a free purulent expectoration the mucous membrane may, when washed, show no perceptible alteration from the healthy standard as examined by the naked eye.

In the *capillary form* the blocking up of the smaller tubes by a tenacious frothy mucus, and by the false membranes which form complete casts of many of the tubes and the partial consolidation (collapse) of circumscribed pyriform masses of lung tissue with which such tubes communicate form the chief features on examition after death.

This state of *consolidation* or *collapse* of lung is frequently seen in simple bronchitis as well. It is then due to the blocking up of one or more bronchia by plugs of tenacious mucus which act as valves, preventing the entrance of air, though it may permit of

its easy passage outward. This state of lung differs materially from the consolidation due to inflamed lung tissue (hepatisation). When cut it does not present the granular appearance of the latter, caused by the exudation into the minute air cells, but the cut surface has an uniform homogeneous aspect aptly likened by Laennec to *muscular flesh* (splenisation). Mendelson, Traube and Gairdner have induced artificial collapse of the lung by introducing foreign bodies into the bronchia of animals.

Emphysema of the margins of the lung is a frequent concomitant of *collapse*. The cause is plain. The portion of lung, the subject of collapse, emptied of its air, does not occupy a tithe of the space it would normally fill. The rest of the lung tissue expands unduly to fill out the vacated portion of chest and the cells become over-distended and ruptured. The emphysematous lung is known by its lighter color, by its irregular bulging surface, by the subsidence of these elevations when pricked with a needle, and by a more marked crepitation when pressed. When the cells have burst and the air escaped into the areolar tissue between the lobes, it appears as dark lines circumscribing small portions of pulmonary tissue and collapsing when pricked.

Treatment. The mildest cases will recover of themselves, especially if care is taken to protect the patients against cold, wet, draughts of cold air, over-exertion, and other injurious causes, and to give a part of the food warm and sloppy. In severe cases treatment must be more active, but it will be borne in mind that severe depletive measures are badly endured. Bleeding dangerously increases the already existing weakness and prostration without affording any corresponding advantage. It is only admissible when from the severity of the symptoms in the early stages suffocation is threatened or when the brain becomes involved in disease.

Causing the patient to inhale water vapor from scalded bran or hay is to be assiduously carried on for half an hour to an hour twice or thrice daily until expectoration has been freely established and the cough and fever alike moderated. The density of the vapor must of course be apportioned to the particular case so as to avoid any approach to suffocation. The addition of the fumes of burning sulphur will often by their astringent and antiphlogistic action on the mucous membrane, render the vaporous application

more effective. A pinch put into a small piece of paper twisted at one end to prevent burning of the fingers may be set fire to and the fumes allowed to pervade the appartment so that they can be breathed freely without inducing cough. A mustard poultice to the neck and sides of the chest should also be applied and kept on an hour or until effusion into the skin is well marked by thickening of its substance. Injections of warm water should be given alike to check or obviate shivering and to equalize the general temperature and to solicit the action of the bowels. In sporadic cases with active fever and full strong pulse a laxative dose of aloes (3 to 4 drachms) may be given, but if with a low fever and during the prevalence of influenza not more than half the dose should be given or enemata alone may be relied upon. As soon as the medicine has set or at once if it is withheld, neutral salts may be given (Liquor Ammoniæ Acetatis 2 oz. or Sweet Spirits of Nitre 1 oz. or nitrate of potass ½ oz. combined with 10 drops tincture of aconite repeated twice or thrice daily). If the cough is troublesome and secretion long in being established, expectorants may be used (oxymel of squill 3 oz., powdered squill ½ oz. or liquorice 1 drachm) with half the doses of the neutral salts.

In the early stages to hasten expectoration such preparations as the following may be given :

Recipe : Potas. Bicarb. 2 ounces ; Ammon. Carb. 2 ounces ; Digitalis Pulv. 2 drams. Mix. Divide into eight powders : give one every four hours.

Recipe : Ammon. Murias. 2 ounces ; Choral. Hydrat. 1 ounce ; Tinct. Hyoscam. 2 fluid ounces ; Aqua 8 fluid ounces. Mix. Give two tablespoonfuls every four hours.

Apomorphia, tartar emetic, turpentine or benzoin may be employed or even pilocarpin, care being taken not to increase prostration unduly. Compressed air, oxygen and peroxide of hydrogen will sometimes relieve.

With the advent of expectoration, or earlier, iodide of potassium in one drachm dose, thrice a day will do much to obviate glandular and other enlargement which would tend to develop *roaring*.

The diet should be laxative, non-stimulating and somewhat spare. Mashes of wheat bran, boiled linseed or boiled barley ; roots such as turnips, carrots, beets ; in summer a limited supply of fresh grass, with little hay at any time and that scalded, may

indicate the nature of the aliments to be used. As a beverage chilled fresh water or linseed tea may be supplied *ad libitum*.

Should the nasal discharge manifest no disposition to cease at the end of 15 or 20 days, as will sometimes happen in young horses, stimulants and tonics must be employed. Gentian (4 drachms), Sulphate of iron (2 drachms), Arsenious acid (5 to 10 grains) or nux vomica (1 scruple) may be given daily as ball, electuary or powder. A full and nutritive diet should at the same time be allowed, and open air exercise enjoined.

CHRONIC BRONCHITIS IN THE HORSE.
BRONCHIAL CATAARH.

As Sequel of Acute: as result of unhygienic environment, diet and usage. Symptoms, breathing accelerated, double expiratory act, short breath, cough husky, or paroxysmal, excited by cold air, water, discharge white flocculent. Percussion, drumlike patches; Auscultation mucous and sibilant râles. Lesions, thick mucus, pale membranes, bronchiectasis, emphysema, ulceration. Thick wind. Treatment, stimulating, tonic, derivative, medicated vapors, careful diet. Tar water.

This may be a sequel of the acute form, or it may appear at once as a catarrhal discharge from the bronchial tubes and without any very marked febrile affection, in animals debilitated by damp stables, overwork and a faulty regimen and diet.

Symptoms. Respiration accelerated, and expiration effected with a double lifting of the flank; the horse is easily blown when moderately exercised; the cough is frequent, soft or rattling and paroxysmal—sometimes hard and deep - is excited when brought from the warm stable into the cold air, and is followed by a whitish, flocculent, purulent discharge from the nostrils, consistent but not sticky (like buttermilk). The pulse is rapid and small in volume.

Percussion manifests a healthy resonance over the greater part or all of the chest, a drumlike sound over given areas, and auscultation over its median part detects a bronchial rattle and in most cases a dry sibilant râle.

After death the bronchial tubes are found to contain an abundance of thick mucus, though abnormal redness of the mucous membrane is by no means a necessary condition. There is always more or less dilatation of the bronchial tubes especially at their points of subdivision where they are often twice their healthy calibre, and an emphysematous state of the lungs is equally constant. Delafond and Rodet have noted minute ulcers on the bronchial mucous membrane and Reynal miliary abscesses and grayish and white indurations of the lung tissue and bronchial glands which may have been glanderous.

It will be observed that the symptoms and lesions closely resemble those of broken wind (heaves), and unless early and suc-

cessfully treated, into this it gradually merges. The chief distinguishing symptoms are the abundance and nature of the discharge, the fetor of the breath, and the presence of the mucous râle in the chest. It is one of the conditions known by the horseman's expression "*thick wind.*"

Treatment. Like its type (dilatation of the bronchia, bronchiectasis) in man this disease obstinately resists treatment. In our efforts to cure it the same general principles must be followed as in acute bronchitis, with this grand qualification that the general aim must be to stimulate and support. Stimulating liniments may be repeatedly applied along the course of the trachea and on the sides of the chest. An equable temperature is desirable and a dry building. Water vapor medicated with various astringents and antiseptics (creosote, carbolic acid, turpentine, tar, or tar vapor) is to be commended. A course of tonic and expectorant medicine is desirable and a highly nutritious and laxative diet is imperatively demanded.

As tonics Gentian may be given daily in 4 drachm doses combined with quaiacum in doses of 2 scruples. In most cases it will be advisable to add to the above or employ separately arsenious acid in doses of 5 to 10 grains combined with three times the amount of bicarbonate of soda, and given daily for a month or longer.

The diet should be as for broken wind, nutritious, in small bulk, of a laxative nature and given at least an hour before work. A moderate supply of grass, roots, bran, oats or barley may be given, but hay must be sparingly supplied and, if exclusively clover hay, dry and dusty, is better withheld. In the north of France horses with chronic bronchitis are maintained in a serviceable condition by a diet of cut straw and cut hay, well sifted to remove all dust mixed with oats and molasses and set aside in a large cask to ferment before being given to the animals. Tar water may be the exclusive drink.

A pint of linseed, well boiled, and given daily for a length of time in succession is often of great value.

BRONCHITIS IN THE OX.

Working oxen most susceptible. Causes, damp buildings, wet, cold, exposed localities. Debility, overwork, poor feeding, close, foul stables, sudden changes of temperature. Previous attacks. Symptoms, mild form, and severe, fever, sneezing, cough, dry, later soft, prostration, retraction of nostrils, labored breathing, hot breath, discharge, watery, milky, purulent. Duration, Complications and sequelæ. Chronic form. Respiratory disturbance, paroxysmal cough, purulent discharge, mucous and sibilant râles, emaciation. Lesions. Treatment, laxative safer, expectorant, derivative stimulant, etc., as in horse. Bronchitis in pigs and sheep.

This is less common than the same disease in the horse, though in working oxen, in which many of the same causes operate, it is frequently seen. It is not infrequent in other cattle in damp buildings or in wet cold exposed situations. Debility from overwork and poor feeding, often brings on the chronic form of this disease. Living out in damp nights after a hard day's work is another frequent cause. The enervating influence of the hot foul air of many cow houses conduces to it and is specially injurious if alternated with a chilling atmosphere out of doors. Previous attacks strongly predispose to future ones.

Symptoms. Some cases are so slight as to escape a cursory observation and subsiding in a few days leave the animal perfectly well. Others are severe and may prove dangerous.

The earlier symptoms are dullness, staring coat or shivering, and sneezing, followed by reaction with hot clammy mouth, general increase of temperature, rapid pulse, reddened nose and eyes, and suspended rumination. The more characteristic symptoms are a hard, dry, hacking cough, not so resonant as in the horse, and soon a mucous discharge from the nose usually cleared away by the tongue almost as rapidly as formed.

If the case increases in severity, and in many cases almost from the first there is great depression, hanging head, semi-closed watery eyes, extreme movement of the nostrils, hot expired air, labored action of the flank, complete loss of appetite, constipation, fæces covered by mucus, cough very hard, painful, occurring in paroxysms and easily excited by touching the larynx or trachea. This is followed by a loose cough, a free discharge

from the nose and a mucous râle on auscultation. Percussion gives healthy resonance. The disease reaches its height on the fifth day and recovery may be almost perfect on the eighth. Its chief danger is from a complication with pneumonia or pleurisy, or from its merging into the *chronic form*.

Chronic bronchitis in the ox is characterized by a persistent disturbance of the respiration, paroxysms of coughing, a white flocculent discharge from the nose, increasing emaciation, palor of the mucous membranes, a mucous râle over the windpipe and median part of the chest and a cooing sound over other points. If left to itself emaciation becomes extreme, the skin is harsh, inelastic, attached to the ribs and covered by vermin, and death usually ensues from diarrhœa or consumption.

After death the lesions are like those seen in the horse, unless there is the complication of tuberculous or other disease of the substance of the lungs.

Treatment. Neither the general care nor the remedial treatment differs materially from that for the horse. The principle difference is in the lesser liability to superpurgation and in the preference to be given to Epsom or glauber salts over aloes as a laxative. Either saline may be given in dose of one pound combined with an ounce of ginger or other stimulants, and followed up by similar diuretics, expectorants and tonics, as in the horse. The *chronic form* is to be treated as in the horse.

Pigs and sheep affected with **bronchitis** must be treated on the same general principles as the ox, only giving one-fifth the amount of the different medicaments, and in the case of the pig oleaginous purgatives and emetics as advised for the dog.

BRONCHITIS IN THE DOG.

Causes, damp kennels, cold and damp after hunting, pampering and exposure, distemper. Symptoms, fever, cough hard, later soft, discharge watery, glairy, purulent. In capillary bronchitis cough more paroxysmal, painful and attended with vomiting. Disturbance of breathing, pulse, temperature. Fatality in different breeds. Treatment, laxative, expectorants, diuretics, heart tonic, calmative, water vapor, chest jacket, stimulant expectorants, stimulants, tonics. Diet.

This is common and severe. Hounds kept in damp kennels, much exposed to cold and damp after being heated in hunting, or subjected to frequent and sudden alternations of temperature are specially liable. Pampered pets kept in warm rooms, overfed and having little open air exercise, are equally subject to its attacks. It is an usual form in which distemper is manifested.

Symptoms. There is roughness of the coat or shivering and a small, hard cough often repeated. If confined to the bronchi the cough soon becomes loose, a free discharge sets in, and with care recovery may be secured in five or six days.

If the smaller bronchial tubes are involved the symptoms are more intense and persistent. The temperature may reach 104° or 105°. To the same early symptoms succeed, a painful cough occurring in paroxysms and sometimes followed by vomiting of a glairy mucus. There is running from the eyes and nose, and reddening of thin membranes. The creature stands with his elbows turned out, his flanks heaving and his heart beating rapidly and tumultuously. In the worst cases when the inflammation has been propagated to the smallest bronchial tubes constituting *capillary bronchitis*, these symptoms are seen in their most aggravated type and the subject often dies of suffocation, or by implication of the lung tissue. Percussion and auscultation are even more applicable than in the larger animals, showing the clear resonance, of the lung tissue, the tubal murmur in the early stages and the mucous rattle in the later ones. In the *capillary form* a distinct crepitation is heard like that of pneumonia. Bronchitis proves most fatal to the higher bred dogs, such as King Charles spaniels, Italian greyhounds, and English terriers, and

according to St. Cyr small dogs suffer more severely than large ones.

Treatment. If the bowels are costive a tablespoonful of castor oil should be given, followed up by nauseating expectorants and diuretics (tartar emetic two grains, nitrate of potass one drachm, and sugar one drachm, mix thoroughly, divide into twelve powders and shake one on the tongue thrice daily). If the cough is very troublesome two grains of powdered digitalis may be added to each dose, or after the nasal discharge has been freely established two to four grains of opium among the tartar. The opiates are of great value in controlling the paroxysms of coughing and the propagation of the disease to the smallest ramifications of the air passages, but as they check secretion they must be used with caution until a free discharge has been established. In the early stages bromides, hyoscyannes or digitalis may be preferred. Muriate or carbonate of ammonia, syrup of Tolu, senega, or guaiac may follow.

Inhalations of water vapor, the maintenance of an equable temperature, and the moist chest jacket, followed by mustard poultices to the throat and chest are not to be neglected. The diet should be simple, oatmeal or Indian corn pudding with milk, soups and the like may be allowed, but as a rule butcher meat is to be withheld. If the patient has previously fed entirely on the latter it should now be given in very limited amount only, and qualified by an admixture of farinaceous diet.

In some cases the prostration becomes so great that the patient must be supported by tonics and stimulants (a teaspoonful each of sweet spirits of nitre and tincture of gentian, or a teaspoonful of port wine repeated twice daily).

In case of persistent discharge, iron, liquor arsenicalis, the same strength as Fowler's solution, or cod-liver oil may be used.

CROUPOUS BRONCHITIS IN CATTLE AND SHEEP.

Causes, smoke, hot air or gas, irritant inhalations, concomitant of infectious diseases. Lesions, intense congestion covered by fibrinous exudates. Symptoms, slowly or suddenly developed, fever, loud, wheezing, stertorous, panting breathing, dyspnœa, dry râles and blowing. Course. Treatment, moist jacket, soothing, expectorant, stimulant inhalations, expectorants, derivatives.

This affection has been found in cattle and sheep from exposure to smoke, hot air or gas, and other irritants, and even from exposure to cold, and without any suspicion of a contagious element. Again it has been seen as a complication in Rinderpest, lung plague and malignant catarrh. The lesions are those of tumefaction and extreme arborescent redness of the mucosa, and the formation of patches of a dense fibrinous exudate of a yellowish color, in some cases completely obstructing some of the smaller tubes.

Symptoms. The attack may come on slowly as in ordinary bronchitis, while in other cases it is sudden. The respiration being loud, wheezing, stertorous and panting and general dyspœna supervening. Auscultation furnishes loud, blowing sounds, dry râles and rouchi, while percussion may show no abnormal change. A strong tremor is felt by the hand on the trachea, and after a paroxysm of coughing false membranes may be expelled. If there is no improvement by the second or third day death is liable to supervene by asphyxia.

Treatment is usually unsatisfactory. The hot, moist jacket, inhalations of vapors of warm water, of carbonate of ammonia, and of ether may be tried, counter-irritants to the chest, and internally liquor ammonia acetatis and iodide of sodium would be indicated. When the membranes are somewhat loosened pilocarpin, or in weaker subjects apomorphine may assist their expulsion.

ACUTE CONGESTION OF THE LUNGS. PULMONARY HYPERÆMIA.

Active and passive congestions. Congestion of incipient pneumonia. Congestion of over-exertion. Causes, lack of condition, fat, plethora, gorged stomach, hot weather, cold rains, cold baths, infectious diseases. Symptoms, dilated nostrils, labored breathing, deep lifting of flanks, panting, pendent head, staring, fixed, bloodshot eyes, pale—later dark red nasal mucosa, rapid pulse, palpitating heart, fine crepitation, cold limbs, tremors, perspiration, obstinate standing, till unable, blood from nose. Fulminant cases. Exposure cases. Course. Termination. Resolution. Lesions, lungs black, gorged, do not crepitate nor collapse, lessened buoyancy, cut surface, compressed bronchioles and alveoli, right heart and veins gorged, blood black, semi-liquid, petechiæ. Nature. Not yet inflammatory, blood engorgement, no cell proliferation, migration, non-exudation, pulmonary vaso-motor paresis, effect of blood pressure, of peptones, etc., of exhaustion. Treatment, relieve respiratory muscles, derivation to surface, stimulants, hot pediluvia and packs, relief of vascular system, bleeding, heart stimulants, digitalis.

Congestion of the lungs occurs in all animals as the percursor of inflammation, but as death may occur without the supervention of actual inflammation a special notice appears to be demanded. The hyperæmia of the lungs may be seen in two forms, active and passive, the latter form being secondary to other diseases, such as valvular diseases of the left heart, by reason of which the blood is forced back on the lungs and creates mechanical congestion. The active form is a pathological process developed in the lung itself, and which often proves fatal through arrest of the circulation through this organ.

Causes. The pulmonary congestions preceding pneumonia are due to the same causes with that disease. The most typical, acute and deadly form of pulmonary congestion is usually due to *over-exertion in an animal that is fat and out of condition.* The English hunting field presents the most typical specimens. A horse that has just left the dealer's hands, or that is plethoric, fat, soft and flabby, is ridden over a heavy country, and though he may perform well for a few miles, he soon hangs heavily on the bit, slackens his pace, and if not pulled up, staggers and falls "all of a heap." A farm horse, taken from grass or other soft

feeding, and entirely out of condition often suffers in the same way, in going perhaps for the veterinarian in case of urgent colic in one of its fellows. Cruzel draws attention to similar congestions from over-exertion in fat cattle, and Trasbot in wild stags and hogs when beechnuts and acorns were abundant, in pampered family horses and in plethoric farm animals generally. Excessive heat (heat apoplexy) is invoked as a cause, and the arrest of hæmatosis and consequent stagnation in the pulmonary capillaries are undoubtedly accessory causes, yet the majority of cases, and the most typical and fatal, occur in the winter season (the hunting season). On the other hand, chills from rains or cold draughts, especially when heated and exhausted, are common causes, and the disease often comes on more gradually, attaining its acme after five or six hours. A horse perspiring after a hard drive and left to face a cold blast unblanketed, or one plunged by accident into ice cold water for ten minutes (Trasbot) are examples of this kind. These cases are ushered in by violent rigors, whereas in those due to over-exertion this is much less marked and is usually only suggested by the coldness of surface and extremities. Another condition which contributes to pulmonary congestion is a full stomach. The plentitude of the abdominal organs leads to compression of the lungs and hampered circulation, and when to this is added over-exertion and exhaustion acute congestion is speedily induced.

Acute congestions are noticed as an accompaniment of other diseases, but these are mostly either the localization in the lung of a specific morbid process (anthrax, influenza, distemper, strangles), or it is due to auto-poisoning, as when the cutaneous transpiration is suppressed by a coating of glue, or to embolism.

Symptoms. In the horse which fails under severe exertion there are the dilated nostrils, the labored breathing, the deep, almost convulsive action of the flanks, the hanging on the reins, the slacking of the pace, the unsteadiness of gait, and lastly the fall. There may now be noticed the protruded bloodshot eyes, the agonized expression of countenance, the extended head, the pallor, and later the blueness of the nasal mucous membrane, the short, panting breathing, accompanied by a roaring noise alike in inspiration and expiration, and the small, weak, rapid pulse often imperceptible at the jaw. If the animal has been stopped

short of having fallen, or if he is able to get upon his feet, he stands with his limbs apart to secure his stability, and with the elbows turned out to facilitate the expansion of the chest. As the breathing becomes panting the respirations are less deep, the ribs are maintained permanently drawn outward, and the flanks rise and fall to a limited extent only but with great rapidity (eighty to one hundred per minute). Auscultation may detect at first an increase in the pitch of the respiratory murmur, and the presence of the finest possible *crepitation sound*. Soon the murmur decreases uniformly. The extremities are cold, and in this coldness the general surface to some extent participates even though it may be covered by perspiration. Tremors or rigors are present. The heart is felt behind the left elbow to beat tumultuously. If blood is drawn it flows in a thin, black, tary stream.

In some cases blood more or less frothy is discharged from the nostrils as the result of rupture of pulmonary vessels.

In the fulminant cases in **cattle** respiration is rapid, even panting, wheezing, the expiration attended by a hoarse grunt, sometimes nasal hemorrhage, great prostration, profuse perspiration, a stupor sets in and the animal falls and dies, with more or less struggling.

In the cases which develop more slowly, and as the result of cold and chill whether in horses or cattle, there is dullness, anorexia, prostration, increasing rapidity and oppression of the breathing, a small, frequent, hoarse cough, and at first distinct pallor of the conjunctiva and pituitary mucosa, with more or less trembling. The head is extended on the neck, toward an open door or window, if available, until prostration and stupor forbid. The pulse is small, thready, often almost imperceptible and much accelerated, while the heart beats are strong, violent, tumultuous. For a time the respiration may be not more than double the normal rythm, but it tends to more or less rapid increase with wheezing or stertorous sounds and shaken by trembling of the respiratory muscles. The nasal discharge is slight and grayish often with streaks of blood. If it increases the cough becomes looser and softer. Quite early the respiratory murmur decreases over the whole lung and a blowing sound from the bronchia or larnyx is heard on the upper middle third of the chest. This may be complicated by

a mucous râle, or when hæmorrhage has supervened by a loud rattling. Percussion shows a lack of resonance over the whole lung, not so flat and definitely circumscribed as in pneumonia but a partial flatness of sound over the whole chest. In pneumonia a limited area of lung is absolutely solidified (hepatized) while the remaining lung is practically normal, whereas in acute congestion often the whole lung is gorged with blood but for some time no part of it is entirely divested of air.

Another marked feature is the maintenance at first of the normal temperature with only a slight rise of about 1°. This serves to distinguish congestion of the lungs from sunstroke (heat anhæmatosis) in which the temperature usually rises to 108° or 110° F. or higher. The temperature rises however as the disease advances and merges into pneumonia. Another distinguishing feature from sunstroke is the early pallor of the mucous membranes which in heat apoplexy are strongly congested. In congestion they become dark red only with the advance of the disease and the advent of asphyxia. These features serve also to distinguish acute pulmonary congestion from contagious fevers, pneumonia and other inflammations of internal organs.

Course. Termination. The more acute (fulminant) forms are promptly fatal. In the exhausted system the lungs have become uniformly gorged with blood, which can no longer be forced through the capillaries by the right heart, the heart in turn is overdistended with blood and ceases to beat and death ensues in a few minutes.

In the less acute cases the patient survives twenty-four hours and upward, the whole lung not being equally implicated but only certain lobules, usually the lower, or the congestion, if uniform in all the lung, being less extreme.

In favorable cases recovery takes place in one or two days. There is a return of life and appetite, a gradual improvement in pulse and breathing, the respirations becoming deeper and longer, and in a few hours all the more violent symptoms may have disappeared. With a more gradual improvement recovery may still be complete in four or five days.

Lesions. When the subject has died suddenly the appearances are essentially those of uniform engorgement of the pulmonary capillaries with blood. The general aspect is a dark red, varying

from reddish brown to black, the darkest shades corresponding to circumscribed areas of actual hæmorrhage. In the worst cases the whole mass may appear like black currant jelly. The lungs do not collapse when the chest is opened, they are more or less friable at various points, and different portions will sink or float in (not on) water, according as it may be more or less airless. A dark liquid blood exudes freely from the torn or cut surface. Sections of the lung tissue hardened and examined under the microscope show the alveoli and bronchioles devoid of exudate, but having their cavities compressed and obliterated by the pressure of the swollen mucosa, and its investing blood clot. The heart is overdistended with fluid blood. In asphyxiated cases the general venous system is filled with black, liquid blood, and the serosæ spotted with petechiæ.

Nature. The nature of this disease is variously understood. It differs from inflammation in the absence of active cell proliferation, and migrations of inflammatory exudation, and of fever at all proportionate to the extent of the lesions. All these may and do supervene if the patient survives but they are practically absent for a length of time at the outset. Some attribute it to paresis of the vaso-motor centres for the lungs, as the result of their over stimulation and of the retrocession of blood from the chilled surface to the internal organs. But congestions caused by cutting the cervical branch of the sympathetic nerve or the sciatic plexas are not marked by a similar blood extravasation and destruction of tissue. The delicate structure of the lung tissue and the comparative absence of mechanical support will account for this in part, the great force of the circulation overloading the capillaries, under the impulse of the heart so closely adjacent, has doubtless a certain effect, and the venous nature of the blood thus forced into the lungs and calculated to arrest all normal function has a potent influence. If we add to this, for the *over-exertion* cases, the sudden advent into the circulatory stream of unchanged peptones and other ingredients of the portal blood of highly fed and plethoric animals we find a sufficiently pathogenic combination. In all acute cases however the adiposity, poor condition and susceptibility to speedy exhaustion must be given their full share of responsibility.

Treatment. Girths, saddles and anything else that may hamper

the movement of the chest must be at once removed and the horse's head turned to the wind, an active stimulant given and the legs well rubbed and loosely flannel bandaged. The nature of the stimulant is of less consequence than its prompt administration. Two ounces of chloroform, of sulphuric ether, or of sweet spirits of nitre; half a pint of whisky, brandy or gin; or a pint of any of the more stimulating wines may be given, diluted in warm water so as to remove their irritating qualities. A drink of warm gruel will often go far to restore warmth to the surface and to unload the overtaxed lungs. Frequent large injections of warm water have a similar effect. Active hand rubbing of the legs and the wrapping of them loosely in flannel bandages previously warmed at the fire is equally valuable. If a roomy, well ventilated, loose box can be obtained the horse should be led to it gently and a light but warm rug placed upon the body. Valuable derivation may be obtained from pediluvia, the feet and legs up to the knees and hocks being put in buckets of water as hot as the hands can bear, and at the same time actively rubbed. If this is impossible the legs may be wrapped in bandages and wet with hot water every few minutes. Or this soothing derivative agent may be applied as well to the surface of the chest. A blanket wrung out of hot (nearly boiling) water until it no longer drops is wrapped round the body and covered up with two or three dry rugs. A second smaller rug is wrung and placed on the neck and covered by a sufficiency of hoods to keep in the heat. The legs are meanwhile hand rubbed and bandaged and the other measures above recommended carried out to restore the circulation in the surface and extremities. The time-honored practice of bleeding freely from the jugular vein is one of the most effective means of relieving the over-charged heart and lungs, and should be resorted to at the earliest possible moment. The blood will at first flow in a small, dark stream, but as the circulation obtains relief the jet will increase in volume and the general symptoms will improve. From four to six quarts may be taken with advantage from an ordinary horse. This is not a pneumonia but an overloaded heart and lungs, threatening speedy death and which the abstraction of blood promptly relieves.

The longer the bleeding is delayed the less effective it is. It should not supersede the other measures already recommended.

There is no real paradox in both bleeding and giving stimulants in such a case, as the essential condition is one of weakness, and if the abstraction of blood has been of use in relieving the clogged heart and lungs, the depression under which these have labored may be still further overcome by agents calculated to rouse their suspended vitality.

Trasbot strongly recommends large doses of tartar emetic and iodide of potassium to reduce the blood pressue in the lungs, an advice which will be received with hesitation by those who dread the already paretic condition of the heart. His combination of iodide of potassium with digitalis will be more confidently resorted to. One drachm of the former may be given with a half drachm of the latter twice daily.

With the advent of marked fever and other signs of pneumonia, the treatment for that disease should be resorted to.

PULMONARY ŒDEMA.

Causes, pneumonia, extra force of right heart or weakness of left, insufficiency of mitral valves, deflection of blood by obstruction in one portion of lung, pressure of tumor on pulmonary veins. Malignant œdema. Malignant catarrh. Bright's disease. Anæmia. Parasitism. Symptoms, abnormal heart sound, or urinary secretion. Parasitism. Percussion shows flat sound auscultation lowered, respiratory murmur, heightened blowing. No crepitation. Expectoration serous. Prognosis grave. Treatment, attacks primary disease. Posture. Elimination. Dry capping. Heart tonic.

A dropsy of the lung tissue may supervene in weak conditions, in the course of inflammatory disease of the lungs; it may also depend on an imperfect balance in the forces of the right and left heart respectively, which leads to the habitual throwing of blood pressure back upon the lungs. Still more frequently the congestion and dropsy depends on insufficiency of the mitral valves by reason of which a reflux of blood toward the lungs takes place at each heart-beat. The pressure of tumors on the pulmonary veins may have a similar action. Obstruction of circulation in one portion of lung may cause an extra blood pressure on an ad-

jacent one, and œdema so caused may be found especially in cattle and pigs in which the interlobular connective tissue is specially abundant. This may be seen in miliary tuberculosis in cattle, and it probably contributes to produce the extraordinary liquid collections that characterize lung plague. In cattle also malignant œdema may affect the lung, and an œdematous condition is sometimes met with in malignant catarrh. Bright's disease is another cause, the uræmic dropsy finding a favorite seat of election in the loose lung tissue unsupported by solid tissues. The anæmia resulting from parasitism like distomatosis may similarly affect the lung.

The *symptoms* of pulmonary œdema will usually be complicated by those of the affection causing it. Thus modification of the first heart sound or of the urinary secretion, or the existence of parasitism, would furnish valuable indications.

The *physical signs* of lung disease vary. If pneumonia is present it is betrayed by its characteristic symptoms. In the absence of inflammation there is dullness on percussion over the affected area, and on auscultation an absence of the respiratory murmur, and perhaps abnormal clearness of bronchial, cardiac and other sounds from deeper parts. It differs from pneumonia in the absence of fever and of any crepitation surrounding the consolidated portion. The expectoration is serous or watery, rather than rusty or purulent.

The *prognosis* is always grave in proportion to the incurable nature of the primary disease. Chronic valvular or Bright's disease, miliary tuberculosis or malignant tumors would render the case hopeless, while in acute pneumonia, or nephritis or parasitism there may be some hope. The *treatment* will largely consist in the therapeutics of the primary disease, yet we may also seek to relieve the dangerous symptoms of œdema. The frequent change of position may serve to limit hypostatic accumulation. Diuretics or purgatives in strong patients will favor absorption. Pilocarpin more than any other agent secures temporary absorption but cannot be continued owing to its depressing effects. Digitalis is often valuable in improving the heart's action, and acting freely on the kidneys. Dry capping on the chest acts as a derivative.

ATELECTASIS. COLLAPSE OF LUNG.

Atelectasis in bronchitis, congenital, etc. Airless condition in the absence of exudation. Causes, congenital persistence in butcher animals. Blocking of air tubes by exudate—ball valve. Desquamation of ciliated epithelium. Compression by hydrothorax, pneumothorax, and false membrane. Symptoms. Percussion flatness, juvenile respiration elsewhere, blowing sounds loud. Drum like sounds on emphysema and pneumothorax. Cyanosis. Lesions, depressed, flesh-like, non-crepitating lobules or lobuletes, sink in water, dilatable. Treatment, rouse respiratory centres, douches, cold and warm, slapping, electricity, forced inspiration, diet, massage. Treat attendant disease.

This has been already referred to as a result of bronchitis, but it deserves special mention as a sequel of that affection, and in various domestic animals, as an independent condition. The condition is one of consolidation of lung by the complete exclusion of air, but without any infiltration of its substance by inflammatory exudate or dropsical effusion. The tissue remains in its normal state apart from the fact that its bronchioles and air sacs are undilated. The affected portion has a solid dark fleshy appearance. The collapsed portion often represents one lobule or group of lobules which communicate with a single bronchium.

Causes. In some instances the conditions remain from birth, the lobule never having been called into use. This is seen especially in cattle and other meat producing animals, in which active breathing is systematically suppressed in the interests of rapid growth and the deposition of fat. In the improved breeds the lungs remain larger than the exigencies of the life demand, and large portions remain out of use. In bronchitis the condition is acquired, and is mainly dependent on the blocking of a bronchial tube with tenacious mucus or a dessicated mass. The pathological lesions of bronchitis favor this since one of the earliest changes in the inflamed mucosa is the desquamation of the columnar epithelium. This removal of much of the cilia and the paralysis of much of what is left annihilates for a time the normal method of clearing away the secretion, and this being now produced in excess blocks the tubes. This secretion virtually acts like a ball valve in favoring the exit of the air during the convulsive expira-

tion of coughing, and hindering its entrance during the succeeding inspiration. The bronchia and bronchioles decrease in size to near their termination, so that, as forced out in coughing, the secretion enters the larger tube and allows the exit of air, which as drawn back in inspiration it enters the smaller tube and closes it against any possible ærial entrance. Mendelssohn and Traube demonstrated this action by introducing a shot into a dog's lung, and in two days the left lung was found collapsed and the right one the seat of complementary emphysema. The violence and frequency of the cough therefore bears a ratio to the occurrence and extent of atelectasis. Other causes are the compression of the lower lobes of the lung by hydrothorax, by pneumothorax (developed by lacerated lung or perforated chest wall) or by a false membrane contracting in process of organization.

Symptoms. As a congenital condition in the improved meat producing animals the condition is rarely recognized in life and cannot be said to be a defect. The collapsed lobule being farther removed from the air may be a more favorable field for the growth of pathogenic bacteria, but on the other hand these do not so readily penetrate it as if the tubes were open. When the collapse is more extensive, the contrast in the flatness on percussion and indistinctness of the respiratory murmur on the affected side, and the marked resonance and loud murmur on the other, may serve to identify the affection. In extensive, traumatic cases this contrast is much more prominently marked, as the expanded portions have to take on extra compensatory work and are not infrequently rendered emphysematous. The drum-like sound in percussion of such parts, and in the upper part of the chest in pneumothorax are pathognomonic of these conditions. Again in hydrothorax the horizontal upper level of the area of dulness betrays a liquid cause. Severe cases are marked by cyamosis.

The *lesions* seen in atelectasis consist in depressed areas of a dark fleshy color on the surface of the lung, usually sharply limited by the borders of the lobules, and in strong contrast with the bulging, light colored lobules adjacent, which are often emphysematous. The collapsed lobule may usually be dilated when air is forced into the bronchium, but if it has been of some standing this is often difficult or impossible. If it has resulted from bronchitis or compression of a previously inflated lung it will often

float in (not on) water, from a little retained air, but in congenital atelectasis it is airless and sinks to the bottom.

When *treatment* is demanded it will vary according to the cause. In congenital atelectasis the respiratory centres must be roused. The new born animal may be sprinkled alternately with ice cold and hot water, or the chest may be slapped with the palm of the hand or a wet towel. The nostrils must be cleared of mucus, and the lungs inflated by blowing or bellows, the larynx being pressed back against the gullet to prevent inflation of the stomach. If available electricity may be applied to the chest walls. These measures may be repeated at intervals and the systemic weakness overcome by nourishing food, stimulants and friction of the skin.

In acquired atelectasis we should seek to correct the disease to which it owes its existence. In bronchitis the measures already indicated for the liquefaction and removal of the expectoration will be in order ; in hydrothorax a judicious paracentesis and in pneumothorax the aspiration of the gas, and the closure of any traumatic opening through which that gas has gained access.

HÆMOPTYSIS.

Causes, over-exertion in plethoric, glanders, pulmonary tubercle, petechial fever, embolism, aneurism, ulcerated new formations, anthrax, septicæmia, hæmorrhagic diathesis. Symptoms. Inappetence, cough, cold limbs, rigor, hard pulse, jugular pulse, violent heart beats, unsteady gait. Discharge, bloody, crimson, frothy, with cough, without acid, excited breathing, debility. Indications from pre-existing disease. Treatment, quiet, elevated head, cold irrigation, ice bags, acetate of lead, opium, ergot, matico, tannin, iron, oil of turpentine, laxatives, cool stable.

The term *hæmoptysis* (αἷμα, blood, πτυω, I spit,) is now entirely restricted to bleeding from the lungs and lower air-passages. It is a very rare complaint in the lower animals, but is sometimes seen in both horse and ox. In very plethoric subjects the overloaded circulatory organs give way in the delicate membrane, lining the ultimate bronchial tubes and the air cells. The exciting cause in such cases is usually some severe effort of draught, a violent gallop, or other unwonted exertion. It occurs in glanders from rupture of caseated pulmonary nodules. It does not appear to be so common in phthisis in the lower animals as in man, but one case occurred under the eye of the writer in which the bursting of a large tubercle in the lung of a cow involved the rupture of a considerable vessel with a fatal result. Pulmonary embolism and infarction, petechial fever, aneurism, ulcerated neoplasms, anthrax, and septicæmia are additional causes. Lastly hæmoptysis sometimes takes place in hæmorrhagic subjects without any appreciable rupture of vessels, the blood sweating from the surface of the bronchial mucous membrane.

Premonitory symptoms are sometimes noticed, such as dulness, and lassitude, loss of appetite, a frequent short cough, coldness of the limbs and surface, shivering, full, hard pulse, pulsation in the jugulars, tumultuous action of the heart, and unsteadiness of gait.

More commonly it comes on suddenly as the result of severe muscular strain or excitement. The blood flows from the nose, and rarely from the mouth in solipedes, but indiscriminately from both in other animals. It is bright red, clear, frothy, or mixed with mucus, and variable in amount. It is easily distinguished

from nasal hæmorrhage, which is not frothy, and from bleeding from the stomach, which is clotted and blackened, with an acid odor from the presence of the gastric juice. The cough of hæmoptysis contrasts with the sneezing of epistaxis and the retching of hæmatemsis. The rattling cough increases the discharge, as does also a dependent position of the head. Besides the cough there is usually an anxious countenance, accelerated breathing and considerable lifting of the flank. When the loss is excessive there is weakness, giddiness, rolling of the eyes, and pallor of the visible mucous membranes.

The previous ill-health of the patient, the presence of tubercle as ascertained by auscultation and percussion, and the hæmorrhagic constitution as shown by occurrence of bleeding from other parts of the system will lessen the chances of a favorable termination. Sometimes, too, the flow is so profuse that the blood cannot be coughed up, and filling the bronchial tubes it destroys life suddenly by suffocation.

Treatment. When brought on by severe exertion absolute quiescence will usually check hæmoptysis. Keeping the head in an elevated position favors its arrest. The application of cold water to the head, neck and thorax, and the giving of iced water, strongly acidulated by vinegar or one of the mineral acids may sometimes be required. In threatening or obstinate cases one drachm of acetate of lead may be given thrice daily to check by its astringent effect on the vessels, and the addition of a drachm of opium is of great value in suppressing the cough. Ergot, tannin, matico, and oil of turpentine have each been employed with advantage, and when costiveness exists a saline laxative (one pound sulphate of soda) may be usefully resorted to. The patient should be kept in a cool, airy dwelling, and should rest for fifteen or twenty days after an attack.

PULMONARY APOPLEXY. HÆMORRHAGIC INFARCTION.

Different forms. Embolism with infarction. Embolism from arteritis. Rupture of blood-vessel. Changes in color. Symptoms. Repair.

Hæmorrhage into the lungs may be: 1st. Petechial in infectious diseases. 2d. *interlobular* as from ruptured vessels. 3d. *Infarction* or apoplexy. *Infarction* results from embolism of a branch of the pulmonary artery, which may in its turn be due to clots formed in a diseased heart or in the systemic veins and carried to the lungs in the blood stream. It may also result from inflammation of the inner coat of the pulmonary artery. A virtual stasis occurs beyond the embolism, and the blood filtering in through the anastomosing capillaries fills and blackens the affected lobule. With rupture of a considerable vessel the blood escapes *en masse* and appears like black currant jelly. As it ages it becomes granular and changes to a yellow color, or it may form a necrotic mass enclosed in a cyst as in lung plague. The symptoms, apart from the absence of respiratory murmur and resonance, are not diagnostic. It may take months to undergo liquefaction and absorption. Iodide of potassium, bitters and stimulating diuretics may be given.

PNEUMONITIS; PNEUMONIA; INFLAMMATION OF THE LUNGS.

Definition. Inflammation of the spongy tissue of the lungs uncomplicated by that of the bronchia or pleura.

Divisions. This affection has been variously divided according to *seat*, *nature*, and *complications*: thus:

Single Pneumonia: Affecting one lung: *right* or *left.*

Double Pneumonia: Affecting both lungs.

Lobar Pneumonia: Affecting one lobe or by lobes.

Lobular Pneumonia: Affecting by lobules.

Acute Pneumonia: Subacute Pneumonia. Chronic Pneumonia.

Croupous or Fibrinous: With fibrinous exudate.

Catarrhal: With exudate rich in cells and granules.

Hæmorrhagic: With extravasation of blood.

Purulent: Tending to pus: abscess.

Necrotic: Tending to gangrene: sequestra.

Desquamative: With great proliferation of alveolar epithelium.

Interstitial. Interlobular: Affecting mainly the interlobular connective tissue.

Hypostatic: Dependent on gravitation of the blood.

Metastatic: Due to embolism.

Parasitic: Caused by parasites. Due to wounds or foreign bodies.

Contagious and *Traumatic Pneumonia.*

Many of these are, however, but localizations of the same affection and others are manifestly microbian diseases which in the present state of pathology it is not always easy to early distinguish sufficiently for clinical and therapeutic purposes. For the sake of convenience therefore pneumonia will here be treated of generally, and under the headings devoted to etiology, pathology, therapeutics, etc., attention will be given to distinctions. Those pneumonias that are but pulmonary manifestations of other diseases—influenza, glanders, tuberculosis, strangles, contagious pneumoenteritis, lung plague, septicæmia, pyæmia, swine plague, hog cholera, petechial fever, actinomycoses, and neoplasms will be considered under these respective headings.

ACUTE CROUPOUS PNEUMONIA. PNEUMONITIS IN THE HORSE.

Definition. Differentiation from acute vascular congestion. Predisposing causes, age, sex, stabling, training, diet, impure air, low health, previous lung disease, plethora, climate, season, exciting causes, chill, fatigue, leucomaines, sudor, draughts, plunging in or spraying with cold water, clipping, inhalation of irritant smoke, gas, dust, drawing of food, irritating or insoluble drugs into the lungs, neoplasms, parasites, contusions, fractured ribs, punctures, contagion, plurality of germs, bacillus of Friedländer, micrococcus of Talamon and Fräukel, diplococcus pneumoniæ equina of Schütz, diplococcus pneumoniæ equina of Cadeac. Symptoms, chill, hyperthermia, dullness on percussion and crepitation in the lower part of the lung, reaction, congested mucosæ, accelerated labored breathing, excited circulation, pulse oppressed, cough deep, patient statant, elbows everted, nose protruded, nostrils dilated, approaching door or window, pinched countenance, skin dry, harsh, adherent, partial sweats, loins insensible, nasal discharge rusty, dependent part of lung largely non-resonant, with peripheral crepitation. Blowing in abnormal situation over hepatized lung. Decubitus, its significance. Course. Results. Favorable indications in pulse, breathing, face, temperature, appetite, decubitus, clearing of lung. Unfavorable indications in breathing, pulse, fever, face, uneasy movements, pawing, cold limbs, prostration, nervousness, weakness. Sabacuto Pneumonia. Terminations of pneumonia, death, resolution, splenization, abscess, gangrene, red hepatisation, gray hepatisation, fibrinous consolidation. Lesions. Congestion, exudation and cell growth, hepatization—red and gray, deliquescence, abscess. Blood, loss of red globules, increase of white, excess of fibrine, glandular swelling, pleurisy, degenerations in other organs, laminitis, rheumatism. Treatment, adapted to strength of subject and type of disease, hygienic, anti-rigor, antiphlogistic, expectant, stimulant, antipyretic, febrifuge, sedative, moist compresses, derivatives, laxatives. In subacute form tonics, heart stimulants, febrifuge. In chronic cases add rich digestible diet, and easy open air life.

This consists in inflammation of the spongy tissue of the lung involving mainly and primarily the walls of the alveoli and interlobular connective tissue with their respective trophic centres (nuclei). The acute congestion of excessive heart action and debilitated pulmonary capillaries described above, is primarily a disease of the bloodvessels which become over distended and may or may not lead to the inflammatory processes in their walls and the tissues adjacent. Pneumonia on the other hand is essentially

inflammation of these tissues and nuclei, with exudation usually of a fibrinous material into their substance.

PREDISPOSING CAUSES. 1. **Age.** A very early age is nearly exempt, and from 6 years upward there are fewer cases relative to the equine population, variations that may be better accounted for by stabling, training and acquired immunity than by the mere fact of age. Of 237 cases, 2 were 3½ years; 32 were 4 years; 19 were 5 years; 131 were 6 to 11 years; 46 were over 11 years; and 7 of uncertain age (Trasbot). 2. **Sex.** No visible effect. 3. **Stabling, training, change of food.** While the young colt at pasture is practically immune, the period of stabling, transition to a dry and grain diet, and to the nervous excitement attendant on training and unwonted work as shown in the statistics of Percivall and Trasbot determine an enormous increase of cases. In a cavalry regiment Percivall found that 56.6 per cent. of all lung diseases occurred before the 5th year, and Trasbot found that at the Alfert Veterinary College 13.5 per cent. of all equine pneumonias occurred in the 4th year. 4 **Hot Stables. Impure Air.** These two conditions usually coexist and prove potent causes especially in young horses brought from the fields. We cannot, however, separate this cause as usually observed from the action of pathogenic germs which are preserved and concentrated in such places. 5. **Poor Health.** Debilitating diseases, insufficient and poor diet, overwork, exposure to cold draughts or darkness and any other cause which lowers the vitality predisposes. 6. **A Previous Attack.** This usually leaves some structural or functional change which renders the lung more susceptible to a subsequent invasion. Against this must be placed the immunity which follows the contagious forms, but as this is usually exhausted in the course of six months it does not invalidate the position that the permanent impairment of pulmonary integrity is a predisposing cause. 7. **Plethora** Tending as this does to congestion it must be accepted also for the next pathological step—pneumonia. 8. **Climate and Season.** This is notoriously an important factor. At Paris, Trasbot met with 237 cases in the nine months from October to June inclusive and but 8 cases in the summer months—July, August and September. In Great Britian, where the vicissitudes are less severe Percivall had in the cavalry horses in the seven months from October to April

inclusive 146 cases = 20.85 per month, and in the 5 months from May to September inclusive 62 cases = 12.4 per month.

