

## POSTOPERATIVE ILEUS.

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FOR postoperative ileus we should substitute postoperative paralytic obstruction, partial or complete, temporary or permanent; for the word "ileus," in its etymology, has no pathological significance and should be eliminated from surgical nomenclature.

Partial or complete intestinal obstruction may be either primarily paralytic or mechanical, followed by loss of motility and peristalsis if operation is delayed.

The primary paralytic form of obstruction may be caused (1) by violent inhibitory impulses transmitted to the muscle walls of the intestines from the central nervous system by the splanchnic nerves through the sympathetics; (2) by impairment or destruction of the energy in the neurenteric plexus, and the myogenic force in the walls of the intestines on which motility and peristalsis depend, resulting from the poisonous action of bacterial enzymes from the intestines or the peritoneal cavity on the nerves or muscles in the bowel walls, or from traumatism; or (3) by the combined action of the central inhibitory splanchnic impulses, with impairment or destruction of the energy in the neurenteric plexus, and myogenic force in the walls of the intestines, caused by arterial poisons or traumatism.

Mechanical intestinal obstruction may be caused by adhesions, constricting bands, volvulus, intussusception, hernia, etc., and if not relieved promptly by operation, the

bowel above the obstruction becomes dilated and then paralytic, and the ileus cannot usually be differentiated from the primary paralytic form.

In order better to understand paralytic bowel obstruction, and also mechanical bowel obstruction, simple, or followed by paralysis, it is necessary to study the research work done on the gastro-intestinal tract bearing on its anatomy, physiology, motility, and bacteriology. While these observations have been made on animals, usually in good health, it has been shown that in treating such obstructions in man we may, with few exceptions, be governed by the results obtained. It may be urged that these experiments were on healthy animals, while in paralytic or mechanical obstruction we are treating patients with a pathological condition of the gastro-intestinal tract, and, while this is true, it is also equally true that we cannot correctly understand the pathology until we learn the normal anatomy and physiology. In studying the anatomy and physiology of the gastro-intestinal tract, while we recognize form, we must not forget that to understand better the physiology and motility, and often the pathology, we must make the division by function and not by form from the inlet to the outlet. This is in keeping with the embryology of the digestive tract, which arises from the foregut, the midgut, and the hindgut. From the foregut is developed all the tract to a point in the duodenum below the entrance of the bile and pancreatic openings; in this part of the tract digestion is nearly completed, but there is but little absorption. From the midgut is developed the lower part of the duodenum, the jejunum, the ileum, and cecum, ascending colon, and the transverse colon to the splenic flexure; in this part of the intestine nearly all absorption of digested products results. From the hindgut is developed the splenic flexure, the descending colon, the sigmoid flexure and the rectum, the part of the intestine used for storage and the expulsion of feces and gas.

The motility of the gastro-intestinal tract is involuntary, except at the inlet and the outlet, and this is essential, because there must be a voluntary control of what enters the stomach and of what passes out of the rectum. The upper third of the esophagus is entirely controlled by the central nervous system, this control disappearing in the middle third, and is entirely abolished in the last third. The sigmoid flexure is nearly entirely controlled by the central nervous system, and the rectum is entirely so. In all other parts of the tract, the motility is involuntary, and a nervous system, known as Auerbach's plexus, lies in the muscle walls and imparts energy to the circular and longitudinal muscles, giving them force to propel the food and liquids through the tract after fixed laws.

The innervation to the stomach and intestines from the central nervous system is from the vagi, the fibers of which enter and become interlaced in the medulla oblongata, and the splanchnics, right and left major and minor, from the thoracic ganglia, which transmit impulses through the sympathetics. The impulses through the vagi to both stomach and intestines are mainly motor, but under certain conditions inhibitory impulses may be transmitted, but probably these are merely temporary, the inhibition being quickly followed by increased motility. The impulses transmitted through the splanchnics both to the stomach and intestines are inhibitory, and this inhibition may be so increased by injury to the testicles, kidneys, posterior structures of the peritoneal cavity, or in a state of profound asthenia, etc., as, temporarily at least, to control the neuroenteric energy and myogenic force in the intestinal walls. When all inhibitory impulses from the central nervous system have been abolished by the division of these nerves, gastric and intestinal peristalsis continues practically normal, and the food products may enter the colon previous to such division, and sometimes sooner. It will also be noted

that in cases in which the splanchnics are cut the relative characteristic rapid carbohydrate discharge and the slow proteid discharge is not materially changed. This may continue for weeks with but little change in the nutrition of the animal.

