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VETERINARY MEDICINE SERIES

No. 14

Edited by D. M. CAMPBELL

SPECIAL EQUINE THERAPY

By

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PREFACE

In this work special attention is given to diseases and conditions which are unnamed, atypical, or of infrequent occurrence. It is such conditions that are troublesome and difficult both of diagnosis and treatment.

The discussions herein are entirely from the viewpoint of the general practitioner; diagnosis and treatment are emphasized, while all quibbling over super-scientific details are ignored.

All conditions and diseases, as well as their treatments, are considered wholly from an American standpoint. Such diseases as occur only in foreign countries are not considered.

Diseases of very common occurrence, such as the practitioner handles almost daily, are also omitted.

The volume is intended to serve the busy practitioner chiefly as a reference work for diseases and conditions with which, because of their infrequent occurrence, he is not familiar, as well as an aid in the diagnosis and treatment of other more common, yet troublesome, diseases.

M. R. S.

1917.

*In every work regard the writer's end,
Since none can compass more than they intend;
And if the means be just, the conduct true,
Applause, in spite of trivial faults, is due.*

—Pope.

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PART I

INTRODUCTION

THE LIMITATIONS OF THE PRACTICE OF VETERINARY MEDICINE FROM A COMMERCIAL STANDPOINT

The practice of veterinary medicine is peculiar in the fact that in many instances the veterinarian's greatest usefulness lies in the early recognition of an unfavorable termination of a given case. But by an unfavorable, or an unsatisfactory termination we do not mean the death of the patient from the effects of the disease.

In the practice of veterinary medicine the veterinarian must nearly always measure his results on a basis of dollars and cents. While there are many dangerous and grave pathological conditions which the veterinarian could succeed in bringing to an ultimate recovery, he is frequently forced to abandon treatment and recommend destruction of the horse. The time required to bring the case to cure, the loss of the animal's services during this time, and the veterinarian's and druggist's bill, would in many cases exceed the value of the horse several times over.

The ability to foresee an unsatisfactory outcome such as this, as well as the exercise of good judgment in limiting the expense of useless treatment, is of especially great importance in a city practice. Many extraordinarily successful city practitioners owe their unusual success

to this ability as much as to their ability as practitioners of veterinary medicine and surgery.

In a country practice, while the same money basis of case handling must be always borne in mind, it is not of quite so much importance. The country practitioner's clients usually have ample stable room and plenty of feed for their horses. Neither is the serviceableness of work horses on the farm computed at day wages, as is the case with most city horses. In addition to this, there usually remains the alternative of consigning the animal to procreative function should the patient be of the female sex. The latter alternative is the gateway to the ultimate recovery of many grave cases in a country practice, cases that in a city practice would inevitably end in the destruction of the horse.

While it is of very great importance that the attending veterinarian call a halt in the handling of cases of this sort, it is of no less importance to be patiently persistent in some cases that, although presenting a most discouraging clinical picture, are known to terminate in a manner that warrants the expenditure of a considerable amount of time and money. This is especially true in the handling of certain cases in young horses, horses that will yet "grow into money."

An item that is worth considering along this same line of thought is that pertaining to the fee charged by the veterinarian when a difficult, protracted case terminates in death. While it may seem that the veterinarian's fee is not based upon the life or the death of the animal, but wholly upon the service rendered in connection with the case, it may be questioned whether this is entirely correct.

Young graduates who are just beginning to establish themselves in practice in a community must give considerable thought to such matters. It does not help to in-

crease the doctor's popularity if he adheres too strictly to an iron-bound schedule of fees in cases like those under discussion. The failure of beginners to give thought to these matters and to use good judgment is one of the commonest handicaps in the race with an older and more experienced competitor.

The diseases and other pathological conditions that will be discussed in the following pages of this volume are nearly without exception cases in which the attending veterinarian must make use of a goodly amount of judgment of this character.

While it is usually not so very difficult to foresee the termination of a given case, it is nearly always quite a troublesome matter to so adjust the business relation between veterinarian and client that no friction will result. An agreeable relationship of a commercial character can usually be maintained by the application of a degree of flexibility in the doctor's schedule of fees—an application that ultimately brings good returns.

SYSTEM IN MEDICATION

It may be appropriate to devote a few pages to the discussion of what might be termed medical exhibition. I mean by this the individual dosage in a particular case of any disease. I believe that most of our veterinary doses are excessive. I also believe that most of our doses are unnecessary. With exceptional opportunities for becoming familiar with a much greater than usual number of medicinal agents, I find that I am able to meet most ordinary pathological conditions almost without the use of drugs.

When I do find it necessary to employ medicinal matter I find that I get the best results from the use of doses that are in nearly every case much smaller than the accepted standards.

This is not because I have greater knowledge of the action of drugs. I believe it is wholly from the fact that I make better use of the natural assistance inherent in our patients. More clearly, I give my patients the opportunity to benefit by this inherent assistance; I give them a chance. I have no ear for homeopathy! But neither have I for the automatic gradation of doses, based on human medicine, still employed too largely by the veterinary profession.

I have taken extraordinary steps along this line. I have had the good fortune to have been in a position in which I could put theory into practice, and I am sincerely satisfied that the average veterinarian does his medical cases but little real good. By far the majority of recoveries which he credits to his treatment are not the result of his treatment, but rather the result of his patient having had sufficient stamina to overcome not only the disease, but the injudicious dosage of drugs as well. Our patients differ markedly from the human pa-

tient in the fact that, *with very few exceptions*, the diseases to which our patients are subject have a tendency to terminate in recovery; not because the diseases are more mild than those affecting human beings, but because our patients are less vulnerable. Our patients, with an occasional canine exception, lead simple existences. They do not consume either liquids or solids deleterious to their well-being; they do not become saturated with nicotine or other poisons. Their nervous systems are not developed at the expense of their physical power. And, of greatest importance, is the fact that, while possessed of a brain, they have no highly developed mind.

The resisting power against disease with which our patients are endowed frequently borders on the miraculous.

The practitioner who, by cultivating his powers of observation of these characteristics, uses in his treatment of disease doses just large enough to assist this power will get *results* which will very frequently border on the miraculous. In this lies the answer to the question why bacterins accomplish what they do. In this lies the answer why iodid of potassium can work such wonders; it is one of the few drugs that establishes its own limit of dosage by the production of iodism.

We do not pay enough attention to the individual in veterinary practice. To most of us a horse is a horse. With hundreds of pounds of difference in size, we merely administer "a dose for a horse." We accept as doses for individual use what were only intended for general standards.

But it is not only in the dosage that we are in error. It is just the same in the selection of a drug in a given case. Most of our treatments smack of heroics. Instead of gracefully bending a case to the accompaniment of

natural efforts on the patients' part, we endeavor to "make or break" all too commonly. We select agents, the action of which is, frequently, too direct. And, again, all too frequently, our remedy, instead of attacking the disease, does its heaviest work upon tissues not in the least involved, or else upon tissues and organs already bearing the brunt of the attack. Most of us can remember the old forms of treatment for acute indigestion. With a stomach already so full of ingesta and gas that it was nigh unto bursting, and frequently it did burst, we persisted in pouring in dose after dose. Many times several gallons of oil and other liquids have been added. Today we do just the opposite; we use a stomach tube and remove the fermenting mass, and deaths from rupture of the stomach following an attack of acute indigestion are today exceedingly rare. In dozens of other diseases we perform just as unreasonable (if not quite such evident) capers in the line of treatment. Instead of assisting the natural powers of the body (and in our patients these most assuredly deserve recognition), we make it doubly hard for them.

The reader will note the simplicity of treatments recommended in this volume. Adherence to these principles, and their adaptation to other conditions than those here discussed will prove a revelation to most practitioners who are unfamiliar with them. Every method of treatment named in the following pages has been thoroughly tested in actual practice and found reliable.

No space has been devoted to any other purpose than the expounding of result-getting matter. It is presumed that the reader is fully conversant with the business end of the profession and that no admonition is required upon points relating entirely to the whims of a clientele.

PART II

MISCELLANEOUS AND MORE OR LESS BAFFLING AFFECTIONS

RHINORRHAGIA

A profuse flow of blood from the nose, and having its source in the vessels of the nose, is termed rhinorrhagia.

The treatment of this condition is considered irrespective of its cause, for the reason that the first concern in this condition is always the checking of the hemorrhage. When a case of rhinorrhagia is presented for our attention, we are not particularly concerned about the direct cause of the bleeding; we attend to this, if necessary, after the hemorrhage is under control.

In veterinary practice alarming hemorrhages from the nose are rare; most of these are limited to a harmless epistaxis which usually ceases spontaneously. When, however, rhinorrhagia does occur in horses the condition is especially serious for the reason that these animals violently resist the veterinarian's efforts, and also because of the anatomical arrangement of the turbinated bones.

Severe hemorrhage in the nose must be differentiated from pulmonary hemorrhage. Blood coming from the lungs appears foamy and produces fits of coughing during its emission. In severe nasal hemorrhage the pharynx may fill with blood and also cause some cough. If this creates doubt as to the source of the hemorrhage, the trachea is to be auscultated. If the hemorrhage is

from the lungs, rales and gurgling sounds are heard in the trachea. If it is from vessels in the nose these sounds are not present, unless the hemorrhage has occurred in company with an acute pulmonary disease. The diagnosis must then hinge on the foamy appearance of the blood.

Treatment of Rhinorrhagia. When nasal hemorrhage is severe enough to require treatment in the horse, prompt and active remedial efforts need to be resorted to. The patient should, if possible, be backed into a single stall and cross-tied as for dental work. By means of a long dressing forceps the nostril, or if blood comes from both sides, both nostrils, are quite snugly packed with gauze. To facilitate the removal of the gauze after the hemorrhage is checked, it should be in one piece, and should have been previously saturated with sweet oil. The sweet oil prevents the adhesion of the gauze to the bleeding spots, and also delays putrefaction in the event that the gauze must remain in place for a considerable length of time. As a rule it is safe to remove the gauze twenty-four hours after the hemorrhage has been stopped.

Whenever it is necessary to pack both nostrils, tracheotomy must first be performed. Tracheotomy alone will at times check a nasal hemorrhage, by making snorting and blowing of the nose impossible. This gives an opportunity for clots to form and to remain in place.

If the hemorrhage is not thought of such gravity as to require packing of the nasal cavities, an attempt may be made to check it by irrigating the nasal canal with a solution of supra-renal extract, or by the spraying of solution of supra-renal extract into the nasal cavity with an atomizer having a long nozzle.

Usually it saves time and trouble to proceed at once with the gauze packing. Irrigating, spraying or swabbing usually brings on fits of sneezing or snorting and

coughing, which only have the effect of aggravating the hemorrhage and complicating matters. Most horses will violently resent the irrigating and spraying, while the gauze packing is calmly submitted to in most cases.

CONTAGIOUS PUSTULAR STOMATITIS

Contagious pustular stomatitis of horses is an acute febrile condition whose local manifestations occur chiefly on the oral mucous membranes. While this disease has all the characteristics of an infectious and contagious disease, there is some question about its being a strictly infectious ailment. The infecting agent is not universally recognized, although some investigators have isolated certain strains of micro-organisms, which may, however, be accounted for by a secondary or mixed infection. Other authors consider the disease a manifestation of horse pox, while others, again, attribute the infection to a common cause such as may occur from contaminated feed. There is some ground for the latter assumption because the disease has a tendency to remain enzootic and to affect nearly all the members of a stable in a routine manner.

The specificity of the condition is, on the other hand, fairly well substantiated by the fact that human beings frequently become infected; characteristic pustular dermatitis develops on the hands. Veterinary surgeons are occasionally infected thus while examining the mouth, and the disease has been known to take a very serious course in several cases of veterinarians so affected. (Dr. Jas. Robertson, Professor of Veterinary Dentistry at Chicago Veterinary College, narrowly escaped death from the effects of an infection of this type in 1900.) The period of inoculation in the horse is from six days to two weeks.

Symptoms. The disease begins with a moderate fever, accompanied by mild depression. There is anorexia, but frequently polydipsia. When the opportunity is given to drink, the animal likes to prolong the act, by playing with the lips, deeply immersing the muzzle, and so forth. Some salivation may be noted. Examination of the mucous membrane of the mouth shows in the early stages reddened areas. The mouth is hot and sensitive. The reddened areas later "run together," and are then the seat of small, hard elevations. They appear in various sizes, from that of a mustard seed to the size of buck-shot. These hardened elevations or nodes appear in "crops" day after day for several days. The earlier ones go over into vesicles, break and discharge either a clear serum or pus. The later ones do the same, successively, until the disease has run its course. The broken down vesicles and pustules rapidly fill with granulations and heal over smoothly in a few days. An occasional extraordinarily large vesicle or pustule, or when several of them coalesce, may leave a deep ulcer which heals more slowly and leaves a "pearly" scar. In some outbreaks of contagious pustular stomatitis the pustules also form on the skin of the lips, and some cases have come to our attention in which the sides of the neck and pectoral region were involved.

The first cases in an outbreak are as a rule the most virulent, while towards the end the cases are usually very mild. The duration of the disease, from onset of the fever to complete healing of all pustules and vesicles, is from one to two weeks, depending upon the severity of the attack and upon the care which the animal receives. The fever subsides after the third day. Some deaths have been reported. Fatal cases are extremely rare, and are no doubt due to septic infection of a secondary character when they do occur.

The diagnosis hinges on the course pursued by the disease and its contagiousness. First, fever and general depression; second, reddened areas in the mouth; third, vesicles, pustules; fourth, rupture of the vesicles. The appearance of other cases in the same stable always follows. The vesicles and pustules are peculiar in their small size and the fact that they appear first on the mucous membrane. Later, in some cases, they may appear on the skin.

Treatment. Antiseptic, astringent mouth washes should be used. A saturated solution of the sulpho-carbolates compound is fine, for the reason that it can be used liberally and does no harm when some of it happens to be swallowed. Ulcers which occasionally form are to be painted several times daily with equal parts tincture of iodine and tincture of benzoin. On general principles a dose of mixed bacterins is indicated. The animal should have sloppy feed and constant access to fresh water. The animals are not serviceable for at least one week. An outbreak of contagious pustular stomatitis can be considerably shortened in stables or a community if the early cases are promptly isolated and strictly quarantined.

TYMPANY OF THE GUTTURAL POUCHES

To one who has never seen this abnormality it gives rise to thoughts of apprehension. Cases of distension of the guttural pouches with air appear to be serious to the veterinarian who has had no experience with them. As a matter of fact, it is usually a very benign and decidedly transient affection.

We meet with this condition most frequently, almost always, in mares that have recently given birth to a foal and that have been turned out to pasture after having been confined to the stable for a long period of time, and in debilitated colts of from one to three years of age when turned out to pasture in the early spring when they must graze nearly all the time to get sufficient nourishment.

A few hours seems to be all the time required for the condition to become fully established, although as a general thing the condition is first noticed towards evening of the first day that the animal has been turned out.

It is then seen that the region over the parotid glands, the laryngeal region, and sometimes the space between the rami of the inferior maxilla are immensely swollen. If a halter has been left on the mare while she has been at pasture parts of it are deeply imbedded and buried in the swelling. When the swelling is palpated it is found to be drum-like and very tense. When force is applied to one side by pressing with the flat hand over the parotid region the opposite side bulges out in proportion. If a finger is pressed firmly into the body of the swelling and the pressure is suddenly released by quickly withdrawing the finger great resilience is noted, and no pit remains.

Near the borders of the swelling there is edema in some cases, no doubt due to interference with the circulation from direct pressure.

In cases in which the distension is exceedingly great, there may be a slight degree of dyspnea. In cases showing only moderate distension there is no dyspnea. Even in cases of the most marked type the animal does not, as a rule, exhibit any signs of distress or indisposition. The symptoms are apparently limited to the tumefied condition in the region. The diagnosis must be made from:

First, the history of the case; mare with foal, turned on grass after a long period of idleness in the stable; or debilitated colt as before mentioned.

Second, the nature of the swelling; pneumatic under palpation, and painless.

Third, the absence of other symptoms of disease. The temperature may be raised slightly, but not much. The pulse, if taken, must be taken at some extremity, such as from the coccygeal artery. It will be slightly accelerated.

Treatment. In very marked cases in which there is much dyspnea, immediate relief may be attempted by the exertion of a firm, steady pressure with the flat hands over both sides of the swelling.

Should this fail to give relief it need be no cause for anxiety. The dyspnea will subside markedly after the lapse of a few hours if the patient is at once placed in a single stall and tied short at the halter, so that the head may not be lowered. But by no means should the head be raised above a normal standing-level.

If it is requested that something be done further than this, an oily liniment may be prescribed with which the tumefied area may be gently massaged.

In all cases, almost without exception, coming to the attention of the writer, the swelling had completely disappeared in less than twelve hours. No recurrences have come in the cases noted.

If the condition escapes detection on the first day, or if the animal is allowed to remain in the pasture after

the swelling has been noticed, a marked edema affecting the entire head may result. When this has occurred the treatment consists of confinement in a single stall with the head raised. The easiest manner in which to keep the head raised in some of these cases is by means of a bridle and back-pad, using an overhead check to check the animal high. This is usually sufficient and should be maintained several hours. Downward massage, lightly done, is helpful. Cold packs may also be of benefit. Two or three doses of digitalis improve the circulation and hasten the disappearance of the swelling due to interference with the circulation.

MYXOID DEGENERATION OF BONES

Not many cases of a myxoid osseous degeneration are on record in veterinary science. Whether this is due to the fact that it is a rare affection, or whether veterinarians have failed to report the cases out of their practices, I do not know. I have been able to find but very little literature on the subject. One case that came up in my practice a short time ago I reported in the *American Journal of Veterinary Medicine*. As the description of the case is quite fully given in this report I reproduce it here, as follows:

“Not many cases of myxoid degeneration of the bones of the skull in horses are on record. Apparently it is a very rare pathological condition. Cases which have been reported were mostly in young horses and sucking colts; now and then the condition has been known to occur in mature animals.

“The case which I am about to record is that of a grey gelding about seven or eight years old. He is a fair specimen of the express type and in good condition, weighing about 1,250 pounds. He was raised by the man who owns him now and was supposed to be a ‘whistler’ or ‘roarer’ since colthood.

“This roaring was not typical and, until the evidence of the myxoid degeneration developed, it could not be satisfactorily diagnosed. Now that the true condition has shown itself the roaring, according to other records of such cases, is explained.

“On looking up these cases I found that in nearly every instance the horse was either affected with roaring, snoring or had some other form of respiratory impediment which was puzzling and which could not be satisfactorily diagnosed until the appearance of signs of the myxoid degeneration.

"The case in this grey gelding was brought to us for treatment because of a 'bunch' on his forehead.

"According to the owner's statement it had been forming for several months, being about the size of a hazel nut when first noticed. It had now attained the size of a large hen egg and had about the form of an egg, the apex pointing straight outward, like a horn.

"No abrasion could be seen; the skin over it was perfect.

"The location of the 'bunch' was exactly over the frontal sinus, a little to the left of the median line. To the examining finger it felt very tense, almost hard, and pressure with the finger over it evidently caused considerable pain, evidenced by jerking the head and attempts at rearing up. A cyst was diagnosed; no details as to the nature of the cyst were gone into.

"With a twitch on the nose an exploring trocar was inserted. Instead of pus (which was really what we expected to find), there came a flow of rusty-colored liquid which spurted outward to a distance of six or eight feet.

"As the contents flowed out the apex of the 'bunch' caved in and it could now be plainly felt with the examining fingers that the frontal bone at the base of the bunch had disappeared or become absorbed. The skin could be pushed into the sinus like an inverted glove finger.

"The diameter of the hole in the frontal bone was irregular, varying from one inch at the narrow point to more than two inches at the widest. The edges were very smooth and seemed to have a thickened margin.

"When most of the fluid had been drawn off the area was painted with iodine and the horse sent home, with instructions to bring him in again in three or four days. This gave us time to look up the handling of the condition.

“When the horse was brought in again the ‘bunch’ was just as big as it had been before we tapped it and the owner stated that it had filled up a few hours after he took him home.

“As we had ‘brushed up’ on the condition and its treatment by this time we recommended incision of the bunch, thorough curettement of the sinus and of the edges of the frontal bone. This was refused.

“We then prescribed painting the enlargement and a considerable area around it with iodin, and iodid of potassium internally.

“After two weeks of this treatment the enlargement has gone down considerably, but an interesting change has occurred. There is now a discharge from both nostrils of a fluid the same color and consistency as that removed with the trocar. Evidently the process has progressed and the sinus is now draining itself.

“The gelding shows no ill effects in any other manner and works every day. The owner has now consented to submit him for operation at a later date. If he keeps his promise we will report results.”

The owner did not keep his promise, and the case was lost sight of.

A few practitioners have reported similar cases in younger horses. Curative treatment recommended consists of complete removal of all the affected bone by means of trephining, sawing and curetting. This is followed by irrigation with suitable antiseptics for about six weeks.

The cause of this disease is unknown. In the early stages, before the swelling is fluctuating in character, the condition must be differentiated from osteoma, and in some cases from catarrh of the frontal sinus which has produced a prominent bulging of the frontal and facial bones. The insertion of an exploratory trocar re-

sulting in the evacuation of the characteristic rusty-colored fluid, establishes the diagnosis of myxoid degeneration.

There is no doubt that, under proper treatment, the bone which has disappeared would be regenerated, although the effect of this regeneration of bone might fall short of expectations.

ACUTE INFECTIOUS PHARYNGITIS

In taking up the discussion of this subject I would begin with the statement that I am aware that some authors of works on equine medicine believe this to be a localized manifestation of influenza. A form of acute pharyngitis does occur during some outbreaks of influenza. I am positive, however, that the authors in question are in error when they classify all forms of acute pharyngitis of an infectious and transmissible character under the head of influenza. We have in the United States a form of acute infectious, contagious pharyngitis in horses that occurs as a disease *per se*, unaccompanied by lesions which could bring it recognition as a manifestation of influenza.

The disease under discussion here occurs in an enzootic form as a rule, although during some seasons it has assumed almost epizootic force. It affects both young and old horses, without regard to condition or individual environment, and runs a typical and almost exact course in all cases. This is its first feature of differentiation from acute pharyngitis resulting from a localized influenza infection. Hardly two cases of the latter form appear similarly, while the disease which we have under discussion runs in every instance a typical, characteristic course.

Symptoms. The disease appears almost as a subacute affection. For one or two days before it becomes fully established the animal has a cough. This cough is not heard, however, except during or after drinking water and eating grain. It may be so mild as to escape the attention of the owner or attendant. In from one to three days after the cough has set in the horse has some difficulty with his deglutitory acts. While drinking, some of the water returns through the nose; the animal stops drinking after every few swallows and experiences a paroxysm of coughing and spasmodic gulping. In the beginning of the disease the amount of water that returns through the nose is slight; later it appears as though literally pumped through the nasal chambers with each act of deglutition.

In another day, or at most two, after the inability to swallow water normally, the horse finds it impossible to swallow grain. While the appetite does not seem to be entirely lacking, in fact remaining good throughout the attack, in many cases the grain is refused. At times the animal attempts to eat oats, but after the first mouthful or two the attempt is again given up. Nearly all of these subjects eat hay throughout the course of the disease. All boluses of hay are not swallowed, however. The manger is soon strewn with boluses that have been masticated but again ejected on account of inability to swallow.

Here we have another good mark for differentiating this disease from such cases of pharyngitis as occur from an influenza infection. In pharyngitis resulting from influenza the horse shows genuine anorexia; he does not eat because he has no appetite, no desire to eat. In the disease we are discussing the horse *would* eat if he *could*; his appetite is not gone. He makes repeated daily attempts, but does not succeed in swallowing well on ac-

count of the pain and interference with the proper action of the muscles concerned in the act of deglutition.

When the dysphagia has become fully established, at the end of from one to three days, there appears a fullness in the parotid, pharyngeal and laryngeal region. The swelling is smooth and lies rather below and under the parotid glands, pushing these upward and outward. In some cases abscesses form in this swelling.

The temperature may not be raised more than two or three degrees; occasionally a case will show temperature as high as 106 degrees F., but this is not common. Cases in which the temperature is much elevated are usually those that later have abscess formation in the swelling described. Despite quite high temperature and marked local swelling, soreness on palpation in the swollen region, and marked indications of a severe disturbance in the affected parts, the horse does not appear very sick. Depression is absent and animals remain bright. Here we have another point against attributing these cases to localization of influenza infection. In the latter there is marked depression, drowsiness, and other evidence of genuine sickness.

Acute infectious pharyngitis requires from one to three weeks to run its course. If no abscess formation occurs two weeks suffice to bring even fully developed cases to a satisfactory termination. The mortality is very low, practically nil, under treatment.

The danger of transmission is nominal. In some instances all the horses in a stable become affected; in others only a part of them, and in some instances a single animal. This is not altogether explained by the fact that the infecting organism is lacking in virulence, but rather by the fact that one attack seems to confer a permanent immunity. Animals in the stable already immune from the effects of earlier attacks, will not develop

the disease. In exceptionally severe cases, showing great tumefaction in the region, considerable dyspnea may occur. Hardly ever does it become so serious as to require the performance of tracheotomy.

There is no discharge from the nose in most cases until the dysphagia is fully established. It is then of a muco-purulent character, and not very copious. This would indicate that the discharge is chiefly the result of an irritation of the Schneiderian membranes as a result of contact with water and food particles that flow through the nasal chambers. Cases that develop an abscess in the pharynx have a liberal purulent nasal discharge. On account of the inability to take sufficient nourishment the affected animals lose considerable weight. The patients maintain a standing position throughout the course of this disease.

I wish to call especial attention to what I have said about the order in which the ingested matter is rejected by the pharynx in this disease, namely,

1. Water.
2. Grain.
3. Hay.

This is always the order of sequence in this disease; and did we have no other single mark by which to differentiate this disease from other forms of pharyngitis, we would be able to recognize it thereby. To me this is also positive proof, in the absence of satisfactory bacteriological evidence, that this disease is not in any way allied to influenza. It is evidence that the dysphagia is not so much the result of an active inflammatory lesion in the pharynx at first, but more probably the result of a secondary trophic disturbance. Were the dysphagia the result of a highly inflamed, painful state of the pharyngeal mucosa and surrounding tissues, we have every reason to believe that the order of rejection would be re-

versed; we would expect to have the patient retain the power to swallow water rather than hay.

Treatment. Those animals that have the disease in a mild form are treated with no especial regard for the specificity of the affection. A good "stiff" liniment is rubbed into the throat several times a day. Apply the liniment over the parotid region, over the larynx, and between the rami of the lower jaw. A pail full of clean drinking water should be kept constantly in the manger where the horse can reach it without effort. The water may be impregnated with an astringent such as dilute sulphuric acid, chlorate of potassium, or ordinary magnesium sulphate. Fluid extracts of nux vomica and gentian may be given in small amounts with a dose syringe several times a day, for their tonic effect.

The feed should consist of semi-liquid slops made of ground oats and bran. Affected animals should not be put to work until they have been able to eat and swallow normally for several days. In those animals with a more severe form of the disease the treatment must be somewhat more arduous. In such a case, a good mustard plaster should be applied to the pharyngeal region; if the first one does not "take hold" sufficiently, put on another. When the mustard has ceased to act and has thoroughly dried, smear the entire region freely with vaseline and bandage over this, using plenty of cotton under the bandage.

Every case of this severe type of the disease should be given a full dose of mixed bacterins to forestall abscess formation.

For internal treatment, the best, both for its local effect on the mucous membrane of the pharynx as well as for remote effect, that I have found, is beechwood creosote in glycerin. The dose of creosote here should be about five drops, using about two drams of pure glyce-

erin in which to suspend it. This is then given every three or four hours with a dose syringe. Keep this creosote treatment up until local improvement is seen, after which it should be continued another day or two. Follow with general tonics.

The feeding and watering instructions that were given for mild cases should be followed in all forms. Abscesses are to be opened when the conditions are favorable, and treated as they would be treated in any other region.

If a case should prove indifferent or unusually stubborn under this line of treatment, stop everything and give three or four large doses of potassium iodid, say two drams, every three hours until three doses have been given. Then begin again with the creosote treatment.

When tracheotomy is necessary on account of serious dyspnea, perform tracheotomy as ordinarily done.

Cases of acute infectious pharyngitis occurring in stables, the horses of which are not known to be immune, should be isolated and handled in every way as though affected with a contagious disease.

Fullness in the pharyngeal region may remain in those animals in which the disease has been complicated by the formation of abscesses. This may be dispersed by the use of iodids internally and massage with oily preparations of iodin locally.

In some cases a tenacious cough remains for weeks after all other signs of the disease have disappeared. This cough does not always yield to the same treatment. Some cases respond rapidly to ordinary cough mixtures; some are cured by a few medicinal doses of potassium dichromate, while an occasional case may prove stubborn under any and all forms of handling. In a few instances there has remained a permanent dyspnea of a mild character.

During the course of an attack of this disease, I have

seen no other complication than abscess formation. Deglutition pneumonia, which might be an expected complication, I have never seen occur in company with acute infectious pharyngitis. Neither has the patient, in any case, required feeding by artificial means.

In old horses acute infectious pharyngitis may have to be differentiated from senile paralysis of the pharynx. A confusion is only possible, and even then not very probable, in the early stages. In cases of senile paralysis the history is always to the effect that the trouble has been coming on very slowly. For months the horse has shown signs of a deglutition impediment of an obscure character.

Neither is there any marked degree of pyrexia. Instead of fullness and tenderness in the pharyngeal region, there is rather a lean throat, and soreness is absent.

SENILE PHARYNGEAL PARALYSIS

A paralysis affecting the apparatus concerned in deglutition is occasionally seen in horses as a disease of old age. This paralysis is not one that makes its appearance very precipitately, but its appearance usually means the end of usefulness in the animal. Recovery is very rare.

The first evidence of the approach of a senile paralysis of the pharynx is seen in "quidding"; the attendant now and then discovers a few "quids" or boluses of masticated hay in the animal's manger. The usual effect of this discovery is a visit to the veterinarian for the purpose of having the animal's teeth examined. The teeth are given whatever treatment the attending veterinarian may choose, but the animal persists in "quidding." The condition slowly grows worse. Soon the animal gets poor in flesh, salivation is marked, and a slight nasal discharge is seen. Still later the horse finds it a difficult matter to swallow enough feed to sustain life; even water is imbibed with difficulty. In some cases esophageal spasm, resulting in choke, is repeatedly present. All cases have a cough.

From the outset of the first symptoms to the end in death from inanition, from four or five months to two years may elapse. A few cases improve spontaneously, and occasionally recovery follows proper treatment.

Strychnin in small doses repeated several times daily for a few weeks is useful. Fowler's solution of arsenic repeated in small doses is followed by improvement in some cases.

Cases which resist either of these agents may improve rapidly under a course of potassium iodid. Local treatment, such as light blisters, iodine paintings and massage, are of use in some instances. Other cases resist all efforts toward improvement or cure.

