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ARTICLE IV.—COLIC OF HORSES.

Offices and Experiment Grounds on the Farm of the Ohio State University.

☞*The Bulletin of this Station is sent free to all residents of the State who request it.*☜

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INTRODUCTION.

In the fifth annual report of the Ohio Agricultural Experiment Station, for 1886 (pp. 296–303), I published a brief article on the causes of colic of horses. I then stated that my own observations had confirmed Prof. Bollinger's assertion that nearly every aged horse has an aneurysm* in the anterior mesenteric artery, that such an aneurysm is produced by the presence of a small worm, *Sclerostomum equinum*, belonging to the family of Strongylidae, and that in many, perhaps in a majority of cases, the existence of such an aneurysm must be considered, if not the sole, at any rate, the principal cause of colic.

Although not much that is really new can be added to what I said in the annual report of 1886, and although no important discoveries have since been made, the simple fact that since that report was published such an aneurysm has been found in every one of the sixteen horses that have been killed for anatomical purposes in our Veterinary College, or Veterinary Department of the Ohio State University, and that said aneurysm was found not only in old horses, but also in young horses and in mules, will more than corroborate what I said two years ago, and be of interest to science and of practical value to the farmer and horse owner. As to the occurrence of the aneurysm in young horses, I can state that among the sixteen horses and mules killed for anatomical purposes since the publication of the fifth annual report, were two young horses, one last year and one this winter, which were each less than two years old, consequently mere colts, and that both had big aneurysms containing quite a large number of worms.

Therefore, as colic is one of the most frequent diseases of horses, which, notwithstanding its frequent occurrence, is but little understood, even by the majority of veterinarians, and consequently a disease which is seldom rationally treated, and perhaps oftener than any other a subject of quackery of the grossest kind; further, as it causes every year great losses, partially due to its often dangerous character, and partially to irrational treatment, I think a brief but comprehensive treatise on colic, showing the causal connection between the aneurysm and the morbid process, explaining its true causes, describing the symptoms, etc., giving a rational treatment, and pointing out the means of prevention, will be

* An aneurysm is a soft, pulsating tumor in an artery.

appreciated by the farmers and horse owners of Ohio. Hence, as the principal work of the Veterinary Division of the Station has not yet progressed far enough to give an account of it to the public, I have concluded to offer to the farmers of Ohio the following treatise on colic.

1. DEFINITION.

The names of diseases are not always appropriate, nor indicative of the nature of the morbid process to which they are applied; but, in the first place, it is often difficult to find for every disease a suitable name which at once conveys a correct idea of its peculiarities; and secondly, it is seldom advisable to attempt to replace an unsuitable or meaningless name, that has been in use for a long time, with a new one that is more significant, because both names will then be in use and will be apt to create confusion. In course of time some names of diseases have also changed their meaning on account of the progress made in medical science. Hence, the names of several diseases, in order to be understood, require a definition, i. e., an explanation of their present meaning. Colic is one of them. It undoubtedly is derived from colon, the name of one of the large intestines, and it probably was first used to signify a disease which was supposed to have its seat in that intestine. At the present time the name of colic is applied to all diseases, which manifest their presence by expressions of great or violent pain in the stomach, the intestines or guts, and the peritoneum (or external lining membrane of the intestines and internal lining membrane of the abdominal cavity). From this definition it will at once be seen that what we are in the habit of calling colic must include quite a number of morbid processes, in fact, all such as cause great pain, and have their seat in those organs named. On this account it has been found necessary to make divisions, and to distinguish:

I. True Colics,
II. Spurious Colics, and
III. Symptomatic Colics.

Even these three divisions have to be subdivided again. Thus, for instance, a symptomatic colic, as the name implies, is not an independent disease. The principal seat of the morbid process is not even limited to the stomach, intestines, and peritoneum, and the cause or causes usually affect the whole organism, not only secondarily but primarily. The name of symptomatic colic is applied to the colicky pains often observed in various septic or typhoid diseases, which cause ulceration, and in anthrax or splenic fever, which frequently produces carbunculous processes in the intestinal mucous membranes; further, to the colicky pains caused by

poisoning with arsenic, sugar of lead, tartar emetic, phosphorus, quicksilver preparations, overdoses of carbolic acid, of aloes, croton oil, etc.; also, to such manifestations of violent pain in the digestive canal as may be caused by the presence of large numbers of parasites or entozoa (worms) in the stomach or intestines. Finally, the name of symptomatic colic is applied to such expressions of violent pain as may be caused by displacements, or by morbid changes in the texture of the intestines; hence, by catarrhal, croupous, follicular, and traumatic inflammations; by so-called internal hernia, intussusceptions (telescoping) of the intestines, by new formations (tumors, etc.) and strangulations, by spontaneous ruptures and perforations, etc.

In spurious colics the seat of the disease is not in the stomach and intestines, but in other parts and organs situated in the abdominal and pelvic cavities. Still, it seems that in all cases of spurious colic the colicky pains are caused by an affection of the peritoneum. At any rate, as long as the morbid process, which may have its original seat in various organs, for instance, in the uterus, or in the bladder, has not yet extended to the peritoneum, no colicky pains will be present, and consequently no spurious colic will be existing. These spurious colics, therefore, very appropriately, might be called peritoneal colic, because a morbid or an inflammatory process in the peritoneum constitutes their immediate cause.

True colics, finally, may be caused by a variety of morbid processes in the stomach and intestines, and may owe their existence to a variety of causes; these, therefore, need likewise to be subdivided. So we distinguish:

(*a.*) Colics not produced by material causes, or so-called nervous, spasmodic, or rheumatic colics, and

(*b.*) Colics due to material causes, that is, caused by anomalies of the contents of the stomach and intestines.

The colics due to material causes again are subdivided according to the nature of the anomalies. We thus distinguish:

1. Colics caused by over-feeding.

2. Colics caused by, or attended with an excessive development of gases, so-called flatulent, wind, or, more correctly, fermentation colics; and

3. Colics caused by solid obstructions in the digestive canal, such as hard and solid fæces, concrements, stones, etc.

From the above it will easily be seen that what we are accustomed to call colic is not a single, definite disease, but may be produced by a variety of morbid processes, which, due to various causes, have one thing in common, that is, they all cause violent pains in the stomach or intestines. The causes may be numerous, but in many cases they

remain unknown. If they could become known in every case, the diagnosis could be more definite, the prognosis more certain, and the treatment, very often at least, be more to the point. But as it is, in many cases even the exciting causes remain unknown; in others the attendants may know what has happened, but for reasons best known to themselves may prefer not to tell, and the patients themselves cannot inform us except by objective symptoms. Consequently in many cases the veterinarian has to rely exclusively upon his own observations, that is, upon his interpretation of the symptoms exhibited by the animal. These, however, principally consisting in manifestations of great pain in the stomach and intestines, are not always the same in like cases occurring in different individual animals, because, a good deal like human beings, one horse will express the same kind of pain in one, and another in another way—one horse is naturally of a nervous or excitable, and another of a phlegmatic disposition. Hence, the same degree of pain, or pain caused by the same morbid process, may be shown in a different way, degree, and manner by different individual animals. All this, of course, must be taken into consideration. It is, therefore, by no means sure that in cases of colic the animal which expresses its sensation of pain in the most violent manner, is more severely affected than another one that is less violent in its actions, and *vice versa*.

THE CAUSES.

If we analyze the causes of colic, that is, the influences and agencies able to produce the various morbid conditions usually called colic, we must keep in mind that colic as well as other diseases requires two kinds of causes, namely, predisposing and exciting causes.

As to the former, it is generally supposed that the same consist in the smallness of the stomach, the inability of horses to vomit, the great length of the mesenterium, and the comparatively great width of the large intestines, cæcum and colon. This view, however, is untenable. It is true, horses have a small stomach, and under ordinary conditions they cannot vomit, but it is equally true that a great many horses never have an attack of the colic, while others, especially older horses, are quite often affected. Besides that, the food remains but a short time in the stomach of a horse, and the greater capacity of the large intestines sufficiently compensates for the smallness of the stomach.

As to the inability to vomit, it is, according to Gamgee, much more due to the insensibility of the horse's stomach to emetics than to the anatomical and histologic-anatomical structure or the peculiar insertions of the œsophagus, etc. Still, it cannot be claimed that the peculiar arrangement of the digestive organs of the horse, the comparative smallness of

the stomach, and the great size of the intestines, cæcum and colon, have no influence whatever upon the digestive process, for they undoubtedly have such an influence; but it seems this influence is not such as to produce digestive disorders, if no other more potent causes are acting; though it may, in some respect, facilitate the actions of other causes. To explain this: over-feeding, for instance, will not make its injurious influence felt as soon, if the stomach is large, as it will if the stomach is rather small; and constipation, or a development of gases, it stands to reason, may become more extensive, may therefore have a more general effect, and cause a more severe intoxication with noxious gases, if cæcum and colon are large or of great capacity, than if they are comparatively small like in other animals. In this way, it is true, the smallness of the stomach, and the comparatively great capacity of the cæcum and colon may contribute to make the disease more severe, if colic is existing, but they cannot cause it.

At any rate, an unbiased observer will find that the digestive apparatus of a horse is well adapted to serve its purpose. The digestive apparatus of a ruminating animal is more complicated, and, may be, more perfect, as far as the digestion of voluminous food is concerned, but surely would not do for a horse. The latter, being a work animal, is not intended to consume large quantities of voluminous food like cattle, but rather moderate quantities of more concentrated food. Horses are not digestive machines like cattle, which have to convert bulky vegetation into animal products, such as milk, butter, cheese and beef. Therefore, horses should not be treated as digestive machines. If they are, a predisposing cause may thereby be produced, the same as by want of exercise. In either case the processes of nutrition and circulations will become sluggish and irregular, and all the energy of the body will be appropriated by the process of digestion. Irregularities in the circulation of the blood, and consequently in the process of nutrition, are apt to be the result. A further explanation will be unnecessary.

As to the comparatively great length of the mesenterium, and the free position of the cæcum, which also have been accused of constituting a predisposing cause of colic, it seems to me just the reverse is the case, because they cannot but facilitate the peristaltic motion, and therefore, cannot be a factor in producing colic. But, on the other hand, it must be admitted, if colic is already existing—has been produced by other causes—and is attended with a development of gases, either arrangement—the length of the mesenterium or the free position of the cæcum—may just as well facilitate the abnormal movements of the intestines caused by the morbid expansion of the same by the gases, and thus become contributary

in producing a displacement, etc., as under ordinary circumstances they facilitate the peristaltic motion.

PREDISPOSING CAUSE OF COLIC.

The real predisposing cause of colic, or the real cause why horses suffer so much more frequently from colic than other animals, must be found in the exceedingly frequent occurrence of aneurysms in the anterior mesenteric artery. These aneurysms, as already explained in the fifth annual report of the Station, are caused by the presence of small worms, known as *Sclerostomum equinum*, Rud., or as *Strongylus armatus*, in the artery: I say such aneurysms are exceedingly frequent. Prof. Bollinger found them, in Munich, in 94 per cent. of all horses killed for anatomical purposes, and in our institution in Columbus, we have found them, so far, in over 95 per cent., or in 20 out of 21. Of its cause, the worm, and of its causal relation to colic, I shall speak further on.

EXCITING CAUSES OF COLIC.

Now, as to the exciting causes, which usually are looked upon as the sole causes of colic. In briefly enumerating them, I will follow Prof. Friedberger, (cf. his monograph: Die Kolik der Pferde, Berlin, 1874.)

1. Sudden changes of temperature and exposure to cold—so-called catching cold. It is true that a sudden exposure of a horse, especially if perspiring, to a draft of cold air, or to a low temperature, may disturb the functions of the digestive apparatus, and may, and sometimes does, cause diarrhœa, attended with more or less colicky pains, but without other causes acting at the same time, will never produce real colic. Drinking large quantities of ice-cold water, or consuming considerable quantities of frozen food, or food covered with hoar frost, will more or less chill the intestines, and may have a sinilar effect, that is, cause diarrhœa or digestive disorders. But the morbid process seldom progresses beyond hyperæmia (an abnormal accumulation of blood) in the mucous membranes of the stomach and intestines, and a subsequent increased secretion. From my own experience, I can only cite one case in which the drinking of a large bucketful of ice-cold water, early in the morning, caused a severe case of colic, which became fatal at 4 o'clock in the afternoon. The post-mortem examination revealed an intussusception (telescoping) of the small intestines.

Sultry and foggy weather, a threatening storm, and a low barometer seem to have some influence. I practiced three years in a country in which, from various causes, cases of colic, especially in the fall, were very frequent; but they usually occurred during moist and foggy weather, or when a storm was brewing. I state this bare fact without attempting to

give an explanation. There are several possibilities, and it is easy enough to advance a plausible theory, but theories are not evidence, and positive evidence in explanation of the fact just stated is yet wanting.

2. Absolute and relative overfeeding undoubtedly constitutes a frequent cause of colic, especially in animals in which the necessary predisposing causes are already existing. Too large a quantity of food in the stomach and intestines, not only requires an increased activity of the digestive apparatus, and consequently an increased supply of the digestive canal with blood, but also retards and impedes the peristaltic motion, and by expanding the abdominal cavity and pressing upon the diaphragm, decreases the space in the chest, and thus interferes, more or less, with the functions of the heart and of the lungs, and in consequence, with the decarbonization of the blood, and the regularity of its circulation.

