

ent as the general character of the inflammatory changes would permit an approximate estimate. Some slight change can be noted in some of the fibrils distributed to papillary portion of the corium, but, unlike Schoonheid,¹⁴ I am unable to attach any special significance to such a feature. Dilated capillaries and lymph spaces, and occasional endarteritis was frequently observed, but not more than could be currently expected in chronic inflammation of equally severe and prolonged type.

The prolonged congested and edematous character of the lesions and their exceedingly friable nature readily accounted for the numerous thin-walled capillaries and spaces, which were congested with red corpuscles, in various stages of preservation in the superficial layers of the corium, and also probably accounted for some of the irregular spaces, cavities and artefacts (canalization of Unna), which were also observed. No thrombi were observed in any vessels of the corium, as observed by Fordyce and Holder,¹⁵ and nothing was noted which could in any way lead me to infer that the vessels of the corium were the seat of the first pathologic change.

Diffused hemorrhages in various stages of preservation were frequently observed in the upper layers of the corium, and many thin-walled capillaries gorged with one or more columns of well-preserved red corpuscles. The orifices of hair follicles and sudoriferous glands were almost invariably the seat of an extensive endothelial proliferation which filled their cavities to a considerable depth with dense keratinized plugs, or when accidentally or mechanically removed left them widely patulous and imparted a honey-comb appearance to the stratum corneum. The plugging of the sweat ducts was not altogether unlike that observed in porokeratosis. In some of the more severe cases of rather long duration, well-defined giant cells were occasionally observed among the plasma exudate,¹⁶ which in themselves do not differentiate the affection from lupus and syphilis, as frequently stated. The pathologic differentiation of the affection from syphilis and lupus is facile without this distinction. My personal efforts to establish a specific cause by bacterial investigation were attended by uniformly negative results.

SUMMARY.

1. Lupus erythematosus, in the absence of bacteriologic proof, appears to be a local infectious diseases.

2. The course of the disease varies with the special clinical types, and the severe forms do not yield readily to the present complex and inadequate therapeutic measures. The severe types, when favorably situated and sufficiently well circumscribed, should be extirpated; merely the border if the center is undergoing visible signs of retrogression and cicatricial atrophy, otherwise the entire lesion.

3. The conjunctival mucous membrane may become involved by direct extension of the process, and in severe cases blindness may become a complication. The primary pathologic focus appears to be in the epidermis and its adnexa, rather than in the vessels of the corium. The earliest changes are observed in the hair follicles and sebaceous glands when they are present, in the form of an active proliferation of the cellular structure.

4. These in turn call forth a focal exudation of plasma

cells from the immediate capillaries, which imparts to the affection a characteristic pathology, and is often accorded primary rather than secondary importance. The sweat glands share in the same process. The process is accompanied with free superficial keratinization and plugging of the orifices. In the absence of hair follicles and glandular elements, the stimulus is primarily directed against the epidermis, resulting in its down- and over-growth, and the resulting plasma exudation is more superficially placed and directed against the lower layer of the epidermis. The inflammatory change ultimately calls forth a connective tissue degeneration.

ABDOMINAL PAIN.

ESPECIALLY PAIN IN CONNECTION WITH ILEUS.*

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From my published works it may be gathered that I have not been able to find any abdominal organ, innervated only by the vagus or the sympathetic nerves, which is provided with the sense of pain. Sensations of pain within the abdominal cavity are, according to my experience, transmitted only by the phrenic nerve, the lower six intercostal, the lumbar and the sacral nerves.

My former pupil, M. Ramström,¹ professor of anatomy at Upsala, has given us the first exact description of the course of these nerves within the diaphragm and the peritoneal lining of the anterior abdominal wall. He has shown that some of the older descriptions of the distribution of these nerves are incorrect. For instance, he has not seen any branches of the phrenic nerve running down from the diaphragm to the anterior abdominal wall, nor has he been able to trace a single branch of the phrenic nerve through the suspensory ligament to the capsule of the liver. Similarly he has been unable to find any twigs from the intercostal nerves of the diaphragm which extend to the capsule of the liver.