The diagnosis is made in old horses by observing a gradually increasing dysphagia, unaccompanied by fever or local evidences of pharyngeal disease, such as swelling or soreness. The condition may possibly be confused with the dysphagia occurring in some cases of spinal or cerebro-spinal meningitis, as well as with the dysphagia of meningism. The comparatively rapid development of other signs common to these diseases promptly precludes the existence of senile paralysis of the pharynx.

Cases of pharyngeal paralysis resulting from injuries are ruled out by the rapidity with which they become established, as well as by usual evidences of trauma. Cases of dysphagia, due to stenosis in old horses, are usually accompanied by snoring or whistling.

Senile pharyngeal paralysis is due to sclerotic processes in the brain and spinal cord in all instances.

EDEMA OF THE GLOTTIS

When the submucous tissues of the glottis become infiltrated with inflammatory exudate or with congestive fluids of other character, the condition is one of grave import. While edema of the glottis is, in many cases, merely a complication of laryngeal or pharyngeal inflammations, it does occur at times as a distinct and separate clinical phenomenon. No doubt, occasional instances of sudden death, attributed to other causes in the absence of positive evidence, are due to this condition.

Symptoms. The clinical picture in edema of the glottis is that of asphyxiation. The horse finds it impossible to inhale; exhalation is not interfered with. There is great excitement, the animal frequently "paws the air," throws itself against the sides of its stall; the urine dribbles away, perspiration sets in. In other cases the horse stands quietly enough, breathing wheezily and with great effort. The animal usually resents palpation in the laryngeal region, and pressure on the larynx increases the dyspnea. Edema of the glottis always develops very suddenly and progresses rapidly to a fatal end by asphyxiation in many cases. From the beginning of the first symptoms to the termination of the attack in death may require not more than half an hour.

Treatment. If asphyxiation is near, evidenced by staggering, opening of the mouth in the effort to inhale, cyanotic membranes, tracheotomy alone can save the life of the horse. No time should then be spent in preparing the operative area and the operation should not be delayed because no trachea tube is at hand. In the latter emergency the tracheal incision can be held open by means of artery forceps or sutures until a tube can be procured or a makeshift improvised. The edema may subside within a few hours, or it may remain for several

days. The tube can be removed as soon as normal inhalation is possible.

Cases which are not so near asphyxiation can usually be handled satisfactorily with Dr. Quitman's guaiacol treatment. One dram of the drug is shaken up with an ounce or two of water and given with a dose syringe. Repeat in an hour, if it is necessary. As the guaiacol does not mix very well with the water, it should be added to the water at the time it is to be given, drawn into the syringe and violently agitated by shaking the syringe. It is then rapidly squirted into the mouth and pharynx. The effect of this drug in this condition frequently borders on the marvelous. Local or external applications in the form of counter-irritants of a mild type may be used in conjunction with the guaiacol treatment. The application of ice may also be tried.

If the relief afforded by this form of treatment does not become promptly evident, perform tracheotomy without further delay.

CHOKES

Choke is the name applied in veterinary practice to that condition resulting from the permanent lodgement of food or other matter in the esophagus. In horses choke is always caused by the arrest of either hay or grain in some portion of the esophagus. Choke from solid bodies, vegetables, for instance, can almost be said never to occur in horses. The pharynx of horses seems to positively refuse passage to any matter that has not gone through the process of mastication quite thoroughly.

Choke in horses is classified into cervical and thoracic forms. When the offending matter finds lodgement in that portion of the esophagus anterior to the thorax, it is termed cervical choke. When the lodgement is in the thoracic portion of the esophagus, it is termed thoracic choke. The classification is only of value for purposes of treatment.

Blocking of the esophagus with food in the horse is possible under two distinct pathological types. The most common type, which includes probably 98 per cent of all cases, is that in which the condition is a purely functional disorder. Because of a peculiar disarrangement in the nerve impulse controlling the esophageal musculature, a spasmodic contraction arrests the progress of a bolus of food on its way to the stomach. A less frequent, in fact, a very rare form, is one in which a structural defect exists in the esophagus in the form of a dilatation, or jabot. Dilatations of the esophagus vary from a very slight enlargement in the diameter from atonic muscular areas to veritable sacculations of considerable portions of the tube.

Choke in horses is always a grave condition, requiring the exercise of much good judgment on the part of the attending veterinarian. In most cases the condition, from

a pathological standpoint, is nothing more than a functional disturbance. This does not reduce the gravity of the condition, however, from the practitioner's standpoint. The seriousness of the case lies mostly in the damage inflicted on the patient by the handling that the case is usually subjected to before the veterinarian's arrival.

The prognosis in cases of spasmodic choke is always favorable when the patient has not been the subject of malpractice, such as the entrance of whips, traces, broomsticks and similar objects into the esophagus, at the hands of laymen or empirics. The prognosis becomes unfavorable in direct proportion to the amount of injudicious interference that has been applied. This is the point that the veterinarian must, by all means, keep in mind in beginning the handling of a case of choke in the horse. Place the responsibility where it belongs. Understanding the condition fully, as you do, have no regard for the feelings of your client if he has been guilty of any form of malpractice in connection with the case in hand. It is a proved fact, substantiated by abundant clinical evidence, that injudicious meddling with a case of choke nearly always results disastrously. The attending veterinarian should have no scruples against so informing the client that has so committed himself. Undertake the handling of such cases advisedly; promise nothing, and expect nothing.

Cases of choke in the horse that come into the veterinarian's hands uncontaminated by ignorant practice can be given a favorable prognosis in almost all instances. The exceptions are those few cases in which the condition is due to a structural change, a dilatation or jabot.

To unconcernedly take in charge a case of choke, with no regard for the handling the case has received before

the doctor was called in, shows lack of experience in these conditions.

In my practice I do not hesitate to inform the client who has meddled with a case of choke that he must abide by the consequences. I place all the responsibility connected with the case on his shoulders. I take the case for what it is worth; I do what ought to be done. But I offer no encouragement.

On the other hand, I view with extreme optimism all cases that come to me "first-hand." I do not hesitate to make light of the case. I try in every manner to place my client's mind at ease and to relieve him of all worry about the patient's state. My chief object in doing this lies in the way of obtaining a free hand in the handling of the case. As a graduate veterinarian I realize that non-interference is worth more in cases of choke than injudicious treatment. If I succeed in placing my client in a frame of mind compatible with this knowledge, I have made a big stride towards a satisfactory termination of the case.

Symptoms. (Spasmodic Choke.) The symptoms of choke in the spasmodic form are more acute than in that form caused by a dilatation of the esophagus. The animal stops eating suddenly, backs away from its feed, and shows signs of restlessness. When the animal attempts to swallow, a gurgling sound is heard over the esophagus, and the cervical region goes through an attack of muscular cramps or convulsions. The muscles in the region seem to contract *en masse*, while the esophageal canal stands out in bold relief. If the obstruction is in the cervical portion of the tube, it is usually possible to detect it during one of the cramps. There is then an added prominence or a greater thickness that can be seen over the obstruction. The majority of spasmodic chokes are in the cervical region.

Cases of choke resulting because of a dilatation in some portion of the esophageal tube appear less precipitately. In some instances it requires the lapse of several days before a complete "choke" is established, the animal having continued to add to the obstruction by eating at times. (In spasmodic choke the patient refuses all food, as a rule.) When enough food has collected in the dilated portion of the esophagus to block the passage the animal begins to show symptoms. These symptoms, however, are not violent. There is now no inclination to eat. There may be a slight degree of retching, accompanied by mild muscular contractions in the cervical region.

If the choke is located in the cervical portion of the esophagus, it can usually be detected by palpation. In choke resulting from esophageal dilatation the mass of food is considerable; enough so that it can be both seen and felt in the form of a bulging area in the esophageal canal. When choke from dilatation has existed for several days food is regurgitated, the nostrils are smeared with it, and an offensive odor emanates from the oral and nasal cavities. In some cases the animal continues to eat even then. At the end of variable periods of time the patient succumbs either from an attack of inspiration pneumonia or from gangrenous processes involving the gullet. Choke from dilatation may, under some conditions, be only a partial choke.

Prognosis. The prognosis is favorable in all cases of spasmodic choke not complicated by injuries sustained at the hands of persons endeavoring to correct the condition by means of probangs or other heroic treatment. The prognosis is grave when such treatment has been administered.

The prognosis in cases of choke resulting from dilatation of the esophagus is always doubtful. Eventually all of these cases terminate fatally. A few attacks are sur-

vived, but ultimately death occurs during one of them. Dilatation choke is recurrent. Choke of the spasmodic variety is not. Choke in all forms is more serious when in the thoracic portion of the esophagus than when it is in the cervical portion.

Points of value in diagnosis and of import from the side of prognosis are:

1. In spasmodic chokes (which includes nearly 98 per cent of all cases) the symptoms are acute, urgent. In dilatation choke the symptoms are not so impressive.

2. In spasmodic choke the point at which the food has lodged can only be detected during one of the muscular cramps, in the cervical form. In dilatation choke the fullness over the obstructed portion of the esophagus can be seen or felt at all times, in the cervical form. In thoracic forms it is never possible to make this observation.

3. History of repeated attacks of choke in the same animal is almost positive evidence that dilatation exists, which will eventually cause the animal's death. Spasmodic chokes rarely recur.

4. In dilatation choke the horse may continue to swallow some food even after the choke is fully established. In the spasmodic form feed is refused.

Treatment. Spasmodic Form. The treatment of this form of choke is based on the knowledge that we possess of its pathology. Recognizing this form of choke as a simple functional disturbance, we treat the case accordingly. With the use of agents having such tendencies we endeavor to overcome the spasm in the parts involved. We know that as soon as this spasm is relieved the bolus of food that is in its grasp will pass on. Therefore, we use no probang or other mechanical appliance in the treatment of this form of choke. One grain of pilocarpin hydrochlorid, hypodermatically, will relieve most spas-

modic chokes almost as quickly as the effects of the injection become evident. If no relief is evident at the end of three or four hours the injection may be repeated. All feed must be withheld. Water is allowed. Sloppy food should be given the patient for a few feeds after an attack of choke. Cases that prove unusually obstinate should be given several days' time before it is decided to resort to surgical interference. In the meantime, an injection, hypodermatically, of a grain of apomorphin may be successful in relieving the choke.

Important points in the treatment of a case of spasmodic choke are:

1. Complete, absolute abstinence from food.
2. Access to water must be allowed.
3. Patience. Give the case time. There is no cause for worry if all feed is withheld.
4. Forbid all forms of mechanical interference.

Cases that do not yield to the above treatment within forty-eight hours are almost positive to be dilatation chokes. Judicious use of the probang, or direct surgical aid is then indicated. Cases in which the obstruction is in the thoracic portion of the esophagus, and that do not yield to medication must be patiently subjected to repeated entrances of the probang. The introduction of an ordinary stomach tube and the irrigation of the mass with water through this tube will, in many instances, yield good results if patiently and delicately carried out. The use of force is not permitted, especially in cases due to dilatation of the esophagus. The esophageal wall is very thin in some of these cases and is therefore easily torn by even ordinary pressure. In the cervical portion Merillat's operation is the operation of choice.

Horses that are known to be the subjects of an esophageal dilatation should be fed on semi-liquid feed at all times. Even this, however, does not prevent occasional

obstruction in cases that have a saculation of the esophagus. Dilatation that is diagnosed in the cervical portion of the esophagus would deserve operative interference aiming at the restoration of the normal calibre of the tube. Resection of the atonic area and coaptation of the healthy margins under aseptic precautions might prove successful.

It may occur in rare instances that the veterinarian is called to treat a case of dilatation choke in the cervical region that has been in existence for a week or longer, and that has been subjected to injury of various kinds at the hands of laymen or others. In such cases there is usually a diffuse, rather firm swelling in and around the jugular groove. There are traces of food particles in the nostril and an odorous, creamy or syrupy discharge issues from the nostril. The history which is given with the case usually helps to make the diagnosis quite sure.

Treatment, to be of avail in these cases, must be somewhat heroic. The large swelling must be incised and food particles removed from the tissues if the esophagus has ruptured or necrosed. If the esophageal tube is yet sound the incision must be carried in to reach the esophagus, lay it open and remove the collection of food or other matter. After preparing the edges of the esophageal wall appropriately, this is to be sutured. The patient is to be supported with nutritious rectal injections until healing occurs. The results from this treatment are usually surprisingly satisfactory.

HYGROMA OF THE WITHERS

While hygroma of the withers is essentially a condition for surgical consideration, it is given a place in these pages for the reason that the early symptoms are amenable to medical treatment. Hygroma of the withers has not been given much attention in veterinary literature. The reason for this is the fact that usually it is not diagnosed in its true character. Another possible reason why this condition has received but slight attention at the hands of writers is that the veterinary practitioner does not usually meet with the condition in its original and early form.

The mark of distinction between a hygroma of the withers and fistula of the withers is pus, or the absence of it. Hygroma of the withers occurs most commonly in young horses. The condition is, almost invariably, the result of bruising from ill-fitting collars, and from blows, contusions, or pressure. It makes its appearance in the form of a smoothly rounded tumefaction on top of the withers. In some cases it is apparent only on one side of the median line. This tumefaction varies in size from that of a small orange to the size of a football. On palpation the tumefaction is of varying degrees of tenseness, having the feeling of a "wind puff." There is at first considerable local heat and slight tenderness under pressure. If not treated, gradual enlargement follows. Hardness develops in the margins of the swelling, pus formation follows, abscess and fistulous openings develop later.

In rare cases the condition becomes stationary, or disappears spontaneously. A fully developed hygroma, sometimes of great size, frequently appears within so short a period of time as twenty-four hours. The formation of pus usually requires from three weeks to as many months.

The contents of an hygroma of the withers are, in the beginning, either a bloody serum or an amber-colored, sticky fluid. Later there is found in this fluid variable quantities of flat, or discoid, masses. These resemble adipose tissue, are quite firm and from one-eighth to two inches in their greatest diameters. Pus formation is probably the result of the degeneration of these masses.

Hygroma is differentiated from the tumefaction of fistula:

1. By the history of the case. Hygroma usually develops very rapidly.

2. By the "wind-puff-like" tenseness of the swelling.

Treatment. Hygroma of the withers in existence not longer than three or four days can be treated successfully with the use of prolonged hot or cold baths and astringent, cooling lotions. The animal must be kept as quiet as possible while under treatment. Work or exercise makes the treatment of no avail. The baths may be either hot or cold and should be given at least twenty minutes out of each hour of the day. Several days of such bathing or fomenting, in conjunction with lotions of lead acetate, are in many cases sufficient to cause the dispersion of the swellings. The treatment is then finished with iodine paintings twice daily for a few days. Cases that do not yield to this treatment, or those in which the tumefaction is only dispersed in part, must be treated in a surgical manner.

The swellings are incised at their most prominent points, making a good, free incision, thus allowing the escape of fluid and discoids. The cavity is then irrigated with an antiseptic solution and packed with gauze saturated with equal parts of oil of turpentine and linseed oil. This packing is allowed to remain for twenty-four hours, at the end of which time it is removed and the wound receives no treatment further than that every

three or four days the cavity is irrigated with an antiseptic solution of mild strength. Healing requires from two to four weeks. The horse can do light work during this time.

It is not always necessary to incise the tumefaction on both sides of the withers. In very many of these cases all fluid and solids can be satisfactorily evacuated by incising one side only. In selecting the site for lancing, choose the side on which the swelling runs the farthest anteriorly. It is futile to attempt a cure in cases that have not yielded to fomentations by the use of an aspirating needle or a trocar. While the fluid portion of the contents of the swelling can be drawn off in this manner, the solidified matter in the form of flattened disks remains. Within a few hours after the fluid has been drawn off, the regeneration of a similar, or even greater, amount takes place. If surgical intervention is indicated, nothing but free incision will prove satisfactory.

LAMINITIS

Although laminitis is not a rare disease by any means, a little space devoted to a discussion of its treatment is appropriate here. The symptoms require no discussion in a treatise such as this. There does not seem to be a treatment for laminitis which can be said to be standard. While in some respects all forms of treatment for this disease are similar, yet every veterinary practitioner has some hobby that he indulges in, in the treatment of this disease.

A form of treatment that has many followers is the adrenalin treatment. Here we have a form of treatment that really gives most remarkable results and that has a good foundation to stand on from a pathological and from a therapeutic sense. When used early in the attack, this treatment can be relied upon to shorten the course of the disease and to prevent disagreeable sequelæ. A one to one thousand adrenalin chlorid solution is injected subcutaneously at various points in the coronary region. Some practitioners make the injection into the meta-carpal artery. This treatment, however, is only of value in the very early stages of laminitis.

A treatment that gives uniformly satisfactory results with very reasonable promptness is the following: With a rasp thin out the horn in the coronary region as much as it will stand. The thinned area should go completely around the hoof and should extend downward toward the sole at least two inches. The horse does not offer any resistance to this rasping, despite the painful state of the feet. The shoes, if in place, are to be left in place; I can imagine no exhibition of poorer judgment than that which the veterinarian shows when he insists on removing the shoes from a laminitis patient. When the rasping off of as much horn as possible has been completed, this

thinned area is literally soaked with pure tincture of iodine. It is to be painted on with a small brush, going over the area again and again, until the horn refuses to absorb any more of it. This means at least an ounce to each foot, and the application is to be repeated every twelve hours in a similar manner. Unless the case is more than ordinarily severe, two or three of these applications will free the animal from all acute pain. The temperature comes to normal and the patient resumes a normal condition more rapidly than with any other treatment. The only improvement that I could suggest in this treatment would be to rasp down the entire wall, from ground surface to coronet, and then soak the feet in a tub of tincture of iodine. If I ever meet with a case of laminitis in a horse whose value would justify such a proceeding, I shall try it. I believe that the effect would be marvelous.

I use in every case of laminitis a daily cathartic dose of arecolin. Where this is impractical I use an aloes ball. In addition to this, every one of my laminitis patients gets a moderate dose of sulpho-carbolates compound three times a day for two or three days. With such treatment I do not find it a rare thing to see many of my cases ready for work in a week.

When, for any reason, the rasping and iodine applications can not be made, I resort to water. Hot water constantly applied often gives marvelous results. After wrapping the feet thickly with burlap, I see to it that some one soaks the burlap *every hour* with hot water. This is the only practical and a very humane method of water cure. To resort to the use of the soaking tub is unpardonable in this disease.

Encourage the patient to assume the recumbent position by providing it with a deeply bedded box stall. Instruct the attendants to make it possible for the subject

to eat and drink while down; do anything reasonable to keep the patient off its feet.

Although in laminitis we usually see very marked constitutional disturbance, the greatest effort, therapeutically, is to be applied locally. And, for fine and complete healing, nothing is of such great importance as that portion of the nursing which encourages the recumbent position. From any point of view—pathologic, therapeutic, humane—one could, with full justification, force the recumbent position on the patient by the use of appropriate restraint.

Only in parturient laminitis must we add something to the treatment of this disease that is of greater importance, namely, liberal irrigations of the uterus. Aside from this the treatment of cases of parturient laminitis does not differ, and it is the only form of laminitis in which the treatment given aims directly at the cause.

SPASM OF THE DIAPHRAGM

This pathological condition is commonly termed "thumps." Some writers have alluded to it under the title of palpitation of the heart. The condition, from its pathological side, is not entirely clear. Most authors have, however, adopted the designation of diaphragmatic spasm, and content themselves with giving clinical proof that cardiac palpitation only occurs coincidentally with the spasmodic contractions of the diaphragm. No doubt, there is a close connection between the two conditions. It has been a rather difficult matter in the past, however, to trace this connection.

For all purposes of a practical nature it is sufficient that we understand that there occurs in these cases a pathological state which is, no doubt, due to a trophic nerve disturbance of some sort, and that the symptoms which we see as a result of this disturbance are, in the main, the effect of spasmodic contractions of the diaphragm. We must accept this view if we would have our treatment of the condition conform to at least a semblance of scientific handling.

"Thumps," or diaphragmatic spasm, is seen most commonly in driving horses. Since the advent of the automobile, it has been forced into that class of diseases usually termed "rare," because of the fact that the class of horses which was most frequently affected is now decidedly in the minority. Heavy horses performing slow and methodical work are hardly ever affected. Causative factors in the development of diaphragmatic spasm are over-exertion, especially during hot weather, heat, great excitement in nervous animals, and occasionally debilitating infectious diseases.

The mortality rate is low, but the affection is important from a clinical standpoint because of the fact that the

practitioner is occasionally embarrassed in the presence of cases that prove stubborn under treatment, and because death does occur in some cases quite unexpectedly. Although recovery is the rule in this disease, the practitioner must be reserved in his prognosis. The clinical picture of a case of diaphragmatic spasm is a grave one in the eyes of the client. For the practitioner the case has no terrors, because he knows that most of these cases recover in from two hours to as many days. Now and then one dies, however, after the attending veterinarian has in all sincerity assured the client that the patient was in no danger of losing its life. And, usually, not only is the patient lost in these cases; the client is "lost," too. A safe rule on this point is to "view with suspicion" all cases of this affection that show no improvement within a few hours after treatment is begun.

Symptoms. The most prominent symptoms in "thumps" is the thump. It is a thumping sound, usually heard in company with, or immediately after, the heart beat. The sound can be heard plainly at quite a distance from the horse. The animal is much distressed and uneasy, the respiratory movements are accelerated, and with each "thump" the entire body moves in a jerky manner.

The condition must be differentiated from an acute exacerbation of heaves. This can be done by carefully noting that the abdominal muscles in the flank region remain rounded, lacking the expiratory flatness characteristic of that act in heaves. If further confirmation is desired, a rectal examination can be made. In a case due to spasm of the diaphragm the contractions of the latter can be plainly felt along the borders of the last ribs, and these contractions will be found to coincide with the thumping sounds.

Treatment. Morphin in full doses. From four to eight grains are given hypodermatically. If the diagnosis is correct, the treatment should show marked improvement within an hour. Cases occurring during hot weather from over-driving should be given cold shower-baths. All cases should have fresh air, should not be moved unnecessarily, and should have constant attendance until almost completely relieved.

MELANOSIS

Pathologists tell us that melanosis is a form of cancer. The tumors or nodules presented in this condition are said to contain pigment. The disease affects white and grey horses exclusively, appearing in the form of black nodes or buttons in the region of the anus, the under side of the tail, the sheath and penis, the eyelids, the lips, and at times in the skin on various other parts of the body. These nodes make their appearance at from the sixth to the tenth year of the animal's life. They increase in size slowly, but attain great dimensions, relatively, in very old horses. In the anal region they are frequently seen in clusters, occasionally in such numbers and of such size that the anal opening is displaced or hidden. In other parts of the body these tumors do not attain great size nor appear in such great numbers as they do in the anal region.

The disease is, without question, hereditary. The get of a certain Percheron horse in this state, most of which were grey, are almost without exception affected with melanosis. I have treated five or six of these horses, and every one of them has from ten to thirty melanotic tumors.

Only rarely does melanosis cause serious, or even troublesome, disturbances. In the anal region a melanotic node may suppurate or break down, producing a mild periproctitis. The discharge from melanotic tumors is at first a tarry, black paste. Later, ordinary pus mixed with bloody streaks appears. In some instances the development of a melanotic growth in the rectum or above the rectum in the sacral region may attain such size that it interferes with the fecal evacuations, either from direct blocking of the rectal canal, or from pain produced during the act of defecation.

When melanosis affects an eyelid it must be differentiated from botryomycotic nodes. I have seen the two conditions affect an eyelid simultaneously.

Melanotic growths in the sheath may prevent normal protrusion and retraction of the penis, resulting at times in a severe paraphimosis.

Obscure cases of lameness in aged white horses that have melanosis may occasionally be attributed to pressure from a melanotic tumor on a nerve trunk in the parenchyma of a muscle in the affected limb.

Traumatism in the immediate vicinity of a melanotic tumor nearly always results in serious consequences. Either the growth of the tumor is greatly stimulated thereby, so that a previously passive nodule becomes a rapidly growing, malignant tumor, or the wound itself assumes a vicious and stubborn character. Apparently the tissues surrounding such tumors are already subnormal in vitality and resisting power.

Melanosis in horses under ten years of age is usually passive. After the tenth year a passive melanoma in any part of the anatomy may become active. This should be considered in examining grey or white horses for soundness. A thorough examination for soundness in grey or white horses must always include examination of the rectum, with the object of detecting the presence of melanotic growths in this canal. Their presence in, or surrounding parts of, the rectum constitutes an unsoundness in horses of any age. Some cases of rectal and anal fistula have their origin in a degenerated melanoma. On account of the hereditary nature of melanosis, the breeding of horses affected should be discouraged.

Treatment. None while passive. When an isolated, single tumor becomes active in a region where a thorough enucleation is possible, it should be dissected out and the bed of the growth thoroughly cauterized. Simple dis-

section, without subsequent cauterization of the bed of the tumor, usually means a recurrence within a very short time. The secondary tumor is more active than the primary one whenever a recurrence takes place.

When a melanotic tumor becomes active in a region where a thorough dissection would be out of the question, the treatment can, of course, be only of a palliative nature. What this would be depends upon the location of the tumor and the extent to which it is hindering the function of the parts involved.

While an active melanotic tumor is a grave condition in any part of the anatomy the attending veterinarian must never be pessimistic in giving a prognosis. In many instances these growths suddenly degenerate, discharge their contents, and a spontaneous cure results. This is especially true of these tumors in the anal region, and also when they develop within the rectum. A rectal examination should be the rule in all grey horses suffering from colicky pains which cannot be clearly attributed to acute indigestion. Impaction just anterior to the rectum will frequently be found as the result of a melanoma involving the rectum or its surroundings.

Melanosis affecting internal organs, such as the spleen, the liver, or other important organs, the heart at times, is not considered in this chapter because the symptoms produced do not vary from other malignant processes in these parts. The diagnosis is only made on autopsy. The treatment, even were an ante-mortem diagnosis made, would no doubt be futile.

It is well to bear in mind, however, that obscure abdominal and thoracic affections, especially those of a sub-acute or chronic nature, in white or grey horses may be the result of melano-sarcoma in the organ suspected.

CHRONIC POLYARTHRITIS

Chronic inflammatory processes involving more than one articulation at the same time occur with fair frequency in horses. The condition is most often seen in horses past eight years of age, but younger horses are not entirely exempt. The etiology is not always the same, and in many instances this cannot be ascertained. By far the greatest number of these cases are the result of acute polyarthritis that developed at an earlier period of the animal's existence as sequelæ to infectious diseases. Influenza, complicated strangles, pneumonia and pleurisy can be named as examples of predisposing affections. Some cases are, no doubt, due to a rheumatic diathesis, and are precipitated as the result of unhygienic surroundings, insufficient nutriment, and excessive work. The process is always non-suppurative, and is characterized by a tendency towards malformation by the development of osseous changes, ligamentous and tendinous thickenings, and subsequent muscular atrophy.

There are in this condition periodic exacerbations of lameness. Between these periods the horse is merely "sore." This "soreness" is peculiar in the fact that it disappears to a great extent, temporarily, with exercise. The changes in the joints, consisting of enlargement chiefly, begin to appear at the end of several months. With their appearance comes permanent lameness. Several months, at times a year, later wasting of the muscular frame in the affected limbs begins. The horse eventually becomes "out of condition" generally, tires easily, refuses its feed after strenuous exercise or work, and finally is wholly unserviceable. In this stage most of these horses rarely lie down; when they do at times assume the recumbent position, they require some assistance in getting up.

In the early stages, during the exacerbations of lameness, there is a slight rise in temperature. The diagnosis must exclude osteoporosis more than anything else. This is done by noting that the bony enlargement is confined wholly to the articulations, while in cases of osteoporosis the shafts of long bones, the flat bones, and the axial skeleton generally, are involved. The entire course of chronic polyarthritis in horses requires several years to exhaust itself. The prognosis is invariably unfavorable.

Treatment. In the early stages I have seen some good results from treatment aimed at the supposedly infectious character of the trouble, such as bacterin therapy with strepto-bacterins and staphylo-bacterins, and the administration of such remedies as salol, hexamethylenamin, and arsenic. Locally, active counter-irritants are employed. When marked changes have occurred in and around several articulations, not much remains to be done.

Relief from lameness can be obtained for short periods of time in the latter cases by the administration of a mixture containing potassium iodid, phytolacca and colchicum seed three times daily for a week or ten days. The enlarged and deformed articulations are to be massaged with oily preparations of iodine, or with mild mercurial ointments.

The use of the actual cautery with the object of correcting the joint lesions and overcoming existing lameness is in these cases insupportable.

The joints most commonly affected are the pastern, the ankle, the hock, the stifle, and the carpus. The elbow and shoulder joints are rarely involved. In a few cases I have even seen involvement of the temporo-maxillary articulation.

From the good results derived in the early stages of some cases with the use of bacterins, it is fair to assume

that most cases could be brought to a satisfactory termination in the early stages if an autogenous bacterin were used. The results should be much better with autogenous bacterins than I have been able to get from the use of stock bacterins.

OSTEOPOROSIS

This is the American term for that form of osteomalacia which affects horses in this country. It is what might quite properly be termed a sectional disease. In certain parts of the United States it is a very common disease, while in other sections hardly any cases are seen. The disease is supposed to be due to a serious lack of lime salts in the feed. At any rate, there is a very decided deficiency of mineral matter in the osseous tissue of animals affected with the disease. Recently the disease has been classed as an infectious disease. No satisfactory proof of such an etiology has yet been given, and the clinical manifestations do not in any manner support it.

Osteoporosis is a chronic disease, choosing mature or aged animals for its subjects more frequently than young animals. Its course covers periods of time varying from a few months to several years. The prognosis is in every instance unfavorable.

Symptoms. Osteoporosis makes its appearance insidiously. Once the changes in the bone are recognized, the disease has already established itself quite fully. This is probably the chief reason why the treatment is unsatisfactory. The early symptoms are rather obscure; by the time symptoms of sufficient prominence to make a diagnosis possible are manifest, the disease has already done so much damage that there is little chance for restoring the health of the animal. Even in regions where the disease is common most cases are not recognized until structural changes have occurred in the skeleton, so insidious

is its approach. A common early symptom is obscure lameness. In regions where osteoporosis is of frequent occurrence this disease should be suspected in every instance in which an obscure lameness occurs, especially if in addition to the lameness there is a history of capricious appetite, lack of endurance, a tendency to perspire easily and a tendency toward leanness.

Another symptom that occurs quite early in the course of this disease and which is quite reliable for purposes of early diagnosis is "cracking" of the joints. This "cracking" is a sound that is very similar to a loud snapping of the fingers. It is heard with the first movements that the horse makes after he has been standing at rest for some time, and also at times when a sudden, quick turn is made by the animal.

As the disease progresses the horse becomes unthrifty. His frame assumes an abnormal setting, most noticeable in a decrease in the obliquity of the pelvis. If the disease is active in the bones of the limbs, these seem to increase in thickness, "buck-shins" develop, and the shafts of all the long bones become more rounded and increase in diameter. The bones involved in the disease process are easily fractured, and it is not uncommon for a fracture to occur while the animal is going through ordinary or normal movements. In other cases tendons pull loose from their insertions, ligaments are torn loose, and similar complications are met with frequently.