Exciting Causes. Nearly all the above causes when acting with unusual force may become direct factors in causation. The effect of a sudden and extreme **chill** is especially to be feared. Even in cases that are unquestionably due to a microbe as the essential cause, the nervous disorder manifested in the chill, and the clogging of the pulmonary circulation in connection with the retrocession of blood from the surface of the body furnishes the opportunity for the colonization of the germ. The average horse at pasture will stand with impunity cold storms of rain, snow, and sleet, and transitions from a warm noonday sun to a cold night wind and dew and even frost, but under other conditions of the system, with the fatigue and fret and sudden changes of food and regimen attendant on domestication, or with any derangement of an important bodily function the *chill* is often the manifest occasion of disturbance of the balance of health, and the supervention of pneumonia. **Fatigue**, a system charged with **leucomanies**, and a **free perspiration**, which is suddenly checked by exposure, at rest, to a **cold rain**, or **snow**, to a **draught between door and window**, to **immersion in the cold waters** of a river, or to **sponging with cold water** is quite liable to cause pneumonia. An unduly heavy winter coat, an individual peculiarity or determined by a cold environment in autumn often predisposes strongly to such dangerous chills, by the frequency and profuseness of the perspirations and general relaxation of the system. Clipping of such subjects is a true hygienic measure though it entails the need of extra care in blanketing. Again in the animal that has already suffered from disease of the respiratory organs these chills are more dangerous factors.

Direct irritation by inhalation of smoke and other products of combustion ; or acrid or irritant gases or dust ; by the drawing of food by aspiration into the lungs (as in paralysis of the larnyx or pharnyx, choking, apoplexy, vomiting, etc.) ; by pouring irritant or insoluble drugs (oil, lard) through the nose ; by the pressure of neoplasms (actinomycosis, tubercle, glanders, cancer) ; or by the presence of parasites (strongyles, distomata, echinococci, linquatulas.

Pneumonia from **Contusion** of the chest, fracture of a rib, or puncture or laceration of the lung is recognized.

Contagion. The presence of a *contagium* in pneumonia is today well established. Clinical observation had indicated this even before the discovery of a specific germ, but recent bacteriological investigations and the transmission of the disease by inoculation of artificial cultures have definitely settled the question. It does not follow that all cases are contagious, nor equally so, but the recognition of the contagious form satisfactorily explains the prevalence of the disease in one stable while an adjoining one escapes, and the eruption of new cases in a stable after an animal affected with the disease or convalescent from it has been introduced. It has been objected that many horses stand in the stable with pneumonia cases and escape, but so is it with glanders, cowpox, and many other affections. It merely argues an immunity in the case of some, and for the disease germ a very limited transmissibility through the air. The further objection that the existence of lesions in the lung before the onset of fever, excludes this from the list of infectious diseases, is untenable since many undeniably contagious diseases, like cutaneous anthrax, glanders, lung plague, cowpox, appear locally before any constitutional disturbance occurs, which later as the result of extensive local disease and the circulation of toxins in the blood. It places contagious pneumonia however in that long list of infectious diseases which develop first locally in the seat of infection and later become more or less generalized.

It must be admitted however that the germ of pneumonia is not the same for all cases of the disease and for all genera of animals. It must also be allowed that the same germ does not always maintain the same degree of virulence, and that it may even live for a time on the buccal mucosa of an animal belonging to a susceptible genus without any morbid result. In short we must recognize that different germs of pneumonia may become temporarily non-virulent or only slightly virulent, and remain pathologically quiescent, as for example during the summer months, to reassert itself later when the conditions become more favorable to its pathogenesis.

BACTERIOLOGY.

a. Bacillus of Friedländer. This is a short rod with rounded ends, often merely oval, occurring in pairs, or chains of

four, and under given circumstances surrounded by a transparent gelatinous capsule. It is ærobic, nonmotile, does not liquefy gelatine, nor sporulate, and in gelatine stick cultures has a nail-like growth. This was found by Friendländer, Frobenius, Weichselbaum and Wolf in the pulmonary alveoli in a small proportion of cases of croupous pneumonia in man. The cultures, injected into the lungs of animals, killed one dog (out of five), six Guinea pigs (out of eleven), and thirty-two mice (all the injected). Lesions were intense congestion of the lungs, seropurulent pleural effusion, and enlarged spleen, while the bacillus swarmed in the blood and exudate.

Micrococcus Pneumoniæ Croupos.æ. First found by Sternberg in his own saliva in health, and by Pasteur in the saliva of a rabid child. Afterward found in the great majority of lungs affected with croupous pneumonia in man, by Talamon, Salvioli, Sternberg, Fränkel, Weichselbaum, Netter, Gamalei, etc. Later it was found in meningitis, in ulcerative endocarditis, in arthritis, in otitis media, and in acute abscess in man.

It is a *spherical* or *oval coccus*, arranged in pairs, in fours, or exceptionally in eights or tens. Lanceolate forms are the rule in the blood of animals, and circular in artificial cultures. It stains readily in aniline colors and by Gram's method, grows in ordinary culture media, at 37° C. in the absence of free acid, and in gelatine stick cultures as small, white colonies along the line of culture, without liquefying the gelatine. It dies in ten minutes at 52° C. (Sternberg). Its virulence lessens in artificial cultures, but is restored by passing through the body of a susceptible animal.

Injection into the lungs or trachea of rabbits, mice, sheep and, less certainly, Guinea pigs, produced distinct fibrinous pneumonia filled with the microbe. In dogs, subcutaneously, it caused abscess, but in the lungs an acute fibrinous pneumonia which only exceptionally proved fatal, recovery usually taking place in ten to fifteen days.

Klemperer induced immunity, sometimes lasting six months, by intravenous injection of filtered cultures.

Diplococcus Pneumoniæ Equina. First found by Schütz in the lungs of pneumonic horses in 1887. It is an oval coccus arranged usually in pairs or in threes or fours, and surrounded by

a transparent envelope. It stains in aniline colors but not by Gram's method. It is ærobic and grows in gelatine at ordinary temperature without liquefying it, and in stick cultures forms a line of small, white, separate colonies which do not coalesce by growth. Does not grow on the surface of the gelatine around the puncture. Line cultures on agar are in colonies like minute transparent droplets. In bouillon it develops long chains.

Inoculated on the rabbit, Guinea pig and mouse, it produced death with pneumonic affections (hæmorrhagic congestion or inflammation), but it failed to take in some of the rabbits and Guinea pigs. Chickens and pigs proved immune. Injected into the horse's lung or as spray into the trachea it produced true croupous pneumonia. Fiedaler and others obtained similar results. Peter has found the fæces of pneumonic horses virulent, an important point in connection with disinfection.

Schütz found that 20 grammes of the culture, in an equal quantity of boiled water, injected into the horse's trachea, produced a rise of temperature by two or three degrees, with rigors, cough, accelerated pulse, elevated temperature, dyspnœa and prostration, but that this subsided in a few hours. By repeating this every thirty-six hours, the fourth or fifth would fail to produce a reaction and the subject proved immune.

Cadeac's Diplococcus Pneumoniæ Equina. In the lungs of cases of contagious pleuro-pneumonia of the horse Cadeac found a round noncapsulated coccus appearing in pairs, or rarely in chains, and staining by Gram's method. It grew slowly in bouillon and agar at 37° C., forming on the latter in twenty-four hours, a thick, whitish, oily drop, which, as it grew larger, assumed a silvery whiteness, and dried in the centre. In bouillon it precipitated a powdery sediment. The reaction of the culture medium was unchanged. It lost virulence rapidly in artificial cultures or by a heat of 50° C., and it died in ten minutes at a temperature of 60° C. Virulence was long retained when dried, or even in putrid material.

This proved infecting to the ass, rabbit and Guinea-pig, while the cat and white rat proved immune. Intratracheal injection of the dog produced a transient pneumonia. The ass inoculated with the blood of the infected rabbit died in three days, with a hepatized lung, pleurisy, and swarms of the microbes in the lungs,

blood and internal organs. Rabbits injected intravenously had enlarged spleen, reddish exudate in the serous cavities, urine stained with hæmoglobin, and lungs and kidneys congested. With intratracheal injections the lesions were exclusively pulmonary. The pulmonary lesions were less constant in the Guinea-pig. Weakened virus caused pulmonary lesions only without septicæmia.

It has been suggested that this coccus is at least closely related to that of pneumo-enteritis of the horse.

Symptoms. The onset of pneumonia is not often seen by the veterinarian, who is called in only after the cough, loss of appetite, hurried breathing and rigor has revealed illness to the attendants. Hence perhaps chill and rise of temperature have been placed among the earliest symptoms. The symptoms are more violent in the racer, trotter and other nervous animals. Trasbot positively claims, that considerable pulmonary inflammation and even exudation have taken place before there is any chill or rise of temperature. This is especially the case in the heavy lymphatic races of draught horses, which often according to this author perform their usual work for days after inflammatory exudation has set in. A fair counterpart of this is found in lung plague of cattle and it would indicate that both start from a local infection, which gradually extends until the systemic derangment is induced. As usually seen, and especially when it follows exposure to severe cold, a staring coat or a shivering fit usher in the disease, the degree of the chill bearing some ratio to the coldness of the air and to the future severity of the malady. This may be accompanied by a small, dry cough, but without any other marked sign of lung disease. With the access of the hot stage the characteristic symptoms of lung disease are manifested, at first resembling those of *congested lungs*, but less severe than those given under that head. There is a distinct increase of the body temperature ; the visible mucous membranes are suffused with a blush ; the expired air feels hot upon the hand ; the breathing, 30 to 40 per minute, is short and accompanied by much lifting of the flanks—(*labored*) ; the cough is deep as if coming from the depth of the chest, but not so hard nor so painful as in bronchitis ; the legs are placed apart, the elbows turned out and the head protruded to facilitate breathing ; the nose is turned to an open door

or window if any such is available ; the contraction of the muscles of the face, the dilated nostrils and the retracted angle of the mouth give an anxious expression to the countenance ; the eyes are semiclosed ; the pulse full but soft—(*oppressed*)—, beats from 48 to 70 per minute ; the bowels are slightly costive, the urine scanty and high colored ; the skin inelastic—*hidebound*—harsh and dry, though sweats may bedew it in parts ; the loins insensible to pinching ; and if there is any discharge from the nose it consists only in a reddish—*rusty*—colored mucus.

Auscultation and **percussion** complete the diagnosis. At the outset the inflamed portion of lung, usually near its lower part, conveys a *crepitating* sound to the ear, but as consolidation extends the healthy murmur and the crepitating râle are alike suppressed over the whole extent of the hepatised portion around the margin of which a line of crepitation betrays the limit of the advancing inflammation. A similar line of crepitation encircles th hepatised mass even when the exuded products are being absorbed and when the lung is being cleared up and restored to its healthy state. Thus the advance of the inflammation, and the progress of recovery can be equally followed by the crepitation which, in the different circumstances, betokens active inflammation or active absorption. When both lungs are involved the posterior parts are chiefly implicated, while if the pneumonia is single it may attack the anterior, median or posterior part, or the entire lung may become consolidated. If hepatisation exists in the anterior part of the lung the thick fleshy shoulder will forbid any satisfactory examination, but if in the middle portion only, while the respiratory murmur is lost it will be replaced by a strong blowing sound (bronchial respiration) because the noise of the air rushing through the larger bronchial tubes to the posterior healthy part of the lung is conveyed with greater force to the ear through the consolidated lung tissue. This is audible from the lower third of the chest to the upper limit of hepatization. The respiratory murmur in the healthy lung is always louder than is natural.

Percussion confirms these results. Over the hepatised lung where no respiratory sound remains, a dull, dead sound only is brought out by the impulse of the fingers or closed fist, comparable to that obtained by percussion over the muscular masses of the shoulder or haunch, and forming a marked contrast to that

obtained over the surrounding healthy lung. There is not that tenderness on pressure in the intercostal spaces which characterises pleurisy, but a sharp blow with the closed fist leads to wincing and usually grunting because of the concussion to which the diseased part is subjected. By increasing the force of such blows the deepest parts of the lungs may be tested, since in this way dullness due to consolidation of the deeper portions of the lungs may be detected even though the superficial investing parts are healthy.

The nature of the symptoms will vary according to the extent and character of the inflammation, from mild febrile reaction, with excited breathing and slight crepitation, to the more severe varieties in which the intensity of the symptoms are such as to threaten suffocation.

A marked feature of pneumonia in solipedes is that the patient obstinately stands in one position and never lies down so long as the severity of the inflammation lasts. The sharp crest on the lower border of his breast bone compels the horse to lie on his side, and since in this postion the whole weight of the body has to be overcome in any full dilatation of the chest, he cannot retain the recumbent posture when any serious impediment to breathing exists. Hence it is that the fact of a horse suffering from pneumonia having lain down and remained so for some time is justly accepted as an indication of improvement.

Progress and results of the disease. The general symptoms above noted, remain with more or less intensity throughout. After the first flush of heat, on the occurrence of febrile reaction, the limbs become alternately hot and cold, and in this the general surface partakes to a less extent.

The tendency of pneumonia is to a crisis and recovery. Certain days have been supposed to be critical and on the whole the third, seventh, eleventh and fourteenth are those on which a favorable change is most probable.

Among the more *favorable indications* are the manifest abatement of the high bodily temperature and febrile symptoms generally, the increasing ease and regularity of the breathing, the greater force, distinctness and slowness of the pulse, the permanent return of warmth to the limbs, the softer and more elastic feeling of the skin, the recovery of appetite, and above all, the

turning of the nose from the open window or the retention of the recumbent position for a length of time. These symptoms will become more patent day by day, and the absorption of the effused products and the clearing up of the lung may be traced by the gradually decreasing area of dullness and of the circular line of crepitation as ascertained by percussion and auscultation.

If on the contrary the disease takes an *unfavorable* turn, some such signs as the following will manifest it: Increasing rapidity and embarrasment of the breathing; smallness and indistinctness of the pulse, which is increased to perhaps 100 beats per minute; tumultuous heart's action, the impulse of which is strongly felt behind the left elbow; a more laborious working of the flanks; frequent despondent looking toward the flanks; pawing with the fore feet, lying down, and as suddenly rising again; permanent coldness of the extremities; hanging head with great dullness and despondency of expression; dull, sunken, lusterless eye; hanging lower lip; leaden hue of the nasal mucous membrane; convulsive twitching of the muscles of the surface; reeling in gait, and extension of the crepitation over all the still pervious lung.

Subacute Pneumonia. This term is employed to designate that subdued or milder form of the disease which sometimes arises spontaneously and at others follows the acute.

In this variety the characteristic symptoms may be much less marked and the disease is less easily recognized. There is some acceleration and quickness of pulse, lifting of the flanks and heat of the mouth and body generally. There are alternations of heat and cold of the surface and extremities, a rough, unthrifty coat, hidebound, a dull, listless moping manner and the same symptoms on auscultation and percussion as in the acute form.

The changes take place slowly but the disease may prove obstinate and is often followed by permanent alterations in the lungs. Rheumatic affections of the limbs, inflammation of the feet, and other diseases frequently supervene during the course of this form of the affection.

The **terminations** of pneumonia are:—by **death**; **resolution** with absorption of exuded products:—**splenisation**; **abscess**; **gangrene**; permanent **consolidation** with organization of exuded products. The disease will sometimes lapse into the chronic form.

Death is fortunately the least frequent issue. It may follow on rapidly advancing and general congestion of the lung,—asphyxia; from heart failure, the overworked organ becoming exhausted under the strain of forcing the blood through the virtually impervious lungs; from hyperthermia, the limit of bodily temperature 108°F. having been reached or exceeded; or from collapse and exhaustion.

In **resolution** which is the most favorable termination the febrile and other symptoms subside and the exudations in the effused lung undergo a process of liquefaction and absorption until neither auscultation, nor percussion nor even the examination of the lung after death will show the slightest trace of the pre-existent disease. This is the most common termination in single pneumonia in the horse.

Splenisation is that condition of lung already described under the head of *pulmonary congestion*, and if affecting both lungs throughout, necessarily destroys life by arresting the aeration of the blood.

Abscess. Diffuse suppuration is very common in the stage of gray hepatization. In this the affected lung becomes more or less extensively infiltrated with pus limited by no distinct membrane like the pus of an abscess, but exuding freely from the cut surface of the lung or escaping from its interstices when it is pressed. It is preceded and in its early stages associated with the formation of granular masses and corpuscles. Its existence cannot be certainly ascertained though it may be surmised when after hepatization of a portion of lung a *mucous râle*, a sort of gurgling, is heard in the adjacent bronchium and an abundant muco-purulent discharge takes place from the nose. It threatens extensive destruction of lung tissue.

Circumscribed suppuration or *abscess* is infrequent though occasionally met with in the horse and ox. In this case the excessive exudation at one point liquefies and the surrounding lymph becoming organized into a vascular membrane an abscess is formed. This may burst into the bronchial tubes and be discharged by the nose. In less favorable cases it makes its way toward the pleural surface and opens into the cavity of the chest. It is impossible to detect the existence of a pulmonary abscess though after it has burst into a bronchial tube the existence of the cavity may be ascertained by the amphoric sound heard on auscultation.

Animals may recover from such pulmonary suppurations or if they are too extensive the consequent depletion may induce hectic and death.

Gangrene of the lung is happily rare and has appeared to be connected with close, foul stables, previous ill health, and work after the onset of pneumonia. It is characterized by high temperature (105° to 108° F.) by great dulness and prostration due to the poisoning of the nerve centres, by weakness and unsteadiness, by complete loss of appetite, and at length an intolerable fœtor of the breath as if from putrefying animal matter. In rare cases recovery may take place, the dead portion having become detached and expectorated.

Consolidation from hepatization is the condition in which the inflamed lung is always found, in the second stage of the disease. The lung has then the density and brownish red appearance naturally belonging to the liver (*red hepatization*), which changes on the occurrence of softening of the exuded products to a grayish hue (*gray hepatization*). But after the subsidence of the acute symptoms, the process of liquefaction and absorption is not always complete, a portion of the exuded product becomes vascular, is developed into fibrous tissue and remains permanently impervious to air. Such is the state of the lung in many cases of *thick* or *short wind* in horses when these have occurred as a sequel of pneumonia. A horse suffering in this way has the breathing habitually accelerated and is thrown into a state of great distress by any attempt to make him perform hard work such as galloping, dragging a load up hill and the like. A *chronic cough* may equally accompany this condition.

Pathological Lesions. These differ according to the stage of the disease. In the *first stage*, that of congestion the lung tissue is engorged with blood as described under the head of **congested lungs.** As early as 6 or 7 hours after artificial irritation, the alveoli of the affected part are already filled by exudation and cell proliferation. Until this has taken place the alveoli can still be distended by blowing into the bronchial tube.

In the **second stage** the condition of the lung is that of **red hepatisation**, so called from its resemblance in color and consistency to the liver. There are gradations between congestion and red *hepatisation.* In the earlier stages of the latter, the lung

retains a measure of its softness, elasticity and permeability to air, though it is considerably firmer and less permeable than that which is in a state of congestion and differs further from it in exuding from its cut surface not a grumous, dark bloody pulp, but a clear straw colored fluid. In the advanced *red hepatisation* the lung is of a firm consistency and granular liver like appearance. In color it varies from a bright red to a dark liver hue, the darker shades being chiefly met with in old animals or when the inflammation and fever have been intense and prostrating. Varying shades are seen in different lobules of the same lung. Its air cells are no longer pervious, it no longer crepitates under the pressure of the finger, nor floats in water, and its friability is such that it breaks down readily when the finger is thrust into its substance. Its surface is distinctly granular from the fibrinous plugging of the alveoli. Such a lung does not collapse when the chest is opened but retains its bulk and shape and in some cases the diseased portion may, by reason of the abundance of the exudation, be really larger than the same portion of lung in a normal state of dilatation. Its surface may thus retain the imprint of the ribs. Owing to the stasis of the blood in the vessels a hepatised portion of lung cannot be injected. The exudation which infiltrates the lung tissue and obliterates the air cells contains in the vicinity of the bloodvessels numerous granular masses and corpuscles and in the darker colored portions blood globules, owing to the action of diapedesis of the red cells and the rupture of minute vessels. The smaller bronchial tubes stand out white and empty showing that they have escaped the inflammatory action. Hepatization usually extends from the anterior lobe or lower border upward.

Gray hepatization is a sequel of the *red* and presents the same firmness, friability and usually the same granular aspect; the lack of crepitation on pressure, and the higher density than water. From the cut surface a fatty or purulent fluid exudes spontaneously, or in other cases only when pressure is applied. The granular masses and corpuscles have disappeared, and if supuration is not so abundant as to prove extensively destructive to lung tissue, that is gradually cleared up and restored to health. This state is always a very perilous one.

Abscess of the lung sometimes met with in animals dying of

pneumonia shows a circumscribed area of inflammation and induration with the liquid pus in the centre immediately surrounded by a vascular (limiting) membrane. Abscess may be single or multiple though in the latter case it is commonly a symptom of pyæmia.

In *gangrene of the lung* the part may be in the dried condition of an eschar; it may indicate gangrene only by its altered color, its flaccidity, its fetid smell and the altered appearance of all its microscopic elements; it may be denoted by a putrid softening, the tissue easily breaking down into a stinking pulp of mixed fibrous and granular materials; or lastly there may be merely a cavity with traces of putrid contents, the dead mass having been detached, disintegrated and expectorated.

Modifications of the Blood and Distant Organs.

A marked feature of pneumonia is the destruction of red blood globules. This is early indicated in the staining of the visible mucosæ by the liberated hæmaglobin and by actual count they may be reduced in the horse from 7,500,000 to 6,000,000 per cubic millimeter (Trashot). There is an increase of white globules, an absolute increase, not only in ratio to the red. The hæmatoblasts are enormously increased especially during defervescence. The fibrine (fibrine formers) is materially increased; in the horse from 3.5 to 6.7 or 7.5 per 1,000 (Grehaut). Albumen is diminished. Soda salts are increased. The bronchial lymphatic glands are always congested, swollen and reddened with some serous effusion. They may become the seat of inflammatory cell growth (embryonic tissue) or even of suppuration. The abscess may open into the bronchia or pleura. These are especially to be dreaded from their tendency to implicate the inferior laryngeal nerve and induce roaring.

Pleurisy is inevitable when the inflammation reaches the surface of the lung, hence hydrothorax is often present. Pericarditis and hydropericardium are similarly met with. Endocarditis is occasionally present and may be traced to strain of the valves of the laboring heart, or to direct infection with the pneumonia microbe. Dilatation of the right ventricle is common as a result of the obstructed pulmonary circulation.

Fatty degeneration of the heart and congestions of the intestinal mucosa, liver, kidneys, and spleen are further complications.

Finally laminitis and rheumatoid affections occur as complications.

Treatment. This must be adapted to the nature and condition of the subject and to the character of the disease. A horse in vigorous condition or with an acute type of inflammation may be greatly benefitted by an actively depleting treatment, whereas to the same animal in a low state of health, or during the prevalence of an epidemic form of the malady depletion may be destruction. It is not sought here, as is so often done in the consideration of this disease, to ring the changes, as to the probability of a change of type in disease, or a change of theory on the part of physicians, having affected the practice of bloodletting. True to our primary purpose of rendering the work eminently practical, we shall first notice the general management applicable to all cases, then the treatment of the two great types of the disease, acute (sthenic), and subacute (asthenic), leaving to the enlightened judgment of the reader to apply an appropriately modified system to that large class of cases which occupy an intermediate position.

A pure airy box is first demanded, with the windows or doors toward the south, or at least not turned in the direction of the prevailing cold winds. The craving for pure air, so strikingly shown by the position which the animal assumes, ought never to be ignored nor neglected. We do not advocate the system of the late Professor Coleman who kept pneumonia patients in open sheds exposed to all vicissitudes of temperature winter or summer, and yet the fact that many recovered under such treatment as well as under a more rigorous system, having been turned out into the open fields amidst frost and snow, ought to open the eyes of all to the incomparable value of fresh air in this disease. The box then must be dry, cool and airy but without a cold exposure and without draughts of cold air.

Next in importance to pure, cool air is the comfort of the patient. Any tendency to chill, shivering, staring coat, or coldness of the surface and extremities is to be counteracted as far as possible. One or more blankets according to the condition of the patient and the temperature of the atmosphere are valuable and for the same reason a hood may be put on. Coldness of the limbs is

to be met by active rubbing with the hand or with wisps of dry hay and then wrapping up loosely in flannel bandages. Some apply to the limbs ammonia and oil, spirits of turpentine, and other stimulants and thus by a powerful derivative action obtain an alleviation of the lung symptoms. For the same reason a mustard poultice on the chest, or the hot wet rugs recommended for congested lungs, often prove valuable in the earlier stages. Large injections of warm water and the supply of warm gruels are not to be neglected when they can be employed. Measures such as these directed to check any chill and render the circulation free and uniform in the skin and extremities, if adopted during the cold stages of the fever, will sometimes succeed in bringing about a resolution of the pulmonary congestion and warding off a threatened attack of pneumonia.

The diet should be of a non-stimulating and laxative kind. Bran mashes, linseed, oatmeal, or other gruels, carrots, turnips, scalded hay, or green food, if at the proper season, should be given in small quantities so as not to satiate.

Antiphlogistic Treatment. Half a century ago bloodletting was considered the remedy *par excellence* for pneumonia and it seemed justified by the marked relief to breathing and pulse which usually at once followed a free bleeding. In a short time, however, the fever would rise anew and the distressing symptoms reappear, which led the school of Broussais to repeat the bleeding, *coup sur coup*, as often as the exacerbation appeared. There was no respite for either age or condition, the debilitated city toiler, the babe at the breast, and man of eighty tottering into the grave had alike to submit to the lancet, and when the oppressive symptoms returned, the blood had to flow anew. Broussais himself, however, recognized his error in his later life, and remarkably enough, his conversion was effected through veterinary practice. His two carriage horses were successively attacked by pneumonia: the first was treated by bleeding *coup sur coup* and recovered: the second was put under a more conservative treatment and also got well, but while the first remained soft, flabby, debilitated and susceptible for a length of time, the second was on convalescence at once able to go into active work. The enormous abuse of bleeding, led to its more complete abandonment than would otherwise have been probable, and the contrast between the high mortality

of cases treated by excessive bleeding, and the lower fatality in pneumonias treated without phlebotomy on the expectant (let alone) plan of Dietl or the stimulating method of Todd, Bennett and others, served to hasten its abandonment. Yet in bloodletting we have an instrument for good or evil which is not to be judged on slight evidence. The mere lessening of the blood pressure is to be little considered, as it requires the abstraction of nearly one-third of the entire mass of blood to visibly affect this. The vascular walls at once adapt themselves to the lessened amount. Nor is the mere lessening of the volume a vital point. After moderate bleeding this is made up in a few hours: after severe bleeding in 24 to 48 hours. The loss of adult red globules is more lasting. Bleeding to the extent of one per cent. of the body weight may have the number restored in seven days. The young red globules though rapidly produced have individually less hæmoglobin, and they can convey less oxygen to the tissues. This should mean less oxidation, less heat, less waste, less urea, uric acid, hippuric acid and other poisonous products in the tissues. Yet Baur says that in anæmia there is a greater metabolism of proteids and more excretion of urea. How easy it is to blunder in looking from one single point of view. Again after bleeding there is a great relative increase of the various forms of white blood globules, most of them young and therefore with somewhat altered functions. The paucity of red globules and excess of white are brought about by the pneumonia and independently of bleeding, so that it is difficult to say whether the phlebotomist is enhancing an evil, or helping a natural therapeusis. It seems hopeless to estimate the effects of these and other changes in the blood after bleeding, upon the metabolic processes of nutrition, secretion and sanguification. This digression has not been made to ellucidate the results or the *modus operandi* of bleeding, but rather to illustrate the complexity of the problem involved and to warn against broad and unwarranted generalizations from insufficient premises.

Even to-day practitioners of the soundest judgment meet with a limited number of cases in which they resort to bleeding with advantage. These occur mainly in strong, robust constitutions, in individuals accustomed to an invigorating, open air life, liberal diet and abundant exercise. Even in these this measure is chiefly resorted to, to relieve an acute pulmonary congestion with a

dangerous distension and over charging of a fatigued and overworked heart. In short the condition is one closely allied to acute congestion in which the value of bleeding is all but universally admitted. It is especially warranted early in the disease, though it may still be adopted with caution in a similar condition which has supervened at a later stage. A strong pulse and bright red mucous membranes, are not as has been supposed, essential prerequisites to its employment. The mucosæ may be pale, or more likely cyanotic, and the pulse small and weak, from the over charging of the heart and its tendency to failure, and it is to relieve these conditions that we adopt this most potent of all measures for securing a temporary lessening of the blood pressure in the right heart and pulmonary circulation. Even the transient relief may allow this to right itself and then less radical or dangerous measures may be relied on. Bleeding should very rarely be resorted to save at the outset of the disease; extensive exudation into the lung tissue strongly contra-indicates it; it cannot be safely employed in the very young or old, in weak or debilitated subjects, when the pneumonia has relapsed or supervened on another serious malady, or when occurring in an unhealthy district. Delafond met with a very high death-rate from bleeding in a damp undrained locality. Where bleeding is permissible, the blood should be drawn from the jugular in a full stream, from a large orifice, the finger being placed upon the pulse, and the flow arrested as soon as the blood is felt to pass along the vessel in a fuller, freer current, and the breathing is seen to be relieved. It can rarely be repeated with profit or safety, and in the vast majority of cases can be well dispensed with altogether.

Antipyretic Treatment. When the temperature runs dangerously high, a temporary use of antipyrin, acetanilid, phenacetin, or other potent antithermic remedy may be resorted to. But agents that so profoundly affect the heat centres are not devoid of danger and should not as a rule be continued after the dangerous excess of temperature has been overcome. They may be looked on as valuable to temporarily obviate an extreme danger rather than as a form of regular treatment.

The modern resort of applying ice bags to the chest may be similarly disposed of. In very high fever they have been apparently beneficial, but the danger of chill or injurious reaction is so great

that they must be employed with the greatest possible consideration and care.

Refrigerant Febrifuge. Neutral salts such as saltpeter in 2 drachm doses every six hours may be given in the drinking water. These are valuable for their cooling and eliminating action, and possibly in counteracting the viscidity of the blood and exudations. Acetate of potash, bicarbonate of soda, iodide of potassium or muriate of ammonia may be substituted.

Stimulants. In debilitated subjects or with low fever or oppressed heart the stimulating diuretics like sweet spirits of nitre or liquor of the acetate of ammonia are to be preferred, and this is especially the case during convalescence. They at once sustain the flagging heart and aid in the excretion of morbid products. Digitalis is often of great value in the same sense, and as a heart stimulant nux vomica.

Some follow Todd and Bennett in seeking stimulation from alcohol, ammonia and its salts, ether, etc. When the circulation is weak or flagging these are often of value and they may even act directly on the pathogenic microbe. The inhalation of oxygen, or the solution of proxide of hydrogen given by the mouth has often an excellent effect.

Sedatives. Aconite has become too much of a domestic remedy, nevertheless it may be used with advantage in high fever with excited heart action, to moderate the circulation and relieve the breathing. Veratrum, hydrobromic acid, bromide of sodium or ammonium, or chloral hydrate may be used as alternates or substitutes.

Compresses. Fomentations. Poultices. No measure is safer nor more promising, especially in the early stages, than the poultice jacket or compress. A blanket wrung out of hot water is wrapped around the chest, covered with a thick dry one, and held firmly attached by elastic circingles. Or soaked cotton wool is applied and covered with a dry blanket or a rubber sheet. The more acute the inflammation the more valuable is this measure.

Derivatives. As a derivative the mustard pulp rubbed in and covered with thick paper or rubber is especially valuable. In one hour it may secure a free exudation and material relief to the breathing. It may be replaced by ammonia and oil, with or without a covering, by hot water or by cantharides. This must how-

ever be used with judgment. In the early stage with a high type of inflammation and fever the surface irritation may aggravate this through sympathy ; in such cases therefore the severity of the inflammation should first be moderated before using an active counterirritant. In debilitated conditions, too with an altered or depraved state of the blood and during the prevalence of a low type of the disease, sloughing may ensue from incautious blistering;

The repetition of the blister is often useful, the healing process going on simultaneously in the blistered surface and the diseased lung by virtue of nervous sympathy.

To complete recovery a course of vegetable tonics, such as gentian, nux vomica, calumba, may be given with iodide of potassium for a week or more. Constipation occurring during convalescence must always be corrected by food, (bran mashes, linseed gruel), injections, or oleaginous, saline, or aloetic laxatives. The greatest care should be exercised to secure pure air, comfort, sunshine, good grooming and general hygiene, and to prevent overexertion during convalescence.

In the *subacute* types of pneumonia the fundamental difference in the treatment consists in the avoidance of all depressing remedies and the employment of stimulants and a supporting diet from the beginning. Sweet spirits of nitre and liquor of the acetate of ammonia, carbonate of ammonia or salammoniac with digitalis and strychnia may be used from the first. Vegetable tonics may be resorted to at an early stage, peroxide of hydrogen, and when expectoration is established and the fever moderated even mineral tonics may be employed. Nourishing gruels, mashes, roots, green food, and scalded oats may be used in turn to coax the appetite and not to satiate. In other respects the treatment is the same as for the acute. This form of the disease is liable to prove obstinate and persistent, and there appears to be a greater tendency to complications and so called metastasis, as enteritis, laminitis or rheumatoid affections of the back or limbs. These when they occur must be treated as if they had arisen in ordinary circumstances, having regard meanwhile to the remaining inflammation in the lungs, for that has not necessarily been quite superseded but only alleviated.

Chronic Pneumonia. This has been described but if uncom-

plicated by consumption it appears to be usually only that consolidation of lung, due to the organization of exuded products into fibrous tissue, which occasionally forms a sequel of acute inflammation of the lungs. In such cases an access of circumscribed local congestion is liable to result from overexertion, or a chronic state of irritation is maintained attended with more or less fever, inappetence, mal-assimilation, and often in the long run hectic, under which the animal is worn out. In such cases the chief indications are to avoid overwork or any undue strain upon the breathing organs, to support the patient by nourishing and easily digested food, and to control and remove any local irritation by measures indicated under the head of acute pneumonia.

CROUPOUS PNEUMONIA IN THE OX.

Subacute in many cases. Effect of temperament, and work. Acute form. Symptoms. Decubitus. Unfavorable symptoms. Prognosis. Suppuration frequent : indications. Gangrene. Coliquative Diarrhœa. Lesions, Comparison with those of lung plague. Tubercle. Treatment, bleeding, laxatives, refrigerant salts, derivatives, stimulants, tonics. Chronic form. Symptoms. Treatment.

In the large ruminants this disease tends more towards a subacute type than in the horse, and coming on insidiously from ordinary causes is liable to be confounded with the *contagious pleuro-pneumonia* of the bovine race. As in the horse the nervous animals show more violent symptoms. It is rare in milch cows and young cattle and more frequent in work oxen.

In the *acute form* the symptoms mainly agree with those of the horse. There is the same shivering, followed by a hot stage, hyperthermia, the accelerated pulse, the short quick labored breathing, heaving flanks, cough frequent, deep, hacking, and easily excited, dilating nostrils, redness of the mucous membrane, and the same indications on auscultation and percussion, care being taken to obviate misconception of natural conditions in the chest of the ox. There is in addition a dry muzzle, tenderness of the back and breast bones and wincing when they are pinched between the fingers and thumb ; suspension of the appetite and rumination and in cows suppression of the secretion of milk ; the mouth is often opened and the tongue protruded to facilitate breathing,

and in bad cases each expiration is accompanied by a moan or grunt. In many cases the ox can lie on his flattened breast-bone and maintain the breathing process, but when the disease is severe he stands no less obstinately than the horse, his elbows turned out, his nose protruded and directed towards a window or other opening.

Among the unfavorable symptoms may be mentioned increasing anxiety and distress, a more oppressed breathing, the animal standing constantly in one position with legs apart, elbows turned out, his nose extremely raised, nostrils widely dilating, mouth open, tongue protruded, the expiratory grunt deep and prolonged, the cough infrequent and so weak as to be almost inaudible, being rather like a forced expiration, and the pulse rapid, feeble or imperceptible. The prognosis is favorable in moderate cases subjected to early treatment.

The termination by *suppuration* is more frequent than in the horse. The general symptoms are ameliorated, appetite and rumination return though they remain capricious and irregular, there remains the double action of the flanks, the dry, rough muzzle, the tense, inelastic skin, frequently varying in temperature, the beast shivers at intervals, the cough is weak and often repeated, a yellowish thick discharge takes place from the nose, weakness and emaciation increases and the animal dies in from twenty to thirty days.

Gangrene of the lung sometimes supervenes and is indicated by similar symptoms as in the horse. In severe and prolonged cases a violent fetid diarrhœa often supervenes and hastens a fatal result.

The *post mortem* lesions are similar to those of the horse. The cut surface of the hepatized lung, however, is divided into irregular red spaces by intersecting yellow lines—hence the name of *marbled lung*, from a supposed resemblance to that stone. The red spaces represent the pulmonary lobules and the whitish lines the surrounding areolar tissue which being especially abundant in ruminants and pigs stands out prominently when infiltrated with the yellowish exudation. There is then nothing specific in this appearance as has been erroneously supposed, it is merely the result of the different conformation of the lung in these animals and is always seen in the hepatized lung unless when from extravasation of blood into its substance the

redness is rendered uniform. The amount of exudate into the interlobular tissue is, however, never so great as in lung plague.

The greater frequency of suppuration in the lung of the ox, as well as the greater tendency to tubercular deposit in prolonged cases are additional features in the diseased lungs.

Treatment. Blood-letting should be employed only with precautions, as in the horse. A saline laxative (1 lb. Epsom salts and ½ lb. molasses) may be used with advantage and safety early in the disease though in advanced stages it may sometimes prove dangerous from the tendency to diarrhœa. If constipation appears at a late stage injections of warm water and a mild laxative (6 ounces sulphate of soda) only, should be given. The purgative may be followed by the same neutral salts and in the same doses as for the horse. Counterirritants are of equal value. A mustard poultice may be kept on for several hours, or a mixture in equal parts of oil of turpentine, ammonia, and olive oil may be actively rubbed over both sides of the chest and repeated daily until tender. In Denmark a prompt and efficient blister is made with 1 part of Croton oil and 10 parts each of sulphuric ether and spirits of wine. This is rubbed actively over the chest and washed off as soon as a sufficient effect has been produced. It must be carefully watched to prevent blemishing.

In the low types of the disease and during convalescence stimulants and tonics are to be employed as recommended for the horse.

Chronic Pneumonia. Gellé describes a chronic form of this disease in cows. For about a month the patient became increasingly emaciated, there was a frequent, dry, weak cough, lifting of the flanks, and expiration double and accompanied by a moan. All these symptoms were aggravated by gentle exercise. Percussion detected dullness at the lower part of the lung and auscultation a distinct crepitating râle. The pulse was weak and rapid, the mucous membranes red and tumid, skin dry, ears and horns cold, appetite small and capricious, rumination rare, excrements soft, and milk almost dried up.

The *treatment* is by diuretics with vegetable tonics and stimulants and active counterirritation over the chest. Gellé considers the malady as all but incurable unless active blistering is promptly employed so soon as the malady has assumed the chronic form and before extensive structural changes have taken place in the lungs.

CROUPOUS PNEUMONIA IN SHEEP.

Causes, damp, cold soils, inclement weather, cold rains, hard driving, shearing or washing in cold weather, change to a cold climate, or from a warm barn, hot barns, heavy fleeces, sudden plethora. Symptoms, in congestive cases, in inflammatory. Treatment, preventive, hygienic, antiphlogistic, laxative, febrifuge, derivative.

This disease is not unfrequent in these animals, occurring enzootically in low, wet pastures; or from cold storms of wind, sleet or drenching rains, particularly after hard driving, or shearing; or from washing during inclement weather. Dressing with mercurial ointment in cases of *scab* is a frequent cause of pneumonia and death in Lincolnshire and various other English counties. Lastly M. Seron in Hurtrel d' Arboval's "*Dictionaire*" describes its prevalence in *Seine-Inférieure* among low conditioned sheep subjected abruptly to a very nutritious diet. The hot buildings, heavy fleeces, and sudden plethora, appear to conduce to dangerous pulmonary congestions. The *symptoms* do not differ materially from those seen in the ox except so far as they are modified by the fact that the disease often terminates fatally before hepatization has been established and the symptoms and post mortem appearances are those of congestion and sanguineous engorgment of the lung rather than of hepatization.

This engorged state of the lungs it is which has led Youatt and others to describe them erroneously as "gangrenous" and shepherds to name the disease "rot of the lights." The condition is that of acute congestion and analogous to that seen in congested lungs in the horse.

The *treatment* ought to be chiefly preventive and will consist in the avoidance of the causes above indicated.

When the disease has set in, fresh air, and general comfort, bleeding if in the very earliest stages and in a strong patient, purging (3 ounces sulphate of soda and ¼ lb. treacle in warm gruel) and a free supply of nitre (about ½ an ounce daily to each) in the water or gruel supplied are the leading indications. As a counterirritant aqua ammonia acts well being sufficiently confined by the fleece.

PIG. PNEUMONIA.

Symptoms, chill, burrowing, hot skin, cough, disturbed breathing, indications of exudation. Treatment, laxative, sedative, nauseant, febrifuge, wet jacket, blister.

Hogs are not exempt from this disease. They show the same symptoms of chill with hiding under the litter, followed by a hot stage, cough, hurried breathing, and (if the clothing of fat is not too thick) conclusive results on auscultation and percussion.

As *treatment* bleeding from the ears and tail is sometimes resorted to with questionable benefit. A laxative of three ounces of castor oil or three or four croton beans given in the food is of value. Tartar emetic in doses of ¼ grain and nitrate of potash in 10 grain doses should be shaken on the tongue at least four times daily to keep up a continued nausea and action on the urinary organs. The tartar emetic so worthless in the larger animals is of value in the pig and dog. A damp compress or blister may be used. The skin of the animal is difficult to blister, but by the use of the Danish croton liniment, mentioned for the ox, of hot water, or of a mixture of oil of turpentine and croton, 8 parts of the former and 1 part of the latter, a sufficient effect can usually be obtained.

DOG. PNEUMONIA.

Breeds most liable. Causes, overexertion, cold baths, clipping, exposure in cold, distemper. Symptoms, chill, fever, disturbed breathing, cold extremities, cough. Treatment, dietary, nursing, laxative, nauseant, febrifuge, moist jacket, mustard, stimulants, tonics, heart tonics and careful nutrition during convalescence.

Pneumonia in birds. Causes, exposure, neglect, foul coops, hot, close houses, etc. Symptoms, erect plumage, drooping head, wings, and tail, dark comb, gaping, panting, cough, crepitation. Treatment, hygienic, laxative, febrifuge.

This is a frequent affection in hounds. In hunting or coursing dogs the causes are like those operating in the horse. The

clipping of long haired dogs in inclement weather, swimming dogs in winter without afterwards drying or heating them by exercise, and shutting them out of doors at night, when accustomed to a warm dwelling are occasional causes. It sometimes occurs epizootically and frequently supervenes during distemper.

The chief *symptoms* of chill, fever, and difficulty in breathing are like as in other animals, while the results of auscultation and percussion are more satisfactory than in any other domestic animal. The dog sits on its haunches to facilitate breathing; his elbows turned out, his mouth open and his tongue protruded. Coldness of the ears and a short quick cough are usually marked symptoms.

Treatment. The general care applicable to other animals is equally demanded here. The diet should consist of mild broths, or farinaceous foods with a little gravy if necessary to render it palatable.

Bleeding from the jugular has been recommended and may be admissible at the outset of the disease in a very few appropriate cases. If costiveness exists a tablespoonful of castor oil may be given (more or less according to the size of the animal), following this up by the tartar emetic, nitre and sugar recommended for bronchitis. The poultice jacket is of great value. Mustard poultices may later be applied to the sides of the chest. Stimulants, tonics and nourishing diet may be required during convalescence, or when the disease assumes a low type.

CROUPOUS PNEUMONIA IN FOWLS.

In chickens exposure and neglect are alleged causes. Foul coops and the contrast between the warm building and cold outer air are justly blamed. Ruffled feathers, drooping head, dark colored comb and wattles, trailing wings, a disposition to gape, panting and cough are noticed. Under the wings and over the back crepitations and dulness may be detected. The patient may take a teaspoonful of castor oil, and saltpetre or iodide of potassium may be given in the drinking water. In careful doses the other remedial measures may be attempted.

ACUTE PLEURISY IN THE HORSE. PLEURITIS.

Causes, cold, damp, soils and exposures, as with rheumatism, youth, vigor, heavy diet, digestion, or hepatic disorder, overexertion, perspiration and succeeding chill, wading or swimming rivers, standing in snow, salted snow, rain, sleet, snow, draughts between open doors and windows, clipping, cold sponging of legs, tuberculosis, a common cause in man and cattle is rare in horses, surface pneumonias, cancers, actinomycosis, tumors. Traumas from broken rib, penetrating intercostal wound, blows, contusions, ruptured pulmonary or intercostal abscess. Irritant (infectious) exudate suggests microbes. Symptoms, chill, reaction, partial sweats, pawing, pointing one foot, hyperthermia, hard, jarring pulse, hurried breathing, inspiration catching, pleuritic ridge, uneasy movements, hacking cough, tumors and twitching of chest muscles, tender intercostals, grunting, friction sound, subsiding with appearance of dull area below, signs of effusion, relief, dyspnœa, lifting flanks and loins, perspirations, stocking limbs, pasty swelling on sternum, effusion of same level on both sides, creaking sounds, splashing, gurgling, metallic tinkling, weakness, sinking. Signs of adhesions, compression of lung, abscess. Duration. Lesions, early formation of false membranes, pleuritic effusion, its composition, its color at different stages, dry pleurisy, serofibrinous, sero-fibro-purulent, hydro-pneumothorax, tubercle. Prognosis. Treatment, during the chill, warm air, clothing, drinks, injections, compresses, pilocarpin during early inflammatory stage, derivatives, dry cupping, mustard, cantharides, hot water, or air, cold applications, laxatives, calmatives, anti-rheumatics, alkaline agents, with bitters, diuretics, heart tonic, iodine, mercury, thoracentesis.