These anatomic observations of Ramström agree with my own experience in regard to the sensitiveness of the liver. Even a strong faradic or galvanic current applied to the surface of the liver above the gall bladder does not excite pain. In some cases in which the position of the liver was low I have separated the attached surface of the gall bladder as far as the cystic duct without causing any pain, whereas the patient complained as soon as I tilted the liver or dragged on the common bile duct, thus putting the cerebrospinal nerves of the abdominal wall on the stretch. Not only the sense of pain, but also the other modalities of sensibility—pressure, cold and heat—are absent from the liver and gall bladder as well as from the stomach and intestines.

We have often been able to ascertain that viscera involved in disease are quite as insensitive to operative measures and to electric stimuli as are sound ones. Thus the old theory defended by Flourens has been destroyed.

After many investigations—some of which have been attended by the well-known physiologist, Hj. Öhrvall, and several by Professor Ramström as recorder—we had come to the conclusion that the parietal peritoneum of the anterior abdominal wall possesses only the sense of pain, not the senses of pressure, cold and heat. (In

14. Arch. f. Dermat. u. Syph., liv, 163.

15. Med. Record, 1900, p. 41.

16. Hyde and Montgomery (Text-Book 1904, p. 687) state that there are no giant cells in lupus erythematosus as in lupus. Schoonheid (Arch. f. Dermat. u. Syph., liv, p. 16) states that "Riesenzellen waren niemals vorhanden."

* Read in the Section on Surgery and Anatomy of the American Medical Association, at the Fifty-eighth Annual Session, held at Atlantic City, June, 1907.

1. Untersuchungen über die Nerven des Diaphragma, Aus den anatomischen Heften, Fr. Merkel und R. Bonnet, 1906, xxx, 92

most cases it is the peritoneum behind the recti muscles from a point 4 to 5 cm. above the umbilicus to a point midway between the umbilicus and the symphysis pubis which has been examined.) Should this view of ours prove correct, it speaks decidedly in favor of the specific character of the nerves of pain. In other words, it goes to prove that the entire parietal peritoneum is provided only with nerves of pain—a condition previously known to exist in the cornea alone.

It is my opinion that all painful sensations within the abdominal cavity are transmitted only by means of the parietal peritoneum and its subserous layer, both of which are richly supplied with cerebrospinal nerves around the whole of the abdominal cavity, possibly with the exception of a small area in front of the vertebral column lying below the crura of the diaphragm and between the two chains of sympathetic nerves. Here, as far as I am aware, no cerebrospinal nerves have as yet been demonstrated,² and on a few occasions I have observed that within this area the patient does not respond to hard pressure with a finger or with an instrument, nor, furthermore, does he experience any sensation when a small portion of the mesenteric attachment at this point is put on the stretch.

The opportunity is given during operations of observing that the manipulations which cause pain are those which occasion stretching of the parietal peritoneum as well as of the parietal attachments of the mesenteries. For example, pain is occasioned by the placing or removal of gauze compresses between the viscera and the parietal peritoneum, by the dragging forward of the cecum, of the vermiform appendix, or of any other organ whose normal attachment to the abdominal wall is put on the stretch; and the same principle applies to the stretching of any abdominal adhesions which may connect the viscera with the abdominal wall. On the other hand, should a compress lie between the viscera without coming in contact with the abdominal wall the patient experiences no sensation when it is removed. Similarly no sensation attends the stretching or breaking up of adhesions which have no connection with the abdominal parieties. As far as I can judge from my observations, the parietal peritoneum along the thoracic aperture and around the foramen of Winslow is especially sensitive to stretching, displacement, etc.

A slow and gradual stretching of all the layers of the abdominal wall by ascites or meteorism occasions distress rather than pain, although a high degree of meteorism may be attended by great discomfort. If in a severe case of paresis of the bowel one succeeds in emptying the intestine by means of a typhilitic, a jejunal or gastric fistula, the procedure is followed by such evident relief that the great distress of the previous condition is emphasized. That a maximal degree of rapidly-forming meteorism is an extremely painful condition and one which may rapidly endanger life I have witnessed in the case of a young student who had a coincident volvulus of the ileum and acute dilatation of the stomach. Four hours and a quarter after the appearance of the first symptoms he was pulseless from intense pain attended with a sensation of bursting.³

Infectious processes involving the abdominal viscera (ulcerations, acute inflammations, etc.) are attended by

lymphangitis and lymphadenitis of the mesenteries. The infection spreads along the lymph-vessels to the subserous tissue of the abdominal wall, and inasmuch as the lymph-vessels follow the course of the arteries the lymphangitis very soon reaches the sides of the aorta, along which it then may continue up to the thoracic cavity. A lymphangitis of the parietal subserous connective tissue greatly increases the sensitiveness (excitability) of the cerebrospinal nerves to any manipulation which occasions pain even under normal circumstances.