When the disease affects the bones of the skull we see what is usually termed "big head." The facial bones seem to puff out, giving the face a full appearance. As a result of changes in the facial and nasal bones there may be obstruction of the lachrymal duct; narrowing of the nares and involvement of the turbinates may produce difficult breathing. The rami of the inferior maxilla are rounded and thickened. The molars become loose, so

that mastication is imperfect, if not entirely impossible. We remember the case of a Shetland pony in which the course of the disease covered nearly four years; every year during this time one or more molars dropped out or were extracted. At the end of the fourth year not a tooth remained. When we last heard of this animal it was being kept alive on gruel. This is one characteristic of osteoporosis when it affects chiefly the bones of the skull, namely, that it frequently confines its ravages to these bones alone. The animals may, in such cases, live for years, showing no other manifestation of the disease except the so-called "big head."

Cases of osteoporosis presenting symptoms indicating general involvement of the osseous elements either slowly go on to death from inanition and exhaustion, or they come to an end from such complications as fractures, ruptured tendons, and so forth. In some instances the disease seems to become checked suddenly, remaining stationary for years, and then suddenly becoming active again.

Treatment. When the characteristic changes have occurred in the bones treatment is useless. If the diagnosis is made early the addition of lime salts to the feed, or the feeding of foodstuffs obtained from regions where the disease does not exist, will frequently stop the progress of the affection.

MANGE

Mange, or scabies, or scab, in horses is usually of the sarcoptic form. It is a disease affecting the skin and hair, and at times has a general devitalizing effect upon the subject. While the disease is contagious, it also occurs sporadically.

Symptoms. The disease begins with itching, as a result of which the horse rubs the infected parts against any convenient object. The parts first infected are usually the head and neck, shoulders and withers. Later the entire skin may be involved. Following the itching stage there appear numerous pimples or nodules of small size, which later burst and form scabs. The hair in the region comes out in tufts or patches, so that in a well-marked case considerable areas of the skin become hairless. When the disease is generalized and has existed for a long time the patient becomes emaciated and unthrifty.

Treatment. Affected horses should have isolated stalls, individual brushes, curry-combs, and harness. So that treatment may be most effectual, the animal should be clipped first. The affected area should be thoroughly scoured with soap and warm water, so that all scabs are thoroughly removed. The diseased skin is then to be thoroughly anointed with a mixture of equal parts of oil of cajuput and cottonseed-oil. This anointing should be done quite vigorously, and is to be repeated again three days later, but omitting the washing with soap and water. Usually, after the second application the affected area shows signs of healing, and another application or two completes the cure.

Fresh eruptions on other parts of the body are treated similarly as they appear. In chronic cases the internal administration of Fowler's solution of arsenic helps to bring about a more rapid recovery.

EPIDROSIS

Abnormal activity of the sweat glands is termed epidrosis. It is a condition that occurs in horses as a clinical entity and, while not of *very* frequent occurrence, it does occur frequently enough to be given some consideration. The condition usually proves to be stubborn under treatment, and therefore cannot be said to be a benign disturbance.

Epidrosis occurs commonly as a symptom of various constitutional diseases, in which form its treatment depends upon the disease that it accompanies. In this chapter we are considering excessive sweating as a condition *per se*, a condition entirely different from the epidrosis seen with other diseases.

Symptoms. A horse that is otherwise in perfect condition perspires freely when the atmospheric temperature does not affect other horses in this respect at all. In some cases the entire body is moist with sweat, while in others only certain parts, such as the neck, withers, flank, or the abdomen, alone become moist. This occurs while the horse is at rest as well as while it is active. In some cases the condition is only evident at night, and it persists even during the cold weather of winter months. In every other regard the horse is normal.

Treatment. If the coat is "woolly," the horse should be clipped. The medicinal treatment is empirical and experimental. Some cases are benefited by repeated small doses of aconitin, giving one one-hundred-and-fiftieth of a grain three times a day for a few days, then twice a day for several more days, and finally only once a day for another week.

Other cases may be completely cured by bathing the entire body several times with a mild solution of alum. The sweating can always be temporarily checked by small doses of belladonna, repeated two or three times.

INTERNAL HEMORRHAGE

Bleeding from a large vessel in one of the cavities of the body occurs occasionally in equine subjects. A hemorrhage of this kind may follow a hard pull on a heavy load after a period of idleness, and in such instances may be confused with azoturia. Moderate internal bleeding does not provoke marked symptoms aside from the altered character of the pulse. Serious internal hemorrhages are accompanied by trembling of muscle groups in all parts of the body, the horse appears drowsy, and may perspire in spots. The pulse is thready and fast; the temperature subnormal by a degree or two. The visible mucous membranes soon become pale or blue-pink, and the respirations rapid.

The prognosis is not unfavorable as long as the pulse can be felt at the maxilla. When the pulse becomes imperceptible in the glosso-facial artery the condition is in grave danger of being fatal.

The important factor in the handling of an internal hemorrhage is the prompt diagnosis thereof. If the case is at first confused with something else and considerable time is lost, it may terminate fatally, although it may have come into the veterinarian's hands early enough for a favorable outcome had the proper treatment been given at once.

The first thought in every inaccessible internal hemorrhage should be atropin. From one-fourth to one-half grain of atropin sulphate should be given hypodermatically. If convenient, the water used for the solution should be warm, so that absorption will begin the instant it is injected. The patient should not be moved; treat him where you find him, and do not allow him to be moved until all alarming symptoms have passed off. When it appears safe to the attending veterinarian, the patient

may be placed in comfortable quarters. The animal must be tied short so that the standing position will be maintained for at least twenty-four hours. Warmth should be applied to the body in the form of warm blankets, and if the hemorrhage has been an extensive one, tincture of iron chlorid should be administered for a week. In valuable animals quantities of normal salt solution may be given per rectum or subdermally. A dose of lime water, two to four ounces, may be given internally to assist the atropin sulphate. Lime salts favor coagulation. In applying warm blankets care should be taken to lay them on loosely; do not cinch them up. Loosen the halter, if tight, and allow no binding of any kind on any part of the horse. The object of this is to lay no hindrance in the way of the greatest amount of blood to come to the body surface, thereby lessening the quantity and the pressure in the region of the hemorrhage.

In aged horses considerable time may elapse before they again attain their former vigor and stamina after a severe hemorrhage. Young horses, as a rule, recuperate rapidly; frequently a few days of rest and good care suffice to bring them back to normal condition.

PSEUDO-HYSTERIA IN THE UN-SEXED MALE

In geldings we may occasionally meet with a set of symptoms, apparently nervous symptoms, peculiar to themselves. The subjects thereof are without exception high-strung, spirited animals. In every instance that has come to my attention they have a nervous temperament, and are possessed of one or more bad habits, such as biting, striking or cribbing. Lacking these, they are what is usually termed "cranky." Only geldings possessed of "defective" temperaments such as these develop attacks of pseudo-hysteria.

Symptoms. The attack comes on suddenly, and usually while the animal is at rest in the stable. The manifestations range in severity from a mild effort at obtaining freedom to methodical outbursts of studied viciousness. In rare cases the manifestations take the form of ridiculous antics, such as shying in the stall at imaginary objects, striking at imaginary objects, and so on. In one case under my care the gelding would stand for hours swinging and jerking his head in exact imitation of a horse resisting bot flies. Another case persisted in standing on the edge of his manger with his forefeet. When taken out of his stall he acted normally. As soon as returned he again assumed the position on the edge of the manger. He did this for three days, after which he again became normal. During the time that the attack was present he showed nothing that could be called abnormal, aside from his mania for standing as described. Other cases refuse to remain tied, or even object to confinement in a box-stall. A gelding that I saw recently gave every indication of being violently insane when forced to remain in the box-stall he had occupied willingly for many months. This mania lasted nearly three days, and abated temporarily as soon as he was turned

into a lot. After three days of such exhibitions he again became as normal and docile as he ever had been.

Attacks of pseudo-hysteria persist, with intermissions of quiet, for from one to three days. They are to be differentiated from staggers, various forms of poisoning, and rabies. The chief difference is that in staggers there is lack of orientation. The hysterical gelding is fully aware of what he wants to do, and he does whatever he wants to do (or what his particular form of hysteria makes him do), methodically and persistently. The horse with staggers of a violent type has apparently lost all sense of direction and proportion.

Poisoning, belladonna, hyoscin and cocain poisoning are to be considered.

Rabies can be excluded from the fact that there have been no premonitory signs, and that the actions in the disease under discussion, although at times violent, are methodical and apparently performed with a distinct purpose.

Treatment. If the patient's actions are of a violent nature, he should be given his freedom in a lot or pasture. Some cases respond satisfactorily to an eighth of a grain hyoseyamin, hypodermically. Others improve rapidly on small doses of atropin sulphate. Chloral hydrate and bromides are of benefit in those of a violent form. A drastic purge will check some cases with marvelous rapidity.

Prognosis. This is always favorable under treatment. The duration of an attack can, with all propriety, be predicted as terminating under three days. The pathology is unknown, or disputed.

PERMANENT INTERNAL HYDROCEPHALUS

The presence of fluid in abnormal and permanent amount within the ventricles of the brain produces a pathological state in the affected animal. This pathological condition has been described by various writers under such names as chronic internal hydrocephalus and chronic dropsy of the ventricles. Among horsemen the horse so affected is called a "dummy."

Permanent dropsy of the ventricles may follow any acute disease of the brain and spinal cord, or their meninges. In other instances it develops gradually as the result of slowly progressing anatomical changes in the region of the ventricles. In all cases the symptoms produced ensue from the effects of increased intra-cranial pressure.

Symptoms. The best description of the symptoms in this condition is conveyed by the name which horsemen have given it, namely, "dummy." Horses that are the subjects of a permanent internal hydrocephalus appear to possess only a fraction of normal alertness and vivacity. The physiognomy at once betrays the low degree of consciousness; the expression is almost comparable to that of idiocy. The horse performs a variety of abnormal movements in connection with feeding, drinking and other ordinary functions. Grotesque and odd positions of the limbs are frequently assumed. The horse at times leans against objects, rests the head on railings, posts or mangers, appearing partly asleep in the act. While eating, the animal frequently pauses, standing idiotically for variable periods of time with a partly masticated bolus of feed or hay held in the mouth. While drinking, the horse frequently immerses the head to the eyes, suddenly and surprisedly jerking up the head as it finds the water entering the nares.

The condition runs a slow course, becoming more marked month by month. Soon the animal finds it impossible to move in a straight line, and usually backing becomes difficult. If the animal is forcibly and quickly shoved backward it will either fall or flounder clumsily about.

In some cases the horse has spells of violent attacks. These last for several hours at times, during which the animal presents a set of symptoms generally described as staggers. It plunges about, falls, rises again, and goes through similar antics frequently. In one case occurring in my practice a violent spell of this sort lasted nearly forty-eight hours, during which time the horse succeeded in completely demolishing its stall and eventually broke loose and ran amuck. It was later recaptured in an exhausted, sleepy condition.

"Dummies" eventually become wholly unserviceable. The condition is always incurable. In the early stages it is recognized by testing the alertness and the acuteness of sensibility, as well as by noting the progressive development of permanent sluggishness. These horses always have a slow, full, soft pulse; and in nearly every case the respiratory motions are abnormally slow and deliberate. The horse is not easily excited by noise that formerly alarmed it, and it responds slowly or not at all to slapping, or to commands. Violent spells, such as have already been described, can frequently be aroused by making the horse perform unusually hard work, and at times by confinement in hot stables during extremely hot weather. Because of the possibility of an unexpected violent spell of this kind, horses so affected should be considered unsafe and dangerous.

Treatment during these spells is wholly symptomatic. Treatment of the condition itself is useless.

MENINGISM

Meningism has been defined by various writers as a sub-acute or mild meningitis, which may be cerebral or spinal and which quite frequently is cerebro-spinal. The etiologic factor is said to be, in almost every instance, an auto-intoxication of intestinal origin. It affects horses in nearly all parts of the United States, showing a preference for young animals, from sucking colts to those just short of maturity. A predisposing cause is idleness and an unbalanced ration. Most cases are seen during the winter months.

Symptoms. Animals affected at first show slight indisposition, appear sluggish, are "off feed," and do not lie down. At this time there is a rise in body temperature of from three to five degrees Fahrenheit. The pulse is full and slow. Within a few days the animal shows some difficulty in swallowing. There is beginning a lack of coördination in the movements of the hind legs. Those which lie down are unable to arise without assistance. The temperature becomes sub-normal in most cases after the inaugurating fever subsides, usually at the end of several days. Constipation is usually evident. In some cases the early symptoms are so mild that the first thing noticed is the lack of coördination in the posterior members. The animal has a "wobbly" gait and, in turning quickly, may fall.

A few cases may be ushered in with more force. The animal appears distressed, shows signs of wanting to "forge ahead," pushing forward against the manger or into a corner. There may be a slight rigor. After twenty-four to forty-eight hours the case assumes the more sub-acute aspect first described. The symptoms enumerated slowly disappear, with the exception of the lack of coördination in the posterior members. This

may hang on for months, even with treatment, and in some cases remains permanently. The mortality is very low, probably not more than two per cent. Cases in which the early symptoms have passed unnoticed, may be found down, unable to rise.

The condition must be differentiated from azoturia, and from injuries to the lumbar or sacral region. In some exceptionally well marked cases there is ptosis affecting one eye-lid.

Treatment. Cases that are found down should be given from one-fourth to three-fourths grain of atropin sulphate, hypodermically, before anything else is done. This gives marvelously prompt results in many of these cases; the animal frequently rises without assistance in an hour or two thereafter.

In those cases which come into the veterinarian's hands in the early stages, a purging dose of arecolin or eserine is indicated, but it is a question whether this is good treatment, everything considered, because of the additional effusion that may occur into the meningeal spaces as a result of the action of these drugs. I have treated animals affected with this disease by giving arecolin and coördination was more interfered with than in cases where arecolin was not used. For that reason I say that a purging dose of arecolin or eserine (basing our treatment on the intestinal stasis) would be indicated. While the early signs of the disease are in almost every case promptly checked by a dose of either of these drugs, I do not recommend their adoption as routine medicament. I much prefer a cathartic dose of aloes.

Intestinal antiseptics are to be administered from the beginning and should be kept up for at least a week. Either salol or the sulpho-carbolates compound can be used, giving half-dram doses of the former and sixty-grain doses of the latter.

In the beginning, a few large doses of hexamethylenamin often prove very beneficial. A two-dram dose can be given freely diluted with water, until at least three doses have been given, allowing two or three hours to elapse between doses. The intestinal activity should be encouraged by repeated small doses of fluid extract of cascara sagrada, or by small doses of magnesium sulphate.

In those cases in which the initial symptoms are marked by a tendency to move forward and by pushing the head against objects, blood-letting gives fine immediate results. The patients should be kept in dry, warm and roomy quarters and should have free access to fresh water.

In most cases of meningism, during the course of which the animal assumes a recumbent position and then is unable to rise, the animal will gain the standing position with slight assistance, such as lifting at the tail.

Those cases in which the lack of coördination persists after all other signs of the disease have disappeared should receive a prolonged course of treatment with iodid of potassium. If the progress towards improvement is slow, Fowler's solution of arsenic should be alternated with the iodid of potassium. The iodid can be given for a week and then a week of arsenic administration. This should be continued until improvement is evident, after which the iodid of potassium is to be given alone until the recovery is complete. While under this treatment for the correction of the coördinative loss the animal should have light exercise. In a few cases complete recovery seems impossible. If recovery has not taken place after four or five weeks of potassium iodid treatment, the case is usually incurable.

The differential diagnostic mark between meningism and true cerebro-spinal meningitis is the lack of opisthotonic fixation of the body. In true inflammatory conditions of the meninges, opisthotonos occurs when the

patient is urged to get up. The head is raised, the neck is flexed in a backward direction and the fore-legs are stiffly extended. In meningism this does not occur. The body remains flaccid. This diagnostic mark is positively reliable.

VERTIGO OF YOUNG HORSES

This is a condition that is not very well understood from an etiological, or from a pathological, standpoint. It is an affection that confines its appearance almost wholly to colts, from the first to the fifth year of life. Breed, sex and condition do not seem to have any bearing on its occurrence. It is seen in poorly nourished colts with no greater frequency than in well fed individuals.

Symptoms. The symptoms in this condition appear intermittently and irregularly. Between the attacks the colt is in perfect health and quite careful examination fails to disclose any evidence of the disease. An attack of vertigo is ushered in with symptoms of confusion. This is of very short duration, not longer than a minute or two, and is immediately followed by staggering, stumbling and decubitus. The colt lies quietly on the ground for a few seconds, apparently only partly conscious. It rises, shakes itself; it may then whinny, and appear to be in perfect health.

Another attack may come within a few days thereafter, or it may take several weeks before another attack occurs. As the colt becomes older the attacks become milder, so that the animal merely stumbles to knee position but does not lie. The periods between the attacks are prolonged until at maturity the attacks are no longer noticeable. From this it would seem that the condition has something to do with glandular secretions bearing upon the attainment of maturity.

Vertigo in colts is only serious when it occurs in ani-

mals that are already old enough to work. The precipitation of an attack while the animal is in harness may result in injury to the horse as well as to the driver thereof.

Treatment. As the attack rarely lasts over a few minutes, nothing is needed in the line of treatment that can be of service during the attack. The veterinarian, if called, never arrives in time to witness an attack. It can do no harm to endeavor to prevent the recurrence of attacks by the use of alterative and tonic treatment, although it is extremely doubtful whether any form of treatment would accomplish this. It usually suffices to assure the client that the attacks are not serious and that they will eventually subside altogether.

The development of a somewhat nervous disposition is an occasional sequel of vertigo in young horses. Whether this is a direct effect of the disease, or merely apprehension psychosis, I am not able to say.

COUGHS

Coughing in horses is a subject worthy of a separate chapter in any book on equine therapeutics. To properly diagnose and correctly treat coughs in their varied forms is no small accomplishment. It is to be regretted that for many veterinarians a cough is merely a cough. The same "cough mixture" or treatment does service for some practitioners whether the cough be due to a cold, laryngitis, or heaves.

While in a great many cases a cough is merely a symptom of other disease conditions, there are presented for our consideration fully as great a number of cases in which the cough is the chief symptom—in fact, the only symptom that we are expected to correct. All of us are familiar with the "Doctor, can you give my horse something for his cough?" question; we hear it almost daily. If we are anxious to do our client a good turn, and our profession credit, we do not settle this question with a dose of made-to-order "cough mixture." We make an effort to get a good history of the case first of all. How long has the horse been coughing? When does he cough most; in the morning, after eating, while eating; or does he cough at all hours with equal severity?

When we have ascertained this, we are ready to examine the patient, and the examination usually follows a line that has been suggested by the client's replies to our questioning. We always take the patient's temperature before we do anything else. The thermometer can be relied upon to indicate whether we are dealing with an acute or a chronic condition; and many times that is the most important item to consider in the treatment of a cough. The client's statements are not always reliable.

When we have assured ourselves that the cough is not the result of an acute disturbance, such as colds, bron-

chitis, laryngitis, etc., we make an effort to classify it by forcing the patient to cough. This can usually be done by firm pressure in the laryngeal region exerted by gripping the parts firmly with the whole hand. If this fails to bring a cough from the patient, a smart tap over the middle of the trachea will often produce it. In still other cases a squirt of cold water into the pharynx by the use of a dose syringe will arouse a cough. Our examination further includes careful auscultation of suspected regions—larynx, trachea and thorax. In cases pointing to thoracic lesions, percussion often tells us all we need to know for purposes of treatment. When diagnosis has been made, treatment is prescribed to suit the particular condition affecting the individual examined.

If we are satisfied, from the history and findings of our examination, that the patient coughs most markedly in the morning, because of a catarrhal condition of the pharyngeal mucous membrane, the cough being an effort to dislodge mucus accumulations that have collected over night, we give small doses of dichromate of potassium.

A case that we decided has resulted from an improperly treated or a neglected distemper we give iodid of potassium.

The patient that has a chronic cough and at the same time gives evidence of its becoming a roarer is given one ounce of a two per cent aqueous solution of tincture of capsicum several times daily.

The patient with a bronchial cough is given Fowler's solution of arsenic.

The cough that "hangs on" after an attack of pneumonia usually does not remain long if we give the patient small doses of beechwood creosote in glycerin several times daily.

Occasionally we meet a case wherein there exists a dry, hacking cough, that is the result of an irritable state in

the pharynx, because of a "bad mouth." The necessary dental attention properly executed ends the cough.

A short, choppy, dry cough with spells of dyspnea may be due to a heart lesion. This must be classified and properly treated if possible. This class of coughing patients usually come with a history of occasional epistaxis.

In this whole list of varied pathological conditions, and it is only an incomplete list, the predominating symptom and the symptom that brings the patient to the veterinarian, is the cough. The practitioner that is satisfied to treat, and tries to satisfy his client by treating all coughs with stock cough mixtures is not only doing poor practice, but is also doing poor business. Many times he tries to cure a ten-dollar case with a fifty-cent bottle of "cough mixture." His treatment is frequently no more effective than so much water. To hit a target one must aim before firing.

The object of this random chapter on coughs and their treatment is to impress upon the general practitioner the futility of attempting the correction of conditions having such a varied pathology by the use of a common agent. One would show just as poor judgment were he to attempt the cure of all forms of lameness by the use of liniments. The use of stock cough mixtures by the practitioner is the result of "getting into a rut" more than anything else, and the effect of the tendency, now so general, of using ready-made pharmaceuticals. The alluring formula of the average proprietary "cough mixture," containing sometimes a dozen ingredients, that is offered for veterinary use by most pharmaceutical houses at a cheap price, makes it a "best seller." The average veterinarian, with little trouble, could demonstrate both a chemical and a physiological incompatibility in most of these cough remedies. He uses them against his own better judgment.

PNEUMONIA

We shall confine this discussion of pneumonia to that of treatment. There is in all veterinary practice no other disease in the treatment of which good judgment is so valuable as in this. If it can be said of any disease to which the horse is subject that "Dr. So and So killed him," pneumonia is that disease. Were I the owner of a valuable horse afflicted with this disease and given my choice, in the selection of a veterinarian to treat the case, between a recent graduate with a grip full of drugs and an old practitioner with nothing but a head full of good judgment, I would say, "Keep your young fellow with his grip full of drugs and give me the man with his head full of good judgment." I would be the last person on earth to lay anything in the way of the young graduate's progress, but I firmly believe that in most instances he is seriously handicapped in the handling of pneumonia because of a lack of practical experience in this particular work.

So many young practitioners seem to forget what their instructors told them about pneumonia almost as soon as they graduate; at least, they seem to forget the most important point from the side of treatment, and that is that *you can not abort a case of pneumonia!* If a practitioner knew nothing else about this disease, but had it indelibly ground into his brain that pneumonia is a self-limited disease, and that he is only lessening the chances of his patient for recovery when he attempts to cut short or interfere with its regular course, he would be sufficiently qualified for all practical ends. Such a man would at least do no harm to the patient. Just as soon as the treatment in a case of pneumonia takes the form of heroics it is harming the patient. I do not intend to modify or qualify this statement under any circumstances.

Another item that seems to have some effect on the young graduate's methods of treating pneumonia cases is that pertaining to the classification of the disease into different forms, such for instance as croupous pneumonia, lobular pneumonia, contagious pneumonia, pleuro-pneumonia, and so on. On this item we can adopt another slogan; we should think, for purposes of treatment, of pneumonia as an inflammation of the lungs and their covering. That is enough—aside from safeguarding the health of other animals in cases of contagious pneumonia, by enforcing regional quarantines, and the possible use of specific or biologic agents (for prophylaxis only), the treatment applicable is the same in all cases.

The reader will note particularly that I say pneumonia is to be considered as an inflammation of the lungs *and their coverings*. I say this because I do not believe that a case has ever occurred, or that a case ever will occur, in which the pleura is not a participant. We recognize the involvement of the pleura only when the symptoms produced by its involvement equal or over-shadow those of the pneumonitis. When the participation of the pleura is so nominal that the symptoms do not betray the involvement, we are content to believe that there is no pleurisy.

From what has just been said I do not wish to have the reader arrive at the conclusion that a case of pneumonia requires no care. On the contrary, the correct and successful handling of an attack of pneumonia is not a very simple matter.

The treatment of pneumonia should have as its main object:

1. To give such assistance to the defensive and constructive forces of the patient's system as will make it more easily possible for the patient to survive.
2. To incorporate into this assistance such treatment

as will have a tendency to prevent complications and disagreeable sequelæ.

3. To shorten the period of convalescence.

Under 1: Stimulants, tonics, hygiene, suitable diet, nursing.

Under 2: Bacterins, serums.

Under 3: Alteratives.

The clinical evidence, and the post-mortem findings in fatal cases, absolutely rule out depressants in the treatment of pneumonia. This evidence demonstrates conclusively that nearly all antipyretics are heart depressants and they are therefore contra-indicated.

In my own practice I start the handling of every case of pneumonia with a counter-irritant, usually a common mustard plaster. While I can not point out any good or authoritative reason for this use of a counter-irritant, I do know that it has much good in it. This good effect may be only of a transient character; one would almost dare say that it was psychological in character. Nevertheless, it is very evident, and very promptly so. Immediately after the application of a good mustard plaster there is a change in the patient; it seems to put him into shape for the fight he is about to enter. Case after case I have had in which the horse, in my opinion, owed his recovery from the attack of pneumonia to this old-fashioned mustard plaster, more than anything else.

As routine internal medication, *nux vomica* or strychnin must be given the first place. The constancy of the effect produced by repeated correct doses of *nux vomica* is very gratifying. The same effect can not be obtained with strychnin alone. My method is to give from one-half to one dram of fluid extract of *nux vomica* every three hours during the day. I do not increase this dose unless there are signs of an impending crisis. I then give one-fourth grain strychnin sulphate hypodermically

at such times as the symptoms (chiefly the heart action) call for it. If strychnin alone is used throughout, the appetite does not remain so good as it does when nux vomica is used, and the effect of strychnin alone is not so constant.

I would give second place to spartein sulphate as routine internal medication. Given in about twenty-grain doses, orally, it has shown evidence of producing some fine results. I do not think that it gives as uniformly gratifying results as nux vomica. Spartein sulphate seems to greatly relieve the heart in those cases marked by a genuine crisis; but apparently it exacts the limit of heart power from the very beginning, leaving no reserve force that can be called into action when it is of the most vital importance. In this reserve force and its being called upon and brought into action at just the proper time lies one secret in the successful handling of pneumonia cases. It is doubtful whether it is possible to gain this knowledge in any other manner than by actual contact with the disease in practice.

It is good practice to inject every patient in pneumonia cases with a moderate dose of polybacterins. This has clinically been shown to be of great worth for the prevention of complications that might result from the activity of pus-producing micro-organisms. The bacterins should be given on the day that treatment is begun. All other medication in pneumonia patients takes the form of symptomatic treatment.

If constipation develops, fluid extract of cascara sagrada is given in half-ounce doses morning and evening as long as necessary. The effect of the cascara in these cases is enhanced if each dose is given with two ounces of sodium thiosulphate, in aqueous solution.

A troublesome diarrhoea is most easily subdued with hourly thirty-grain doses of sulphocarbolates compound

in conjunction with twenty drops of tincture of capsicum. The sulphocarbolates alone do not give the desired result.

Patients that do not eat can in many instances be brought into good appetite by a few doses of lemon juice. The juice from two or three lemons makes an average dose, and can be repeated a few times. It is to be given with a dose syringe. Others are tempted with fresh vegetables, of which carrots are usually the most acceptable. A good, seasoned bran mash containing a few finely sliced carrots will frequently be eaten when everything else is refused. Feed that is not promptly eaten by the patient should be removed from the manger. A pail of fresh water should be constantly within reach of the patient. The patient may be blanketed or not, depending upon the temperature of the stable, the presence of air currents, etc.

One feature of great importance in the successful handling of pneumonia cases is the supply of fresh air that the patient must have. Were I to choose between treating a case of equine pneumonia in a poorly ventilated stable and the open, even in extremely cold weather, I would choose the open. Ventilation of a stall is not always practicable, especially in the ordinary basement stable usually built on farms. In a stable such as this fresh air usually means cold air admitted in such a manner that it will chill the patient. Under these conditions better results will be obtained from the use of an out-house or shed to which fresh air has free access constantly. When this sort of an abode is resorted to the patient must be amply provided with blankets, a hood, and leg wrappings. The wrappings applied to the legs should consist of a layer of cotton under flannel bandages, loosely put on. Under all conditions the patient should be subjected to a daily application of curry-comb and brush, thoroughly manipulated.

Cases in which resolution is tardy can usually be hurried along by the administration of five or six doses of iodid of potassium, giving a dram at each dose, at intervals of four to six hours. This may "knock out" the patient's appetite; if it does, the iodid administration is to be stopped at once.

Cases complicated with pleural effusion of such amount as to produce dyspnea should be tapped. This operation can often be delayed until the patient is in fair condition to go through the ordeal. A quarter to a half grain of strychnin sulphate given hypodermically half an hour before the operation will fortify the patient against ensuing shock.

Cases complicated by the development of empyema do well under large doses of echinacea. If conditions are favorable for properly irrigating the pleural cavity after the contents have been drawn off it should be done, using either normal salt solution or a very mild solution of chinosol.

Coughs that remain after recovery from an attack of pneumonia are best treated with beechwood creosote in glycerin.

Cases that run a slow and indifferent convalescent course should receive potassium iodid and Fowler's solution of arsenic alternately.

Some cases that are slow in regaining their former vigor can be brought to prompt improvement by the injection of one large dose of anti-streptococcic serum.

In summing up the items of importance in the handling of patients afflicted with pneumonia, I would mention again:

1. Avoid anything and everything the effect of which is depressing. Do not allow yourself to be moved into using antipyretics, especially coal-tar products, in your efforts in controlling the fever. The fever subsides spon-

taneously if you follow the instructions already outlined. It would be a physiological impossibility for the patient to overcome the pathological condition attacking him without the presence of a rise in body temperature.

2. By good nursing, sanitation, hygiene and enticing feeds, endeavor to keep the patient cheerful. As long as he is cheerful he will eat; and as long as he is eating well, he is making good progress. In no other disease is the appetite such a good index of the patient's condition.

3. If it is not possible, or practicable, to arrange sufficient and proper ventilation in the stall, have the horse put out of doors. Fresh air is half the treatment.

4. Make it a point to observe your patient with especial care from the fifth to the eighth day of his attack. While many cases of pneumonia in horses come to resolution without marked crises, nearly as many do go through a genuine crisis. This is generally forecast by an additional rise in temperature, by increased respiratory difficulty, restlessness and capricious appetite. Occasionally there is a slight rigor. When these signs appear on the fifth to the eighth day, meet them with strychnin. Give small doses hypodermically every four or five hours if need be. If it is not convenient to administer hypodermically instruct the attendant so that he may give it per rectum.