If the food is not too voluminous, but difficult of digestion—for instance, too rich in nitrogenous compounds—also a greater activity of the digestive apparatus, and a more liberal supply with blood are required, because the gastric and intestinal juices must be increased. This can be done up to a certain limit, which, of course, differs in different animals. If this limit is passed, a disorder in the circulation of the blood is apt to follow, especially, as I shall show further on, if an aneurysm is existing. The digestion will then be interrupted, the peristaltic motion will cease, a decomposition of the contents of the digestive canal will supersede the process of digestion, gases will develop, and colic will be the result. All this will happen the sooner, the less the digestive apparatus is accustomed to extraordinary quantities of food, or to food, very rich in nitrogenous compounds, or difficult of digestion. No part or organ of the animal body is apt to cease to perform its functions if only required to do an amount of work to which it has been accustomed, but if this amount of work should suddenly be augmented to an extraordinary extent, or much beyond the accustomed limit, a more or less complete cessation of functions will often be the result.

3. Food which has a tendency to ferment. For instance, young, green grain, wilted down, or rank and watery grass, grown under the shadow of trees, or any other succulent food plants of a rapid growth and rich in water, if cut and thrown into heaps so as to become partially wilted; further, new grain and new hay that have not passed through the "sweating process," and are not yet cured and perfectly dry, have a great tendency to ferment in the stomach. This tendency must be overcome by an increased (extraordinary) activity of the digestive apparatus. Such an increased activity means an additional secretion of the gastric and intestinal juices, and more vigorous peristaltic motion, consequently an augmented supply of blood to the digestive canal. And here again, especially if such

an increase is a sudden one, the existence of an aneurysm, as I shall show later, constitutes a very potent, predisposing cause of colic.

Feeding a heavy meal of grain, or food difficult of digestion, either immediately before exercise, consequently at a time when a larger quantity of blood will be appropriated by the locomotary apparatus at the very moment at which also an increased amount is needed by the digestive canal, or immediately after severe muscular exertion, when most of the blood is yet circulating in the muscles. In either case a sudden change in the distribution of the blood has to take place, and this change often leads to serious consequences, and, as will further on be explained, often constitutes an exciting cause of colic, if the principal predisposing cause, an aneurysm in the anterior mesenteric artery, is existing.

4. It also has been claimed that swallowing considerable quantities of air, as is sometimes done by cribbers and wind-suckers, may produce flatulent or wind colic. This, however, seems to be a mere supposition, lacking any real foundation. At any rate, a horse that has become a cribber or wind-sucker, and has learned how to swallow air, has also learned how to belch and to get rid of it. At least experience teaches that if a wind-sucker or cribber gets bloated—his stomach expanded by air or gases—there is seldom any danger, because he very soon will commence to belch, and will thus expel the air and gases.

5. Mechanical obstructions in the intestinal canal interfering with the circulation of the blood, or hindering the passage of the contents, constitute a not infrequent cause of colic. These obstructions may be of various kinds. They may consist in accumulations of fæcal masses, which have become more or less firm and solid by an absorption of their fluid constituents, and have thus become lodged at some place in the intestinal canal, where, especially if wedged in, they will press upon the blood vessels in the wall of the intestine and in adjoining parts, so as to interfere with the circulation of the blood, particularly in the capillary system of the mucous membrane. But not only that, they will also abstract the lumen of the intestine, and thus prevent the passage of its contents. Local stagnation of the blood in the mucous membrane, and stagnation and subsequent decomposition of the food or fæces in the intestine will be the necessary result.

If meal, shorts, or bran are fed, and not sufficiently wetted, or not moistened at all, and the horse happens to be a greedy eater, sometimes hard and compact balls are formed, which may become lodged in an intestine, and thus constitute such a mechanical obstruction.

The same effect is produced by foreign, insoluble, and indigestible bodies when lodged, or wedged in, at some place in the intestinal canal. As such foreign bodies may be mentioned: accumulations of earth and

sand (quite frequent in colts and old horses grazing on pastures almost destitute of vegetation); concretions and stones (often found in horses fed with excessive quantities of bran or other food rich in inorganic compounds, especially if the water for drinking, too, is hard); other insoluble inorganic substances; for instance, large quantities of black antimony in horses that have been compelled to consume large amounts of "condition powders," of which black antimony and other insoluble and indigestible substances are frequently the principal ingredients. The concretions and stones occurring in the digestive canal of horses are principally composed of phosphates of ammonia and magnesia, and are found most frequently in the horses of millers, and in such as are largely fed with bran and mill stuffs.

Worms, particularly large numbers of *Ascaris megalacephala*, sometimes constitute the cause of colic, especially in weak, emaciated, and stunted colts and one and two-year-olds, and in old and lean horses. In such cases we find the worms at the post-mortem examination in enormous numbers in the duodenum and in the stomach, and occasionally even in the œsophagus. Whether the worms constitute the cause of the emaciation and the weakness, whether the same find a more suitable soil in an already weak and emaciated animal, or whether the usual causes of weakness and emaciation: the poor, innutritious, and often spoiled or decayed and unclean food, contain the worm brood, is a question, which to discuss here would lead too far.

Prof. Friedberger mentions a case in which large numbers of another worm, *Sclerostomum tetracanthum*, in the cæcum and rectum constituted the cause of colic.

There are still other entozoa (intestinal worms); for instance, *Spiroptera megastoma*, and *Tænia plicata* and *T. perfoliata*, which have been accused of occasionally causing colic.

6. In every-day life a great many cases of colic are attributed to the presence of the larvæ (bots) of *gastrus equi*, or the bot-fly, in the stomach. The truth is, a great many horses, perhaps all who have been pastured during the summer, have a larger or smaller number of bots, and still have no colic. Veterinarians since the time of Delabere Blaine, therefore concluded that these larvæ, or bots, are entirely innocent and productive of no damage. Some even claimed that their presence is wholesome and promotes digestion. This, undoubtedly is erroneous. They are parasites; they feed upon material which is furnished by their host, and therefore are not apt to be a benefit to the latter. On the other hand, as to the question of doing serious damage, a "Scotch verdict" (not proven) will have to be rendered, in most cases at least. Still, there are some cases in which the evidence is against them.

The stomach of the horse may be divided into two portions, the anterior, left, or cardiac portion, and the posterior, right, or pyloric portion. The former is lined with a firm, whitish-looking mucous membrane, coated with pavement epithelium, and destitute of glands. It is a continuation of the mucous membrane of the œsophagus. The posterior, right, or pyloric portion of the stomach is lined with a mucous membrane entirely different. It has a velvety, reddish-gray appearance, is rich in blood-vessels, contains numerous glands, and is coated with cylinder epithelium. This latter mucous membrane is much more sensitive; its numerous glands secrete the gastric juice, and it therefore constitutes an important factor in the process of digestion, whereas, the anterior or cardiac portion of the horse's stomach is only a preparatory organ. The larvæ of *gastrus equi*, or so-called bots, are very small when they invade the stomach, and then, as a rule, fasten themselves with their hooks to the mucous membrane of the anterior, cardiac portion, and feed upon the exudation produced by their presence. This exudation, it seems, also accumulates around their heads, and forms a kind of a wall, so that when such a larva is removed a cup-shaped disk (concavity) remains behind in the mucous membrane.

If these larvæ are disturbed, or caused to let loose where first attached, either from insufficiency of food (exudation) or from other causes, then they often attach themselves anew to the mucous membrane of the pyloric portion of the stomach. But when they do this, they are not any more the small embryos they were at first; they have grown larger and stronger, and finding no circumvallate disk, they, it seems, wound, and burrow into, the numerous membrane, and thus undoubtedly interfere with the functions of the latter. That a grown bot is able to wound the mucous membrane, there is abundant proof, because at some post-mortem examinations a few are sometimes found in the abdominal cavity; consequently they must have worked their way not only through the mucous membrane, but also through the muscular and serous coats of the stomach. It is true, they very likely do this only after their host has died, or when the flow of exudation ceases. But it is equally true that they may, and can do precisely the same thing where from some cause or another the flow of exudation becomes insufficient during the life of their host. And even if not, when attached to the pyloric portion of the mucous membrane of the stomach, there can be no doubt that damage is done, as they necessarily must interfere with the functions of that membrane, the secretion of the gastric juice. This, of course, the more, the larger their number. I think, it will be safe to say that bots, as a rule, do not cause essential damage, but where from some cause or another a digestive disturbance is taking place in the stomach, and the bots are caused to let loose, and to

attach themselves to the pyloric portion, they will then not only increase the gastric disturbance, but may convert it into a very serious, and even fatal colic.

In some cases the larvæ or bots have been found attached in uncommon places, for instance, in the larynx, or in the œsophagus, and have caused serious trouble. If present in large numbers in the stomach, and caused to detach themselves, or when matured and ready to make their exit from the stomach, they sometimes accumulate in the pylorus (the opening of the stomach into the intestines) or in the duodenum (the intestine proceeding from the stomach), close the opening, and thus constitute the direct cause of colic.

7. That spoiled, decomposed, or damaged food, organic and inorganic poisons, overdoses of drastic medicines, etc., can produce colic, sometimes of a very serious or even fatal character, is self-evident, and may not need any further explanation.

8. Morbid changes in the texture of the stomach and intestines necessarily interfere more or less with the functions of these organs, and consequently with the process of digestion. They may even effect an obstruction of the passage, and thus, like other obstructions already mentioned, become a direct cause of colic. They may also interfere with the circulation and distribution of the blood, and in that way, more indirectly bring on an attack, or act as a predisposing cause.

9. Wounds, penetrating into the stomach or intestines from the outside, or being produced from within by sharp or pointed bodies that may have been swallowed with the food, or by concrements, ulcerative processes, etc., usually cause peritonitis, and the colicky pains produced constitute what we have called spurious colic.

10. Displacements of an intestine, a so-called internal hernia, an intussusception, or telescoping of one part of an intestine into another, a torsion, a stricture, a spontaneous rupture, etc., are usually secondary morbid changes, that is, the result, and not the cause of the attack of colic. They, of course, if produced during the life of the animal, will cause the colic to become fatal. But, in by far the majority of cases, all these changes, displacements, strictures, torsions, volvuli, etc., or what else they may be called, do not take place while the animal is yet alive. They nearly always are produced after death, and are due to the post-mortem movements of the bowels caused by the rapid development of gases and the excessive expansion of some parts of the intestines after the peristaltic motion has entirely ceased, and decomposition has full sway and is proceeding with great rapidity. If produced while the animal is yet alive, the displaced, strangulated, twisted or telescoped parts invariably show

local congestion, swelling and exudation, whereas, if produced after death no such local congestion, swelling and exudation will be existing.

If we investigate the true relation of the possible causes, above enumerated, to the cases of colic actually occurring, we will find many cases of colic in which none of these causes can be accused, and will find, also, numerous instances in which one or some of these causes are present or acting, and no colic results. So, for instance, it happens very often that of two horses, constituting a well-matched team, one has frequent attacks of colic while the other is never affected, notwithstanding that both horses are kept and fed in every respect precisely alike, and are in every other way, as to age, size, constitution, breed, temperament, sex, condition, etc., as nearly alike as possible. Then again, sometimes cases of colic occur when one or another of the causes enumerated was present, but not in such a degree as to produce by itself a severe attack. Or else, it quite often happens that one or another of the causes, especially one of those enumerated under heads 1, 2 and 3, is acting, and another disease, founder, for instance, and not colic, is the result. Hence, there must be some predisposing cause, which either alone, or in combination with some external agency or exciting cause, produces at least all those cases of colic that cannot otherwise be accounted for.

Although colic is, and always has been, a very common disease, this principal predisposing cause remained unknown, or was overlooked, or a long time. It is Prof. Bollinger, in Munich, who first brought it to light. (Cf. his treatise: "Die Kolik der Pferde und das Wurmaneurysma der Eingeweidearterien," published in 1870).

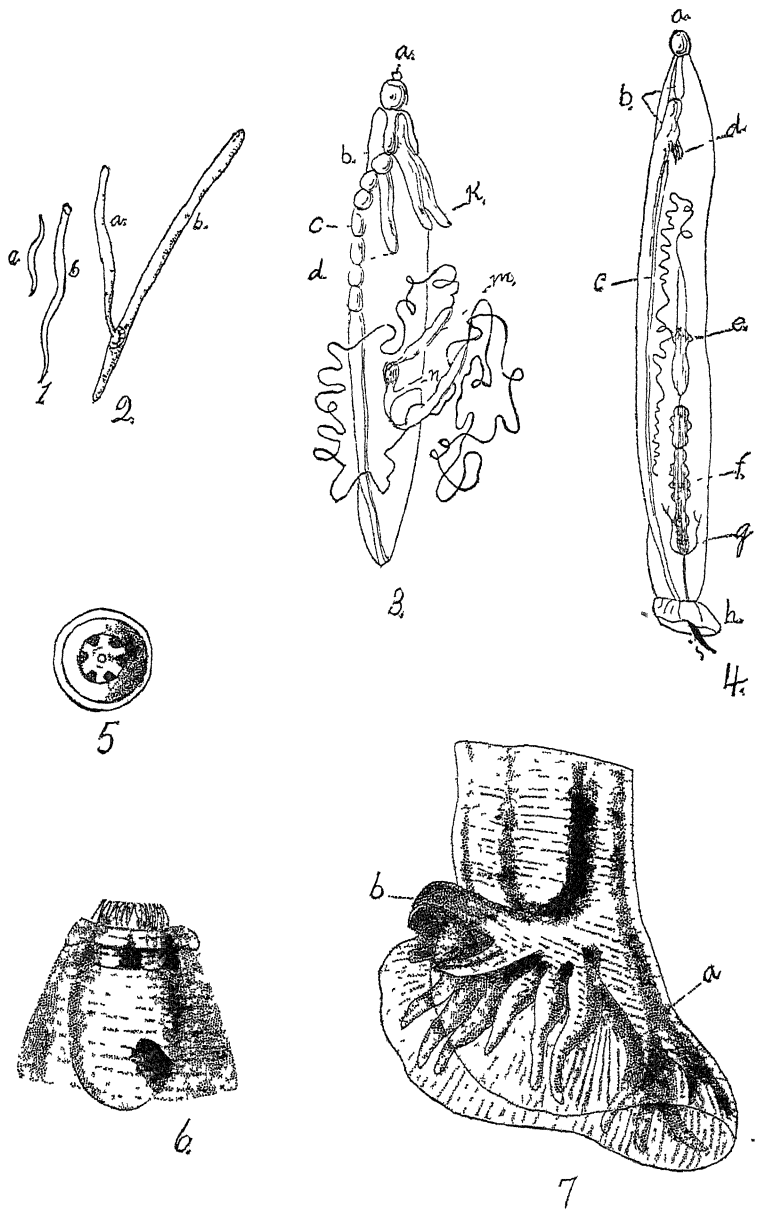
Bollinger found in his investigations that the anatomical and functional changes in a vast majority of cases of colic, at any rate in such as have a fatal termination, are essentially the same as those which follow a rapidly developing obstruction by embolism or thrombosis in the intestinal arteries. He then found that most old horses killed for anatomical purposes have a worm aneurysm in the anterior mesenteric artery. He found such an aneurysm in 90 to 94 per cent. of all aged horses in Munich. (In Munich, like in most other large cities in continental Europe, a great many horses are butchered for meat, but before this meat is allowed to be sold the horses must have been examined by a veterinarian). Bollinger also established the causal connection between the aneurysm and the frequency of cases of colic.