All that we know of lymphangitis and lymphadenitis attending affections of the mouth, of the pharynx, of the extremities, etc., applies equally well to corresponding processes within the abdominal cavity. We know, for example, that the severity of pain attending a lymphangitis and lymphadenitis of the above-named regions varies according to different infections, and the same thing is true for the abdominal cavity.

The irritability of the nerves of pain of the parietal peritoneum is much increased even by a slight peritoneal inflammation. In the case of a serous peritonitis (*peritoneale Reizung*) the boundaries of the hyperemic zone of the parietal serosa can be mapped out almost to the centimeter by gentle palpation of the abdominal wall. With further increase of the hyperemia and of the inflammation the sensitiveness is at first almost proportionally increased. The fact that so many infectious processes within the abdominal cavity begin with diffuse abdominal pain may be explained by (1) an increased sensitiveness of a large portion of the parietal peritoneum owing to the lymphangitis or peritonitis, (2) a considerable increase and irregularity of peristaltic action which, in addition to pain, often produces a feeling of sickness, vomiting and one or more actions of the bowels at the commencement of these illnesses. On account of the increased sensitiveness the movements of the stomach and intestines against the parietal peritoneum and the stretching of their respective mesenteries are felt as severe pains. In most cases, however, the general peritoneal irritation soon passes off. Only the part more especially infected remains in a condition of inflammation, and the abdominal pain will become localized at this spot. In those cases where the infection spreads over a large portion of the peritoneal cavity, thus giving rise to a more or less general peritonitis, the pain will diminish as soon as the bowel becomes parietic and the nerve endings of the parietal peritoneum have been more or less destroyed by the severe inflammation.

PAIN IN CONNECTION WITH PERFORATION.

In case of visceral perforation or of an abscess which ruptures into the free abdominal cavity the primary pain is caused by the contents of the organ, or of the abscess, coming into contact with the parietal peritoneum. The severity and character of the pain depends on the nature and quantity of the escaping fluid, the extent to which it immediately comes into contact with the parietal peritoneum, and, lastly, on the intensity of the contractions of the stomach and bowels brought on by the irritation of the peritoneum.

Many clinical observations are explicable if one bears in mind the fact that only the parietal peritoneum can transmit painful sensations. For example, the primary pain occasioned by a duodenal perforation may be referred to the iliac fossa. Again, the paroxysmal pains in connection with a gastric ulcer are elicited by the movements of the stomach, that is to say, by its dragging on a parietal serous membrane which is hyper-

2. Here we find only more or less transversal running nerves uniting both sympathetic chains, and no immediate cerebrospinal nerves.

3. Recovery after Gastrotomy. Reposition of the Ileum. Jejunostomy. Gastrotomy, and Later Gastro-Jejunostomy, *Deutsche Zeit. f. Chir.*, 1906, lxxxv, 151.

esthetic on account of a lymphangitis from an infected ulcer. If the stomach is put at rest by the aid of a jejunostomy the pains cease. Further, in the case of a patient with an inflammatory focus surrounded by small intestines and covered by a thick omentum, pressure on the abdomen will disclose no tenderness, whereas palpation per rectum may cause pain.

THE HYPOTHESES OF NOTHNAGEL AS REGARDS COLIC.

In human beings suffering from intestinal fistulas and to whom no anodyne has been given, sensation of pain can not be evoked by means of chemical, thermic, mechanical or electrical stimuli applied to a portion of the gut lying outside the abdominal cavity as long as the stimulus or contractions which it causes only affect the bowel. When, on the other hand, the contracting bowel drags on adhesions connecting it with the abdominal wall, it at once produces pain.