5. Remember that a single dose of mixed bacterins given early in the attack will almost positively prevent empyema.

6. Begin the treatment of all cases with a good mustard plaster.

No great strides have been made in the treatment of pneumonia. Even in human medicine, where every opportunity and facility for improvement exists, the treatment of pneumonia is today practically the same as it was a generation or two ago. This is not because no

efforts have been made to find a different or a better treatment—in fact, the subject of the treatment of pneumonia has received fully as much study as any other. The disease itself is the only answer—*it can not be aborted*; it is self-limited. The nature of the tissue which is the seat of pneumonia is such that an inflammation once established within its cells can only terminate by resolution after certain stages (fully understood by all pathologists) have been passed through. All improvement in the treatment of pneumonia must therefore end, as a matter of course, with such innovations having to do with the prevention of complications and sequelæ.

Just as soon as the practitioner comes to look at his pneumonia cases from this angle he has only occasional losses. As long as he adheres to heroic treatments, endeavoring to abort the attacks or to bring them to a rapid termination, he experiences only occasional recoveries of his patients.

ACUTE YELLOW ATROPHY OF THE LIVER

This is a rare disease in general. In the neighborhood of smelters and paint works it occurs often from the ingestion of foliage soiled with poisonous fumes or smoke. As an accidental disease it may occur in any region at any time after an animal indulges an abnormal appetite on articles containing phosphorus or other active poisons. Also, as a sporadic disease, it may develop from unknown causes at any time in any locality. However, as has been pointed out, the disease is a rare one. In a life-time of practice one might see half a dozen cases.

From a pathological standpoint the disease is an acute fatty degeneration of the liver cells, which runs its course in a few days, and in almost every instance terminating in death.

The onset of an attack of acute yellow atrophy of the liver is always sudden. Usually the attack begins with a mild colic. The pains are not severe and do not remain long, an hour or two at the most. They are at once followed by the symptom complex about to be described.

First, with the disappearance of the colicky pains, there appears great depression. This is so marked as to resemble severe shock. The pulse is lost, and the temperature, which was raised several degrees during the time the colic persisted, begins to drop. In a few hours it may be several degrees subnormal.

Second. The visible mucous membranes are icteric. At first only a slightly yellow tinge is seen, later they become a dirty brown, or a greenish brown.

Third. An uncontrollable diarrhoea sets in. The evacuations are at first normally colored, later they become pale yellow, and finally have a gray tint. Before death occurs these evacuations assume a thin, watery cast and give off a very disagreeable odor. Evacuation follows

evacuation; the patient stands now in an exhausted, resigned attitude. Muscular tremors and perspiration appear, and soon recumbency and death. A horse in the grip of this disease is a pitiable sight to behold. The disease runs its course in from two to four days. During this time the patient will not refuse all feed, but from time to time may nibble some oats. These appear, undigested, in the evacuations within a few hours. The urine is very dark in color.

Nothing will check the course of the disease so far as is now known. In one case which occurred in my practice, I had ample opportunity and every facility and inducement to attempt a cure. Everything was useless. Until the etiology and the pathological changes which occur are better understood, we can expect to make but little impression on an attack with our treatment. The disease runs such a rapid and violent course that any form of handling, to be successful, "must hit the nail on the head." Until we can do this these cases will continue to be always fatal.

On account of the infrequent occurrence it may not be possible to solve the problem presented by this disease for a long time to come. Experimental handling of these cases would, therefore, be entirely ethical and permissible by veterinarians in attendance on animals so affected. Under our present form of treatment and understanding of this disease, death is always a foregone conclusion. At autopsy the liver is greatly shrunken or reduced in size and very friable.

JAUNDICE

Jaundice, or icterus, is the name given to the condition produced by the repression, or else by the reabsorption, of the biliary secretion. The condition results from repression of the biliary secretion in such affections of the liver as cirrhosis. It results from reabsorption of the bile when there is an obstruction in the bile duct, such as calculi, new-growths, catarrhal inflammation, etc.

Symptoms. These vary in severity with the amount of interference in the biliary output. When jaundice is fully established it is frequently a grave and stubborn condition. The horse eats little or not at all. There is an alternating rise and fall in the body temperature; slight exertion frequently produces attacks of tachycardia. The clinical picture may change several times a day. At certain hours the patient is apparently in fair health, while possibly at other hours of the day the symptoms again become marked. The visible mucous membranes, especially the conjunctiva, are of a cast from yellow to yellow-green. The color of the urine which the patient voids is either dark brown or greenish in color. The feces are at first slightly lighter than normal in color; later they become greyish. After the ingestion of food the animal may exhibit slight colicky pains. An attack of jaundice may disappear in a few days, or it may remain for several months, depending upon the etiology.

When the jaundice is clearing up there is in some cases a severe pruritis. The animal will mutilate itself in its attempts to scratch itching portions of the body, frequently resorting to the use of its teeth. No doubt some of the cases of self-mutilation that have been reported were due to jaundice. During the entire time that the

condition persists the affected animal is weak, listless, and incapable of performing its usual work.

The prognosis is favorable in cases due to catarrhal obstruction or calculi. It is unfavorable in all cases due to structural changes in the liver or its ducts.

Treatment. No matter in what form the condition appears, one of the most essential features in the treatment is rest. Even slight exertion has a tendency to delay recovery and aggravate the symptoms.

Because of the difficulty of differentiating the various forms clinically in the horse, all attacks of jaundice are given such treatment as would appear most appropriate in the catarrhal form. The nature of all other forms is such that, in the horse, their correction is at least impracticable if not impossible.

Repeated doses of epsom salts, of sodium phosphate, or of other saline laxatives, are given daily. These doses are to be just large enough to produce a mild laxative effect; purgation is rather more harmful than beneficial. The feed should be nutritious and easily digested. Stomachics and some form of internal antiseptic should be given together.

Some cases that resist the above line of treatment respond quickly to repeated doses of sodium bicarbonate and infusion of rhubarb. Small doses of essence of peppermint enhance the effect of the two latter agents.

Some cases that have resisted all other treatment will recover with the use of iodid of potash, phytolacca and colchicum.

When the pruritis incident to the convalescent period begins, the horse must be prevented from injuring itself. Alkaline body-washes may be used to lessen the pruritis. Usually, however, there is not much benefit derived from any sort of applications for this purpose.

The case may be considered on the road to recovery

when the urine clears up and the feces again assume a normal color. The icteric cast of the mucous membranes may persist for a long time after all other symptoms of the disease have disappeared.

LEUKEMIA

This disease has only very rarely been reported in horses, being, no doubt, frequently mistaken for other diseases having a similar general course. The actual diagnosis of leukemia in any form is only possible by laboratory methods.

Leukemia occurs in two forms; first, myelogenous leukemia, which affects chiefly the leukocytes; second, lymphatic leukemia, in which the lymphocytes are chiefly concerned. The etiology is unknown.

Symptoms. Leukemia affecting horses is said to occur always in a very gradual, insidious manner. There are at first only signs of general debility, lack of endurance and weakness. There may be a degree of pulmonary dyspnea. Later the mucous membranes assume a pale, almost white color. Heart lesions develop, edematous swellings appear in various regions, and the lymphatic glands throughout the body become very much enlarged. Splenic hypertrophy is always present. There may be general soreness in movement, said to be due to involvement of bone marrow.

When the disease has become well established there is a tendency toward hemorrhage, such as epistaxis. The blood which so issues is pale and thin. Death occurs from inanition, cachexia, or from internal hemorrhage. The course of the disease may extend over a number of years. The prognosis is unfavorable.

Treatment. Leukemia is only rarely treated as such because a diagnosis is not often made of the true condi-

tion. And, even when the condition is actually diagnosed, the treatment is wholly of a symptomatic character. Good food, light work, tonics such as arsenic and iron, and good hygienic surroundings accomplish all that can be expected. The termination is almost without exception fatal, and it is only possible to slightly delay this termination.

DIABETES INSIPIDUS

For some reason or other this has come to be a very rare pathological condition. In my opinion this reason can be found in the now prevalent custom of treating grain with formaldehyd to prevent mould. While the exact pathology of diabetes insipidus was not fully understood, it was quite generally held that it was the result of an infection with some form of mould taken in on the feed. The prevailing custom of treating grains with formaldehyd and the coincident decrease in the number of cases of diabetes insipidus would, to a certain extent, strengthen this hypothesis.

The predominating symptoms in this disease are great thirst and frequent urination. The affected animal suddenly develops a great thirst. It will drink from four to six times the normal quantity of water, and at every opportunity. If no drinking water can be reached the animal will drink anything liquid, such as sewer water, rain water, urine, etc. At the same time micturition is frequently performed, great quantities of urine being passed at each act. After the condition has existed for three or four days there is much straining and dribbling of urine at the end of each urinary act. The appetite now becomes capricious, and in another day or two the patient refuses all food, retaining only an unquenchable

thirst. Exhaustion, emaciation and death are next in order.

Diabetes insipidus must be differentiated from simple polyuria. The latter is a transient condition free from constitutional disturbances, and disappears spontaneously. Diabetes insipidus persists for weeks, slowly increases in severity, and frequently terminates in death if proper treatment is not instituted.

Treatment. Begin the treatment of a case of diabetes insipidus with the administration of a dram of resublimed iodine. Give this in a capsule with a little powdered gentian. With few exceptions this is all the medicine that is required.

The remainder of the treatment is concerned with limiting the supply of drinking water. Allow at first about twice the normal amount; gradually reduce this to normal and eventually to less than normal for a day. This controlling of the amount of drinking water imbibed by the patient must be done very gradually, reducing the quantity in proportion to the reduction of urinary excretion. With the administration of the iodine and sensible control of the drinking, even very marked cases come to a satisfactory termination within two or three days. If improvement does not show within twenty-four hours after the iodine has been administered the same dose may be repeated once.

When the appetite returns the grain supply should be fresh, so as to prevent reinfection in case the animal had been fed mouldy grain. While the iodine treatment and the control of the water supply promptly puts an end to the alarming symptoms, the patient usually remains in a weak, unthrifty condition for some time. Even when the attack has persisted only for a few days the horse is usually much the worse in condition at its termination. When an animal has gone through a marked

attack of diabetes insipidus, a long rest is usually required before the customary well being is reestablished.

DIABETES MELLITUS

Diabetes mellitus may be described best as the "sugar disease." It is characterized by an excess of grape sugar in the blood, which excess is excreted in the urine in its true state as grape sugar. Diabetes mellitus is much more common in horses than is generally held to be the fact. True, it is, comparatively speaking, a rare disease; but it appears frequently enough to make a knowledge of its manifestations essential.

The disease runs a slow, chronic course, lasting from a few months to several years. While spontaneous recovery is possible, the termination is nearly always fatal.

The pathology is not really understood. Pancreatic insufficiency is quite generally presumed to be a factor in the causation.

Symptoms. The first signs of diabetes mellitus are, what describes it best, a "lack of pep." The horse does not come up to his usual standard in condition and workability. Signs of fatigue are common at ordinary exertion. The animal perspires more easily than it should, and usually the client says that "his feed does not seem to do him any good," despite the fact that his appetite does not seem to be interfered with. The condition may remain unchanged for months. Tonics, or whatever else may be given, do no appreciable good to the animal. As the disease progresses, the horse loses in condition. The appetite remains good, and very often becomes almost ravenous. Thirst is almost constant; the horse accepts every opportunity to indulge this sense.

It is now noted that the animal is becoming "tender-

skinned''; wherever the harness rubs, abrasions occur. The points of the hips and the sides of the elbow, the sides of the carpus, hock and head are bruised from the slightest contact with parts of the stall in lying down and rising.

At this stage of the disease a very common manifestation is a suppurative coronitis, frequently affecting all four feet. We have not noticed in our patients the development of ocular lesions described by some authors. During the entire course of this disease there is polyuria. The degree of polyuria is not always equal; at times the urinary output only slightly exceeds the normal, while at other times great amounts of urine are passed every hour or two. The urine is clear in color and has usually a sweet-sour odor. If voided on wooden floors it leaves the soiled area covered with a whitewash-like film after evaporation. Most cases of diabetes mellitus do not come to an end in death as a result of the disease itself. In most cases the horse dies from complications which do not show a very clear connection with it. Cases that run to fatal termination as a result of the progress of the disease terminate in marasmus, decubitus and death.

Diagnosis is made positive by urinalysis, demonstrating abnormal presence of grape sugar.

Treatment. Up to the present time the results from treatments generally resorted to have been of no avail. The disease is apparently incurable. In human beings fine results have recently been reported with what is known as the starvation treatment. Having noticed that the sugar content of the urine decreased when abstinence was enforced, Allen began to treat his cases of diabetes in human beings by starving them for variable periods of time. The results were good. A modification of the starvation treatment might be tried in horses. Feed

rich in starches must be forbidden. Very light exercise in conjunction with the starvation gives better results than complete rest or idleness.

LUPINOSIS

Lupinosis occurs in sections where alfalfa is fed on pasture. The disease is quite common in the northwestern states. It results from the ingestion of lupine grasses containing lupinotoxin.

Symptoms. While there may be an initial constipation, as claimed by some writers, the first symptom in my cases has always been violent purgation. The evacuations occur very frequently and at last are wholly fluid. This purgation may continue for several days, at the end of which time the horse appears greatly emaciated. There is now a wobbly gait, lack of coördination behind, and the horse stands about listlessly. The eye has a peculiar, bright, glassy stare. When the disease has been in existence for three or four days the buccal mucous membrane becomes the seat of irregular ulcerated areas, varying in size from that of a pea to a twenty-five cent piece. They have a tendency to spread and enlarge, and stubbornly resist ordinary treatment. The horse retains some appetite throughout, but may not begin to eat normally as long as the mouth is the seat of extensive ulcerated areas. The animals consume immense quantities of water if permitted to do so. In some cases there are also ulcerated areas on the skin of the lips, head or neck.

In most cases edematous swellings appear in various parts of the body. The case may terminate in death in from a week to two weeks, during which time the purgation continues, and at the end of which time the horse is in a pitiable state of emaciation. Cases may recover

completely under treatment, but convalescence is very slow. In some instances a stubborn polyuria supervenes.

Symptoms that occur in some cases, but that are not seen in all cases, are icteric membranes and high temperature. In most cases the temperature is only elevated a few degrees Fahrenheit. Rare cases show some cerebral disturbance, of which they give evidence by standing about, grinding their teeth.

Treatment. The horse must be taken off pasture or, if in the stable, the lupine-content must be withheld from the ration. The symptom that gives the most concern in the greatest number of cases is the violent purgation. In the control of this I have had fine results from oil of eucalyptus. An ounce of oil of eucalyptus is given in capsule three times on the first day, twice the next day, and once a day for two days longer. The first dose or two is accompanied by a full dose of tincture of opium.

The ulcerated condition in the mouth yields promptly to paintings of pure tincture of benzoin. Should any single ulcer prove more stubborn it may be lightly cauterized with nitrate or silver, and the benzoin paintings then resumed.

Hypodermic administration of medicines in cases of this disease frequently results in abscess formation and sloughing at the point of injection. Although the greatest care be taken regarding antisepsis and although the injected substance may not be irritating, such abscesses will occur frequently.

Horses regain their former vitality very slowly after an attack of lupinosis. When the acute symptoms have been completely controlled the animal should be permitted to move about in an open lot or yard, and tonics may be administered. In some cases a recurrence of all the symptoms may take place, just when the horse seems

about to improve. The same treatment must then be repeated.

The use of oil of eucalyptus was adopted as the most satisfactory treatment after I had failed to accomplish much good with various other medicaments. I have seen remarkable benefit in very grave cases of lupinosis from a single dose, especially when combined with opium.

THROMBOTIC COLIC

Obstruction of the mesenteric arteries by a species of *Sclerostoma* is a recognized clinical phenomenon in the horse. Thrombosis resulting as a consequence of such obstructions produces attacks of colic that are termed thrombotic colics. Cases of colic of this type are only reported when the attack terminates fatally, when post-mortem examination reveals the thrombotic vessels.

That an obstruction in the mesenteric arteries will produce colicky pains is not a theory. Experiments performed on animals by ligating and limiting the blood supply in the mesentery have shown that two pathological conditions will result, one of which is immediate and the other remote. The immediate result occurs within an hour or two after the blood supply has been cut off, and consists of violent peristaltic movements in that portion of the intestine from which the blood supply has been shut off. This violent peristalsis will continue until:

1. The obstruction to the blood supply is removed; or
2. The regional vessels assume the work of the obstructed artery; or
3. The section of intestine is paralyzed.

Under 1, there occurs an attack of "spasmodic" colic of moderate duration.

Under 2, we see a prolonged siege of colicky pains and varying degrees of flatulence in the small intestines.

Under 3, we see first what has just been said of 2, plus the grave developments of enteritis and peritonitis, terminating usually in death.

The remote effect of obstruction in the mesenteric arteries is obliteration of the arteries and accompanying lack of tone in the section of gut formerly served by the obliterated artery. The result here, also, is colic. Despite the fact that the manner in which colic can result

from thrombosis of the mesenteric arteries is well understood, the diagnosis of such forms of colic is not easily made positive. Colic resulting from thrombotic mesenteric arteries is fairly characteristic in its semeiology, yet the diagnosis can be made only problematically in clinics. Thrombotic colics are recurrent, always.

Symptoms. There is practically only one form of colic resulting from thrombosis that can be diagnosed with *any* degree of positiveness, and that is the form which results under "1" mentioned above. A colic of this character comes on with no regard for digestive periods; it may come just before a feed, or it may come at midnight or any hour of the day. The symptoms hardly vary from those of a typical spasmodic colic, but there is always quite active peristaltic sounds to be heard in the small intestine. This active peristalsis in the absence of diarrhœa may be said to be practically diagnostic of this form of colic. If, in addition to this, there is a history of previous similar attacks, colics occurring at variable hours from no apparent cause (such as change of feed, etc.), the diagnosis is made sure.

This is the one form of colic in which exercise is justified as part of the treatment. The horse, by being ridden or driven, so raises its blood pressure that it may have the effect of disintegrating the thrombus, or of hastening the establishment of a collateral circulation. Either accomplishment terminates the attack of colic. This occurs in from a few minutes to a few hours.

Medicinal treatment includes morphin, or tincture of opium, and other anodyne agents. Cathartics are contra-indicated in this type of thrombotic colic.

When the obstruction is not removed promptly, or if collateral circulation does not become rapidly established in the neighboring vessels, the attack of colic is prolonged. At the end of four or five hours a degree of

flatulence has been added to the clinical picture, and there is now either a diarrhœa or frequent evacuations of feces of normal consistency. At times there is a trace of bloody mucus mixed with or covering these.

In treating the case now volatile oils may be administered internally, and anodyne or analgesic agents which are used should be such as do not constipate. The attack may yet terminate favorably at the end of a few hours, or it may hang on for several days.

If the thrombus does not yield, or if the regional vessels do not carry the load for the thrombotic vessel, the case becomes serious. We then get either intestinal paralysis, or enteritis. When paralysis of a section of the intestine occurs the symptoms suddenly change. The horse ceases to exhibit signs of acute pain, but gives evidence, on the other hand, of dull, drowsy uneasiness. Flatulence increases, and muscular tremors and twitchings occur. Still later there are staggering, high temperature, filiform pulse, and fetid evacuations. Death comes at the end of twelve to eighteen hours.

Treatment in this stage can only be symptomatic. Symptoms of enteritis are well known.

Cases of colic that occur from the remote effects of thrombosis (atonic sections of the small intestine) are usually prolonged, atypical, recurrent colics. Varying degrees of pain are exhibited by the patient, and there are at times some evidences of flatulence. The attacks are of short duration.

Attacks of colic occurring in horses repeatedly without cause are always to be suspected as being due to thrombosis of the mesenteric arteries.

The prognosis is doubtful in all cases of colic resulting from thrombosis in the mesenteric arteries. While a horse may survive attack after attack, it is impossible to forecast what the termination will be in any particular

attack. While the prognosis is always doubtful, it does not become grave or unfavorable until there is evidence that the intestinal disturbance has been transformed from a functional derangement to a structural pathologic process.

DISEASES OF THE HEART

Veterinarians, as a rule, do not find diseases of the heart of sufficient importance to give them much study. When a distinct heart lesion is encountered in horses there is really only one important point in the diagnosis, and that concerns the fact whether the lesion is functional or structural. A structural heart lesion in horses means, almost in every instance, that the horse has become unserviceable. A possible exception to this statement might be found in a case where a compensatory hypertrophy had occurred. From a practical standpoint it is a very reasonable statement to say that heart diseases in horses, accompanied by noteworthy organic lesions, are never worthy of treatment.

In the interests of the client the best practitioner under such conditions is the one who can be relied upon to appreciate this at the earliest possible moment, and to its fullest extent. In no other class of diseases is this so true as in diseases of the heart. While it is many times not a very difficult matter to so handle the case that the horse will apparently regain a fair semblance of health, it is almost a foregone conclusion that the symptoms will return as soon as the horse resumes its regular duties, if the heart substance has suffered structural damage. Luckily, heart diseases *per se* are quite rare in the horse, and they may almost be said to occur only as a sequel to some acute infectious disease.

Functional heart diseases are always transient in horses, and in most instances disappear without treatment. The evidences of organic heart lesions are chiefly:

1. Persistence of the symptoms.
2. Involvement of other organs in the symptom complex, especially of the kidneys.

3. Edematous swellings in the subcutaneous tissues of dependent parts.

4. Recurrence of the symptoms when the animal resumes work, although a cure may have been simulated by their disappearance after the initial sickness.

Whenever two or more of these features become identified with evidences of a heart lesion the disease may with certainty be classed as organic. The prognosis is then always unfavorable.

Organic heart disease occurring as a sequel to infectious diseases is not nearly so common as formerly; since the use of biologic therapeutics has been quite generally adopted in the treatment of infectious fevers, and the use of depressants is less popular, the heart is spared.

Although a few authors have given the consideration of diseases of the heart considerable space, it can be said without fear of contradiction that the average practitioner gives such discussions scant attention. This is not so because of lack of appreciation on the part of practitioners, but only because diseases of the heart are really of minor importance in the average practice. When a case does occur the practitioner usually finds little trouble in recognizing it, although in most instances no great effort is made along curative lines. The performance of even the most ordinary work expected of horses is of such a strenuous character that the results following the treatment of organic heart lesions usually fall short of the requirements. In veterinary practice it is oftentimes a case of "service or death;" sentiment plays a very small part in our branch of medicine and, possibly excepting canine and feline patients, the practitioner's results are nearly always judged on a commercial basis.

AZOTURIA

No one disease of horses has been the subject of so many theories, theoretical treatments and hypothetical suggestions as this one. It is doubtful whether, out of the thousands of veterinarians in practice in America, fifty could be found who agree to any extent on the pathology and treatment of azoturia.

Probably one reason for this is the great variety of forms in which azoturia attacks horses.

Azoturia in the city horse, for instance, differs considerably from the azoturia seen in the country horse. The city horse develops a more severe, acute and, usually, rapidly fatal form.

Cases seen in the country are of a milder type, and do not run such a rapid hyperacute course.

This, then, would explain to a certain extent the disagreement among veterinarians in regard to several points of importance concerning this disease.

The fact that azoturia presents different clinical pictures under different circumstances, environment, locality, feeding customs, breeds and individuals, further explains why veterinarians are not agreed on its features.

Lastly, all argument is explained away by the fact that no one has so far succeeded in demonstrating satisfactorily what azoturia really is. I refer, of course, to its identity from the standpoint of pathology. From the standpoint of the clinician, the diagnostician, the subject is quite clear. Assume that your horse has azoturia. Call in a hundred veterinarians; or two hundred, if you wish. Each and every one of them, without exception, will recognize the condition and make exactly the same diagnosis—azoturia!

But question them in regard to the pathology of azo-

turia! Probably not ten of them would hold the same belief.

And were you to get from each of these veterinarians a prescription for the treatment of this case of azoturia, you would no doubt have several dozen different treatments.

It does not take a very learned man to arrive at the conclusion that, granting the above is true, azoturia must, indeed, be a formidable foe, both of the veterinarian and his patient.

As every veterinarian is familiar with them it is unnecessary to discuss at any great length the symptoms and diagnosis of azoturia. I shall therefore confine my remarks to some of the vagaries of the disease, its probable pathology, and its treatment.

Briefly, azoturia is an acute disease of horses, characterized by great nervous excitement in its early stages when it occurs in a typical form, and terminating in complete prostration of the subject attacked. Secondary clinical phenomena are hematuria, or rather hemoglobinemia, delirium, and prolonged decubitus. Death is either sudden, coming at the end of a few days, or results after variable periods of time from complications or the effects of decubitus.

It attacks young, thriving horses as a rule, though occasionally aged horses are affected.

The disease is a cold weather disease, but odd cases are seen at all seasons of the year. The greatest number of cases come during the winter months, from November to May, and an attack most frequently follows a short period of idleness. From one to three days of idleness, on regular rations, is a predisposing factor. Longer periods of idleness are not usually followed by an attack of azoturia. The foregoing remarks have reference to typical cases of azoturia.

To record in detail the various atypical forms and changed clinical pictures of this disease would be the work of a lifetime. We shall, however, attempt a review of the more important features presented in atypical attacks, and shall begin the discussion by noting the differences between azoturia cases in city horses and in farm horses.

Azoturia as seen in horses by the city practitioner, is, as a rule, a more serious condition than that which the country practitioner sees in his patients. City horses develop azoturia nearly always in a typical form. The attack comes, is barely noted before the horse goes down, and death comes nearly as quickly, after a day or two.

The country horse develops an attack in a more leisurely manner. In over half the cases seen in the country the disease gives fair notice that it is on the way. There is, first, lagging, possibly some perspiration; then, if the animal is stopped, symptoms are seen which point towards acute indigestion. If properly handled, the condition stops here, and the horse is again in good health after the lapse of a few hours. Country horses that go down with azoturia have usually gone down because the driver lacked the good sense to stop the animal as soon as he noted that it was not well. But even cases of azoturia in the height of their development in country horses, when the animal is down, are milder in form than the same cases would be in city horses.

The explanation probably lies in the more concentrated feed upon which the city horse lives, and in the more strenuous existence he is forced to lead. Country azoturia cases that receive ordinary care and treatment while the animal is yet standing will make a complete recovery in a few hours ninety-eight times out of a hundred. The city practitioner does not find it so. Many of the cases to which he is called while the horse is yet on its feet go down in spite of the best care and treatment.

The cases he sees before the animal goes down are not many at the most.

Here we have the explanation for some of the beliefs in the treatment of this disease.

We can dispose of the discussion of the part which the city horse plays in this disease by saying that the attacks are nearly always typical, that they are more grave than in country horses, and that the mortality is much higher.

Aside from the less acute character of azoturia in country horses, it presents other vagaries. The country practitioner sees more atypical cases of this disease than typical.

Probably the most common cases are those in which the horse is standing up when the doctor arrives; he shows signs of abdominal pain, looks around towards his sides. He may be breathing a trifle fast; the nostrils are dilated. There is a slight trembling of some muscle groups, but so slight that unless the veterinarian suspects azoturia he will not notice it. Sometimes, but not very often, there is some swelling in the gluteal region. Now and then a case shows swelling in the shoulder muscles.

If the catheter is used, the urine that is withdrawn shows darker than normal; occasionally, in these cases, it is typical coffee colored, but more often it is just a trifle darker than normal.

Properly handled, most of these cases are again normal within a few hours, except that in many of them swelling appears after eight or ten hours in the gluteal region, and the urine is not yet clear, or is even of a more typical azoturia color than it was in the beginning. This proves the diagnosis.

Out of a hundred cases of the character described above, a single one may become progressively worse until complete prostration and a fully developed case is established. The remaining ninety-nine cases will make an uneventful recovery within a few hours' time.

Another atypical form of azoturia common in country practice is that form affecting only one hind limb.

In these there is apparently acute pain; the animal is very uneasy, may lie down, and again rises to its feet with considerable difficulty. There is marked fullness in the gluteal region of the affected limb and trembling of the muscles in the crural region. The limb does not seem able to support its share of the bodily weight, and the animal works itself into a state of extreme exhaustion in its efforts to stand normally. While a goodly number of these cases go down eventually, few of them succumb. A few days of judicious handling restores the animal to usefulness.

A form of azoturia is often seen in country horses affecting chiefly the front limbs. There is in these cases immense tumefaction of the shoulder region, and the brachial muscles. This swelling in many instances persists for several days, although all other evidence of the disease may disappear within a few hours after its initiation.

Of all the atypical forms of azoturia, the one which causes the practitioner the most anxiety is that form in which the predominating symptoms are of a nervous character. Although the animal is standing up and the usual azoturia symptoms are mild the horse appears to be suffering from extremely heightened nervous tension. Constant shifting about from one leg to the other is gone through; the animal repeatedly backs up or attempts to move forward; the head is kept going from side to side, or is jerked nervously upward and downward. In this form there is frequently a degree of trismus, making it almost impossible to administer medicaments orally. This form of the disease frequently assumes a very serious course. The constant moving about tends to aggravate

the condition, and not a few of these cases ultimately go down.

An atypical form of azoturia which is not so common is a form affecting isolated muscle groups. One crural group, or one brachial group only, may be involved, showing marked swelling and extreme hardness. The animal exhibits no other symptoms except the full, rapid, tense pulse, and a "dopey" or sluggish countenance. The urine, when drawn or voided, confirms the diagnosis of azoturia by its consistency and coffee-like color. These cases, while not severe or serious, occasionally prove stubborn under treatment.

For the purposes of identification and proper treatment we should classify the various forms of azoturia as follows:

First. Cases during the initial stage of which symptoms of digestive organs predominate.

Second. Cases during the initial stage of which muscular symptoms predominate.

Third. Cases during the initial stage of which nervous symptoms predominate.

A careful adjustment of this classification to any given case in its early stages and the administration of adjunctive treatment on the basis of this classification will do much towards reducing the hazard, or the tendency, towards an aggravation.

Azoturia in mares seems to run a more mild and favorable course than it does in geldings.*

* The reader is reminded that the conclusions and averages arrived at in this thesis are not only those gained by the author in his own experience, but they cover the reports and opinions of hundreds of veterinarians obtained during a period of four or five years from all parts of the United States and Canada, by the author in his capacity of Veterinary Director for the Azolysin Company, manufacturers of the Azolysin treatment for azoturia.

The classification just made is only applicable in the early stages or in the mild cases that do not go down. Once the patient is permanently prone ("down" expresses it better than anything), we have azoturia *per se*, and no classification of predominating symptoms is required.

We now arrive at the discussion of that part of the subject of azoturia which, aside from the treatment of the disease, is of the greatest interest to the practitioner, namely, its pathology.

In the beginning of this chapter I stated that so far no one had satisfactorily demonstrated just what azoturia really is from a pathological standpoint. I now desire to modify this statement in so far as my own satisfaction is concerned. I am thoroughly satisfied that the theory which I here present is rational, sane and practical.