Causes. Pleurisy is common in all domestic animals and especially so in cold, damp, exposed localities which suffer equally from rheumatism. It occasionally extends to the fascia of the limbs, the joints, or the navicular or other trochlea as a rheumatic affection. The disease is prevalent among young and vigorous horses, four or five years old, on stimulating feeding. Here hepatic derangements and poisons, over-exertion, perspiration and succeeding chills are especially to be suspected. Plunging the limbs in ice cold water as in wading a river (Fromage), standing in snow and above all in salted snow, or facing a cold rain, sleet, or snow when perspiring or fatigued, are recognized causes. A full drink of ice cold water when freely perspiring, and followed by standing in the frosty air, or in a cold current indoors. Exposure unblanketed after clipping in winter (Field,

Trasbot), and even sponging the body or legs with cold water when heated or fatigued or both. St. Cyr found that pneumonias stood to pleurisies as 3 : 1, Trashbot as 10 : 1, yet the latter draws attention to the fact that in cavalry horses habituated to the stable and sent out into camps in the depth of winter, the pleurisies are more numerous than pneumonias. This may suffice to show the importance of the rôle filled by cold and chill in the production of pleurisy. Yet many physicians look upon the chill as a predisposition only, while the true origin of disease is microbian. And in man a large proportion of pleurisies appear to be distinctly tuberculous. Bowditch traced 90 cases of acute pleurisy and found that 32 had tuberculosis. The objection to generalizing too largely on this for the lower animals is that the horse and dog, in which tuberculosis is rare, are by far the most common subjects of pleurisy, whilst cows which are very prone to tuberculosis show few cases of simple pleurisy. Again we find pleurisy in the horse as the result of other diseases localized in or adjacent to the pleura, and where there is nothing to indicate tuberculosis. Thus it follows pneumonia approaching the surface of the lung, cancers, actinomycosis and other tumors, and traumas—a pulmonary abscess bursting into the pleura, a broken rib scratching and lacerating the lung, a perforating wound of the intercostal space, or in cattle a sharp pointed body advancing from the reticulum toward the heart.

But the presumptive absence of the tubercle bacillus in the great majority of pleurisies in the horse does not prove the absence of all pathogenic microbes. Trasbot, who rejects the microbian theory, found that the injection of a little of the exudate into the pleural cavity of a sound horse, always determined a generalized pleurisy. Injections of distilled water with the same antiseptic precautions, made separately by himself and Laborde, had no pathogenic effect. Trasbot attributes the pleurisy vaguely, to the irritant effect of the exudate, but if it should finally be shown that this exudate contains microbes, though they may not be those of tuberculosis, the irritant action will be much more clearly explained. There are forms of pleurisy which are unquestionably the result of microbes, as in lung plague, influenza, canine distemper, glanders, tuberculosis, pneumo-enteritis, actinomycosis, and theoretically it might be supposed that in our ordinary acute

pleurisies, other germs that have been lurking harmless in the system may take occasion by reason of the lowered vitality induced by a chill, or a trauma, to colonize the thoracic serosa and develop pleurisy. Under such a theory, the predisposing and microbian element would remain equally effectual, but only operative when conjoined, neither being pathogenic without the other.

Until the constancy of the microbian factor is demonstrated we must recognize the time honored doctrine, that pleurisy may be due to cold, exposure, over exertions, to traumatic injuries, blows, concussions, fractures, penetrating wounds, and to extension by contiguity from adjacent diseases.

Most commonly pleurisy is unilateral on the right side but is often on the left or on both stdes.

Symptoms. There is the early symptom of shivering followed by a hot stage in which the limbs participate and partial sweats bedew the surface. There are first uneasy movements of the fore limbs with some lifting of the flanks and this discomfort increases until the patient is panting with pain and occasionally glancing round at his heaving flanks and even pawing as in colic. If the pleurisy is confined to one side the corresponding fore limb is often advanced before the other. The temperature is 102° and upward. The pulse is quick, hard and incompressible being usually compared to a jarred wire and beats from 48 to over 60 per minute. The breathing is highly characteristic. It is hurried, is carried on chiefly by the abdominal muscles to avoid the rubbing of the inflamed pleuræ on each other, and has the inspiration short and suddenly checked by an audible closure of the glottis while the expiration is slow and prolonged. This character of the breathing is well observed when the ear is placed against the false nostril. The laboring abdominal muscles stand out as a ridge from the outer angle of the ilium along the lower ends of the last ribs (pleuritic ridge). A tremor on this line is often noticeable in the early stages. It may also be felt by the hand laid on the costal region. The horse does not stand obstinately still as in pneumonia, but frequently moves as if seeking an easier posture. The short, hacking cough contrasts with the deep, rare cough of pneumonia. The expired air is not so hot, nor the mucous membrane of the nose so red as in the last named disease and there is no nasal discharge. A twitching of the muscles of

the chest is sometimes seen and if the intercostal muscles are pressed upon, the animal winces and frequently grunts. This last symptom is likewise seen in rheumatic disease of the intercostal muscles (pleurodynia) but the absence of the fever, the cough, and other chest symptoms sufficiently distinguish this. Auscultation detects in the early stages in addition to a healthy respiratory murmur, a friction sound audible in inspiration only in short jerks near the close of the act and comparable to the rubbing of the palm of one hand over the other laid over the ear, but this is no longer heard when effusion of liquid has taken place into the pleuræ. Percussion in the early stages detects no change from the healthy chest resonance.

If not relieved in from twenty-four to thirty-six hours, a remarkable modification of the symptoms takes place indicating the occurrence of effusion. The violent symptoms are suddenly relieved. The quick catching breathing which is in many cases accompanied by a grunt, becomes easy and though fuller than natural is comparatively regular. In particular the inspiration is free and full and comparatively painless, the sudden check and the grunt by which it was arrested having alike disappeared. The tension of the abdominal muscles and the tucked up appearance of the flanks give way ; the pulse acquires a softer character, the haggard pinched countenance is relaxed, and a general appearance of comfort and even liveliness prevades the animal. This temporary improvement is often so great that the horse will take to feeding as if he had all at once recovered.

The apparent recovery is, however, only transient. Soon the pulse becomes more frequent and loses its fulness, the breathing is more laborious and attended with a characteristic lifting of the flanks and loins, the nostrils are widely dilated, the limbs outstretched and the elbows outturned, the eyes stare and project and the countenance has a haggard appearance indicating threatened suffocation. Partial sweats may break out on the surface, due to the state of nervous excitement and general relaxation and supplementing in some degree the impaired exhalation from the lungs. Auscultation over the lower region of the chest shows a complete absence of the respiratory murmur, rising to the same level precisely at all points. Percussion elicits no resonance on the same region. If the effusion has taken place slowly or existed

for some time, the dulness and absence of sound will usually indicate that the liquid rises to the same level on both sides. So thin and permeable is the posterior mediastinum in its lower part that unless thickly coated by new solid exudations, the effusion readily passes through it and rises to the same height on both sides. If gas as well as liquid is produced in the pleural sac a gurgling or splashing sound may be heard on auscultation, and occasionally, after rising or other change of position, a *metallic tinkling*, due to droppings from the shreds of false membranes above into the fluid below.

As the disease proceeds dropsical effusions are observed beneath the skin of the breast and abdomen, a mucous rattle is heard in the trachea, the nose, ears and limbs become cold, the pulse increases in rapidity and weakness, shows the distinct anæmic tremor or thrill, and becomes rapidly imperceptible; the horse moves unsteadily and often falls suddenly dead.

This early fatality is, however, only seen in the worst cases. In those about to terminate favorably improvement is shown usually about the fourth day. The lifting of the flanks and loins becomes moderated, the ribs move more freely, the grunt ceases, the pulse is fuller, softer and less frequent, and auscultation and percussion show a steady decrease in the effusion. Appetite meanwhile returns, the horse moves more freely, lies down for a length of time in succession, and convalescence lasts from two to three weeks.

In the less fortunate cases structural changes more or less permanent, keep up symptoms of illness for a variable length of time. Sometimes after the liquid effusion has been absorbed the lung remains attached to the side of the chest by newly formed tissue (false membrane) and while this is undergoing a drying and organizing process, it gives rise to a leathery, creaking sound heard on auscultation and easily mistaken for crepitation. Sometimes an abscess forms on the surface of the pleura or in the newly organized false membrane, and either bursts into the pleural sac (empyema) where it serves to increase and sustain the irritation, or it makes its way through the intercostal spaces and is discharged externally. In this last case its advance toward the surface is heralded by an extensive inflammatory infiltration and pasty swelling much more tender to the touch than the dropsical swell-

ing already referred to. Another condition is that in which false membranes of considerable thickness invest a lung and, following the law of all fibrous structures in process of organization, they contract and cause a compression and partial collapse of the contained lung tissue. A flattening of the corresponding side of the chest and a muffled and almost inaudible respiratory murmur is the result of this condition. In some measure these symptoms are present during convalescence in all cases of pleurisy since the lung never expands to its full size till some time after apparent recovery, but it is only when the organ is invested with false membrane that the symptoms are very apparent.

In all such cases of prolonged pleurisy from protracted structural change there is continued illness without the violent symptoms by which the acute form of the disease is manifested. The acute suffering, the restlessness, the grunt, and even the catching breathing may be absent ; the temperature may be almost reduced to the healthy standard, the pulse small and tolerably soft, the appetite considerably improved and the different secretions tolerably normal ; yet the pinching of the intercostal spaces causes sharp pain, and measurement, auscultation and percussion testify to the persistence of disease. The animal is hidebound, unthrifty and unequal to any exertion. The cough is weak and painful and sometimes accompanied by a grunt.

Besides the changes connected with exudation and effusion, and organization or suppuration in the exuded products, *gangrene* sometimes results. A case of this kind is related by *Percivall.*

The *duration* of pleurisy may thus extend from two days in very acute cases to several weeks, or even months if we estimate it by the continuance of *hydro-thorax* in the chronic cases.

Post Mortem Appearances. These consist mainly in the presence of false membranes lining the pleura and hanging in cobweb like shreds into the cavity of the chest, and of the liquid effusion which fills up the chest at its most dependent part. The pericardium also contains fluid in many cases. The periods at which exudation takes place, and when the principal changes take place in the exuded materials have been well investigated by Dupuy, Hamont, Delafond and St. Cyr. They induced pleurisy by injecting irritant liquids into the chest, and noted the regular sequence of changes.

Dupuy injected two drachms of oxalic acid dissolved in three ounces of water. Symptoms of pleurisy at once came on, with the friction sound characteristic of its early stages. Next day friction sound had ceased and evidence of effusion existed. The same experiment repeated on several horses showed that if killed at any period subsequently to this, considerable exudation had already taken place. In one horse in which the disease was of 50 hours' standing the chest contained 43 pints of citrine-colored fluid, and abundance of yellow, thick, false membrane enveloping the costal and pulmonary pleuræ.

Hamont injected seven ounces of a weak solution of tartaric acid into the left pleural sac, repeated the injection next morning and destroyed the horse twenty minutes afterward. The chest opened immediately showed a small amount of liquid on the affected side, and the pleura injected and reddened.

Delafond made twenty-two experiments with the same general result.

Percivall found recent adhesions between the lungs and side so early as seventeen hours after the commencement of the pleurisy.

Andral injected rabbits with acetic acid and in nineteen hours found in the injected pleura soft, thin, false membranes traversed by red anastomosing lines, and in certain cases a serous or puriform fluid.

W. Williams found a false membrane formed twenty-fours after the injection of the irritant.

St. Cyr in a series of 43 experimental and casual pleurisies in horses, found that in a very few hours there was marked local congestion and swelling of the pleura speedily followed by the formation of soft, pulpy, friable false membranes, largely amorphous and granular but impregnated with many cells and nuclei. These adhere feebly to the pleura but may accumulate with prodigious rapidity so as to cover in three or four days the whole pleural surface on one or both sides. The attendant serous effusion was bloody, turbid, or lactescent. The pleural surface under the false membrane was highly vascular and studded with fragile, red conical elevations projecting into the membrane. Exceptionally the sub-serous connective tissue became the seat of exudation as well.

From the sixth to the ninth day the false membranes began to

become vascular and from the tenth to the fourteenth day commenced to organize into the connective tissue. With the advent of this stage, the inflammatory action tended to subside, and the reabsorption and repair to ensue.

Pleuritic effusion. This varies greatly at the different stages of the disease. As effused it has a composition resembling that of the blood :—

Water, - - - - - - - - - - - 911 to 924
Albumen, - - - - - - - - - - 63.33 to 82.50
Fibrine formers, - - - - - - - 2.16 to 12.50
Extractive matter.
Salts.

The progressive changes from the hæmorrhagic effusion to the limpid hydrothorax and their relation to the different stages of the disease and the subsidence of the inflammation are of the greatest importance in deciding questions of responsibility, when the animal has recently changed hands. St. Cyr has classified his cases in the following instructive table :

Duration of the Disease.	Effusions.				
	Port Wine	Sero-sanguineous.	Muddy or Grayish.	Limpid.	Total.
From 1st to 7th day.	9	6	3		18
" 8th to 15th day.	2	3	4	6	15
" 16th to 30th day.		1	1	5	7
After 30th day . . .				3	3
	11	10	8	14	43

Up to the 7th day 50 per cent were dark red ; after the 7th day only 13.3 per cent ; and after the 15th day none. Up to the 7th day 83.3 per cent were either dark red or sero-sanguineous and not one had attained to translucency. After the 7th day only 8 per cent were of port wine hue, and by the 15th day 24 per cent. of all cases of over seven days standing were already transparent. Of all cases of over 15 days standing, 80 per cent. were perfectly translucent and none showed the dark red hue. Finally after the 30th day all remaining cases were limpid. This of course must not be applied with the same confidence in both directions. While translucency of the effusion bespeaks seven days standing and

probably fifteen or twenty, the dark red hue must not be held to imply a recent date for the attack. A relapse in the course of convalescence may easily and quickly stain anew a liquid that was already limpid, or had advanced far toward this condition.

The appearance of the lung tissue in a case of confirmed pleurisy is characteristic. The lung is of a dull red color, shrunken, slightly collapsed, flabby, scarcely crepitant under pressure and heavier than water or floating in water. It is tough, not friable like hepatized lung, and its cut surface is dry, smooth, and presents the interlobular septa very well marked. This is due to the compression by effused liquid, and by the organizing and contracting false membranes covering the lung and implies nothing more than simple condensation. The air cell may be collapsed, but contains no new product and has not parted with its epithelium and the lung can be inflated through the bronchia.

Differentiation according to the nature of the effusion. Pathologists have divided acute pleurisy into the *dry*, *sero-fibrinous*, and *sero-fibro-purulent*.

1. **Dry or fibrinous pleurisy** has usually a more acute type and the exudate containing an excess of the fibrinogenous elements forms a coagulum or false membrane on the affected surface tending to bind that to the part adjacent—the lungs to the costal pleura. The serum, small in quantity, is in the main retained in the exudate or if set free is actively reabsorbed by the healthy pleura.

2. **Sero-fibrinous pleurisy.** This form is usually less acute and more extended involving perhaps an entire pleural sac, or even both sides of the chest. This is the common form of pleurisy and is that referred to in the experiments of St. Cyr and others above. The earliest lesions in experimental cases (with chloride of zinc solution) in dogs are an uniform bright red congestion, with a bright, shining surface as yet perfectly dry. There is already shedding of patches of the endothelial cells, swelling and proliferation of the superficial connective tissue cells and the formation of a few pus globules. This is seen in from half an hour to six hours after the application of the irritant.

Next follows the exudation of fibrine and serum, which respectively coagulate as false membrane on the inflamed membrane, or drop to the bottom of the sac as liquid. The fibrine appears as

granules, little knobs and threads between and on the endothelial cells and entangling a few pus cells. The changes are now much more marked in the connective tissue cells, which are more numerous, larger, nucleated and often stellate or polygonal. Changes are well advanced in twenty-four hours. The cells go on increasing to the fourth or fifth day, when new blood vessels are formed into the membrane and may be injected from the pleura. From this time, in favorable cases absorption of the liquid proceeds, and the fibrine is organized, and by the fourteenth day is transformed into connective tissue, the superficial cells forming endothelium and the deeper, branching connective tissue cells. The result is the thickening of the pleura and the formation of adhesions. The case, however, may prove fatal, or it may be protracted through the continued production of fibrine and serum, or it may pass into empyema.

3. **Sero-fibrino-purulent pleurisy. Empyema.** This is usually very dangerous as well as complicated. It may supervene on the last described form. It may depend on rupture into the pleura of abscess of the lung, bronchial glands, liver, diaphragm or intercostal space and the infection of the chest cavity. It may in the same way follow the laceration of a bronchium by a broken rib, the perforation of the intercostal space by a foreign body, or (in cattle) the penetration of the chest by a sharp-pointed body from the reticulum. It may follow at once on pleurisy of a very high grade. Probably in all such cases there is infection of the pleura by pus microbes. When there is a communication with a bronchium, the reticulum or the external air there are usually septic germs in addition, and the contents of the chest become fœtid.

The purulent fluid may accumulate in the lower part of the pleural sac, or it may be confined in abscess form in the false membrane, and extend thence into surrounding tissues. The pus-containing pleura, or cavity infected by the pus germs, assumes the appearance of a granulating surface, or of the lining membrane of an abscess, and continues to produce pus in greater or less amount.

The formation of pus in the pleura is known as *empyema*. When air enters the pleura through a wound perforating the chest wall, or when gas is formed in the pleura, the condition is **pneu-**

mothorax. As liquid is usually present as well it is hydro-pneumothorax.

Tubercular and other forms of pleurisy have in certain cases been superadded to the specific local lesions, by which such diseases are individually characterized.

Prognosis. Occurring in an otherwise healthy system and especially if confined to one side of the chest, pleurisy is not frequently fatal, and under appropriate treatment recovery is oftentimes rapid and satisfactory. A certain number of cases merge into chronic hydrothorax, the inflammation apparently subsiding, but reabsorption failing to take place. The hydrothorax may last for months or even a year.

Treatment. If seen during the *chill* and before inflammation has been definitely established every effort must be directed to secure its abortion, if possible. No time should be lost in placing the patient in a warm comfortable stall or box, covering him with woolen blankets and actively rubbing and loosely flannel bandaging the legs. Warm drinks and warm injections must be given. Half an ounce or an ounce of camomile or boneset in infusion in two or three quarts of hot water, or in the absence of this any of the carminatives, or etherial, alcoholic or ammoniacal stimulants may be given. Pilocarpin in 7 grain dose hypodermically may promptly secure a revulsion of blood to the skin and at once overcome the chill and prove a most effective derivative from the pleura. Placing the legs in buckets of hot water, or the whole animal in a hot air bath will often act equally well. Packing the chest and even the abdomen in a blanket wrung out of very hot water and covering it closely by one or two dry ones, or, better still, by a rubber or other impermeable covering, will long retain both heat and moisture, securing free cutaneous circulation, and soothing in a most effective way the irritation in the chest. This may be maintained as long as requisite to relieve the patient, and then the body may be uncovered, a part at a time, rubbed dry and covered with a dry woolen blanket. By using elastic circingles over the compress they are adapted to the respiratory movements and any restriction in the movement of the ribs is beneficial by limiting the friction, pain and irritation in the pleura.

In the second stage, when inflammation has already set in, the same general measures of derivation toward the skin and hot bath

or soothing derivative compress are still demanded though they may be substituted by more stimulating derivatives. The bleeding of the patient into his own vessels is sought in various ways. On the continent of Europe stimulating embrocations (essential oils, ammonia and oil, mustard, etc.) are applied to the limbs. In America and England similar agents are more commonly applied to the walls of the chest and dry cupping in the same region has been resorted to. Metallic cups with small mouths and having a capacity of about a pint each, have the air rarefied by plunging into each a spirit lamp, and, on its withdrawal, suddenly applying the mouth of the cup on the skin of the costal region previously well coated with lard. Another form of cup is made with a tube and stopcock in its otherwise blind end by means of which it is exhausted with a syringe after its mouth has been applied to the skin. In the absence of both a narrow mouthed glass tumbler may be employed, the air is rarified by inserting into it a burning spill of paper or wood for a few seconds and on its withdrawal the cup is instantly inverted on the skin. If the animal is very hairy or very thin it may be necessary to shave the part, and smear with oil and even to select a very narrow mouthed cup. When applied the cup is cooled with water or otherwise and owing to the partial vacuum the skin is strongly drawn up into it and the blood accumulates in and under the skin. It may be kept on for half an hour at a time and with ten or twelve cups on one side the patient tends to profuse perspiration establishing a strong revulsion toward the skin, and great relief. In dangerous cases three or four applications may be required in twenty-four hours.

Next to this the mustard application is perhaps the safest and most valuable. The best ground mustard (black by preference) is made into a very thin pulp with tepid or cold (never hot nor boiling) water and rubbed in against the hair so as to soak the surface of the skin; it is then closely covered with paper and with a rubber or other impervious covering or, in default of better, with a close blanket and left on for two hours. By this time the skin should be thickened to the extent of at least a quarter of an inch and the derivation and relief will be very manifest.

Cantharides is sometimes used but like most other severe irritants, is liable to induce sympathetic irritation in an already severely inflamed pleura, and thus to obviate all benefit. Cantharides is also liable through extensive absorption to irritate the kid-

neys. To counteract this Bouley gave ½ drachm doses of camphor with alleged good effect.

Some practitioners make local applications of hot water and of aqua ammonia (confined) but unless very closely watched these are liable to destroy the hair follicles and produce permanent blemish.

The hot air, steam bath, and hot compress have the advantage over the mere irritant derivatives that their action is from first to last soothing and free from all risk of inducing sympathetic irritation and yet as derivatives they are eminently efficient. Next to them in safety and efficacy comes dry cupping.

The irritant derivatives are often the most valuable, but must be used with great judgment. They are always dangerous when the pleural inflammation runs very high and when the local irritation and suffering are specially acute. Under such circumstances it is usually desirable to adopt other measures to moderate the severity of the inflammation, and to fall back on baths, compresses and cups until the irritation is alleviated before vegetable or animal vesicants are resorted to. In acute and severe attacks these latter are especially applicable to the early stages before the inflammation has been fully formed, or after the stage of free effusion has set in.

With high fever and no benefit from hot local applications, cold irrigation or refrigerant compresses to the walls of the chest, have proved useful, but considering the rôle filled by cold in causation and the suggested relation between pleurisy and rheumatism this is not to be followed as a general practice.

If the patient has been a hearty feeder and if there is evident costiveness a purgative (aloes or sulphate of soda) is often desirable at the outset, but if the disease is of a low type this is always dangerous, owing to susceptibility of the intestinal mucosa and it is safer to correct constipation by injections or at most by a pint of olive oil.

When the suffering is very acute and is aggravating the fever, a hypodermic injection of morphine will often greatly relieve and even favor a revulsion of blood toward the skin, but as it tends to suppress the action of both bowels and kidneys it should be avoided unless it seems absolutely necessary, and above all it should not be given by the stomach. Cocaine hypodermically may be used to relieve pain.

Both fever and suffering can sometimes be greatly relieved by large doses (2 drachms 3 or 4 times daily) of salicylate of soda, which again suggests a close relation of the disease to rheumatism. Acetanilid or phenacetin may be used to fill the same indication.

Next come the questions of alkaline and diuretic treatment. Some cases do well if given nitrate of potash freely in the drinking water. Some prefer the alkaline diuretics, such as acetate of potash or ammonia, bicarbonate of potash or soda, biborate of soda, or the vegetable diuretics such as colchicum, squills, etc. Fraënkel found that, while comparatively ineffective alone, these proved most efficient (in man) when combined with cinchona or other bitter. The hint should be useful to the veterinarian. Diuretics in the stage of effusion should be pushed as far as the strength of the patient will warrant.

Friedberger and Fröhner recommend pilocarpin, and no agent produces an equal secretion from the natural emunctories and an equal tendency to reabsorption. It is however so profoundly exhausting that it must be used with the greatest judgment and caution.

Digitalis has often an excellent effect. Though not primarily a diuretic, it is a powerful tonic of the heart and circulation, and by increasing the blood tension it usually produces a free flow of urine. In combination with the diuretic salts it may be used from the first but it is especially valuable, after effusion and when attention must be given mainly to securing reabsorption. Care is demanded that we avoid its cumulative action, and in place of continous large doses, a strong infusion applied over the loins will sometimes have a good effect. It may also be combined with bitters and even with ferruginous tonics in the advanced stages.

In combination with neutral salts and digitalis, iodide of potassium would seem to be indicated. Results however do not show a great superiority to other diuretics in favoring absorption.

Tincture of iodine, painted upon the chest over the affected parts, and repeated until tender, acts more or less as both a derivative and deobstruent. A liniment of iodide of potassium and soap is a convenient form of application.

The inunction of the chest walls with mercurial ointment has strong advocates both among physicians and veterinarians, and is combined in such cases with the exhibition of calomel in-

ternally. Unless the good effects are shown in a day or two it may well be abandoned.

When effusion becomes dangerous through excess, and in advanced cases when it fails to yield to medicinal measures thoracentesis is called for. (See under *hydrothorax*.)

PLEURISY IN CATTLE,

Milch cows and work oxen most liable. Causes. Damp buildings and locations, sudden transitions from heat to cold, exposure when fatigued, etc. Symptoms, rigor, reaction, cold horns and limbs, later hot, excited pulse, catching breathing, hyperthermia, 104° to 105°, tender chine and intercostals, friction sound, later dulness, creaking, weaker murmur, subacute cases often tuberculous, effusion unilateral, chronic cases. Lesions, as in horse with superficial marbling of lung. Treatment, laxative, warm drink, compresses, derivatives, sedatives, diuretics, heart tonics, diuretics, thoracentesis.

This is not common in young growing cattle, but is more frequent in milch cows and work oxen. It is due to the same causes as in the horse, and especially to chills when heated, damp buildings and locations, cold draughts between open windows or doors, and cold storms. The greatest danger comes from hot, close stables, like many distillery stables, approximating to the temperature of the animal body and from which the stock are suddenly turned out of doors, or shipped by car or boat with a temperature near zero, and above all if furnished ice water to drink. Such animals taking no exercise to increase the circulation and heat, are especially liable to shiver and contract illness. Rigors too are easily induced in animals standing in hot buildings, when, in connection with the cleaning, an adjacent door is thrown wide open or two on opposite sides of the house. Working oxen heated with exercise and then exposed to extreme cold and compulsory inaction are endangered.

Symptoms. The attack is manifested by the same general symptoms as in the horse. The rigors are often very well marked, especially over the shoulder; the tenderness of the chine and intercostal spaces is striking; the breathing is catching but there

is rarely the same restlessness as in the horse; the bowels are costive, appetite and rumination impaired or suspended, and the paunch is often distended with gas. The tenderness of the intercostal spaces, the friction sound of the pleura, and the maintenance of the respiratory murmur and the normal resonance of the lung, become the ultimate diagnostic symptoms. The pulse may be 70° and upward, the temperature above 104° to 105°. In some insidious cases indeed the fever is very slight and besides the general wasting of the animal, the indications obtained by physical examination alone enabled us to recognize the malady. Tuberculous pleurisy which is very common in cattle is to be suspected in such cases.

Effusion is recognized by the dulness of the lower part of the chest up to a certain line, and often unilateral, by the softer pulse, by the dilated nostrils, or open mouth, the contracted facial muscles, by the glazed eye, and anxious expression, by oppressed breathing and often by engorgement under the chest and in the limbs.

When the disease lasts over ten or twelve days it tends to pass into the chronic form. Or a chronic pleurisy of a subacute type may begin *de novo* and pursue an insidious and latent course.

If the disease commences as a subacute affection there may have been for a month, capricious appetite, general illhealth and falling away before any other symptom is noticed. Now the breathing is manifestly excited, a small, short cough is heard at intervals, the pulse is accelerated but weak, and pinching auscultation and percussion detect unequivocal signs of pleurisy. From this the symptoms become more decided though for a length of time they are very slight, the animal meanwhile becomes increasingly emaciated, and perishes ultimately in a state of great weakness. Such insidious cases are always to be suspected of tuberculosis.

The *post mortem appearances* resemble those of the horse. The surface of the lung beneath the diseased portions of pleura, however, often presents a marbled appearance from the infiltration of the areolar tissue between the adjacent pulmonary lobules. The organization of the false membranes begins on an average about the tenth day.

Treatment. The same general principles must be followed as

in the horse. Bleeding can rarely be employed, partly because the disease so often assumes a subacute form, and partly because when first seen considerable effusion has often already taken place and severe depletive measures are thereby contraindicated.

A laxative dose (1 lb.) of sulphate magnesia, may be given in warm gruel, and the same means by compresses, hot fomentations and counterirritation adopted, and the same sedative and diuretic medicines given as in the horse. In the advanced stages and in the low types of the disease the stimulating diuretics (sweet spirits of nitre, and liquor of the acetate of ammonia) and vegetable and mineral tonics are especially indicated. The diet in these last types must be nutritive, laxative and easily digested.

Tapping of the chest is equally applicable as in the horse, (*see Hydrothorax.*)

In the chronic forms everything is to be done to support the general health whether by food stimulants or tonics, and counter-irritants may be applied several times.

PLEURISY IN SHEEP.

Causes, exposure, after clipping, washing in cold weather, alternations from hot buildings to cold fields, shedding of the wool. Symptoms, hyperthermia, troubled breathing and pulse with catching inspiration, tender intercostals, friction sound, and signs of effusion. Treatment, preventive, shelter, febrifuges in food or water, aqua ammonia to sides.

The causes of pleurisy in sheep may be largely included in the general statement—exposure. Cold washing and exposure after clipping is especially injurious. Devieusart saw 300 cases of pleurisy and thirty deaths in a flock of sheep shorn in February. If kept secluded in warm buildings sheep may be shorn in midwinter, but any reckless exposure, and any sudden reduction of the temperature of the building is liable to be disastrous. *Scab* and other skin affections which lead to a shedding of the wool in inclement weather may also be the occasion of widespread attacks. Otherwise the causes are essentially those of the same disease in the larger animals.

The *symptoms* resemble those of pneumonia, but with the peculiar sharp, short arrest of the inspiration, and the marked tenderness of the intercostal spaces as above described. The cough is short, dry, hacking and infrequent or suppressed as much as possible. Auscultation and percussion signs, corresponding to those found in other animals, are easily got in the newly shorn sheep. In the unshorn the wool must be parted and a stethescope employed.

The *treatment* is mainly *preventive*, or when the disease is present, of a general nature applicable to flocks. A warm barn, with pure air, blanketing, wet compresses, to which may be added extract of henbane, and nitrate of potash in the drinking water give examples of general medication. As a derivative, aqua ammonia and oil may be applied in lines on the chest exposed by parting the wool or generally on the shorn. Where the patient can receive the requisite attention further treatment should be on lines laid down for cattle.

DOG. PLEURISY.

Causes, exposure to cold, etc. Chill, reaction, disturbed breathing, catching inspiration, rapid, hard pulse, hyperthermia, tender chest, friction sound, later dullness at lower part of the chest in any position. Treatment, as in pneumonia, with antirheumatics and diuretics freely. Thoracentesis.

This is occasionally seen in the dog as the result of exposure, and like other diseases of the chest is easily recognized. It owns the same causes with pneumonia.

Symptoms. There is first dulness, shivering and some excitement of respiration. To this follow the more acute symptoms, the hard pulse, the rapid, catching breathing, the animal standing or sitting on his haunches, the open mouth, pendent tongue, the injected mucous membrane, the costiveness, but above all the tenderness of the intercostal spaces, the early friction sound on auscultation, the pain and normal resonance on percussion, the muscular twitchings and the short, suppressed, painful cough. When effusion has occurred its amount may easily be estimated by turning the animal alternately on its feet, back and haunches, and observing how high the dullness extends in these various positions.

The same *treatment* may be adopted as in *pneumonia*, with this difference that salicin may be given freely, and when effusion has taken place active diuretics are specially indicated, and hence tincture of squils (a teaspoonful) may be made to replace the nitre. In advanced and obstinate cases, or where danger exists from rapid effusion, the liquid may be drawn off with a cannula and trochar as in other animals.

PLEURO-PNEUMONIA. BRONCHO-PNEUMONIA. BRONCHO-PLEURO-PNEUMONIA.

Though we often meet with typical forms of *bronchitis*, *pneumonia* and *pleurisy*, it is much more common to find them combined more or less with each other. Thus combined inflammation of the bronchial tubes and pulmonary substance is frequent; inflammation affecting both the lung and its investing pleural membrane is no less common; and cases are seen in which all three structures are involved. These conditions are to be recognized by the presence of the symptoms of both the coexisting maladies but particularly by the indications furnished by touch, auscultation and percussion. The predominance of one disease over another will decide the nature of the treatment which must be adapted to the peculiar character of each case whether *mainly bronchitic*, *pneumonic*, or *pleuritic*. It is these mixed cases that test the ability and judgment of the practitioner as he must carefully individualize each case, ascertain the different parts affected, the grade of the inflammatory action, the nature of the attendant fever, the presence or absence of epizootic influence, etc., and having all these conditions in view must apply remedial measures accordingly.

It must be evident that particular directions cannot be supplied for all of these cases. General principles only can be inculcated and their adaptation to the varied phases of different cases left to the judgment of the student.

HYDROTHORAX.

All animals liable. Causes, pleurisy, obstruction to pulmonary or intercostal veins, heart disease, Bright's disease, anæmia, parasitic or otherwise. Effusion reddish gray or clear straw color, inflammatory and dropsical. Symptoms, troubles of respiration, as a secondary disease complicated by dropsies elsewhere, signs of hydrothorax without fever, shedding of hair. Treatment, diuretic, tonic, derivative, thoracentesis, trochar and cannula or aspiration, point of election for puncture, method, asepsis, drainage by aspirator, or into an antiseptic solution, eligible cases, dangers attending thoracentesis, shock, rupture of false membranes and lung, infection of pleura, injection of antiseptics.

Hydrothorax or *water in the chest* is common to all domestic animals. It is as we have seen one of the most ordinary results of *pleurisy*, and may persist long after that disease has disappeared. It likewise occurs independently of inflammation as a dropsical effusion. Thus when the return of blood by the bronchial, pulmonary or intercostal viens, is hindered by any cause such as tumors in the bronchial glands or subvertebral region a passive effusion may take place through the coats of the vessels. In imperfection of the mitral valves the regurgitation of blood in the pulmonary veins during each cardiac systole equally causes such transudation. Chronic disease of the kidneys (Bright's disease) with the retention of effete and injurious materials in the blood leads to dropsy of the chest as in other parts of the body. Again in many debilitated conditions parasitic and otherwise, with a tendency to general dropsy the chest participates and a collection of fluid takes place in the pleuræ.

The nature of the contained fluid will vary according to the conditions in which it has been effused. If the result of inflammation there are the different stages already indicated : *first*, of a yellow citrine color or red from contained blood ; *second*, grayish and muddy either from contained pus or other changes taking place in the fluid ; and *third*, clear limpid and translucent as seen in the later stages. If merely a dropsical effusion the fluid is watery clear and transluent or with a slight straw color. The inflammatory effusion contains fibrine or fibrinogenous elements, is associated with the formation of false membranes, and though it

may remain fluid so long as it is retained in the chest, it coagulates rapidly when withdrawn. The dropsical effusion rarely contains fibrine, and then only in very small amount, and it does not coagulate when drawn off from the chest. The inflammatory effusion usually contains a greater proportion of common salt, phosphates or albumen than exist in the blood, and floating granules, particles and cell forms, none of which conditions characterize the dropsical effusions. The most prominent feature of the inflammatory effusions is thus seen to be their power of coagulation, by virtue of the contained fibrine, when exposed to the air.

Symptoms. When a sequel of pleurisy it is manifested by the symptoms already mentioned under that head as indicating the occurrence of effusion.

The dropsical cases may come on rapidly and present all the signs of troubled respiration together with the results of auscultation and percussion that characterize rapid inflammatory effusion but without the fever and acute symptoms of pleurisy. More usually it comes on insidiously, the lung accommodates itself to the gradual increase of the fluid and it is only when the accumulation has become excessive that the symptoms become prominent. In heart or kidney disease the filling of the legs and infiltrations of the eyelids and of the skin beneath the chest and abdomen are precursors or early concomitants of the disease, but in all cases the accumulation in the chest is to be measured by the height of the line of dulness on percussion and the extent of chest surface giving forth no respiratory murmur on auscultation. As the liquid rises on both sides of the chest, as it always does in such cases in the horse, the breathing becomes short and labored, being chiefly effected by the action of the diaphram and the flanks—the ribs moving only slightly. The nostrils are widely dilated with each breath. The previously existing want of vigor and energy, the weak pulse, the poor appetite and the pallor of the mucous membranes become aggravated ; the animal becomes very weak and prostrate, the loins insensible, the permanently tucked up flanks labor tumultuously, the loins rise in inspiration, the face is pinched and haggard, the eyeballs glazed and protruding, and death is preceded by the same general symptoms as in rapid effusion after pleurisy. A prominent feature of this, as of all dropsi-

cal affections, and one usually seen in the hydrothorax of inflammation as well, is the ease with which, even at an early stage of the disease, the long hairs of the mane and tail may be pulled out. In many cases they come out in handfulls when the comb or the fingers are passed through them.

Treatment. The treatment must be of the actively diuretic kind recommended for the effusion of pleurisy. It is modified however in one respect. The inflammatory action having subsided or nearly so and the condition being now essentially one of weakness a free use of tonics is demanded. Many a patient dies in such circumstances from the actively depletive treatment to which it has been subjected and the want of attention to its need of generous diet and other support. The agents prescribed for the advanced stage of pleurisy may be given, or the digitalis or other diuretics and bitters may be combined with iodide of potassium in one or two drachm doses, the amount being apportioned to the strength of the animal. Iron in the form of sulphate, perchloride or iodide may be freely given combined with gentian, quassia, or other vegetable tonic, and above all a liberal and easily digested diet must be allowed. Good will sometimes result from repeated applications of strong iodine ointment to the sides with active friction.

When the condition is dependent on disease of the heart, kidney or other organ, these must be attended to according to their special requirements.

Disconnected from such complications hydrothorax will often give way to an active treatment similar to that indicated above. In some cases however our only hope of even temporarily prolonging life lies in the operation for drawing off the fluid.

Tapping the chest or as it is technically called **thoracentesis** or **paracentesis thoracis** has proved sufficiently successful in the lower animals to warrant its continuance in cases that resist other modes of treatment. It is highly probable that the larger proportion of unsuccessful cases is due in great part to the hopelessly advanced stage at which it is often had resort to, to the insufficient precautions adopted in its performance, and to the want of appropriate dietetic and medicinal treatment. Dr. Bowditch's treatment by *paracentesis* saved in the human subject at the rate of about two patients in five and we ought by availing of similar precautions to reach the same standard.

The cannula employed in veterinary practice is a silver tube two inches in length, a quarter of an inch in diameter and furnished with a shield of the same metal at one end. The trochar by which it is introduced is of steel or brass. To carry out Dr. Bowditch's system the operator must supply himself with a syringe of a somewhat smaller bore and an intermediate brass piece of a size adapted to fit accurately into the cannula and supplied with a stopcock. By an instrument of this kind the fluid can be drawn off by means of the syringe without any risk of the introduction of aerial germs which always tend to induce suppuration and even a putrefactive decomposition in the contained fluid.

The point selected to operate on is, in the horse, ox or dog, in front of the anterior border of the ninth rib, at its lower end or close to its union with the cartilage. The point of the trochar should be directed slightly upward and forward to avoid the possibility of injuring the diaphragm. The skin is first rendered aseptic by shaving, followed by a thorough soapy wash and a free use of mercuric chloride solution (1:500). It is then pricked with a lancet, then drawn aside that the wounds in the skin and muscles may not correspond after the cannula has been withdrawn. The trochar is then pushed steadily through the intercostal space till all obstruction has been overcome, when it may be concluded that the pleural sac has been reached. The trochar is now withdrawn and the fluid allowed to flow from the cannula until there is presumably some risk of the introduction of air, when the brass piece is to be applied and the remainder drawn off with the syringe or aspirator. As a substitute for the aspirator a caoutchouc tube, eighteen inches long, put on the cannula or needle and having its lower end plunged in a solution of boric acid will prevent the entrance of germs. A prob has often to be introduced to prevent plugging of the cannula by floating false membranes, and a new puncture in a different place may be necessary. In the case of excessive accumulation it is often advisable to draw it off at two operations, as recommended in large abscess of the pleura and for the same reasons. The need for such a precaution will be understood when it is stated that in bad cases the chest contains as much as six or seven ordinary stable bucketfuls of the liquid. If, however, it is limited in amount it may be all withdrawn at once.

The most successful cases in the horse have been upon young, vigorous animals, from four to eight years old, during the first month of illness, and where the pleurisy has been confined to one side.

Dr. Bowditch lays down the following rules for the adoption of paracentesis in man (*Clinical Medicine*, by Prof. W. T. Gairdner) :—

"I now never operate unless I find some distension or rounding out of the chest, and filling up of some of the intercostal spaces, so that the chest presents a uniform curve, and not alternate depressions and elevations as in the healthy chest. I operate under the following circumstances when I feel certain there is fluid :

"1. When there is *severe permanent dyspnœa*—orthopnœa—however acute the disease if I find fluid filling the pleural cavity, or nearly filling it.

"2. When there are occasional attacks of orthopnœa threatening death, even if there be not sufficient to fill more than half of the cavity. If the fluid seems to be the cause of the dyspnœa I operate, because occasionally I have lost a patient while waiting for more extensive physical signs. This rule I apply to acute and chronic cases.

"3. I use the trochar after three or four weeks of ineffectual treatment, without any absorption being produced.

"4. In chronic idiopathic hydrothorax, a latent pleurisy with simply physical signs to indicate *extensive* effusion, but when the rational signs are either very slight or none at all save a general malaise and weakness."

The use of iodide of potassium and vegetable and mineral tonics must be perseveringly employed and the strength further supported by a generous diet, to secure the animal against the dangers of extreme prostration, of suppuration, or other undesirable conditions of the exuded product.

Among the dangers attending thoracentesis are fainting as a result of shock on the sudden withdrawal of so much liquid, rupture of the false membranes, and even of the enclosed lung tissue or of blood vessels, under the sudden expansion of the partially collapsed lung confined by the investing false membrane, and the introduction of pus or septic germs into the pleural cavity. To obviate the first named dangers tight bands (circingles) around

the chest will give support and limit sudden expansion. In case of excess of liquid the withdrawal of one-half or two-thirds at a time will allow opportunity for accommodation. Hæmorrhage may be met by the internal use of chloride, sulphate or nitrate of iron, matico, hamamelis or tannic acid, and a weak solution of boric acid or other antiseptic agent may even be injected in small amount into the pleural cavity.

In obstinate and chronic cases the injection of a weak solution of iodine and iodide of potassium is often of service. In other cases a normal chloride of sodium solution (previously sterilized) may be introduced as soon as a partial evacuation causes uneasiness, and by a succession of such evacuations and injections the residuum liquid may be rendered clear and largely aseptic on a single occasion.

In the smaller animals the selection of the most dependent part for insertion of the trochar is not so essential, as the body may be turned to facilitate the drainage.

On completion of the operation the wound may be again treated antiseptically and coated with aristol or collodion.

PNEUMOTHORAX. AIR OR GAS IN THE PLEURA.

Causes, decomposition of liquid effusion, perforation from a bronchium, the stomach, a thoracic wound. Symptoms, metallic tinkling, splashing, succussion, drum-like resonance, suppressed respiratory murmur, distance of cough sound, distress, anxiety, dyspnœa, bulging intercostal spaces, sometimes a wound. Treatment, closure of wound, calmatives, aspiration of gas. Treatment for pleurisy.

The collection of air or gas in the cavity of the pleura has already been noticed as co-existing with liquid effusion in some cases of advanced pleurisy. It may arise from other causes, among which may be noted: 1. When a mass of pulmonary tubercle connected with a bronchial tube has opened into the pleural sac. 2. When a communication has been established between the pleural cavity and the alimentary canal, as in combined rupture of the stomach and diaphragm, or of the double colon and diaphragm. 3. When a rib is fractured and the broken end penetrates the lung tissue and opens into one or more small bronchial tubes. 4. When a wound has been inflicted penetrating the walls of the chest and forming a valvular orifice through which air is drawn inward during each inspiratory act, but out of which it cannot pass when the thorax collapses.

The amount of gas present may be extremely slight, or in a case such as that from a valvular wound it may cause complete collapse of the lung, filling up the entire half of the thorax and bulging into the opposite half.

The *symptoms* are often very obscure. If with liquid the metallic tinkling after rising, in small animals the splashing when shaken and the other sounds of auscultation and percussion will point it out as described under *pleurisy*. In the case of a broken rib the distortion, swelling and tenderness, will lead to suspicion. A penetrating sound will be sufficiently evident, and in the case of tubercle previous cough and ill-health will have been manifest.

The specific signs of uncomplicated pneumothorax are: 1st, A drum-like resonance on percussion over the seat of the gas, usually at the upper part of the chest; 2d, A partially suppressed or distant respiratory murmur over the same area; 3d, A muffled or suppressed sound of the cough; 4th, Sometimes, especially if the

gas is abundant, prominence of the chest on that side ; 5th, There are also more or less distress and anxiety, difficult breathing, quick, weak, rapid pulse, and other signs of illness.

Some cases of this kind recover spontaneously or with the liquid effusion with which they are associated ; in traumatic cases the wound is sometimes sealed up by a pleuritic exudation which here becomes a curative process ; while in some examples of valvular wound of the lung or walls of the chest death may ensue in a period varying from a few minutes and upwards to weeks.

Treatment is limited to the prevention of the ingress of air through an external wound where that exists ; the employment of opiates and other agents to moderate attendant suffering ; to measures calculated to moderate the intensity of resulting pleurisy, and, in cases where there is imminent danger from accumulation of gas, to the puncture of the chest and the careful withdrawal of the gas by aspiration. If necessary sterilized air may be made to replace the aspirated gas.

PYO-PNEUMOTHORAX, EMPYEMA.

Causes, septic cocci entering through wound or blood. Symptoms, those of hydrothorax, with prostration, fœtor, and it may be issue of pus. Treatment by antiseptic injections.

A purulent fluid in the pleural cavity may be found in ordinary pleurisy, but is much more likely to supervene in traumatic forms, in which the pus cocci reach the cavity through the wound of the bronchia, alimentary canal, or chest walls.

The symptoms are essentially those of pneumothorax, with greater prostration, and in certain cases a distinct feverish smell or fœtor of the breath, or the escape of pus through a wound. In treatment the difference from pneumothorax is mainly in the antiseptic character of the injections and the freer employment of stimulants and tonics. Salt, salicylic acid, borax, peroxide of hydrogen, aluminium acetate, or potassium permanganate solutions may be used. Tonics (quinia) and antiseptics (sulphites, salicylates, iron) may be given.

CHRONIC PLEURISY.