Both theories of Nothnagel are hereby disproved, for the colicky pain can not be due to pressure on the nerves of the bowel wall in consequence of a tonic spasm of its muscular coat, since the intestinal wall can be crushed with a strong pair of forceps without eliciting any sensation whatsoever. Further, the pain can not be due to anemia of the intestinal wall due to a spasm of its muscular coat, since it is possible by means of electric stimulus to produce so powerful a contraction of the bowel that it becomes of tumor hardness and assumes a yellowish-white color from anemia, without the patient experiencing any sensation even of being touched.

Wilms, in an article⁴ and later in his splendid work on ileus (1906), tried to show that all those pains which we are accustomed to term "intestinal colic" are entirely due to stretching of the mesenteric attachments. In my first publication (1901) I made the statement that every distension or contraction of a gut which is attended by a pull on its attachments to the abdominal wall is necessarily painful, as it involves a stretching of the cerebrospinal nerves of the parietal serous and subserous layers. With regard to the duodenum, the duodeno-jejunal flexure, the three flexures of the large intestine, and the most distal part of the ileum, it goes without saying that these portions of the bowel can not contract on their contents in front of an obstruction without giving rise to a powerful dragging of their mesenteries and at the same time a painful stretching of the nerves of sensibility of the parietal serosa. In similar fashion powerful intestinal contractions are bound to involve a painful stretching of the parietal serosa in case the bowel has become fixed to the abdominal wall by adhesions and there exists at the same time some obstruction at this point.

The pains in connection with ileus, due to kinking, volvulus, etc., were thus easy to understand. On the other hand, it was my opinion that the stretching of a high and free mesentery of the small intestine or of the transverse colon could not account for the pain attending a stricture of these parts of the bowel when the stricture has not become adherent to the abdominal wall or in some way fixed, because I could not think that a contraction of the bowel around its contents in front of the stricture could drag on the parietal serosa and subserosa by that high and free mesentery. Wilms also accounts for the pain in these cases as being only the result of the stretching of the mesentery proximal to the stricture. He considers that a loop of bowel, contracting on its contents at the proximal side of a con-

striction, endeavors to assume a straight form in the same way as does the gut in sausage-making. The mesentery, however, prevents the bowel from assuming the shape of a straight cylinder. The result is a stretching of the mesentery, and this is what causes the pain. In this connection an observation was made a year ago in a case in which I had to resect more than one meter of the ileum, together with the cecum and a large portion of the colon, this portion of the intestine having been excluded some months previously by means of an ileo-colostomy, on account of multiple fistulas and adhesions which could not be loosened.

Under local and a short ether anesthesia the excluded portion of the bowel was completely freed from all adhesions, only the normal mesenteric attachment being left; the patient being awake, a piece of the ileum about 40 cm. long was clamped and inflated with air. The bowel straightened, the mesentery got stiff and assumed a fan-shaped form. While in this position, standing out from the vertebral column, the patient complained of pain, but this passed off as soon as the bowel was emptied of air and the mesentery was allowed to resume its normal position. The experiment was repeated with a considerably shorter piece of bowel. The result was the same, for the mesentery again became tight and stood out from the vertebral column, occasioning pain. Lastly a piece of the intestine only 5 or 6 cm. long was shut off. Although it was inflated *ad maximum*, causing the serous membrane to burst and the bowel to sink between the two layers of the mesentery for fully 1 cm., the patient felt nothing. As soon, however, as the mesentery was stretched for a few moments with the fingers pain was felt. These observations are in full accordance with the above mentioned view of Wilms as regards the stretching of the mesentery, a view which I believe to be correct.

The pain in connection with a volvulus of the intestine or of an ovarian cyst naturally increases in proportion as the twisting comes on quickly and more parietal peritoneum is drawn into the pedicle.

PAIN CAUSED BY DISPLACEMENT OF THE SEROUS MEMBRANE OF THE ANTERIOR WALL.