This theory was published in the October, 1912, issue of the *American Journal of Veterinary Medicine* under the signature of the writer and Dr. R. W. McCracken, a physician. As far as we have been able to learn the theory, which we named "the mechanical theory of azoturia," is entirely original with us, and up to the present moment has not been successfully challenged or exploded. Upon this theory was based the "azolysin treatment," a treatment also original with us, and one which probably has more recoveries to its credit than all other forms of treatment combined. In my opinion the theory that has the least ground for existence is the theory based on a toxemia. How anyone can believe that a toxemia *of a nature so active* that it can, in a few minutes, wholly incapacitate a horse otherwise in the "pink" of condition, *and that will suddenly cease* its action when the horse is properly handled (as we have frequently seen in cases quite well developed, with no direct treatment aimed at a

toxic poison, often nothing more than absolute quiet) can exist is beyond my comprehension.

I can appreciate a form of toxemia violent and active enough to bring on an attack of azoturia; but my entire professional judgment rebels against the acceptance of the second factor, namely, the spontaneous cessation of activity of a toxemia, or a toxin, so active. We *cannot* believe in the theory of a toxemia as the excitant in the disease if we have thoroughly mastered the fundamentals of certain branches in the study of veterinary science! And still less can we accept this theory when we fully understand the disease, and when we endeavor to make the theory fit the condition as we know it.

The "mechanical theory" elaborated by us has not been born in idle speculation. It is the result of a careful weeding out of other theories, and diligent study of azoturia from a clinical standpoint. The theory is the result of the interlocking understanding of a theory-forming, finely-tuned physician's brain and the more materialistic, hard-headed, veterinary judgment.

I give it here as it appeared in the issue of the *Journal* already mentioned.

"Our theory is, and our success with the treatment based on this theory proves its soundness, that the condition is a mechanical one entirely, if we may use the word in this sense. We mean by this that toxins, enzymes, bacteria, ferments or extraneous forces of any nature whatsoever have no hand in the production of this disease.

"The theory which nearly every veterinarian with whom we discussed this suggested as the most plausible was that azoturia is a toxemia. In the summing up given below we make special mention of this to show how the symptoms in certain stages of this disease absolutely exclude toxemia as a cause of azoturia. To come to the

point, we believe that the condition in azoturia is first a viscosity of the blood; later, if the condition does not abate, genuine clotting or thrombosis in the vessels of the parts involved. Death occurs either early as the result of embolism, or late from decubitus and its accompaniments. Carefully follow the form below, in which we work out the theory.

“1. A horse at rest a number of days on full rations.

“2. A modified atony of the muscular coats of the vessels results, accompanied by a hyper-nitrogenous condition of the blood and internal secretions.

“3. In adverse ratio to this atony is the accelerated tone of nervous elements, causing the horse to ‘feel good,’ as it is commonly expressed.

“4. When this horse is put to work he is keyed up to the highest pitch as regards volition; in other words, he has ambition forced upon him by the heightened tone of his nervous system. (Here could be inserted the reason why the disease does not occur or is so rare in summer.)

“5. Responding to this stimulation the cardiac action as a result of a few minutes’ exercise assumes the force which it would, under normal conditions, attain only at the end of the most strenuous exertion, at running or severe pulling in harness. This does not produce objective tachycardia because of the flaccid condition of the muscular apparatus of the vessels.

“6. From this atony or flaccidity the vessels in the extremities are the last to recover; in fact, if the horse is not stopped at once they (the vessels) do not recover. Gravity plays a slight part here.

“7. Soon there is a real blocking of the circulation in these parts.

“8. The large muscles in the region attempt to assist the vasoconstrictors; they contract in a firm spasm

around the vessels, thus bringing about the swelling in the gluteal or the brachial region.

“9. From viscosity to thrombosis is a short step. The patient goes down, and if thrombosis has occurred and the clot is not fixed, the patient is dead in a few days from embolism, dying with symptoms of apoplexy.

“10. When the condition is less severe, halting at viscosity, the patient recovers or lingers along and finally dies from the various results of prolonged decubitus. (A good example of a very mild case of azoturia we experience when we sit a long time in one position and a leg or an arm ‘goes to sleep.’)

“Theoretical proof of our theory and proof of the absolute falsity of the theory of a toxemia lies in the following:

“If this horse is exercised very mildly and then allowed to rest for a half hour before he is put to work, thus allowing the muscular tone of the vessels to regenerate itself preparatory to prolonged exertion, the disease does not occur.

“We will go still farther and say the horse, instead of being first exercised, is at once put to work and is attacked by the disease, but the driver stops him as soon as he sees the symptoms coming on. We all know that this horse will not go down and will recover with no further treatment than absolute rest. This, because it gives the circulatory muscular apparatus an opportunity to revive and fortify itself before the vessels are pumped so full of blood that absolute, or practically absolute, stasis occurs.”

The greater portion of this theory is amply substantiated by the clinical manifestations of the disease. This is more than can be said of any other theory applied to it in the past. Parts of it could be a little finer drawn, and still remain sound; on the whole, it meets with the

approval of the general practitioner more readily than other theories.

The fact that the treatment which was based upon it has proved remarkably successful is the best evidence that this theory has merit. Accepting this theory as our basis we searched about for an agent that would prevent coagulation and that would, if it were possible, bring about a resolution of blood already coagulated. After much digging in textbooks on physiology, and after a series of experiments, we chose oxalic acid, believing that after ingestion it would rapidly form oxalates of potassium in sufficient amount to serve our purpose. The dose which we found most satisfactory for repetition was four grains.

Oxalic acid is an active agent, and while this dose may appear small, it is sufficient for all practical ends when repeated hourly.

This, then, is the active principle of azolysin—oxalic acid.

For business motives several other ingredients were added to azolysin while it was on the market as a secret preparation.

From reports gathered over a period of four or five years we have evidence that this oxalic acid treatment will save over eighty per cent of the bad form—cases in which the horse is down. Veterinarians who used azolysin, as made by the Azolysin Company, did not use the treatment in mild cases; it was too expensive. For this reason I do not hesitate to say that the percentage of recoveries will be over ninety per cent when the treatment is used as a routine treatment.

While oxalic acid alone will save many cases, I would recommend the use of symptomatic treatment in conjunction.

Cases showing a predominating nervous trend should

have bromides, gelsemium or chloral. Dr. E. L. Quitman advises lobelin for the nervous symptoms. Cases showing symptoms of digestive disturbance should have salicylic acid, carminatives, and evacuants.

Cases with immense swelling of certain muscle groups should have massage. It is well to start the treatment of all severe cases with a purgative. Water should be given in abundance, drenching the patient with several quarts every few hours if he refuses to drink it freely.

The oxalic acid should be administered in four-grain doses every hour until seven or eight doses have been given. It must be given highly diluted with water to prevent any local action on the mucous membranes. It is not caustic, but has a softening or degenerative effect in concentrated solutions. After seven or eight hourly exhibitions, it should be given every two or three hours. In exceptionally grave cases the first dose or two may be doubled. The catheter may be used for the purpose of emptying the bladder in the early stages. Later it is neither necessary nor beneficial.

Cases which are down should not be molested or urged to get up before the lapse of at least eighteen hours, unless they make voluntary attempts to arise. In raising a horse, down with azoturia, two good men lifting at the tail can accomplish all the aid needed. If the animal is not able to get up with this assistance its condition has not yet improved sufficiently to make the standing position desirable. A few strokes with a whip may be required in some cases to induce the animal to make a strong attempt to arise.

The use of slings is not recommended. The practitioner who uses slings for the purpose of raising up horses, down with azoturia, should lose his right to practice; he understands neither the disease nor the horse.

I have personally seen a number of deaths directly attributable to the use of slings in this disease.

If the patient remains down for several hours or longer he should be turned over every two or three hours. Grain should be fed sparingly, if at all.

All cases terminating in recovery should have a course of diuretic and alterative treatment extending over a short period of time, say four or five days. Local or regional applications in the form of hot packs may be beneficial in cases showing great muscle swelling. Warm quarters and quiet surroundings aid all forms of treatment.

In concluding the discussion of the treatment of azoturia I wish to remark that I do not wish to give the impression that the oxalic acid treatment is perfect. As one eastern veterinarian, who has used the treatment for four years in a large practice, says: "We get better results with this treatment than with any other, but it is a long way from being a specific."

Neither do I wish to give the impression that this is the best treatment that we will ever find for this disease. But I do believe that any improvement in the treatment must come along the lines of the "mechanical theory" of the pathology of the disease, or of a modification of this theory. Possibly the mechanical theory is only on the verge of truth and soundness. However, it is the most reasonable, the most sane and sound theory yet expounded for azoturia; and the fact that it has bred a treatment of such merit as the oxalic acid, or "azolysin" treatment, is good enough evidence to satisfy the general practitioner.

Possibly, at some time in the near future some laboratory expert will give us an agent that will more satisfactorily perform the work which we now rely upon oxalic acid to do. Someone may even find a means of

isolating and making synthetically the exact agent required. Until this happens, however, most of us will rely on oxalic acid for effect in severe cases of azoturia. A factor very much in favor of the oxalic acid treatment is its inexpensiveness. Another is its simplicity, and the promptness with which it accomplishes results in the great majority of cases. In this regard it is like any other treatment; the better one becomes acquainted with the treatment, the better will be the results.

An important factor in the successful treatment of azoturia with oxalic acid lies in beginning the treatment as early in the attack as possible. If the horse has been down more than twelve hours the treatment cannot accomplish much.

Prognosis. I can point to no particular clinical signs which can be relied upon to forecast the termination in well-marked cases of azoturia. Oftentimes the case which appears to be most violent in character makes a rapid, uneventful recovery. On the other hand, cases are frequently seen which, in the beginning of the attack, appear mild and ordinary, and yet terminate fatally.

The color of the urine cannot be relied upon as a prognosticating factor. With the oxalic acid treatment the prognosis is very unfavorable if the horse does not get up within forty-eight hours after the treatment is begun. Cases in which marked delirium persists in spite of treatment are usually fatal cases.

Cases that remain down longer than forty-eight hours do not usually make a complete recovery; most of them suffer later from one or more of the sequelæ common to this affection.

Sequelæ. Attacks of azoturia, mild in character, during which the horse remains on its feet, rarely leave after-effects of any consequence. Now and then, after a mild attack, a certain stiffness or awkwardness remains in the

members affected. This usually passes off in a short time without treatment of any kind.

In grave attacks, during which the horse has been down for a number of days, the most common after-effects are bed sores. These come on the points of the hips and other prominent places. Some of these sores are very resistant to treatment, and they may leave permanent scars. They are commonly treated with antiseptics and astringent lotions, healing powders or ointments. In obstinate cases an occasional swabbing with a caustic solution of silver nitrate will hasten healing. Those having centers of dead or gangrenous tissue should first be curetted.

Another common sequel of azoturia takes the form of muscular atrophy. This sequel is sometimes seen after cases that were not severe by any means. The degree of atrophy varies from a slight flattening of the muscles to an almost complete disappearance of the muscle involved. Most commonly it is the gluteal region or the crural group; sometimes both. This atrophy is very stubborn under treatment and often requires several months for its correction. I have seen no particular or noteworthy benefit from any form of treatment. Now and then a certain case will respond marvelously under one form of handling; when another case is given the same treatment nothing whatever is seen of a beneficial result.

The usual treatment for this sequel consists of setons, blisters, massage, injections of strychnin or of irritant agents, tonics, alteratives, and occasionally actual cautery.

In my experience one is as good as another. I have seen a few cases in which this atrophy remained permanently. After several weeks of hospital treatment the animals were turned out to grass for several months. On their return to the stable at the end of this period not a

particle of improvement had been made. After another month or so of treatment the animals were in each case destroyed.

Lack of coördination in the posterior extremities is another rather frequent after-effect of azoturia. This ranges from a slight awkwardness in turning or backing, to a ridiculous wabbling in all gaits. It makes its appearance from a few weeks to a month after recovery from the attack of azoturia.

Prolonged courses of potassium iodid treatment will effect a cure in a good many of these cases. A somewhat rare, but noteworthy, sequel to azoturia is a relapse. After the animal has apparently recovered from an attack, another attack is ushered in. As I have already remarked, this is a rare occurrence; it has happened once or twice in my practice, and other practitioners have reported it also.

A few years ago I had a patient that developed an attack of azoturia several times each year. This animal, a mare, was down for two or three days on several occasions with a well-marked case of azoturia, and on at least one occasion her condition was very grave. She made a complete recovery after at least four attacks to my own knowledge. She was then sold to a farmer a few miles away, and had no more attacks for at least two years, after which I lost sight of her. In one instance this mare apparently had recovered entirely from an attack. She got up, was normal in every respect for nearly twenty-four hours, and then suddenly went down again. She remained down for nearly two days with all symptoms of azoturia well marked. At the end of the second day she got up and made a rapid recovery. None of the attacks from which this mare suffered were followed by after-effects of any kind.

It is my opinion that the treatment used in combating

an attack of azoturia has considerable bearing on whether sequelæ develop or not. Since I have been using the oxalic acid treatment I have seen very few cases that developed serious or permanent sequelæ. The recovery under this treatment is as nearly complete as could be desired.

TRANSIENT CRURAL PARALYSIS

In this, we have a condition which is very confusing to the practitioner who has not had previous acquaintance with it.

It occurs almost always in young horses, usually during the fourth or fifth year. It makes its appearance precipitately, and as a rule disappears within a number of hours after its development. It is important chiefly from the viewpoint of prognosis.

The affected limb is held in a limp, flaccid attitude; the hock is extended to the limit, the ankle fluxed completely. In moving about the animal drags the leg loosely, the anterior face of the ankle touching the ground. The paralysis apparently includes the motor nerves only; sensation is not impaired in the parts.

The condition must be differentiated from the following affections:

1. Cramp.
2. Rupture of the flexor metatarsi.
3. True crural paralysis.

It is differentiated from cramp by the loose, flaccid condition of the muscles. A sudden shock, such as a stroke with a whip, will dispel cramp. Not so with this condition. In cramp, the entire limb is held rigid.

From rupture of the flexor metatarsi it is differentiated by the absence of trauma, lack of swelling and soreness on palpation in the region of the tendon of the flexor.

True crural paralysis differs from the condition under discussion in that it makes its appearance gradually, or else follows injuries sustained in falling, in being cast, or from other noted accidents. A history of this nature can usually be obtained from owners or attendants in cases of true crural paralysis. A history that now and then goes with a case of transient crural paralysis is that the

patient has for some time been a bit "stringy." Evidence of stringhalt has especially been noted in horses when they are first started in harness in the morning.

As has already been said, the condition is important only from the standpoint of prognosis. When presented for the veterinarian's attention, a case of transient crural paralysis appears quite formidable. From the appearance of the patient a grave prognosis seems necessary to the inexperienced. Rarely does this condition persist longer than four or five hours. Even without treatment of any kind most cases terminate favorably in a few hours. Recovery is spontaneous, almost instantaneous, and complete. However, recurrence is common. Some horses have an attack of transient crural paralysis every few weeks or months. In some instances each attack is more marked and persists for a longer period of time than previous attacks. The stringhalt symptom is also aggravated.

The repetition of attacks abates with maturity. After the sixth year few cases are seen. In horses developing repeated attacks of this affection treatment aimed at a constitutional disturbance is valuable. Fowler's solution of arsenic, iron, or iodides may be tried in turn. In my experience iodides gave the best results. Stimulating liniments, blisters, and massage may be used locally.

The pathology of this condition is not known. No doubt it is due to a functional aberration in one of the body glands concerned in controlling growth. Possibly the thyroid gland is involved. From the salutary effect obtained from the administration of potassium iodid in recurrent attacks one may suspect the thyroid gland is affected in these cases.

LYMPHANGITIS

We will discuss in this article that form of lymphangitis which, despite modern bacteriologic thought, is best described as an *idiopathic* lymphangitis. We choose to confine our discussion to this form of the disease because, in our opinion, all other forms are preceded by this one. Further, this is the most common form of lymphangitis. We do not agree with those over-enthusiastic bacteriologists who ascribe all attacks of lymphangitis in horses to infection following an injury. In a practice covering a considerable period of time, we have not been able to adopt this view; the clinical evidence has not been sufficiently ample to demonstrate the correctness of such a theory. On the other hand, so markedly contrary to this theory is the clinical evidence that we consider the old name of "Monday morning sickness" most appropriate.

Clinically, lymphangitis in horses is, in most instances, a functional disturbance, primarily innocent of bacteriologic taint. When we refer to lymphangitis in veterinary subjects, we mean, as a rule, that typical form of lymphangitis affecting a pelvic limb. While lymphangitis, or inflammatory processes in lymphatic vessels, is possible in any part of the body, the term lymphangitis in veterinary literature has come to stand almost wholly for that form affecting a pelvic limb. In rare instances lymphangitis, or a generalized congestion and inflammation of the lymphatics, has been reported as occurring in a pectoral limb.

While lymphangitis is somewhat more common in young horses, it occurs with almost equal frequency in mature or aged horses that are in thriving condition. Unthrifty, weakly animals are not often affected by this disease in any of its forms. Animals heavily fed on grain with an under-proportioned share of work are predis-

posed to lymphangitis to a lesser extent than heavily fed horses that are also heavily worked. An abrupt cessation of activity in the latter, occasional holidays, may at any time precipitate an attack of lymphangitis.

Symptoms. Lymphangitis in most cases makes its appearance without particular premonitory signs. In some cases there may be a rigor which ushers in the attack. Most frequently, however, the disease makes its appearance with speed minus stormy symptoms. Following upon a day or two of rest the horse is found some morning to have a considerable enlargement of one limb. The swelling extends from the groin to the hoof, is painful and hot, and has a line of demarcation near the body. The animal refuses its feed and has a body temperature of from 103° F. to 106° F. There is usually some constipation.

The swelling is quite evenly firm, except near the line of demarcation, where it is more "doughy"; in the internal aspect of the thigh this "doughy" consistency of the swelling is most marked. The swelling above the hoof may be of such proportions that the coronary region appears to extend anteriorly over the hoof for several inches.

As a result of infection with pyogenic bacteria, abscess formation may occur at various points on the affected limb. Mild cases of lymphangitis recover spontaneously. In many cases, however, some enlargement remains in the limb permanently. Well marked cases require careful and judicious treatment. Recurrent attacks of lymphangitis are not rare. One patient in our practice has had about six attacks within two or three years. Repeated attacks of lymphangitis may ultimately leave the affected limb permanently enlarged.

Treatment. The treatment of lymphangitis, in the idiopathic form, should include:

1. Depleting internal medication.
2. Local or regional applications.
3. Prophylactic bacterin therapy.
4. Dietary regulations.

1. Despite recent improvement in the line of hypodermatic medication, for cathartic effect we have found nothing that has a more satisfying effect in cases of lymphangitis than aloes. From an ounce to an ounce and a half in bolus form, is the ideal cathartic in these cases. For the best effect the bolus should be hard. Powdered aloes given loosely in capsule is not satisfactory; neither have we found the effect of aloin nearly so satisfactory as that of aloes. As soon as the cathartic effect of the aloes is established we begin the administration of small doses of salol or sulpho-carbolates compound. This administration is kept up until the case assumes a favorable aspect, usually four days. If the appetite is entirely absent, an occasional dose of bitter tonics may be given.

2. Local applications consist of prolonged fomentation, at least twenty minutes out of each hour. The water must be as hot as the patient will bear it. These fomentations are followed by the local application of solutions of lead acetate, or other astringents.

3. Every case of lymphangitis should receive a prophylactic dose of mixed bacterins to forestall abscess formation.

4. Dietary measures consist of total abstinence from grain. Hay may be eaten freely. Water may be given *ad libitum*.

During the first four or five days the patient must not be moved; absolute rest is imperative. At the end of this time, if considerable improvement in the general condition is noted, the animal may take a few steps. The amount of exercise is to be gradually increased, and at the end of a week the horse may be turned loose, or put to

light work. At the end of the first four or five days, if the swelling is not receding satisfactorily the fomentations and astringents are to be discontinued and a solution of potassium dichromate is painted over the entire limb once or twice each day. In recurrent attacks inunctions of mercurial ointment once daily will dissipate the swelling more promptly than anything else.

Cases of lymphangitis that prove obstinate under treatment can usually be brought to a satisfactory termination if iodid of potassium is administered in full dosage for several days. Abscesses which complicate an attack of lymphangitis are treated by incision and swabbing of their cavities with tincture of iodine. Abscess formation near the foot is best treated by soaking in antiseptic solutions, immersing the entire lower extremity in a pail full of the solution. Exuberant granulations in abscess cavities are removed by actual cauterization.

EPIZOOTIC LYMPHANGITIS

This term is slightly misleading in a veterinary sense. The veterinarian's comprehension of a lymphangitis is classically portrayed in the clinical picture of the idiopathic form of lymphangitis discussed in the preceding chapter.

Epizootic lymphangitis is confined chiefly to the superficial lymphatics, and may make its appearance not only in an extremity, but in any portion of the body surface. It is caused by infection with a yeast mold. It is a very rare disease in this country, having been reported as prevailing in a few states only. Because of the similarity of some of its manifestations to cutaneous glanders, some of the cases reported may have been the latter, instead of the epizootic form of lymphangitis.

Epizootic lymphangitis is a sub-acute and, at times, a chronic, affection. It is presumed to be transmissible. It is more probable, however, that a series of cases in a given district are the result of a common or regional etiologic factor.

The symptoms are chiefly confined to the local manifestations. In protracted attacks the affected animal may show some systematic disturbance, such as pyrexia, inappetence, emaciation. The duration of an attack varies from one month in mild cases to seven or eight months in severe cases. The infection almost invariably occurs in an abrasion or superficial wound. As a result, healing of the wound is retarded, and the establishment of an ulcerated area takes place. A thickening immediately around this ulcer slowly forms, increasing in size to that of a goose egg in some cases. The swelling is not very painful, and from it, in many directions, are cord-like radiations. These are enlarged subcutaneous lymphatic vessels. The swelling at the original site of infection eventually breaks

down, emitting a rich yellow, tenacious pus. New swellings form along the course of the radiating lymphatic vessels. These also break down and discharge later. Healing may occur in some of these broken down areas. At times this healing is permanent, but in many instances new abscesses form on the site of the old ones. Those that heal leave little or no scar. In this manner the disease may continue for months, either confining its activity to a restricted area, or else involving a considerable portion of the integument. In very mild cases the manifestations are not so pronounced, limiting themselves to pustular, pimply areas that resist treatment indefinitely.

Epizootic lymphangitis must be differentiated from:

1. Cutaneous glanders.
2. Botryomycosis.

The absence of other symptoms of glanders is established by clinical examination. If the result of such examination is not decisive, malleinization is essential. The absence of the characteristic granules in the active area differentiates the condition from botryomycosis.

Treatment. The treatment of this disease has not proved very satisfactory. Enucleation of affected lymphatic glands and the thorough dissection of involved lymphatic vessels may terminate the condition, if performed early enough. Later cauterization and persistent antiseptic treatment, both internally and externally, must be used. All cases should be handled as an infectious disease, and proper precautionary measures should be taken to prevent the transmission of the disease to other horses. Infection is by direct contact only, from the use of currycombs, brushes, and other agents.

FOOT SCAB

Foot scab in horses is a localized form of mange caused by the *Dermatocoptes communis*, and is important for the practicing veterinarian because it must be differentiated from scratches. Foot scab is most prevalent during the winter months and affects the region that is commonly the seat of scratches, the posterior face and sides of the pastern region.

Before any visible lesion is present there is considerable itching, which the horse manifests by stamping and gnawing or licking the parts. Shortly after this the skin in the region desquamates and becomes scurfy and raw, followed by the formation of large crusts. The skin becomes thickened, and transverse wrinkles or folds are formed. Eventually these assume a tough, horny character and remain even after the disease has been eradicated.

Treatment. In the itching stage one or two applications of mercurial ointment, well rubbed in, will usually put an end to the trouble. Most cases, however, do not come to the veterinarian for treatment until actual skin lesions are present. These respond quite promptly to treatment with applications of coal-tar antiseptics or sulphur ointment. Transverse, horny wrinkles or folds can be slowly removed with a ten per cent ointment of salicylic acid, which should be applied daily.

When it is not possible to treat the horse as above described, a complete cure can often be obtained by smearing the affected pastern regions liberally with pine tar. One application will accomplish the desired result in the early stages of the disease. When the disease has become chronic and the skin is marked with tough wrinkles and folds more careful treatment is required. Foot scab is transmissible through the agency of brushes, currycombs, cloths, ankleboots and attendants' hands.

FOOT-ROT OF COLTS

This disease has also been described as canker of the foot. As it differs markedly from true canker of the foot I prefer the name "foot-rot," which not only differentiates the condition from true canker of the foot but also describes the condition more accurately.

Foot-rot affects colts usually from the first to the fourth year of life. I have seen only one case in the mature horse. While I have not been able to disprove the infectious nature of the condition, I do not believe that it is infectious. It appears nearly always in isolated cases and shows no tendency towards transmissibility. Apparently, it is a pathological process incident to colthood. Cases have been seen under sanitary as well as unsanitary stabling conditions.

Symptoms. There is at first a slight "soreness" in movement of the affected foot. This may be any one of the four, although the disease shows a slight preference for the posterior extremities. Occasionally all four feet may be involved. About the time that attention is attracted to this soreness in movement, there is also noted a slight thickening in the region of the coronet. This thickening extends, in some cases, up to, and may even include, the ankle. If the swollen member is now carefully examined it will be discovered that the sole of the foot is the seat of a degenerating process. There is an area, varying in size from an inch in diameter to two inches, in which the horn has degenerated into a black, mushy substance. If this area is curetted thoroughly an uneven layer of raw, easily-bleeding, granulating tissue is found below. The degenerating process slowly spreads, so that at the end of one or two months the entire sole of the foot may disappear, leaving the black, mushy substance already described, and under this an expanse of

granulating soft tissue. Aside from the edematous swelling in the coronary and pastern regions no other symptoms are produced.

If the condition is allowed to continue without appropriate treatment a "drop-sole" usually results, making the horse unsound.

Treatment. With a hoof knife and a curette the bottom of the foot must be thoroughly cleaned up, removing all degenerated parts of horn. The entire area is then to be wiped over a number of times with sponges of cotton so that it becomes perfectly dry. By means of a small brush or cotton swabs the whole of the diseased area is now thoroughly painted over with equal parts of formaldehyd and glycerin. The colt is then to be confined in a dry stall. No bandages or dressings need to be applied.

After three or four days the foot should be reëxamined. If there are any new areas of degeneration these must be pared out, and the entire diseased section of the foot is then treated again with formaldehyd and glycerin. This performance is repeated every three or four days, and must be continued until there is a good growth of new horn beginning to appear. In mild cases one or two treatments may suffice, while in more extensive lesions seven or eight treatments may be required.

An important feature in the handling of a case of foot-rot in a colt is that of keeping the foot as dry as possible. No washing of any kind must be employed and the colt should stand in a perfectly dry stall until the disease is under control. All the cleaning that is required must be done with the hoof knife, curette, and wads of dry cotton or oakum. With unruly colts it may become a difficult matter to do this thoroughly unless they are first cast. If there are good reasons why the colt should not be cast in any individual case, the paring and cleaning part of the treatment can be omitted, and the formaldehyd and

glycerin applied only. Recovery will, however, be considerably retarded under these conditions. The thorough removal of all of the diseased horn before the medicine is applied is a very important part of the treatment.

In some cases that have existed untreated for a considerable period of time, some swelling may remain in the coronary and pastern regions after all other signs of foot-rot have disappeared. This swelling, while it appears to be nothing more than an edematous swelling, is, at times, very persistent and stubborn under treatment. A cooling astringent lotion should at first be prescribed and used for some time. If no improvement is shown after it has been used for ten days or two weeks, put the colt on a course of potassium iodid and apply tincture of iodine locally. The colt should have moderate exercise during the time it is being treated for the removal of this swelling.

UMBILICAL HERNIA

Umbilical hernia in colts should not be considered as a surgical condition before the sixth month. This form of rupture is amenable to medical treatment in probably 90 per cent of all cases if treatment is begun not later than the second month of life.

An umbilical hernia that gives no evidence of becoming reduced, or, on the other hand, that increases in size by the time the colt is two months old, should be treated. Umbilical hernia that decreases in size can be left alone until the colt nears the age of six months. Many cases of navel hernia disappear spontaneously before the sixth month. In all cases a cure should be attempted by medicinal agents before surgical intervention is thought of. The risk with medical treatment is practically nil, and if the result should not be completely satisfactory, the colt is still a good subject for the surgical procedure. I repeat that, if treatment is begun not later than at two months of age, a cure results in 90 per cent of all cases. About one in ten will refuse to yield to such measures as I am about to outline, and appropriate surgical interference will be required for the correction of the abnormality in these.

Treatment. Very small hernias will disappear with the use of ordinary blisters. The treatment that I have found the most cleanly, and that accomplishes uniformly satisfactory results with reasonable dispatch, consists of the application of fuming nitric acid. To make the application properly, the colt must be laid on his back. Before the acid is applied, a good sized area around the hernial enlargement, which area should not get nearer than one inch to the outer circumference of the enlargement, is liberally smeared with vaseline. Lard should not be used; it is too light in body and does not remain adherent

long enough. The acid is then painted with moderate generosity over the enlargement itself. The colt is allowed to arise immediately. There is now and then a colt that will give slight evidence of pain from the application, but this is rare.

Within a day there appears a diffuse swelling in and around the hernial enlargement. This remains for variable periods of time, usually a few weeks, and when it disappears it is noted that the hernia is also gone. There is usually a necrotic area in the skin where the acid has been applied. This area requires no attention, and as the location is always in a place where it can not be noticed, the slight scar that remains is no reason for objection to the treatment. One application is all that is required. Cases that do not yield to this application will not yield to any other form of medicinal treatment that I know of. This treatment is especially to be remembered because of its

1. Ease of application.	3. Safety.
2. Practicability.	4. Regularity of cure.

Hernias that have not entirely disappeared when the swelling caused by the nitric acid is gone, should not be turned over to the surgeon with too much haste. Some that at first apparently have not been cured will yet slowly reduce in size during the following weeks. If, then, at the end of another two weeks no improvement is evident, surgical measures are in order. Such cases that still show some degree of hernia when the swelling from the acid treatment has disappeared will, if examined, be found to contain in the bottom of the hernial sac a hardened disk of consolidated effusion. This should not be disturbed by manipulation, as on its endurance depends the beneficial action that may yet ensue. If no improvement has resulted when this disk has been entirely absorbed, none is to be expected later.

PARALYSIS OF THE CAUDA EQUINA

Injuries inflicted to horses in the region of the croup, that are of sufficient violence, at times result in hemorrhages into the neural canal. At other times fractures of the caudal vertebræ inflict direct injury upon the terminal portion of the spinal cord. As a result of such injuries or hemorrhages there occurs a chronic interstitial inflammatory process, accompanied by the formation of variable amounts of fibrous tissue. Paralysis of the tail, and at times of the anal and vesical sphincters, follows. The tail hangs loosely and flops about when the animal is trotted. There is total loss of motor power in the tail. In some cases this is only unilateral; the horse is able to switch flies on one side only. When there is also a paralysis of the anal and vesical sphincter the case is, indeed, a complicated one. The rectum becomes blocked with feces in its posterior portion, some of which drop out during movement of the horse at rapid gaits. The urine dribbles away constantly. There is also loss of sensation in a limited area in the region of the anus, which can be demonstrated by pricking with a sharp instrument.