Animals liable. Causes, irritation through effusion and exudate acting on susceptible pleura, or by other disease products in lung or pleura. Unhygienic surroundings and management predisposes. Frequent chills in cold water. Symptoms. unthriftiness, easily blown, fatigued, or sweated, cough, paroxysmal under exertion, pallor of mucous membranes becoming congested on slight exertion, difficult breathing when recumbent, percussion and auscultation signs of pleurisy and hydrothorax. Lesions, great liquid effusion, clotting on exposure, with much albumen and cell-forms. False membranes partially organized. Treatment, tonic, diuretic, derivative, diet nourishing, counterirritants, paracentesis.

In all domestic animals acute pleurisy may merge into the chronic form, the irritation being maintained by the presence of the residuum liquid and the false membranes and adhesions which interfere with the free dilatation of the chest. The pleura too, having been once inflamed, retains an increased susceptibility to such disturbing conditions. In other cases the affection is symptomatic of other chronic affections, as tuberculosis, glanders, and neoplasms of various kinds. It has been seen especially in old, weak and debilitated subjects, kept in confined, impure stables or habitually exposed to undue cold and damp. Hence dairy cows in unhygienic conditions, and hunting dogs, which plunge in water when heated, are among the most frequent victims.

Symptoms are often obscure. The affected horse may be bright and lively, showing little respiratory disturbance unless under exertion. Yet there is a general appearance of unthriftiness, with erect, dry hair, hidebound, and a small, dry cough. Under work there is hurried breathing, early exhaustion, ready perspiration, and aggravation of the cough which then occurs in paroxysms. Auscultation and percussion give characteristic signs according as there may or may not be false membranes or effusion at particular points. It is usually bilateral in horses, unilateral in other animals.

In cows in addition to the corresponding symptoms, there is pallor of the mucous membranes when at rest, quickly transformed into congestion under exercise, suppression of the milk, and weak heart beats unless when excited. In the advanced condi-

tion the animal has difficulty of breathing when recumbent on the sound side and subcutaneous infiltration is felt or seen beneath the sternum or in the limbs. The affected side shows an increased dimension, vertical and longitudinal, of the chest, and the intercostal spaces in their lower part bulge out and fluctuate.

In cows and indolent animals there may be a quiescent condition or very slow progress, but any violent exertion is likely to give a sudden stimulus to the morbid process.

Lesions. The liquid effusion, usually unilateral, except in the horse may amount to 40 quarts in the latter animal, 30 quarts in the ox (Rigot), and 5 to 6 quarts in the dog. Unless there has been a recent sudden accession of inflammation it is of a pale straw color, with, in the dog, a slight rosy tinge. It clots loosely on exposure to the air and contains a large amount of albumen and few cell-forms. The false membranes are thick and white at some points and red and vascular at others. In the main they are completely organized. The lung is more or less collapsed and the right heart dilated and attenuated.

Treatment must be in the main tonic, diuretic and derivative. Food must be nourishing, digestible and in liberal amount; diuretics and bitter tonics with digitalis and, (if there is little fever) preparations of iron are to be pushed as far as the strength will allow; and the counterirritants applied to the sides of the chest a number of times in succession. Iodides may be used internally and externally, and *paracentesis* must be employed unless early improvement is manifested.

PLEURODYNIA.

Definition. Symptoms, stiffness, pointing of fore limb, catching inspiration, tender intercostals, less fever, cough, and hardness of pulse than in pleurisy, no friction sound nor signs of pleuritic effusion. Treatment, antirheumatic, derivatives, colchicum, alkalies, salicylate, salol, phenacetin, warm (steam) bath, warm building and clothing.

Definition. Rheumatism of the intercostal muscles. This has been occasionally observed in the horse, and is liable to be mistaken for pleurisy, which it closely resembles in its symptoms. There are the same stiffness of the fore limb on the affected side, the same short breathing, the same fixed and inactive appearance of the ribs, and the same extreme tenderness on pressure as in pleurisy ; but the high type of fever, the cough and the full hard and accelerated pulse are usually absent ; the tenderness tends to shift from one point to another, there is no shivering nor friction sound in the early stages, and no subsequent absence of sound and deadness on percussion over the lower part of the chest as result from effusion. When associated with fever it is very difficult to distinguish from pleurisy, and its recognition can only be made by these physical signs just mentioned.

Treatment. This must be the same as in rheumatic attacks in general. Rub the chest actively and repeatedly with a mixture of equal parts of spirits of turpentine, laudanum and olive oil, give ½ drachm doses of powdered colchicum daily and bicarbonate of potass freely in the water drank. Or give four times a day 2 drachms of salicylate of soda, or 1 drachm of salol, or phenacetin. A warm building and warm clothing are essential elements in treatment.

BRONCHIAL ASTHMA IN THE DOG.

Definition. Pathology, neurotic origin, bronchial spasms, swelling of mucosa, fibrinous inflammation of bronchioles, Berkart's streptococcus, irritants formed in indigestion, overfeeding, inactivity, plethora, constipation. Symptoms, obesity, sluggishness, recurrent paroxysms of dyspnœa, hard cough, tense abdomen, constipation, piles, depilation of skin, tartar covered teeth, fœtid breath. Retching, vomiting, a glairy mucus, emaciation may follow. Lesions, emphysema, fatty deposits in mediastinum, old standing diseases of the heart, lungs and digestive organs. Treatment, antispasmodics by lungs or rectum, stramonium, nitre fumes, emetic, purgatives, vegetable diet, exercise, sedatives, blisters. Asthma in the horse.

Definition.—A neurotic affection mainly affecting the pneumogastric nerve, and leading to paroxysms of stenosis or constriction of the bronchioles and attacks of dyspnœa. In its initial stages it is associated with corpulence and disordered digestion, and later with congestion and swelling of the mucosa of the bronchioles, emphysema, and dilatation of the right side of the heart.

Pathology. Asthma is generally attributed to spasm of the bronchial muscles (Williams), and though recent observations have failed to sustain this it must be admitted that in the majority of cases it is of decided neurotic origin. Again it is attributed to erythematous swelling in patches of the bronchial mucosa, (Clark). Another theory is that it is a fibrinous inflammation of the mucosa of the bronchioles, the tenacious exudate blocking the tubes more or less completely and relief coming with a more diffluent secretion. Berkart found a streptococcus in the sputa which he supposed to be the final cause. Again it has been held to depend on the circulation in the blood of deleterious matters introduced during digestion. Again it has been attributed to a neurosis roused by constipation and the accumulation of irritant matters in the intestine. Whatever local conditions may be operative, there can be no doubt that in dogs it is almost exclusively confined to those kept indoors, overfed, without exercise, plethoric and constipated. The disease seems to originate in and persist by nervous disorder propagated from the digestive organs.

A change of diet or any disturbing cause may bring on a paroxysm.

Symptoms. The disease is one of pet dogs, kept in towns, deprived of exercise, fresh air, and of the opportunity to relieve the bowels at will, and gorged with highly spiced meats, and sweets at least three times a day. Sluggishness and obesity are marked characteristics of the dog when first attacked though in the advanced stages the violence of the paroxysms and their frequent recurrence may have induced extreme emaciation.

The affection is usually ushered in by a cough, at first slight, but soon becoming frequent, hard and sonorous, as in the early stages of bronchitis. The cough becomes very troublesome and the breathing habitually labored, but at irregular intervals a paroxysm comes on which threatens death by suffocation. The dog stands or sits on his haunches with open mouth, pendent tongue and staring eyeballs, panting for breath, and has his condition aggravated by every change of position or other source of excitement. By the frequency and severity of the attacks may be estimated the danger of the patient.

An examination in the intervals of the attacks detects some disturbance of the digestive organs. The tense and distended condition of the abdomen usually manifests the existence of overloaded stomach and bowels, of indigestion, tympany and constipation. Piles are often present as a result of long continued costiveness. The skin is dry and unthrifty, and often in patches denuded of hair. The teeth are covered with tartar and the breath fœtid.

Retching is occasionally seen to occur during a violent access of coughing, but only a little glairy mucus is brought up.

The cough, hurried breathing, and paroxysms of dyspnœa become aggravated, the general health suffers largely, and death often ensues in a state of great weakness and emaciation.

On *dissection* of such cases the lesions of various old standing diseases of the heart, lungs, or abdominal organs have been met with at times, and such disorders have doubtless assisted in maintaining and aggravating the asthma. The most constant lesions, however, are emphysema of the lung, and accumulations of fat in the mediastinum.

Treatment. 1st. **During a paroxysm.** This is confined to

the administration of antispasmodics either by inhalation or as an injection to avoid the additional suffering of swallowing. Ether or chloroform may be inhaled from a sponge, but the employment of these should be guarded especially in advanced cases when besides the prevailing weakness there is reason to suspect structural changes in the heart. The same agents in doses of one, two or three teaspoonfuls, or laudanum in double that quantity may be thrown up as an enema, and may be combined with a couple of ounces of castor oil when costiveness exists. The fumes from burning stramonium or from burning brown paper which has been previously soaked in a strong solution of nitrate of potass, will in many cases suddenly cut short the paroxysm. If on the other hand there is reason to believe that the stomach is overloaded the attacks will often be suddenly cut short by giving an emetic. For this purpose a grain of tartar emetic may be shaken upon the tongue, or a dessert spoonful of wine of ipecacuan, or of antimony, poured over the throat. 2d. **In the intervals between the paroxysms.** Attention must be given to counteract any inflammatory action in the chest by which the disease may be maintained. Our chief object, however, must be to divest the animal of its superfluous fat and bring the digestive organs into a healthy condition. Unfortunately the propensity to fatten in some dogs seems to be a morbid condition. The food appears to be stored up as fat at the expense of muscular and other tissues even when the animal is kept on the borders of starvation. All flesh must be withheld and coarse vegetable fare alone allowed. A well boiled pudding (porridge) made with oatmeal or Indian corn meal, water and a little salt, with a small quantity of skimmed milk or buttermilk, is an excellent diet in such cases. The amount must be small, though the hitherto pampered favorite will rarely seek to fully replenish his stomach until he has forgotten his former extravagant habits.

A good deal of open air exercise must be given, not violent, but gentle and long continued, and this though the patient may appear physically unfit for it. Exercise should be given three hours or more after a full meal. Purgatives (one ounce castor oil) should be administered twice a week. A clean bed, not too soft nor luxurious, should be allowed in a dry, airy place. The skin should be well brushed daily and occasionally washed thor-

oughly with soap, care being taken to dry the coat completely afterwards. Sedatives should be given daily, such as a half grain each of stramonium and tartar emetic, and in advanced stages with weakness and emaciation vegetable tonics will be demanded.

Blaine strongly advocates a course of emetics, given every alternate day, and Mayhew lauds frequently repeated ammoniacal blisters to the sides. Such measures will be especially applicable when there is irritation and discharge from the bronchial mucous membrane. Strong subjects can alone, however, bear such treatment.

All cases of asthma in the dog are obstinate and critical and require much judgment in treatment.

ASTHMA IN THE HORSE.

Hering records a case of *spasmodic asthma*, in a strong cart horse. Besides the oppressed and difficult respiration, the animal was excessively dull and had no appetite, but the pulse was almost of the natural standard. The animal was not benefited by opening medicine but improved under active doses of extract of hyoscyamus. Quillaume reports two asses attacked apparently in the same way, and Delwart and Robertson refer to other cases. They recovered under antispasmodics. These are at least closely related to heaves, which is largely a neurosis at first.

ASTHMA. BROKEN WIND. HEAVES. DYSPNŒA.

Definition, neurotic affection with digestive and respiratory disorders. Causes, no racial exemption, disease largely coextensive with leafy hay from clover, alfalfa and other leguminous plants, musty hay, cryptogams, overloading the stomach, active work on a full stomach, overdriving, bronchitis, chronic bronchitis, emphysema. Nature, a neurotic affection, starting with derangement of some part of the vagus, dilatation of the right heart, congestion of the bronchioles. Symptoms, double expiratory action, flatulence, weak, husky cough, wheezing, glairy, grayish nasal discharge, wheezing, increased resonance along the margins of the lungs. sibilant râle, heart's impulse strong, even felt on right side, aggravation with overloaded stomach, costiveness or muggy atmosphere, improvement on laxative (green) food. Treatment, succulent green food, natural pastures, relieve any abnormal state of lungs or bowels, pure air, heart tonic, diet, arsenic, special diagnosis, guard against masking of symptoms by narcotics, privation of water, shot, lard, recto-vaginal fistula, diagnostic signs, dilated nostrils, auscultation and percussion signs of emphysema, relapse under hay and water.

Definition. A chronic affection of the equine species, manifested by a hurried, wheezy breathing, greatly aggravated by close, muggy weather, a full stomach, certain kinds of diet, or by exercise; by a double lifting of the flank with each expiration; by a small, weak, dry cough, often occurring in paroxysms and easily excited by a drink of cold water, exposure to cold air or a fibrous quality of food; and lastly, by a marked disorder of the digestive organs.

Causes. This disease is essentially the result of faulty feeding and working, though preexisting diseases of the air passages and sudden violent muscular efforts no doubt occasionally contribute to its development.

It has been alleged that some races of horses are exempt from this disease. Among these the Arabian, Persian, Barb, Spanish and Portugese are especially named but their immunity in all probability depends on the feeding and management rather than on any peculiarity of breed. The countries where these horses are met with are not subject to a prolonged winter but yield green food throughout the greater part of the year, and it is a notorious fact that no horse becomes brokenwinded at pasture. The Arabians

moreover "feed their horses on the scanty plants which the borders of the deserts supply and when these are wanting they are fed on a little barley with chopped straw, withered herbs, roots dragged from the sands, dates when these can be obtained, and in cases of need the milk of the camel. They drink at long intervals and in moderate quantities," (Low). Since an habitually overloaded stomach is the most common cause of *heaves* the absence of the affection in the Arab is not surprising. But the Arab unfortunately enjoys no such security in England or America. Concerning the Barb, Delwart remarks that after a day's hard work, fasting, he is fed on six or seven pounds only of barley, and without the cut straw that the Arab is allowed in similar circumstances.

In Spain and Portugal horses at work are fed on broken wheat and barley straw, from twelve to twenty-five pounds, and barley from six to twelve pounds daily, according to the size of the animal and the demands upon his strength. The mares are constantly at pasture and according to the rainfall they are starved or in abundance. Green food and a limited straw and grain diet are precisely the conditions in which broken wind does not appear. Rodriguez, veterinarian to the queen says that the disease was unknown to Spain until the cultivation of red clover, lucerne, and sainfoin. At Aranjuez, horses fed on the hay of these plants, lost vigor and wind and several became decidedly brokenwinded. All were, however, restored to health and vigor by a return to their former diet. Count Cardenas found that his horses gained in flesh on the new fodder, but that symptoms of broken wind developed themselves rapidly.

In France, M. Demoussy records similar facts. In Segala, where the aliment is substantially hay, brokenwinded horses abound, whilst in the adjacent district of Causse where horses are fed through the winter on straw and barley broken by the mules feet in the act of threshing or treading out, the disease is virtually unknown.

Lucerne and sweet trefoil are indigenous and grow abundantly in Causse and Caussergne but eaten green or after their seed has been shaken off and the stems have acquired a dry ligneous character these are innocuous. When however condemned to stand in the stable through a severe winter, with their racks constantly

filled with hay, they will eat from thirty to thirty-five pounds of this daily and many become broken-winded. The breeding mares which get little hay, seldom become affected though the plenitude of their abdomen and the impaired respiratory function might be thought to conduce to the affection.

In England broken wind is much less prevalent than on the European Continent and it is deserving of notice that lucerne and sainfoin hold no place among the British green crops, that red clover hay is only exceptionally met with owing to the amount of land that is *clover-sick*, that natural hay is largely used, and that when horses are largely fed on hay it is qualified by such laxative agents as turnips, carrots, beet, etc.

All this throws light on the immunity of horses on our western prairies and plains. Feeding on the indigenous grasses fresh or made into hay, they are saved from the noxious influence of those artificial products which are found in all countries to determine the development of broken wind. It needs not that we adopt the popular notion that any special plant growing in these pastures ensures the safety of the equine races. It is merely a repetition in the Western Hemisphere of the experience so long before obtained in the case of Spain. Parallel with the progress of cultivation in our western lands, we see this malady advancing. Fifty years ago it was virtually unknown in Michigan and adjacent states whereas now these states can almost emulate New York in the relative number of their victims. It must not however be supposed that this cultivated fodder is the sole cause of the westward march of this malady. With improved agriculture have come better roads, spring wagons and driving at a pace which was comparatively unknown to the early settlers.

In California the condition of Spain was for long pretty accurately repeated. With no winter worthy of the name, troops of horses were left at pasture throughout the whole year and those that were stabled subsisted chiefly on natural hay in which the indigenous grasses were commingled with white—but no red—clover. California long retained the reputation of having no broken-winded horses.

In our Eastern states where the disease was thirty years ago so notoriously prevalent, the fields of luxuriant red clover might well have excited the envy of the English farmer. The hay made

from this, full of seed and dust was given without stint to the farm horses, which during the rigor of the winter were often shut up in stable for a length of time continously and dangerously gorged themselves with this provender. In the Eastern States with a steady falling off in the red clover, there is also a corresponding reduction in the number of cases of heaves. The grain allowed them, a mixture, supposed to consist of Indian corn, oats and buckwheat, given as a dry coarse flour, was little calculated to counteract the effects of the clover hay, and the entire absence of turnips and other succulent roots as a farm crop precluded their use as a preventive of the malady. We need not forget the prevalent ambition to possess a fast trotter, nor the effect of the climate on the air-passages (See *chronic bronchitis*) in estimating the causes of this malady in the Eastern states.

The mere overloading of the stomach is a potent cause of the development of heaves. The horse is above all other animals compelled to undergo hard work on a full stomach. Coleman cites the experience of the coaching days when each horse had 20 lbs. of oats daily and not more than 5 lbs. of hay with no water before work. These horses were driven fast for long stages yet they never contracted broken wind under this treatment. Farmers' and millers' horses on the other hand were most subject to the disease because gorged continually with hay chaff and mealy food, and worked in this condition. "Nimrod" who confirms Coleman's statement says "I have taken some pains to ascertain this fact by my own personal inquiries. One proprietor who has nearly fifty horses at work—many of which are in as fast coaches as any that travel on the road—assured me lately that he had not a broken-winded horse in his yard; whereas before he stinted them in their hay he generally had one to five in that state." Percivall testifies to its comparative infrequency in the English cavalry horses, which have their diet carefully regulated. Hay musty from bad harvesting or other cause and such as is rank from growing in low wet localities are *cæteris paribus* more injurious than good hay.

Every day observation shows that driving a horse upon a full stomach often causes broken-wind and nothing will more surely aggravate it, when it does exist. The same remark may be made of the drinking of large quantities of water after feeding and just

before going to work. Gross feeders are above all others the subjects of the complaint.

The question arises how a disturbing cause operating directly upon the digestive organs should affect the respiratory, in such a marked and permanent manner. It cannot be because of the gastric and abdominal distension since pregnant mares though in a state of much greater plentitude, are not thereby rendered liable to broken wind, and if they have previously suffered from this infirmity, the symptoms are usually less marked when breeding. The explanation first advanced by Dupuy appears to be the correct one. The lungs, the stomach, and certain other organs derive innervation from the vagus nerve, and certain disturbances of the stomach and intestines so impair the function of this nerve that the lungs are affected, at first functionally and afterwards structurally. In support of this view is the fact that broken wind is usually associated quite as much with digestive as respiratory derangement. The horse though a heavy feeder becomes unthrifty, hidebound and emaciated; his dung is passed in an undigested state like so much chopped straw, and flatus is continually passed from the bowels. Indeed the almost incessant passage of wind and fæces, during the first mile or two of a journey, is a disgusting evidence of the malady. The power of doses of shot, fat and other agents to temporarily allay the symptoms may be held to point in the same direction.

Beside causes operating on the side of the digestive organs others undoubtedly superinduce the disease, and among these severe exertions and chronic bronchitis ought to hold prominent positions.

Overexertion induces overdistension and rupture of the air cells by the forced retention of air within the lungs, by the closure of the glottis, while the chest is strongly compressed by the respiratory muscles. It is an essential condition to all severe exertion in man that the breath should be held, and though the horse appears equal to the same efforts of draught after the operation of tracheotomy has deprived him of the power of holding the breath, yet he would seem to be sooner exhausted (Goubaux, Colin, Bouley), from which it may be inferred that this power is frequently exercised, and it probably always is in any sudden severe effort as in starting a heavy load, or jumping a five-bar gate.

This retention of air in the lungs during violent compression of the chest walls is precisely the condition met with during an access of coughing, and in both cases alike there is the tendency to overdistension of the minute tubes and air cells until they have lost their power of contraction, or they may even give way and allow the air to pass out and lodge in the lung tissue.

Another mode in which violent effort injures the lungs is by the rapid and continued inhalation of great quantities of air during rapid breathing, so as to dilate the lungs suddenly to their fullest extent. Sometimes from irregular distribution of the ærial current or from the want of tone in a particular part of the lung that gives way under the pressure and the air cells become overdistended or ruptured. This condition is especially met in the more rapid paces. It is well exemplified in the results of the deep breathing after cutting the vagi nerves.

In either case the result will be more certain if the effort is made upon a full stomach or with the functions of the vagus nerve impaired by a previous faulty diet.

That broken wind is a frequent concomitant or sequel of *chronic bronchitis* is undeniable, and theoretically nothing is more likely to cause dilatation and rupture of the air cells and consequent impairment of the innervation and contractility of the lung than violent fits of coughing, while the bronchial tubes have thickened and friable walls, or are partially plugged by tenacious mucus.

Broken wind is mainly a disease of old horses, though I have seen several cases in five-year-old animals, and Bouley records a case in a two-year-old colt out of a badly broken-winded mare. This would seem to indicate an hereditary proclivity, and there is no doubt that the shallow, narrow, weak chest predisposing to this as to many other pulmonary complaints is transmitted from parent to offspring.

Nature of the Disease. Emphysema of the lungs is the most constant structural change met with in the bodies of animals which have suffered from broken wind. This condition of the horse's lung appears to have been noticed by the early Greek writers. It was advanced as the cause of broken wind by Riding in 1704 (Pathologie Veterinaire), by Floyer in England in 1761 (Treatise on Asthma in Man), by Vitet in France in 1783 (Medi-

cine Veterinaire, Lyon), by Freutzel in Germany, and Bracy Clark in England in 1795. It was only, however, after the admirable discoveries of Laennec that the question was systematically investigated by Delafond, who has furnished the most comprehensive data on the subject. Out of fifty-four broken-winded horses dissected by him no less than forty-five had the lungs extensively emphysematous. This emphysematous lung differs according to whether the emphysema is *vesicular* or *interlobular*.

In **vesicular emphysema** the smallest bronchial tubes and the air cells have become distended beyond the natural standard and remain permanently so, the lung tissue having lost its power of contraction. If such a lung is inflated and dried, and a thin slice taken from the surface of the emphysematous part the size of the minute orifices on the cut surface will show its condition. These fine openings are only the air cells cut across, and in their healthy state they will admit no larger object than the point of a needle or a fine bristle. They are slightly larger in adult and especially in old horses than they are in the young. If affected by emphysema they will often admit a hempseed or even a small pea. On opening a healthy chest the lung collapses, contracting on itself and expelling the contained air; if the lung is emphysematous the diseased portion does not collapse and if the entire lung is affected it continues to fill the chest and may even bulge outward after it has been opened. The color of the emphysematous lung is of a brighter red than are the healthy portions. If a diseased lung has been left exposed to the air for twenty-four to thirty-six hours and then cut across in all directions, the diseased lobules may be distinguished at a glance by this lighter shade, and such light portions if near the surface will be found to correspond to elevations above the general level of the lung. If the diseased lung is placed in water it floats on the surface like an inflated bladder scarcely at all sinking into the fluid. If the lung is blown full of air the emphysematous part is first filled causing the bulging on the surface to be still more marked than before. Vesicular emphysema rarely affects an entire lung; it is usually confined to the anterior lobes and to the thin lower and posterior borders of the organ.

Interlobular emphysema is the extravasation of air into the connection tissue between the lobules owing to rupture of the air

cells, and smaller bronchial tubes. It may occur independently of the vesicular emphysema but more frequently, it is preceded by that form and results from it. It is manifested on the surface of the lung by irregularly formed transparent elevations movable from one place to another under the pressure of the finger contrary to what is the case in vesicular emphysema. These vary from the size of a pea to that of a hen's egg. When the air exists in the cellular tissue between the lobules it appears as intersecting lines circumscribing irregular spans, and seemingly dark colored to a superficial glance but seem to be transparent on a closer examination. Like the elevations on the surface these collapse on being pricked.

When a lung in this state has been inflated and dried it presents on the diseased parts the union of several air sacs into one by the rupture of their intervening walls so that a pea may be lodged in the cavity; it further shows wide and prolonged canals on the surface and in the intervals between the lobules—the dilated areolæ of the connective tissue. These abnormal conditions like the vesicular emphysema are chiefly met with in the anterior lobes of the lungs along their free borders and on their inner surface near the entrance of the bronchi.

One or both of these two forms of emphysema may be considered as essential conditions in all forms of broken wind. It does not follow that this is the primary disease; we have already seen that the cause of the malady is usually to be sought on the side of the digestive organs, and that impaired innervation, on the part of the vagus nerve or of the ganglia in the brain presiding over it, leads to these functional and structural changes in the lungs. If these changes are results and not causes, their extent will not necessarily bear a constant proportion to the intensity of the disease, though in reality they are generally found to do so.

From a series of injections of lungs from broken-winded horses M. Demoussy arrived at the conclusion that the essential lesion of broken-wind was an aneurismal dilatation of the capillary vessels of the lung. This is like the condition of the mucosa found in asthma in man and is explainable in both cases by the impaired innervation, as dilatation of these minute vessels is a natural consequence of the loss of vaso-motor nervous power, and contact with air saturated with carbonic acid.

Dilatation of the smaller bronchial tubes is frequently present and especially characterises such cases as supervene on chronic bronchitis. These dilated tubes contain a plastic, whitish, inodorous mucus.

Another frequent concomitant of the emphysematous lung is a dilatation of the right cavities of the heart, especially the auricle, and an attenuation of their walls. The same condition is noticed in pulmonary emphysema in man and like this is probably due to the slow and imperfect circulation in the diseased lung.

Collating these structural changes with the different causes of the disease we find that they harmonize with the theory of impaired function on the part of the vagus nerve or its presiding ganglia, whether this functional disturbance has its origin in disorder of the digestive organs, as in the great majority of cases, in severe muscular efforts, or in chronic bronchitis.

Section of the vagi nerves affords an exaggerated instance of their paralysis and its results. These are mainly emphysema, capillary dilatation, blood extravasation, inflammation and pulmonary collapse. Emphysema is the first result and due to the slow, deep respiration (Boddaert) and loss of contractibility (Longet); capillary dilatation results from the extreme distension of the air cells and the retention in them of air highly charged with carbonic acid (Donders); the other lesions occur later and own very different causes.

That this is the true nature of the disease would further appear from the occurrence of emphysema without broken wind, two cases of which are recorded by Percivall; and from the existence of broken wind without emphysema. Cases of this last variety have been recorded by Godine, Volpi, Rodet, D'Arboval, and Delafond in France; and by Sewall, Dick, Smith, Hallen and Gloag in Britain. In connection with this last class of cases it must be noted that dilatation of the right cavities of the heart sometimes gives rise to very similar symptoms, and that the signs of chronic bronchitis are often scarcely distinguishable from those of broken wind. In catarrhal bronchitis too, after the air tubes have been washed, it is sometimes impossible to decide whether the lining membrane has been the subject of inflammation or not.

Symptoms. The most prominent are the double lift of the flank with each expiratory act, in the absence of fever, the short, weak,

dry and almost inaudible cough, the wheezing noise in breathing when that is accelerated by exertion, and the intestinal flatulence with the frequent passage of gas.

The **cough** usually heralds the advent of other symptoms. Often the character of the cough draws forth the remark that an animal is becoming broken winded and though no other symptom is seen at this time they thereafter rapidly develop themselves. At this early stage of the disease the cough is paroxysmal, coming on in fits during work or after a drink of cold water. Once the disease is established the horse rarely coughs more than once at a time. The cough is extremely short, weak and low and followed by a sort of wheeze. So specific is it that if once heard it can readily be recognized. The sudden effort made in coughing usually leads to the expulsion of gas from the flatulent bowels.

The **double lifting of the flank** in expiration is not peculiar to broken wind. It is seen as well in most diseases of the lungs and even of other organs (enteritis, peritonitis) which interfere with the freedom of the respiratory act. If however it is not attended by fever but associated with the brokenwinded cough, the wheezing respiration, the disordered and flatulent state of the bowels, the tumultuous beating of the heart against the left side after exertion, and the slight flow of clear, watery matter from the nose, it is pathognomonic. The act of inspiration is quick and free, that of expiration is not uniform and continuous as in health, but consists of two stages interrupted by a momentary arrest. In the first stage the posterior part of the abdomen is slightly raised and and it falls in laterally; then comes an almost imperceptible period of inaction, followed at once by the further lifting of the flanks to complete the expulsion of air from the lungs. The first stage seems the natural collapse of the walls of the chest and forward movement of the diaphragm, the second a contraction of the abdominal muscles partly due to an exercise of will to overcome the obstacle to the expulsion of air.

In very bad or advanced cases these symptoms are more marked. The inspiration is sudden and manifested by a rapid expansion of the chest, and dropping of the belly previously supported by active contraction of the abdominal muscles. The two stages of the expiratory act are quite distinct. The first is manifested by a sudden falling in of the walls of the chest so that the ribs no longer

stand out prominently beyond their interspaces; the abdomen equally rises inferiorly and falls in laterally so that a projecting ridge is formed from the lower end of the last rib to the point of the hip. This is specially marked during the period of inaction, and this is succeeded by the second effort quick and almost convulsive. These movements are so extensive that they are conveyed in a striking degree to any vehicle to which the animal is attached, especially if it has only two wheels, and a rider on horseback feels the movement still more disagreeably. When a horse is in this state the alternate rising and falling of the abdominal organs imparts a synchronous movement of protrusion and and contraction to the anus and in thin subjects a rising and falling of the muscles on each side of the root of the tail. The nostrils too are kept constantly dilated.

There is a *nasal discharge*, but this is very inconsiderable in the early stages of the malady. It is a clear watery or slightly grayish albuminous material without any visible admixture of pus globules, and on drying it leaves a scarcely perceptible crust. At first it appears intermittently and in minute quantities, but in bad cases it becomes almost constant, and is especially profuse after exercise.

Abnormal respiratory sounds are marked symptoms in the advanced stages. The wheezing noise of the breathing, especially when that is excited by exertion, may be heard at a short distance from the animal. The increased resonance on percussion along the lower border of the lung is only heard when the emphysema is extensive. The dry sibilant or whistling râle heard over the same parts is equally a symptom of the advanced stages. When there is much discharge a moist rattle is often heard over the lower end of the windpipe or immediately behind the middle of the shoulder. The overlaying of the anterior lobe by the thick, muscular shoulder, and the complication of results obtained at the free border of the lung by the abdominal noises and resonance render these results less conclusive in the earlier stages and slighter cases.

The application of the hand to the side of the chest behind the left elbow may detect a strong impulse of the heart with each beat. If the patient is actively exercised for some time this may be felt on the right side as well. This symptom indicates the existence of dilatation of the right cavities of the heart.

The symptoms of **indigestion** are also very manifest. The dung passed is like so much chopped hay and oats, and does not at all resemble the fæces of a healthy horse. The abdomen is tumid, tense and filled with flatus, which is frequently passed *per-ano*, and has no doubt given rise to the name of **broken wind.** This expulsion of gas from the rectum usually takes place whenever the animal is excited to cough. When first started on a journey, the frequent passage of wind and dung for the first mile or two is one of the most disagreeable features of the disease. When the animal has thus *emptied himself* he usually goes much better for the remainder of the journey.

Broken-winded horses are always greedy feeders, and if they get little work they manage to maintain their flesh. But they are soft and flabby, and if put to active work they fall off rapidly, becoming emaciated and hidebound, a true indication of their impaired digestion.

The symptoms are liable to occasional aggravation. If the stomach and bowels are overloaded they are invariably so. If the patient is kept in a hot, close stable, the same result follows. Thick, muggy weather has the same effect. After a more than usually severe day's work all the symptoms may be intensified, and this may continue for several days or a week. Bouley attributes this to an extensive rupture of air cells and a sudden increase of emphysema, and the gradual subsidence of the symptoms to the partial absorption of the displaced air and the accommodation of the lung to its new condition.

Light and laxative diet on the other hand alleviates the symptoms and a broken-winded horse usually improves at grass.

Course. The general tendency of broken wind is to persistent aggravation, but by a judicious regimen many cases may be checked in their progress and greatly relieved, or even cured.

Treatment. We have already seen that broken wind is virtually unknown on natural pastures where the grass is short, green and succulent. Turning out on such pastures will improve or even temporarily cure mild cases. The same may be said of the lexative systems of diet. (See that recommended for *chronic bronchitis*). Feeding on dry grain only, with a very limited supply of water, will enable many broken-winded horses to do ordinary work with comparative ease and comfort. In such

cases, however, improvement is only due to the empty and unclogged condition of the digestive organs and the symptoms return with all their former intensity when the original diet is restored. By way of palliation much may be secured by avoiding accidental causes of aggravation. If catarrh or bronchitis has supervened it should be treated in the ordinary way. If the stomach and bowels are overloaded and costive, a small dose of aloes and enemata will relieve. If the stable is close a free admission of air will be beneficial. The temporary excitement in these cases may be further alleviated by sedatives, of which opium and digitalis have been mostly employed. The last agent will sometimes control the breathing to such an extent that the horse may be thought to have completely recovered. Professor Dick believed that he had effected a cure in one case by the administration at a single dose of a drachm each of camphor, opium, calomel and digitalis. Temporary results only can, however, be expected from such agents, except in the case of an aggravation due to a cause acting for a limited time only, in which case the partial improvement may be lasting.

By adopting such measures to check accidental complications and confining the animal to a rigid system of diet a broken winded horse may be worked with comfort to himself and his master. The aliment should be principally or exclusively of oats, bran or barley, though good succulent grass, turnips, carrots, beet, and potatoes may be allowed, as may also wheat or oat straw in limited quantity, but no hay and above all none prepared from red clover, alfalfa, sainfoin, or allied foreign plants and none that is musty or otherwise injured by keeping. No food nor water must be allowed for one or two hours before going to work, and the pace must be slow at first and gradually increased as the horse empties himself, and the breathing gets less embrrassed. If meadow hay, straw or other bulky food is allowed in small quantity this must be after the horse has returned from his day's work.

If the food above recommended is boiled or pulped, and mixed with some saccharine agent as molasses its restorative action is enhanced.

If, however, we add to these hygienic and dietetic measures a prolonged course of arsenic, the symptoms generally disappear. From five to fifteen grains of arsenic made into a powder with a

drachm of bicarbonate of soda may be given daily in the food until improvement is noticed or symptoms of the poisonous action of the agent appear. When these are manifested in loss of appetite, colicy pains or red and watery eyes the medicine must be suspended and begun again some days later in smaller doses.

The therapeutic value of arsenic in this case is probably largely due to its action on the nervous system, which has long been recognized. As early as the first century of the Christian Era, Dioscorides, recommended its use in asthma and in recent times it has acquired a considerable reputation for the treatment of neuralgia. Another—though perhaps an allied—physiological action of arsenic no doubt adds to its value in this equine disorder. This is its power of retarding the waste of tissues. This property it possesses in common with tea, coffee and some other agents, but to a greater degree. This has led to its extensive employment by the peasants in Lower Austria, Styria, and the mountains separating Austria from Hungary, who found that it improved their personal appearance, increased their weight and enabled them to sustain greater exertions in climbing without fatigue. It was the revelations of Dr. Tschudi concerning the Styrian arsenic eaters that first led Professor Bouley to try this agent in broken wind.

Examination of Broken-winded Horses. Though the symptoms enumerated above are sufficient to detect broken-wind in all ordinary cases, yet it may not be time thrown away to caution the reader against pronouncing it absent when the more prominent symptoms are not seen. Unscrupulous dealers do not hesitate to avail of a variety of devices to conceal the symptoms and make the animal pass for a sound horse. Digitalis and other sedatives are so employed, but are mostly rejected because they render the horse dull and sluggish. By some the bowels are unloaded by a dose of physic, the horse is kept on a spare diet of oats, beans and other grain, water is withheld, and on the morning of sale one or two pounds of leaden shot or of bacon fat are administered. The inconvenience attendant on the presence of these agents in the stomach makes the animal desist as much as possible from moving the abdominal organs, and the double lifting of the flank is thus more or less completely hidden. With the veterinarian however this measure like the last defeats its own

purpose, for such horses are always intolerably thirsty and if allowed to regale themselves at the nearest watering trough, the charm is broken, the double lift returns and with it all the symptoms of the malady.

A brutal practice existed among ancient farriers, of making an artificial opening into the rectum to allow the exit of the flatus upon which they conceived the disease to depend. This was effected either by cutting through the sphinctor ani with a knife or by making a new opening to one side of it with a red hot iron. According to Ferguson this has been improved upon by the modern Irish jockey, in the case of broken-winded mares. With the knife an artificial communication is made between the rectum and the vagina, of sufficient size to insure that it will remain open and large enough to allow pellets of dung to pass into the vagina. The double lifting of the flank forces the fæces through this artificial opening, and to avoid the inconvenience of their presence in the vagina the animal carefully refrains from this action. This orifice further allows the free escape of any gases generated in the rectum and thus materially relieves the flatulence. Ferguson says he has seen broken-winded mares that have been operated on in this manner, that breathed so freely that even professional men have failed to detect the affection.

In all cases of broken wind, no matter how masked there will be manifest, on slight exertion, a permanent dilatation of the nostrils—*i.e.*, alike in inspiration and expiration,—and when any such suspicious symptom is seen the horse should be carefully examined, especially the state of his lungs as ascertained by auscultation and percussion, his breathing after he has freely partaken of water and hay, and, if there is suspicion of drugging, after he has stood over night in a hot stable plentifully supplied with both hay and water.

It should be borne in mind that mares advanced in pregnancy often show no double action of the flank though decidedly broken winded.

POLYPUS OF THE BRONCHIAL TUBES.

Like other mucous membranes, that covering the bronchial tubes, is liable to diseased growths, which may each remain attached by a broad base, and form a morbid elevation of the surface, or it may become loosened and retain its connection with the mucous membrane only by a neck or pedicle. An interesting case of the latter variety is recorded in the *Edinburgh Veterinary Review* for January, 1864, by Mr. Parker of Birmingham. It was attached to the wall of the right bronchus about an inch below the bifurcation of the trachea, and had an ovoid form measuring 8½ inches in its longest circumference by 4½ in its shorter. The pedicle was 1½ inch long and allowed the tumor to pass freely upward into the lower part of the windpipe, threatening instant suffocation. It had a fibrous structure and was continous with the interlacing bundles of yellow elastic tissues which cover the cartilaginous rings of the bronchus.

From its frequent displacement upward the tumor gave rise to paroxysms of hurried and difficult breathing apparently threatening suffocation, but when these passed off, respiration was tolerably tranquil and easy. The fits of dyspnœa came on after any unwonted excitement, but above all after a cough. In such cases the tumor had been coughed up into the lower end of the windpipe and until it slid back into its former position, the animal seemed on the very verge of death. The paroxysms had appeared very frequently during a period of five months that had elapsed since her purchase, increasing steadily in severity and finally causing death. The lungs contained many small abscesses the result doubtless of the frequent paroxysms of dyspnœa.

Cases of this kind are not usually amenable to treatment, nevertheless as they are pretty certain to end fatally if neglected, it would be quite permissible to perform tracheotomy in the lower part of the neck and attempt to snare the tumor with an elastic wire passed through a single or double tube. The tumor might even be seized by a vulsella and twisted off, provided it could be reached.

DISEASES OF THE BRONCHIAL AND MESENTERIC GLANDS.

These are the seat of congestions, neoplasms, and parasites, including pentastoma, actinomycosis, tubercle, glanders, etc., which will be treated under their respective headings.

PARASITES OF THE AIR PASSAGES, LUNGS, AND PLEURA.

Nearly all the domestic animals are subject to parasites of the lower air passages. These give rise to verminous bronchitis in cattle, sheep, horses, swine, goats and camels, to gapes in birds, and to pulmonary acariasis in fowls. The lungs and pleura are invaded by distomum, echinococcus, cysticercus, aspergillus, etc. (See parasites).

DISEASES OF THE HEART AND ORGANS OF CIRCULATION.

Susceptibility in different genera. Reasons for partial immunity of the quadruped, special and general causes in quadrupeds, violent, forced work, fatty degeneration, swallowing of pointed metallic bodies, difficult diagnosis in the animal. Position of the heart in the horse, ox, sheep, pig, carnivora, birds. Structure of the heart as a pump. Results of imperfect structure or action. Heart-walls. Table of size of the heart. Capacity. Weight. Pulse in each healthy genus, according to age, size, environment, temperament, proximity to parturition. Morbid conditions of the pulse, frequent, slow, quick, tardy, full, strong, weak, feeble, indistinct, small, hard, wiry. thready, oppressed, leaping and receding, intermittent, unequal, irregular, anæmic, venous. Percussion. Palpitation. Auscultation. Healthy sounds. Morbid sounds, in unusual place, force, intensity, rythm, repetition of 1st sound, of 2nd sound. Murmurs, synchronance with given stages of heart movement, their significance, pericardial murmur. General symptoms of heart disease, cold extremities, passive congestions, dropsies of limbs, etc., shortness of breath, venous pulse, vertigo, dulness, sluggishness, corpulence.

The lower animals are perhaps less subject to heart disease than mankind, but the comparative immunity generally assumed for them is far from being a real one. The horizontal position of the quadruped largely obviates that special tax upon the heart demanded by the erect position of man, and especially by the elevated place given to his more ample and vascular brain. Animals too are comparatively free from those mental and moral influences which so largely affect the regularity of the circulation in the human subject. But on the other hand many physical causes of heart disease affect the lower creation equally with their lord, while some undoubtedly operate with special force on the brute. All animals are subject to diseases of the heart as of other internal organs, from exposure ; this organ is occasionally involved from its contiguity with other diseased structures or from interdependence of function as we have already seen in certain diseases of the lungs (congestion, brokenwind, etc.); the tendency to heart disease frequently runs in a particular family of animals, especially with the rheumatic constitution, which is transmitted from parent to offspring as surely as the color of the skin the turn of

the horn or the depth and spring of the rib. The lower animals are further subject to congenital malformations and imperfections and to deposits of morbid material around the heart or in its substance so as to impair its healthy action.

Horses and dogs have special predisposing causes in the violent and prolonged exertion to which they are habitually exposed. The quiet sluggish and nonexcitable ox and pig meet with dangers no less real though of a different kind in the overfeeding which induces fatty degeneration of the heart as of other muscular tissues. The larger ruminants are further endangered by their propensity to swallow needles and other sharp pointed bodies which ultimately reach and penetrate the heart.

The prevalence of heart disease in animals may be deduced from the fact that out of 150 horses, oxen and dogs dissected at Montfaucon by Leblanc in 1840, not less than one twentieth presented cardiac lesions. The supposition of an immunity of the lower animals has been largely due to the heavy muscular shoulder of quadrupeds which covers the upper and anterior regions of the heart shutting them out from physical exploration. In man the entire heart and connecting blood vessels are so open to examination that the physician can pronounce with the greatest accuracy not only concerning the existence of disease, but also its precise locality and nature. In the quadruped no such facility is open to us, and veterinarians have too generally refused to face the difficulty, preferring to ignore heart diseases, or still worse seeking to cover their ignorance by the assertion that such affections rarely exist. Now however we not only know that heart diseases are much more frequent in the lower animals than heretofore believed, but that as a general rule they are sufficiently manifested and recognizable by their distinctive symptoms.

Position and exposure of the heart. In the **horse** the heart has only its apex and a small portion of its left ventricle approached to the surface of the chest, at a point where it is felt to beat behind the left elbow. The apex approaches the surface in the interval between the fifth and sixth ribs and close above the breast bone. The posterior border of the ventricle follows a nearly vertical line upwards from this point, while the anterior border has a direction upward and forward crossing diagonally over the fifth rib. The part of the ventricle exposed extends about three

inches upwards from the apex, and is about two inches in its transverse diameter. The great mass of the organ is covered by lung substance.

In the **ox** about the same extent of heart tissue is exposed. In **sheep** a portion about an inch in height and one and a half inches in breadth is left uncovered by lung. In the **pig** the heart is exposed only in a triangular space of about an inch across.

In the **carnivora** the heart lies more directly in the median line of the chest. It appears as if tilted forward so that its apex is directed backward and its base forward, while the body of the organ lies directly over the breast bone. The lungs invest it on both sides preventing any approximation to the walls of the chest laterally, and it can best be auscultated by applying the ear over the sternum.

In **birds** the heart is situated in the centre of the chest and enveloped by lung tissue so that its exploration is about equally difficult at all points.

The larger blood vessels at their origin from the heart are not open to examination in the lower animals except to a limited extent in the dog.

Internal arrangement and structure of the heart.. In all warm blooded animals the heart is composed of two portions, the internal cavities of which are perfectly distinct from each other and contain blood in different conditions; the right portion holding the impure, purple or venous blood which has just circulated through the body, and the left portion being filled with the bright crimson or arterial blood, which has been ærated by circulating through the lungs. Each of these portions is divided into two distinct cavities, an *upper* (auricle) which receives the blood from the veins, and a *lower* (ventricle) which receives the blood from the auricle and transmits it into the arteries. The auricle is separated from the ventricle by a transverse musculo-membranous partition having a large central orifice furnished with valves (auriculo-ventricular), the free borders of which are turned downward so that they allow the blood to flow freely downward from the auricle but completely close the orifice and prevent any reflex when the ventricle contracts. The great artery which originates from the base of each ventricle is likewise furnished with a system of valves (semilunar) having their free borders turned

into the artery, so that they allow blood to flow freely into that vessel during the contraction of the ventricle, but prevent any reflux into the heart when the ventricle again dilates. The apparatus may be likened to a force pump with two systems of valves, one to prevent the return of any water from the pump into the fountain; the other to hinder any reflux from the delivery pipe into the pump. Any interference with either of these valves entails a very serious and usually a fatal disorder of function.

These *orifices* differ considerably in size. Those between the auricles and ventricles are considerably larger than those at the commencements of the great arteries. Those on the right side of the heart too are greater than those on the left. They vary with the form of the heart. Thus in dilatation of an auricle and ventricle on one side of the heart, the auriculo-ventricular opening becomes equally widened and the valves remaining disproportionately small the blood is allowed to rush back into the auricle during ventricular contraction. The left auriculo-ventricular opening has been known to become contracted in some very flat and shallow chests; the blood failing to circulate freely through the lungs and to reach the left side of the heart in a full supply this orifice accommodates its size to the amount, and may become so narrowed that it forms a serious obstacle to the blood flow and a series of morbid changes result following the backward course of the circulation. The auricle first becomes overdistended and its muscular walls increase in thickness and consistency; the lungs tend next to suffer from a passive congestion, and lastly the right side of the heart becomes engorged and enlarged.