THE ANTERIOR MESENTERIC ARTERY AND ITS ANEURYSM.

The anterior mesenteric artery is a short but strong vessel, only about an inch long. It proceeds from the lower wall of the posterior

aorta beneath the first lumbal vertebra, and furnishes the blood for the small intestines (duodenum, jejunum and ilium), the large intestines (cæcum and colon), and the anterior portion of the rectum. From the short trunk proceed from 18 to 21 branches, the intestinal arteries, which descend in the mesenterium to the small intestines, from the duodenum to the ilium. Close to the intestines these arteries form anastomoses, that is, connect with each other in the following way: each one divides near the intestine into two main branches, of which one curves forward and the other backward. The forward branch of each one unites with the backward branch of the preceding artery, and the one curving backward unites with the forward branch of the following artery. In that way artery No. 1 unites (anastomoses) with its forward branch with a branch of the cœliac artery, and with its backward branch with the forward branch of artery No. 2. No. 2 anastomoses in the same way with No. 1 and with No. 3, and so on, until the last one, No. 18 or No. 21, as the case may be, anastomoses forward with the backward branch of the preceding artery, and backward with the artery of the ilium, a branch of the ilio-cæco-colic artery. From the arches thus formed between each two intestinal arteries, proceed the smaller arteries which pass directly to the intestine, and ramify in its walls.

The next branch proceeding from the common trunk, the anterior mesenteric artery, is the anterior artery of the rectum, which, however, proceeds quite often from the superior artery of the colon, and then, of course, not from the common trunk. The anterior artery of the rectum, like the intestinal arteries, divides into two branches, one anastomosing backward with the middle artery of the rectum, a branch of the posterior mesenteric artery, and the other anastomosing forward with a branch of the superior artery of the colon. The next branch proceeding from the anterior mesenteric is a very large one; it is the superior artery of the colon. It passes to the upper layer of the colon, gives at once a strong branch to the middle lobe of the pancreas, and then passes along to the posterior curvature of the colon, where it forms an anastomosis (unites) with the lower artery of the same intestine. The remaining trunk of the anterior mesenteric artery, or the last and strongest branch, is called the ilio-cæco-colic artery. It soon gives off the lower artery of the colon, which has already been mentioned as anastomosing at the posterior curvature of the colon with the superior artery of the same intestine. The remaining trunk of the ilio-cæco-colic artery soon divides into three branches, of which one passes to the ilium and anastomoses with the last of the intestinal arteries, and two to the cæcum as external and internal artery of that intestine. These two form numerous anastomoses with each other.



In order to explain what an aneurysm is, and how it is produced, it will be necessary to give a brief description of the worm that does it, and also the natural history of the same as far as it is known.

Sclerostomum equinum s. armatum, Rud., or Strongylus armatus, is a round worm and belongs to the Strongylidæ. The mature worm inhabits the cæcum and colon of horses, and occasionally also occurs in the duodenum, the pancreas, the free abdominal cavity and the vaginal sac of the scrotum.

The larvæ or immature worm, which is best known under the name of Sclerostomum equinum, inhabits the larger arteries of the posterior part of the body, but especially the anterior mesenteric artery and its principal branches, the ilio-cæco-colic and the superior colic artery, and in comparatively rare cases only the other main branches of the posterior aorta, for instance, the cœliac artery, the arteries of the kidneys, the posterior mesenteric artery, and the large arteries of the pelvic cavity.

In the following I will give a brief description: Head, globular; mouth, circular, armed with a double wreath of sharp teeth, which gives it the appearance of a Trepan crown (fig. 6). From the mouth proceeds the so-called mouth-capsule, which leads to the bulb-shaped œsophagus, *b*, fig. 3 and 4, which is provided with teeth, and acts as a sucker. It is lined with a continuation of the chitinous layer of the skin, which undergoes the same casting process in the œsophagus as on the skin.

The body is round, red, or red brown, and tapering towards the tail. The latter is provided in the male worm with a funnel-shaped bursa, *h*, fig. 4 and fig. 7, which, if spread out, presents three parallel posterior, and separate intermediate ribs. The spiculum, i, fig. 4, is double. The tail end of the female, l, fig. 3, is obtuse; the sexual opening, n, fig. 3, is about 0.4 inch from the end of the tail.

The length of body of the mature male worm (a, fig. 2), is 0.8 to 1.2 inches, and that of the female (b, fig. 2), is from 0.9 to 2.0 inches. At the copulation the male embraces with its bursa the sexual opening of the female, so as to form with the body of the latter an acute angle, and often remains in that position for a long time (fig. 2). The eggs are elliptic, constricted in the middle, and from 0.003 to 0.004 inches long.

The larvæ, or immature worm, as found in the anterior mesenteric artery, and occasionally in some other large branch of the posterior aorta, measures from 0.5 to 0.8 inches in length, and only possesses rudimentary sexual organs. Fig. 1*a* presents a small, and fig. 1*b* a larger, almost full-grown larva with rudimentary sexual organs. The mouth opening of the larva is surrounded by a six-lobed rosette, fig. 5. The drawings, fig. 1 and fig. 2, present the worm in its natural size; fig. 3 and 4 present an enlarged outline drawing of the arrangement of the inner organs. In these figures *a* presents the mouth, *b* the œsophagus, *c* the intestine, *d* longitudinal vessel of the body, *e* and *f* testes and seminal ducts, *g* muscular fibres of

the lower end of the seminal duct, *h* the caudal bursa of the male, *i* the spiculum, *k* glands, *l* anal opening, *m* ovarian sac, and *n*, female sexual opening. Fig. 5, 6 and 7 are considerably enlarged. The figures are after Zuern.

In regard to the natural history and development of Sclerostomum equinum, interesting researches have been made by the great entozoölogist, Leuckart. He found that the mature worms copulate in the cæcum and colon, and that the female worm deposits her eggs in the intestine. The eggs then pass off with the excrements. If the latter are deposited in warm weather on moist ground or in mud, the same will hatch in about 3 to 4 days. Then the embryos, enveloped only by a mere shell, try to get into water or mud, and there exist as free nematods (Rhabditides). If they get into water, and the water is drank by a horse or a mule, the larvae again reach the intestines, and from there enter the blood-vessels. How they accomplish this, that is, find an entrance, is not definitely known. But as they are found in the anterior mesenteric artery and its main branches, and occasionally also in the cœliac artery, in the arteries of the kidneys, in the posterior mesenteric, and even in the larger pelvic arteries, it seems, they find an entrance into some of the arteries which ramify in the intestinal mucous membrane, by penetrating the inner layer of the latter and the wall of such an artery, and then ascend in the latter until they reach a strong countercurrent of blood, which probably induces them to fasten themselves with their armed mouth to the inner surface of the wall of the artery. Usually from a few to a dozen or more are found in the anterior mesenteric artery, but in some cases as many as 100, or even 120 have been found in a single aneurysm. Wherever the immature worms fasten themselves, morbid changes in the wall of the artery will be produced, and an aneurysm will be the result. These aneurysms, of course, vary in size, according to their age, and the number of worms that have caused them. They have been found as large as a child's head.

The aneurysm itself, it seems, is produced in the following way: The worms wound the interior membrane of the artery, and in that way cause inflammation and exudation. The exudation causes the membrane to swell, and also accumulating on its surface, would make the lumen (opening) of the artery narrower, if the pressure of the blood column would permit a contraction of the arterial tube, or not expand the same. Such an expansion, yielding of the arterial walls, is facilitated by their inflamed and swelled condition, and therefore decreased resistance. Gradually the walls, in those places, at least, at which the worms attack them, become more and more degenerated, the artery increases in size, and although at certain spots, cavities, or an enlargement of its internal diameter, may be produced, the passage or clear tube opening is not enlarged,

but the interior surface of the arterial wall becomes uneven and rough. In some cases, such an aneurysm extends not only through the whole length of the anterior mesenteric artery, which is but an inch, or a trifle more, but also several inches into the ilio-cœco-colic or superior colic artery. Usually also, more or less coagulation is found in such an aneurysm, which not always presents the appearance of a post-mortem change, but very often appears as if it has been produced during the life of the animal. This becomes the more probable, if it is taken into consideration that the worms are rather lively, often change their place, and thus interfere with the regular current of the blood, and give an occasion for a formation of clots.

The mere existence of an aneurysm does not seem to do any visible damage, unless pieces of the fibrinous exudation, or some clots, become detached by the pressure of the blood, and are carried by the force of the current into one or more of the branches of the main artery. In such a case, the exudation, or the clots of coagulated blood are carried further and further, until they get stuck, form a thrombus, close the artery, and obstruct the circulation, but about this afterwards. In other, though rare, cases the walls of the aneurysm may become degenerated and weakened to such an extent as to become unable to withstand the pressure of the blood current, and then the artery ruptures, and the horse dies of internal hemorrhage.

It probably will not need an explanation that there is no possibility of removing or killing the worms, once safely lodged in the anterior mesenteric artery, where no medicines can touch them. The mature worm, while in the colon or cæcum, or in any of the other intestines, might be destroyed by some worm medicine, but the trouble is, we have no means of knowing when they are there, because in the intestines they do not cause any sufficient disturbance to produce observable symptoms.

The only way to protect horses against an invasion of these worms, and the subsequent formation of an aneurysm, is never to allow them to drink any stagnant water from pools and ditches, or even flowing water from creeks and streamlets, but to water them exclusively from a good well, and when that is not possible, to have some kind of a filtering apparatus that will retain the worm-brood, and to filter every drop of water before the horses drink it.

THE FREQUENCY OF THE ANEURYSM.

Prof. Bollinger, as said above, found a worm-aneurysm in 49 per cent. of all, and in 90 to 94 per cent. of all aged horses. He therefore concluded that it is a frequent occurrence. He further found that 40 per cent. of all internal diseases of horses are cases of colic, and that 40 per cent. of all

horses that die of disease, die of colic. He further claimed that 75 per cent. of all cases of colic are directly or indirectly due to the existence of a worm-aneurysm.

We have endeavored during the last three years (cf. Annual Report of the Ohio Agricultural Experiment Station for 1886, page 296 ff.,) to ascertain, as far as circumstances would permit, the truth of his statements. In the winter of 1886–87, we killed for anatomical purposes four old horses and two mules, and found that only one mule was free from such an aneurysm. In 1887–88, we killed in all, four old horses, one colt, 22 months old, and two mules. Every one of them had an aneurysm. Of two of them we injected the arteries, and were very successful in injecting the intestinal arteries, so that the finest ramifications were plainly visible, but found that in each case two or more of the intestinal arteries had become obliterated. In the present winter of 1888–89, we again killed seven old horses and one colt (yet a stallion and less than two years old), and every one had a worm-aneurysm. We intended to make injections to see how many and what arteries had become closed, but in the fore part of the winter we could not do it, because the carcasses were needed for the study of the muscles, and the latter cannot very well be prepared when the arteries have been injected, because the injection material, gypsum, will immediately dull every knife; and in the latter part of the winter we could not do it, because the burning of one of our University buildings (last February) destroyed all our instruments and apparatus.

To sum up, we found a worm-aneurysm in the anterior mesenteric artery in twenty out of the twenty-one animals that were examined. We endeavored to learn the history of every animal we killed, but as we obtained most of them through the Humane Society, and some from people who had owned them but a short time, we succeeded in this only in regard to a few, and these few, we learned, had repeatedly suffered from colic. The mule that had no aneurysm never had any colic as far as we were able to learn.

THE CAUSAL CONNECTION BETWEEN A WORM ANEURYSM AND THE MORBID PROCESS OF COLIC.

The question may be asked, in what way does such an aneurysm cause colic, or, since it is known that most horses have an aneurysm, and yet do not all suffer from attacks of colic, and as the exciting causes of colic, as above enumerated, are and have been recognized as such by every author and by every practitioner, by what process does an aneurysm in the anterior mesenteric artery become a predisposing cause of colic?