On many occasions when performing a laparotomy I have passed into the abdominal cavity a finger covered with a thin, smooth India rubber glove dipped into saline solution and exerted a slight pressure on the anterior abdominal wall. Of this the patient has no perception, but as soon as the serous membrane is displaced against the muscles or aponeuroses of the abdominal wall the patient has a feeling sometimes of touch, more often of pain, according to lesser or greater sensitiveness of the individual, and according to the degree of pressure and displacement employed. When asking the patient, "Have you ever felt anything like this?" I have usually received answers such as these: "It feels like colic;" "It feels as if the bowel is being expanded by wind;" "It's like bad griping pains;" "It is worse than gripes."

Such sensations are occasioned by displacing a small, limited area of the serous membrane at nearly any point of the anterior abdominal wall. On account of these observations I believe that a displacement of the serous lining takes place and gives rise to pain as soon as a loop of intestine contracts on its contents, hardens, rises and presses against the parietal peritoneum. According to my opinion, these pains occur not only in connection with ileus, but also in connection with tem-

4. Münch. med. Wochschr., 1904.

porary irregularities of the peristaltic movement of the bowels in people not suffering from abdominal diseases. I have myself felt these gripping pains and I have been strongly inclined to localize the same at the anterior abdominal wall, most often to the left lower quadrant. As a rule I have attributed them to the contractions of the sigmoid flexure.

Here I may mention a case illustrating the displacement of the serous membrane of the anterior abdominal wall at the thoracic aperture. It was brought about by a subserous myoma of the fundus uteri of about the size of a mandarin, in a woman eight or nine months pregnant. She had suddenly been taken ill with severe pains, vomiting and inability to pass flatus. A diagnosis was made by her medical attendant of a twisted ovarian cyst. As soon as the myoma had been removed under local anesthesia all symptoms disappeared. The ovaries were normal. The pregnancy went on to its normal termination. I have record of another patient who had a small subserous myoma of the uterus and in whom, after the seventh month of pregnancy, abdominal pains were produced by any movement. They were felt as a very painful friction and disappeared as soon as the tumor was removed.

If the bowel wall on the proximal side of an obstruction is considerably thickened and the serous coat rough the *Darmsteifung* is naturally attended by a much more extensive displacement of the parietal peritoneum than in the case of a normal intestinal wall. Contrary to Wilms, I consider this factor also to be the cause of the colicky pain which attends intestinal obstruction. I have consequently come to the conclusion that "gripes" are due partly to a stretching of the parietal attachments of the bowel and partly to a displacement of the serous lining of the abdominal wall.

REFLEX RIGIDITY OF THE ABDOMINAL MUSCLES.

The normal response of the abdominal muscles to an acute sensation of pain which originates in the parietal peritoneum or subserous tissue lies in a reflex contraction (*défense musculaire*). In cases of violent and very extensive peritoneal irritation (hyperemia, slight edema) the abdomen assumes a board-like rigidity and the respiration becomes costal in type, for the abdominal muscles and the diaphragm are in a state of tonic contraction. In this way the range of movement of the abdominal organs is greatly diminished and consequently the abdominal pain is greatly lessened. Compare with this the endeavor of patients suffering from severe abdominal pains to get relief by lying on the "belly" or by fixing something tightly round the abdomen. The extension of the reflex muscular rigidity corresponds closely to the area of peritoneal irritation of the parietal serosa. We know little as yet about *défense musculaire* in connection with a mechanical ileus before the onset of peritonitis. It is necessary to observe with care every case of severe colicky pain attending an intestinal obstruction in order to see whether a tonic contraction of the abdominal muscles and diaphragm takes place with the object of diminishing the movements of the bowels and indirectly the severity of the pains.

In acute inflammatory diseases of the abdomen I have not observed the presence of cutaneous hyperalgesia so often as one might expect, especially in consideration of the "*triade douloureuse*" of Dieulafoy (1899) which he regarded as necessary for the diagnosis of an acute appendicitis—cutaneous hyperalgesia, reflex muscular rigidity, tenderness on pressure. Before applying Head's

theory of cutaneous hyperalgesia to a given case one ought to consider the question whether in that special case an infectious lymphangitis along the posterior abdominal wall and around the vertebral column might not cause a hyperesthesia of the sensitive nerve trunks and spinal ganglia of that region. It will, of course, be clear to every one that this is only a working hypothesis. In those few cases of acute appendicitis with cutaneous hyperalgesia which have come under my care since I began to pay more attention to this matter I have observed co-existing tenderness on deep pressure in the angle between the twelfth rib and the erector spinae or somewhat lower down at the border of this muscle.