If the motor impulses of the vagi be destroyed by vagus section, leaving the splanchnics intact so as to allow the continuous inhibitory impulses, the discharge of both carbohydrate and proteid food is distinctly retarded, but the same relative rapidity of discharge is maintained. This slowness of discharge is, however, partially recovered from after several days.

When all extrinsic impulses are removed by both vagus and splanchnic section, the gastric and intestinal motility is nearly normal, indicating that the relatively slow discharge of food from the stomach and intestines after vagus section is caused not alone by the absence of vagus motor impulses, but also by the depressing or inhibitory influence of the splanchnics.

The characteristic rapid discharge of carbohydrate food, and the slow discharge of proteid food through the pylorus into the duodenum, after destroying central motor or inhibitory impulses, shows that the discharge from the stomach is mainly under local control, the peristaltic wave in the body of the stomach and the antrum being induced by the mechanical irritation of the food, while the opening and closure of the pyloric constriction is under local reflex control, induced by the presence of acid chyme. While in much of the gastric secretion, and probably in all the secretion of the digestive enzymes in the intestines, the glands are activated by a hormone or secretin, we have not been able to show that chemical mechanisms have caused motility in any part of the gastro-intestinal tract, this being entirely local reflex in the nerve plexus in the intestinal walls, caused by distention. Why these nervous mechanisms controlling motility should

be so adaptable, allowing the food to remain in the stomach, the duodenum, the small intestine and colon, each a sufficient time to complete normal digestion and absorption, we have not been able to learn. We have learned, however, that this orderly march may be easily disturbed by central nervous influences, caused by excitement or shock, or by injury to certain organs or retroperitoneal structures in the abdominal cavity, or by traumatism to abdominal viscera, especially the stomach and intestines.

That we may better appreciate the meaning and the significance of paralytic obstruction, we should first correctly understand the motility in the intestines as seen in normal peristalsis after food is taken into the stomach, which passes on, observing fixed laws—"the laws of the intestines." The food remains in the stomach or any part of the intestines until it is acted on properly. The proximal bowel then develops onward peristaltic waves of contraction, and simultaneously the immediate distal bowel relaxes so as to allow the food to move toward the least resistance. The onward contraction peristaltic waves and the distal relaxation continue in a regular order until the food is passed into the cecum and colon, but these coördinating movements are easily disturbed by irritation or injury to the intestines, and in some cases of intestinal wounds by projectiles the bowel becomes temporarily paralyzed, thereby greatly lessening the amount of fecal matter that would otherwise empty into the peritoneal cavity and increase the dangers of infection. This conservatism is so protective as to allow a patient to recover from a timely operation who would otherwise die. But if the bowel is simply irritated at any point, it immediately contracts in the part oral to this point, and relaxes in the part aboral, which relaxation may extend a considerable distance. In all forms of so-called ileus, temporary or permanent, there must be a partial or complete destruction of peristalsis, which may be restored promptly,

slowly, or continue until death. If the obstruction is complete, the peristalsis aboral to the obstructed part may be but little if at all disturbed, and we often see the distal bowel collapsed and apparently in a state of health, when the proximal bowel is greatly distended with feces and gas, its walls filled with infarcts, or in a state of death. We see the same condition in mesenteric thrombosis, in which there is neither mechanical obstruction, peritonitis, nor inflammation of the bowel walls. "Dynamic" signifies "pertaining to motion as the result of force;" hence the word should be excluded in any form of intestinal obstruction, unless there be cases caused by a temporary or continued spasmodic contraction in the ileocolic sphincter, or in some part of the bowel. Starling may be correct in his observation that, while splanchnic stimulation may cause complete relaxation of both the circular and longitudinal muscles of the small intestine, thereby destroying peristalsis, it produces a strong contraction of the muscle fibers of the ileocolic sphincter. While vagus stimulation may cause an initial inhibition of gastro-intestinal peristalsis, quickly followed by an increased motor force, it does not cause contraction at the ileocolic junction. Antiperistalsis is normal in the cecum and the ascending colon, and, while it is contended that there is no antiperistalsis in the small bowel, we have positive proof that in both mechanical and paralytic obstruction the intestinal contents, if not spontaneously expelled through the esophagus, or taken away by repeated lavage, may be forced into the stomach, greatly dilating it, the bowel contents in these cases moving in the direction of least resistance. This is an important fact to remember in abdominal surgery, for lives may be saved by the repeated use of the stomach pump, just as we may cure a patient with acute dilatation of the stomach, with apparent spasmodic contraction of the pyloric ring, and a loss of motility in the gastric muscle fibers.