This question I shall endeavor to answer. In the first place, it is an admitted physiological fact that activity in any part of the body causes

more waste of tissue (consumes more material) than idleness, that waste of tissue calls for repair, and that the material for repair must be furnished by the blood. Hence, every organ or part of the animal body requires, and if possible receives, more blood when active or performing its functions, than when idle or inactive, and increased or forced activity requires in the same ratio, in which it is increased, also an increased supply of blood. Further, if the increase of functions is a sudden one, the demand for more blood will likewise be a sudden one, and vice versa. Hence, the current of blood in the artery, or arteries, of an organ or part of the body will be stronger, if the organ or part is active, than it will be if the same is inactive, and it will be the stronger, or the more forcible, the greater the activity of the organ. Also, if the functions or activity of an organ are suddenly augmented, the increase in the force of the blood-current will likewise be a sudden one. If an aneurysm is existing in which fibrinous exudation, and, perhaps, coagulations, are adhering to the ragged and uneven interior surface of the wall of the artery, an even current of the blood through that artery is not apt, perhaps not forcible enough, to detach the coagulation or parts of the exudation, but a stronger current may, and often will. If coagulations are existing, which it seems is not often the case, the same occasionally may become detached, even if the force of the blood current is not abnormally strong.

Now, then, at what time, or by what process is the force of the current of the blood through the anterior mesenteric artery extraordinarily augmented? The answer is easy. It is when the intestines have increased functions to perform—in other words, if extraordinary demands are made upon the digestive powers of the intestines. If we now analyze the effect which most of the above enumerated exciting causes will have upon the digestive apparatus, we will find that nearly every one of them necessitates an extraordinary digestive activity—makes abnormal demands—consequently causes a greater rush of blood to the intestines, and, of course, a more forcible current through the anterior mesenteric artery. Some of the exciting causes, it is true, are able to cause colic, or at least disease attended with colicky symptoms, without the assistance of an aneurysm as a predisposing cause. As such I will mention all mechanical obstructions, such as stones, concrements, solid food balls composed of imperfectly moistened meal, bran, etc.; large numbers of entozoa, bots obstructing the passage, displacements, intussusceptions, so-called internal hernia, strangulations, torsions, etc., but if we investigate the cases of colic that come under our observation, we will find that very few of them indeed are due to the causes just mentioned, and that in a large majority of cases none of them can be accused. What exciting causes do remain? It is catching cold, or a sudden chilling of the intestines; over-feeding, quant-

itatively and qualitatively; food difficult of digestion; a heavy feed (of grain) immediately before or immediately after some exercise or hard work; food rich in indigestible constituents, and food that has a tendency to ferment. All these make extraordinary, and some of them suddenly increased, demands upon the digestive powers; consequently all of them cause a change in the distribution of the blood, and an irregular, or more forcible, current of the blood through the anterior mesenteric artery—hence, the very thing that is apt to detach, by increased friction, portions of exudation from the walls of the aneurysmal artery. If some exudation is torn loose, the current of the blood will carry it along into one of the branches, toward the intestine, until it gets stuck; then the artery will be closed, and that portion of the intestines supplied by it with blood will be deprived of its supply. As a consequence, its functions will cease; the peristaltic motion will stop; the contents of that intestine will not move on, and the digestive process will be interrupted, because a part of an intestine, as well as any other part of the body, if deprived of its supply with blood, will be dead for the time being. As a further consequence the peristaltic motion in front of the paralyzed portion of the intestines will meet with an obstacle and be unable to proceed any further. Cramping and griping will be the necessary result, and even displacements and intussusception may follow.

Fortunately all the intestinal arteries form anastomoses with their immediate neighbors, and if it happens to be one of the smaller arteries that becomes closed by a thrombus, circulation will soon be restored through the anastomoses. Hence, some cases of colic, although for the time being the cramping and griping pains may be severe enough, will terminate in speedy recovery, which, of course, takes place as soon as the circulation is restored, and long before any medicines that may have been administered, can have any effect. If in the next attack it happens that the thrombus (piece of exudation or clot of coagulated blood), becomes lodged in an adjoining artery, in No 2, the recovery will be somewhat slower, and the morbid changes produced will be severer, because a larger portion of the intestines is thrown out of circulation, and when finally the latter is restored through the anastomoses, two arteries will have to furnish the blood which formerly was furnished by four. If it then should happen that in the next attack artery No. 3 becomes closed, the colic will be of still longer duration, and the gravity of the situation will be increased, because the circulation can be restored only by two arteries furnishing the blood formerly provided by five, and it may even happen that meanwhile—until the circulation has been restored through the anastomoses—the morbid changes produced have become serious enough to endanger the life of the animal. If, however, instead of one of the comparatively

small intestinal arteries, one of the larger ones, for instance, the superior artery of the colon, or even the largest, the ilio-cæco-colic artery should become closed by the thrombus, the first attack may become severe enough to endanger the life of the animal, because it will require a much longer time until the circulation can be restored through the anastomosing arteries. Besides that, the large intestines usually contain a great deal of food, which will soon begin to ferment and to decompose under the influence of warmth and moisture, if the intestine is deprived of its supply with blood, and therefore paralyzed and unable to perform its functions. The gases, principally carbonic acid, carburetted hydrogen, etc., which are the product of the decomposition and fermentation processes, will diffuse through the walls of the intestine and will be absorbed—it must be kept in mind that the veins and lymphatics are not closed by embolism—and pass over into the blood, which thus becomes surcharged and poisoned, with them. The lungs will soon become unable to eliminate these gases as rapidly as they are developed. As a consequence the respiration will become more and more accelerated, and the lungs themselves will become hyperæmic (filled up with an abnormally large quantity of blood). This hyperæmic condition will still more interfere with the performance of their functions. At the same time the contractions of the heart, and consequently the pulse, will more and more increase in rapidity in order to overcome the obstacles to the circulation, caused by the thrombosis in some of the intestinal arteries, and by the stagnation of the blood surcharged with noxious gases in the overburdened lungs. If at the same time the development of gases in the affected intestine is so rapid as to cause an abnormal distension of the latter, still further impediments to the circulation of the blood will be the result, because the gases not only will compress the blood vessels in the wall of the intestine, and in adjacent parts, but the abnormally expanded intestine itself will press upon the arterial and venous trunks in the abdominal cavity, and, by pressing upon the diaphragm, will materially reduce the space in the chest available for heart and lungs. In that way heart and lungs will be compelled to perform their steadily increasing functions under still greater disadvantages, until either of them gives out, or until the blood becomes surcharged with noxious gases to such an extent as to become incapable of furnishing nutriment to the various parts of the body. Of the latter it is principally the brain and the nerve-centers that become first and most affected, and unable to perform their functions. Colic patients, therefore, often become unconscious, soporose, and even delirious, take no notice of their surroundings, and tumble about, and throw themselves without regard to anything. If the morbid process still continues, either the nerve-centers will cease to perform their functions, or heart and lungs will give

out, and the animal will die of suffocation. If all this happens before the circulation in the intestinal artery, or arteries, is restored, the colic terminates in death; if not, and the circulation is restored through the anastomoses before it comes to the extreme, the animal will recover.

A fatal termination may also result from a different immediate cause. If the development of gases is very rapid, for instance, in cases in which the food consumed has a great tendency to ferment, the expansion of the intestine may become so excessive as to cause the latter to rupture; then its contents will pass into the abdominal cavity, and an inflammation of the peritoneum will be added to the already existing morbid changes. In such a case recovery is out of the question.

Other morbid changes of a somewhat different character, are produced at the same time. The processes of diffusion, endosmosis and exosmosis do not cease when the circulation of the blood is interrupted; on the contrary, they seem to increase. The membranes, which constitute the walls of the intestines, become penetrated with exudation, the fluid parts of the contents of the intestines, and dissolving blood, and thus present, at a post-mortem examination, a wine-colored, brownish, or even blackish appearance; and some parts, especially such, in which the interruption of the circulation was complete, may even be gangrenous.

In other cases the peristaltic motion, brought to a stop at the paralyzed part of the intestines, may cause intussusception (telescoping of the intestines), or the expansion (bloating) caused by the rapid development and the imprisonment of gases in an intestine may cause a displacement, or a twisting of the intestine around its longitudinal, or even (in rare cases) around its transversal axis. Usually, however, such displacements, twistings, strangulations, torsions, etc., are not produced during the life of the animal, but after death, when fermentation, decomposition, and chemical processes in general have full sway in the contents of the whole intestinal canal, and when the development of gases and the expansion of the intestines proceed with great rapidity. Then such an expanded or bloated intestine seeks room wherever it is, and the epiploön, or caul, and the mesenterium, already in a state of semi-decomposition, easily tear. The same applies to most cases in which the stomach or an intestine is found ruptured at a post-mortem examination. It is, however, easily ascertained whether any one of these anomalies has taken place during life or after death. If during life there will be more or less local congestion, exudation, swelling, and, perhaps, adhesion, either of which will be absent if they have occurred after death.

It is a well known fact that colic occurs most frequently among horses which are kept a part of the year (in the winter) in the stable, and another part of the year (in the summer) on pasture; also, that cases of colic are

comparatively not frequent among horses which, like army horses, for instance, kept in barracks' stables, are always regularly fed, receive the best of food, and are never, or very seldom pastured. It is the regularity of feeding, etc., that has been advanced as a reason why colic is of less frequent occurrence among the latter class of horses than among the former. It undoubtedly has something to do with it, but it does not seem to me to be the sole nor even the principal cause. Horses always regularly fed with sound food, never or very seldom allowed to graze and to *obtain their water for drinking wherever it may be found, but always watered from a well or with hydrant water*, have less opportunity to pick up the larvæ (worm-brood) of *Sclerostomum equinum*, and therefore are not so often affected with an aneurysm as horses that are pastured part of the year, and obtain their water for drinking from stagnant pools, ditches, small currents, etc

Prof. Friedberger, in his already cited treatise, says: "Worm-aneurysms, surely, are not found less frequently in military horses than in others, and the fact they (the military horses) are generally not so old, does not alone suffice to furnish an explanation." Prof. Friedberger does not advance any reason upon which he bases the above opinion, and seems to have left out of consideration the fact that the embryos of the worms live in water, but only in water that is exposed, and therefore accessible to them, and that these embryos do not occur in well water, or in water not exposed, and therefore not accessible to the worm-brood. Hence, as the embryos are picked up by horses with their water for drinking water, and in no other way, they cannot very well invade a horse that does not drink any water that contains them.

I perfectly agree with Prof. Friedberger when he indorses Prof. Bollinger in the following language: "At any rate we must agree with Bollinger when he says: 'Partial or complete paralysis of a portion of intestine, and thus produced intestinal stenosis undeniably constitutes in herbivorous horses, in a majority of cases, the initial point of those symptoms which we comprehend under the name of colic.

"'Intestinal paralysis and a hindered moving on of the contents of the intestines constitute, in a majority of cases, the cause of the phenomena'" (symptoms).

THE SYMPTOMS.

As colic is only a collective name for different morbid processes, which have but that in common that they have their primary seat in the digestive organs, and are attended with manifestations of great pain, the symptoms, naturally, must vary in different cases. They usually make their appearance rather suddenly, and become somewhat characteristic by the behavior, and especially, the restlessness of the animal, but as to degree

and intensity they widely differ, and, of course, not all of those below enumerated will ever be found united in any single case of colic.

I will first mention such as are most conspicuous and characteristic, and therefore of diagnostic value.

1. *Changes in the behavior of the animal and signs of pain.* A colic patient has no appetite, neither for food nor water; paws with the fore feet; strikes with the hind feet towards the abdomen; steps to and fro from one side to another; switches with the tail; looks back towards the seat of pain in the abdominal cavity; puts all four feet close together as if attempting to lie down; hesitates, but soon attempts again; does lie down, then perhaps lies quiet for a moment or two, but feels uneasy, and rolls, or jumps up, but soon goes down; becomes more restless, repeats jumping up and lying down; when up shows increased restlessness; sometimes acts as if trying to make water, but none is voided; perhaps makes a few steps forward, or moves in a circle; paws more violently; lies down and jumps up more frequently; when down shows great uneasiness, rolls and tumbles from one side to another; when rising remains perhaps for a short time in a half-raised position, or may not rise and may get down again; paws and kicks when lying as well as when on its feet; when up paws the bedding together in one heap, and often acts as if intending to lie down with the abdomen right on the heap of bedding that has been pawed together, but usually misses it. Gradually all the actions become more violent and more reckless, and although the animal at first apparently looked for a suitable place to lie down, the same, very soon, if the pain grows worse, goes down, even throws itself anywhere, without regard to anything. When down it often strikes and bumps the head on the ground; the rolling, tumbling, kicking, pawing and striking becomes more violent, and thus it often happens that the patient throws or works itself into a very uncomfortable or awkward position, from which it may not be able to rise without help. In other cases, but especially if the disease has continued for some time, the animal may lie rather quiet, or in a soporous condition—be perfectly indifferent to its surroundings—and can only with difficulty be induced to get on its feet. If driven up, some restlessness, manifested by pawing, kicking, groaning, etc., may again appear, but soon the patient will lie down and relapse into its former stupor. Meanwhile, the indifference to surroundings increases, and the horse, if driven up, may show just as much stupor when standing, as when lying down. In other cases the patient may lie on its side, stretch out all-fours, and groan, and grate its teeth. Others, again, while standing or lying down, but usually when resting on the breast-bone, may hastily pick up a few straws from the bedding, but they will soon be dropped and will not be eaten.

In some cases the patients, when down, assume, sometimes persistently, very unnatural positions; for instance, sit on their haunches like a dog, or lie on their back, all-fours extended upward, or else the joints may be flexed and the feet be almost resting on the abdomen. Still others stand up with their hind feet, but forward rest upon their knees like a cow attempting to rise. These unnatural positions are usually looked upon as an indication of the presence of a mechanical obstruction in the intestinal canal, such as is caused by a stricture, an internal hernia, an intussusception, a displacement, a torsion, etc. Still, in some cases, animals showing these symptoms have survived, and in other cases one or another of the just named morbid changes has been found at the post-mortem examination, and these unnatural positions were not assumed while the animal was sick. On the whole, however, I do not like to see these abnormal positions, and regard them as a bad sign.