The prognosis is very unfavorable, as the condition becomes aggravated from month to month in most cases. As a result of involvement of the posterior portion of the rectum, there is rectal impaction, from which the animal suffers almost constant distress and sometimes colicky pains. Cystitis may develop in a serious form from involvement of the bladder. At the end of a few months, or a year at most, the horse is useless.

Treatment. In most cases this is of no avail. An occasional case may become stationary at the end of a month or six weeks, and the paralytic symptoms may slowly clear up. Most cases, however, become progressively

worse in spite of anything that is done for them. If it is desired to keep the horse alive for any particular reason, the rectum must be emptied manually several times daily to prevent impaction and colic. To allay the irritable condition in the bladder, salol, saw palmetto, belladonna or stramonium should be administered. Treatment directed at the lesion in the cord should include potassium iodid.

The horse must have nutritious food and good care in hygienic surroundings. In view of the fact that the whole trouble is the result of fibrous tissue formation in and around the cauda equina, it might be possible to accomplish something with fibrolysin. I recommend its trial in this condition. Fibrolysin is made by Merck & Company, and its active principle is thiosynanim, which has a selective action on fibrous tissue and brings about its resorption. I have already referred to results obtained with it in paraplegia following distemper in dogs in "Special Veterinary Therapy." While it would be desirable to bring the fibrolysin in direct contact with the lesion, this is not absolutely necessary. It can be given hypodermically in any portion of the body.

AGALACTIA IN MARES

Absence of lacteal secretion, or agalactia, is a condition that occurs in mares with considerable frequency. It has at times been known to assume the proportions of an enzootic trouble, especially during recent years. The cause seems to lie in feed that is deficient in nitrogenous matter, the affection being most common during years in which oats are "light" and other grain feeds are of inferior quality.

There occur two distinct varieties of this affection and their identification and recognition is important for reasons bearing on the prognosis.

1. In the first form the mare "makes a bag," but the mammary development is checked at about the ninth month of pregnancy. In some cases the mammary glands decrease in size from this time on, and remain only slightly enlarged until parturition. The enlargement has a doughy, clammy feeling and, when the teat is stripped, its contents have the appearance of a blackish jelly. This form of agalactia in mares is never benefited by treatment, although at the next pregnancy the mammary glands may perform their functions in a perfectly normal manner.

2. In the second form, the mammary glands show no sign of engorgement, or at least so little that it is barely noticeable, until a week or so before parturition. They then become slightly rounded; they occasionally show wax on the teat orifice, but contain no milk. If the teat is stripped a fluid is abstracted that has the appearance and consistency of very thin maple syrup. It is just a trifle sticky and has a light yellow tint. This form of the affection can be benefited by treatment, and at times the glands can be brought to normal function. For best results the treatment should begin before the end of the

gestation period, and should be brought to a climax on the date of parturition. In most cases the owner consults the veterinarian sufficiently early to make this possible.

Treatment should be commenced a week or, better still, two weeks before parturition and should consist of:

1. Dietary regulation and exercise.
2. Local massage.
3. Appropriate internal medication.

1. The mare should be fed concentrated, nutritious feed. If good oats are not to be obtained, oatmeal must be fed. The amount of grain should be such as would be necessary were the mare performing hard work. Light exercise must be given with regularity. The best is driving, going at a jog trot for periods not to exceed half an hour twice daily.

2. Massage of the udder must be practiced. This, to have effect, must also be done regularly. The act should consist in quite vigorous manipulation of the glandular portion or body of the udder. Do not strip the teats. This udder massage should be given morning, noon and night.

3. Internal treatment up to the day of parturition should consist of tonics and mild laxatives. A good combination is *nux vomica*, *gentian*, *cascara sagrada*, and *licorice*, in doses suitable to the weight and age of the mare. The amount of *cascara sagrada* should never exceed one dram daily; in small doses this drug seems to have the action of a galactagogue.

On the day of parturition the treatment is brought to a climax by the hypodermatic administration of *pilocarpin hydrochlorid*, one grain, and *strychnin*, a half grain.

Most mares that are affected in this manner will secrete enough milk to amply nourish their colts. Without treatment the lacteal fluid is entirely absent in every instance. In a few cases in which it was not possible to

begin the treatment before the colt was born, I have been able to excite the secretion of milk in a mild degree by rapidly repeated doses of pilocarpin on the day of birth. When this treatment is resorted to, the colt in almost every instance develops a diarrhœa. This is usually of a mild type and responds to ordinary treatment.

The occurrence of agalactia in brood mares, especially on small farms, is a veritable calamity. The veterinarian should warn his clients of the possibility of this occurrence and should impress upon them the great importance of early intervention in those cases of a curable form.

In the first form described, I have never been able to accomplish anything with treatment. There seems to be in those cases not only an atony of the mammary glands, but also an aberration of function, and, although the glands sometimes respond to treatment by showing signs of renewed engorgement, the fluid which they secrete remains the same—a blackish jelly. In either form of the affection, the mare should not be discarded for breeding purposes; the milk flow may be normal at the next pregnancy. On the other hand, I have had two or three patients that developed agalactia in two successive pregnancies.

STRANGLES

Strangles can almost be said to be one of the commonest diseases of horses. Although this is true, and despite the fact that this volume is confined almost wholly to the discussion of rare diseases, strangles is given a place here for the reason that it very frequently has an influence on the veterinarian's success in a community. The loss of a colt from strangles is a "black eye" that the veterinarian finds hard to cast off.

Strangles is better known by its common name, distemper. It is an acute febrile disease resulting from infection with the streptococcus equi. Judging from the frequency with which horses suffer from this disease, one is led to think that this organism is universally distributed and that it must of necessity be very tenacious of life. The disease seems to occur wherever horses are found, in all parts of the world. In itself a mild pathological process, this disease at times assumes a very grave character, not infrequently proving fatal because of the development of complications and the localization of pus foci in various parts of the body.

Strangles, or distemper, is a disease that most frequently attacks young horses, from sucking age to maturity. Occasionally older horses become affected. After an incubation period of from three days to a week the horse refuses to respond to command as promptly as formerly; the animal appears slightly depressed, or "dopey." The appetite lacks sharpness, and a little later the horse will not eat. There is now a discharge of muco-purulent matter from one or both nostrils; there may be a cough; and there is an enlargement of the submaxillary lymph glands. This enlargement is usually very painful under pressure. The temperature may be elevated only one or two degrees, or it may

rise four or five degrees above normal in well-marked cases.

After four or five days the appetite improves, the horse brightens up, and the glandular enlargement slowly disappears.

This is about the clinical picture of strangles in probably ninety per cent of all cases. It is safe to say that only a very small proportion of these cases ever come to the veterinarian's attention.

The common belief among horsemen seems to be that all colts are bound to have this disease sooner or later, and that usually it will take care of itself. This is practically true in both instances, except that occasionally a case will become complicated; and there are not many other diseases that appear in such a multiplicity of vicious pathological manifestations as one of these complicated, or "rotten" cases of distemper.

It may so happen that a case of distemper will take an irregular, complicated course under the care of a veterinarian and the horse may die. This is a calamity in the career of a beginner in the practice of the science of veterinary medicine and surgery. It is something that many farmers, especially, can not comprehend—that a horse could die with distemper! And nearly every time the veterinarian gets the blame. In the case of an old, established practitioner, this sort of an occurrence does not amount to very much. But with the recent graduate it frequently makes a big difference, often creates a serious hindrance in the path of his successful career in the community.

Strangles in an irregular form begins at times in the usual manner, while at other times it appears in a form that makes its recognition difficult. When strangles deviates from its usual, mild course we see an aggravation of all symptoms. The temperature reaches higher bounds

and persists indefinitely, at times dropping a degree or two and then suddenly rising again. The submaxillary enlargement goes over into abscess formation; the formation of abscess in the sub-lingual, retro-pharyngeal and other lymphatic glands follows. The parotid glands may next show enlargement, followed by pus formation. As a result of swelling which accompanies the formation of multiple abscesses in the laryngeal region and pharynx, there may follow serious dyspnea, so that tracheotomy may have to be performed.

In a number of cases the trouble does not stop here. There may follow an inflammation of the lungs, or pleurisy accompanied by empyema may develop. The facial sinuses may become the seat of a purulent process, becoming filled with pus. In several cases I have seen abscess formation in various abdominal viscera. Abscess formation occasionally occurs in lymphatic glands in distant parts of the body, and skin lesions are not exceptional.

The course of the disease in an irregular form is very protracted, sometimes requiring two months' time before all manifestations of the disease have disappeared. Usually the abscess formation goes forward in a successive manner, one forms after another; it is astounding in some cases to witness the progress. In the region of the head and neck I have seen as many as twenty individual abscesses. At the same time it is remarkable how well a colt will pass through, even a most severe case, at times. Permanent sequelæ are quite rare. One attack of this disease is generally presumed to make the recovered horse permanently immune.

Treatment. For the handling of strangles in any form, from the most ordinary attack to the most exaggerated, we have two means that alone can be relied upon. There are others, but the two that I am about to name are the

sine quo non of strangles treatment. With these two alone we can accomplish all that is possible to accomplish, and we do this in a very direct manner. These two agents are:

1. Bacterins.
2. The abscess knife.

If bacterins had absolutely no other ground for existence, their effect in this disease alone would give them a high place in the catalogue of veterinary therapeutics. Either plain strepto-bacterins, or better still the mixed pus bacterins, are to be used in strangles. Every case of this disease that is serious enough to require any attention whatever should be given at least one full dose. Cases of greater severity should receive two doses forty-eight hours apart, and several doses thereafter every third day until the case is well under control.

Abscesses that form should be lanced freely and promptly. On general principles the patient may be put on tonic treatment. Complications of a functional character are treated symptomatically as they arise.

The early use of bacterin treatment has proved its worth as a preventive of complications and as a rapid control of existing lesions in hundreds of cases in my practice. So reliable have I found their action, and so unquestionable, that I would not hesitate to undertake the handling of strangles in any form with them alone.

INFLUENZA

There is no one disease to which horses are subject that can present a greater variety of clinical pictures than this one. Influenza is essentially a contagious catarrhal fever. It is presumed to be due to infection with a microorganism of doubtful identity, and is presumably the equine type of "La Grippe." The characteristics of influenza are very similar in equine patients to those in human beings. They are fever, great depression, catarrhal changes in mucous and synovial membranes, and frequently grave complications such as pneumonia, and involvement of various organs in uncertain degree.

It is a question whether there is such a thing as a typical attack of influenza. The disease rarely follows a typical course; irregular developments are the rule rather than the exception. While affection of a distinct organ usually occurs in a typical form, it is only rarely that the same parts of the anatomy are invaded in a series of cases. When regular involvement of an organ, or a set of organs, occurs in an outbreak of influenza, the practitioner is very apt to be led astray in his diagnosis. The best example of this sort of an occurrence we have in the name "pink eye." This is nothing more than a panophthalmitis, now and then a mere conjunctivitis, being presented in the form of a localized influenza infection. Not many years ago "pink eye" was considered as a separate and distinct disease of horses. In like manner, we have witnessed outbreaks the cases of which were characterized by enteric symptoms, and in which the practitioner usually failed to see influenza infection. The most disastrous outbreak of this form of the disease occurred in the late '90's.

Quite regular involvement of other parts of the anatomy have been observed in various epizootics of this dis-

ease. Most commonly, however, influenza in equine patients runs an irregular course, involving various organs and tissues in uncertain degree.

In my opinion a typical case of influenza limits its manifestations to pathologic changes in mucous membranes. Whenever the case presents symptoms denoting involvement of an organic nature in other tissues, I believe it is atypical and due to a mixed infection. Infection with the virus of influenza gives, in my opinion, a typical attack of influenza characterized by high fever, depression, anorexia and catarrhal inflammation of mucous membranes. The addition of pus producing microorganisms—various strepto- and staphylococci—making a mixed infection, gives us what are usually termed complications: in other words, atypical cases. This opinion is in great part substantiated by a fact with which many practitioners are well acquainted, namely, that many times these complications are more violent than the true influenza symptoms, and frequently manifest themselves when the symptoms of influenza proper are already under control.

A division of influenza infections into catarrhal and pneumonic or pectoral sets, is not rational. A division can only be made on the basis of typical and atypical manifestations, classing all cases with symptoms that deviate from those of an acute, catarrhal inflammation, and its recognized phenomena, as atypical. Thus we may have a typical enteric influenza or a typical pectoral influenza, or a typical influenza infection of any other organ or tissue. But the course of the infection in any organ or tissue can only be typical when the pathological invasion is true to type; that is, when the infection is not a mixed infection. Just as soon as the virus of influenza is reinforced by staphylo- or streptococcus infection in an organ or tissue, symptoms

are produced which are atypical of influenza, and the case is then to be classified, of course, as an atypical case.

If we are ready to adopt this view and this method of classifying the disease, we must then admit that influenza, typical or true to type, is usually a very transient and benign affection. It is only those cases which are atypical—in other words, those cases in which the addition of pus-producing organisms has occurred—that are serious. From the practitioner's standpoint, this conclusion, based on the foregoing classification, will prove to be of merit, and of great assistance in the handling of both the typical and the atypical forms of this disease. Of course, as long as the identity of the infecting agent in influenza is in doubt, no classification from a bacteriological standpoint can be made with certainty. From a clinical standpoint, however, the foregoing classification is rational.

Symptoms. Typical form. A well-marked influenza infection usually begins with a chill or rigor. The temperature rapidly ascends to from 104 degrees to 106 degrees F. The horse gives evidence of great depression, appears "dopey" and listless. There is usually a mucopurulent nasal discharge and pertussis. The appetite is entirely lacking or at least indifferent. The visible mucous membranes are considerably injected and have nearly always a jaundiced cast. There is lachrymation and varying degrees of photophobia. The feces are coated with mucus; the urine is scanty and high-colored. The pulse is irregular and increased in frequency.

Depending upon the degree of involvement of tracheal and bronchial mucous membranes, there is a corresponding deviation from normal respiratory sounds and movements. Catarrhal pneumonia is not an unusual typical manifestation.

All of the foregoing symptoms vary in individual cases from very mild exhibitions proceeding to a favorable ter-

mination at the end of four or five days, to most violent presentations requiring several weeks to come to a favorable end; or else terminating in various complications and sequelæ, or even death.

Atypical cases are inaugurated in a variety of phases. Some of them begin as typical cases, later becoming atypical as a result of the invasion of a mixed infection. Others make their appearance known by the development of purely local lesions, such, for instance, as edema in certain parts of the anatomy, synovitis, tendinitis, or even lymphangitis. There may be manifestations of varying degrees of dermatitis, laminitis, or panophthalmitis. Pleurisy with purulent exudate, resulting in empyema; pleuro-pneumonia with necrotic tendencies; suppurative hepatitis; parenchymatous nephritis; all of these have been known to develop in atypical influenza either primarily or secondarily.

In the presence of influenza enzoötics or epizoötics the practitioner must suspect this disease as the excitant in every instance of abnormality that can not be referred to a recognizable cause. One attack of influenza does not confer permanent immunity. It is even doubtful whether a temporary immunity exists after recovery from an attack of this disease.

Typical influenza must be differentiated from simple catarrhal fever, colds, etc. Atypical cases may occasionally be confused with distemper in young horses, strangles, and, under certain conditions, glanders in the acute form. The period of incubation in influenza varies from three to ten days.

Treatment. Mild cases of the typical form in influenza require no special medication. Good warm quarters, proper ventilation, and tonic treatment bring these cases to a satisfactory end within four or five days.

More severe cases of the typical variety are treated

symptomatically. In my practice every case of influenza is given a dose or two of mixed bacterins, the idea of this being to forestall the invasion of pus-producing micro-organisms. The results have always been highly satisfactory. Medicinal treatment varies with the symptoms presented, but is in the main of a supportive nature.

As a routine treatment, no matter in what form the disease occurs, I find the administration of intestinal antiseptics of great benefit. Salol or the sulphocarbolates compound may be used for this purpose in suitable dosage for repetition two or three times daily. In atypical cases the treatment is also symptomatic. Depressants are to be avoided in all forms. As a routine form of treatment I would suggest mixed bacterins, intestinal antiseptics and tonics. Complications are to be met with appropriate treatment as they arise. The bacterins may be replaced by the anti-influenza serum in some cases—cases in which debility is marked. There is also on the market now a special influenza bacterin, composed of those streptococci which are usually found in atypical or complicated cases. This bacterin is also of value for purposes of prophylaxis.

Sequelæ. Influenza is frequently followed by after-effects of a permanent character as well as transient pathological effects. One of the most common after-effects is purpura hemorrhagica, although the occurrence of this sequel is a good deal less frequent under the bacterin treatment. In the treatment of cases of purpura hemorrhagica that follow on an attack of influenza, the practitioner must endeavor, more than anything else, to avoid any depleting agents. Aim to do everything possible towards building up the debilitated state of the patient's system. Warmth, pure air, nutritious feed, tonics and good nursing are to be featured. Influenza is to be considered always as a very readily transmissible disease. Cases developing in stables among other horses should

immediately be isolated. Cases of this disease are presumed to be contagious for variable periods of time even after all symptoms of the disease have disappeared. This period of time probably never exceeds ten days.

New horses, especially green horses, added to the members of stables are always to be considered as infected. They should receive a prophylactic dose of anti-influenza serum or influenza bacterins, and isolated for a period of ten days. If the disease has not made its appearance at the end of this time the animal may be considered free from the infection.

INFECTIOUS CEREBRO-SPINAL MENINGITIS

While there is no tangible evidence to show the specificity of any grade of inflammation in the coverings of the brain and spinal cord in Equidae, there is no doubt about the infectious character of certain types of this condition from a clinical standpoint. A type of cerebro-spinal meningitis has on occasions taken on an epizootic character among horses, and enzootic outbreaks of the disease are seen in various parts of the United States almost every year. By far the most cases occur in the spring and in the fall. Both young and aged horses are affected.

The disease presents symptoms the severity of which coincides with the extent and gravity of the inflammation in the meninges and with the amount of effusion that occurs as a result of this inflammation. As both the degree of inflammation and the quantity of effusion are variable, so also do the symptoms vary in particular cases from violent, almost maniacal attacks to dull, mediocre or indifferent manifestations.

In the most active form the disease begins with a high temperature in company with a rather slow, full pulse. There is from the beginning a certain degree of psychic bias. I mean that the horse shows some peculiarity of action, of countenance, of attitude, which can only be attributed to a brain and spinal cord lesion. There develop in rapid succession, sometimes simultaneously, dysphagia, lack of coördination, "staggers," delirium, decubitus and death. Opisthotonos prevails throughout the attack. Saliva drools from the mouth, the horse has difficulty in maintaining a comfortable attitude; he may plunge violently about, fall, and die in convulsions. The onset of symptoms to the end in death may take not more than twenty-four hours.

Cases of such virulency are not so common as those in which the disease begins with a moderate rise in temperature. Some difficulty in swallowing is present; the facial expression is dull, vacant, stupid; the head hangs heavily or it is pressed firmly against a wall or other object. If the animal is made to move, the movements are made sub-consciously and without recognition. There are moments when the animal appears brighter, but these become less frequent and of shorter duration. At the end of two or three days the animal goes down, remaining in the recumbent position for variable lengths of time until recovery or death terminates the case. During the entire period of recumbency (which may last from three to fifteen days) there are spells of delirium alternating with lucid periods. Before death occurs there is in most cases marked coma with subnormal temperature.

Favorable cases begin to improve after a few days of semi-comatose periods; the horse recognizes voices, calls for feed, later rises to chest position and with assistance will rise. Dysphagia, lack of coördination, facial paralysis, paraphimosis and other troubles may remain indefinitely, as complications or sequelæ.

Cases with violent symptoms must be differentiated from rabies. Anthrax must be considered in diagnosis of hyper-acute cases. Mild cases must be differentiated from simple meningism. The differential point here has been pointed out in the chapter on meningism.

Treatment. I begin the treatment in every case with fifteen grains barium chlorid intravenously. This provokes rapid elimination of the intestinal contents and, if administered early enough in the attack, changes the entire aspect of even a serious case. In my hands the results from this handling have been very gratifying. The remainder of the treatment consists of alternate doses of Fowler's solution of arsenic and potassium iodid

in amounts varying according to the weight of the patient. I give half an ounce of Fowler's solution and a dram of potassium iodid at each dose for a twelve-hundred-pound horse. Beginning with the Fowler's solution, I have the attendant administer a dose of the iodid three hours later; again three hours later the arsenic solution, and so on every three hours from early morning to late at night. I keep this up until an ounce of the potassium iodid has been given; then I continue Fowler's solution alone every four or five hours.

With this treatment I have had some remarkable results in this disease. I pay no attention to the patient's general condition, such as the appetite, temperature, and so on. Neither do I worry about the position of the patient; if recumbent, I have him turned over every three or four hours.

As a prophylactic measure, hexamethylenamine should be administered to other horses in the stable where a case exists. This drug can be relied upon to prevent the development of this disease if it is used before infection has occurred. From one to two drams should be given in a pail of water twice daily.

As it is quite generally conceded that the infectious matter gains entrance to the body in feed or drinking water, both should be changed if possible. On general principles all cases of this disease should be handled as are those of transmissible diseases, even though doubt exists about the transmissibility.

In exceptional instances the infecting agent seems to be so weak or inactive that the symptoms produced are very mild and diagnosis is difficult. In such cases, if one is not familiar with the vagaries of this condition, the cases are easily mistaken for influenza. Doubt can usually be dispelled, however, if the horse is excited. Cracking a whip and threatening the patient with it, will

nearly always make the animal show signs of the disease suspected, such as involuntary jerking of the limbs, twitching of the facial muscles, and at times opisthotonic position. The temperature in such cases is nearly always subnormal, sometimes as low as 97 degree F., reaching a degree or two above normal, after the disease is fully established.

Some of these mild, doubtful cases later assume a very serious turn, running a prolonged sub-acute course. In many of them recovery is incomplete, lack of coördination in the hind quarters remaining for variable periods of time. About twenty per cent of cases terminate fatally. The mortality in the hyper-acute cases is from sixty to eighty per cent.

In the presence of cases of this disease the attending veterinarian is not infrequently questioned by the client in regard to the relationship between this disease in horses and infantile paralysis of children. While it was for a long time presumed that domestic animals could develop and transmit infantile paralysis to human beings, Simon Flexner has conclusively shown that the forms of paralytic meningitis that affect animals have no relation to similar diseases in humans and, therefore, can not be transmitted to them. It has only recently occurred in my practice, however, that a case of infantile paralysis developed and caused the death of a child on a farm where, two weeks previously, I treated two well marked cases of cerebro-spinal meningitis in horses. No doubt this must be viewed as a coincidence only.

ERYSIPELAS

This is an inflammatory skin disease which affects horses occasionally. It is caused by a streptococcus infection and is an acute, febrile disturbance.

Symptoms. Erysipelas is frequently ushered in with a rigor. The temperature rises to 103 degrees F or even higher. Simultaneously with this there develops upon the infected region of the body a diffuse, painful thickening. In two cases which occurred in our practice the infection was on the side of one hip. Within a few hours after the swelling develops there appear in the area numerous pea-sized vesicles. In another few hours these rupture and discharge a sticky, pale serum. The vesicles now coalesce and are soon covered with a dry, thin scab. In some cases there is great pruritis at this time.

If the infection is on an extremity there is usually great lameness. In white horses the skin in the infected area is intensely reddened. The swelling and vesicle formation may spread progressively over a large area within a few days, or may limit its activity to the original area of infection. When the vesicles have burst and scab formation has taken place, the process of repair seems to begin immediately under the scab. In a few days the scab lifts off, leaving temporarily bare spots. Later these are again covered with hairs.

Erysipelas is differentiated from other skin diseases by the force with which it makes its appearance. Its local manifestations appear in rapid order:

1. Diffuse, painful swelling.
2. Vesicle formation.
3. Rupture of vesicles.
4. Desquamation.

Treatment. A purgative dose of aloes is given first, on general principles. Specific treatment in the form of streptobacterins gives fine results. The local manifestations respond most promptly to painting with tincture of chlorid of iron once or twice daily. This brings even severe cases to a prompt and satisfactory end.

Erysipelas is one of the examples found in veterinary pathology that serves as a good illustration of the difference existing in similar infections between animals and human beings. Clinically, erysipelas in animals does not compare with the same disease in human beings, and this is one of the chief reasons why erysipelatous infections in animals are usually not recognized as such clinically. The erysipelatous infection that is most commonly seen in animals is not nearly so grave in character as the usual erysipelatous infection occurring in human beings. I make this notation particularly for the reason that most text-books attempt to make an even comparison between the erysipelas of horses and the erysipelas of human beings. This tendency is the result of the all-too-common custom of permitting physicians to conduct the course in pathology in veterinary colleges.

SEPTICEMIA

We shall take up this condition from its pathological standpoint first. The term septicemia literally means poisoned blood. The term is used in a rather broad sense, clinically. Speaking in a strictly pathological sense a septicemia would only include those conditions characterized by the presence of septic matter, or pathogenic bacteria, in the blood stream. Cases in which the morbid process is the result only of toxins in the blood stream can not, strictly speaking, be termed cases of septicemia. Furthermore, just at what moment or under what conditions, an ordinary infection of a wound ceases to be an ordinary infection and becomes a septicemia, is a doubtful point. From the practitioner's standpoint a septicemia is any serious or grave condition resulting from the infection of the organism with pathogenic bacteria. When the infection is not severe enough to produce marked systemic disturbance the practitioner is content to term it an ordinary infection.

Clinically, for reasons bearing on the treatment of the condition, a septicemia needs to be differentiated chiefly from a pyemia. Pyemia makes its identity plain by the presence of localized pus collections, which are absent in cases of septicemia. A good definition for septicemia would read somewhat like this: Septicemia is an acute, febrile disturbance resulting from infection of the organism, either through a wound or otherwise, with pathogenic microorganisms. But, again from a clinical standpoint, the term septicemia is reserved almost always for systemic participation in wound infection of such a degree that it creates a clinical entity. In other words, whenever the systemic disturbance overshadows the local or traumatic lesion, the term septicemia finds application. The only differentiation to be made, and then from

the viewpoint of treatment only, is from pyemia, and, under certain conditions, malignant edema.

Symptoms. Septicemia appears in various degrees of severity, depending upon the virulency of the infecting agent and the resisting power of the animal infected. It develops early in the course of wound lesions in contradistinction to pyemia which appears later and in a less precipitate manner. Septicemia is ushered in with a chill, which may, however, be mild in character and pass unnoticed. The temperature during the chill, or immediately thereafter, rises rapidly and may reach as high as 107 degrees F. in a few hours. Temperatures of 105 degrees F. and 106 degrees F. are ordinary in this condition. The patient appears generally indisposed; the respirations are increased, and the pulse exceeds its normal rate considerably. Feed is refused, and the patient gives other evidence of the gravity of its condition. The discharge from the wound has either ceased entirely, or it has assumed a thin, watery consistency. The immediate vicinity of the wound is the seat of a tense, firm, hot and painful swelling. Cord-like ramifications of this swelling may be seen following the course of the lymphatic vessels in some instances. In well marked cases this swelling rapidly increases in size and area, and in the course of a few hours it may involve an entire limb, or a great portion of the body. If the wound from which the infection developed is situated on one of the limbs, great lameness, entirely out of proportion to the extent of the trauma, develops.

The swelling, when incised or tapped, emits no discharge other than a few drops of serum in the early stages. Later a rather free dripping of serum may follow when an incision is made. In exceptionally severe cases the patient may succumb within the course of seven or eight hours, collapsing suddenly and dying in a few

minutes. In less virulent infections the condition terminates in decubitus and death after a few days, violent delirium preceding the end in some cases, and pronounced coma in others.

Cases that are promptly and judiciously treated recover after variable periods of time, depending upon the severity of the infection, the treatment applied, and the recuperative powers of the animal. In exceptional instances the infection is so virulent, and so rapidly and so grossly disseminated throughout the body, that the animal succumbs before great swelling or other changes occur in the wound or its vicinity. These cases are rare.

Differentiation. Pyemia: Pyemia is less tumultuous in its approach. It appears rather as a secondary development, often after the original seat of infection is under control. It is characterized by metastatic foci of pus. These vary from small abscesses to large pockets of purulent material.

Malignant edema: The diagnostic feature of this condition is the characteristic odor, the gangrenous trend, and more especially the presence of gas in the tumefaction, evidenced by crepitation.

Lymphangitis: The swelling in lymphangitis is more circumscribed, has a line of demarcation, and is of a doughy consistency.

Treatment. The successful handling of a well developed case of blood poisoning requires, in my opinion, the best professional judgment. The favorable progress of a given case is frequently interfered with by the most trivial circumstance in the method of treatment. It is my belief that many cases of septicemia terminate in death chiefly as the result of over-dosing with medicinal agencies. The practitioner too frequently allows his diagnosis of septicemia, "blood-poisoning," to take possession of his mental faculties to the extent of a veritable

panic. He over-reaches in the attempt to save the life of his patient; he does too much. His treatment is too heroic, or it does not follow a sound line of therapeutics.

What little reserve disease-resisting stamina the patient has left, the veterinarian wipes out with his treatment. If there is one condition in which heroics do harm, it is in this one. Every move on the practitioner's part in the treatment of a case of septicemia should go no further than an effort at assistance. Do just enough to help the natural efforts inherent in the organism, and don't try to help too much.

We can do all that should be done, all that it is possible to do in a really beneficial sense, with three things:

1. Mixed bacterins; or anti-streptococcic serum.
2. Iodin.
3. Nux vomica or strychnin.

With this give me a lance, and I am ready to do battle with any case of septicemia resulting from wound infection.

Begin the treatment, if the case is ordinary or mild, with a full dose of mixed bacterins. If the case is more than ordinarily severe, and you fear that bacterins will be too slow, use anti-streptococcic serum along with the bacterins. The serum will dilute and hold in abeyance the toxins in the blood stream until the bacterins can induce the elaboration of anti-bodies. There is no question in my mind in regard to the value of bacterins in acute infectious diseases. In fact, I have had much better results from bacterins in acute diseases than in chronic. I have used bacterins since 1906 (the first were "home-made") and, although my practice has probably not been so large as that of some veterinarians, I have used several thousand doses of them. I have so much faith in bacterins and depend upon them to such an extent that I would find it a most difficult problem to practice without

them. I believe that the veterinarian who gets no results from bacterin treatment, or the one who disputes their value, has not learned the art of their successful application, and does not know enough about them to make a good judge of the matter.