Any obstruction in the aorta which conveys the blood from the left side of the heart equally leads to dilatation of its internal cavity and abnormal thickness of its walls.

The imperfection of the valves is one of the most serious results of such changes in heart structure. The sounds by which such imperfection may be recognized will be presently noticed, meanwhile the mode of testing this in the heart of the dead animal will be referred to. If due to structural changes in the valves themselves, the new deposits, the cicatrices, the lacerations, etc., will be visible to the eye. Though no such disease changes are seen the valves may still manifest imperfection by failing to fulfill

their normal function when put to the test. Water is poured into one or other of the great arteries which arise from the ventricles, the vessel being held vertically, and if it fails to descend into the heart the valvular action is perfect. The auriculo-ventricular valves may be equally tested by filling the ventricle and observing whether there is a reflux into the auricle.

The **thickness of the walls of the heart** varies in disease. The auricular walls are invariably thin and flaccid except as above noted with diminution of the auriculo-ventricular orifice. The walls of the right and left ventricles differ in thickness in accordance with the distance to which they have respectively to propel the blood and the propulsive effort demanded. Thus the walls of the *right ventricle* which is only called upon to propel the blood through the lungs are only about ⅛ an inch in thickness and are thinnest at their lower part. Those of the *left ventricle* which have to send the blood to the most distant parts of the body are from 1 to 1½ inches except at the lower part where they form the apex of the heart, and are reduced to a tenuity resembling the walls of the auricles. They are thickest at the median part, and diminish slightly in an upward or downward direction. The bulk of these walls is excessively muscular, the fibres arranged as an elaborate double spiral and connected with a layer of white fibrous tissue placed in the interval between the auricles and ventricles and surrounding the auriculo-ventricular openings and the orifices by which the great arteries take their origin. It is at this point, where the muscular fibres of the ventricles are connected with the white fibrous rings, where rupture of the heart usually takes place.

The following measurements may be held to refer to medium sized animals of the different kinds mentioned.

	Longitudinal Diameter. Inches.	Transverse Diameter at the Base of the Ventricles.		Circumference. Inches.
		Antero-Posterior Inches.	Transverse Inches.	
Horse	10	$7\frac{1}{2}$	$5\frac{1}{3}$	$19\frac{1}{2}$
Ox	$9\frac{1}{5}$	$6\frac{3}{5}$	$4\frac{1}{2}$	$17\frac{1}{2}$
Sheep	4	3	2	$7\frac{1}{2}$
Pig	$4\frac{1}{2}$	$3\frac{3}{5}$	$2\frac{1}{3}$	$9\frac{1}{5}$
Dog	$3\frac{1}{3}$	$3\frac{3}{5}$	2	$8\frac{1}{2}$

The **internal capacity of the ventricles** is so modified by the amount of *post mortem* contraction that it differs widely from the actual capacity during life. The left ventricle of the larger domestic quadrupeds usually admits from 3½ oz. to over 5 oz., while the right ventricle whose walls are so much thinner and more lax will contain double that amount. In the smaller animals about a tenth of these quantities will be admitted.

The **weight of the heart** too can only be stated as an average or for medium sized animals. In the *horse* it may be from 4½ lbs. to 9 lbs.; in the *ox* from 3 lbs. 5 oz. to 4½ lbs.; in the *sheep* from 5½ oz. to 7 oz.; in the *pig* from 9½ oz. to 14 oz.; and in the *dog* from 5 oz. to 7 oz. This statement must be understood to apply to dogs approximating in size to the shepherd's.

Taking into account the size of the particular animal any considerable deviation from these measurements and weights may be accepted as abnormal. The ratio to the body weight is about :—horse and dog 1 : 100, ox, sheep and pig 1 : 220. This necessarily varies with condition—fat or lean.

The **pulse** offers valuable indications in disease of the heart.

The *number* of the pulse in healthy full-grown animals may be set down as follows per minute :—horse, 36 to 46; ox, 38 to 42 (with loaded paunch or in a hot stable up to 70); sheep, goat and pig 70 to 80; dog 80 to 100; cat 120 to 140; goose 110; pigeon 136; chicken 140. In old age the pulse is less frequent. This diminution may extend to 5 beats per minute in the larger quadrupeds and to 20 or even 30 in the smaller. Youth and small size again are associated with a greater rapidity of the pulse. The pulse of the foal, at birth, is about three times that of the horse; in the colt of six months it is double; at a year old about one and a half times; and at two years old one and a quarter.

The smaller the animal, *cæteris paribus*, the more rapid is the pulse. Hot buildings, exertion, fear or any other exciting cause likewise accelerates it. It is more frequent with the nervous temperament, as for for example in the English race horse, or the greyhound, than in the dull lymphatic cart-horse or mastiff. In advanced pregnancy it is increased in number. In the cow and mare it undergoes a monthly increase of four or five beats per minute after the sixth month. (Delafond)

Independently of these conditions a rapid pulse indicates febrile

excitement attendant on active inflammatory or other disease, or a state of weakness and debility. In this last condition the heart beats more frequently to secure a more rapid circulation in the capillary blood vessels, and thus make up to the craving tissues by frequency of contact, what is wanting in the quantity and quality of the nutritive fluid. This point cannot be too much insisted upon, as the fatal doctrine that a rapid pulse indicates force of the circulation is very misleading as to treatment.

The **force and character of the pulse** differ in the various species. In the **horse** it is full, moderately tense and elastic. In the **ass** and **mule** it is smaller and harder, with an inequality of force in successive beats, and sometimes even a beat is suppressed or imperceptible. In the **ox** the pulse is full, soft and regular, appearing to roll forward beneath the fingers. In the **sheep** and **goat** the pulse is small but with a peculiar quick or sharp beat. The **pig's** pulse is said to be firm and hard. That of the **dog** and **cat** is firm and hard coming with a sharp impulse against the finger. In the dog, however, successive beats are not always of the same force and an intermission or complete absence of a beat is by no means an indication of disease of the heart or other serious malady. It often attends the slightest excitement in a perfectly healthy animal.

In disease the *pulsations* may become :—**frequent** or increased in number ; **slow** or decreased in number ; **quick** or striking with a sharp impulse against the finger ; **tardy** or without sharpness of stroke and as if they rolled slowly past under the finger ; **full** and **strong** when the impulse is forcible and not easily compressed by the finger ; **weak, feeble** or **indistinct** in the opposite conditions ; **small** when though perfectly distinct and forcible they are wanting in fulness ; **hard**, when forcible and jarring (this is sometimes called **wiry** or, if smaller, **thready**) ; **soft** when though the artery may be full the beat is devoid of hardness and easily compressible so as to be unfelt ; **oppressed** when with a full rounded artery, the impulse is jerking though not hard and as if the distended vessels opposed the transmission of the impulse ; **jerking and receding—leaping**, when with empty and flaccid arteries the pulse seems to leap forward with each beat of the heart—(this pulsation may be visible to the eye in the carotids) ; **intermittent** when after a number of beats at regular in-

tervals there is a complete pause extending over that period of time which would have been occupied by a full beat; **unequal** when some beats are strong and others weak; **irregular** when without any distinct intermission for a period equal to that of a single beat, the intervals between successive beats are of varying length. The pulse further has a peculiar **thrill** or **tremor** in states of great debility with deficiency of blood and imperfect filling of the vessels.

Of these the **leaping**, the **intermittent**, the **unequal** and the **irregular** pulses are of special importance in their bearing on heart diseases.

The **jerking and receding** pulse is felt in cases of imperfection of the semilunar valves at the commencement of the great aorta, and which allows blood propelled into the arteries by the contraction of the ventricle to flow back into the ventricle during its state of relaxation. This pulse is met with in other conditions as in aneurism of the aorta, but if from heart disease it is distinguished by the presence of a *blowing murmur* with the second sound of the heart.

The **intermittent** pulse indicates functional derangement of the heart but it does not as is generally believed betoken structural disease. It is frequently observed in healthy asses and mules, and in dogs however slightly excited whether by fear or joy, or by the mere fact of their being handled, it is so common as to be almost the rule rather than the exception. It may be seen in a healthy horse as the result of excitement. During the early stages of convalescence from inflammatory affections of the lungs in the horse the pulse is often intermittent. The pulsations are at the same time unequal. There is a regular cycle of beats gradually decreasing in force and extending over a complete respiratory act. The cycle commences with the strongest beat during or immediately after the act of expiration, and the succeeding four or five beats are less and less forcible until the chest is fully expanded when there is a quiescent interval corresponding to the period of one beat. In many such cases there is no other indication of heart disease and the phenomenon appears due to the interference with the circulation by the hepatized lung, to the impaired nervous energy of the heart and to its compression between the distended lungs. A pulse simply *intermittent* and not asso-

ciated with any further sign of heart disease does not then possess the significance generally attributed to it, but a careful examination of the heart should invaribly be made when this functional disorder is observed. It exists or may be brought about by slight excitement in the great majority of heart diseases.

In case of intermittent pulse it is useful to ascertain whether there is also an intermission of the heart's beat, since in softening of the heart, that organ may beat without being able to transmit the impulse along the artery.

A pulse at once **unequal and irregular** is a much more serious indication than a merely intermittent pulse. It is observed especially in fatty degeneration of the muscular substance, and with imperfection of the valves on the left side of the organ, though it may be present in other cardiac diseases independent of the existence of those lesions.

In *hypertrophy of the left ventricle*, the pulse is full and strong and the impulse appears prolonged, because of the greater length of time taken up by the ventricle in the act of contraction. When *dilatation* coexists with *hypertrophy* the impulse is still full and strong, more blood being transmitted through the vessel; but when *dilatation* is combined with *attenuation* of the ventricular walls the impulse is soft and weak by reason of the feebleness of the contractions.

The pulse at the radial artery should be about synchronous with the beat of the heart. If retarded it may be held to indicate the existence on the anterior aorta or its primary divisions of an aneurism with elastic walls or more probably an imperfection of the aortic valves, which allows a regurgitation of the blood into the heart.

Venous pulse. A venous pulse seen in the lower end of the jugular veins is common in the domestic animals. In the ox it is quite compatible with health and is only to be judged by its amplitude and force. In other animals it often coexists with congestion of the lungs which impedes the circulation through the right side of the heart and leads to engorgement of the venous system. In the absence of this condition it frequently indicates an imperfection of the auriculo-ventricular valves in the right heart and a reflux of blood from the contracting ventricle which checks the descending current in the veins.

Percussion. In the **horse** a dull, dead sound is emitted when percussion is made over the left side for about four inches above the breast bone and in the space corresponding to the lower ends and the cartilages of prolongation of the fourth, fifth and sixth ribs. In the **ox** this dulness is less marked on the level of the sixth rib. The same results can be obtained on the right side by imparting heavier blows to the chest walls so as to derive the sound from the deeper parts.

The area of dulness is *increased* in cases of *hypertrophy* or in *dilatation* of the heart when the enlarged organ presses aside the lung tissue and exposes a greater amount of its substance to the chest walls. The same result takes place in hydropericardium.

The area of dullness is *diminished* in cases of ruptured air cells (as in "heaves") when the inflated and expanded lung tissue envelopes the heart more completely and gives out its own clear resonance where the dull sound of the heart is usually obtained.

Application of the hand. Palpation. In conditions of health and in quietude the hand applied on the side of the chest, close behind the left elbow only just perceives the beat of the heart with each contraction. If the animal is excited whether from fear, joy or physical suffering the heart's impulse becomes more powerful and by this alone the state of its function may be very satisfactorily ascertained. The impulse is strong in all active fevers and extensive inflammations of important organs, but it is especially marked in diseases of the heart and lungs. Irregularity in the force of successive beats is seen in various heart diseases and debilitated conditions are recognized in the same way.

Any want of harmony between the heart's action and the pulse may be observed by laying the right hand over the region of the heart and applying the fingers of the left on the radial artery. In debility and especially if from a deficiency of blood the violent or tumultuous action of the heart contrasts strangely with the weak jerking and compressible pulse. The same symptoms are noticed when the valves of the heart close their orifices imperfectly. In convalescence from lung diseases and in certain diseases of the heart a beat may be felt by the right hand for which no corresponding pulsation is felt in the radial artery by the left.

When the heart is hypertrophied the impulse is stronger and is associated with a full, strong, and rolling pulse. When it is atro-

phied the impulse on the chest and pulse beat are equally weak. When water exists in the pericardium the heart strikes the ribs with less force.

Sounds of the Heart. Synchronous with each beat of the heart two distinct sounds are heard, separated by a short interval, inappreciable to most ears, and followed by a period of silence. These sounds are distinct alike in character and duration. The *first sound* is dull and prolonged; the *second* is short and quick. Some idea of these sounds may be formed by the pronunciation of the two syllables, *lub—tip*, but an acquaintance with the sounds themselves is essential to a correct conception of them. The period of time occupied by the first sound is double that taken up by the second and in man and the smaller quadrupeds the subsequent period of silence is of equal duration with the second sound. Dividing the time belonging to one revolution of the heart into four equal periods the first two are taken up by the first sound, the third by the second sound and the fourth by the interval of silence. In the horse the silence is more prolonged, and occupies the entire latter half of the period of a revolution. The relations stand thus:—the first sound extends over two-sixths of the time, the second sound over one-sixth, and the silence over three-sixths.

The *first sound*, sychronous with the beat of the heart against the ribs corresponds also in point of time with the contraction of the ventricles, the closure and tension of the auriculo-ventricular valves and the rush of the blood into the great arteries. The second sound corresponds to the reflux of blood in the arteries and the closure of the valves between them and the heart. The period of silence represents the period of rest during which the heart is being filled from the veins.

In the horse, at rest, the first is the only sound that can be distinctly heard in many cases, but during the excitement of exercise, or in febrile conditions the second is sufficiently apparent and any deviation from the natural character is easily noted.

These sounds are most distinct over the lower end of the fifth and sixth ribs on the left side, but they may be heard distinctly behind the middle of the shoulder on either side when the corresponding limb is advanced. In birds they may be heard beneath the wings but above all and most clearly over the breast-bone.

In disease these **sounds** may be heard in unusual situations, they may be altered in force duration or rythm, or they may be associated with other sounds or superseded by them.

The sounds may be heard in new situations, in displacements of the heart from tumors or effusions in the chest, structural changes in the lungs, pleuræ, or pericardium, aneurism of the aorta, etc., etc.

The heart sounds are clearly heard over any part of the chest when the lung tissue intervening between that part of the surface and the heart is solid (hepatized). They are heard distinctly behind the median part of the right shoulder, when liquid effusion into the left pleural sac has displaced the heart to the right; and when the right cavities of the heart are extensively dilated as exists so commonly in the advanced stages of "*heaves.*"

The **extent** over which the sounds may be heard is *increased* when the lung surrounding the heart is solidified (hepatisation, splenisation, etc.), or when liquid effusion exists in the chest. A liquid but more especially a solid is a better conductor of sound than the spongy lung. Enlargement (hypertrophy) of the heart equally increases the area of sound. The *area* of sound is lessened by atrophy of the heart, and by an emphysematous condition of the lungs by which the heart is more extensively covered and further separated from the walls of the chest.

The **force** or **intensity** of the heart sounds is increased in high fever, in acute inflammation, in increase of the muscular walls of the heart with enlargement of the internal cavities, in functional disturbance from fear or other exciting cause, and in palpitation. Often in a weak and bloodless patient the heart sounds can be clearly heard at several yards distance from the animal. The *intensity* of the sounds is diminished in debility when not associated with palpitation, in atrophy of the muscular substance of the heart, in hypertrophy of the muscular tissue of the heart with diminution of its internal cavities, in *broken wind* when the the emphysematous lung more completely envelopes the heart, and in cases of extensive liquid effusion into the pericardium which prevents the apex of the heart from striking against the side of the chest.

The regular **rythm**, normally manifested by the two sounds and the silence, may be modified in the unequal irregular or in-

termittent contractions of the heart. Küssmaul's paradoxical pulse is one in which the pulse is more frequent but less full during inspiration than expiration. Seen in weak heart, during recovery from chest diseases, in chronic pericarditis, and when fibrous bands encircle the root of the aorta. Bigeminal and trigeminal when two or three beats follow each other rapidly, and are separated from the preceding and succeeding beats by longer intervals. This occurs in disease of the mitral valve, and in other weak states of the heart. Fœtal heart rythm in which the pause is shortened and the two sounds of the heart are almost identical, is seen in the later stages of fevers, and in extreme dilation. A curious aberration of rythm is the *repetition* of either the first or second sound. If of the first sound (anapestic bruit) each beat will be accompanied by three sounds the first two of which resemble the first sound of health. If the second sound is repeated (dactylic bruit, bruit de galop) the first sound only will be prolonged and the last two sharp and quick. The repetition of the last sound is probably due to impaired nervous supply which allows the completion of the contraction of the ventricle and the closure of the arterial (semilunar) valves sooner on one side than the other. If due to diminution of the arterial orifice which retarded the emptying of one of the ventricles, the first sound would probably be accompanied by a blowing murmur. If the auriculo-ventricular valves on one side were imperfect, allowing a reflux into the auricle and a more rapid emptying of the ventricle a blowing murmur would equally accompany the first sound. In either of these two last mentioned cases the murmur would mask or hide the first of two doubled sounds.

The repetition of the 1st sound is often due to dilitation of one ventricle, which in consequence is longer in reaching the same sensation of plenitude, and in receiving the stimulus to contraction.

Morbid Sounds. Murmurs. The distinct and superadded sounds heard in disease are usually designated murmurs. They originate in the interior of the heart (endocardial) or externally to the heart (pericardial). The **endocardial** sounds mostly arise from some abnormal conditions of the valves or orifices and consist in a blowing or rushing noise which usualiy accompanies or displaces one of the heart sounds, though it may precede or succeed these. The following table modified from that of Bartle

and Roger presents at a glance the relations of these different sounds and their significance.

Blowing or Hissing Murmurs.

Blowing murmur before the first sound.		Narrowing of the auriculo-ventricular orifice. Vegetations or coagula on the valves.
Blowing murmur with the first sound.	Strongest toward the base of the heart. Propagated along the great arteries.	Narrowing of the aortic opening.
	Strongest toward the apex of the heart. Not propagated in the great arteries.	Narrowing of the pulmonary artery or insufficiency of the auriculo-ventricular valves.
Blowing murmur with the second sound.	Double rushing sound heard over the great arteries. at each beat of the heart.	Insufficiency of the arterial (semilunar) valves.
Blowing murmur after the second sound.	Double rushing sound in the arteries with each beat of the heart.	Aneurism (dilatation of the great aorta.

From the table it will be seen that each orifice in the heart may become the seat of two perfectly distinct and independent murmurs; one due to constriction of the orifice in which case the sound is produced with the onward progress of the blood wave; and one due to dilatation of the orifice or insufficient closure of it by the valves, when the sound is due to a recoil or regurgitation of the blood. There is a further sound due to mere roughness of the valves in cases of disease when the sound will be with the normal current of blood, though a second or regurgitant hiss is often heard from the valves being at the same time insufficient to close the orifice. Another blowing murmur is usually heard over the heart and coincident with its first sound in the bloodless state (anæmia). This is not necessarily connected with any diseased condition of the heart itself.

The nature of these murmurs differs in special instances. They may resemble the soft whisper of the words *who* or *awe*, of the double letter *ss*, or the single letter *r*, according as they are soft or hard and purring.

The **pericardial murmur**, caused by the rubbing of the dry roughened surface of the serous membrane covering the heart on the correspondingly dry rough surface of the same membrane, re-

flected on the investing sac, resembles that caused by passing the palm of the one hand over the other which lies on the ear. It is distinguished from the friction sound of pleurisy by its coinciding with the movements of the heart and not with those of respiration. It is usually heard alike during the sounds of the heart and during the period of silence or in other words during the movements of contraction and dilatation in that organ.

General Symptoms of Heart disease. In the acute inflammatory affections there are the signs of general constitutional disturbance attending similar affections in other organs. The decision as to the true nature of the disease must be arrived at from the special character of the pulse, heart sounds, etc. as already noticed.

In the chronic forms of the disease however a particular class of symptoms usually point towards the organ affected. In cattle, sheep and pigs raised only for slaughter, and as far as possible protected against active exertion, serious heart diseases may exist for a length of time without making themselves manifest by any prominent symptoms. Thus in cows, pins and other sharp pointed bodies swallowed with the food frequently make their way to the heart and lodge for a length of time in its vicinity without material derangement and when at last the animal dies a sudden death they are found transfixing the walls of that organ. In the horse or other animal subjected to exertion the symptoms are usually very patent.

When the heart is enlarged the pulse strong and the circulation full and free, apoplexies or hemorrhages especially on the brain or other soft organs where the resistance is least, are liable to occur. When on the other hand the circulation is weak from atrophy or fatty degeneration of the heart, or from insufficiency of the valves there is a tendency to coldness of the extremities, and to passive congestions with their consequences :—serous effusions, dropsies, and difficult breathing. The imperfect supply of blood to the muscles of the extremities sometimes brings about an unsteadiness of gait in the hind limbs when the animal is trotted for a short distance and sometimes cramps supervene.

Continued coldness of the limbs, and a filling or thickening first of the hind limbs then of the fore and lastly of the chest and belly and of the skin beneath their dependent parts are useful indications.

Shortness of breath and inability to proceed when trotted or galloped on hard ground or when walked up hill, the animal being in fair condition, without fever or cough, but subject to cold extremities and a venous pulse in the jugulars, almost certainly indicates insufficiency of the auriculo-ventricular valves on the right side of the heart.

Vertigo megrims or giddiness may be caused by heart disease The horse without having sustained any pressure on the veins of the neck by the collar, and having had no previous symptom of brain disease suddenly reels in harness and perhaps falls. There are the cold and engorged limbs or a tendency to their engorgement as in the former case. The attacks recur, when the horse is put to the same exertion, and he proves utterly worthless. In such cases a careful examination of the pulse and heart sounds will complete the chain of evidence.

An almost constant feature of chronic heart disease is a condition of dulness, sluggishness, and in many cases, curiously enough, a tendency to lay on fat, so that although the patient is unfit to work, he appears to enjoy excellent general health to which a period is only put by sudden death.

Affections of the heart are primarily divisible into *functional* and *structural* disorders.

PALPITATIONS.

Convulsive contraction of the heart, functional or structural diagnostic features of these. Significance of the functional disorder, genera most liable. Treatment, quiet, heart tonic, digitalis, correct other disorders.

These consist in a sudden violent and convulsive beating of the heart, not connected with any appreciable structural disease. They differ chiefly from the palpitations of organic disease of the heart in the absence of any apparent local change to account for their occurrence. The following table from Bellingham furnishes a number of criteria equally valuable in the lower animals as in man.

PALPITATION DEPENDENT ON ORGANIC DISEASE OF THE HEART.	PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART.
1. Palpitation usually comes on slowly and gradually.	1. Palpitation usually sets in suddenly.
2. Palpitation constant, though more marked at one period than at another.	2. Palpitation not constant, having perfect intermissions.
3. Impulse of the heart usually stronger than natural, sometimes remarkably increased heaving and prolonged; at others irregular and unequal.	3. Impulse neither heaving nor prolonged; often abrupt knocking and circumscribed, and accompanied by a fluttering sensation (visible jerking or lifting) in the precordial region or epigastrium (flank and abdomen).
4. Palpitation often accompanied by the auscultatory signs of diseased valves.	4. Auscultatory signs of diseased valves absent; bellows sound often present in the large arteries and a continuous murmur in the veins.
5. Rythm of the heart regular, irregular, or intermittent; its action not necessarily quickened.	5. Rythm of heart usually regular, sometimes intermittent; its action generally more rapid than natural.
6. Mucous membranes often reddened and congested; dropsy of hind limbs common.	6. Mucous membranes generally pale; dropsy of hind limbs rare.
7. Palpitation increased by exercise, by stimulants and tonics, etc.; relieved by rest and frequently also by local and general bleeding and an antiphlogistic regimen.	7. Palpitation increased by close confinement; by local and general bleedings, etc.; relieved by moderate exercise and by stimulants and tonics, particularly the preparations of iron.

Palpitations in the lower animals not dependent on any structural disease of the heart are usually due to some violent mental emotion, such as fear or joy. The author once possessed a fast and clever cob, having no sign of organic disease, and equal to the severest work on the road without showing signs of exhaustion, but which nevertheless was affected by palpitation when threatened with a cane in his stall, or if he had been guilty of some wilful misdemeanor for which he dreaded punishment. In such cases the heart's action was accelerated and the beating could be heard loudly for a distance of several yards. They are especially common in dogs under strong mental emotion (joy, fear), and in dogs and pigs suffering from digestive disorder (worms) or chest diseases.

Percivall collects a series of cases from the *Veterinarian* under the head of *Spasm of the Diaphragm*, a diagnosis originating in the jerking movement of the flank, a symptom which, as is shown in the above table, is common to man as well. Leblanc equally collects cases from the French veterinary journals and acknowledges their true character. One of these observed by Coulbeaux is thus described: "The respiratory movements are interrupted by a violent lifting of the flanks, confined to the upper part, and so intense as to be appreciated by the hand as well as the eye. The lifting of the flank which is limited to a few square inches of surface precisely in the hollow of this region, is perfectly isochronous with the beats of the heart, which cannot be appreciated but by the ear."

A case reported by Levrat at greater length may also be given. The subject a mare, fifteen years old, and kept for hire, was attacked without any known cause. "There was a jerking of the whole body produced by the extraordinary force of the beats of the heart which struck violently against the back towards the upper ends of the first asternal ribs on the left side. The shock is very distinct and does not appear to be due to any tumor interposed between the heart and the ribs; its force is such that it causes a movement of the whole body, which can be seen at a great distance. The beats of the heart very regular, numbering fifty per minute, were heterochronous with the pulsations of the submaxillary artery, which are of the same number." (The pulsation was evidently retarded). "The

pulse was soft and regular. The breathing was peculiar. On approaching the ear to the nostrils the animal is heard to make three successive inspirations which coincide with the beats of the heart; each of these inspirations is followed by an expiration so short and feeble that the expired air can not be felt; the fourth is followed by full expiration sustained during three beats of the heart." This form of respiration was continued without intermission for an entire day.

In three cases observed at the Lyons Veterinary School the disease continued for eight days. In all these and twenty more observed by Leblanc, the patients invariably recovered. The steady persistence of the disease for several days and the subsequent complete recovery under the unaided action of digitalis would seem to warrant the conclusion that such cases were really accompaniments of structural diseases of the heart and not mere functional disorders. Even inflammation of the lining membrane of the heart often exists without any obvious fever or other manifest symptom of illness, and in the dissecting rooms of medical schools nothing is more common than to find traces of pre-existing heart disease in patients whose whole life had been passed without the suspicion of such a malady.

A number of such cases observed in England and on the European Continent are adduced to prove spasm of the diaphragm or of the abdominal muscles, (Delafond), and great importance is attached to the fact that the convulsive movements of the abdomen and loins are heterochronous with the beats of the heart. This lack of exact coincidence however does not seem to amount to more than a perceptible delay after the heart beat, just as the maxillary pulsation is delayed in case of aneurism of the aorta. This has been my own experience with such cases. The flank movements have been equal in number to the heart beats or have corresponded to certain beats in the heart cycle, and have been perceptibly retarded in accordance with the necessity for time for the transmission of the blood wave along the posterior aorta and the development of the reflex action which set the phrenic and abdominal muscles in motion. We must of course accept the convulsive action of the phrenic and abdominal muscles, only it would seem that each such movement has its starting point in the contraction of the heart. In cases that show no relation in number

nor succession with the heart beats, we can freely acknowledge a neurosis starting at a point different from the heart, but in all examples which are manifestly connected with heart movements, and readily curable by the heart-tonic digitalis, the recognition of the cardiac derangement as an initial factor is sound alike in pathology and practice.

Nervous and purely functional palpitations are probably confined to cases in which they are manifested at intervals appearing under the influence of some transient excitement, and continuing only for a few minutes at a time.

In the anæmic or bloodless condition the palpitation of weakness is often observed under the slightest exciting cause. It is then associated with a pallid state of the visible mucous membranes, a weak, trickling pulse and a blowing murmur in the larger veins.

Palpitations are much more frequent in pigs and dogs than in horses.

Treatment. Quietness and avoidance of all excitement are first to be secured, then the action of the heart is to be calmed and regulated by the use of digitalis. To the horse 15 to 30 grains of the powered leaves may be given thrice daily, and to the dog from two to four grains at equal intervals. When the disease is associated with anæmia this agent may still be used in combination with the various tonic remedies recommended, but in the smallest doses only as the heart is usually morbidly sensitive to external influences. Chloral hydrate has been used with success. Bromides, valerian, and belladonna may be tried in obstinate cases. Any other deviation from a healthy condition must be noted and corrected, especially any disordered condition of the stomach or bowels.

ANGINA PECTORIS. BREAST PANG.

W. Williams describes a case of illness in the horse to which he gives this name. When standing idle he had twitchings of the pectoral muscles, and when exercised these and adjacent muscles became violently convulsed, the left fore limb being alternately fixed by spasm, and paralyzed so that it was useless and the animal fell if compelled to move. There was "venous pulse, great irregularity of the heart's action, a loud cooing or blowing sound and strong impulse indicative of hypertrophy and a want of correspondence between the cardiac energy and feeble pulse." It was unfortunate that the murmur was not associated by the observer with a particular heart sound, and with the right or left side of the heart, and that hypertrophy was not diagnosed by percussion, since the case can be of little value as it stands.

Breast pang is usually associated with some disease of the heart: obstruction (usually calcification) of the coronary arteries, insufficiency of the aortic valves, calcareous degeneration of the aorta, aortic aneurism, or fatty degeneration of the heart. Loomis, basing his view on dissections made by himself and others, resolves all of these into concurrent ischæmia of the heart, the circulation in the coronary arteries being seriously interfered with. "That the sudden withdrawal of a supply of blood to a part may occasion neuralgia is shown by the intense pain in the limb which directly follows embolism of the femoral artery. Moreover general anæmia, as is well known, favors the recurrence of neuralgia in various situations." Inability of the heart to propel the blood is to be explained in the same way.

The difficulty of endorsing Williams' diagnosis lies in the fact that the disease, so far as it is a distinct disease, is functional and manifested by pain, the nature of which can only be inferred in the case of the lower animals (not by spasms of the pectoral muscles), and that hypertrophy of the heart is not likely to be present in case of insufficiency of blood supply to its walls.

In true angina pectoris of the horse, treatment is useless. Absolute rest is a prime requisite, and anodynes, stimulants, heart tonics, and nerve tonics are indicated. But the horse at rest with no prospect of final recovery is simply a source of expense.

FUNCTIONAL IRREGULARITY IN THE RYTHM OF THE HEART.

Associated or not with palpitation, irregularity in the force or frequency of the heart-beats is sometimes met with at intervals or independently of any further indication of structural disease. Particularly in the greyhound and certain other breeds of dogs the temporary occurrence of intermitting action of the heart is a frequent though a very transient condition. It may be excited by some emotion or excitement such as the attentions of the owner, or the straining anxiety in the immediate anticipation of the chase. Here again digitalis is pronounced the great panacea though it need not be resorted to unless the habit interferes with the usefulness of the animal. If in any degree dependent on weakness, that must be counteracted by a systematic tonic treatment.

CONGENITAL MALFORMATIONS AND DISPLACEMENTS OF THE HEART.

Ectopia Cordis. Cyanosis, pervious foramen ovale. Symptoms, blue mucosæ, coldness, staring coat, unthriftiness. Unequal to exertion, palpitations, murmur before the first heart sound. Obstructed circulation in the lungs as a cause of cyanosis, cyanosis as a cause of lung disease.

These have been much less frequently observed in the lower animals than in man. The anomalies observed in mammals include the following: The displacement of the heart to the right side of the chest; displacement entirely out of the chest (ectopia cordis); permanent communication between the right and left auricles (cyanosis); entire absence of heart; two hearts; one common ventricle communicating with two auricles as in reptiles; three ventricles; only one auricle; absence of one or several valves; absence of the pericardium; variations in the mode of connection of the heart and large vessels, etc.

Displacements of the heart have been especially studied by Hering on calves. The breast bone remained as originally developed in two lateral halves, and the heart remained outside connected with the interior of the chest only by its large vessels. The heart thus exposed and covered only by its investing membrane (pericardium) afforded an excellent opportunity to study its action, of which Hering freely availed himself. Animals affected in this way survived their birth but a very short time. An approach to this condition was thirty years ago made familiar to the medical world in the person of M. Gouz, a German mechanic, the movements of whose heart could be easily watched through a fissure in the breast bone.

Permanent communication between the two auricles. Pervious foramen ovale. Cyanosis. Previous to birth there is an opening between the right and left auricle, allowing the blood to flow from the former into the latter in place of ,as in after life, descending into the right ventricle and thence circulating through the lungs. At birth this is contracted, and in a few days is completely closed in accordance with the new life, which demands that all blood must circulate through the lungs in order to

its aeration. Sometimes this fails to be effected, and venous blood from the right side of the heart continues to mix with arterial in the left, deteriorating it in quality and unfitting it for nutrition, secretion, calorification, and other essential processes. The semi-venous blood circulating in the arteries gives a bluish hue to the visible mucous membrane, hence the name of *the blue disease*. This blood is unfit for sustaining the vital changes essential to the production of animal heat, so that the animal suffers from coldness of the surface and extremities, staring coat and general unthrifty appearance. Such subjects grow badly, and refuse to lay on flesh, but are said to arrive at maturity in some instances and to have their imperfection recognized only because of the short breathing, and irregular heart's action when subjected to exertion. A heart murmur preceding the first sound of health is usually present, as in anæmia.

Drs. Abernethy and Wardrop draw attention to the frequency of previous *foramen ovale* in the human subject in connection with pulmonary consumption, and opine that it is reopened as a consequence of this disease. The coincidence has not been observed in the lower animals, though if it were found to exist the question would arise whether the deterioration of the blood and general health in open *foramen ovale* did not also favor the deposit of tubercle in the lungs. When from deficient ventilation the atmosphere and blood become impregnated with carbonic dioxide the production of tubercle in man or in animals is correspondingly frequent.

The subjects of previous foramen ovale die young or prove worthless when they arrive at maturity. Nothing can be done to ameliorate the condition.

HYPERTROPHY OF THE HEART.

Simple, eccentric, concentric. Ventricles chiefly affected. Causes, increased functional activity, from obstruction to the circulation, or continued extra exertion. Right ventricular hypertrophy-obstruction in the pulmonary circulation; left ventricular hypertrophy-obstruction in the systemic. Auricular hypertrophy-insufficiency of the auriculo-ventricular valves. Pericarditis as a cause. Abnormal weights. Symptoms, beats more forcible and prolonged, 1st sound low, prolonged, 2nd sound clear, often doubled, increased dulness on percussion, diagnostic signs of hypertrophy, dilatation and a combination of the two. Simple hypertrophy rarely dangerous, with dilatation grave, threatens congestions and apoplexies. Treatment, rest, laxatives, sedatives, in irregular heart action digitalis, arsenic.

An enlargement of the heart from increase of its muscular substance is by no means uncommon in the horse. It may exist without any change in the capacity of the cavities of the heart (simple hypertrophy) or it may be associated with dilatation of one or more of these cavities (hypertrophy with dilatation;—excentric hypertrophy). A third variety has been described in which the capacity of the cavities is decreased but Cruveilhier and Budd have satisfactorily shown the nonexistence of this condition except as a congenital deformity.

It is in the ventricles that the increase is chiefly observed, the reason of which is to be found in the causes of the malady. These usually consist in some obstruction to the circulation such as chronic congestions in the lungs or elsewhere, rupture of air cells in the lungs, tuberculous and other abnormal deposits in the chest and elsewhere, tumors which by their position interfere with the circulation through the larger vessels, and the like. Where by some such cause the blood is impeded in its outward course, one or both ventricles are called upon to contract more vigorously to force a sufficient amount of blood onward and in accordance with the inherent adaptability of the animal economy, there takes place an increase of the muscular walls of the ventricle proportionate to the required energy of the contractions. The condition then is essentially due to a more active nutrition and growth of the muscular substance and finds its exact parallels in the well-de-

veloped legs of the ballet dancer or the brawny arm of the blacksmith. All alike occur in accordance with a general law that whenever there is habitually demanded of any organ an unusual activity of function, which stimulates without exhausting its power, nature adds to the active element of such organ till the required labor can be accomplished without the overwork of any particular part.

Keeping this in view we can easily explain the increase of one part of the heart without immediate implication of another. The ventricles are more commonly enlarged than the auricles because upon them devolves the work of overcoming the obstruction, whether this exists in the lungs or the system at large. The auricles fulfill little more than a passive function in receiving the blood from the veins during the contraction of the ventricles and allowing it to pass down into these when their relaxation takes place. The closure of the auriculo-ventricular valves during the ventricular contraction protects the auricles from the internal tension to which the lower part of the heart is subjected and thus all tendency to increase is obviated.

The hypertrophied part corresponds to the locality of the obstruction. If it exists in the lungs (heaves, consumption, hepatisation, chronic bronchitis), pulmonary artery, its valves at its origin from the heart, or if it consists in contraction of that orifice, the enlargement takes place primarily in the right ventricle, the right auricle remaining unchanged so long as the auriculo-ventricular valves act perfectly. The ventricle, however, tends to dilate as well as enlarge in thickness of walls, and as soon as this dilatation has proceeded so far as to widen the orifice between the auricle and ventricle and render its valves insufficient, the auricle also begins to dilate and its walls often increase in thickness. But the vicious chain does not end here. Should the animal survive and the original obstruction persist, the veins throughout the system become habitually congested because of the reflux of blood from the right auricle and ventricle, dropsies appear in different parts, the congestion of the veins is continued through the capillary blood-vessels to the arteries, the difficulty of propelling the blood comes to be experienced by the left ventricle and a corresponding series of morbid changes taking place on that side, as have already ensued on the right, the vicious

circle is soon completed, and the entire organ becomes diseased, each constituent part of the organ operating injuriously on that which preceded it in the track of the circulation, and every new change forming but a stepping stone to a more dangerous modification.

On the other hand the obstruction may exist in the general circulation, on the course of the aorta, or its branches, in its valves at its origin from the heart, or in the narrowing of its orifice. Then the increase takes place first in the left ventricle, is propagated to the left auricle, leads to congestion of the veins, capillaries and arteries of the lungs, and lastly to disease of the right side of the heart. Here there is a different starting point, but the progress of the disease-changes in a direction opposed to the course of the circulation is the same.

The disease may, however, begin with the auricles, owing to disease of the auriculo-ventricular valves impeding the flow of blood into the ventricle, or to simple narrowing of the auriculo-ventricular opening. The auricle is then primarily enlarged, the corresponding veins congested, this is propagated to the capillaries and arteries, and lastly the ventricle on the opposite side of the heart is involved. This is chiefly seen with fibrinous deposits on the valves or in the case of polypus hanging into the auriculo-ventricular opening. Aneurisms, embolisms, neoplasms, atheroma and calcic degeneration of the arterial walls may be effective factors.

In addition to these causes Bouilaud and Leblanc attach a high importance to chronic inflammations of the serous membranes, which by reason of the contiguity of the latter to the muscular structure bring about a more abundant circulation in this and an increased nutrition. Another cause is unintermitting hard work which necessitates excessive exertion of the heart, to supply blood more freely to the muscular system and the lungs. Many hunters suffer from this affection it is believed because of their extraordinary exertions. The stallion Helenus had a heart of 14 lbs.

Weight of the heart. The heart in the horse which rarely weighs over 9 lbs. is increased from 10 lbs. to 14 lbs. in this disease and in one case in a cart horse, recorded by Stephenson in the Veterinarian for 1861, it is said to have reached 32 lbs.

Stephenson probably weighed the heart while filled with blood. A diseased heart weighed in this way by Thomson amounted to 34 lbs., one by Gerlach, 19 lbs., an ox's heart by Herran 36 lbs. In Stephenson's case there was further an extraordinary dilatation of the anterior vena cara. Haycock (Veterinarian, 1850), records a case in which though the heart only weighed 10 lbs. 8 oz., yet the walls of the ventricles were double the normal thickness, those of the left being 2¼ inches while those of the right were 1 inch. An estimate from the thickness of the walls, it must be borne in mind, is not so satisfactory as the absolute weight taken after the removal of the large vessels, the superfluous fat and the contained blood.

The usual coincidence of other complaints supports the statement that it is mostly due to obstruction to the circulation. A few cases will illustrate. Dyer reports the case of a hunter in which with general hypertrophy and dilatation of the right auricle, the pulmonary artery was so large as to admit the fist. (Veterinarian, 1861). Halloway relates a case in which there were extensive internal deposits of melanotic material, especially in the mesenteric glands, liver, spleen, and kidneys (Veterinarian, 1850). Haycock records a case with thickening of the auriculo ventricular valves on the right side, and of several of their tendinous cords, also a diseased liver which had ruptured before death (Vetenarian, 1850). Percivall publishes a case associated with pleurisy and rheumatism (Veterinarian, 1858). Henderson furnishes a case associated with diseased right auriculo-ventricular valves and enlarged liver weighing 55 lbs. (Veterinarian, 1847.)

Symptoms. In simple hypertrophy the heart beats are more forcible and prolonged so that the period of silence or rest is shortened. This is due to the greater length of time taken up in the contraction of the ventricles. For the same reason the pulse which may or may not be accelerated, irregular or intermittent, is full and rolling or as it were prolonged. The first sound of the heart is prolonged and low or muffled, sometimes almost inaudible while the second is unnaturally loud. Sometimes when one ventricle only is enlarged that may complete its contraction later than the other and the second sound is repeated as in the syllables *lub—tip tip*. A duplication of the first sound only is less common. If the sounds are heard over a greater extent of the chest's

surface than is natural, the lungs being healthy, it is probably due to hypertrophy of the heart. If very clear on the right side they indicate increase of the right ventricle. The heart's impulse is usually strong and may be felt on both sides, and it may be over the whole chest.

Percussion usually shows a more extended dulness in the region of the heart but the blows must be pretty forcible to bring out the deeper resonance, otherwise it will come only from the thin layer of lung. These results are of the greatest value in the dog.

The pulse is usually regular and if excited to irregularity and intermission quickly returns to its natural state when the patient is left at rest.

As hypertrophy is usually associated with dilatation of the heart the following table abridged and modified from Dr. Walshe will prove valuable by presenting side by side the signs indicating hypertrophy with and without dilatation, and simple dilatation.

TABLE CONTRASTING THE MAIN SYMPTOMS OF HYPERTROPHY AND DILATATION.

A. General Physical Signs.

SIMPLE HYPERTROPHY.	HYPERTROPHY WITH DILATATION.	SIMPLE DILATATION.
Heart's impulse slow and heaving as if pressing steadily against an obstacle—in rythm regular, in force unequal.	Force increased, sharper, more knocking, may impart a shake to the body. May be felt on the right side.	Impulse conveys a feeble undulatory sensation ; force of successive beats unequal ; rythm irregular.
First sound is dull, muffled, prolonged and weakened almost to extinction. Second sound full and clanging ; period of silence shortened.	Sounds gain greatly in loudness and extent of transmission, especially if the valves are not thickened.	First sound short, abrupt and clear. Second sound not specially affected.
Murmur with the first sound present at one time and absent at another.	Murmur with the first sound may be present, from altered direction of the orifice of the aorta.	Murmur with the first sound from insufficiency of the auriculo-ventricular valves.

B. General Functional Symptoms.

SIMPLE HYPERTROPHY.	HYPERTROPHY WITH DILATATION.	SIMPLE DILATATION.
Strength unimpaired. Power of continued exertion (especially uphill) limited by shortness of breath.	Strength tends to become impaired.	Strength fails.
Visible mucous membranes healthy or of a bright red.	Purpleness and lividity of the mucous membranes proportionate to the valvular or pulmonary obstruction.	Lividity of the mucous membranes. Dropsical effusions of the limbs and other dependent parts which pit on pressure.
Difficulty of breathing occasional.	Difficulty of breathing occurs in paroxysms.	Difficulty of breathing great and constant, with occasional aggravations.
Pulse full, strong, firm, tense, resisting and prolonged without jerk or thrill.	Fullness of pulse continues but strength and power of resistance lost.	Pulse small and feeble, much later than the heart beat. Regular or feeble, fluttering and irregular. Venous pulse in the jugulars.
Rarely and never rapidly the direct cause of death.	Indirectly and more or less rapidly fatal.	Palpitation frequent. Faintness occurs from time to time, and may lapse into fainting and sudden death.

Pure hypertrophy rarely implies imminent danger unless dependent on some pre-existing structural disease which impedes the freedom of the circulation. If excessive, however, or if associated with dilatation the animal is short-winded and unfit for all but the slowest work. It predisposes to congestion or apoplexy of the lungs when its seat is the right ventricle, and to congestions and hemorrhage in other parts of the system, brain, kidney, lungs, liver, bowels, if in the left.

Asthma (dogs), heaves (horses), emphysema and tuberculosis in cattle are occasional complications attended by grave symptoms.

Treatment. In advanced cases and such as are dependent on irremovable structural changes in the lungs or elsewhere no treatment is of any avail. In recent and uncomplicated cases in the horse and cow and in some more advanced conditions in other animals,

not used for work, a palliative treatment may be profitably adopted. This consists in a nitrogenous restricted and gently laxative diet, perfect rest in fattening oxen and other animals, or in the horse moderate and carefully regulated work, and as a medicament the use of digitalis or aconite. No known remedy has any power to directly check the growth of the heart and the utmost that can be expected of these agents is to lessen the activity of the heart's action and retard its growth. Digitalis may be given as recommended for palpitation, or aconite in the form of tincture 20 drops for horses and cattle and 1 to 2 drops for dogs, repeated four times daily. Strophanthus may replace digitalis. When depletion seems advisable purgatives or diuretics should be given as appears most applicable to the particular case. Iodide of potassium has been strongly recommended,

When extreme dilatation exists with the hypertrophy, sedatives should be given cautiously and their effects carefully watched as the heart is often dangerously susceptible to depressing influences. When the disease has advanced so far as to cause abundant dropsical effusions it is futile to resort to treatment as amelioration can rarely be looked for, not even to the extent of allowing an animal to be fattened.

The value of arsenic in most cases of broken wind (heaves) has suggested the inquiry whether it does not operate directly on the heart. Leblanc who advances this query might have quoted in explanation the known power of arsenic to retard and arrest tissue change, with its natural consequences, the diminished amount of effete matter thrown into the blood in any given time, and the lessened necessity for an active circulation to supply any great waste of structure. It may benefit such cases in this way but does so probably to a far greater extent by an influence on the nervous function analogous to its action in neuralgia and other purely nervous disorders. Dilatation of the heart which usually exists in *heaves* is usually benefited by tonics which like arsenic are destitute of stimulating properties.

ATROPHY.

Simple, eccentric, concentric. Usually eccentric. Causes, effusion in pericardium, obstruction of coronary arteries, by false membranes, etc., general inanition. Symptoms, beats weak, sounds loud, clear, decreased area of dulness on percussion, pulse slow, weak, under excitement unequal, irregular, intermittent with palpitation, dropsy of limbs, etc., murmur with 1st sound. Treatment only in early stages by removal of the cause.