Again, other patients, while lying down, show a tendency to rest on the sternum, and stretch out their fore-legs forward, and others, when standing, stretch themselves and bend the back downward or sideways, while in some, considerable weakness in the hindquarters may be observed.

The facial expression of a colic patient, too, is somewhat characteristic. It shows that the animal suffers intense pain; especially the look of the eye is often staring and wild, or in an advanced stage of a very severe attack, sometimes dull and broken, or glassy.

The visible mucous membranes do not show conspicuous changes in the beginning, or in a slight attack; but if the latter becomes severe, the mucous membrane of the mouth, particularly the gums, become dry and sticky, and if the attack is a very severe one, but especially toward a fatal termination, the same exhibit a bluish or purplish-brown color, with a livid seam around the incisors. The nasal mucous membrane usually looks as if coated with dirt, and is otherwise more or less reddened, according to the severity of the attack, and if the disease is very severe, or the animal about ready to die, a cyanotic color may be observed. The conjunctiva is usually more or less reddened. As a rule, the visible mucous membranes show increased redness in various degrees, but cases occur, in which the same are abnormally pale or anaemic. This happens, if nearly the whole blood is accumulated—locked up by the morbid process—in the abdominal cavity, and therefore must be looked upon as an unfavorable sign.

As a symptom of great prognostic value, must be considered the pulse. In very mild attacks, it is but moderately accelerated, and almost normal in quality, but if the attack continues and becomes severer, and if the animal gets very restless, rolls and tumbles a good deal, the pulse rapidly increases

in frequency, and may run up to 50, 60, 70, or even 80 beats in a minute. The acceleration of the pulse, it seems, depends principally upon two things: First, upon the extent and the gravity of the disturbance of the circulation in the abdominal cavity; and secondly, upon the degree in which the blood has become poisoned with noxious gases. So it may happen that in very severe cases, the pulse may rise to 80, 90, 100 beats in a minute, or even higher. As to its quality, but little changed in the beginning, or if the attack remains a mild one, soon a change will be observed if important morbid changes make their appearance. Then the pulse often becomes hard, irregular, weak, or wiry, and finally, when a fatal termination is approaching, not seldom imperceptible.

I said the pulse is of great prognostic value. It may be taken as a rule, which suffers but few exceptions, that as long as the pulse is below 60 beats in a minute, there is very seldom any danger; between 60 and 80, the dangerous stage commences; cases, in which the pulse runs above 80 beats in a minute, must be considered as decidedly dangerous, and requiring prompt attention, if anything can be done; and finally, cases in which the pulse is 100 or above, must be considered as hopeless. I will say, however, that as to the number of pulses in a minute, an examination made immediately after the animal has been down, or has got on its feet, cannot be relied upon, because the pulse will be fluttering, and, at least in severe attacks, be considerably higher than it will be a minute or two later. Hence, if the pulse is examined immediately after the animal has been down, the examination, in order to be reliable, must be continued until the pulse becomes steady, or does not show any decrease in, say, half a minute. The quality of the pulse, too, is of prognostic value. As long as the quality is normal, or nearly so, there is no danger. A hard, and also an abnormally weak pulse indicate that severe morbid changes are existing; a wiry pulse indicates imminent danger, and if the pulse becomes imperceptible, it is to be regarded as a sign that the end is approaching.

The best place to examine a horse's pulse is on the inside of the foreleg, close to the body, on the radial artery, which is easily found, and large enough to distinctly show the qualitative differences, which are much less plain in the smaller arteries. But if a colic patient is very unruly, it is sometimes impossible to get at that artery without incurring considerable danger. In such cases the pulse is best examined on the maxillary artery, which bends around the lower jaw-bone in the region of the third and fourth molars. If it should be impossible or dangerous to get at this artery, too, the coccygeal artery in the median line on the lower surface of the tail is nearly always accessible, but it is insufficient to distinguish fine qualitative differences, and can only be used to ascertain the number of pulses.

The beating of the heart can be felt in some cases, while in others it cannot, but if the disturbance of the circulation, and the arterial pressure is very great, it is not seldom throbbing.

The temperature is undoubtedly very much above normal in all severe cases, except, perhaps, immediately before death, but for obvious reasons it will be next to impossible to apply the thermometer a sufficient length of time; neither is it necessary, because other symptoms give us all the clues needed for the prognosis.

The respiration is not essentially accelerated at the beginning, or in very mild attacks, but will become so, if the animals get very unruly on account of the pain. It will be the more rapid, difficult, and even dyspnœic, the more violent the attack, but especially, the greater the surcharge of the blood with the noxious gases developed by the contents of the intestines as the cessation of the digestive process. It also will be the more accelerated, the greater the pressure upon the diaphragm by the distended intestines. Hence, as a rule, the respiration is the more accelerated, and the more dyspnœic the more rapid and the more extensive the development of gases. Still, in some cases, the respiration becomes extremely fast and even distressing, may increase to 80 breaths in a minute, without any conspicuous expansion of the stomach or intestines being present; but in such cases the poisoning of the blood with carbonic acid and other noxious gases is of the utmost degree, and a fatal termination is imminent, especially if the pulse, at the same time, runs up to about 100 beats in a minute or over, and is wiry or imperceptible. It may not need any mentioning that previously existing respiratory disorders, heaves for instance, will very much contribute to accelerate the respiration. In extreme cases, a few minutes before death, the animals sometimes breathe through the mouth.

The flanks are usually more or less full or even tympanitic, while the abdomen is often drawn up, because the abdominal muscles are in a state of contraction. It will also hardly need any mentioning that if the stomach and intestines are expanded by gases, the flanks will be full and tympanitic in a corresponding degree.

The peristaltic motion of the intestines is in most cases considerably diminished, and only in comparatively few morbidly increased, while in others, especially in such, in which the contents of the intestines are not moving at all, no peristaltic noise whatever can be heard. In some very severe cases, finally, particularly if a fatal termination is approaching, a peculiar noise, resembling that produced by a drop of water falling into an empty kettle, is the only thing that can be heard. It seems like "ting," "ting," and according to my observations, must be regarded as a very bad sign.

Appetite for food and drink does not exist. If either is taken, it is almost always a sign that the animal is recovering, or that the disease is not a true, but a spurious colic; in other words, that the seat of the morbid process is not in the digestive canal, but in some other part—in the peritoneum, for instance. The same applies to the voiding of excrements and the discharge of urine. If either takes place, the animal is either not severely affected, is recovering, or the disease is spurious colic.

If in true colic the stomach or intestines are expanded by gases, some horses will commence to belch, or will pass wind, and this is usually a favorable symptom, because considerable quantities of gases may thus be discharged. In other cases, retching and even vomiting may be observed; neither can be considered as favorable, especially vomiting, which is looked upon as a bad symptom, and usually regarded as an indication of a rupture of the stomach. Still, I have seen horses vomit and recover, consequently the stomach cannot have been ruptured, and I have also seen horses vomit and die, and at the post-mortem examination no rupture could be found. Vomiting, therefore, is not a sure sign that such a rupture has taken place. If the animal retches, the mucous membrane of the mouth, usually dry and sticky in severe cases, is often abnormally moist. Vomit is usually discharged through the nostrils on account of the great length of the soft palate in horses. In some cases, however, through both nose and mouth.

I remember one case, in which a horse had become somewhat bloated in the pasture, and when it was taken home and put in the stable, it vomited a bucketful of grass and fluid. There were no real colicky pains, and the horse perfectly recovered.

Except in very mild cases, nearly always more or less perspiration will be observed, especially if the patients are very restless and uneasy. Still, a profuse perspiration is not, under all circumstances, a sign of great danger. But if the perspiration is not general, but partial, and the pulse and respiration, at the same time, are accelerated to the utmost, or if the sweat is clammy, and the skin cold to the touch, or if the perspiration suddenly ceases, and the skin becomes cold and hard to the touch, a fatal termination may soon be expected.

If the symptoms, just described, are analyzed, it will be found that some of them, which happen to be the most conspicuous, and are always present, though in different degrees, consist merely in manifestations of intense pain—cramping and griping—in the digestive canal; that others, not always present, but in some cases dangerous, are the immediate result of the interruption of the digestive process, and that still others, which present themselves fully developed only in very serious cases, and always indicate imminent danger, proceed from an intoxication or poisoning of

the blood with the products of the decomposition and fermentation processes, consisting of noxious gases and other delcterious matter.

COURSE AND TERMINATION.

An attack of colic, like that of any other disease, will last as long as the causes which produce the morbid process continue to act. It will terminate in recovery, if the causes are removed, or cease to act before the morbid changes produced have become such as involve a cessation of some vital function, or have developed to such an extent as to become irrepairable. In some cases morbid changes fatal to life—for instance, a rupture of the stomach, or of one of the large intestines, an excessive poisoning of the blood with the products of fermentation and decomposition, etc.—will be produced in a short time, and then the attack will speedily terminate in death. In other cases the causes—constipation, for instance—are not, or cannot be, speedily removed, but the morbid changes may develop rather slowly, or do not soon become irreparable or dangerous to life, and then the attack may last quite a while—twenty-four hours, or even longer—and still terminate in recovery.

The shortest duration is usually observed in those cases, in which the cause consists in embolism or thrombosis in one of the intestinal arteries, and in which the thus interrupted circulation is soon restored through the anastomoses with the nearest arteries; because as soon as this takes place, the morbid symptoms will disappear. So it may happen that an attack of colic, in which the manifestations of pain are rather violent, will terminate in recovery in half an hour to a few hours, and, may be, even sooner. In such a case, however, pulse and respiration do not become seriously affected. If the embolism or thrombosis, however, takes place in an artery which furnishes blood for a large portion of the intestines, for instance, in the superior colic, or in the ilio-cœco-colic artery, or if the circulation cannot be restored through the nearest anastomosing arteries, because already obliterated, irrepairable or dangerous morbid changes, for instance, a severe intoxication with the products of fermentation and decomposition, a rupture, a displacement, a torsion, an intussusception, a strangulation, or even gangrene of an intestine may result, before the circulation is restored. In such a case the disease will have a fatal termination, but its duration will depend upon the nature of the morbid change. The shortest duration, if the termination is a fatal one, is usually observed, if large quantities of fermenting food constitute the cause. In such a case the process of fermentation rapidly develops large quantities of noxious gases, which not only expand the stomach and intestines, and in that way cause great pressure upon the diaphragm and upon the large blood vessels in the abdominal cavity, and, may be, a rupture of the intestine itself,

but also a rapid poisoning of the blood by the diffusion and absorption of the noxious gases.

Thus it may happen that such a flatulent, wind, or fermentation colic sometimes terminates in death within half an hour; but then the immediate cause of death is always either a severe intoxication with the products of fermentation and decomposition, the noxious gases, or hemorrhage. If the stomach or one of the large intestines becomes ruptured, the termination, unless hastened by a simultaneous absorption of poisonous gases, and subsequent suffocation, or by hemorrhage, is somewhat slower, because death, in such a case, will not be the immediate result of the rupture, but be caused by the peritonitis (inflammation of the serous lining of the intestines and the abdominal cavity), which invariably will develop if an intestine is ruptured.

If the stomach or one of the intestines is ruptured while the animal is yet alive, the rupture is always produced by internal, and never by external forces, and exactly in the same way as it is if it takes place after death, viz.: by the expansion of rapidly developing gases, and not by rolling and tumbling. In the living animal a rupture of the stomach is more frequent than a rupture of one of the intestines. It nearly always takes place in the large curvature, and comparatively very seldom in the small curvature or in the lateral wall of the stomach. But it occurs only when the animal has colic, and when the stomach is full of food and gases, and its normal elasticity and contractibility are impaired, either by the weight of its contents, or by other causes. The rupture itself is always due to an excessive pressure from within, caused by the expansion of the gases. It is brought about in the same way, in which an excessive expansion of steam will cause the bursting of a boiler of an engine. If there is a weak place in the wall of the stomach, caused, for instance, by previously existing morbid processes producing degeneration, ulceration, etc., a rupture will require less force from within, and may be produced by an expansion of gases that would not seriously affect a healthy stomach. It is just the same as with a damaged boiler, which also will burst from much less pressure than one that is in a perfect condition. It is also claimed by some that an anti-peristaltic motion, due to some obstruction in the intestines, retching and attempts to vomit, may cause a rupture of the stomach. This, I think, is hardly probable, unless the walls of the stomach are damaged by a previously existing morbid condition.

For 100 years or longer a great many writers on colic have copied, one from another, the supposition that a rupture of a stomach expanded by gases might be produced by the sometimes violent actions of a colic patient, notwithstanding that not the slightest proof of the correctness of this supposition can be furnished. On the contrary, if the development of

gases, and the distention of the stomach are so great as to cause a rupture of the latter, the patient, as a rule, will be in a soporous condition, and will not act in such a violent manner as in other cases, because the stupor caused by the intoxication with poisonous gases is too great; and on the other hand, a rupture of the stomach, produced during life, is never found, if the actions of the animal have been very violent until the bloating (expansion of the abdomen) ceased to increase, which it will at once as soon as a rupture has taken place. At any rate, in all those cases, in which the stomach is found ruptured at the post-mortem examination, the animals have ceased to be very violent before the expansion or swelling of the abdomen reached its height.

If a rupture of the stomach is effected, usually the external or serous membrane will break first, the middle or muscular coat will break next, and the internal or mucous membrane, because the largest, will be the last that will give way to the pressure. Hence, the margins of such a rent in the stomach nearly always present a serrated appearance. In comparatively rare cases, if the force of the rapidly developing gases becomes suddenly very great, it may happen that all three membranes rupture at once. In such a case the rent usually is a large one.