When considering the pains connected with infectious diseases of the liver and gall bladder one has to remember that well-known embryologic facts, as well as my own researches and the investigations of Ramström, all lead to the assumption that the liver, the gall bladder and the extrahepatic bile passages do not possess nerves of pain. One has further to consider the distribution of the lymph vessels from these organs to the posterior abdominal wall and diaphragm as well as their anastomoses with the lymphatics of the duodenum and pancreas. One can then easily understand that infectious diseases of the liver and gall bladder are apt to be followed by spasms of the diaphragm and that the movements of the common bile duct, the duodenum and stomach may be attended by pain. When the gall bladder contracts spasmodically in order to expel its contents there results a stretching of the cystic and common bile ducts and consequently a displacement of the parietal peritoneum and the extremely sensitive retroperitoneal connective tissue around the common bile duct. If a tube has been fixed (water tight) into the gall bladder in a case of cystostomy for cholecystitis, 100 c.c. or more of saline solution may be made to pass from the gall bladder into the duodenum (the biliary passages being free) without the patient feeling anything so long as the solution is being slowly injected into the gall bladder. If the injection is made with a little greater force the patient almost immediately complains of colicky pain in the back.

With a shrunken gall bladder and a very wide common bile duct biliary colic due to the stretching or distension of the common bile duct is inconceivable.

It is quite necessary to consider carefully the account which the patient gives of his pain. Lately a patient of mine suffering from an acute hemorrhagic pancreatitis stated that the attack of pain began with a sensation as if the large blood vessel at the back had burst near the pit of the stomach. The autopsy showed, in addition to the pancreatic hemorrhages, a large retroperitoneal extravasation around the celiac artery and the aorta. One must never forget how difficult it is to localize pain and to how great an extent a correct interpretation of the site of painful sensation is a matter of practice.

SUMMARY.

In estimating abdominal pain, and especially in connection with illnesses giving the symptoms of "ileus," we must bear in mind, briefly, that:

1. Pains do not originate within the abdominal organs which are supplied only by sympathetic fibers and the vagus nerves.

2. All pains originate in the abdominal wall, more especially in the parietal serous membrane and subserous connective tissue structures which are innervated by the cerebrospinal nerves.

3. Stretching of the parietal (mesenteric) attachments of the stomach and intestines, as well as of string or band-like adhesions to the abdominal parietes, invariably elicits pain.

4. The same thing holds true for the displacement of the parietal serosa from its normal relation to the muscles or aponeuroses of the abdominal wall.

5. Most of the diseases connected with ileus are, at their commencement, attended by increased and, as a rule, irregular peristalsis.

6. Chemically different substances, such as the contents of the stomach, gall bladder, intestine or abscesses, give rise to severe pains when they come into contact with a healthy or hyperemic parietal peritoneum (pain due to perforation).

7. Even that form of acute peritonitis which goes under the name of peritoneal irritation (*peritoneale Reizung*) greatly increases the sensitiveness of the parietal serous membrane.

8. The sensitiveness of the parietal peritoneum at first increases *pari passu* with the inflammation, but later decreases again when the inflammation has reached a certain high degree, and in many cases may ultimately cease altogether. (Compare herewith erysipelas of the skin, more especially the gangrenous kind.)

I believe, finally, that we are on the way to completely understand the pains of ileus, though a great amount of work still remains to be done. I consider it to be a very happy thought to bring together anatomists, physiologists, physicians and surgeons for the discussion of this question, and I feel not only greatly honored, but also deeply grateful for the invitation to contribute this introductory paper.

PHYSIOLOGIC OBSERVATIONS ON EXPERIMENTALLY PRODUCED ILEUS.*

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In his discussion of ileus, Nothnagel divides the condition into two classes: 1, Mechanical ileus, in which there is occlusion of the intestine; 2, dynamic ileus, in which there is a syndrome of occlusion of the bowel, without, however, closure of the lumen. The second class of cases occurs whenever the intestine becomes paralyzed, parietic or spastically stenosed.¹ Since an earlier paper in this discussion is concerned with intestinal obstruction, we shall not repeat here our observations, already published,² on certain physiologic changes following mechanical closure of the bowel. In that publication we raised the question as to the location of dynamic ileus. Is it of central or peripheral origin? In this research an attempt is made to answer that question.