The loss of substance in the muscular walls of the heart is either *simple* when there is no change in the capacity of its different cavities :—**eccentric** when the chambers of the heart are enlarged ; or **concentric** when these chambers are reduced in size. Like hypertrophy it may affect the walls of one chamber to the exclusion of the others.

Atrophy is much less frequent in the lower animals than hypertrophy and in nearly all cases on record it was associated with dilatation.

The *causes* are not always very evident. Effusion into the pericardium is one of the most frequent, the compression of the heart impairing its nutrition and decreasing its size. Especially is it hurtful when several layers of false membranes deposited on the surface of the heart become organized, preventing its sufficient dilatation and compressing its nutrient blood-vessels. A case of this kind in a dog occurred to Leblanc ; the right auriculo-ventricular opening was surrounded by thick organized layers of false membranes which by their contraction had largely diminished the opening and even pressed on the coronary artery cutting off to a great extent the supply of blood to the walls of the ventricle. Another alleged cause is a prolonged insufficient nourishment to the entire body. Leblanc has also observed this in dogs the subjects of long continued wasting maladies.

Symptoms. In pure atrophy these are the opposite of those seen in hypertrophy. The beats of the heart are weak or inappreciable to the hand placed on the side of the chest behind the left elbow. The sounds of the heart are loud and clear, their intensity being proportionate to the thinning of the walls and the dilatation of the chambers. Percussion so far as it can be made effectual, which is chiefly in dogs, shows a diminished area of dul-

ness. The pulse is slow, weak, or indistinct, compressible, becoming accelerated, unequal, irregular, and intermittent when the patient is excited. Palpitation is frequent, breathing is difficult or easily embarrassed and there is a tendency to dropsy of the limbs and dependent parts. These symptoms are usually associated with considerable prostration and depression.

These are often complicated by symptoms of valvular disease or dilatation.

Atrophy progresses slowly and rarely causes death in the earlier stages. In its advanced stages when dropsy has supervened little can be done even in its mitigation. In the earliest stages only can good be done by employing measures calculated to remove its causes and thus put a stop to its progress.

DILATATION OF THE HEART.

Result of obstruction to circulation. In right ventricle usually. In auricle from narrow auriculo-ventricular opening. Pure dilatation from sudden extreme blood pressure as in inflammations of the lungs. In fat cattle from fatty obstructions around the heart and great vessels. Weakness of cardiac muscles in fatty degeneration, fevers, debility, etc. Symptoms, dyspnœa under slight exertion, unsteady walk, cold, dropsical limbs, venous pulse, pulse small, weak, irregular, intermittent, with palpitations. Treatment, in early stages arrest the causes, arsenic, digitalis, fatten for butcher.

Dilatation of the right cavities of the heart is one of the most common heart diseases of the horse. It is an almost constant condition in advanced broken wind, and is a frequent concomitant of hypertrophy and an occasional one of atrophy of the heart. Its usual direct cause is some obstacle to the free escape of blood from the cavity affected. Thus in broken wind the difficulty of the circulation through the lungs causes accumulation in the pulmonary artery and right ventricle of the heart, the walls of which are distended because of the unwonted internal pressure. When the dilatation of this ventricle reaches a certain stage the auriculo-ventricular opening is equally widened, the valves become insufficient to close it and the right auricle and venæ cavæ par-

ticipate in turn in the internal pressure and dilatation. The right ventricle is more often affected than the left, because of the greater frequency of obstruction in the circulation through the lungs than in that through the general system, and because of the thinness of its walls which more readily give way under internal pressure. Dilatation may result from disease of the great arteries, from diminution of their calibre by the pressure of tumours, or by narrowing of their openings at the heart, whether as the result of diseased valves or other morbid condition. As affecting the auricles primarily its usual cause is narrowing of the auriculo-ventricular opening from some abnormal deposit. The extreme thinness of the walls of the auricles allows these to give way under internal pressure even much more readily than the right ventricle.

The causes it will be seen are similar to those inducing hypertrophy, and hence the frequent coexistence of the two. Pure dilatation occurs especially when internal pressure takes place suddenly and to excess, and while the nutritive functions are to a great extent in abeyance. Such conditions are found in acute inflammations of the respiratory organs, or of the inner or outer membranes of the heart, and the rapid deposit in the lungs of tubercles or other abnormal material.

Dilatation of the right side of the heart is a common complaint in overfed cattle, and is apparently due to the diminished power of resistance in the walls of the heart, the muscular substance of which is partly replaced by fatty granules, and to the obstruction offered to the circulation by the extraordinary accumulation of fat around the base of the heart and the commencement of the large blood vessels. Though a diseased condition this rarely shortens life or interferes with the uses to which cattle are put.

The heart walls are similarly weakened and yield more readily to the internal blood pressure in endocarditis, myocarditis, pericarditis, high fever, infectious diseases, poisonings, anæmia, and debilitating diseases generally. Debility and incapacity to resist the blood pressure is the essential prerequisite to dilatation.

The *symptoms* which have been already enumerated in the table given under the head of *hypertrophy* are mainly these: Loss of appetite, spirit and endurance; faintness and difficulty of breathing on the slightest exertion; habitual coldness with a

tendency to dropsy of the extremities; loss of control over the extremities when walked or trotted far; venous pulsation in the jugulars; heart's impulse weak and undulatory or tremulous, or under exertion tumultuous or palpitating; murmur often present with the first sound; the first or more commonly the second sound may be doubled; pulse small, weak, irregular, and often intermittent, and frequently livid spots in the nasal mucous membrane. Paroxysms of unsteady gait from irregular circulation in the brain is frequent, and Dyer asserts that he has repeatedly seen blindness as a result of this condition.

In *treatment* the main purpose should be to put a stop to the cause of the disease before it has been developed to a dangerous extent. When the malady is manifested by the symptoms above enumerated the subject is rendered permanently unfit for service and will probably die suddenly under some slight exertion. Fattening animals in a condition of quietude will often lay on flesh for an indefinite length of time notwithstanding that the heart is considerably dilated. (See note on digitalis, strophanthus and arsenious acid in dilated heart, under the head of *hypertrophy*). To relieve the asthmatic attacks attending on an overtaxed heart Zuill strongly recommends the combination of iodide of potassium, digitalis, nux vomica and coca. But heart tonics are often much more affective after the bowels and portal system have been unloaded by a laxative.

INFLAMMATIONS IN THE HEART.

These are among the most common diseases of this organ and moreover lead to many of the changes in structure to be hereafter noticed so that it is convenient to treat of them here. According to their relative frequency they may be ranged:—1st. Inflammation of the external covering of the heart—pericarditis; 2d. Inflammation of the internal lining membrane of the heart—endocarditis; and 3d. Inflammation of the muscular substance of the heart—carditis.

PERICARDITIS.

Definition. Frequency in different genera. Causes, rheumatic, traumatism, extension from pleurisy. Unwholesome buildings and localities, debility. Symptoms, chill, reaction, pleuritic symptoms, hyperthermia, tenderness behind left elbow only, friction sound synchronous with heart beat, later it is lost and heart sounds are muffled, increased area of dulness on percussion, oppressed breathing, venous pulse, patient statant, little fever in chronic cases. Traumatism from the stomach, digestive disorder, grunting, dropsy under the sternum with little fever at first. Lesions, as in pleurisy, obliteration of pericardial sac. In traumatism from stomach the foreign body is formed in the cardiac end of a band of lymph extending to the reticulum. Treatment, in chill, after reaction, medicinal measures as in pleurisy, local applications to the region of the heart. Paracentesis, insertion of needle, antiseptic precautions. Chronic pericarditis in oxen.

Definition. Inflammation of the strong fibro-serous sac in which the heart is contained and which is reflected on the muscular substance of that organ so as to form its external covering.

This is the most common inflammatory disease of the heart and has been met with more frequently in horses and cattle than in the smaller quadrupeds.

Causes.. It frequently coincides with or follows other diseases such as *influenza*, *pleuro-pneumonia* and above all *rheumatism*. In cattle and goats wounds from sharp pointed bodies, (needles, pins, nails, etc.), which have been swallowed with the food and have passed through the walls of the second stomach, the diaphragm and pleura to the heart constitute a frequent cause in cattle. The pericardium has been punctured by a fractured rib and has been implicated in inflammation attendant on an abscess or other lesion in the walls of the chest. Besides these the general influences which cause uncomplicated attacks of the disease are the same as those producing pleurisy, peritonitis, rheumatism and inflammation of serous membranes generally. These are sudden changes from heat to cold, cold winds, cold draughts, drenching, chilling rains in animals already overheated and exhausted, or prolonged exposure in severe weather, in low states of the system. Leblanc justly remarks that "with the morbid influences which appear specific, there often coincide intemperate seasons,

badly arranged buildings, a want of sufficient attention to the conditions of health, and in the case of herbivora, wet, cold, and badly exposed pastures." In other words whatever deteriorates the health and vitality predisposes.

Symptoms. These are less characteristic than in man owing to the smaller portion of the heart exposed, but they are usually marked enough to permit a recognition of the disease. *Acute form.* The affection is ushered in by chill, general fever, hyperthermia, (103° to 104°), staring coat, hot, dry mouth, dilated nostrils, excited, difficult breathing, double lifting of the flank with each expiration, the existence of a prominent ridge from the lower end of the last ribs along the flank to the outer angle of the hip bone, as in pleurisy, pinched, anxious expression of countenance, fixed eyes, accelerated, full, hard and often wiry pulse, and tenderness when the ribs behind the left elbow are pinched or struck. The same tenderness is noticed particularly in the ox and smaller quadrupeds when pressure or compression is made beneath the breast bone. Auscultation over the lower ends of the fifth and sixth ribs and their cartilages detects a friction or rubbing sound in the early stages and until liquid has been thrown out into the pericardial sac. This sound may be at first the finest possible creaking, afterward increasing to a distinct rubbing, is synchronous with the beat of the heart, and usually with the first sound. It is distinguished from the friction sound of pleurisy in occurring rythmically with the sounds of the heart and not with those of breathing, and from sounds produced in the interior of the heart by its absence when auscultation is made over the carotid or other large artery. This friction sound is lost when serous effusion takes place into the pericardium, but reappears when the liquid is absorbed in the process of recovery. Until effusion takes place the impulse of the heart is strong, often irregular, in force, and sometimes accompanied by a purring tremor or, according to Leblanc, a metallic tinkle.

When effusion has taken place the pulse is weaker and softer, irregular or intermittent, the impulse of the heart is weaker, the friction sound is lost, and the area of dulness corresponding to the heart is increased. Percussion shows it to extend higher than three inches above the breast bone in the horse and more than two or two and a half inches transversely. It is distinguished from

the effusion of pleurisy in this, that the dulness is confined to the anterior part of the chest, having the outline of an inverted cone, and does not extend backward along a horizontal line, and, in solipedes, in not showing equally on both sides. In the smaller animals it may be distinguished by not always occupying the dependent part of the chest when the animal is placed in different positions. As the effusion increases, the heart's sounds, previously strong, become first muffled, then more and more distant until they may become altogether imperceptible. The difficulty and oppression of the breathing increases, the nose is protruded, the eyes more rigidly fixed, and the face more haggard ; a venous pulse, apparently due to the compression of the heart and large veins by the fluid, is seen in the lower ends of the jugulars, and the animal obstinately stands as indeed the solipeds do all through the disease. At this advanced stage dropsies of the limbs, sheath, and other dependent parts of the body are frequent.

A painful cough is sometimes though by no means invariably present throughout the disease. Emaciation takes place rapidly and in the more acute cases death ensues in five to eight days. A fatal issue may be delayed until after three weeks or the affection may merge into a *chronic form*.

Chronic Pericarditis is sometimes seen in the *ox* without any preceding acute attack. This is manifested by the local symptoms without the accompanying acute fever. Along with a *slight* fever, there is the oppressed breathing aggravated by exertion, the weak irregular or intermittent pulse, the weak or distant heart sounds, the absence of respiratory sounds and the dullness on percussion over a space represented by an inverted cone at the anterior part of the chest on each side, the venous pulse in the neck and the general tendency to dropsy.

If the *pericarditis* has been the result of sharp pointed metallic bodies swallowed and afterwards making their way to the heart, it is sometimes preceded by eructations, tympany, difficulty in swallowing or in rumination, and by dropsy under the sternum, but more frequently the heart symptoms are the first to be noticed. It is not attended by the high fever of other pericarditis.

Post Mortem Appearances. These do not differ materially from those of pleurisy, to which accordingly the reader is referred. The effusions and false membranes are of course localized in the

sac of the pericardium. A frequent termination is a permanent adhesion of the pericardium throughout more or less of its extent to the surface of the heart. In cases of death the serous effusion is commonly colored with blood though mostly from a *post mortem* infiltration of blood from the congested lungs. The effusion has been known to measure fifteen litres in the horse. It may be purulent or combined with fœtid gases, particularly in traumatic cases. After mild attacks white patches (milk spots) are often left extending, it may be only through the pericardium and in other cases reaching into the muscular substance. At a less advanced stage the false membranes are yellow, with a rough or villous surface, they may be softened from fatty degeneration or they may be more or less completely calcified.

When the cause has been perforation by a metallic body, it will be found surrounded by exudate enveloping a canal or band extending to the diaphragm or stomach.

Treatment. Pericarditis often proves fatal but it is by no means invariably so in uncomplicated cases. There is especial danger when serous effusion is excessive, when it occurs in a weak and debilitated subject, or when it is complicated by pleurisy, influenza or rheumatism. The preliminary chill may be met by the measures advised for the rigor of pleurisy, but if the malady is developed other treatment is required. The medication is still essentially as for pleurisy, only the primary disease (rheumatism, influenza, pneumonia) must be specially attended to when such is present. Acute pain may be met by carefully graduated doses of opium or aconite and by the moist jacket or fomentations. Some employ icebags to soothe at once inflammation and pain and in the absence of rheumatism these may be resorted to. In the small animals leeches may be applied over the cardiac region. Dry cupping is a good alternative applicable to all.

An active purgative is demanded unless the affection is attended by a low type of fever or has occurred during the course of an epizootic disease (Horse 5 to 7 drachms aloes, cow 1 to 2 lbs. Epsom Salts, dog 1 oz. castor oil). After the walls of the chest have been well fomented they may be enveloped in a large mustard poultice which must be continued until a considerable effusion has taken place beneath the skin. To moderate and control the heart's action give digitalis (horse and ox ½ drachm, dog 2 to 4

grains) four times a day. After the purgative has acted an ounce of nitrate or acetate of potass may be given daily to the larger quadrupeds (1½ drachms to sheep and pigs, and 20 grains to dogs) in the drinking water. These agents together with the digitalis must be pushed to the largest doses when the effusion has taken place abundantly and when it threatens to dangerously interfere with the heart's action. Pilocarpin is a dernier resort, to be used with caution. In similar circumstances, ointment or tincture of iodine should be freely applied over the chest in the region of the heart. Mustard and other vesicants repeatedly applied often greatly hasten the reabsorption of the liquids.

From the first the animal must be warmly clothed and every means employed to obtain free circulation and warmth on the surface. The legs must be well rubbed and wound in warm flannel bandages, or this failing, may have mustard freely applied to them. Warm injections must be at the same time thrown into the rectum and will benefit by soliciting the action of the bowels as well as in raising the temperature of the surface generally. The food allowed should be warm mashes of wheat bran, boiled linseed and similar agents in small quantities.

If the amount of effusion threatens a fatal result, it may be drawn off by a cannula and trochar introduced between the cartilages of the fifth and sixth ribs, by a valvular wound and with antiseptic precaution (see hydrothorax), care being taken to avoid puncturing the heart itself.

The trochar or aspirator needle should be pushed in a direction upward and inward until resistance ceases or it is felt that the heart has been touched. A caoutchouc tube may now be attached to it and allowed to depend twelve or eighteen inches, and its lower end should be plunged in a weak solution of boric acid or other antiseptic. This avoids the entrance of air and insures against the introduction of ærial bacteria.

When the vital powers are being exhausted stimulants must be given to support the animal, combined with iodide of potassium. (See advice concerning the allied condition in *Hydrothorax*).

In the *chronic pericarditis of oxen* the fatality is greater. Treatment consists mainly in counterirritants and powerful diuretics employed in doses determined by the strength of the animal, and combined with stimulants and tonics as in the advanced stages of the acute disease.

In complicated forms of pericarditis attention must be given mainly to the constitutional affection, thus in *influenza* a stimulating and supporting treatment is demanded, and in *rheumatism* colchicum, acetate of potass, salicylate of soda, salol and similar agents must be freely administered, though not to the exclusion of counterirritants to the region of the heart, and other measures demanded by the heart diseases.

ENDOCARDITIS.

Definition. Psthology and lesions, congestion of the endocardium covering the valves, valves liable through friction and strain, exudation in or on the serosa rendering it opaque, coagula of fibrine on the surface, secondary endocarditis mycotic, microbes, changes in serosa, distortions and degenerations of valves. Symptoms, as in pericarditis, with violent heart impulse of varying force, clear metallic sound, blowing murmurs, weak pulse decreasing in force, irregular, intermittent, absence of local tenderness, no friction sound, no increase in area of dulness, if lesions are in right heart—venous pulse, venous congestion, dropsies. Valve lesions, in mitral valve—general heart symptoms and murmur with 1st heart sound, 2d sound may be repeated and exceptionally a venous pulse—in tricuspid valve—same with constant venous pulse, venous congestion and dropsy ; narrowing of the mitral orifice—general heart symptoms and blowing murmur before the 1st sound ; narrowing of the tricuspid orifice—same with murmur sometimes audible on the right side ; insufficiency of aortic valves—general heart symptoms and murmur with 2d heart sound, double rushing sound in arteries and delay of pulse beat at jaw ; lesions in pulmonary valves—same but without double rush in arteries, or delay of pulse beat at jaw. Loose coagula. Embolism. Causes, as in pericarditis and strain on valves, and poisons and microbes in the blood. Prognosis grave. Treatment, as in the early stages of pericarditis, antirheumatics and germicides more, and diuretics less desirable. For clots iodides, alkalies.

Definition. Inflammation of the serous membrane lining the chambers and covering the valves of the heart.

Pathology and Morbid Anatomy. The causes and symptoms will be better understood after the diseased conditions have been comprehended. The earliest changes are the reddening and thickening of the lining membrane of the heart but above all of that covering the valves. The valves are particularly exposed to in-

flammation by reason of the friction of the blood when violently forced through the narrow opening in excited conditions of the heart, by the strain thrown upon them from the violent contractions of the heart or the recoil of blood in the arteries, and by their susceptibility in common with all other fibrous structures to rheumatic inflammation. The redness is of the ramified or branching kind characteristic of inflammation, and is neither removable by washing the surface nor does it correspond in position with the colored portion only of a clot which the cavity in question may contain, as seen in bloodstaining occurring after death.

There is further exudation of plastic lymph into and beneath the serous membrane, rendering it opaque, white and thick, or on its surface forming granular elevations, and in the case of the valves becoming moulded into ridges or festoons by the mutual pressure of the different flaps on each other. The inflamed surfaces are further liable to be covered by masses of blood clot in successive layers, deposited by the action of the fibrinogenous matter developed in the inflamed part. These clots sometimes accumulate in considerable masses, firmly adherent to the heart's walls or valves by their attached surface, but soft and filamentous on their free aspect. These clots or polypi, as they have been called, are soft and loose on their free surface, and become firmer toward their points of attachment. In other words their consistency is in direct ratio to their age. If of old standing they are usually pale yellow or white and streaked with red, while if recent they are mostly red throughout. They vary in size from a thin film to a mass filling up nearly the entire cavity in which they are lodged, and as they frequently extend through the auriculo-ventricular openings or become applied against this or the opening of the great artery, they seriously and sometimes fatally interfere with the circulation. Leblanc asserts that large masses of this kind may be deposited in a few days or even hours, causing sudden deaths, and especially in dogs. He has found other circumstances than endocarditis to cause these fibrinous deposits, and especially the absorption of pus, or the sudden suppression of a long standing discharge, as in catarrh of the air-passages. If death does not immediately ensue, these fibrinous deposits may become vascular, as is the case with false membranes in the the pleuræ, becoming organized into fibrous tissue, or even de-

generating into calcareous matter, necrotic debris, or pus, several instances of which as occurring in horse and cow are on record.

These cases illustrate *endocarditis by infection* (*mycotic, malignant, or ulcerative endocarditis*), which occurs independently, or as an extension of a bacteridian disease, primarily localized elsewhere in the system. Thus it is a secondary lesion in infectious omphalitis, pneumonia, pleurisy, arthritis, abscess, pyæmia, etc. Beside the general lesions of endocarditis and a great tendency to molecular death of the new formations and the underlying tissues, there is the presence of specific germs which have been the occasion of the disease. Among these the staphylococcus pyogenes aureus, the streptococcus pyogenes, and the diplococcus pneumoniæ, have been particularly noted. In case the valves were already diseased, they become especially liable to be colonized by any such bacteria that may be circulating in the blood.

In the early stage there may be a mere swelling of the valves, with as yet a smooth, unbroken surface, but with enlargement and increase of the connective tissue cells, later fungous vegetations start out from the surface, and on these the fibrine of the blood is deposited in layers.

Besides the formation of clots on their surfaces other changes occur on the cardiac valves as the result of inflammation. The organization of the exuded lymph within and upon them leads to rigidity, loss of elasticity, unevenness of their surface, contraction and puckering so that they can no longer approximate to each other, but leave the orifice imperfectly closed. They may, moreover, have gristle or bone deposited in their substance. The osseous degeneration of such new products appears to be the most common cause of those ossifications of the heart, of which specimens are to be found in nearly all veterinary museums.

Chronic valve disease is thus found to be a common result of endocarditis, and from the obstacle presented to the flow of blood through the different cardiac orifices by the rigid, inelastic and distorted valves, hypertrophy of the heart frequently supervenes.

In our domestic quadrupeds ante-mortem clots and fibrinous polypi have been chiefly formed in the right side of the heart, and diseased valves in the left.

Symptoms. The general symptoms agree in many respects

with those of *pericarditis*. There are the same general symptoms of fever (temperature 102° to 106°), the same pinched, anxious countenance, the same shortness of breath and oppression when moved, the same violent heart's action, and the same rapid, excitable pulse tending to be irregular and intermittent. Among the more specific symptoms are a very violent impulse of the heart against the left side, varying in force, however, in successive beats; a metallic tinkling accompanying the impulse and sometimes heard at some little distance from the body, a blowing murmur as soon as the changes in the valves render them insufficient to close the orifices, and, if the obstruction exists on the right side, venous pulse, general venous congestion, and dropsical swellings.

The pulse may at first have considerable force but, as insufficiency of the valves ensues, it becomes small and weak, its weakness forming a most marked contrast to the violence of the heart's impulse against the side. The irregularity and intermission of the pulse is to be ascribed at first to the impaired nervous energy of the heart though later it is often due to the obstacle presented by clots to the flow of blood from the heart, so that a beat sometimes takes place without a corresponding pulsation. It may reach 80 or 160 per minute in horse or ox.

The blowing murmur when heard is one of the most characteristic symptoms but must be carefully distinguished from other allied heart sounds. If very loud it may be confounded with the friction sound of pericarditis, but may be differentiated by its invariable coincidence with some particular portion of the heart's beat. The absence of local tenderness is another distinctive symptom. Again in pericarditis effusion takes place early annuling friction sound, and diminishing alike the impulse and the sounds of the heart.

It is of less practical value to be able to distinguish the precise seat of the murmur, yet the following data will guide to such a conclusion.

Simple induration or insufficiency of the Left Auriculo-ventricular (Mitral) valve. Paroxysms of palpitation, oppression, and difficulty of breathing; vertigo with loss of control over the limbs and vacilating gait; stupor, coma; slight tremor and blowing noise with the first sound of the heart; heart's im-

pulse, violent, but irregular in force, sometimes double; pulse feeble, irregular, unequal, or intermittent; sometimes though not at all constantly a venous pulse in the lower end of the jugulars. In chronic induration of this valve, or in osseous, or cartilaginous degeneration the same symptoms are shown. The more general symptoms may, however, require exercise to develop them.

Induration, etc., of the Right Auriculo-Ventricular (tricuspid) valve. The symptoms are almost identical with the last. Venous pulse is constant, and, particularly after exertion, the veins generally are distended. Dropsies are more common.

Narrowing of the Mitral orifice. In addition to the same general symptoms as the last named lesions, there is a sighing, blowing, purring or rasping sound, according to the degree of narrowing, heard *before* the first sound of the heart. It is the noise of the blood rushing through the narrowed orifice between auricle and ventricle. It is usually loudest behind the middle of the shoulder on the left side. Feeble pulse, frequent imminence of suffocation and filling of the limbs, etc., are nearly constant.

Narrowing of the Tricuspid orifice Symptoms nearly identical with the last. Venous pulse more constant. Blowing murmur sometimes loudest on the *right* side of the chest.

Induration or insufficiency of the aortic valves. Blowing murmur with the second sound of the heart. Double rushing sound in the carotid with each heart's beat. There is an appreciable interval between the beat of the heart and corresponding pulsation at the jaw.

Induration or insufficiency of the pulmonary valves. Blowing murmur with the second heart sound, but no corresponding double sound in the carotid, nor any marked retarding of the pulse.

Loose coagula in the heart or *adherent ones* (*polypi*) produce one or other of the above class of symptoms, according to the particular orifice they tend to block or the valves whose function they impair.

Anæmia and leukæmia may have blowing murmurs with the first or second heart sound.

Embolism. Plugging of arteries. Another class of symptoms sometimes supervenes because of loose clots being washed on into the arteries, and blocking them when they reach those

that are too small to transmit them. These symptoms will be as varied as the organs whose arteries are plugged. If in the brain there may be dulness, stupor, vertigo, somnolence, delirium; if in the liver, biliary and digestive derangement; if in the lungs, cough with the other signs of pneumonia and abscess; and if in the limbs lameness and paralysis, (brought on or aggravated by exercise, and often removed by a few minutes' rest), wasting of the muscles, etc. (*See Embolism*).

Causes. These are in the main the same as those of *pericarditis*. Weak health, exposure to extremes of weather, punctures with foreign bodies, but above all, the rheumatic constitution are common causes. Indeed rheumatism appears more prone to attack the serous membrane lining the heart cavities than that enveloping it externally. One reason for this is to be found in the great and incessantly recurring strain on the fibrous structure of the valves, and particularly in hard worked horses and hunting dogs in which the strain is often extreme. It has been argued that the increased blood pressure caused by digitalis is an appreciable cause. Its frequent connection with rheumatism is shown in the rheumatic lesions of joints and fibrous structures seen in carcasses dead of endocarditis.

Diseases in the muscular substance of the heart as cysts, abscess, etc., frequently extend to the endocardium.

Among other causes must be mentioned disease-changes in the blood. These may act on the valves directly as in the case of lactic acid injected by Dr. Richardson, into the peritoneum with the view of producing rheumatism and successfully as regards the lesions of the cardiac valves; or indirectly by determining coagulation and irritation of the lining membrane coming into contact with the clot. The very fibrinous and plastic state of the blood in extensive inflammations is a probable cause of the occurrence of clots in the heart, and the frequency of such clots in the dog has been ascribed to the plasticity of his blood (Leblanc). The injection of pus into the blood or the absorption of microbes from diseased surfaces will sometimes produce ulcerative disease of the valves. The same is true as regards the germs of omphalitis, pneumonia, arthritis and other infectious diseases.

Lafosse records certain cases of endocarditis due to extension of the disease from inflamed veins.

Prognosis. Endocarditis is always attended with great danger to life, but it is more likely to terminate in chronic valvular disease which quite unfits the animal for useful work. Mild cases may terminate in complete recovery.

Treatment. This is in the main the same as that adopted in the early stages of pericarditis. Absolute rest is of prime importance. Laxatives, sedatives and counterirritants are to be mainly relied upon. Belladonna and chloroform on the chest behind the left elbow may be used. As there is not the same danger from effusion, diuretics need not be pushed to the same extent. Digitalis must be avoided if possible until the high fever subsides. In infective cases quinia, salicylate of soda, salol, or hyposulphite of soda may be given. Later give tincture of muriate of iron.

In rheumatic cases, treat as for an acute attack of rheumatism. Frequent large doses of salicylate of soda or salol, large doses of acetate of potass and colchicum, warm clothing and counter-irritants to the region of the heart are especially demanded. (See Rheumatism.)

When clots are suspected, and when endocarditis threatens to lapse into the chronic form, it is recommended to give iodide of potassium (horse and ox 1 drachm, dog 5 grains, twice daily) with carbonate of ammonia or of potass and bitter tonics. A lengthened rest after apparent recovery is essential to avoid permanent valve lesions.

CARDITIS. MYOCARDITIS.

Definition. Rare. Complicates pericarditis and endocarditis, wounds of the heart, and tubercular and other deposits. Symptoms. Treatment.

Definition. Inflammation of the muscular substance of the heart.

This is a rare affection and is necessarily limited to a small portion of the heart's substance, otherwise, the cardiac contractions must cease in obedience to the general law that the normal function of an inflamed organ is for the time abolished. It is mainly seen as a concomitant of endocarditis or pericarditis, and extends only to the superficial muscular layers; or it results from a wound as in the penetration of the heart by a needle or other sharp-pointed body and is then equally circumscribed. It has been seen as a complication in infectious diseases—aphthous fever, pyæmia, septicæmia, pneumonia and tuberculosis.

The evidences of the existence of carditis are chiefly the lesions met with after death. 1st, The existence of abscesses in the heart's substance associated with polypus (Gowing, Leblanc, etc.,) or otherwise (Reynal). Also diffuse suppuration in the heart's substance (Puze, etc.) 2nd, Softening of the muscular substance a state occasionally met with when an animal has died of ruptured heart. 3d, Ulceration of the walls of the heart as reported by Mercier in a case of endocarditis. 4th, Transformation, and induration of the heart's substance whether into fibrous tissue, cartilage or bone. This last condition of the walls of the right auricle and ventricle has been repeatedly seen in old horses, the change being in certain cases so extensive that one is left in wonder as to how circulation could have been carried on. Three specimens of this kind were preserved in the museum of the Alfort Veterinary College, Paris, and the Royal Veterinary College, London. Lafosse records two cases of gangrene of the internal layers of muscle in endocarditis.

The *symptoms* are those of acute heart disease generally modified somewhat by the precise location of the inflamed spot, and *treatment* need not differ materially from that applied for inflammation of the investing membranes, inner and outer, and for the infectious disease which it complicates.

CHRONIC VALVULAR DISEASE OF THE HEART.

This, as already noticed, is a common result of endocarditis, the valves being most obnoxious to disease in such cases. The *symptoms* are those mentioned under endocarditis as characterizing disease of the different valves, such as incapacity for exertion, difficult breathing, palpitation, irregularity or intermission of pulse, venous pulse, abnormal heart sounds, unsteadiness of the limbs when driven, and dropsical swellings in the limbs and elsewhere. The reader is referred to endocarditis for particulars, it being borne in mind that these symptoms are not in this case associated with fever.

Horses affected in this way are useless. Cattle may sometimes be partially fattened by preserving them from all sources of excitement, by keeping the bowels regular and by combating any paroxysms with sedatives, such as aconite, veratrum, hydrocyanic acid, or opium, and with digitalis.

FATTY DEGENERATION OF THE HEART.

Causes, improvement in the direction of easy fattening, inactive life, best breeds of butcher cattle and pigs suffer. Symptoms, weak, irregular, intermittent pulse, palpitation, unfitness for exertion, general heart symptoms.

In addition to the fibrous and bony transformations to which the substance of the heart's walls is subject, a fatty metamorphosis is frequently met with. In most cases the fat accumulates in great masses externally, but in others the muscular tissue has to a greater or less extent lost its natural structure and fatty granules have taken the place of the sarcous elements. In overfed oxen the right cavities of the heart rarely escape dilatation, and this condition is very often accompanied by the fatty change. Virchow has shown that highbred English pigs imported into Germany are subject to a similar affection of the heart and of the entire muscular system. It may occur during wasting diseases and from phosphorous poisoning.

The *symptoms* are weak, irregular and intermitting pulse, palpitation on excitement, weakness of the heart's impulse in the intervals, incapacity for exertion, sighing, Cheyne-Stokes respiration, loss of control over the limbs when hurriedly driven and tendency to dropsy. It is often associated with dilatation, is rarely distinguishable from it in life, and is equally beyond remedial measures. The feeding animals most commonly affected can usually be fattened if removed from all sources of excitement. In case of phosphorous poisoning improvement takes place when the poison is stopped.

NEW FORMATIONS IN THE HEART. TUMORS. PARASITES.

Glanders, abscess, melanosis, tubercle, polypus, nævus, parasites—echinococcus, cysticercus tenuicollis, cysticercus cellulosa, trichina, sarcocyst, filaria immitis, strongylus subulatus, strongylus vasorum.

1st. **Deposits of Glanders.** In many cases of glanders and farcy in horses the specific product is deposited in the heart as well as in other internal organs. Such deposits are small but numerous, infiltrating the muscular tissue; their cut surface is dry, finely granular and of a yellowish white color.

2d. **Abscesses** are sometimes formed in the heart from the colonization of microbes from suppurating surfaces.

3d. **Cancer of the heart** has been noticed chiefly in dogs by Leblanc. It occurs only consecutively to cancer in other parts of the body, yet it has sometimes acquired considerable dimensions and interfered materially with the movements of the heart.

4th. **Melanosis** of the heart has been repeatedly noticed in the horse. Some if not all such cases should be classed with cancers, as these internal deposits of black coloring matter in solipedes, have, in our experience, mostly possessed malignant characters, though they are usually simple tumors as developed in the skin of the horse. These black masses usually project beneath the pericardium or endocardium.

5th. **Tuberculous deposits** have been met with in the substance of the heart in cases in which the lungs or other organ were the seat of this disease.

6th. The *fibrous growths* or *polypi* due to the deposition and organization of fibrinous material from the blood have been referred to under *endocarditis*.

7th. Gamgee reports the existence of a vascular tumour of the right ventricle of a horse in the museum of the Turin Veterinary School. It consisted of varicose veins ramifying beneath the endocardium which in its turn was healthy.

8th. The **parasites found in the heart** are various. *a*. One, the **Echinococcus Veterinorum**, has been repeatedly found in the substance of the heart or projecting from its inner or outer sur-

face. *b.* Another, the **cysticercus tenuicollis**, has been met with in the pericardial sac of a calf (Reed). *c.* A third, the **cysticercus cellulosa** infests the muscular structure of the heart of measly pigs. *d.* The heart like other voluntary muscles of hogs occasionally contains **trichina spiralis**. *e.* **Rainey's cysts** (**sarcocysts**) are microscopic ovoid bodies usually found in the hearts of oxen and other animals. *f.* A round worm, **filaria immitis**, first described as filaria papillosa hæmatica by Delafond and Gruby, lives in the blood of the dog, is one millimeter thick by fifteen to 30 centimeters long. It may obstruct the pulmonary artery (Serres) or the mitral orifice (Silvestre). It may cause various nervous disorders and even sudden death. Its mode of entrance is unknown. *g.* **Strongylus Subulatus**, 1 to 2 mm. long by 70 to 90 μ. in thickness was found in numbers in a nodule of a dog's lung, and the dorsal vein of the penis of a dog (Leisering). *h.* **Strongylus Vasorum** in the right auricle and ventricle of a dog, in pea-like blood clots. It is 14 to 21 mm. long by 1 m. in thickness (Serres).

RUPTURE OF THE HEART.

In the lower animals ruptures of the heart have been observed as the result of (a) extraordinary exertion, (b) violent concussion, and (c) ulceration and degeneration. The rupture of the fatty heart in the lower animals is not common.

Rupture during severe exertion occurs in the perfectly healthy heart. The ruptures take place in the weakest point, and most commonly in the fibrous ring which encircles the base of the heart and attaches the great aorta. This is occasionally seen to happen in very spirited horses during a severely contested race or when a heavy load is being dragged up hill. Percivall mentions the case of a horse at a Woolich racing meeting, which had just lost a heat by half a head and which died just after passing the winning post, with ruptured right auricle.

Cases occur during coitus (Hering), tympany (Anacker, Mayer, Perdan) and operations (Stockfleth).

Rupture from Concussion more frequently implicates the muscular walls which have not the same power of resistance when they receive the blow in a relaxed condition. Parker met with a case of rupture of the right auricle at its base or at the line of its union with the ventricle. The subject was a pony which ran away down hill and struck his right shoulder violently against a cart wheel. In other instances the rupture takes place in the posterior vena cava, and particularly if its walls have been the seat of disease. Gamgee found rupture of the commencement of the azygos vein in oxen killed by pithing in the slaughter houses of Ferrara, and Professor Maffei subsequently found that out of 3095 oxen killed in these abattoirs 57 had this vein ruptured. Gamgee's explanation of the occurrence is that "the instant the animals are pithed the walls and contents of the chest become paralyzed, the heart becomes an inert bag filled with fluid, the jerk of which as the animal falls, causes rupture of the containing vessel at its weakest part and this is in truth the vena azygos whose walls are thin and only protected externally by the pleura." Hertwig gives other cases resulting from falls.

Perforation of the heart from ulceration is sometimes seen

in cows when sharp-pointed metallic bodies from the stomach make their way into its substance. An alleged case of rupture following ulceration of the walls of the right ventricle is recorded by Gaullet.

Inflammation, softening, fatty and calcareous degeneration, dilatation, atheroma, and the presence of parasitis in its substance render the heart more friable and predispose to rupture.

Lesion. The rupture is often at the fibrous ring encircling the aorta or pulmonary artery; in other cases in the muscular wall of ventricle or auricle.

Symptoms. Death may be practically instantaneous. If delayed there is hurried breathing, anxiety, weakness, pallor of the mucous membranes, staggering, trembling, vertigo, stupor, and convulsions.

DISEASES OF ARTERIES.

The chief morbid conditions seen in arteries are: Wounds, inflammation, thrombosis, embolism, degeneration, and aneurisms. Wounds belong essentially to surgery.

ARTERITIS. EMBOLISM.

Internal and external arteritis. Thrombosis, from inflammation. Embolism. Bruising. Stretching. Ligature. Lesions. Extension of clot, color, consistency, adhesion, lamination. Composition of clot. Condition of vessel. Changes in muscles. Causes: muscular tension, embolism. Heart clots, venous clots. Infecting debris. Symptoms: paresis or paralysis on exertion. Local suffering, tenderness, firm swollen artery, derangement of nutrition and function, atrophy. Chronic arteritis: Atheroma. Changes in serosa. Fibrous thickening, atrophy, dilatation. Strongylus. Treatment: rest, anodynes, alkalies, massage.

Inflammation of arteries has been divided into *external* and *internal* arteritis, according as it affects the fibrous sheath of the artery or its inner lining membrane.

In **external arteritis** the exudation of lymph often forms a protecting layer around the vessel, while the inner coats continuing sound the current of blood remains unimpaired. Even when suppuration takes place in the vicinity of a large artery, that vessel may pass through the center of the abscess and convey the blood as freely as before. The nutrition of the vessel thus detached from the surrounding tissues is maintained by its accompanying nutrient artery, though if the abscess is large there is danger of a deficient supply. The frequent presence of such arteries traversing an abscess should make the surgeon careful how he breaks down the bright pink bands occasionally seen to stretch across such cavities.

Internal arteritis, or *inflammation of the internal coat of an artery* is incomparably more serious and mainly because it determines the coagulation of the contained blood and consequent *plugging* of the vessel. This is but one manifestation of the general law that in inflamed tissues the fibrine forming elements are produced in excess, and when blood comes in contact with these it tends to coagulate (thrombosis). On the other hand the inflammation in the arterial coats may ensue from the pressure of a

blood clot formed in the veins or heart and carried on with the current until it reaches an artery too small to admit it (embolism).

The inflammation may be confined to a limited space as when an artery is bruised, stretched so as to tear through its inner coats, or interrupted by a ligature. It may on the other hand be diffused over a greater extent of the vessel, and in some cases two inflamed portions are separated by intervals of sound artery.

Anatomical features of the inflamed artery. In active inflammation of the internal coats of an artery, it contains blood clots, and if the inflamed surface is not very limited in extent the vessel is completely plugged and the clot forms up to its nearest transverse branch on the cardiac side, precisely as if the artery had been tied. The resulting clot is sometimes tubular, so that an impaired circulation is still carried on. The clot varies in length according to the extent of vessel inflamed, or the distance from the inflamed spot to the nearest diverging branch. The clot is usually fusiform in outline and is firmly attached throughout more or less of its diameter, and occasionally so firmly that it is all but inseparable from the serous membrane. The narrowed ends of the clot mostly float free in the liquid blood and portions from the end most distant from the heart will sometimes get detached, and by blocking up smaller arteries give rise to new centres of disease. This is a true instance of *embolism* or *plugging*.

The clot has nearly always a grayish or yellowish white color in the larger vessels, such as the posterior aorta, and an uniform pink or red streaked with yellow in the small. It is possessed of great firmness and elasticity. That portion of the surface which was not attached to the arterial walls, during life, is clear, smooth and glistening, while the portion which adhered to these walls is rough, irregular, and broken into shreds. It is usually composed of concentric layers showing its mode of formation.

Clots of this kind analyzed by M. M. Lassaigne and Clement were composed of water 74 parts; fibrine and albumen 25 parts; and alkaline salts 1 part.

The vessels filled by these clots are very irregular in their outline being thick and bulging at one point and thin and constricted at another. The outer coat is rarely the seat of morbid change, though it sometimes shows branching redness and thickening from exuded lymph. The internal coat where the clot was

attached is intensely and unnaturally red, and a rough granular surface has given place to the healthy, smooth glistening appearance. In old standing cases the clots can only be separated from such surfaces by dissection with the knife. Other portions of the surface than those to which the clot adheres are usually smooth and polished, though rough granular and injected patches are sometimes met with independently of clots.

The muscles formerly supplied with blood by the obstructed arteries are pale, discolored, unnaturally firm, and if some time has elapsed since the plugging their fibrillated structure is made out with difficulty.

Causes. The causes of arteritis are often obscure. Goubaux conceived that it was frequently determined by extreme muscular tension. In support of this view he adduced the facts that it has been mainly observed in the horse, in which such stretching of the muscles is greatest, and that its most common seats have been where the muscles and vessels are most liable to stretching. Thus it is frequent in the posterior aorta towards its termination or in other words where the adjacent muscles (psoæ) are very liable to laceration from slipping backward or from efforts to disengage the limbs when fixed in soft ground; the femoral and auxillary arteries are likewise frequent seats of inflammation and are likely to be overstretched when the limbs slip outwards.

Embolism or **Plugging** of the arteries must be accepted as another cause. This is referred to under *endocarditis*, as an occasional consequence of the detachment of clots and fibrinous substances from the internal membrane of the heart. The detached mass in this case passes from the heart into the aorta and thence through its divisions until it reaches a vessel too small to receive it, when it is at once arrested and determines inflammatory action in the plugged vessel. When arrested in some soft organ such as the lungs, liver or brain the resulting inflammation often gives rise to extensive suppuration and abscess. In other situations its effects may be confined to inflammation, the shutting off, of blood from particular parts, the impairment or loss of their function and nutrition, and finally atrophy and degeneration.

But the heart is not always the primary source of such clots. Virchow and others have demonstrated by *post mortem* examina-

tions in cases of plugging and by a number of experiments on the lower animals, not only that such clots may have their place of nativity in some distant and diseased part of the body and proceed in the veins to the heart, and thence through the arteries to other distant parts of the body where they plug the vessels and induce a train of morbid changes ; but that such embolism arteritis and abscesses can be produced at will by the introduction into the circulation of solid and insoluble (infecting) bodies. Fragments of decaying and suppurating tissue and the elements of tubercle and cancer may be thus equally carried onward in the current of the circulation, and reproduce themselves at those points where their course is arrested. This is a mode in which secondary deposits of these morbid matters are determined. Embolism and arteritis in the body and limbs occuring in this way necessarily have their point of departure in pre-existing disease of the lungs. The clots loosened from the capillaries or veins of the lungs are carried through the left side of the heart into the arteries of the body at large to be arrested in some of the smaller vessels. I have seen plugging of the digital arteries of the hind limbs, to occur in this way in a horse that had been suffering from inflamed lungs.

Microbes and toxins may pass harmlessly through healthy parts, including the pulmonic circulation, to establish colonies and embolism beyond where the tissues have become debilitated. Thus Gamgee records a case of embolism of the anterior mesentric, right external iliac and right femoral arteries, supervening on an attack of strangles.

Symptoms of acute arteritis. These consist largely in impaired muscular power in the part, indications of acute local suffering, such as trembling and tenderness to the touch, if the obstructed vessel lies within reach it can be felt as an exquisitely tender cord-like mass, and the limb on the distal side of the embolism and dependent on the diseased vessel for its blood supply is anæmic and cold. In the distal portion of the embolic artery and its branches pulsation has ceased. If the lesion is extensive there may be more or less fever, but a limited arteritis in a small vessel may escape this complication. If the disease is of long standing there is atrophy of the tissues formerly supplied by the embolic vessels. The secondary derangement of nutrition and function

are as varied as the organs affected and will be noted below in the special article on thrombosis and embolism.

Chronic arteritis. Atheroma. This is an indolent inflammation supposed to result mainly from strain and overwork, and manifested by thickening and clouding of the serosa, with cell proliferation, softening and fatty degeneration. The diseased substance becomes soft, pultaceous, slightly greasy, and under molecular degeneration it breaks up and is even in part washed on in the blood stream. Other degenerations may occur in the inflamed walls of the artery. The exudate may become organized, constituting fibrous thickening. It may become the seat of calcareous degeneration. It may yield to the blood pressure, becoming slowly attenuated (atrophy), and even dilated (aneurism by dilatation). As a cause of chronic internal arteritis in the horse should be named the presence in the vessels of the larva of the strongylus armatus. The posterior aorta and anterior mesenteric artery which are the most commonly infested by these parasites are frequently attenuated, dilated and calcified in this connection.

Treatment. Acute arteritis should be treated like any other local inflammation, by rest, soothing applications (fomentations, astringents, icebags), and alkaline salts. It has been proposed to manipulate the affected artery and contained thrombus, but this can only tend to block the smaller arteries farther on, and perhaps with even more injurious results. The liberal use of alkalies on the other hand, if effective in dissolving any portion of the clot, returns this to the blood stream in a condition that will not endanger further embolism. The agents usually employed are carbonates of ammonia, potash or soda, and iodide of potassium.

THROMBOSIS AND EMBOLISM.