As soon as such a rupture has taken place, the animal, for the time being, will act as if somewhat relieved, and apparently be more at ease, but there will be no other improvement. On the contrary, it is only a temporary relief which merely signifies the beginning of the end.

If the rent is only a small one, and happens to be covered by another intestine, so that but very little of the contents of the stomach will pass into the abdominal cavity, the animal may live from one to two days, but if the rupture is extensive, or if a large quantity of the contents of the stomach is discharged into the abdominal cavity, the absorption and effusion of the products of fermentation and decomposition will be rapid, and the animal will die in a short time, may be in half an hour, or, in most cases, within two or three hours. A staring, glassy look of the eye; a relaxation of the facial muscles; driveling of saliva and food-particles from the mouth and nose, or retching and vomiting; cold and clammy sweats, usually partial; a rapid, wiry, irregular, or imperceptible pulse; accelerated and dyspnœic breathing; coldness of the extremities, and loss of the vital turgor in the legs; a venous, purplish, or even livid redness of the visible mucous membranes; trembling and conspicuous weakness in the voluntary muscles; great depression of the sensorium, and reeling and staggering, indicate the rapidly approaching end. The visible mucous membranes, however, will be pale or anæmic, if the rupture extended to a

larger blood vessel, and if, in consequence, considerable hemorrhage into the abdominal cavity has taken place.

A rupture of any of the intestines—duodenum, jejunum, ilium, cæcum, and colon—wherever it occurs, is due to the same causes, an expansion of the rapidly developing gases, and it will not be necessary to explain why such a rupture usually occurs in a part previously damaged by an inflammatory, or any other morbid process

DISPLACEMENTS, STRICTURES, TORSIONS, ETC.

Although these are, in most cases, post-mortem changes, that is, changes produced after death by a rapid development of gases after putrefaction has set in, yet they are, if occurring during life, the product of the same internal forces, viz : an expansion of parts of the intestines by gases, imprisoned by the interruption of the peristaltic motion in an adjoining part of the intestines. They are never caused by any violent action, rolling, tumbling, etc., of the patient, because there is, outside of the intestines, which themselves are very elastic, no empty space in the abdominal cavity; therefore, if the former expand or contract, the walls of the abdominal cavity will do the same, and if the latter are exposed to concussion—producing external violence, the former, by acting as an elastic cushion, will effectively break the force. External violence, if acting with sudden force, a kick, for instance, may sever the continuity of the wall of the abdominal cavity—cause a hernia—but will never cause a displacement, torsion, or stricture, etc., of the intestines. Such morbid changes require forces acting from within. In all cases of true colic, there is at least one portion of the intestinal canal, in which the peristaltic motion is interrupted either by paralysis or paresis, or by some mechanical obstruction; in which, consequently, the contents are not in motion. This part, therefore, constitutes a fixed point. In other portions, anterior to this fixed point, especially it expanded by gases endeavoring to escape, the peristaltic motion often will be violent, or, at any rate, irregular, because it cannot proceed beyond the paralyzed or paretic part, and if then the gases which cannot pass on, continue to expand, the moving part of the intestine will work itself in, where, on account of the irregular peristaltic, another part will temporarily yield; thus the former may become wedged in, or be turned around its longitudinal, or rarely, and perhaps only, if it happens to be the cæcum or blind-gut, around its transversal axis. So-called internal hernia are produced in the same way. An interruption of the circulation, an inflammatory process, or an imbibition of serum, etc., may weaken the texture of the epiploön or of the mesenterium, so as not to be able to withstand the pressure of an intestine expanded by gases, or the tension caused by its violent motion. A rupture may thus be produced,

a part of a violently moving intestine may find its way through the rent, and may thus become wedged in or be imprisoned when filling up with gases or with more solid contents.

Intussusception, or telescoping of an intestine, is brought about in a similar way, by an interruption of the peristaltic motion in one part, and an increased or violent peristaltic motion in the adjoining portion of the same intestine—usually the jejunum or narrow gut—anterior to the paralytic or unmoving portion. A further explanation to show how it is brought about, will hardly be necessary.

All these conditions, just mentioned, lead to death. At first, a severe inflammation, then gangrene, and a subsequent absorption and diffusion of deleterious material will be the result, and the fatal symptoms, mentioned elsewhere, will make their appearance

A comparatively rare complication, though worth mentioning, because usually leading to death, consists of a rupture of the diaphragm. It is caused by an excessive development of gases in the stomach and intestines, and by great pressure of these organs, thus to the utmost expanded, upon the diaphragm. If the rent, which usually occurs in the membranous portion, is only a small one, and covered by a portion of the epiploon, the animal may live; but if the same is large, so that the stomach, a part of the jejunum, or portions of the large intestines pass through into the chest, the animal will die, and will die very soon, especially if, at the same time, the stomach, too, is ruptured, and its contents emptied into the chest. Such a complication as just described, however, only occurs in extreme cases of so-called wind or flatulent colic, in which stomach and intestines are expanded to the utmost.

As said above, a rupture of the diaphragm effected while the animal is yet alive, is a very rare occurrence. It happens more frequently after death, and is thus due to the same causes, an excessive development of gases, especially after putrefaction has set in.

At a post-mortem examination it is easily decided, though, whether a rupture of the stomach, of any of the intestines, or of the diaphragm, or any of the other complications mentioned, for instance, a displacement, a torsion, an internal hernia, or an intussusception, etc., is simply a post-mortem change—has been produced after death—or has taken place while the animal was yet alive, as has been explained elsewhere.

DIAGNOSIS.

The general diagnosis is easy enough, so that one who has some experience, can often decide at the first glance, whether a horse has colic or not. Still, there are diseases or morbid conditions, in which the symptoms are somewhat similar. Among these I will mention:

1. All cases of so-called spurious colic, in which the seat of the morbid process is in the peritoneum, in the sexual organs of females, or in the urinary organs. Still, in all these cases of spurious colic the real colic symptoms, that is, the manifestations of severe pain, are not so predominating as in cases of true colic, while the feverish symptoms, accelerated pulsation and respiration, etc., are more prominent. Besides that, the appetite for food and drink, but especially for the latter, is not so entirely absent as in true colic, and finally, the mucous membrane of the mouth, but particularly of the gums, is not so dry and sticky, as is usually the case in severe colic. In regard to all those diseases, in which colicky pains constitute a subordinate symptom, the fact that other symptoms, indicating the existence of another disease, are present, guards against mistakes.

2. Certain cases of poisoning, if it is not known that a poison has been given, might be mistaken for colic. The principal symptoms are as follows:

a. Poisoning with arsenious acid (arsenic). Thirst; retching; constipation, followed by fœtid diarrhœa; a much accelerated, small, wiry, irregular, and finally imperceptible pulse; very rapid, dyspnœic respiration; colicky pains, manifested by pawing with the fore-foot, looking toward the abdomen, restlessness, a frequent lying down, and a painful look of the eye; diffuse redness of the conjunctive and of the other visible mucous membranes; driveling of saliva from the mouth; a relaxation of the facial muscles and therefore drooping of the lips; depression of the sensorium; dilation of the pupil, trembling, cold sweats, and finally paralytic phenomena.

b. Poisoning with salt. Thirst; hot and dry mouth; bloating; diarrhœa; colicky pains (griping); accelerated pulse; enlargement of the pupil; redness of the visible mucous membrane; trembling; spasms; coma.

c. Poisoning with carbolic acid. Spasms and trembling; great difficulty of breathing; colicky pains; diarrhœa; dark olive-colored urine; loss of consciousness.

I might mention the principal symptoms produced by other poisons, but as the same deviate still more from those of a true colic, the above will suffice to show that always some symptoms are present in cases of poisoning in which the colicky symptoms may present some similarity to those of colic, which do not occur in the latter disease. A colic patient, for instance, never drinks any water until he has about recovered Where there is thirst there is no true colic.

The differential diagnosis is often rather difficult. In other words, it is not always easy to decide, which of those rather numerous diseases, usually called colic, is present in a given case, nor is it always possible to

determine what causes have been acting, and what morbid changes have been produced. In some cases such a decision may be immaterial, but in others it is of great importance, because the treatment may have to depend upon it. Without going into minute details, I will, therefore, briefly state the characteristic symptoms, as far as there are any, of the different kinds of colic.

1. *Spasmodic colic*, or colic caused by a sudden interruption of the peristaltic motion in some part of the intestinal canal. It probably is the most frequent form, and it seems to be especially this form, in which thrombosis or embolism, produced by some fibrinous exudation or a clot of coagulated blood, torn loose from the wall of an aneurysm in the anterior mesenteric artery, must be accused, if not as the sole, at any rate, as the principal cause, since, as a rule, none of the other agencies, usually accused as the causes of colic, has acted in sufficient force to produce the morbid process without the aid of something else as a predisposing cause. In this form the attack is usually of a short duration and not very severe, if the thrombosis has taken place in one of the smaller arteries, or at such a place of one of the larger ones, at which the circulation of the blood is only temporarily interrupted, because soon restored through the anastomoses. If, however, the superior artery of the colon, or the ilio-cæco-colic artery is closed near the common trunk, the anterior mesenteric artery, so that a large portion of the colon, or nearly the whole cæcum, is deprived of its supply of blood, and, in consequence, unable to perform its functions, the case may become serious enough, and the animal may die before the circulation can be restored through the anastomoses. On the whole, however, the attack is not of long duration, but the manifestations of pain, the pawing, kicking, rolling, and other restless actions of the animal are usually very lively, even violent, but the uneasy spells are often divided by quieter intervals of a few minutes' duration, and the other symptoms, such as a much accelerated pulse and respiration, etc., are comparatively less conspicuous, unless the food consumed has a tendency to ferment, is difficult of digestion, or the attack is uncommonly severe. In such cases all the symptoms enumerated elsewhere as proceeding from an intoxication with noxious gases will become fully developed.

2. *Colic caused by over-feeding* can be diagnosticated with certainty only, if it can be learned what, and how much, the animal has eaten. There will be a tendency to develop gases, but this, of course, will depend largely upon the kind and quality of the food consumed. There will be no peristaltic motion, and consequently no peristaltic noise, if there is any impaction in any part of the intestinal canal. If there is not, some peristaltic noise may now and then be heard. In some cases, however,

especially in such in which the food contained in the digestive canal has a tendency to ferment, but the passage is unobstructed, a very lively peristaltic noise may be heard. Such a case usually terminates in diarrhœa.

A peristaltic noise is also absent in many other cases, in fact, in all such in which the passing on of the contents of the intestines is prevented, either by paralysis or paresis of some part of the intestinal canal, or by some mechanical obstruction, for instance, by a stone, a concrement, a ball of meal or bran, an impaction, a large number of worms, a torsion, a stricture, a strangulated internal hernia, an intussusception, etc. All these mechanical obstructions do not, besides causing peristaltic silence, produce any other characteristic symptoms, except that the horses often show a tendency to assume unnatural positions, such as sitting on their haunches like a dog, lying on their backs, all-fours extended upward, or the feet dropping down on the abdomen, or kneeling down on their fore-knees, and standing up on their hind feet like a cow in the attempt to get up. Still, such abnormal positions are not, under all circumstances, a reliable indication, because it has happened that a horse assuming any one of these abnormal positions, during an attack of colic, has recovered, and in others that did not assume them, one or another of the mechanical obstructions above mentioned, has been found at the post-mortem examination. But after all, where a horse affected with colic repeatedly or persistently places itself in any one of these abnormal positions, we are justified in suspecting the existence of a mechanical obstruction, and may expect a fatal termination.

3. *Wind, flatulent, or fermentation colic* is easily diagnosticated, because the whole abdominal cavity will be expanded (bloated) to the utmost, and the flanks, in particular, will be tympanitic, like a drum-head. Its severity will depend upon the quantity and quality of the fermenting food in the stomach and intestines, and upon the completeness of the obstruction of the passage.

4. *Worm colic.* An animal suffering from worm colic always harbors a large number of worms, which, in most cases, are ascarides. Consequently such an animal is not only more or less emaciated, but also presents a somewhat cachectic appearance, and previous to the attack has shown symptoms of having worms. If all this is taken into consideration, the diagnosis is seldom very difficult. Other characteristic symptoms—except that the behavior of the animal, that is, the manifestations of pain, are seldom very violent, owing, undoubtedly, to the already weakened condition of the animal—are not existing.

In the literature of colic a great many other distinctions, some of

them very fine, have been made. I regard them as superfluous; at any rate, they cannot be diagnosticated in the living animal, and therefore are of no practical value. Besides, the fact, already mentioned, that different individual animals, as well as men, are apt to express a sensation of pain in a different manner, must not be lost sight of.

The most valuable aid to a differential diagnosis in a case of colic, consists in a knowledge of what has happened. If one knows under what conditions a horse is, and has been, kept, what and how much and when the same has been eating and drinking, what kind of work has been performed, what, at what time, and how soon after or before having been worked the animal has been fed, to what work, meal hours, and food as to quantity and quality the same is accustomed, whether the horse had an attack of colic before or not, or whether the same is, perhaps, subject to frequent attacks or not, and last, but not least, what has been done by way of treatment, the special diagnosis, in most cases, is not very difficult.

The weather prevailing when the attack of colic took place, as already mentioned, is not without influence. At least, it is my experience, that when everything else is equal, the most cases of spasmodic and wind-colic will occur during cloudy and foggy weather, and also when the air is sultry, or a storm is brewing.

PROGNOSIS.

It will not be necessary to say much under this head, because almost everything that has a bearing upon the prognosis has already been stated in the preceding chapters. Therefore, a recapitulation of the most essential indications will suffice.