In dynamic ileus we are dealing directly with a failure of normal peristalsis. True peristalsis, according to Bayliss and Starling, is a coördinated movement dependent on this local reflex. The reflex has its seat, as Magnus has shown,³ in Auerbach's plexus. Although the alimentary canal can perform its motor functions for some weeks almost normally, when wholly disconnected

from the central nervous system,⁴ yet, according to Magnus, all spontaneous and reflex movements cease when Auerbach's plexus is removed. Consequently anything which would injure this myenteric plexus would necessarily result in a cessation of peristalsis.

The extrinsic innervation of the stomach and intestines has recently been much clarified by the investigations of Bayliss and Starling and by men working with them and with Langley.⁵ It seems fairly certain now that the splanchnic nerves, acting through the sympathetic, are purely inhibitory nerves for the intestine. Concerning the effect of these nerves on the stomach, there is still controversy, but one of us has observed inhibition of gastric peristalsis during emotional excitement when the only connection between the stomach and the central nervous system was the splanchnic supply.⁶ Strong impulses through the splanchnic nerves, therefore, may be regarded as another cause of gastric and intestinal inactivity.

From physiologic studies, therefore, we should expect failure of peristalsis to follow inhibitory impulses through the splanchnic nerves, or to result from injuries or disturbances to the myenteric plexus, or from trauma to the muscles themselves. In other words, the cause of dynamic ileus may be general, by way of the central nervous system, or it may be local, in the inefficacy of Auerbach's plexus or the muscles which that plexus controls.

DYNAMIC ILEUS THROUGH THE CENTRAL NERVOUS SYSTEM.

The Effects of General Asthenia.—When the nervous connections between the alimentary canal and the central system are intact, nothing is more remarkable than the responsiveness of the canal to conditions of general asthenia. In the asthenia of animals afflicted with "distemper," for example, food will lie in the stomach or intestine all day without the slightest sign of a peristaltic wave passing over it. There is total stoppage of the motor activity of the digestive organs.

The condition is quite different when the canal is disconnected from the spinal cord and brain. In such a state, with the stomach and small intestine entirely deprived of their extrinsic innervation, these organs have been observed exhibiting their normal activities, although the animal was to the last degree exhausted and toneless. In one instance an animal, very limp and anæsthetic, was fed by stomach tube 25 c.c. finely divided lean beef mixed with 5 gm. subnitrate of bismuth, reduced to a mush by the addition of water. Inspection by means of the Roentgen rays at intervals of an hour for five hours revealed normal peristalsis of the stomach and normal motor activity of the small intestine at each observation. At the end of five hours, although the animal was moribund, gastric peristalsis was still prominent. At the end of the sixth hour the animal was dead. Autopsy revealed a deep septic process in the muscles of the neck.

If the nervous connections with the cord and brain had been intact in this case, the persistence of the activity of the alimentary canal, according to our experience, would not have been seen. The general bodily condition, therefore, may affect the canal unfavorably through the nervous system. The pathway of these unfavorable influences is probably the splanchnic, for normal activi-

* Read in the Section on Surgery and Anatomy of the American Medical Association, at the Fifty-eighth Annual Session, held at Atlantic City, June, 1907.

* From the Laboratory of Physiology in Harvard Medical School.

1. Nothnagel's *Diseases of the Intestine and Peritoneum*, English translation, Philadelphia, 1904, p. 377.

2. Cannon and Murphy: *Ann. of Surgery*, 1906, **xliii**, 512.

3. Magnus: *Archiv. f. die gesammte Physiol.*, 1904, **cll**, 362.

4. Cannon (W. B.): *American Jour. of Physiol.*, 1906, **xvii**, 429.

5. *Jour. of Physiol.*, 1899, **xxiv**, 99; 1901, **xxvi**, 125; May, 1904, **xxx**, 260; Elliott and Barclay-Smith, 1904, **xxxi**, 272.

6. Cannon (W. B.): *American Jour. of Physiol.*, 1905, **xiii**, **xxii**.