In cases of septicemia I repeat the giving of bacterins on the third day; not three days later, but on the third day from the first dose. A third dose is then given three days later.

As supportive treatment I rely wholly on nux vomica or strychnin. I give preference to the fluid extract of nux vomica and order it given in half-dram doses every three hours during the daytime. If for any reason oral administration is impracticable, it can be given per rectum with just as good effect, excepting for its beneficial action in creating an appetite.

I have long ago discarded combinations of iron, quinin, and other hematinics. Their action is too slow and in nearly every instance accompanied by some disagreeable by-effect. Iron, for instance, constipates; quinin is a heart depressant when given in sufficient amount to be of benefit.

For the local treatment of the seat of infection I can recommend tincture of iodine without reserve. But it must be used generously; don't be stingy with it. Paint the swelling and a considerable area beyond it freely with pure tincture of iodine twice daily. Literally soak the parts with it, and keep up this soaking twice every day until the case is terminated.

If the wound is of such a nature that it does not drain well, make proper drainage, if possible, with the knife. For injecting the wound, in place of watery solutions of antiseptics, I highly recommend tincture of iodine, one part, in seven parts ether. This can be used without fear of doing damage in the most delicate tissues, and its

action is very gratifying. The wound should not be molested further than to provide drainage, and an application once or twice daily of the ether-iodin solution.

Every practical means must be used to keep the patient eating. The *nux vomica*, when given orally, plays no small part in this. The animal should have warm quarters and should not be subjected to unnecessary excitement. A good nurse is more than valuable. Under this treatment, with the addition of good nursing, even serious cases of septicemia are well on the road to recovery in a week.

After-treatment, consisting of administering Fowler's solution of arsenic, or the elixir of iron, quinin and strychnin phosphate, may be indicated.

During the time that the disease is active the patient must be confined in a single stall, and should not be moved out of this stall under any consideration until the disease has been conquered so far that the animal is again eating normally, and the swelling is receding. The animal may then be turned into a box stall. If no box stall is to be had the patient should be carefully led at the halter, walking not over half a block the first time out. The amount of exercise is then gradually to be increased. This is a very important point in the favorable termination of a case of this kind.

In cases in which the immense swelling involves the under side of the abdomen and the sheath, a degree of paraphimosis may result. This rarely requires any special attention. The swelling and consequent protrusion of the organ does not remain long enough to be of any moment. As soon as the case has reached the stage in which exercise may be given, the swelling leaves, and the paraphimosis corrects itself. If the paraphimosis really becomes serious, the sheath may be scarified and the penis supported with appropriate bandages.

The bacterins that are used in the treatment of a case of septicemia should contain not less than four billion killed staphylococci and one billion streptococci in each dose. All must be of equine source and must be polyvalent or mixed. The injection of the bacterins must be made only subcutaneously, *never* intravenously, and then only in a region remote from the diseased area. Never inject them near the lesion. The anti-streptococcic serum may be given intravenously. The dose is from 20 to 40 mils. daily.

MALIGNANT EDEMA

Horses seem to be more susceptible to this disease than other domesticated animals. It is an acute febrile disturbance characterized by the development of an edematous and gaseous swelling at the seat of inoculation. The infective organism is an anærobie bacillus, which is said to be universally present in the soil and in animal excrement. Despite the general distribution of the bacillus, the disease is comparatively rare. The infection develops most commonly in puncture wounds, contusions, or insignificant abrasions of the integument. Infection may also result from injuries to the mucous membrane of the mouth, nose, or eyes. The latter is rare.

This disease runs a rapid and frequently fatal course. After the period of incubation has expired, an extensive swelling or edema develops at the seat of inoculation, within a few hours. It is usually a rather smooth swelling, blending gradually with the normal regions around it. At the same time the animal gives evidence of marked constitutional disturbance. The respirations are rapid; the pulse increased in frequency so that it may reach 60 or 70 beats per minute, and the temperature may rise to 106 degrees or 107 degrees F. in the course of the first few hours.

When the swelling at the point of infection has fully developed, it becomes emphysematous. When palpated it crepitates. This is due to gas formation in the subcutaneous tissues as a result of the bacterial invasion and the establishment of necrotic processes. At the point in the swelling at which the disease is most active a gangrenous area develops. This area is frequently darker in color than the remainder of the tumefied area, and, when cut into, this part of the tumefaction proves to be non-sensitive. At least, the affected animal gives no evidence

of pain, from the knife stroke. In grave cases, or those which are not favorable, the tumefaction may spread over a considerable portion of the body. In certain sections of the South, malignant edema is frequently seen affecting the region of the parotid gland. The cases are nearly always fatal.

Differentiation. The symptoms of malignant edema must be differentiated from subcutaneous emphysema following injuries, and from septicemia following an injury. Subcutaneous emphysema resulting from injuries, and which is merely caused by suction created from movements of the thorax during breathing, is not accompanied by any systemic abnormalities. The temperature is normal, and the horse is not depressed or dejected. Usually, the wound in company with subcutaneous emphysema is rather large and gaping. The wound in cases of malignant edema is nearly always a small puncture wound, and in most cases can not even be demonstrated. Septicemia resulting from wound infection differs from malignant edema in the fact that crepitation is absent in the swelling of the former. Neither does septicemia show the gangrenous area at the active point in the swelling.

Prognosis. This is unfavorable in probably fifty per cent of all cases, and in over ninety per cent of the serious cases. Death may come within four or five hours in some of the most severe cases. In moderate attacks it may come in a day or two, while mild cases recover after variable periods of time. Cases that live over forty-eight hours may be considered favorable after the lapse of such a period of time.

Treatment. The treatment of malignant edema is not very satisfactory in results. Some practitioners have made reports of good results from the use of oxygen gas. A small oxygen tank, such as is used by some in the treat-

ment of parturient paresis in cows, must be at hand. By means of rubber tubing and a hypodermic needle the oxygen is forced freely into various parts of the tumefaction.

Other practitioners claim good results from incising the active region of the swelling freely, and then packing it with gauze or wads of cotton previously saturated with hydrogen peroxid. All the cases that I saw were treated by simply cutting freely into the active area of the tumefaction and liberal irrigation with peroxid of hydrogen. All died. Cases reported as recovering are said to make a slow final recovery. Usually the gangrenous center of the tumefaction sloughs out, leaving a difficult wound to heal.

In future cases of malignant edema in my practice I shall resort to treatment similar to that used in poisonous snake bite, namely, complete extirpation of the active area followed by the application of crystals or a saturated solution of permanganate of potassium. The internal treatment should consist of supportives.

PURPURA HEMORRHAGICA

Purpura hemorrhagica is a pathological state of the animal organism characterized by the appearance of hemorrhages into the subcutaneous and submucous tissues, in company with infiltration of these tissues with serum, and in some cases desquamation of parts of the integument. It is said to be an infectious, but not a contagious, disease. At one time it was presumed to be contagious.

The disease, from a veterinary standpoint, may almost be termed more correctly a sequel. Purpura hemorrhagica in veterinary practice occurs almost always as a sequel to some other infectious, debilitating disease. The most common of these is influenza. Other cases follow an attack of strangles, or a case of distemper. A number of diseases seem to possess the faculty of so disarranging the animal organism that purpura hemorrhagica supervenes. Just what this disarrangement of vital phenomena must amount to, is not easy to judge. Occasionally purpura hemorrhagica will follow an attack of influenza of ordinary grade, while another case that has been much more serious will not be followed by this sequel. In some instances only very mild or ordinary attacks of distemper have preceded the development of purpura hemorrhagica. To a certain extent this feature strengthens the belief that the disease is infectious, although no proof of a specific etiology has yet been produced. On account of the usual infectious nature of the disease to which purpura hemorrhagica is a sequel, it becomes a most difficult matter to obtain satisfactory clinical evidence of the specificity of any organisms that might be isolated therefrom. Various streptococci have been blamed for this disease, but it has never been successfully transmitted by artificial inoculation with such cultures.

Symptoms. Most cases do not come to the veterinarian's attention until the disease has become quite well developed. The earliest sign is usually an edematous enlargement of the nose and face; occasionally the first swelling appears just in front of the trachea in its middle third. Cases which are not so often seen are those in which swellings appear distributed over the body in spots, somewhat resembling pronounced urticarial swellings. Simultaneously with the appearance of these edematous swellings, appear petechiæ on the visible mucous membranes, and hemorrhagic discharges are emitted from the natural openings. These discharges are thin and watery in consistency, and frequently have a rusty instead of a hemorrhagic tint.

The edematous swellings become gradually more extensive, so that at the end of three or four days they cover almost the entire animal. They are most marked in the extremities, the head, sides of the abdomen, and the pectoral region. The surface of some of these swellings, as well as the flexion surfaces of such joints as the hock, carpus and pastern, exude the characteristic rusty or hemorrhagic fluid.

The temperature in cases of purpura hemorrhagica is high early in the attack, reaching 106 degrees F. quite ordinarily. Later in the course of the disease, the temperature is what might be termed "fitful"; today it may be 104 degrees F. and tomorrow 102 degrees F., while the next day will record a temperature of 105 degrees F. or any number of variations. The general clinical attitude of the case rises and falls sympathetically with the temperature variations; today the patient presents a favorable aspect, tomorrow an unfavorable one. This could be explained most plausibly as a result of hemorrhages into internal organs of considerable importance, or of the out-pouring and absorption of exudates in the region of

physiologically important regions of the body. The patients frequently have a good appetite throughout the entire course of the disease, although the great swelling of the lips may make prehension of food almost impossible. The edema may be so severe in the nasal region that breathing becomes difficult and suffocation may threaten.

Affected animals maintain the standing position throughout the course of this disease. A terminal symptom in some cases is desquamation, occurring in patches.

Complications. Complications are the rule rather than the exception in cases of purpura hemorrhagica. Why this should be is easy to understand when we recall that purpura, which is in itself a serious disease, frequently or almost always follows some other equally debilitating disease. The affected animal has not much resisting power left, and therefore easily becomes the subject of invasion by other destructive processes.

Pleurisy, with hydrothorax, is a very common complication. Pneumonia and peritonitis are not uncommon. Laminitis may complicate a case of purpura hemorrhagica.

Other cases develop recurrent diarrhœas; and now and then attacks of acute indigestion may transform an otherwise favorable case into an unfavorable one. As a result of the pain due to acute indigestion, the animal is forced into recumbency and this is followed by death. Sloughing of parts involved in some of the swellings may occur as a result of secondary infection in some cases. Acute iritis terminating in total or partial blindness is another complication that may develop. One or both eyes may be involved.

Paraphimosis, constipation or actual impaction, and now and then intestinal flatulence, may aggravate a case of purpura hemorrhagica.

In fact, the more experience one has with this disease, the less does he marvel at the appearance of complications of any sort. One may expect anything in a case of purpura hemorrhagica. As has already been pointed out, the resisting power of the animal is at a low ebb.

Differentiation. Purpura hemorrhagica is not easily confused with any other affection. In cases in which the swellings appear in spots it must be differentiated from urticaria. Examination of the visible membranes will disclose patechiæ in cases of purpura hemorrhagica, whereas in cases of urticaria these are absent. Violent pruritis common to urticaria is not exhibited in purpura hemorrhagica. The swellings of urticaria are transient, lasting at most a day or two. The purpurial swelling becomes more pronounced during this time.

Treatment. My experience with purpura hemorrhagica has shown it to be a self-limiting disease, the treatment of which is chiefly symptomatic. I have not been able to abort or shorten the duration of cases by the use of treatments that have been reported as doing this. In my experience I have found that it requires about one month for a well developed case of this disease to run its course to a favorable termination. Very mild cases may recover in half this time.

I would place the rate of mortality at about forty per cent in the well marked cases, and at about twenty-five per cent in all cases. This death rate can not be said to be direct, because most of these animals die not from purpura hemorrhagica, but from complications incident thereto.

Human patients have been recently reported promptly cured of purpura hemorrhagica by the intramuscular injection of large quantities of fresh blood. While I do not believe that this disease in human beings is the same as that which veterinarians find in their patients, I would

suggest that this form of treatment be tried in animals. Other physicians have recently reported fine results in the treatment of this disease in human beings from the use of emetin hydrochlorid.

Dr. E. L. Quitman claims excellent results from potassium dichromate, orally, in three to six-grain doses every few hours, in purpura hemorrhagica of horses. He claims that the disease comes to a favorable termination in a much shorter time under this treatment. I have not been able to substantiate this, although I have not used this treatment in a sufficient number of cases to either recommend or condemn it.

The best results that I have had were obtained with mixed bacterins. I treat my cases very much like I treat cases of septicemia. Bacterins and supportive medication consisting of nux vomica and bitter tonics, some iron in the form of the tincture chlorid, and the handling of complications as they arise, will have as good results as other more troublesome and expensive procedures.

Tracheotomy may be indicated in some cases. This should be resorted to at the earliest moment that it becomes evident as a requirement. The tube must be kept perfectly clean, and may be removed and the wound allowed to heal as soon as the danger of suffocation has passed.

The convalescence from purpura hemorrhagica is slow. Fowler's solution of arsenic, preparations of iron, quinin and strychnin, good feed and nursing, are important in after-treatment.

Mild cases, promptly given bacterin treatment, may make a complete recovery and be ready for work in three weeks. Severe cases may require as long as two months before they are again in working condition. Mares in foal may pass through an attack of purpura hemorrhagica and give birth to healthy colts. In one instance that

came to my notice a mare suffering from an exceptionally grave case of purpura hemorrhagica gave birth to a fine colt, in perfect health, while the attack was at its height. During the birth the mare assumed the recumbent position, from which she was raised with difficulty, dying a day later.

ACUTE INFECTIOUS DYSENTERY

In the northern half of the United States horses are the subjects of this disease. It is a hyper-acute, sometimes rapidly fatal, form of diarrhœa occurring most frequently in young horses during the cold months of the year. While the etiology of this disease is as yet unknown, it is no doubt infectious in character, and is transmissible. It occurs in an enzoötic form and may affect all the young horses in a series of stables.

That it is not the result of errors in diet is easy to demonstrate. It occurs in stables widely separated and in which the feed and methods of feeding are not at all similar. It affects young horses indiscriminately in a stable or region, while the old horses fed on the same feed and under the same conditions are not affected. When a case develops in a stable in a young horse, other young horses in the stable can not be prevented from contracting the disease, although the method of feeding be changed immediately after the development and recognition of the first case. The earliest cases in an outbreak are usually the most severe. Towards the end of an enzoötic series of cases, only mild attacks are seen.

Symptoms. There are no premonitory signs that are noticeable. The horse may be attacked while idle or while in harness at work. Suddenly a diarrhœa begins. The evacuations are at first only a trifle more soft in consistency than normal, but an evacuation is followed by another every five or ten minutes. After three or four evacuations have occurred thus, the feces that are passed in following evacuations become more and more soft and watery. At the end of an hour the evacuations are merely water with a trace of feces in it. This liquid is passed every five or ten or, at most, every twenty, minutes, and spurts outward with considerable force.

The horse shows no sign of pain, but soon, in two or three hours, signs of exhaustion appear. The patient perspires freely, the respirations are rapid and shallow, the pulse is almost imperceptible. If it can yet be counted by feeling over the course of an artery, the beats may register sixty to seventy per minute.

The temperature usually stands near 103° F., although in some cases it is somewhat higher. If the case is not now taken in hand and given proper treatment, the animal soon succumbs. The facial expression becomes drowsy. Shifting of the weight from side to side follows; there occur a few plunges or awkward straddling movements; the animal falls or clumsily lies down and death comes quickly. Autopsy shows an empty, slightly congested alimentary tract; nothing else.

I have known cases of this disease to run their complete course, from first symptom to death, in so short a period of time as four hours. However, I have never known one to die, not even of the most severe form, when proper treatment was given during the early hours of the illness. Mild cases will recover without any treatment, but evidences of the disease remain for weeks in the form of inappetence, drowsiness, loss of weight, and in some instances edematous swellings affecting the limbs, head, etc. Even severe cases recover more completely under proper treatment than mild cases do if not treated. Furthermore, it is impossible to tell in the beginning of an attack how violent or severe the affection will become. The symptoms may improve spontaneously within a few hours; on the other hand another few hours may mark the death of the subject. It is therefore imperative that all cases be promptly treated.

The diagnosis hinges on the history, namely, sudden and rapidly increasing diarrhœa without a sign of previous illness, change of feed or other evident etiological

factor; and on the early exhaustion in the absence of signs of pain. The feces have no abnormal odor; no blood or mucus accompanies the evacuated matter. The intestinal sounds heard by auscultation are not riotous, but have the sound of being compressed and wrung; very much such a sound as is emitted on crushing a bunch of grapes quickly in the hand. The sound approaches nearer to being a high-pitched, squashy crunching than any other description of it that I can give.

Treatment. Experience with this disease under a variety of circumstances has demonstrated the fact that acids will check the course of an attack with almost marvelous rapidity. Acetic acid gives the best results, besides being very cheap and easy to handle. The dilute acetic acid is given in from one- to three-ounce doses every twenty minutes until the evacuations occur with less force and regularity. As soon as this is apparent from a half to one ounce of tincture of opium is given in capsule. This completes the handling of the case. If the opium is given before the acetic acid has controlled the condition it has a tendency to cause flatulence and pain.

If a case should come into the veterinarian's hands quite exhausted and apparently moribund, friction and warmth should be applied to the body, and half an ounce of spirits of camphor should be administered immediately. Then begin at once with the acetic acid doses. The acetic acid can be given in capsules in mild cases. In the severe cases, give it with a dose syringe with an equal amount of warm water.

I would warn the practitioner against giving an unfavorable prognosis in these cases even when the patient seems near collapse before the treatment is begun. Withhold prognosis until you have given a few doses of the acid.

Mild cases treated should not be permitted to eat or drink anything for at least twelve hours. Well marked or serious cases should be made to fast for twenty-four hours after treatment. A course of tonic medicine is usually indicated as after-treatment in all cases.

If a troublesome constipation should follow an attack of this disease, I would recommend that it be corrected by the use of either castor or linseed oil. Do not use salts.

Cases treated as outlined above usually make a rapid and complete recovery.

TETANUS

Because of the average practitioner's familiarity with this disease we will limit our discussion to a few interesting points regarding the pathological anatomy of tetanus and then consider the treatment.

Tetanus is the result of an intoxication of the nervous elements with the toxin of the bacillus of Nicolaier. The bacillus is anaerobic, and the most favorable wounds for its activity are small wounds, deep punctures, and all traumata of such a character that will prevent free contact of the exposed or injured tissue with the air. Large, open wounds are not favorable for the propagation of a tetanus infection.

The period of incubation may be as short as forty-eight hours and as long as three weeks. An average can be reasonably placed at about eight days. This is influenced by the temperament of the animal, by the amount of infective matter, and the virulency thereof.

Tetanus is one of the diseases in which the infecting organism remains at the site of inoculation; the symptoms are the result of toxins which the bacillus elaborates. In tetanus these toxins travel along the nerve trunks, and no disturbing symptoms are occasioned until a sufficient quantity of toxins has been fixed in the brain cells. That this is not a mere theory has been shown by several investigators in the following manner: A horse is artificially inoculated with a virulent culture of tetanus bacilli, making the inoculation in the foot of one leg. A surgical division of all the regional nerve trunks is then made. The animal so treated will present no tetanus symptoms. Tetanus, for this reason, can not be strictly termed a toxemia. The exciting toxins do not travel in the blood stream. More properly speak-

ing it is an intoxication of the nervous tissue of the body.

An attack of tetanus does not confer a permanent immunity. Animals surviving an attack of this disease do acquire a certain degree of immunity, but the periods for which the immunity holds are uncertain in length. A second infection resulting in the development of a second attack of the disease has been reported in as short a time as two months after recovery from a primary attack.

Tetanus can be prevented with almost positive certainty by the proper use of prophylactic doses of anti-tetanic serum. A dose should be given as soon as possible after a wound of favorable character is inflicted. A second dose should be given eight days later. It is quite generally agreed that 500 units constitutes an ample prophylactic dose, if a similar dose is given eight days thereafter.

Tetanus appears in a more severe form in the Northern states than it does in the South. Cases occurring in the South nearly always terminate favorably, many of them making spontaneous recoveries. Tetanus in the colder climate of the North, especially in winter months, is a very grave condition, accompanied by a high death rate. The duration of an attack of tetanus is from four to six weeks.

Treatment. There is no treatment for this disease which is accorded universal recognition. More than this, there is no known form of treatment which will give satisfactory results with any regularity.

In giving credit for reported cures in this disease the practitioner should heavily discount results reported from the Southern states. Tetanus in the South, as has already been remarked, is a mild disease. I make this statement out of first-hand experience with this disease

in various parts of the South. Most of the reports of good results from anti-tetanic* serum come from the South.

That tetanus can not be cured, nor even benefited, by the use of anti-tetanic serum was proved to me beyond all doubt while I was yet a student of veterinary medicine. I was serving at that time as an assistant to two veterinarians in the city of Milwaukee. These gentlemen were in the employ of a live-stock insurance company that gave free veterinary services to the animals insured. The company had at that time, as well as I can remember, more than 3,000 horses insured in Milwaukee, all of them in charge of the two veterinarians to whom I was assistant. No expense was ever thought of by these two gentlemen when it was a matter of saving the life of an insured horse; the insurance company paid the drug bills, and the watchword with them was, "go the limit." If ever anti-tetanic serum was given a thorough trial as a curative agent, these two veterinarians gave it. I, myself, under their supervision, used more anti-tetanic serum in a single case of tetanus than I have used all told during the past twelve years, and more than I expect to use during the remainder of my career as a veterinary practitioner. Case after case was treated in this manner with not a shadow of benefit that could be attributed to the serum. True, some cases recovered; but not more rapidly, nor with less trouble, than under the carbolic acid treatment which was in vogue at that time. And the cost of treatment with anti-tetanic serum was, therefore, and is today, entirely out of reasonable proportion to the benefit derived.

The conclusion arrived at by the veterinarians referred to above was that any case of tetanus that recovers under

*Anti-tetanic serum is the proper term. There is no tetanus *anti-toxin* used, or on the market.

the serum treatment would also have recovered under other treatment. This conclusion was arrived at after a more thorough trial than could be generally accorded a remedy. The veterinarians were not hampered through considerations of expense; they had hospital facilities, ample assistance; in fact, everything was favorable for the serum, if it had a trace of merit. In the light of this and later experience with anti-tetanic serum, I can only condemn it as a curative agent. (As a prophylactic, I am just as positive that it has great value.)

I use anti-tetanic serum now in my tetanus cases, but I use it in a different manner. I give two or three large doses of it in the beginning, from 2,000 to 3,500 units at a dose. I expect in this manner to get no results of a curative nature. What I have in view in administering the serum in this way is an effect that might be described as a "shock absorber." I expect this quantity of anti-tetanic serum to hold the toxins in abeyance, to dilute them, at least long enough to enable the patient to assemble all his bacteriolytic faculties. In plain words, I give him an opportunity to "catch up" with the infection. This is all that can be expected of anti-tetanic serum, all that can be claimed for it. And even this is only a theoretical merit; it is not apparent.

It is a sorry fact, but a true one, that for almost our entire knowledge of the adaptability of various biologic agents, we must rely on the manufacturers of such agents. Only on this account do some of them continue to be exploited and used. Anti-tetanic serum for curative purposes is one.

Other forms of treatment. The treatment of tetanus has in the past taken the form of fads. We have gone through courses of treatment with phenol, magnesium sulphate, tallianine, and, just now, we are in the grasp of the lobelin fad. All of these, as well as most of those

not named and including the *Passiflora* treatment recommended by the author since 1905, are truly nothing but symptomatic treatments. In all truth and candor, from the clinical standpoint (which is always and only the viewpoint of the practitioner), we must confess that whatever we have of value in the treatment of tetanus is distinctly of a symptomatic nature. We try our best to control the disease and its symptoms; but we are not remarkably successful even in this.

I will briefly discuss the most usual forms of treatment in vogue for this disease at the present time.

Phenol treatment. Commonly termed the carbolic acid treatment. This form of treatment has survived longer than any of the other forms. It consists of the hypodermic injection of from two to five per cent solutions of carbolic acid in water. Sometimes a little glycerin is added. From thirty to sixty mil. are injected once a day, or every other day. Some veterinarians inject it at intervals of three days. Many cases recover with the use of this treatment, but so do they with the use of other treatments. The most noteworthy disadvantage in connection with this method is the formation of abscesses at many points of injection. The most noteworthy feature of an advantageous nature is its low cost and the ease of application.

Magnesium sulphate treatment. This treatment is quite as ancient as the carbolic acid method, but it has recently been again brought out as a new treatment. Various amounts of a solution of magnesium sulphate are given by hypodermoclysis. No standard strength or quantity of solution has ever been established for veterinary use. In my opinion this treatment has less ground for existence than any of the others.

Lobelin sulphate treatment. The use of lobelin sulphate in the treatment of tetanus is quite recent in origin.

At first used only as an adjunctive agent with serum, it is at the present time being used by some practitioners as the entire treatment. Recoveries have, of course, occurred and have been reported in veterinary journals. Its most ardent advocates up to this writing, however, have been the pharmaceutical houses that have it to sell. It is to be given in doses of one-twentieth to one-tenth grain, hypodermically, at least once each day until the symptoms are receding.

Passiflora incarnata treatment. The use of fluid extract of *Passiflora incarnata* in the treatment of tetanus in horses is, so far as I know, original with Dr. Wendell A. Knight, formerly state veterinarian of Texas. While Dr. Knight's cases were of the mild type seen in the South, I can say from personal experience that equally good results are obtained with this agent in the more marked cases seen in Northern horses. I have used this treatment with more than ordinary success in cases of tetanus in extremely high altitudes during cold months of the year. I make mention of this because the disease is seen in its most active form in such regions.

The one great disadvantage that this treatment has is in the fact that its administration is troublesome. It must be given with the dose syringe. One ounce of the fluid extract is given, just as it is, every three hours until the patient is free from excitement and marked spasms. After this it is given just often enough to keep up this effect. Tetanus cases treated with *Passiflora* recover more quickly and come out of the attack in better condition than they do with any other form of treatment with which I am familiar.

If I should be asked to state to the best of my professional knowledge what I would do were I to outline a treatment for tetanus I would say, "Use heat and Pas-

siflora.” In my opinion tetanus symptoms are milder in a Southern climate merely because of the climate. I do not believe it is so because the tetanus infection in the South is less virulent, or because Southern horses are not so susceptible to the action of its toxins. I believe that the climate is entirely responsible. In some parts of the South the natives take advantage of the beneficial effect of heat on tetanus symptoms. When a horse develops tetanus he is at once confined in a small, almost air-tight enclosure having a tin or sheet-iron roof. The openings are draped with sacks or blankets to keep out the light. This is all the treatment the patient gets. But it is a real treatment, for when a high-noon Southern sun gets action on a tin roof, a Turkish bath establishment isn’t in the running. And it is nothing out of the ordinary for a complete recovery to take place in so short a time as two or three weeks with no other treatment than heat, darkness, and quiet.

I was on one occasion forced to use this treatment in one of my patients while I practiced in the South. The subject was a fat, chunky, 1,000-pound stallion. Each day at noon when I entered the little shed where he was confined, I feared the heat would overcome him. In less than three weeks he was again in harness, and his case had been rather a severe one, too.

Veterinarians who contemplate the erection of a hospital should include in their plans a veritable “dungeon” stall that can be heated by steam until its temperature registers 100° F. or more. Tetanus cases only should be treated in such a stall. If in addition to this the patient is given the Passiflora treatment, the results, I am sure, will be most gratifying.

Regarding the general handling of tetanus cases, it is only necessary to emphasize the enforcement of quiet and the provision of darkened quarters. Attendants should

not be changed; the same attendant should administer all medicines, feed, water and give other care. If the site of infection is evident on some portion of the patient's body, it should be treated antiseptically if it can be done without unduly worrying and exciting the subject. I have heard of practitioners who cast a tetanus patient to treat the nail wound in the foot when they can yet find it. This shows about the same degree of common sense as is exhibited in the use of an operating table to save cocain. If it is impossible to treat the wound without aggravating the tetanus symptoms, leave the wound alone. Very little good results from local treatment of the case under any conditions, and unless the wound is in a region where it can be treated easily, I would advise non-interference.

Much remains to be accomplished along the lines of a uniformly satisfactory treatment for this disease. The most that we can do with our present knowledge of this condition is to make the patient as comfortable as possible; in other words, treat the symptoms to the best of our ability.

Before closing the discussion of the treatment of this disease it can do no harm to repeat what has been said about the value of anti-tetanic serum as a prophylactic agent. Every practitioner of veterinary medicine and surgery should make it a routine practice to inject a prophylactic dose into all patients presenting injuries of a character favorable to tetanus infection. No treatment of a puncture wound is complete, either from the standpoint of judgment or law, unless it includes a prophylactic dose of anti-tetanic serum. For sure prophylaxis a second dose should be given eight days after the first one. In localities where the clientele does not readily submit to suggestions on the part of their veterinarians, the practitioner is in duty bound to inform a client of the risk

which goes with a puncture wound when anti-tetanic serum is not used.

ANTHRAX

Anthrax, called charbon in the south, is a highly infectious disease, which frequently runs a hyper-acute course and has a very high mortality rate. It results from infection with the *Bacillus anthracis*, a relatively large micro-organism that is not very tenacious of life.

Anthrax appears in various degrees of severity, from a hyper-acute form commonly termed apoplectiform anthrax, to an ordinary, sub-acute, less fatal form.

Anthrax makes its appearance most commonly in various parts of the country, in what are known as anthrax districts, where it usually assumes an enzootic character. These districts are almost always low-lying sections, such as river bottom land which is subject to annual inundation. The *Bacillus anthracis* lives normally in the soil of such sections. Horses feeding on pasture are most frequently affected. On occasions the disease will, however, make its appearance even among stabled animals.

The manner of infection is usually by way of the alimentary tract, resulting from the ingestion of infected fodder. The period of incubation is from forty-eight hours to ten days. Young animals are slightly more susceptible than aged ones.

Symptoms. Hyper-acute, or apoplectiform anthrax. The course of the disease in this form is usually so rapid and so violent that death may come within a few hours after the first sign of sickness. A horse which is apparently in perfect health suddenly stops feeding, shows a few moments of excitement and goes down in a few minutes. The animal lies in a stupor, breathing stertorously. Sanguinous fluid issues from all the natural openings,

and the visible membranes appear cyanotic. In various parts of the body edematous swellings begin to form rapidly, convulsions set in, and death comes in the second or third hour. In some cases death occurs before any swellings are formed. The dead animal begins to bloat almost immediately after death. Rigor mortis does not become fully established in carcasses of animals dead from anthrax.