The prognosis is favorable, and a speedy recovery may be expected, if the pulse is normal in quality and but slightly accelerated in freqency, if the respiration is but little affected; if the look of the eye is rather natural (not staring), if the visible mucous membranes present a normal color, and if the peristaltic motion is not interrupted—that is, if the ear applied to the sides of the abdomen can hear, what I would like to call, a "coarse" peristaltic noise or rumbling. In such a case it makes no difference how uneasy the animal may be, and how much the same may paw and roll. If a horse affected with colic, even if the attack is, or has been, a severe one, shows a tendency to eat, accepts water offered to drink, or makes dung and passes urine, the disease, if true colic, has taken a favorable turn, and the prognosis, although at first, perhaps, doubtful, or even unfavorable, is thereby changed to favorable.

Of course, it must be kept in mind, that in almost every colic the symptoms are not as severe in the beginning as afterwards, and that a case, which at first appears like a mild one, may soon become severe enough,

but in such a case the symptoms rapidly increase in gravity, and the "coarse" rumbling can very seldom be heard even in the beginning.

If it is a case of wind-colic, the prognosis must be made with a little more caution, and will, to some extent, depend upon the kind, quantity, and quality of the food that has been eaten. The prognosis, on the whole, is favorable, if the expansion of the abdominal cavity does not increase, in, say half an hour; if considerable peristaltic noise (rumbling) can be heard; if the animal commences to pass wind (a very good sign) or to belch; if pulse and respiration are not very much accelerated, and especially, if the visible mucous membranes present a natural color.

The prognosis is less favorable, if pulse and respiration are considerably increased; if especially the former rises above 60 or up to 80 beats, and the respiration above 20 or up to 30 breaths in a minute; if the visible mucous membranes show increased redness; if the gums are dry and sticky, if the animal is much perspiring, and if the peristaltic motion is retarded, that is, if but little peristaltic noise, or only an interrupted, somewhat distant-sounding rumbling can be heard in the abdominal cavity.

The prognosis becomes doubtful, if the pulse rises above 80, and becomes rather hard, and the respiration above 30; if the animal shows plain signs of delirium or unconsciousness of its surroundings; if the visible mucous membranes, but especially the gums, show a plainly developed venous redness, if the eye becomes staring, looking into space at nothing; if the sweating becomes partial or profuse, but the skin is hot or moderately warm, and at any rate, not yet cold, and if only a faint peristaltic noise—a kind of "fine" rumbling—can be heard at shorter or longer intervals of time.

The prognosis also is rather doubtful in all such cases, in which the existing symptoms remain the same without any abatement or visible improvement for six to eight hours. In wind-colic the prognosis becomes doubtful, if the expansion or the bloating of the intestines continues to increase, and if, at the same time, but little peristaltic noise can be heard, and no "wind" is passed, or not enough to stop a further expansion of the abdominal cavity.

The prognosis becomes unfavorable, if the pulse becomes hard, small and wiry, and increases in frequency considerably above 80, or up to 100 beats in a minute; if the respiration also becomes more rapid and dyspnœic, if the visible mucous membranes become cyanotic (bluish, or reddish-brown); if the sweating ceases, and the skin becomes dry and leathery, or if the sweating continues, but the skin, at the same time, becomes abnormally cold to the touch; if no peristaltic noise can be heard; if the look of the eye becomes more staring and the facial muscles morbidly relaxed so as to give the face a lengthened appearance; if the deliriousness and the

unconsciousness increase to such an extent that the animal commences to throw itself anywhere without regard to anything, and cannot any more be restrained in its actions, and if the vital turgor in the legs commences to disappear. The prognosis also becomes unfavorable, if the animal ceases to make any more violent movements, and lies down in a stupor, perfectly indifferent to anything that is going on. Retching and vomiting, as has already been mentioned elsewhere, must also be considered as symtoms indicating great danger.

The prognosis, finally, becomes absolutely bad, if the hard and wiry pulse becomes accelerated to above 100 beats in a minute, or if it becomes imperceptible; if the respiration already accelerated to the utmost, becomes very dyspnœic and distressing, or if the animal commences to breathe through the mouth; if the visible mucous membranes, especially the gums, become cyanotic in the highest degree, or bluish-brown with a plainly developed livid border around the incisors; if either no peristaltic noise whatever, or only a "ting"—"ting" sound, as if a drop of water is falling into an empty kettle, can be heard; if the eyes are drawn back into their sockets and look broken or glassy; if the sweat, usually partial, becomes cold and clammy, and the skin abnormally cold and parchment-like; if the last rest of vital turgor has disappeared from the legs, which then not only are cold, but appear to the touch as if the skin sticks almost immovably to the bones and tendons; if the animal lies in a stupor entirely unconscious of everything, and cannot be roused any more to make an effort to get up, or if the same is on its legs, but is hardly able to stand, staggers to and fro, or leans for support on a wall, etc.

Whenever these symptoms, just enumerated, are present, no matter whether other unfavorable, or usually fatal symptoms, such as vomiting, etc., are added or not, the case must be considered a hopeless one.

MORBID CHANGES.

I cannot give an exhaustive account of the morbid changes found after death, at a post-mortem examination, because want of space forbids it; neither will it be necessary, because the morbid changes taking place can easily be inferred from what has already been said. A brief account therefore may suffice:

First the blood. It is invariably dark-colored, and even blackish-brown, especially in all such cases in which the animal died of suffocation, resulting from a poisoning of the blood with noxious gases, and in which, in consequence, the dyspnœa or difficulty in breathing, and the acceleration of the respiration was very great. In some of those cases, in which a paralysis of a part of the intestinal canal, produced by thrombosis or embolism in some branch of the anterior mesenteric artery constituted the principal

causes of the disease, the dark color and the imperfectly, or not at all, coagulated condition of the blood constitute the only important or conspicuous morbid change, if we except some hypostatic redness and an exudation of bloody serum in the walls of the intestines and of the abdominal cavity. As other morbid changes often occurring, either singly, or two or more combined, may be mentioned: Inflammation products and inflammatory changes of different degree and extent in the intestines and peritoneum; necrosis in the intestinal mucous membrane, and even perforation of the intestinal wall, produced by wedged-in stones and concrements, etc.; perforation of the stomach or an intestine caused by ulceration; displacement of the stomach or an intestine; a stricture, a torsion, or an invagination of an intestine; strangulation of a part of an intestine that may have found its way through a rent in the mesenterium or diaphragm, etc.; a stricture of an intestine by a strand of connective tissue or by a tumor, a lipoma, for instance; a rupture of the stomach, of the cæcum, or of the colon; a rupture of the liver, or of the diaphragm; blood extravasations or hemorrhage, either in an intestine or in the abdominal cavity; paralysis of the stomach, etc.

Besides these morbid changes, just enumerated as occurring in the abdominal cavity and its organs, some others, also the direct product of the morbid process, are usually found in the chest, especially if suffocation produced by an intoxication with noxious gases constituted the immediate cause of death. The same consist in congestion and œdema, and sometimes emphysema of the lungs, an incipient degeneration of the heart, ecchymoses, blood-extravasations, etc.

Some morbid changes, finally, which are not directly connected with the morbid process of any of those diseases or morbid conditions usually called colic, may yet deserve a mentioning. They are often found in the lungs of horses, which, while sick, were drenched with some kind of oil, or with fluid, which contained insoluble or undissolved powders in suspension. The same are produced, if a part of the drench is poured down through the larynx and trachea (windpipe) into the lungs, and consist of congestion, exudation, incipient hepatization, blood-extravasations, and, in some cases, of incipient ulceration, or even gangrene in the bronchi, bronchial tubes, and lung-tissue. In many cases of colic, which terminated in death, but would have ended in recovery, if the horses had not been drenched, and the medicines been poured down into the lungs, these morbid changes constitute the real cause of death.

CHRONIC OR HABITUAL COLIC.

Some horses suffer from what may be called chronic or habitual colic, and it may not be amiss to say a few words about it. Such horses have

repeated and rather frequent attacks, often not more than a few days apart. These attacks, in most cases, are due to a paresis (incomplete paralysis) of the cæcum, but may also be caused by a great many other morbid conditions; for instance, by an adhesion existing between an intestine and the wall of the abdominal cavity, or between the stomach and the diaphragm; by the presence of stones, concrements, or an accumulation of earth or sand, or other insoluble or indigestible substances in the stomach or in one of the intestines; by a morbid distension and partial paralysis of the stomach; by a rupture of the diaphragm, etc. But, as said above, the most frequent cause consists in paresis of the cæcum, produced by an irregularity of the blood-supply on account of embolism in some of the branches of the ilio-cæco-colic artery. Usually several, and, may be, many attacks take place, but one will be the last by becoming fatal. At the post-mortem examination, a rupture and paralysis of the cæcum or the colon will be found, in most cases at least, while in others the morbid changes will correspond to the causes.

TREATMENT.

It will hardly need an explanation, that the treatment will depend upon the differential diagnosis, if the same can be made. But as this is often impossible, it has to be based, in a great many, perhaps in a majority of cases, upon general principles. In all cases of colic the peristaltic motion, and the moving on of the contents of the intestines is more or less interrupted, consequently the treatment, in all cases, must be such as is best calculated to promote a restoration of the peristaltic motion, and a moving on of the fæces, and everything that has a contrary effect must be avoided. Other existing symptoms, especially such as are dangerous, or may become so at any time, also require our attention. So, for instance, where gases are developing in the stomach and intestines, the life of the animal may become endangered at any time.

The first thing to be done in a case of colic is to put the patient, if possible, into a spacious stall or loose box, not occupied by any other animal, and containing nothing on which the usually restless and unruly animal can injure itself; to provide there an abundance of dry bedding (straw or saw-dust), and then to allow the horse to roll and paw as much as he pleases. The rolling, etc., has usually some good effect, in so far as it promotes a restoration of the locally interrupted circulation and peristaltic motion, probably more than anything else. But, of course, the attendant must see to it that the animal does not injure itself, and does not get into an awkward position. Some authors, and even some practitioners, are afraid that rolling, etc., might cause a rupture or a displacement of an intestine, a torsion, or an internal hernia, etc., and do not allow it, or en-

deavor to keep the horse on his feet. These fears, however, are without foundation, because: 1, there is not a single case on record, in which, on an unprejudiced investigation, a displacement, rupture, torsion, internal hernia, intussusception, etc., found at a post-mortem examination, can be attributed to the rolling, etc., of the animal. 2. All the intestines of a horse, notwithstanding the comparatively great length of the mesenterium, and the rather large size of the cæcum and colon, are so well fastened and secured, and so elastic, that on a well-littered floor no rolling or other violent movement of the animal itself can produce any rupture, displacement, torsion, internal hernia, etc. There is no empty space in the abdominal cavity, because all the space available is occupied by the intestines, and the latter themselves are very elastic, which enables them to resist any external force that does not meet the surface of the body with greater swiftness than that caused by the actions of the animal. 3. In most cases all such ruptures, displacements, torsions, internal hernia, etc., as may be found at a post-mortem examination, are produced after death, while the animal, most assuredly is not pawing, rolling, or acting in a violent manner. 4. These morbid changes, if produced during the life of the animal, owe their existence to the same internal forces, acting from within, in the interior of the intestines, viz.: excessive development of gases, unable to escape and forced to expand by an interruption of the peristaltic motion, which produce them after death. 5. There is not a solitary case on record in which such morbid changes have been produced in a healthy horse by any amount of rolling or other violent movements. Hence, there is no need whatever of preventing such voluntary movements of a colic patient; on the contrary, it can very often be observed that they increase the peristaltic motion, or promote its reappearance where it is absent. That they are well calculated, perhaps more than anything else, to act favorably upon the circulation and distribution of the blood, and to promote a removal of existing obstructions by the alternate contraction and relaxation of the muscles, which become active when the animal lies down and jumps up, rolls and paws, etc., there can be no doubt.

It has often been recommended to exercise a colic patient. If it is simply walking exercise, there may be no objection, but if, as often done, a man or boy is put on the horse's back, who, by constant whipping, compels the suffering animal to trot or to gallop, there are very grave objections. In the first place, such an exercise has not, and cannot have, the massage-like effect upon the circulation of the blood in the interior of the body as the rolling, jumping up, lying down, etc., because different muscles are acting, and their activity is in a different direction. Secondly, such forced exercise will still more accelerate the pulsation and respiration, in most cases already high enough, and compel heart and lungs to perform

their functions under still greater difficulties. At any rate, experience teaches that nearly all cases of colic have a fatal termination, in which the animals are treated to a good dose of such forced exercise. I consider it far better not to restrain them in their voluntary movements, even if they should become rather violent. Still, if the restlessness should become excessive, so as to seriously accelerate pulse and respiration, or to exhaust the animal, a subcutaneous injection of hydrochlorate of morphine—three to seven grains dissolved in distilled water, and to be injected beneath the skin on both sides of the neck—usually will soon quiet the animal. There is only one objection, and that is, morphine has a tendency to retard the peristaltic motion. Besides that, the effect of morphine is not the same in all horses. If the dose just mentioned should not have the desired quieting effect, it should under no circumstances be repeated more than once, because in some horses it has, instead of its usual, narcotic, an irritating effect, and causes greater excitement; an overdose, therefore, is apt to make such a horse perfectly wild and unmanageable.