Definition. Thrombosis—clotting in the vessel. Embolism, blocking of the vessel. Thrombus may form in any bloodvessel. Embolism occurs in arteries. Clot follows the blood current. Causes of clotting—fibrinogen, paraglobulin, fibrine ferment; foreign bodies; parasites; air; blood that has been exposed, (transfusion; aerial germs; disease germs; chemical coagulants; high and low temperatures; breaches of endothelium; congestion or inflammation of the serosa; stasis of blood and extension of clot; ligature near a branch vessel: deoxidation and carbonization of blood, marasmus; neoplasms; traumas of the vascular coats. Infarction, causes of blocking; disintegration of clots, softening, liquefaction, ulceration, action of microbes; excess of white globules; air; fat; parasites. Pathogenesis; complete occlusion of vessel; infarction; sequestrum; colateral circulation; embolism of external iliac or femoral artery; effects on pulse; during rest; atrophy; lameness comes on with exertion; disappears under rest; circulation inadequate to sustain active function. Embolism of internal iliac artery; effect on pulse; on tail and pelvic organs. Embolism of axillary artery; effect on pulse, action, nutrition. Embolism of mesenteric artery; verminous; effects on innervation and circulation; spasms, congestions. paresis; involution. Treatment: expectant; alkalies; gentle exercise, time.

Definition. **Thrombosis** is the blocking of a blood vessel by a clot formed in its interior by the deposition of layer above layer on its inner coat.

Embolism is the blocking of a bloodvessel by a clot or other solid body formed at a distant point of the circulation floated on in the blood stream, and arrested when it reaches a vessel too small to transmit it.

A *thrombus* may be formed at any point of the circulatory apparatus (heart, arteries, veins) whenever the conditions are such as to determine coagulation of the blood. An *embolism* on the contrary is a disease of the arteries since in these the blood current, proceeding centrifugally from the greater to the lesser, inevitably carries the moving solid to a point too narrow to allow of its further progress. Thus clots originating in the systemic veins or right heart pass to the lungs and produce embolism of the pulmonary arteries whereas those formed in the pulmonary vein or left heart are arrested in some part of the systemic arteries. Clots formed in the portal vein however are arrested in the hepatic vessels into which that trunk breaks up.

Causes of Thrombus. The production of a thrombus may be due to the condition of the blood or of the vessels. The researches of Buchanan, Schmidt, Hammersten and others show that two albuminoid elements, fibrinogen and paraglobulin, present in the living blood, and a fibrine ferment mainly derived from the white corpuscles in process of change or destruction, determine powerfully the formation of fibrine and clot. Hewson, Brücke and Lister have shown that blood may be maintained fluid for many hours in an unimpaired vein, or turtle's heart though it may have been removed from the body, the important condition being that the vein shall retain its vitality and suffer no derangement of its endothelium. Lister has even shown that blood may remain fluid for many hours in a sterilized glass tube which has been filled by passing the tube carefully into such a vein without disturbing its lining membrane, or imparting motion to the liquid. In such a case a thin film of coagulum only, forms on the interior of the glass tube. In healthy blood, without addition of any extraneous matter, and kept perfectly still, the plasma and globules retain their integrity, and the former its fluidity for a length of time. But if shed into a basin it coagulates at once.

a. *Changes in the blood.* Contact with foreign bodies generally determines this change and prompt coagulation. Transfixing the artery with a needle, even a silver one, the entrance of parasites (actinomycosis, strongyli, filaria), the presence of pus, and of certain infectious microbes and their products, the introduction of solid particles and even of air into the vessels, the transfusion of blood which has been exposed to receive aerial germs, or which contains microscopic clots, or the globules of which have become modified by contact with a basin or other vessel, even the transfusion of defibrinated blood may cause coagulation. The danger is always greater if the blood is drawn from a different genus and unfitted to live in the blood of the recipient. Disease germs are especially dangerous if adapted to colonize the serosa of the vessel and destroy its epithelium. A decrease of the density of the blood favors coagulation, a lowering of one thousandth rendering it syrupy, and various chemical agents induce or favor coagulation, thus acetic acid, valerianic acid, alcohol, the salts of iron, and above all the salts of lime act in this way. Very high and low temperatures throw down the fibrine as a grumous precipitate, but the clot remains soft.

b. *Changes in the vessels.* Any disturbance or alteration of the endothelium sets free the so-called fibrine ferment, and precipitates coagulation. Lister found that contact of ammonia with the interior of an otherwise living vein caused a thrombus. So in all endarteritis and phlebitis coagulation takes place on the serosa and quickly blocks the vessel. Even in the capillary vessels the same principle holds, and in inflammation minute coagula (thrombi) form in the capillary network throughout the whole inflamed area. This explains not only the capillary blood stasis but the thrombosis of inflamed arteries and veins. In these two latter the clot increases and extends in the direction of blood stasis:—in the artery toward the heart as far as the next colateral branch, and in the veins away from the heart as far as to the next colateral trunk. On the distal side of the arterial thrombus the blood flows off freely toward the capillaries, but on the proximal or cardiac side it is absolutely stagnant up to the next branch through which it can freely flow into the capillary plexus. Into this stagnant blood the fibrine ferment, produced by the altered white globules in the clot already formed, slowly extends until the whole has formed a firm coagulum. Beyond this the actively moving blood carries off and dilutes this ferment so rapidly that it can exert no appreciable effect on the fibrine-forming elements. The principle is an important one in surgery, as the clot formed entad of the ligature will be extensive in proportion to the distance from the first colateral trunk, and in inverse proportion to this clot will be the danger of secondary hæmorrhage. In veins the same rule holds, with this difference that as the blood is flowing toward the heart it empties the vessel on the cardiac side, and stagnates on the distal side up to the next colateral branch. Hence it is that a thrombus in a vein always extends away from the heart, while that of the artery extends toward it.

Another cause of coagulation is the deoxidation of the blood and the excess of carbon dioxide. This occurs in the stagnant blood in the vessels and above all in the capillaries. The normal trophic changes in the serosa, fail to take place in contact with blood in this state, and the resulting changes in the white and endothelial cells set free fibrine ferment and determine coagulation. Stasis of the blood from any cause (ligature, pressure, embolism), tends to this condition and the extension of the coagulum.

A thrombosis of marasmus has been observed in anæmic and debilitating diseases, and apart from the microbian invasions in a certain number of those affections, this may be looked on as due in part to the lessened density and other changes in the blood and to the debility of the serosa of the vessels.

The compression of the vascular walls or their penetration by neoplasms, tumors and ulcers, is another cause of coagulation and thrombus, also a varicose or aneurismal dilatation, with weakening of the vascular walls, or dilatation of the heart with structural changes in the endocardium as stated under that heading, or compression of the smaller vessels and capillaries by an exudate in process of organization, or a similar obliteration under the action of extreme cold. Injury to the serosa of the vessel by stretching, bruising, laceration or section determines a thrombus starting from the injured endothelium. In the smaller vessels the thrombus is usually deep red from the entangling of a large quantity of red globules, whilst in the larger arteries the greater part of the globules pass on and the coagulum is largely buff or straw colored.

Again in obstruction in the smaller arteries, the inactive capillary plexus and the tissue beyond are liable to become gorged with blood with excess of red globules, from the adjacent capillary network, constituting *infarction*, and ending in gangrene. In cases in which this is prevented by the action of intense cold the part may remain pale, as *white infarction*.

Causes of Embolism. As already stated embolism results from a detached portion of a thrombus passing to a smaller vessel and obstructing it. Such detachment is favored by molecular softening, liquefaction or suppuration in the clot or beneath it, by the destructive action of microbes, or by friction or manipulation of the obstructed vessel. Excess of white globules (leukæmia) favors the formation of minute coagula and embolism. Bubbles of air, globules of fat, or cholesterine crystals block the fine pulmonary capillaries, and the debris from atheromatous patches, ulcers, and tumors opening on the inner wall of the artery form emboli in various parts. Finally parasites, especially the larval strongyli in solipedes and filaria and spiroptera in dogs, themselves obstruct the vessels and determine coagulation.

Pathogenesis. In the larger arteries (aorta, radical stump of

the mesenteric artery) clots (as from strongles) rarely produce dangerous obstruction. In the smaller vessels stenosis is complete and anæmia and gangrene are liable to occur unless the blood supply is partially maintained by anastomotic vessels. When the embolism affects a number of smaller arteries or capillaries in a vascular organ like the lungs, the blood filters in from the adjacent capillaries, in which circulation is still carried on, and this passes through the softened and ruptured capillary walls so that the tissue is charged with globules and constitutes a *black infarction*. In the lung this usually affects one or several lobulettes, forming a pear shaped mass corresponding to the distribution of the obstructed vessel. The cut surface is black, compact and granular. The lymph thrown out around it forms an organized fibrous sac, and the unclosed sequestrum undergoes a slow necrobiosis, blanching and liquefaction into a pus-like fluid which is removed by absorption. Such results are met with in the parenchymatous organs (lungs, liver, spleen, kidneys, etc.) and less frequently in the limbs. The symptoms will correspond to the particular organ invaded.

In the fore or hind limbs the result is usually less radical. The vessels below the obstructed trunk are connected more or less freely by anastomosing branches, so that the circulation in the tissues below, though somewhat restricted, remains active enough to sustain a fair measure of nutrition. Apart from the suffering, attendant on the preliminary inflammation, the morbid phenomena are largely confined to the absence of pulsation in the lower part of the limb and the inability of the muscles to sustain active contraction.

Chronic Embolism of the External Iliac or Femoral Artery. In this condition the pulsations in the digital arteries are imperceptible, if it has been of long standing there may be obvious atrophy of the muscles of the thigh, but when standing quietly or walking there is usually no lameness. In continuous rapid walking and above all in the trot, however, he soon begins to halt on the affected limb, and this rapidly increases, the joints bending under his weight, the toe dragging and the animal threatening to drop altogether. If stopped and allowed to rest for ten or fifteen minutes he gradually recovers and may be led quietly back to his stable without a sign of lameness. But if again trotted fifty or one hundred paces the lameness develops anew and disapppears in

the same way when left at rest. The circulation in the muscles is enough for a moderate nutrition but altogether inadequate to sustain active work.

Chronic Embolism of the Internal Iliac Artery. In this case the control of the muscles of the limb may be perfect but there is some indication of paresis of tail, bladder, rectum and anus. Impaction of the rectum is liable to occur. By examination through the rectum the pulsations are felt to be strong in the aorta and external iliac, but imperceptible in the internal iliac blocked by the embolus.

Chronic Embolism of the Axillary Artery. Here there are the same general symptoms, the absence of the radial and digital pulsations, the wasting of the muscles of the forearm, and the intermittent lameness, developed rapidly by exercise and recovering promptly under rest.

Acute Embolism of the Mesenteric Arteries. This will be fully treated under the title of verminous colic in solipedes. The blocking of the branches, usually of the anterior mesenteric artery, leads to derangement of the innervation, congestions, spasms, involutions and other disorders. The presence of the strongyli in the fæces, the general symptoms of intestinal worms, and the recurrence of the indigestions and spasms would serve to indicate the nature of the complaint.

Treatment of Chronic Embolism. As affecting the arteries of the limbs the repair must be largely left to nature, and we must place the patient in condition, favorable to such repair. Except in the early stages absolute rest is not necessary. Gentle exercise stimulating to a freer circulatoin solicits a slow enlargement of the anastomosing vessels (arterial or capillary), and when this has reached a given stage, weak pulsations may again be felt in the vessels beyond and the muscles will once more stand moderate work without lameness. Alkalies and iodide of potassium may be given to solicit solution of the clot, but this can rarely be counted on to the extent of rendering the vessel once more pervious. A small paddock in which the patient can move around quietly is desirable, and in a few months a tolerable recovery may have taken place.

Embolism in other organs must be treated on the same general expectant method, and a considerable time is usually necessary to secure a fair recovery.

ANEURISM.

Definition. Divisions, true, false, dissecting, arterio-venous, mixed, traumatic, spontaneous. Causes, violence, rupture, debilitated vascular walls, strains, stretching, force of blood current, overloaded intestine, strongyli, contiguous inflammation, embolism, microbian invasion of the walls, arteritis, concussion. Symptoms, soft tumor pulsating with the heart, a double rushing sound, diagnosis from abscess, nervous disorders through pressure, cramps, palpitations. Treatment, when desirable, rest, moderate, laxative diet, iodides, bromides, icebags, compression, ligature, galvanopuncture, wire coils, injections.

Definition. A pulsating swelling on an artery, consisting of a sac filled with arterial blood.

Divisions. **A true aneurism** (**aneurism by dilatation, arteriectasis**) is a simple dilatation of the artery, the tumor being surrounded on all sides by the distended arterial walls. It is usually fusiform or cylindroid, but may have the form of a more or less rounded sac.

A **false aneurism** is where the wall of the artery has been lacerated and the blood is enclosed in an adjacent sac of condensed connective tissue and communicates with the interior of the vessel. The same name has been given to cases in which the inner coat only has given way, and the middle and outer coats constitute the walls of the sac. From its liability to extend and separate the tissues this is further known as a **dissecting aneurism. Arterio-venous aneurism** in which an intervening sac communicates with both artery and vein, has been found in the human subject.

Mixed aneurisms are those in which a dilatation of the artery is complicated by the presence of an outside pouch.

A distinction has also been made according to origin into *traumatic* and *spontaneous*. The former is of necessity *false*, whereas the latter may be *false* or *true*.

Causes. Apart from rupture of the arterial coat by direct violence, the common cause is a debility and loss of resistance in the walls. In horses a far larger proportion of aneurisms are deep-seated than in man, in whom forced muscular effort is less common. Yet even in horses the most common seat—the posterior aorta—is liable to overstretching and to inflammation and softening by reason of contiguity to dorsal sprains. The posterior

aorta too, from its size and direction on leaving the heart, is in the direct line of the strongest blood current, and under long continued, forced and violent efforts (as in racing, hunting, and heavy uphill draughts), has to sustain an extraordinary blood pressure. Bouley claims as an additional cause the pressure of a loaded colon. This is also the point of all others where the vessels suffer from the presence of the larval strongyli. From whatever cause originating, congestion of the arterial coats leads to more or less attenuation, softening or lack of cohesion, and they tend to yield under the blood pressure. Similar conditions operate on the smaller vessels in different parts of the body, and thus overstretching, contiguous inflammation, and excessive blood pressure cause such lesions in the chest, trunk and limbs.

Another cause is embolism which by blocking an artery at once increases the tension in the vessel on the cardiac side of the obstruction, and develops inflammation in the arterial coats, robbing them of their cohesion and resisting power.

Eppinger has shown the importance of infectious microbes in weakening the arterial walls and predisposing to aneurism.

The larval strongylus armatus already referred to is the most potent factor in solipedes. They accumulate in the anterior mesenteric artery, leading to clotting of the blood, inflammation of the serous coat, and dilatation, so that in some verminous localities nearly every old horse shows a lesion of this vessel.

All forms of arteritis, and disease of the vascular walls which entail attenuation or weakening, predispose to aneurism.

Of direct traumatism may be mentioned an aneurism of the arch of the aorta in a horse struck by a wagon pole, during a sharp descent (Jacob), and two with aortic aneurism after violent blows on the back with shafts of wagons.

Symptoms. An aneurism within reach of the hand is to be recognized primarily by the pulsation of the swelling synchronously with the beats of the heart, and by a double rushing sound with each beat of the heart, observed on auscultation. An abscess over a large artery lying on a bone may pulsate but it is to be distinguished by the presence of a single in place of a double rushing sound on auscultation, by the possibility of causing more or less complete collapse under pressure, and by the history of an active phlegmonous inflammation followed by softening which steadily extends from the centre of the previously dense mass.

In a case of aneurism of the gluteal artery of the horse reported by King and in one observed by the author the symptoms were unmistakable. Other similar examples on the popliteal artery and other failed to be recognized during life though attended by lameness.

In internal aneurism the symptoms are mostly indefinite. Ollivier found tympany and vomiting in a goat which at the necropsy showed an aneurism of the anterior aorta as large as the closed fist and enclosing a sewing needle. A more careful diagnosis should have detected a retarding of the maxillary pulse and a double rush over the carotid with each beat of the heart. Pressure on the vagus doubtless led to the symptoms noticed. In aneurism of the posterior aorta there have been noticed a loss of life and energy, dulness, lack of appetite and stiffness of the loins. Torpor of the bowels, expulsion of fæces with effort and groaning, intermittent colics, lameness in one or both hind limbs, and finally cramps in the hind limbs, and palpitations. In one case Maillet was able to reach the aneurismal tumor through the rectum.

Treatment. The treatment of internal aneurism will be seldom called for in the lower animals, as the disease is seldom diagnosed, is beyond reach of mechanical applications, and survival without certain power of endurance would seldom be desirable. In some valuable breeding animals it might be worth while to seek prolongation of life. The most promising measures are absolute rest, and low, non-stimulating diet of a laxative nature and in small bulk. Iodide of potassium is often useful in man, and although in the lower animals there is not the excuse of specific disease, yet the rest to the circulation and reduction of blood tension are not to be undervalued. Bromides may be given with the same object.

Other measures applicable only to aneurisms, within reach and essentially of a surgical nature include: Ice bags and compression. The compression should as a rule begin at the distal end of the limb and be concentrated by suitably shaped pads on the swellings. Ligature of the diseased artery above or below or both above and below the tumor. Galvano-puncture of the aneurism with the object of inducing coagulation. The introduction of coils of fine wire through a hypodermic needle with the same object in view. In both horse and dog the persistent compression with the finger seconded as it is by the plasticity of the blood has succeeded in checking the flow from large arterial

orifices, and offers great encouragement in the application of this measure to aneurisms. The injection into the sac of tincture of chloride of iron with firm compression to prevent motion of the blood is another available resort.

ARTERIO-SCLEROSIS.

Fibrous thickening of the arterial coats and calcification are well known lesions in the posterior aorta particularly of the horse. Commencing in congestion or degeneration which lessens the resistance of the vascular walls, the condition tends to dilatation, and if this is checked by compensatory thickening, the condition of sclerosis is induced. The combination of a slight fusiform dilatation and fibrous or calcareous sclerosis is well known in the posterior aorta of the horse. Unless it advances to marked aneurismal dilatation the condition is not often recognized. If diagnosed, rest and quieting of the circulation are especially indicated. Should it occur in other parts of the body the symptoms would correspond to the organ invaded.

ANGEIOMA. CIRCOID ANEURISM. ANEURISM BY ANASTOMOSIS. VENOUS TUMOR. NŒVUS.

These are forms of dilatation and elongation of the network of small arteries, or veins, and even of the intervening capillaries. In man these constitute the unsightly red patches and swellings that appear on the face and hands. In animals with dark skins and hairy covering they can only be recognized by the swelling, the feeling as of a bag of worms when the hand is passed over it, and by the rushing sound when auscultated. The trouble is usually subcutaneous and is essentially a surgical one. The most promising treatment is by persistent pressure, by electric current supplementing the pressure, by electro-puncture, and by injections of muriate of iron. When the nœvus is not too extensive a double thread drawn by a needle through beneath the tumor at short intervals, then cut and each point tied separately, so as to completely stop circulation is most effective.

PHLEBITIS.

Divisions, traumatic and idiopathic. Causes, punctures, defective blood supply in walls, debile coats, thrombus, infection, overstretching, injury or disease of serosa, irritants in blood, microbic infection. Lesions, exudation, cell growth, breaches in serosa. Adhesive phlebitis, desquamation, granulation, occlusion. Suppurative phlebitis, infection, pyæmia, erysipelas, metritis, ulceration, neoplasms, phlebolites. Symptoms, local, firm, corded, swollen vein, extends entad, venous congestion, dropsy, gangrene, diagnosis from lymphangitis. Fever, venous congestion in vicinity. Treatment, germicide, rest, cold, antiseptics, blisters.

Inflammation of veins as seen in the lower animals has usually been a sequel of bleeding and is hence a purely surgical lesion. Animals as well as man however are subject to idiopathic phlebitis which as affecting the deeper seated veins may be held to be a medical subject.

The *causes* of **idiopathic phlebitis** are varied. Injury to the walls like the punctures made in bleeding; if they result in the exposure of a raw, and above all an inflamed, surface to the blood, tends to the formation of a thrombus, and of local inflammation. Even the inflammation of the outer coat tends in the same way to thrombosis and phlebitis, and the experiment of Nicasse showed that the dissection of its sheath from a vein, thus robbing it of its vascular and nervous supply promptly induced coagulation of the blood in the denuded part. The debilitated or devitalized walls evidently give off fibrinogen and fibrine ferment in amount that is incompatible with the maintenance of fluidity. All other forms of direct injury to the veins, leading to disturbance of the endothelium or cell enlargement or exudation in the intima, will operate in the same manner. Sometimes as in puerperal phlebitis the inflammation extending from the adjacent tissue to the walls of the veins, determines thrombosis, and the invasion by pus microbes determines suppuration. Bruises, over-stretching, pressure with over-distension, and the circulation in the blood of irritant matters may lead to changes in the wall, thrombus, and inflammation. Such irritants may be septic or other bacterial products, or they may arise from the colonization of bacteria on or in the venous coats with the same final result.

The *lesions* in the vein are often primarily of the nature of exudation and cell growth in the coat, without at first any change in the serosa or endothelium. Later the changes implicate those, thrombosis follows and one of various ulterior processes.

In **adhesive phlebitis** which is most frequent as the result of purely mechanical injury, the endothelium is disquamated and granulations from the denuded surface extend into the clot and finally occlude the vein. A recovery takes place by the organization of this new product and the contraction of the vessel into a simple fibrous cord.

In **suppurative phlebitis,** which occurs especially in connection with infection (erysipelas, metritis), the inflammation, though starting in the same way in the vascular coats, advances rapidly to suppuration, and the intima, lying in contact with the resulting thrombus may become itself the seat of the suppurating process. Cases of this kind are almost of necessity in the nature of an infection and the danger is greatly enhanced. Small abscesses formed in the vascular coats may burst into the vein and passing on with the blood produce general infection (pyæmia). Even when the pus enters the vein at a point covered by the thrombus, it may escape by the partial loosening of the clot from the serosa, or through the interior of a honey-combed coagulum and thus lead to general infection. This is especially liable to follow in erysipelas and metritis, in which the tendency as in the solid tissues is to diffuse suppuration without any investing limiting membrane. There are other forms of bacterial colonization of the vascular walls, of ulceration, and of the extension of morbid growths into or through the venous walls, producing inflammation more or less localized, and leading or not to general infection. The presence of phlebolites in the vein is a conceivable source of phlebitis, though no such case has been so far recorded.

The *symptoms* in localized cases of simple adhesive phlebitis may be purely local. The vein if within reach may be felt like a firm, rounded cord, which extends in a direction from the heart. If there are no free anastomosis with neighboring veins on the distal side of the thrombus, venous congestion and dropsy of the tissues ensue, and in some cases moist gangrene. When, however, such anastomosis is abundant these peripheral symptoms

may be absent, especially if the affected vein returns blood from a higher level than the heart, and then the symptoms are confined to the vein and its immediate surroundings. From lymphangitis which shows similar hard cords, it is distinguished by the absence of an extended network of diseased vessels, by the lack of a diffuse, doughy swelling, and by the fact that the adjacent lymph glands remain free from inflammation, pain and swelling. In the more extended cases there is fever, which may be of a very high type and may merge into pyæmia. In deep-seated cases it may be difficult to identify the disease, but it may be suspected if in the course of erysipelas or metritis there is a sudden increase of fever with pain and swelling, and distension of veins leading into the part.

The *treatment* of idiopathic phlebitis is largely that of the particular infecting disease on which it depends. In simple cases due to trauma absolute rest and the application of ice and antiseptic solutions, or where these cannot be applied, the use of antiseptics internally, will be indicated. Hyposulphite of soda and sulphide of calcium are especially indicated. From the early days of veterinary medicine, flying blisters of Spanish flies, over the inflamed vein or veins have proved very successful, and under the lead of Nonat the same was in 1858 and since adopted with gratifying success in the human subject. Abscesses formed in accessible situations should be promptly opened and treated antiseptically, and swelling of the affected part should be checked by elevated position, or if that is impossible, by a smoothly applied bandage. Rubbing and active movement are dangerous, as tending to detach clots which float off to start new emboli and inflammations in the lungs.

VARICOSE VEINS. DILATED VEINS WITH ALTERED WALLS.

Rare in animals. Angioma Varix. Superficial. Deep. Causes, obstructed circulation, compression, congestion. Symptoms, enlargement, elongation, tortuosity of veins, stiffness, lameness, complications. Treatment, compression, coagulants, cauterization, ligature.

Varix is not so common in the lower animals as in man, and is generally observed in the superficial veins, so that it comes under the domain of surgery. In the form of angioma, which affects the veins, there is extensive dilatation and elongation, but it involves a large group of connecting and anastomosing veins, whereas varix usually affects but one or a few connecting vessels. In the horse the most common seat of varix is in the saphena vein, as it passes obliquely over the inner side of the hock. Less frequently it appears on the flank or other superficial part. In cattle the mammary veins are the most frequent seat. Varices, however, occur also in deep-seated veins and in connection with normal venous plexuses, as in the buccal, palatal, and peneal. Anatomically they may be simple fusiform dilatations, as in the saphena; dilated, elongated and tortuous, branching trunks, as in the mammary veins; or dilated veins with thickened walls and pouch like dilatations.

Causes. There is usually some obstruction to the circulation through the affected vessel, it may be by pressure by a tumor, or a constrained position, obliteration by a phlebitis and thrombus, extension of inflammation from adjacent organs, increased blood pressure by gravitation, or from diseased heart or lungs. Whether from the extension of contiguous inflammation, from external pressure, or from blood tension, the morbid process has much in common; the circulation and nutrition in the vascular walls are interfered with, degenerations set in (softening, fatty, connective tissue), which predispose to dilatation under the blood pressure. The pouch-like dilatations of the jugular consequent on bleeding, are essentially traumatic. The impaired innervation which lessens the resistance of the vascular walls is not to be forgotten. Varix of the saphena is usually an attendant or sequel

of tibio-tarsal synovitis, and is the result of combined pressure and congestion. Mammary varices are manifestly connected with the coagestion and exudations which affect the udder and environment at the time of parturition, or with a casual mammitis.

The *symptoms* in superficial vessels are visible enlargement, and often elongation and tortuous direction of the vein or veins, with or without tenderness. Deep-seated varicosities may be attended by stiffness of the part and a halting in progression with or without pain on pressure. These cases may recover spontaneously as the result of adhesive phlebitis, or they may develop phlebolites, suppuration, inflammation, ulceration and hæmorrhage.

Treatment. Superficial varices have been treated by compression, cauterization, coagulating injections, and ligature. It is not often that interference is demanded but in such cases, pressure with elastic bandage having failed, ligature with antiseptic precautions is indicated.

PHLEBOLITES. CALCAREOUS BODIES IN THE VEINS.

Nature. Location. Mode of formation. Calcareous plates in two inner coats. Phlebotomy. Altered sanguification. Treatment. Extraction.

Calcareous bodies have been repeatedly found in the veins of man and several observations of the same kind have been made in the horse. Spooner found them in the abdominal veins and Simmonds in the jugular. Much difference of opinion has existed as to the mode of formation of these bodies whether by calcareous deposit in a coagulum or by degeneration of a neoplasm in the vascular wall. Andral held the latter opinion, and Tiedemann and Cruveilhier found the bodies connected to the inner coat of the vein by a fine membrane. Morton's cut of one of Simmonds' specimens (Calculous Concretions) shows a structure in successive layers having their centre at one end, evidently corresponding to a former connection by pedicle. Cornil and Ranvier says "sometimes there are seen in chronic varices, calcare-

ous incrustations in the form of plates, nodules or spheres with concentric layers . . . calcareous infiltration is seen in the form of spheres or phlebolites in the varicose diverticula. An extensive calcareous induration several centimetres in length, is also sometimes observed, the vein being transformed into a calcareous tube with the ramifications also varicose.

The calcareous plates of the vein are developed in the fibrous and internal portion of the middle coat. At the beginning they consist of granules deposited in the fasciculi of the connective tissue or between them ; these soon unite and form transparent plates with granular striæ."

Phlebolites in the jugular suggest a connection with the pouch-like dilatations, and transformations in the vascular walls that have been subjected to phlebotomy. It is probable however that there is usually a morbid condition of sanguification and nutrition which predisposes to their formation. In Simmonds' case the jugular was impervious below the bodies, there was hepatitis and arthritis of the fetlock joint.

When recognized during life these may be extracted with due antiseptic precautions. If the vein can be dispensed with it may be ligatured above and below, if not an attempt may be made to preserve it, extracting through a clean cut longitudinal incision and securing as perfect coaptation of the edges of the wound as possible.

HÆMORRHAGE.

Arterial, venous and capillary hæmorrhage belong almost exclusively to the domain of surgery. Internal hæmorrhages will be considered in connection with the organs in which they take place.

HÆMOPHILIA.

Definition. Causes, lack of plasticity of the blood, thin walls, blood tension, cardiac erethism, hypertrophy and neurosis. Sex. Heredity through the female. Treatment, depletive, styptic, astringent. Transfusion.

This is a constitutional infirmity, usually hereditary and characterized by the occurrence of profuse and continuous bleeding as the result of otherwise insignificant injuries or even apart from any recognizable lesion. It has been attributed to a slow coagulation of the blood, but at the start of a hæmorrhage the blood is rich in corpuscles and coagulates firmly. It has also been ascribed to extreme tenuity of the vascular walls, but this has only been met with in a certain proportion of the cases. Another potent factor is a permanent over-filling of the blood-vessels (Immermann, Delafield, Prudden). The same writers attach importance to cardiac erethism, cardiac hypertrophy, and certain neurotic influences which temporarily increase the habitually congestive diathesis. In man the majority of victims have been males, perhaps because most subject to traumatisms. On the contrary the hereditary transmission is mainly through the female members of the family. The families are very prolific, a condition counterbalanced by the death of the majority of the victims at an early age. Among the lower animals it has been observed in horses consequent on castration (Siedamgrotzky, Kohne, Friedberger and Fröhner), setoning (Kohne, Dieckerhoff), and an ulcer of the leg (Kohne).

Treatment consists in combating plethora and constipation by saline purgatives. The subject should be carefully protected from injuries. Locally use styptics such as matico, muriate of iron, tannin, alum with pressure. Internally ergot, lead acetate, iron chloride, tannin, alum, or muriate acids. Transfusion is a denier resort.

DISEASES OF THE BLOOD.

Obscurity of blood changes. Red globules, biconcave, embryonic. Source. Escape of immature red globules. White globules, eosinophile, neutrophile, uninuclear, multinuclear, lymphocyte, granular amœboid, strap-nucleated. Conditions of increase. Relation to microbes and their products. Blood plates. Destruction of red globules in the liver. Numbers in animals, in different vessels and conditions.

The blood is the common medium through which all nourishment is conveyed to the tissues, all material to the glands for secretion, or transformation, and all effete matter to the various emunctories for elimination. It is beside the carrier of oxygen for the respiration of the tissues, and the seat of changes, as yet little known, effected through the white globules. The activities of the various processes, carried on by the fixed tissue cells and nuclei would suggest, that any disease or derangement of these processes would be at once cognizable in changes shown in the blood. Yet so perfect is the balance of sanguification and elimination on the one hand, and of the remaining vital processes on the other, that it has hitherto been impossible to detect in the blood such changes as would identify the great majority with morbid processes. Some morbid changes are however recognizable and it is important that the significance of these should be known.

The blood is a liquid, consisting of a plasma holding in solution serum albumen, serum globulin, fibrine-forming elements, sugar, urea, salts, and a variety of other soluble bodies, and floating a series of semi-solid organized bodies, the red and white globules.

The red globule is however seen in two distinct forms. 1st. The biconcave disc, non-nucleated, containing a colorless stroma, and the coloring matter—hæmoglobin. 2d. The embryonic red globule, large, nucleated and rarely biconcave. The latter is found in the blood of the fœtal man or animal and persists to a slight extent for some time after birth. These are believed to be formed from the embryonic cell and from the cells of the embryonic liver, spleen, and marrow, whereas after birth they are derived from the marrow cell, and in healthy conditions pass the nucleated stage before they escape into the blood. In pathological anæmia and

after severe hæmorrhages they escape more rapidly, probably from both spleen and marrow, and appear in the blood, even of the adult, of the gigantic size and nucleated appearance of the embryonic red globule.

The white blood globules (leucocytes) are spherical, about twice as large as the red globules, and are readily divisible by the acid eosin stain into two kinds: 1st. Cells which are deeply stained by eosin—eosinophile; and 2d. Cells that do not take on the eosin stain—neutrophile (Ehrlich).

Howells further divides these white globules into uninuclear and multinuclear. Of the uninucleated he describes three varieties: *a*. The lymphocyte which is non-granular and without amœboid movement; *b*. The granular cell with a plotoplasmic envelope and amœboid movement; and *c*. The granular with strap-shaped, horseshoe or spiral nucleus. Like Lovet he considers the multinucleated as on the way to disintegration.

We cannot as yet speak with confidence of the pathological significance of these respective forms of white globules, but they increase greatly in numbers in connection with certain diseases of lymph plexus, and glands, of the spleen and other blood glands, and in foci of inflammation, and they perform most important functions in connection with the resistance of microbian invasion and in elaborating the antitoxines which confer immunity from second attacks.

The next form of blood solids are the *blood-plates* of Bizzozero, the hæmatoblasts of Hayem. These are nucleated (Semmer) discoid, less than half the diameter of the red globules, and cluster together in granule masses when the blood is drawn. Their true significance is uncertain though it has been surmised that they are intermediate corpuscles (Semmer), that they are the disintegrated nuclei of the leucocytes, and that they furnish paraglobulin to the circulating blood (Schmidt, Howell).

The liver is one centre for the destruction of red blood globules and in the blood of the hepatic vein there may be a reduction of a million to a million and a half of red globules per cubic centimeter, as compared with the portal vein.

Malassez gives 4,500,000 as the number of globules in a cubic millimeter of blood (dog and horse 7,500,000, Nocard). The white globules are to the red in the proportion of about 1 to 300

(domestic animals 1 : 800, 1 : 1100, Nocard). The variation in different parts of the vascular system and at different times of the day is striking and suggestive.

In the blood of the splenic vein 1 : 60 ; in the hepatic vein 1 : 170 ; in the portal vein 1 : 740 ; in the morning, fasting 1 : 716 ; half an hour after breakfast, 1 : 347 ; in boys 1 : 226 ; in girls 1 : 389 ; in men 1 : 346 ; in old men 1 : 381 ; in menstruating woman 1 : 247 ; in pregnant woman 1 : 281, (Stricker).

PLETHORA. POLYÆMIA.

Definition. Transitory only. Causes, kidney disease, drinking freely, rich feeding, profuse secretion, polycythemia, hyperalbuminosa, excess of fibrine, sugar or fat. Ratio of blood to body. Variations of globules. Symptoms, general, local. Appearance of blood. Prevention. Treatment.

Definition. An excess of blood, of the blood globules, or of the albuminoids.

Formerly accorded an important place in pathology, plethora has been entirely eliminated from some recent works. The actual amount of blood varies greatly at different times, rising after a free consumption of food or drink, and falling during a period of abstinence. A healthy activity of the secretory and excretory organs secures a fair uniform average in the plenitude of the circulatory system. Moreover, large variations are not in themselves rapidly injurious. Worm-Müller and Cohnheim introduced into dogs ten to twelve per cent. of the body weight (fifty to eighty per cent. of their blood) of canine blood without inducing fatal results. More than this was fatal. In non-fatal cases a reduction to the normal standard is speedily secured.

But we cannot count on absolute immunity in all circumstances. Disease of the kidneys, or drinking water to excess, determines a surplus of water and urinary salts (serous plethora, polyæmia aquosa). In cases of rapid gain in condition from rich feeding, and above all after profuse watery secretion (diarrhœa, diuresis,

perspiration), the red globules are relatively increased (plethora polycythæmica). After hearty feeding there is a large increase of albumins (plethora hyperalbuminosa). Fibrine-forming elements are apparently in excess during rheumatism, pneumonia and other acute inflammations. Sugar is in excess after a saccharine or farinaceous meal, fat in obese individuals, after consumption of fat, after injuries to the bone marrow, and after severe diseases with much destruction of albumen.

The ratio of blood to the body weight is: In birds 1:12; in Guinea pig 1:19; in rabbit 1:20; in cat 1:21; in dog 1:17; in horse 1:18; in sheep 1:24; in pig 1:26; in ox 1:29 (Colin). As showing the variation under even different normal conditions of the system Bollinger found the blood but 2.2 per cent. of the body weight in a fat pig, whilst it was 13.5 per cent. in a draft horse. Colin found it 2.4 per cent. in the fat ox instead of the usual 3.4 per cent.

The excess of red globules and usually also of albumins is seen as a temporary condition, in lean but vigorous animals put suddenly on an abundant diet, rich in assimilable albuminoids, in working animals, put in confinement to feed, and above all in high conditioned cows after an easy parturition, when the uterine blood has been suddenly thrown on the general circulation and the emunctories have failed to establish a balance. Also in the lymphangitis occurring after a day or two of rest, in a horse that has been hard worked and heavily fed.

It should be borne in mind that the number of red globules varies considerably in the different animals. In the dog it was by weight 148.3 grammes per 1000; in the pig 105.7; in the horse 102.9; in the ox 99.71, and in the sheep 98.2 (Andral, Gavarret, Delafond). By count the horse has 5,500,000 per cubic millimeter (7,500,000, Nocard); and the dog 5,000,000.

Symptoms. Under a sudden dangerous increase of the volume or the organic elements of the blood, there are usually dulness, lassitude, dropping of the head, strong, full, hard pulse, extra force in the heart beats, thirst, elevated temperature, and redness of the visible mucous membranes. At first there is no indication of local disease, but unless relief comes by free secretion some local complication is likely to ensue. This may be epistaxis, congestion or apoplexy of the brain, parturition fever, lymphangitis,

or congestion of some internal organ, etc. A drop of blood colors deeply the finger or other object, it clots firmly in three to five minutes, and shows more than usual of a buffy coat.

Treatment. As dangerous plethora is usually a very transient condition the main attention should be given to *prevention*, in keeping the diet low and the emunctories active in high conditioned parturient cows; in lowering the diet and securing free secretion, or in giving exercise to high fed, hard worked horses that have been laid off work; in changing only by slow gradations thin, vigorous animals to a rich diet, etc. When the danger is imminent prompt relief can be secured by the liberal abstraction of blood. Purgatives, diuretics, and restricted diet may be applied to less urgent cases.

HYDROÆMIA. ANÆMIA OLIGÆMIA.

Definition. Causes: bleeding, watery repair, hydroæmia, repair of globules, changes in red globules, in bone marrow. Cause of chronicity: profuse secretions,; moplasms; parasites; chronic exhausting diseases; defective diet or hygiene; diseases of jaws or throat: overwork: toxic substances. Symptoms: pallid mucosæ, weakness, perspiration, soft tissues, small pulse, palpitation, anæmic heart—arterial and venous murmurs, depilation, indigestion, costiveness, urine clear, abundant, emaciation. Lesions: blood poor in globules, embryonic, and other abnormal red globules, fatty degeneration, blood-clot. Treatment: remove causes, diet, hygiene, sunshine.

Definition. Bloodlessness; Deficiency of blood; Lack of red blood globules. The last named is the condition to which the term is habitually applied.

Causes. Anæmia is not so much a disease, as a result of a great many debilitating and exhausting conditions. **Hæmorrhage** the most direct cause of anæmia determines at first an actual lack of blood (oligæmia) and of blood pressure, which may be sufficient to cause fainting and death. In case of survival the amount of blood is rapidly made up by absorption from all available sources of liquid in the economy, but the blood so restored is essentially hydroæmic having an excess of water and a lack of glob-

ules and dissolved solids. If however the loss has been moderate the quality may be restored in a few days. Buntzen found that after moderate bleeding the volume is restored in a few hours; after a profuse hæmorrhage in 24 to 48 hours. After bleeding to 1.1 to 4.4 per cent. of the body weight the increase of the red globules may be noticed after 24 hours, and is completed in 7 to 34 days. It is noteworthy that during this repair the bone marrow becomes much redder and more cellular, and that new red cells found in the blood are nucleated (Neumann) and contain less hæmoglobin (Ott). The absence of hæmoglobin is nearly in proportion to the amount of the hæmorrhage (Bizzozero, Salvioli). If the hæmorrhage is slow and continuous this repair is counterbalanced and the anæmia is much more persistent.

Profuse secretion as of milk (cows, goats, ewes, bitches, on poor feeding), of liquid fæces, urine, or pus often determine a marked and even dangerous anæmia.

The rapid growth of multiple **tumors** as of *melanosis* in gray horses has been noticed to cause profound anæmia (Bouley).

Perhaps no cause is more potent than the attacks of **parasites** and especially such as live by sucking the blood. The numerous strongyli of the lungs, stomach, and intestine, the tricocephalus, and allied round worms, the trematodes of the liver, and the cytodites of birds furnish striking examples of the bloodless and debilitated condition which they may produce. In man ankylostomata causes anæmia in Egypt, Italy (St. Gothard) and elsewhere, and bothriocephala in different countries.

Chronic exhausting diseases especially those which affect the digestive organs and mesenteric glands are prolific causes. So with Bright's disease.

Connected with these are **defects in diet or hygiene.** Starvation, unsuitable, innutritions, or indigestible food, too laxative food, damp, dark, draughty or unventilated stables, and irregularity in feeding, watering and work are all potent factors in inducing anæmia.

Diseases of the masticatory apparatus (broken jaw, diseased teeth,) preventing the preparation of food, and pharyngeal troubles interfering with deglutition are other causes. Finally overwork is not to be forgotten.

Toxic anæmia may occur from the ingestion of lead, mercury, or arsenic.

Symptoms. These may be little marked at the outset in slowly developing cases. Extra pallor of the mucous membranes, fatigue and even breathlessness on slight exertion, a small, weak, pulse, with a tendency to become rapid, with violent heart beats, when excited.

At a more advanced stage the mucosæ, especially the buccal, are pale and thin, the muscles are soft, flabby and weak, fatigue and perspiration are easily induced, the feet are advanced more nearly in the median line of the body, and the toes strike on any obstacles, the pulse is weak, small and quick, and the heart easily excited even to palpitation, and with an occasional anæmic murmur with the first heart sound. Arterial and venous murmurs may be present. The hairs are easily detached. Appetite and digestion fail, there is costiveness, a full secretion of urine of a clear aspect, the subcutaneous fat disappears and the skin feels thin and limp (paper skin in sheep), the hair dry and lusterless, the wool flattened (clapped). The weakness and emaciation go on increasing and dropsies appear in the limbs, under the trunk and jaw and in the internal cavities.

Pathology. Lesions. Apart from the causes, the morbid conditions are mainly found in the blood. The watery state of the blood, the lack of red globules (even to but 2,000,000 per cubic millimeter), the absence of albumen (76 per 1000 in place of 83), the loose coagulum with excess of buffy coat, and the excess of serum are characteristic. The presence of large, nucleated (myelogenous) red cells, of spherical bodies smaller than the normal red cells (microcytes), and of irregularly shaped red calls (poikilocytes) is characteristic, the latter especially of pernicious anæmia. As the disease advances fatty degeneration of heart, liver, kidneys, and other organs are complications and tend to aggravate the disease, by counteracting repair of the globules—thus establishing a vicious circle. All the organs are pale and flaccid, the arteries empty, the veins contain a little blood, forming pale clots. In the cases considered, all the result of another disease, the lack of blood and of the solid and vital elements in that which remains, entails imperfect function in all the vital processes, including sanguification itself, and in this way an anæmia once established tends to perpetuate and aggravate itself.

Treatment. The anæmia above considered being largely symptomatic, or resultant from other diseases, the first consideration as regards both prevention and treatment is to prevent or cure such diseases. Where dietetic or hygienic, a liberal diet, and good hygiene will meet every demand in the early stages. In the warm season an open air life is most important. In case of a drain by over-secretion (milk) this must be judiciously checked. In bitches it will often be needful to wean several of the puppies. A rich and very digestible diet (oats, beans, linseed, oil meal, milk, gruel), in small compass, and suited to the genus and individual, with iron and bitters, and in the herbivora carminatives, will suit many cases. Muriate of iron, with strychina or nux vomica; iron sulphate, sodium chloride and nux; or dialysed iron, or some other soluble ferruginous salt, with quinia, gentian, or some other bitter will serve a good purpose. For the dog saccharated carbonate of iron or citrate of iron and ammonia with quinia or strychnia, in pill form, is convenient. With poor digestion muriatic acid and pepsin may replace the iron at first. Beef teas may often be given with advantage, even to the herbivora, and injections of defibrinated ox blood night and morning have proved of service. In extreme anæmia, as from hæmorrhage, transfusion, or its equivalent, must be resorted to. A normal saline solution (0.6 per cent. NaCl), boiled, may be thrown into the peritoneum or subcutaneous connective tissue, or defibrinated blood, may be injected into the peritoneum. Transfusion is the dernier resort.

PROGRESSIVE PERNICIOUS ANÆMIA. IDIOPATHIC ANÆMIA.

Definition. Causes, obscure, faulty diet, hygiene, microbes, glycerine, pyrogallic acid, hæmoglobin, deranged sanguification, parasitisms. Symptoms, of anæmia of obscure origin. Treatment as for anæmia, special measures, for intestinal fermentations, dietetic.

Definition. Anæmia which is without any pre-existing appreciable cause.

Causes. As in the corresponding disease in man the real starting point of pernicious anæmia is unknown. Faults in diet and in general hygiene have been adduced, and while in Berne this appears to be sustained, in Ireland, in the poorest classes, the disease is little known, and in Montreal, it find its victims largely in a class of well to do artisans (Osler.) In the domestic animal it is described on all soils, and on the most varied dietary (Bouley and Reynal). Zschokke and Friedberger and Fröhner in cases occurring enzootically in stables, found a minute bacillus in the patients, which would remove these cases into the list of symptomatic anæmia. The same is true of the anæmia (Surra) of horses and mules in India and Siberia, in which Evans, Burke, Steele and Ignatovsky, found a motile spirilloid organism which destroys the red globules. Other forms that are apparently purely idiopathic have been attributed to a failure in the cytogenic processes in the bone marrow especially. Back of this we know only of the various debilitating causes in food, hygiene, building, location, work, etc., operating on a specially susceptible system, in which, once started, the morbid process tends to perpetuate itself and increase.

Ponfick induced anæmia experimentally by the intravenous injection of glycerine, pyrogallic acid, solutions of hæmoglobin, etc., which dissolve the blood globules. This suggests the probable pathogenesis by the production of unidentified blood solvents in cases of deranged sanguification, but it still leaves us in the dark as to the exact seat of such derangements (liver, blood glands, bone marrow, etc.) and as to the cause, parasitic or otherwise, which determines such disorder. Pathological investi-

gation has enabled us to differentiate, according to their respective causes, a number of diseases (distomatosis, lung worms, ankylostomiosis, internal acariasis, chronic trichinosis, strongyliasis, etc.,) which were formerly classed as anæmias, and it seems altogether probable that the onward progress of medicine will enable us to go farther in the same direction and to allot the remaining unclassed anæmias to their proper etiological places. Some may be unrecognized helminthiasis, others microbian disorders, and still others, disorders in nutrition and sanguification from different causes.