Ordinary, or subacute forms. In this form the disease begins with a very high temperature which reaches 106° or 107° F. within an hour or two. The horse appears at first slightly excited, but soon shows symptoms of great depression. It stands fixedly in one spot until it either lies down or falls in an attempt to change its position. Labored respiration begins, the mucous membranes appear almost cyanotic, and stupor rapidly develops. Edematous swellings now develop on various parts of the body; under the neck along the trachea, flank region and parotid region. Colicky pains sometimes appear. There may be bloody scours, with protrusion of the rectal membranes; the eyes, nostrils and the mouth discharge bloody secretions. The urinary secretion is usually hemorrhagic. Death comes in convulsions; the horse dies usually as though he were being strangled. The entire course of the disease in this form is from one to two days. Recovery is very rare.

Differentiation. Anthrax is not easily mistaken for any other disease, but because of the high mortality rate it is not difficult to obtain material for proving the diagnosis, and this should always be done. In the apoplectic form an ante-mortem diagnosis is hardly ever made because the patient usually succumbs before the veterinarian is on the ground. In making a postmortem examination of carcasses in anthrax cases, every precaution should be taken by the veterinarian against infection.

No part of the carcass should be touched with the bare hands; heavy rubber gloves should always be used.

The diagnosis can be made positive postmortem upon the following findings:

1. Multiple hemorrhages into the connective tissue are seen immediately the integument is removed.

2. The blood appears tarry-black in all the vessels and it does not coagulate.

3. The edematous swellings, when cut into, are jelly-like in consistency.

4. The lymphatic glands, when cut into, have a "strawberry" cast; they appear very much like the cut surface of a strawberry.

5. The spleen is very much enlarged, and its parenchyma is easily broken down.

6. Rigor mortis is only partial, or even entirely absent.

In addition to these cardinal lesions there are hemorrhagic areas in nearly every organ, serious infiltration is evident in nearly all tissues, and in some cases there are even necrotic areas. The diagnosis can be clinched by microscopic examination of blood smears; the large bacilli can be seen under low power, formed in short chains.

Carcasses and all excreta of horses dead from anthrax should be burned. The infective agent exists in the alvine discharges and other excreta.

Treatment. The treatment of anthrax in horses can be summed up in one word—*prevention*. Anthrax is nearly always fatal in horses. While cattle oftentimes recover from ordinary anthrax infections, horses nearly always die. The whole hope lies, therefore, in preventive vaccination, a proceeding that is quite generally recognized.

Recent developments along the line of curative treat-

ment are those having to do with serums. Very promising results have been obtained so far by Eichorn and others, and the treatment of animals afflicted with anthrax will no doubt be made practically possible with immune serum in the near future. The present disadvantages, high cost, etc., will be overcome in some manner.

RABIES

This disease in nearly every instance runs a rapid and fatal course in the horse. It is characterized by symptoms of great excitement, violent exhibitions, paralysis and death. Rabies is a disease that has been the subject of much controversy in regard to its true character in animals. Some pathologists of repute have even held that rabies, as a disease, does not exist in the lower animals. The greatest confusion in arguments and theories along this line originates from the fact that an effort is usually made to make rabies conform to the manifestations of hydrophobia as seen in human beings. An intelligent conception of rabies is only possible when all connections which it is presumed to have with hydrophobia are ignored *from a clinical standpoint*.

It is now quite generally held that the bodies of Negri are the etiological factors in this disease. At first looked upon as coincidental to the pathological changes occurring in the brain as a result of the disease, they are now accepted as the true causative agents.

The period of incubation in this disease is from one to ten weeks; an acceptable average can be placed in practice at three weeks. The period of incubation is shortest when the site of inoculation is on parts of the head. Belief is no longer held in exceptionally long periods of incubation. Cases of the disease which occur many months, or even years, after *known* exposure, are no doubt due to exposure or inoculation which was contracted later in an unknown manner.

Horses are infected most frequently as a result of being bitten by dogs afflicted with the disease; more rarely from bites of members of their own species. Relatively speaking, the horse is a very rare subject of this disease. The percentage of animals which develop rabies as a result of

being bitten by other infected animals is comparatively small; I would place it at less than 25 per cent in the case of horses. (A capable veterinary pathologist of my acquaintance held that the infection is never transmitted in the bite of a horse. It was later possible for me to explode this theory in my own practice, to his entire satisfaction.)

Symptoms. Almost without exception rabies in the horse takes what is known as the furious form. Usually a history can be obtained that connects the development of the symptoms with a dog bite or, occasionally, a bite from a horse. Although many writers report itching in the region of the bite at the first symptom, I can not say that this always holds true. In some cases in my practice this early symptom was entirely absent. In my experience the most constant and reliable early symptom has been a nervous, spasmodic retraction of the commissures of the mouth. Simultaneously with this, or very soon thereafter, there is ptosis of one eyelid, sometimes so slight that it is barely noticeable; but it is there. With this there is, in quite a few cases, a marked difference in the size of the pupils. I have been able to demonstrate one or all of these symptoms very early in the course of this disease in every instance coming to my attention.

Soon after the manifestation of the foregoing symptoms the horse becomes restless; the ears are moving almost constantly, and pawing, neighing, defecation, urination and other acts are performed repeatedly in rapid succession. There are contractions of the skin, which occur spasmodically, in various parts of the body. (In not a few cases there are symptoms at about this stage which resemble an attack of acute indigestion. This phase does not remain long, however; the colicky pains gradually assume greater violence and end in maniacal outbursts of ferocity.) If the horse is now offered a drink

of water, it will be noticed that dysphagia has developed. When this symptom becomes fully established there is very active salivation. The horse, in the general run of cases, requires from eight to twelve hours to reach this stage in the course of the disease.

From this on there are symptoms which point quite plainly to the nature of the disease. Although the patient may have been normally very docile, he now assumes vicious tendencies. When approached, he attempts to nip, strike, or kick his attendant. This vicious tendency becomes rapidly more marked; the animal plunges about, rearing, striking and biting. Towards the end the animal often attacks its own flesh with its teeth, tearing out pieces of hide and muscle. Progressive paralysis now develops, the animal goes down and dies in convulsions. Death simulates that resulting from asphyxiation. The entire course of the disease usually consumes from twenty-four to forty-eight hours. While some writers have reported recoveries from this disease, I do not believe that recovery ever takes place.

The treatment of rabies is impractical and would no doubt be useless, if attempted. Prophylactic treatment, according to the method of Pasteur, is a reliable preventive if resorted to promptly after the horse has been exposed to infection. The veterinarian's whole efforts, when handling a case of rabies in a horse, are aimed at:

1. Safeguarding other horses in the stable by so confining the patient that he will be practically harmless.
2. Instructing attendants or owners, who may have been bitten, in the proper steps towards preventing the development of the disease in their own persons.
3. The employment of the Pasteur treatment in horses that may have been injured by the patient.

1. As soon as rabies is suspected the horse should be securely tied and, if possible, boxed in. This must be

done with the expectation that the patient will become unmanageable within a few hours, and that great harm may result if the patient should run amuck.

2. Persons that have been bitten should be urged to take the Pasteur treatment without delay. Therein lies their only hope of escaping a horrible death. While only about two per cent of those bitten by horses have (on record) developed hydrophobia, I advise against taking a chance. On this point the veterinarian is sometimes approached by persons who have accidentally come to some injury about the patient for variable periods of time before the animal developed symptoms. They desire to know whether they should submit to the Pasteur treatment. Persons who received wounds through some act of the horse in question, accidentally or with vicious intent on the part of the horse, ten days or less before the horse showed symptoms of rabies, should take the Pasteur treatment. Pathologists have found the saliva infectious eight days before symptoms were evident.

3. If the rabid horse has attacked and actually bitten other horses in the same period of time or during the active stage of the disease, they should be isolated for sixty days. If no symptoms have developed by that time they may be presumed non-infected. If the bitten horse, or horses, should be of great value, the Pasteur treatment should be begun at once.

Cases of rabies occurring in mares with foal at side are cause for isolating the colt for a period of sixty days, for the reason that the disease has been conveyed, in some cases, through the milk. The thorough disinfection of the stall in which the rabid animal was confined is imperative. While infection in any other manner than through a bite is almost unknown, it is possible that the disease could result from wounding of a part of the body with objects contaminated by saliva or other materials.

TUBERCULOSIS

Tuberculosis seems to be a very rare disease of horses. Whether this is true in fact, or whether it is merely apparent because it frequently goes unrecognized, is hard to say. Those cases that have been reported have been diagnosed postmortem. The symptoms produced by the disease do not seem to be very characteristic, and can easily be mistaken for the manifestations of glanders.

Tuberculosis is suggested by such symptoms as chronic cough, nasal discharge, respiratory impediment resembling pulmonary emphysema, inanition and weakness. Enlargement of lymphatic glands, irregular temperature, and otherwise unexplainable functional disorders of a chronic type must also be considered as having a possible tuberculous origin.

When the disease is suspected the animal should be subjected to a test with tuberculin. It is possible that the routine testing of horses with tuberculin, in a manner similar to such tests in cows, would show that the disease does occur with greater frequency than is generally conceded. If the horse does, on the other hand, possess such marked natural resistance to this disease as the scarcity of recorded cases would now indicate, the identity of the secret of this immunity should be investigated. Its recognition might be of incalculable benefit in the prevention and treatment of tuberculosis in other species.

DOURINE

Dourine, also called *mal du coit*, is a disease of horses that interests the practicing veterinarian chiefly from the standpoint of diagnosis, its treatment having been discountenanced by legal enactment. Dourine is probably the only true venereal disease of which horses are the subjects. It is characterized by local manifestations in the generative organs, with subsequent development of various degrees of paresis and disturbances of nervous function generally.

The infection is transmitted almost entirely in coitus. The infecting agent is the *Trypanosoma equiperdum*. Trypanosomic infections are usually associated with hot climates, and the fact that dourine affects horses in cold climates also stamps it as one of the rare infections occurring in the United States.

Because of the infrequent occurrence of this disease in the United States, there were, until the last occurrence in the Northwestern states, comparatively few veterinarians in this country who were considered qualified to make a diagnosis of dourine clinically. I recall an instance that occurred in the Southwest, in the Mesilla Valley, in which even one of these qualified experts confused a botryomycotic process on the sheath and penis of a stallion with dourine. In another instance that occurred in my experience as assistant to the state veterinarian of Texas, a series of experiments and postmortem examinations, which were made chiefly for the purpose of establishing a period of incubation for quarantine enactment, resulted in blank. One of the men in charge of the work was presumed to be an expert in the diagnosis and postmortem identification of the disease because of previous experience with it in other lands. From what I

saw happen at this meeting of experts, I came to lose all faith in them.

Certain parties, whose identity must remain secret, made it an issue to fool the expert diagnostician from the very beginning, and he remained "fooled" throughout the experiments. Since that time, however, the disease has become better understood in most of its phases, especially during the past four or five years; and its diagnosis is frequently made, clinically, by many practitioners. The period of incubation may be as short as one week. Under some conditions it may be six weeks.

The symptoms as I give them here are as observed by me personally in two cases in the stallion and one case in the mare.

Stallion 1. This was a pure-bred imported Percheron horse five years of age. The first symptom calling attention to his case was frequent urination. This began two or three weeks after he had served the mare whose case is hereinafter reported. When the penis was protruded in the act of urination it was noted that the urethra was very prominent, appearing congested and flattened laterally around the orifice. At the same time an edematous swelling began to appear in the sheath, which progressed quite rapidly and involved the true prepuce to such an extent that a degree of paraphimosis resulted after a few days. The swelling in the sheath was smooth and doughy and characteristically edematous. Within a day or two after the paraphimosis developed the glans penis became the seat of a process simulating a moist eczema.

On the anterior face of the glans, close to the urethral orifice, appeared what can best be described as a chancre, which broke down at the end of four or five days and formed a deep ulcer. Other similar chancres appeared on various parts of the penis during the course of the next week. Some of these healed over with rapidity.

Where they had been, a white spot remained. Others refused to heal even under treatment, especially those near the urethral orifice.

When the disease had been in existence for two or three weeks weeping areas appeared on the neck and in the crural region. These areas were from the size of a nickel to a dollar and appeared at first as a moist, weeping spot slightly elevated above the surrounding tissues. Within a few days this took the form of an ulcer, which healed kindly. The area was depigmented when healed. On various parts of the trunk, and also in the gluteal region, this stallion showed the typical whip-lash edema. This edema was migratory, appearing in different parts at times. At the end of nearly two months the most marked of these early manifestations had disappeared.

There now began to be shown a slight lack of coördination in the posterior limbs, beginning at first very much like a case of "shivering." Within another four or five weeks this had progressed to a real "wobble," so that the horse had difficulty in walking in a straight line.

Stallion 2. This was a rather small Clydesdale grade, nearly ten years of age when he came to my attention. From the history given by the owner he had passed through the early manifestations in a typical manner. When I examined him he was so "wobbly" on his hind legs that in attempting to mount a mare he fell flat several times, rising each time with great difficulty. The glans was the seat of several depigmented areas, and the urethral orifice was deformed. A microscopic examination of the semen showed only a few live spermatozoa.

Mare 1. This mare was known to have had dourine for several years, having infected two different stallions belonging to the same owner. When I saw her she was in a pitiable state of emaciation. There was a "gluey," mucilaginous collection in the lower commissure of the

vulva; the clitoris was very prominent and hypertrophied. One side of the vulva and spotted areas on and near the clitoris were depigmented. Although very emaciated and lacking good coördination of movement, this mare appeared to be a nymphomaniac, accepting the service of the horse promiscuously.

Some writers claim to have seen the development of the secondary symptoms—lack of coördination, etc.—without any marked local lesions primarily.

Microscopic diagnosis should be called for in all suspected cases of dourine. The parasites exist in the blood and secretions. They are said to disappear very rapidly after the death of the animal; postmortem microscopic diagnosis would therefore be unreliable. Antemortem, a blood smear is to be made, using Romanowskie's stain.

With the present system of eradication in use by the Bureau of Animal Industry against this disease, dourine will ultimately be completely eradicated. The systematic inspection of imported horses of European origin will do much to prevent the bringing in of new cases. At the present time the disease seems to be well controlled in the United States.

GLANDERS

Glanders of horses is an infectious disease that appears most commonly as an insidious, chronic manifestation of lesions involving chiefly the respiratory tract. Less frequently it appears as a local disease in the skin and regional lymphatics, and still less frequently it appears in an acute form. It is a disease that is generally held to be incurable and against the treatment of which the various states of the Union have enacted regulations.

Glanders, like tuberculosis, seems to be a disease that follows the trail of civilization; in fact, formerly many able veterinary pathologists considered glanders to be the equine form of a tubercular process. It is now a proved fact, however, that such is not the case, and that glanders is a distinct disease caused by an infection with the *Bacterium mallei*. The disease is said to be unknown in regions where modern commerce and the interchange of horses among alien peoples are not actively indulged.

Like dourine, glanders is important to the practicing veterinarian chiefly from the standpoint of diagnosis. As a result of active regulation and modified methods of eradication this disease is not nearly so prevalent as it was twenty, or even ten, years ago. In former times glanders frequently made its appearance among bodies of horses in a form that could almost be termed an epizootic. No doubt an occurrence of this sort was possible only because of lax quarantine and improper prophylactic measures. The universal recognition of the true infectious character, and the most common sources of contamination, is having the effect of an ultimate total eradication of this disease.

Symptoms. In discussing the symptoms of glanders it becomes necessary to classify the disease into three

forms: (1) Chronic glanders; (2) Acute glanders; (3) Farcy, or glanderous lesions in the skin.

1. *Chronic glanders.* This deserves to be discussed first because it is by far the most common form. Chronic glanders appears so insidiously that, when marked symptoms really do become evident, the horse has already become generally vitiated by the infection.

Probably the earliest objective sign is a nasal discharge. This may be uni- or bi-lateral, and is at first a thin, viscid fluid; later it becomes greenish, muco-purulent, and is occasionally streaked with blood. This streaking is the result of ulceration in the Schneiderian membrane. Some of these ulcers are visible to the eye near the anterior portion of the nasal septum, appearing as clear-cut, rounded, saucer-shaped depressions. When these heal, which they frequently do spontaneously, they leave a star-shaped, pearly cicatrix.

The submaxillary lymphatic glands in chronic glanders are enlarged in a peculiar manner. The individual glandular lobules seem to become isolated from one another and then enlarged. This gives the mass the feeling of a layer of small peas. This glandular alteration and enlargement is not painful under pressure, and appears to be adherent to the overlying skin.

As the case progresses, the nasal discharge becomes more plentiful. At times it may be malodorous, from necrotic processes in the nasal septum or in the sinuses of the head. The discharge now adheres to the nostril, making the nose appear filthy and smeary. As a result of confluent ulceration, there may occur perforation of the septum nasi. If the horse is now carefully examined there will be found an enlargement of nearly all the superficial lymphatic glands. In the lungs changes can be noted by auscultation and percussion. The horse becomes emaciated, has a general unthrifty appearance,

and is finally unserviceable. Localized suppurative areas in the region of lymphatic glands may appear eventually.

Acute glanders. Acute glanders is, luckily, very rare. Its diagnosis, clinically, presents difficulties. An attack of acute glanders does not differ materially from an attack of strangles in a serious form. Possibly the symptoms in acute glanders are slightly more vicious from the very beginning.

There is an occasional diagnostic feature, namely, the appearance of blood in the nasal discharge. A suspected case of strangles should be considered as glanders whenever the nasal discharge is streaked with blood. This symptom does not occur in all cases of acute glanders, but when it does occur, it is quite reliable.

Now and then a case of chronic glanders may become acute as a result of an overwhelming elaboration of toxins, to which the already lowered resisting power of the horse offers but little impediment. Such an occurrence is not so difficult to recognize as a primary acute attack of glanders. It is also well to suspect as acute glanders any malignant form of strangles, or what appears to be strangles, in horses that are over six years of age.

Farcy, or localized glanders. When a glanders infection confines its activity to a circumscribed area of superficial tissues, it is called farcy. The most frequent localization of a glanders infection is in a pelvic limb. The manifestation at first somewhat resembles "grease-heel." There is fullness and thickening of the integument, and one or more areas of ulceration. These areas begin as a "bud" or node; they break down and remain as a crater-like area, of a mouse-eaten appearance. Some may heal over, while at the same time others appear; or subsequently others make their appearance higher up on the limb, preferably in the seat of the popliteal lymphatics. Cord-like "runners" or swellings radiate from the ulcer

in the subcutem. The limb eventually becomes a mass of ulcers and scars, becomes thickened and indurated. The horse succumbs as a result of septicemia, or the case becomes one of generalized glanders, which terminates fatally eventually.

Diagnosis. The diagnosis of glanders can be made positive with the use of mallein, and while a clinical diagnosis is frequently possible, mallein should always be used to verify the clinical diagnosis. The *modus operandi* of malleinization is practically identical with the application of tuberculin in tuberculin testing. Within the past few years the ophthalmic mallein test has become official, being used by the Bureau of Animal Industry in official tests of horses for glanders.

The ophthalmic method is a great improvement on the subcutaneous method, being more simple and more rapid. It consists of depositing a quantity of mallein in the conjunctival sac by means of a camel-hair brush, or dropper. The presence of glanders in the horse gives a reaction in the form of varying degrees of conjunctivitis with purulent exudate or lachrymation. The eyes of horses that are free from glanders remain normal, or, at most, lachrymate only slightly.

Treatment. Glanders is never treated in the United States. The infected animal is to be destroyed in conformity to the mandates of the legislative body in the state in which the case occurs. Most states indemnify the owner of the horse to a certain extent.

Without a doubt the most important factor in the control of glanders has been the compulsory mallein testing that has been imposed by most of the states on horses coming into their boundaries from other states. This, in effect, virtually gives one state a sort of check upon another. Another feature that has had great bearing on the control of the spread of glanders among horses is the

increase in the number of graduate veterinarians and the decrease in the number of non-graduate practitioners in all states.

It was not an uncommon occurrence for a non-graduate to treat a case of glanders under the supposition that it was nasal gleet. Cutaneous glanders or farcy was usually treated as a case of chronic grease heel by most empirics. It is possible for glanders to be entirely eradicated in the course of time by the systematic use of mallein if, at the same time, empirical practice of veterinary medicine is prohibited.

PART III

POISONINGS

Accidental poisoning of horses with drugs, chemicals and other substances occurs occasionally. In some rare instances intentional poisoning is the result of feuds and neighborhood quarrels.

In the following pages is given a list of the most usual forms of poisoning, together with their symptoms and their treatment. The recognition of the effects of either an accidental or an intentional poisoning quite often depends upon the understanding which the veterinarian has of the more remote effects of the various poisons.

The veterinarian must rely also to a great extent upon his powers of observation; some of the effects of some poisonous matters simulate the manifestations of certain diseases. The successful handling of these cases depends upon the veterinarian's alertness in detecting the true etiological factor; a clue to the situation is at times quite apparent if the veterinarian is observant. On the other hand, the practitioner should never be rash in giving out his opinion that the patient has been poisoned. It is always best to add a proviso to such an opinion so that, should the diagnosis be in error, those concerned will not be unnecessarily aroused. A reckless diagnosis of intentional poisoning has, on a number of occasions, led to murder.

The veterinarian is between two fires here. To protect himself and his professional reputation, he must acquaint the client with the fact that his horse has been poisoned, and yet he must do it in such a manner that no

one will come to an untimely end as one result of the occurrence.

On general principles, the diagnosis can always be made "ptomaine poisoning" until some one forces the issue. The treatment is strictly the veterinarian's own business and he will, of course, treat a case of arsenic poisoning as it should be treated, although to save his client an unpleasantness he may diagnose the case "ptomaine poisoning." A good veterinarian must also be a good man.

Coal Oil or Kerosene Poisoning

Poisoning of a horse with coal oil is a very common accident in regions where this agent is used by the laity for the treatment of colics. It is usually the result of giving an over-dose; on occasions a quart may be given a horse with the object of "curing a colic."

Symptoms. There is great depression. The pulse is irregular, thready, or it may even be imperceptible. The respirations are short and hurried, the extremities are cold, and the skin feels clammy. If a fold of skin is ridged up it returns very slowly to normal position. Temperature is subnormal. The marked odor of kerosene removes any doubts about the diagnosis.

Treatment. Give stimulants, such as aromatic spirits of ammonia, brandy or whiskey. Provide the patient with warm quarters and apply blankets. Employ some one to rub the extremities. Remain with the patient and administer to it until the respirations approach normal and the pulse is improved.

Death from kerosene poisoning comes very suddenly when a lethal dose has been given, and a lethal dose does not necessarily mean a very large dose if the horse happens to have a heart lesion of some kind.

Poisoning with kerosene frequently results in perma-

nent sequelæ, such as pulmonary emphysema, roaring, and vertigo.

Carbolic Acid Poisoning

Poisoning of horses with carbolic acid is usually accidental, by mistaking the acid for some harmless drug, placing carbolic acid in mis-labeled bottles, or misconception of the effects of the acid when given internally.

Symptoms. The mouth shows white patches on the mucous membrane where the acid came in contact with it. Contracted pupils. The horse is listless, has muscular tremors, lack of coördination when forced to move. There is salivation, dyspnea, weak and rapid pulse, and usually a subnormal temperature. If the dose was lethal, collapse and deep coma precede death. The urine gives off the odor of the acid.

Treatment. Alcohol is the antidote for this poison; give it freely. The best physiological antidote to be given in conjunction with the alcohol is atropin. If collapse threatens, use strychnia. Avoid giving anything oily. The direct chemical antidotes for the treatment of carbolic acid poisoning are the sulphates of magnesium and sodium. They form sulpho-carbolates. The best results are, however, gotten from alcohol.

The three important items to remember in the treatment of carbolic acid poisoning are:

1. Alcohol.
2. Atropin.
3. Strychnin.

In districts where alcohol is not easy to obtain use magnesium or sodium sulphate. If neither alcohol nor one of these salts can be obtained, use vinegar. It is said to be almost as valuable as alcohol in this form of poisoning. The alcohol has, however, the additional action of a stimulant, an action that is by no means superfluous here.

Aconite Poisoning

Poisoning of horses with aconite results from overdosing by laymen in the treatment of colic, fevers, colds, or, in fact, in most any disease. Aconite has a very wide therapeutic field in the minds of laymen. Juggling with the official strength of the tincture of aconite, reducing its strength from thirty-five to ten per cent, may have resulted in some cases of poisoning by the giving of a new-strength dose of old thirty-five per cent tincture. The change made did more good than harm, however, and without question automatically prevented the poisoning of many horses by the use of this drug in the hands of laymen. I have known of instances where half-ounce doses of tincture of aconite were given repeatedly.

Symptoms. Great weakness. Pulse slow and small. The horse seems to be blind. There is a cold, clammy sweat. Champing of the jaws, producing a foamy salivation, and "a peculiar clicking sound in the pharynx." The horse is bloated; belches gas. The muscular weakness progresses if the dose has been lethal, and the animal goes down. Motor paralysis, loss of sensation in the skin, rapidly falling temperature, cardiac and respiratory paralysis, and death. The horse remains conscious to the end.

Treatment. Use the stomach tube and wash out the stomach. Give nitro-glycerin hypodermically. If this is not at hand, give freely of other rapidly acting stimulants. *Keep the horse warm*, and avoid unnecessary movement. Treat the case as though you were momentarily expecting the horse to die from heart failure.

Cocain Poisoning

The toxic effects of cocain at times become evident to an alarming degree after the injection of cocain solutions for local anesthetic effect. Other cases of poison-

ing occur in a race track practice, where cocaine is sometimes administered for its exhilarating effect.

Symptoms. Great excitement, restlessness, muscular twitching, free perspiration. Frequently simulates the early stages of azoturia, with violent muscular trembling. The pupil is widely dilated. Lethal doses result in delirium, and produce death suddenly by cardiac exhaustion.

Treatment. Confine the horse in a quiet place, away from noise and excitement. The best effects are obtained from a full dose of chloral hydrate, given well diluted. If chloral hydrate is not to be obtained, give a full dose of morphin hypodermically.

Arsenic Poisoning

The toxic effect of arsenic results from the accidental eating of preparations intended for use as an insecticide; from the administration by laymen of excessive doses of arsenic in the treatment of various diseases; from the eating of vegetation contaminated by fumes from ore smelters; and, lastly, as a result of malicious, willful poisoning.

Arsenic is the favorite poison of the criminal, and horses are occasionally poisoned with it by cruel and revengeful persons.

Symptoms. Arsenical intoxication, or chronic arsenic poisoning, is seen quite commonly in horses pastured near smelting works, where the grass becomes coated with arsenical compounds from the smoke and fumes. The horses so affected become emaciated, appear "mouse eaten," and drink great quantities of water. There is general soreness in motion, as a result of the development of neuritis; as a result of articular inflammations, there is great swelling in certain joints and marked lameness. Finally, necrosis occurs in osseous tissue. The teeth

appear almost black in color, become loosened and may even fall out. Eventually complete paralysis results.

Acute Arsenic Poisoning. Acute symptoms of arsenic poisoning usually take the form of a severe gastro-enteritis. The horse appears near collapse, trembles and suffers intensely. There is vomition and active purgation, occasionally blood is passed. The urine is also bloody when passed, but may be retained. Before death there is edema of dependent parts. The animal dies in coma.

Treatment. In chronic arsenical poisoning the cause should be removed and the horse given a prolonged course of potassium iodid to hasten the elimination of arsenic.

In acute poisoning the stomach tube should be used to evacuate the stomach. The direct chemical antidote is hydroxid of iron. To be effective this must be fresh, and given in from one to two-ounce doses every fifteen minutes. If this antidote is not available lime water should be given in generous potions. Oils and demulcents should also be given. To relieve the intense pain which the animal suffers, large doses of morphin are to be given hypodermically.

Acute arsenic poisoning in horses usually results fatally, unless the chemical antidote is administered early enough to prevent the absorption of a lethal quantity of the poison. Even then the case will frequently terminate in death from the effects of a violent gastro-enteritis.

Poisoning with Mineral Acids

Poisoning of horses with one of the mineral acids occurs now and then accidentally, as a result of mis-labeling the contents of bottles or mistakes in interpreting directions. The most common acids used are hydrochloric, sulphuric, and nitric acid.

The symptoms of poisoning with any of these acids are soreness in the mouth, dyspnea, colicky pains. The

dyspnea results from swelling in the pharynx as a result of burns from the action of the acid, and may be so serious as to make tracheotomy necessary.

The burns in the mouth are:

Black from sulphuric acid;

Tan or yellow from nitric acid;

White from hydrochloric acid.

Treatment of poisoning with these acids is by the use of alkalies, such as magnesium sulphate, sodium bicarbonate and emulsions of soap. Water must not be given.

If called early enough, use the stomach tube. Stimulants are to be given hypodermically to overcome depression.

Gelsemium Poisoning

In sections where gelsemium is used by the laity in the treatment of colics it is a common occurrence to be called to cases of poisoning with this drug.

Symptoms. The horse appears as if asleep in a standing position. If made to move he may fall. The pulse is very small and weak, occasionally skipping a beat. The pupils are dilated; the lower lip hangs loosely pendent. Respiration is labored.

Treatment. Give large doses of aromatic spirits of ammonia or whisky. Strychnin may be given hypodermically in small doses. Use friction and slapping; blanket warmly. When the horse becomes somewhat aroused give gradually increasing walking exercises.

Acute Lead Poisoning

Acute lead poisoning occurs in horses now and then as a result of the accidental or vicious ingestion of paints. Because of the gravity of the symptoms produced and occasional fatal consequences the condition deserves mention in a work of this kind.

Symptoms. Within a few hours after the substances have been ingested slight colicky pains develop. These are followed by a profuse diarrhoea, which is at first composed of fluid feces, later of clear liquid. The evacuations emit the odor of the particular lead-containing vehicle, usually oil. At the end of from twenty-four to forty-eight hours the animal appears in a state of collapse. The pulse is imperceptible, the integument cold and **clammy**. There are in some cases, crepitating areas under the skin, on various parts of the body. The horse now prefers to stand fixedly in one spot. When forced to move, the action resembles that of laminitis.

If proper treatment is not promptly given, death comes at the end of two or three days after the lead-containing substance has been eaten. Before death occurs there are edematous areas in the extremities, and varying degrees of paralysis in some groups of muscles. The symptoms described appear in various modifications depending upon the amount of lead contained in the ingested substance.

Treatment. If the animal comes into the veterinarian's hands within a few hours after the poison has been eaten, the stomach tube may be used. From one to three ounces of magnesium sulphate is to be administered in watery solution every twenty or thirty minutes until a pound has been given. This is the most satisfactory antidote, chemically. Later a mixture of tincture of capsicum one dram, tincture of opium one ounce, is to be given to check the purgation. If necessary this dose may be repeated in an hour. The animal should be blanketed and quartered in a warm place. Exhaustion is counteracted with alcohol, brandy, port wine, or strychnin.

When the acute symptoms have been controlled, the animal should receive a course of iodid of potassium.

Some of these cases become progressively worse and die in spite of anything that may be done. The diarrhoea

is especially difficult to control at times. Some cases retain a paralysis of certain muscle groups indefinitely after making a good recovery from the acute symptoms.

Animals that have ingested enough lead-containing substance to provoke marked symptoms are infirm and lack stamina for long periods of time after the symptoms of lead poisoning disappear.

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