If a rapid development of gases is taking place in the large intestines, and great danger is apprehended on that account, the bloated intestines may at once be relieved by puncturing. As a rule, the operation should be performed at the right side, at a point equidistant from the last rib, the transversal processes of the lumbal vertebræ, and the external angle of the ilium—the hip. The instrument to be used should be a small, round trocar, made especially for that purpose, and used for nothing else. It should be about 4 inches long, and one-sixth of an inch in diameter. The operation, if properly performed, is not dangerous. First, the hair should be cut away at the point at which the trocar is to be inserted, at a place as large as half a dollar, then the skin at that place should be washed (disinfected) with a weak solution of corrosive sublimate (1:1000), then with a small, perfectly clean lancet a cut, say one-fourth to one-third of an inch in length may be made through the skin, so as to facilitate the introduction of the trocar, especially if its stiletto is without a shoulder. This done, the perfectly clean and disinfected trocar (it is best disinfected with a 5 per cent. solution of carbolic acid) is placed at a right angle to the surface of the skin, with its point in the incision, and pushed through. The stiletto is then withdrawn, and the gases are allowed to escape. If the tube of the trocar should get clogged by particles of food, etc., the obstacle is removed by temporarily inserting the stiletto. When the trocar is to be removed, the stiletto must first be inserted again, so that no gases, fluids, or parts of the solid contents of the intestine may follow the withdrawal of the trocar, and enter the abdominal cavity, or contaminate the wound, and care must be taken to place thumb and finger on the skin at the sides of the trocar, and to press back, when the latter is pulled out, so

as to prevent a separation of the skin from the subcutaneous tissue. After the trocar has been removed, a little iodoform, or a little tar may be applied to the wound. This, however, is not absolutely necessary, if all other precautions have been taken, because the wound itself will be very small. The animal, in a majority of cases, will feel immediate relief. The intestines will have collapsed; the pressure upon the blood vessels, caused by the expansion of the intestines, by the gases, will cease; the circulation of the blood will be freer, and as a large quantity of gases have been removed, the absorption of the same will cease, or at least be very much diminished; the respiration, therefore, will be slower, and be less distressing. Consequently such a puncture of the intestine will not only avert immediate danger, for instance, a rupture of the expanded intestine, or a fatal poisoning of the blood with noxious gases, but it may also, indirectly, contribute to effect recovery, at least in so far as a slower and easier respiration and circulation will favor it. It is true, the operation does not directly remove the source of the gas production, neither can it save the life of the animal, after the intestine is ruptured, or after the other morbid changes, for instance, the poisoning of the blood with noxious gases, or the development of gangrenous processes, etc., have already passed the fatal point—in other words—where the operation is performed too late. If executed in time, and with the necessary precautions, it may not save the life of the animal in every instance, but it will do no harm under any circumstances, and therefore is indicated in all such cases, in which an excessive development of gases demand speedy relief.

Where the difficulty of the respiration is very great on account of an excessive accumulation of blood in the lungs, and where, at the same time, the visible mucous membranes show plainly developed venous redness, it may become necessary to bleed the animal from the jugular vein. But if bleeding is resorted to, it should be done in such a way as to cause a rapid flow of blood, and the fleam to be used should not be too narrow nor too pointed.

Bleeding, in such a case, will afford immediate relief, especially if suffocation is threatened, provided, of course, it is resorted to in time, while the blood is yet able to flow. This, however, does not mean that every colic patient should be bled; on the contrary, bleeding should not become a rule, and not be done unless strictly necessary.

As in nearly every case of colic, the peristaltic motion of the intestines is retarded, or in some part of the intestinal canal altogether interrupted, it must be one of the principal objects of the treatment to promote, and, if possible, to restore the peristaltic motion. Hence, injections of warm soap-suds, or of oil into the rectum are indicated. It is true, they may, even in a majority of cases, have but little influence, but they can do

no harm in any case, provided, of course, the injections are not made in a rude and careless manner, and are not overdone.

Friction applied to the walls of the abdominal cavity by rubbing the same with a wisp of straw or hay, or with a piece of a woolen blanket, etc., will have a tendency to promote the peristaltic motion, and the restoration of a normal circulation of the blood in the intestines, and is therefore of great value, usually underestimated.

If the peristaltic motion is very tardy, or is totally interrupted, but especially if the existing colic has been caused by overfeeding, a good physic, a good dose of aloes for instance, will very naturally suggest itself, and, indeed, will be the very thing best calculated to set the contents of the intestines in motion. If aloes is used, the de Barbadoes deserves preference, because its effect upon horses is more reliable than that of any other kind of aloes. Still, even the de Barbadoes acts but slowly, and in many cases too slow to save the life of the animal. The dose to be given will depend, first, upon the age, size, and constitution of the animal, and secondly, upon the kind of food the same has been eating. To illustrate, if, for instance, an ounce is the proper dose for a medium-sized or rather small, full-grown horse in the winter, or when kept on dry food, six drachms would be more than enough for the same animal in the summer, or when green and juicy food, grass, has been fed. Aloes, it seems, is the most effective, if mixed with a small quantity of soft soap, just enough to make a pill of proper consistency, provided, the pill is freshly prepared, and not old and hard. Its action, however, is milder, if it is mixed with althææ root powder and water, and given in that way, in the shape of a pill, the danger of giving too much is considerably lessened. It is often claimed that fluid medicines come sooner to action than medicines given in shape of pills or electuaries. The difference, however, is insignificant, provided the pills or electuaries are freshly prepared, as they should be. Besides that, to give medicines in a fluid form, as a drench, to a horse, especially if the respiration is very much accelerated, as is usually the case in colic, is exceedingly dangerous, because fluid medicines often go the wrong way, pass into the larynx, and from there into the trachea and into the lungs, and if then the fluid medicine contains insoluble powders in suspension, or is composed of some oil, or contains grease, the horse will die of pneumonia. The powder will stick to the mucous membrane and lodge in the ramifications of the bronchi, will act as foreign bodies, and even close the ultimate air passages, and as they cannot be removed, a fatal pneumonia will be the result. Oil and grease are just as bad, because they cannot be absorbed, and likewise continue to act as foreign bodies. Of all the oils occasionslly used, not only by quacks, but also by some practitioners, fish-oil seems to be the worst, probably, because taking it is very

objectionable to a horse. Oil, at any rate, is not a suitable medicine for a horse; its physicking properties are very slight; it has a bad general effect upon the process of digestion; it thoroughly destroys the appetite for about a week, and its action is a slow one. Aloes, which is much preferable, it is true, also acts too slow in a good many cases, but the same applies to nearly all other physics, some of which act considerably slower yet. Professor Dieckerhoff, in Berlin, therefore recommends hypodermic injections of sulphate of physostigmin or eserine, dissolved in distilled water. It offers several advantages. In the first place, an hypodermic injection is easily made, and is not attended with any danger, even if the animal is ever so unruly, and secondly, physostigmin, if it acts at all, evacuates the bowels much quicker than anything else; but there are also some disadvantages. Physostigmin or eserine is an alkaloid, which contains the effective, and therefore very poisonous, principle of the calabar bean; it consequeutly is dangerous, and its effect is by no means reliable; besides that, its action upon the nervous system is not always what is wanted. The dose is from one to two grains dissolved in one or two drachms of distilled water. Another medicine, which I have often used in combination with aloes, is camphor. It acts upon the skin, increases its activity, draws blood away from the interior organs to the periphery of the body, besides that, it increases the vitality of the blood, and acts as a tonic upon the nervous system. All this, and its unquestionable antiseptic effect, may explain its favorable actions if given in suitable doses, that is, from one to two drachms, to be repeated in three to four hours.

If inflammatory processes, especially in the peritoneum, are developing, a good and promptly acting counter-irritant, applied to the flanks, will have a tendency to reduce the inflammatory process in the interior of the abdominal cavity. A mixture of equal parts of oil of turpentine and ammonia will answer. If applied, and no reaction takes place in, say, about twenty minutes, it is pretty safe to make an unfavorable prognosis, but if a prompt reaction sets in, the animal, very likely, will recover. Its application, therefore, is of some prognostic value. If the first application does not have a sufficient effect, another one may be made half an hour to two hours later, but to make more than two applications may be productive of a permanent blemish (loss of hair).

It has often been recommended to give internally, in cases of wind colic, that is, where stomach and intestines are distended by gases, some alkaline substance, that will form a chemical combination with these gases, and thus render them harmless. Theoretically, such a treatment appears to be all right, but practically there is very little merit, if any at all, in it. As such alkaline substances have been recommended, Liq. ammonia, of course, sufficiently diluted with water; Hepar sulphuris, or sulphur of

potass., a few drachms dissolved in some aromatic infusion; limewater, etc. But these alkaline substances, at best, only bind a small quantity of the gases, and therefore are of very little use, and their administration, which has to be in a fluid form, involves danger.

Some practitioners give as a physic large doses of sulphate of soda, sulphate of potash, or sulphate of magnesia. There is but one objection to these salts. If given in the shape of pills, or as an electuary in combination with a sufficient quantity of vegetable powders, they are too voluminous, and to drench a colic patient, especially if the respiration is much accelerated, is exceedingly dangerous. I know what I am talking about, when I say that every year more colic patients are killed by careless drenching than are cured by medication.

If in a male animal colic is caused by an incarceration of an existing scrotal hernia, an endeavor must be made to effect a reposition without delay. If it cannot be done without a surgical operation, then the latter must be performed without any hesitation, even if the sick animal should be a valuable stud-horse. To describe the operation would lead too far, and require more space than is allowed me.

From what has been said, especially in regard to the causes of colic, it will appear that in a majority of cases comparatively little can be accomplished by medication. At any rate, without indulging in theorizing, it may be safely said that all those colic patients which do not recover long before most of the medicines, usually administered, can come to action, are in imminent danger.

To sum up, therefore, the treatment, as far as medication is concerned, may be reduced:

1. To giving a good physic whenever an insufficiency or absence of peristaltic motion may call for it, and to give as such either aloes de Barbadoes, or where no time is to be lost, from 1 to 2 grains of physostigmin, or sulphate of eserine, as an hypodermic injection. Croton oil is recognized as the most powerful agency to compel peristaltic, but it is an heroic and powerful medicine, acts even slower than aloes, and cannot very well be given, except in combination with some fat oil, which, being very objectionable to horses, easily goes the wrong way, and is not to be recommended, as has already been explained. The dose of croton oil, for a full-grown horse, is from 20 to 25 drops in four to five ounces of some fat oil. Besides a physic, injections into the rectum with soap-suds, clean water, or oil, may find an application in nearly every case.

2. Where it is desired to quiet an animal that is very restless and in great misery, a subcutaneous injection of morphine is more effective, and less objectionable than giving opiates by way of mouth. The dose of

morphine (the hydrochlorate of morphine is to be preferred) to be subcutaneously injected is from 3 to 7 grains.

3. When the intestines are dangerously disturbed by a rapid development of gases, the trocar should be used without any hesitancy, and before it is too late, but, of course, always with the necessary antiseptic precautions.

4. When a tonic is indicated, a drachm or two of camphor may be given in combination with other suitable medicines, or else a subcutaneous injection of ether may be made.

5. In cases of peritonitis, a promptly acting counter-irritant, applied to the flanks, will prove to be beneficial.

I know very well that a good many other remedies have been recommended by several authors, and are used by several practitioners, but it seems to me, most of them are useless, and some of them are even worse than that.

I therefore will say only a few words more in regard to treatment: In all those cases of colic, in which the cause consists in an obstruction that cannot be removed, or in which irreparable morbid changes have taken place, any treatment will be in vain. A further explanation will not be necessary.

In some cases the horses have a tendency to lie down in a stupor, and if the ear is put on the sides of the abdomen, it will be found that no, or but very little, peristaltic noise can be heard. If such horses are driven up, very often some noise will appear, though, perhaps, only for a short time. Still, where peristaltic noise can be heard, some motion is going on. I therefore never allow such a horse to lie down in its stupor any longer than, say, twenty minutes and have often found that every time the horse is driven up, some more peristaltic noise will appear, until finally the peristaltic motion is restored, and the animal has a passage and recovers.

When a colic patient, especially after a severe attack, shows signs of returning appetite, and other symptoms of recovery, special care must be taken to keep the same for a day or two on a strict diet, to give but little food, and only such as is easy of digestion. Especially the first meal must be such (a light bran-mash, for instance, or a few handfuls of nice, sweet, green grass), as will make no severe demands upon the digestive organs. If after recovery the animal is thirsty, all the water desired may be given, but only gradually, and in small quantities at a time. That an animal after a severe attack of colic should remain exempted from work, until fully and completely restored to health, may not need mentioning.

MEASURES OF PREVENTION.

A prevention of a disease is effected, if its causes are prevented to act, destroyed, removed, or neutralized. In most cases of colic two kinds of

causes, predisposing and exciting causes, can be distinguished, and the disease is produced, if both kinds of causes are present and acting in concert. Consequently, these cases of colic, as well as many other diseases, are prevented, if either one of these kinds of causes is warded off, or not allowed to act. As to colic, it will, on the whole, be easier to ward off, or to prevent, the exciting, than the predisposing causes. A prevention of the principal and most frequent exciting causes will be effected, if the horse is always regularly fed; if the food is sound, wholesome, and digestible; if feeding a heavy meal immediately before, and immediately after, severe exercise is avoided; if no food that has a tendency to ferment, or that is rich in alkalies is given; if the feeding of new grain and of new hay that has not yet passed through the so-called "sweating process" is avoided, or where that cannot be done, if such new hay and new grain are fed only in small quantities and then with a small pinch of salt added to each meal; if no icy food, or food covered with hoar-frost is allowed to be eaten; if no ice-cold water is given to drink, or when it cannot be avoided, only in small quantities, and never when the horse is perspiring or has an empty stomach; and finally, if meal or bran that may be used as food, is never given until it has been thoroughly moistened.

The principal predisposing cause, the aneurysm in the anterior mesenteric artery, can be warded off by preventing the wormbrood of *Sclerostomum equinum* from entering the digestive canal of the horse, but this, it seems, can be accomplished only, if the horse is never allowed to drink any water but what is positively free from the wormbrood. That this will be difficult, will not need any explanation.

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