The *Symptoms* are those of other forms of anæmia, but being more obscure in origin and therefore less open to corrective treatment, it is more likely to grow to an extreme development and fatal issue. The weak pulse, irritable heart, debility, unsteady walk with the hind limbs, hurried breathing and sweating under exercise, watery, puffy eyes, dropsies in limbs and dependent parts of the body, progressive emaciation, and weakness are even more marked than in symptomatic anæmia.

The *treatment* is in the main as for the other form alike in its hygienic and medicinal bearing. In man recoveries have taken place under arsenic combined with the iron. Arseniate of potash and ferrum redactum in pill form, or tincture of chloride of iron, and Fowler's solution in food or water, continued for a length of time. Phosphated pepsin, and peroxide of hydrogen have seemed to do well in some instances, and phosphorated oil is another resort. W. Hunter attributes idiopathic anæmia to toxins derived from microbes in the alimentary canal, as Sir Andrew Clark ascribes chlorosis to a similar cause. He prescribes beta-naphthol as the least soluble and best antiseptic, in a dose of 5 grains daily in mucilage for man (1 drachm for horse or ox). Hunter further found that a farinaceous diet protected the globules against destruction while a nitrogenous diet favored this. It may be noted that long ago Delafond attributed anæmia in animals to the extension of the use of artificial fodders of the natural order leguminosæ which are rich in nitrogen.

CHRONIC ANÆMIA. DROPSY IN CATTLE AND SHEEP.

Definition. Causes, parasitic and microbian. Symptoms. Treatment.

Definition. A progressive anæmia in ruminants and other animals, resulting in general anasarca, and dropsies of the internal cavities.

In veterinary works published on the European Continent this affection is given a special place apart from the same train of symptoms which mark distomatosis, taeniasis, and strongyliasis. The disease is described as prevailing in wet years, after inundations, when the vegetation is rank and aqueous, and of course largely aquatic, in animals that are turned out in early morning before the dew has evaporated, in the conditions, in other words, that favor the ingestion of parasites. It prevails also in work oxen fed on the refuse of sugar factories (beets, turnips) in which the nitrogenous materials are held to be deficient, but in Great Britain where cattle are often fattened on an exclusive diet of turnips, containing even a larger proportion of water, this non-parasitic disease is unknown. It is also ascribed to close, ill-ventilated, unwholesome buildings, and to over-kept and tainted fodder, and so far as a separate disease exists, it seems more reasonable to charge it to the toxins produced by bacterial ferments or cryptogams than to causes which elsewhere appear to be inoperative.

The *symptoms* are essentially those of distomatosis, and the *treatment*, apart from the parasiticides, is the same. When helminthiasis can be certainly excluded *prevention* would include the avoidance of the factory refuse, especially when in a state of decay.

MELANÆMIA. BLACK PIGMENT IN BLOOD.

Definition. Melanin, in normal tissues, abnormal. Melanosis. Bisulphide of carbon subcutem. Decomposition of hæmoglobin in leucocytes. Coloration of tissue.

Definition. Accumulation of granules and scales of blood pigment (melanin) in the circulating fluid, and in various organs (spleen, liver, bone marrow, brain, etc).

Melanin—$C_{44\cdot2}$, H_3, $N_{9\cdot9}$, $O_{12\cdot6}$—or black pigment (a close relative of hæmatin) occurs physiologically in epithelium (choroid, retina, iris, in the deeper layers of epidermis, and on the surface of the dog's lung and of the sheep's brain) and in connective tissue corpuscles (lamina fusca of the choroid).

Pathologically it is found in the blood of the victims of malarious fever, often in great abundance, and in the spleen, liver, bone marrow, brain, lymph glands and some other organs. It is formed abundantly in the black pigment tumors (melanosis) of man and animals, and in extensive melanosis is present in the blood of both man and horse (Schimmeln). So far it has not been found in connection with the extensive destruction of red globules which takes place in anæmia. Schwalbe has developed malanæmia experimentally by the hypodermic injection of bisulphide of carbon in rabbits.

According to one view the melanin is produced in connection with the destruction of red globules in the liver, spleen, etc., and is thence carried into the blood. This is in keeping with the local formation of the pigment in melanosis. Arnstein however urges that in malarious cases the destruction of the red cells takes place in the blood, and that the hæmoglobin, absorbed into the leucocytes, is transformed into melanin, and finally deposited in the tissues by the migrating white corpuscle. Why the hæmoglobin set free in anæmia is not similarly transformed, does not appear. The pigmented organ may be quite black in the immediate vicinity of the blood vessels, and in its general aspect in chronic cases reddish brown, dark gray, or dark olive.

LEUKÆMIA. LEUCOCYTHÆMIA.

Definition. Nature. Result of other morbid processes. Leucocytes polynuclear. Lymphatic leukæmia. Spleno-myelogenous leukæmia. Leucocytes in each. Loss of amœboid movement. Charcot's crystals. Hæmatoblasts. Cell increase in bone marrow. Myelocytes. Enlarged spleen and lymph glands. Hæmorrhages. Lymphoid growths. Susceptible genera. Causes obscure. Symptoms, pallor, listlessness, weakness, apnœa, sweating, thirst, emaciation, weak circulation, anæmic murmur, enlarged spleen, bleedings, diarrhœa, dropsy, excess of white globules, reduction of red globules, buffy coat, beaten fibrine is granular, china-white mucosæ, hurried breathing, stertor, deranged digestion, marasmus. Duration. Not inoculable. Treatment, not hopeful, as for anæmia, good hygiene, tonics, stimulants, antiseptics.

Definition. An excessive and persistent increase of the white blood globules, and associated with enlargement of the spleen, lymph glands or bone marrow.

Nature. This must be distinguished from the leucocytosis which occurs during digestion, or that which attends on tuberculosis, glanders, pneumonia, and other extensive inflammations and profuse suppuration. These forms are transient and the cells are of the polynuclear variety. The cells of leukæmia are various in character, but bear some relation to the particular organ which is the seat of hypertrophy or morbid process.

"In **lymphatic leukæmia** the increase in the number of leucocytes is due to the mononuclear lymphocytes, especially of the small form. As many as ninety-five per cent. of the colorless cells may be of this form. In **Spleno-Myelogenous leukæmia** the eosinophile cells may be especially increased in number, and there are also large leucocytes coming apparently from the marrow of the bones, and called myelocytes. These most nearly resemble the larger lymphocytes of normal blood, but they are usually larger. They have a single large nucleus which stains feebly and their bodies may show neutrophile granules. Larger and smaller nucleated red blood cells may be found in spleno-myelogenous leukæmia. The leucocytes are frequently in a condition of fatty degeneration, and there may be a decrease in the number of red blood cells." (Delafield and Prudden). In splenic leucocy-

thæmia blood plates may be absent and in lymphatic leucocythæmia they may be in excess.

Cafavy claims that many of the leucocytes have lost their active amœboid movements.

Bright white crystals in the form of elongated octahedra are found not only in the blood but in the diseased glands, spleen or marrow (Charcot's crystals). Clusters of discoid hæmatoblasts (blood plates) are present in the blood in variable numbers (Schultze's granule masses).

The bone marrow is marked by an accumulation of spheroidal cells, which tend to pass into a condition of fatty degeneration. Most of them are colorless, larger than the lymphocytes of normal blood and have one large often vesicular nucleus, staining less highly than the lymphocyte nuclei, and with neutrophile granules in the protoplasm (**myelocytes**). There are besides, nucleated red blood cells, spheroidal cells, containing red blood cells, and Charcot's crystals. The marrow may be uniformly red, mottled gray and red, gray, grayish yellow, or puriform (Delafield and Prudden). This may affect one or many bones. The affected spleen is usually much enlarged, at first uniformly, later unevenly, firm or softened, and with thickened white capsule. The cut surface is smooth, brownish red, or yellow, with white lines (thickened trabeculæ) and indistinct Malpighian corpuscles. It contains glutin, glycocoll, hypoxanthin, zanthin, leucin, tyrosin, and lactic, acetic, or formic acids.

The affected lymph glands are somewhat enlarged, red or gray, exceptionally, softened or caseated and otherwise contain an excess of leucocytes.

Slight hæmorrhages may appear in any of these structures. Lymphoid growths may appear in a number of other organs as the liver, heart, lungs, kidneys, bowels, tonsils, the different blood glands, the serosæ and the retina.

Genera affected. It has been seen mainly in dogs, but also in horse, ox, pig, cat and mouse. Nocard has collected the following cases: horse 9, cattle 6, pig 5, dog 22, cat 1.

Leisering found a horse's spleen weighing 28 kilogrammes. Johne found a pig's spleen of 2.4 kilogrammes.

Causes. The primary causes of leukæmia are unknown. As in anæmia all unhygienic conditions are invoked as causes. That

it is not due to simple hypertrophy or irritation of the leukogenic centers is plain, as it does not follow on ordinary diseases and injuries of these parts, but what is the precise nature of the morbid cause has so far eluded us.

Symptoms. Pallor of the visible mucous membranes, listlessness, lack of energy and endurance, breathlessness and perspiration on the slightest exertion, ardent thirst, rapidly advancing emaciation, unsteady gait, stiffness or lameness, lies most of the time, walks with pendent head, and jaws open, small, weak pulse, anæmic murmur in the heart, enlarged lymph glands, or spleen felt beneath the left lumbar transverse processes in the ox, or in the left hypochondrium in the horse. Bleeding from the nose or elsewhere, slight hæmorrhage into the conjunctiva, irritable conditions of the bowels, diarrhœa and dropsies are suggestive. The blood when obtained in epistaxis or drawn by a needle prick may be pale rose, brownish or grayish brown instead of red, and under the microscope shows the enormous excess of leucocytes—the ratio to the red being sometimes 1:2, or even more, in the human subject. In the domestic animals the following ratios have been made by actual count: 1:85 (Leblanc and Nocard), 1:50, 1:45 (Mauri), 1:20 (Nocard), 1:15 (Siedamgrotzky), 1:12 (Forestier and Laforque). The normal average for the domestic animal according to Nocard is 1:900. This great relative excess of white globules serves to distinguish this malady from anæmia, and its persistency is a means of diagnosis from transient leucocytosis.

The red globules are always reduced in number in the horse and dog to 5,082,000, and even 2,050,000 per cubic millimetre, while the normal is 7,500,000 (Nocard).

In clotting, the blood forms an extensive buffy coat, and in solipedes which normally show this, the blood set in a test tube forms three strata, the upper slightly yellow, semi-transparent and formed of fibrine; a median of a dull, opaque white color and formed mainly of leucocytes and blood plates, and a lower of a violet red and formed mainly of red globules.

The amount of fibrine is variable. It becomes granular when beaten. Albumen is variable but usually reduced.

The visible mucous membranes are bloodless and of a clear porcelain white. The walk becomes weaker, fore feet wide

apart and the hind limbs partly flexed, head and neck extended, and breathing labored. The breathing may be with constant stertor, the bowels torpid and tympanitic, or loose and fœtid, dropsies and hæmorrhages ensue, and the patient dies in complete marasmus.

Duration. The disease may prove fatal in less than a month, or it may last for three, six, or eight months. It is mostly fatal.

Not inoculable. Many attempts have been made to transmit it by inoculation, but in no case with success.

Treatment is not successful. All hygienic measures should be adopted, as for anæmia; open air and sunshine, with protection against chills; the treatment of all complications; iron, bitters, phosphorus, arsenic in particular, electricity to the spleen, massage; oxygen inhalation; and locally, iodide of potassium or mercury, generally and locally.

LYMPHADENOMA. HODGKIN'S DISEASE.

Definition. Relation to leukæmia. Causes. Mainly accessory. State of lymph glands, spleen, liver, bone marrow, intestine, tonsils, thymus, kidneys, liver, lungs, bronchial mucosa, pleura, pericardium, nervous system. Symptoms, as in leukæmia, with adenoid hyperplasia, but little leucocythemia. Relation to glanders. Uric acid, low density, no hippuric acid. Tuberculin and mallein tests. Treatment, as in leukæmia. Excision in cases not constitutional. Phosphorus, phosphide of zinc.

Definition. Hypertrophy of the lymphatic glands with little or no leucocytosis. There may further be lymphoid growths in the liver, spleen, bone marrow and other organs.

The visceral lesions in lymphadenoma do not differ in character from those of leukæmia, and as it does often apparently merge into that disease by the characteristic changes in the blood, it is denied by many that it constitutes a separate pathological entity. In his admirable monograph on leucæmia in the lower animals Nocard affirms their identity. The main excuse for keeping up an alleged distinction, is the frequent absence of leucocytosis, and this often supervenes after the lymphadenoma has existed for some time.

Causes. As in leucæmia, no definite cause can be found in the majority of cases. An accessory cause can sometimes be observed where a local irritation gives rise to swelling of the adjacent lymphatic glands and this goes on to distinct lymphadenoma.

Lesions in the Lymph Glands. The hyperplasia may affect but a single group of glands, more commonly a number of groups, and often nearly all. In one case only of leukæmia in the lower animals, a dog, has Nocard failed to find the lymph glands affected. In the horse he has found the sublumbar glands alone weighing 14.5 kilogrammes, 11 k. and 8 k. They compressed the posterior aorta and vena cava and had caused extensive ascites.

The enlarged glands are white, gray or in case of rapid growth veined or pointed with red ; they may be soft or firm according as the hyperplasia has operated most on the trabeculæ or the cells ; they are homogeneous throughout. The scraping of the cut surface gives a more or less thick milky juice containing a great number of nucleated or double nucleated lymphocytes, free nuclei and granules which stain strongly.

Hardened sections show an enormous development of the follicles at the expense of the medullary walls, and double nucleated white globules packed in a rich reticulum of adenoid tissue, whilst the blood-vessels in the connective tissue are crowded with white cells, and there are slight ruptures, old or recent.

Lesions of the Spleen. These are nearly always present. Leisering found a horse's spleen over three feet long and 28 lbs. weight, and Nocard one of 13 lbs. Bollinger found a pig's spleen 3½ lbs. Siedamgrotzky found dog's spleens over 2 lbs. The consistency is usually firm (sometimes soft in dog). Capsule thickened and white, cut surface dry, reddish brown, granular, Malpighian bodies enlarged like a pea, hazel nut or walnut, with contents as in the lymph follicles. The capillaries are enlarged and crowded with white cells.

Lesions in the Liver. The liver is enlarged in one-half of the cases of leukæmia in the lower animals. It has been found to weigh 20 lbs. in the horse, and 4 lbs. in the dog. It is of a grayish brown, or yellowish brown hue, or light red spotted with yellow, or mapped out by anastomosing grayish white lines. There may be enlargement of the acini, or the formation of little nodes of adenoid tissue, or most commonly in the lower animals,

there is an adenoid thickening of the bands of connective tissue extending in from the capsule. These are filled with white cells which stain deeply with carmine. There may also be slight extravasations of blood and infarcts.

Lesions in the Bone Marrow. These noticed in the pig by Fürstenberg, and in dogs by Siedamgrotzky, consist in increased vascularity, great cell hyperplasia, and formation of adenoid tissue as described under leukæmia.

Lesions of the Intestine. These commence in the agminated or solitary glands, which become enlarged, causing thickening of the mucous membrane, and later grow out into more or less rounded masses of lymph—adenoid tissue up to an inch in thickness. They are quite subject to ulcerations.

Lesions of the Tonsils. Bollinger, Nocard and Siedamgrotzky found these enlarged in dogs in connection with adenoma of the spleen. They were soft, friable, grayish, and consisted of a very delicate and fragile adenoid tissue.

In one case Siedamgrotzky found adenoid hypertrophy of the thymus in a cow, and adenoma of the kidneys similar to that of the liver has been noticed.

Similar adenoid hyperplasia has been found in the lungs, the bronchial mucous membrane, the pleura, the mediastinal and bronchial glands, and the pericardium. In man this has invaded the nerve centres, and it seems that at any point where there is a lymph gland or a lymph plexus this adenoid hyperplasia may localize itself.

Symptoms. The general symptoms of failing health are as described in leukæmia. The particular symptoms of this disease consist in the recognition of the adenoid hyperplasia in the absence of a marked leucocytosis. The submaxillary glands are usually the first attacked, and the disease may, in the horse, be confounded with glanders. There is, however, no pituitary discharge nor ulcer, the glands are enlarged symmetrically on the two sides, and a careful search will usually discover other groups with similar symmetrical enlargement. The parotidean, the pharyngeal, the prepectoral, the prescapular, the axillary, the popliteal, the prefemoral, the post and premammary, and the inguinal should be critically examined. The enlarged mesenteric glands may be reached and detected by the hand engaged in the

rectum, or in the small animals by external palpation, as may also the enlarged spleen or liver.

The adenoid hyperplasia in the chest offers very obscure and uncertain symptoms. The enlarged bronchial and mediastinal glands may seriously interfere with the functions of the vagus nerve, causing, in cattle, disturbed digestion and rumination and tympanies, in horses stertorous breathing, and in the carnivora and omnivora a tendency to vomiting. In animals generally the pressure on the cardiac nerves leads to great irritability of the heart, and violent action under any exertion. The prominent dyspnœa in the advanced stages may be explained by these thoracic hyperplasiæ.

Nocard claims that the urine furnishes most important indications in its low specific gravity (horse 1010), its constant acidity, and in the almost entire absence in that of the horse of hippuric acid. When there is any suspicion of tuberculosis or glanders, the tuberculin or mallein test will decide.

Treatment is essentially the same as in leukæmia, and equally unsatisfactory. Arsenic has in the main given the best results. In the very earliest stages when the granular hyperplasia is confined to one group, excision is advisable. This should be avoided in all cases in which the constitutional symptoms have developed. Phosphorus and phosphide of zinc have seemed beneficial in certain hands. Injections into the glands have so far proved useless.

ACUTE LYMPHANGITIS OF PLETHORA IN HORSE. ANGEIOLEUCITIS.

Definition. Symptoms and causes. Genera affected. Causes of plethora. High feeding. Work followed by rest. Fever, hurried breathing, strong, rapid pulse, anorexia, stiffness and swelling in a hind limb, inguinal glands, connective tissue engorgement, corded lymphatics, suppuration rare. Mild forms. Lesions, in lymphatic vessels and glands. Chronic cases. Nature, plethoric, lymph excess, stasis, excess of cells and fibrine, immunity of the fore limb. Season of prevalence. Climate. Diagnosis, from farcy, erysipilas, etc. Treatment, exercise, friction, resolvents, purgative, bleeding, diuretics, astringents, iodine, pressure, diet. Prevention. Treatment of chronic cases.

Definition. Inflammation of the lymphatic vessels and glands of one limb usually in connection with rest.

Symptoms. This affection is common in heavy draft horses of a lymphatic temperament and kept on high feeding and at hard work. It rarely develops however while the subject is kept at steady work. But if, in the midst of such work, the horse is kept at rest in the stall over one, two, or more days on the same generous diet, he is found shivering violently, with rapid, labored, breathing, high pulse and elevated temperature, symptoms which have been frequently mistaken for those of pneumonia. There is complete anorexia, and often ardent thirst. The patient is indisposed to move and if forced to it shows lameness in one hind limb with an extraordinary abduction of the limb at each step, and sometimes so severe as to prevent his putting his full weight upon it. If an examination is now made high up in the groin close outside the inguinal ring, the lymphatic glands will be found to be swollen, hot and tender, so that under even moderate pressure the leg will be lifted and abducted until the patient threatens to fall on the other side.

A little later the shivering may have given way to the hot stage, with it may be general perspiration, and the swelling may have extended down the course of the saphena vein and lymphatics, as a distinct ridge and the lower part of the limb from the foot to the hock may be filled, dropsical and hot. Unless checked the swelling goes on increasing till the lower part of the limb is two

or three times its natural thickness, and the swelling has extended well up on the thigh. The swelling has a soft œdematous feeling, easily receiving and retaining the imprint of the finger and is not only hot, but excessively tender. From the margin of the swelling, firm, tender, rounded cords are found to emerge passing upward along the line of the saphena vein and its branches toward the inguinal glands. These represent the swollen and gorged lymphatic trunks, and may often be traced for some distance into the substance of the general engorgement.

When the inflammation is violent, suppuration may ensue at one or several centers, but more commonly the engorgement goes on increasing and when the febrile attack has subsided the limb is left permanently enlarged and correspondingly liable to a second attack.

Milder cases are met with which are perhaps even more misleading. There may be little or no rise of temperature, loss of appetite or general constitutional disturbance, but under some change of regimen and particularly after one or two days of rest the subject becomes lame in one hind limb, without any of the usual injuries to account for it. Examination of the groin shows swelling and tenderness of the external inguinal glands, with or without, a tender cord-like swelling running down from them.

Between these two types may be found all grades of lymphatic inflammation with a varied degree of attendant constitutional disturbance.

Lesions. The coats of the inflamed lymphatic vessels are thickened by exudate and the outer coat is the seat of ramified redness with minute spots of blood extravasation. The inner coat is dull, opaque, or even thickened. The vessel is dilated and its walls friable. The contained lymph in the intervals between the valves has coagulated into a very thin diffluent jelly-like clot, which in old standing cases may have become granular. The connective tissue from which these vessels lead is infiltrated with liquid and the lacunar spaces distended. Red patches from blood extravasations are numerous. The external inguinal glands and often the internal and sublumbar are swollen, congested, and the seat of active cell hyperplasia. Abscesses are exceptionally seen.

In chronic cases the lymphatic vessels of the affected limb and

especially of the lower part which is permanently swollen, are enormously increased in calibre (lymphangiectasis), and have their walls correspondingly thickened. The connective tissue is the seat of extensive fibrous hyperplasia, and its interstices are greatly enlarged.

Causes. Nature. This disease has not been sufficiently studied to ascertain what toxic agents are produced in the plethoric condition, under the torpid processes of nutrition and sanguification entailed by absolute compulsory rest. A consideration, however, of the relations of the lymph and lymph vessels and glands to other parts will in part explain the pathology of the malady. The lymphatics take their origin in the nuclear spaces of the various tissues, the anastomosing canals of such pericellular spaces together with the latter forming the actual radicles of this set of vessels. They receive, therefore, the surplus plasma which is not used up by the tissue cells in performing their trophic, secretory and other functions. This lymph carried on by the *vis a tergo*, muscular compression and other movements, is delayed in the adenoid tissue known as lymph nodes, and especially in the lymph glands, in which the proliferation of lymph cells is mainly carried on. Thus the lymph cells are very scarce in the lymph radicles of the connective and other tissues, and are found in greater numbers after passing through the lymph nodes, and in still greater after passing through the lymph glands. But the increase of cells is also in inverse ratio with the rapidity of the circulation of the lymph. When this is rapid the cells are hurried on and there is little time for their reproduction. When slow on the other hand, there is time for cell growth and division in the glandular detention cavities, and the ratio of cells to the plasma is materially increased. Consider next that the multiplicity of cells determines an increase of the fibrine factors, so that the more cells the lymph contains there is the more material for fibrine (Landois), and we have one good reason why under enforced rest the overcharged and congested gland may become the seat of fibrinous coagula or lymphatic embolism. Any overdistension, toxic element, or other cause of disturbance, which deranges the functions of the cell or causes its rapid multiplication by division—as in inflammation—at once sets free the fibrine ferment and determines the coagulation. In the disease before

us we have the overfeeding of an animal having a strong digestion, we have an absolute compulsory inactivity, with a suspension to a large extent of the functions of nutrition, sanguification, secretion, and elimination ; we have in consequence an increase of the blood pressure, and of the solids of the blood and of the plasma of the lymph ; we have a suspension of the great motor force of lymph circulation, namely, the muscular contraction, and we have the consequent tardy movement of the lymph, the great increase of lymphocytes, and the distension and engorgement of the lymph glands. As soon as this has reached a certain stage the congestion and incipient inflammation of the gland determines the precipitation of fibrine, the obstruction of the gland, and of the entire circulation of lymph in the lower part of the limb. The fever, the local swelling, and the subsequent steps follow as a matter of course. This view is sustained by the fact that incipient cases can be cured by muscular movement alone. The rarity of the disease in the fore limb may be ascribed to the greater force of the *vis a tergo*, the lesser height of the lymph column, and the stronger action of the aspiratory power of the chest on the lymphatic vessels.

In addition to the causes mentioned above must be noted the following : The disease is an affection of heavy draft horses, in which the tissues are more lax, and the lymph plexus in the connective tissue of the hind limb is much more abundant. It is common in the heavy English, Scotch and Belgian draft horses, and rare in the English racer, the American trotter, and in the average light American horse. The malady is most frequent in spring and autumn, when the work is hardest and the feeding most abundant. It rarely attacks the horse in steady work, but appears after an idle Sunday spent in the stable (Monday morning disease), or after one or more days of compulsory idleness from heavy rains or other cause. The damp climate of western Europe has probably an exciting influence, as it has in producing the lymphatic constitution. In the same line of thought Zundel says that many cold weather attacks would be prevented by clipping off the heavy coat which keeps the entire system relaxed. In some cases a sudden change of food, and in others musty oats have been claimed as causes.

Diagnosis. Lymphangitis is distinguished from a simple drop-

sy of the limb by the acute fever, the great local tenderness especially of the inguinal glands, and by the tender corded lymphatics that enter these. From cutaneous glanders (farcy) it is diagnosed by the more acute fever, by the swelling of the inguinal glands in the early stage of the disease, followed by the swelling of the lower limb, and by the absence of the hard, comparatively insensible and prone to ulcerate, farcy bud. Farcy buds usually appear on the pastern or fetlock, with more or less swelling of the lower part of the limb, while the inguinal glands are as yet normal in size and without tenderness. From erysipelas, with which this has been confounded, it is distinguished, by the suddenness of the onset, under the circumstances above described, by the high type of fever, by absence of early cutaneous inflammation and the formation of vesicles, and by the fact that lymphangitis commences in swelling of the inguinal glands.

Treatment. In cases that are seen in the earliest stages, before the leg has become badly swollen, recovery will usually take place under active exertion continued for hours at a time. The pumping action inside the hoof during exercise, and the alternate compression and relaxation of the lymph vessels by the muscles, tend to establish a rapid current of lymph, to break up coagula and to re-establish a healthy condition. Friction from below upward on the lymphatic vessels and swollen limb will greatly assist in this restoration. Different agents are employed, such as camphorated spirits or oil, iodine, mercurial, and even blistering ointments. These should not replace exercise when this is possible.

When the fever has set in suddenly and runs very high, the abstraction of four or five quarts of blood, and the administration of a purgative (8 drs. aloes) will be in order. In cases occurring in the same stable and in all other respects apparently identical, the subjects of phlebotomy recovered without any permanent swelling of the limb, while those that were not bled recovered with thickened limb.

In cases so advanced that the limb cannot be used, cold irrigation, with friction, may be applied, and when the irrigation is intermitted one may apply some astringent (vinegar, alum, lead acetate), or an iodine lotion followed by an evenly applied bandage.

The purgative should be followed by full doses of diuretics (nitre, bicarbonate of potash or soda, colchicum, iodide of potassium) until fever and local inflammation have subsided.

As soon as the patient can use the limb, walking exercise should be kept up for several hours forenoon and afternoon.

Throughout the disease the food should be of a light and non-stimulating variety. When appetite returns give at first wheat bran, or roots, or sweet grass in small amount, and do not return to a grain diet until fully recovered and ready to go to work.

After one attack there is always an increased liability to a second, and great care should be taken to give the subject daily exercise, or where this is impossible, to reduce the feed, give a dose of saltpeter, and turn into a yard or roomy loose box on the idle day.

In chronic thickening of the limb, an evenly applied elastic bandage, extending from the hoof up, regular feeding and exercise, washing daily with a weak iodine lotion, and the internal use of iodide of potassium and other diuretics, with bitters and even iron tonics may be used.

INFECTIVE LYMPHANGITIS. TRAUMATIC LYMPHANGITIS.

Infection varied, through wounds, autogenous. Simple irritation, simple lymphangitis. Causes, sun's rays, bruises, other injuries, lymph coagulation from heat, cold, chemical irritants, and coagulants. Germs in blood act on debilitated tissues, lymphatic constitutions, anæmic, overworked, or starved. Insect bites, claws, teeth of carnivora, foul instruments, fingers or clothes. Bloodless wounds dangerous. Distal parts of the limbs exposed. Fresh wound exposed, granulating less so. Most microbes enter by the lymphatics. Symptoms, extension from wound, swollen lymphatics, reticular lymphangitis, tubular lymphangitis, farcy, tuberculous case, slough. Fever variable. General infection. Joint infection. Chronic cases. Lesions. Diagnosis, from phlebitis. Treatment, antiseptics, diet, eliminants, antithermics, blisters, mercurial ointment, iodine, lancing, tonics, massage, bandage.

Under this heading must be named not one specific disease but a group of infections entering by the lymphatic vessels and developing inflammation of their substance. They may be divided into two classes: those caused by infection through external wounds and those in which the poison already in the system becomes localized on a weak or exposed tissue.

A third class must be included, in which there is no recognizable poison but simply a local irritation which leads to coagulation or other alteration in the lymph, or disease of the lymphatic vessels.

This subject belongs rather to surgery than medicine but it seems necessary to contrast it here with the plethoric form of equine lymphangitis. Most of its forms pertain to infectious diseases and will be treated in connection with these.

Causes of Simple Lymphangitis. Formerly many forms of lymphangitis were ascribed to mere local irritation; a superficial form will occur from exposure to the rays of the sun, and an inflammation attendant on a bruise or other injury with unbroken skin, may cause local inflammation of the lymph vessels and enlargement of the adjacent lymph glands. As we have seen above coagulation of the lymph and fibrine embolism may induce local inflammation in the walls, and this may occur in connection with

excessive heat or cold or the presence of chemical irritants and coagulants. These cases are however rarely serious and the tendency to-day is to trace nearly all cases to infection, from germs already present in the lymph or blood, or introduced through a wound or sore. The effect of germs already circulating was shown in the beautiful demonstrations of Chauveau in regard to calves subjected to castration by subcutaneous torsion (bistournage). In the healthy calf the simple operation gave rise to little disturbance. The healthy calf injected with septic liquids equally escaped visible trouble. But the calf injected with septic liquids and then subjected to bistournage had a fatal infecting inflammation. There is a strong presumption that, in lymphangitis, starting from an injury with no external sore, the germs were already present in the blood or tissues but were unable to do any serious damage until the injured and weakened part or organ offered an area of lessened resistance to their colonization. Following the same line of thought it has been noticed that animals of a coarse texture, and lymphatic constitution (heavy draft horses and animals raised for the butcher), and such as are debilitated by anæmia, overwork, or poor and insufficient nourishment are above all liable to be attacked by lymphangitis.

The insertion of the septic poison may take place through the bites of insects, the claws, or teeth of carnivora that have been devouring tainted or infecting meat, through the lancet or operating instrument of the surgeon, by his fingers or the dust from his hair or clothes. The wound is perhaps more likely to be infecting if it leads to no effusion of blood, but affects only the thickness of the epidermis, as there is less chance for the washing out of germs by the flowing blood, and there is less care to employ antiseptics. Wounds in the feet and lower parts of the limbs are specially liable to infection by reason of their frequent contact with manure and decomposing organic matter in the soil.

A fresh wound, in which the lymph spaces are exposed, is somewhat more open to infection than one that has advanced to the stage of granulation, the layer of unorganized lymph and cells acting as a slight barrier to the passage of the microbes.

Nearly all microbian diseases make their inroad by way of the lymphatics, where the sparse cells fail to establish as active phagocytosis as do the numerous moving cells of the blood. Hence a

number of infectious maladies are primarily and pre-eminently diseases of the lymphatics, as glanders, strangles, tuberculosis, cancer, anthrax, swineplague, etc.

Symptoms. The most common form is where lymphangitis extends from some pre-existing wound—as pricked or suppurating foot, fistula of foot, withers or poll, chafing of shoulder or back, cracked heels, boil, sloughing bruise, etc. The swelling around the sore or injury involves in fact the radical lymphatic plexus in the connective tissue (reticular lymphangitis). When the swelling extends and becomes more tense, with firm, painful sinuous cords running out of it in different directions, and especially toward the nearest lymphatic glands, and when these glands are slightly swollen and tender, tubular lymphangitis is diagnosed. No more striking example can be found than in skin glanders (farcy). The rigid cords extend from the side of the face, from the eye, and nose down toward the submaxillary glands and with more or less adjacent engorgement. Or on a hind limb, or some portion of the trunk, a more or less turgid swelling with one or more firm nodes (farcy buds) and painful, tortuous cords running towards the lymph glands is very characteristic.

A tuberculous case may show an indolent, hard, comparatively insensible cutaneous cord leading toward the jugular furrow, the prescapular, precrural or inguinal glands, and at long intervals softening, fluctuating, bursting and discharging a thick pus. In a carcinoma there is the old, hard, nodular, and finally ulcerating swelling from which the firm cords extend to the mass of steadily enlarging lymphatic glands.

A simpler form is where a bruise by the harness causes a hard, thick, slough, embracing the entire thickness of the skin, from which the firm corded lymphatics extend in different directions. After the slow process of detachment, the local lymphangitis usually subsides under simple cooling or antiseptic treatment.

But the grade of such lymphangitis is as varied as the particular germ or combination of germs present in the wound, and the susceptibility of the animal attacked, and there will be high, moderate or no fever, according to the severity of the case, and in some cases purely local trouble and in others general infection with purulent or septic localization in distant parts. There is always danger of extension to a neighboring joint with destructive results.

A curious outbreak is described by Wiart as attacking nearly every horse in the regiment that sustained a slight wound. A tubercle looking mass formed in the depth of the wound was slow to heal, and the lymphatics leading out from it became round, corded, turgid, and at long intervals developed along their course fluctuating centres which, whether opened spontaneously or by the lancet, showed the same indolent habit. A single attack would last from two to six months, and the actual cautery had to be used on the sores.

The *lesions* are those already described in the last article for simple lymphangitis. For infecting cases they are those of the particular disease which may be present.

Diagnosis. The general diagnosis of lymphangitis is the distinction from phlebitis. In phlebitis the vein is blocked and cannot be raised by pressure on the side leading toward the heart; in lymphangitis it can be so raised. The swelling and tenderness are both greater in lymphangitis. The inflamed vein is more rectilinear, the lymph vessel somewhat sinuous. If suppuration ensues it is more diffuse in lymphangitis; more restricted and mixed with the elements of blood in phlebitis.

For identification of the particular forms of infecting lymphangitis, the reference must be made to the individual infectious diseases.

Treatment. In general the treatment of lymphangitis is the antisepsis of wounds. Further than this the treatment of each case is that of the particular disease which it represents. For all cases alike it is important to apply vigorous treatment early, so as to cut it short before it can attain a dangerous extension.

For the simpler forms of lymphangitis the wound should first be thoroughly cleansed and disinfected. Washing with soap suds, or carbonate of soda will remove any greasy agent which would prevent a thorough antisepsis. Then it may be washed with the antiseptic lotion :—carbolic acid solution (1:20), or mercuric chloride solution (1:500), or zinc chloride (1:400) or potassium permanganate (1:160). If the infection has been introduced by a small or punctured wound, the sting or bite of an insect, or the prick of a sharp instrument it should be freely cauterized to its depth with lunar caustic incising it if need be to reach the whole of the poison, and the surface afterward dressed with antiseptics.

The diet should be light but nutritious and laxative, and the free action of the bowels and kidneys should be maintained by salines. When fever runs high give quinine, or salicylate of soda. When a large wound has to be dressed it may be requisite to use a non-poisonous agent like acetate of aluminium or boric acid to irrigate it thoroughly. In some such cases packing the irrigated wound with iodoform gauze has often an excellent effect.

When there is a firm inflamed cord, hot and painful, a fly blister along its course followed by mercurial ointment often gives excellent results. Or they may be repeatedly painted with tincture of iodine.

Foci of suppuration must be promptly opened and thoroughly and persistently disinfected.

With suppuration in multiple abscesses or large open sores liberal feeding must be enjoined and iron and other tonics should be resorted to.

The persistent swelling of the part must be met by active rubbing or kneading, by exercise and by uniform compression by a flannel or elastic bandage.

LYMPHANGIECTASIS. DILATED LYMPHATICS.

Result of lymphangitis, of heart disease, of pulmonary arterial thrombosis, of external jugular plugging. Causes, obstruction to lymph flow, compression, increased venous blood pressure, fibrinous lymph coagula, action of sensory nerves, of lymphadenitis, anæmia. Symptoms like dropsy if in plexus, in large lymphatics, moniliform swelling, sacculation, wounds discharge lymph, hyperplasia of connective tissue, fatty deposits, lipomata. Treatment, elastic bandage, cold, astringents, iodine, punctures, ligatures, cauterizations, tonics.

The most striking cases of dilatation of the lymphatics in the lower animals are met with in horses that have suffered repeatedly and severely from the lymphangitis of plethora. Then the lower part of the shank and the postern are enormously thickened to perhaps two or even three feet in circumference, and skin and connective tissue are the seat of a general dilatation of the lymphatic plexus and vessels with great thickening of their walls. Nocard and Barrier record cases of general dilatation of the lymphatics in dogs in connection with heart disease, also the case of a horse with old standing thrombosis of the pulmonary arteries, hypertrophy of the right heart, and dilatation of the thoracic duct to the size of the arm and of the lymphatics of the mesocolon to the diameter of half an inch to nearly an inch. Nocard records two cases in the horse, one of a reticular lymphangioma of the sheath, and the other of dilatation of the lymph vessels accompanying the saphena vein on the inside of the thigh. This formed small, soft, fluctuating, extremely irregular tumors, completely covering the vein for a space of about four inches.

In both cases the dilatations were surrounded by a thick layer of connective tissue filled with liquid. Virchow records a case of a new-born calf in which a thrombosis of the external jugular vein caused obstruction of the mouth of the thoracic duct, and a consequent extreme distension of all the splanchnic lymph vessels with a slightly sanguinolent fluid. The intestines especially were covered everywhere with broad, bead-like canals, arranged so closely together that the intervening tissue could be scarcely recognized.

The *causes* of lymphangiectasis appear to be generally some obstruction to the onward flow of the lymph. Any diseased condition, therefore, that causes compression of the larger lymph vessels may cause dilatation of the smaller ones leading into these. General distension may come from disease of the lungs or left heart and increased venous blood pressure, or from thrombus of the jugular, or a tumor obstructing the thoracic duct, while local engorgments may come from the pressure of tumors, or the occurrence of lymphangitis and formation of fibrinous coagula. In cases of partial obstruction of the lymph vessels the increased secretion of lymph may lead to distension and enlargement. It may be named in this connection that irritation of the sensory nerves in dogs has been shown to determine a larger production of lymph (Krause). Lymphadenitis and the obstruction of the passage of lymph through the glands is an obvious cause, and hence the disease is specially liable to appear in connection with diseases which show a predilection for the lymphatics (tuberculosis, glanders, strangles, carcinoma, etc.)

In his work on dilatation and occlusion of lymph channels Busey shows that in man the majority of cases are in hospital patients in whom blood and general health have been impoverished and reduced by unhygenic conditions. One case gave support to the theory of maternal impression, the pregnant mother having suffered from over-use of the right limb on a sewing machine, and the offspring having shown extensive lymphangiectasis in the right leg.

Symptoms consist in enlargement of the lymph vessels or plexus, and often of the glands. If of the lymph plexus it may appear like a dropsical effusion in the part, with or without saccular dilatations at intervals. If of the larger vessels, their tortuous anastomosing trunks following largely the lines of the veins are usually characteristic. If the distension is slight it is usually moniliform, as the valves are still intact, and the intervals between them stand out as bladder-like masses. If the structure is wounded or if it ulcerates there is the discharge of a straw-colored fluid, often rendered milky by the presence of fatty granules, and at times tinged with blood. There is always a tendency to the increase and condensation of the connective tissue surrounding the vessels, and fatty degeneration and the formation of lipomata are not uncommon.

Treatment. Compression, by flannel or elastic bandage, from the foot upward, is the simplest and most promising treatment when the limb is affected. The local application of cold, astringents or iodine may be added. Punctures, ligatures, and cauterization have not given encouraging results. Ligature of the nutrient artery of the part, has succeeded in one or two cases, but has failed in others. Tonics are to be tried more particularly in cases due to specific debilitating diseases. Sometimes a spontaneous recovery has been noticed when the surrounding connective tissue has increased and contracted in connection with inflammation.

LYMPHORRHŒA. LYMPHORRHAGIA. DISCHARGE OF LYMPH THROUGH WOUNDS OR SORES.

Result of rupture of lymphatics. Milky, fatty lymph. Treatment, ligature, excision, cauterization, of little avail. Compression. Tonics.

Obstruction of a lymph duct may lead to rupture and the discharge of its fluid on the surface or into an internal cavity. Dr. Cayley records a case of fatal peritonitis in man from rupture of the receptaculum chyli, and the formation of lymph fistulæ has been attributed to filaria sanguinis hominis. We are aware of no corresponding case in connection with the blood parasites of the horse or dog. In the larger domestic animals the great thickness and resistance of the skin offers a barrier to the rupture of subcutaneous lymph vessels, but this no longer applies in case of a suppurating or ulcerous wound. The escaping lymph has often a milky hue from the admixture of fat, just as its escape in the kidneys causes chyluria, and in the bowel fatty stools. The escape is often very profuse and persistent, and results in marked debility. Ligature and excision of the fistulous vessel, also caustics—actual and potential, have been tried with rather poor success. Fitzer succeeded in an obstinate case by the extensive application of nitrate of silver and others by simple compression. As the victims are usually debilitated a course of tonics is usually desirable.

LYMPHADENITIS. INFLAMMATION OF THE LYMPH GLANDS.

Result of lesions of tributary tissues. Arrest in glands of microbes and other irritants. Trauma of gland. Inflammation. Symptoms, swelling, stiffness, gland tender, hot, pitting envelope, corded lymph vessels, abscess, fever. Lesions. Treatment, antiseptics, astringents, emollients, vesicants, lancing, antiseptics, antiphlogistics, antithermics. Chronic adenitis. Symptoms, enlarged glands without engorgement, if simple affects a single gland, if infectious, a group. Lesions, gland swelling, induration, shrinking, follicular distension, pigmentation, growth of lymphocytes, caseation, calcification. Treatment, antiphlogistic, antiseptic, iodine, chloride of calcium, iodide of potassium.

Apart from traumatic lesions lymphadenitis virtually implies some lesion of the tissues from which the different vessels of the glands proceed. The glands however have been referred to as filtering agents on the course of the lymph vessels and in this partial view of their functions we find abundant reason why irritants carried in the lymph stream, should be arrested with pathogenic results in the glands. A particle of pigment gaining entrance to the lymph vessels tends to be arrested among the trabeculæ of the gland, and contributes to the pigmentation so common in old animals. Cells and granules from malignant tumors, and bacteria from an infection-atrium are arrested in the glands and make these the great centres of infection-lesions.

Traumatic inflammation comes from bruises, punctures or incisions directly implicating the glands. There result swelling, tenderness and the other general signs of inflammation, and in the case of an open wound possibly lymphorrhagia.

Acute inflammation more commonly supervenes on inflammation in the area drawn upon by the afferent vessels of the gland. In inflammations generally the adjacent lymphatic glands become congested. In lymphangitis it is so in a marked degree. In external parts we can follow this by careful observations during life, in internal organs we often find the glandular enlargement after death.

Symptoms consist in swelling and perhaps stiffness in the region of the gland. Manipulation shows tenderness and heat, the gland being felt abnormally large, round, or oval, tense, loose

from the skin but having a distinct envelope of soft pitting exudate which tends to increase in a downward direction. There may or may not be a corded feeling of the afferent lymphatic trunks. As the pasty swelling increases, it extends into surrounding parts, binds the gland to the skin and adjacent structures, and may even conceal the gland in the excess of its investing engorgement. This is especially frequent in strangles. As the process advances softening may take place in the centre and extend toward the circumference, and this may burst like an ordinary abscess. In some cases the softening is very limited and tardy, and the pus may be pent up and inspissated, or it may appear to be entirely reabsorbed while the gland is in process of induration. Fever which may run high during the process of suppuration, moderates when that has been accomplished.

In the case of glands too deeply situated to be clearly felt the occurrence of purulent fluctuation in their vicinity suggests abscess of the glands, an important induction as the maturation and healing are usually slow in the gland tissue.

Lesions. At the outset the glands are visibly enlarged, softened, and of a dark red hue, with spots of a brighter red. The changes, mainly in the medullary layer, consist in a great proliferation of spheroidal cells in the follicles and also of polyhedral cells in the lymph sinuses. The endothelial cells are swollen, the blood vessels gorged, and extravasations of blood into the follicles and sinuses are frequent. Abscess or fibroid hyperplasia with induration may follow. Much depends on the particular infection (tuberculosis, glanders, carcinoma, etc.) as the special product of each disease will be found in the affected gland.

Treatment is in the main as advised for lymphangitis and will vary with each specific causative disease. Locally antiseptics, astringents, deobstruents, emollients, and vesicants will be requisite in different cases. As soon as pus can be distinctly diagnosed it should as a rule be evacuated, and the cavity treated antiseptically. General treatment may at first be antiphlogistic and febrifuge, but must usually embrace tonics and stimulants in the end.

Chronic Adenitis may be a sequel of the acute, or it may arise independently. In the latter case it is usually the result of some other disease (tuberculosis, glanders, carcinoma, sarcoma

melanosis inveterate disease of the skin, chronic fistula, abscess, or mucous inflammation).

The *symptoms* are those of enlarged glands with no material surrounding engorgement. In the infections of tuberculosis and glanders it shows a tendency to affect the whole group, whereas in simple abscess or in suppuration of the nasal sinuses it may implicate one gland only, the remainder appearing normal.

Lesions. The gland often becomes indurated and even shrunken, the connective tissue elements undergoing a steady increase at the expense of the follicles and lymphoid cells. This is a common condition of tuberculous glands (perl-knoten, grapes) of cattle, but may result from the entrance of pigment or other cause of mild irritation. In other cases pigment entering from without or developed from blood in the congested gland, finds permanent lodgment in its tissue and may give it a gray mottled or quite black aspect. In still other cases, there is a great increase of the round lymphoid and larger polyhedral cells, many of which degenerate becoming strongly refracting, stain feebly, or not at all, and pass into a cheesy degeneration. This is a common condition in tuberculosis and glanders, and the caseous centres beginning as multiple miliary centres may coalesce to form masses of six or twelve inches in their greatest diameter as in bovine tuberculosis. In other cases the caseating mass becomes the seat of calcareous deposit and the necrotic and caseated gland becomes in part calcified. Other degenerative changes such as atrophy, amyloid, and hyaline are met with but have received little attention.

Treatment will be subordinated to the primary cause. If that is a simple local inflammation or irritation its removal will entail a speedy improvement in the gland, and, in the absence of too extensive structural change, a speedy recovery. The infectious cases on the other hand are likely to prove as inveterate as the disease on which they depend. In case the enlargement or congestion of the gland persists after the removal of its primary cause local deobstruants especially the preparations of iodine are usually effective. Tincture of iodine with soap, iodide of lead, and mercurial ointment have been severally used with advantage. Injection of a weak solution of iodine into the gland will at times succeed. The internal use of chloride of calcium or iodide of potassium will often hasten recovery.

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