While there may be shock in most cases of severe or fatal

postoperative paralytic obstruction, this is the result of, and is associated with, the absorption of bacterial toxic products from the bowel or the peritoneal cavity into the intestinal walls and tissues of the body. We have proof of this in the fact that when we remove the mechanical obstruction and allow the virulent poisonous contents above in the distended and paralytic bowel to pass onward, the patient may collapse from the rapid absorption in the comparatively healthy bowel below, the epithelial resistance being quickly impaired. It is further shown by the experiments of Ramzi, Kula, Clairmont, and Albu, that the subcutaneous or intraperitoneal injection of the filtrate of normal intestinal contents gave nearly negative results, but that the injection of the filtrate from an obstructed and distended bowel caused serious or fatal symptoms, in some cases collapse being marked. The absorption of intestinal gas does not seem to have much effect on man in paralytic obstruction, and, as the bowel distends, the absorption of gas and liquid from the bowel is diminished, and finally ceases. In such cases, however, the absorption of bacteria or of toxic bacterial products from the bowel contents into the bowel walls and other tissues is increased, because of the injury to or destruction of the epithelial layer, through which it is probable bacteria cannot pass while it is intact and normal. This absorption from intestinal contents, however, ceases, after death of the bowel. Postoperative peritonitis, which causes intestinal paralytic distention, or probably sometimes narrow constriction in some part of the bowel, is usually caused by the invasion of pathogenic bacteria or their toxic products from the gastro-intestinal tract, the pelvic organs, or by infection through abdominal wounds, or by unclean abdominal operations; a frequent contributing cause is extensive intestinal exposure and rough manipulation of the stomach, intestines, peritoneum, and mesentery. The rough manipulations of the intestines injure the endothelial

layer, the nerve plexus, and the myenteric structures; and tension on the mesentery may result in increased inhibitory splanchnic impulses, thereby causing distention of the bowel by impairing or destroying its motility and peristalsis.

What I have said above and also what follows is intended to apply to both acute and subacute postoperative mechanical or paralytic intestinal obstruction, remembering that the complete mechanical form will always become paralytic if the obstruction is not promptly removed by surgical intervention; hence it will be seen that mechanical obstruction is always surgical, but that with too much delay surgery is of no avail, for the bowel will then have become greatly distended with liquid and gas, which gradually interferes with the nutrition of its walls, destroying the epithelial barrier against bacterial invasion. Unfortunately we are often unable to distinguish between the early or even the late primary mechanical and the purely paralytic forms. While some surgeons have claimed that the differentiation can usually be made, this is contrary to the experience of nearly all abdominal surgeons of broad experience; for this reason it may be impossible to decide if an operation is necessary until it is too late to be of benefit. In such cases as can be diagnosticated, however, the abdomen must be promptly opened, the obstructing cause, whatever it may be, removed when it is possible to do so, the surgeon operating rapidly and committing as little traumatism as possible, for these cases will not resist a long operation or rough handling of the intestines and other intra-abdominal structures. Such patients will often recover if operated on before there is much peritonitis or peritoneal infection, and before the bowel is so distended as to interfere with its circulation and nutrition until its vitality is destroyed by the bacteria and bacterial toxic products absorbed from the intestinal contents. In some cases in which the obstruction can be removed, and in all cases in which it cannot for the time being be removed,

we shall get better results by performing enterotomy or even enterostomy, so as to give immediate temporary or continued drainage of the bowel contents, thereby removing tension on the walls and lessening the absorption of toxic products, thus giving the bowel a better chance to regain its nutrition and motility. In such cases a subsequent operation may be performed to remove the obstruction and to restore the bowel attached to the abdominal wall to its normal condition.

In operations for postoperative intestinal obstruction, it may be better not to enter the abdomen through the original incision, but to select a point that will probably enable the operator to find and remove the obstruction with the least possible exposure and handling of the intestines; by this means he may avoid opening a wound, often infected, and protect the patient from further peritoneal infection from this source. In the absence of any evidence of the seat of the obstruction, it is better to enter the abdomen in the median line below the umbilicus, for through this opening the focus of trouble may be found more easily than through any other incision; but if the original opening was made in or near the median line, then it may be better to open the cavity through an incision in the outer border or beyond the right rectus in the region of the appendix. If, however, there is evidence that the trouble is in the sigmoid flexure from volvulus or intussusception, it would be better to open on the left side. By practically studying the anatomy of the jejunum, the ileum, the mesentery, and the relative positions in the abdominal cavity of the first, second, and third parts of the small intestine, so beautifully demonstrated by Monks, we may better understand the part of the bowel presenting, its direction, and how to handle it quickly with the least possible traumatism. While I have operated successfully in many cases for intestinal obstruction, with the abdomen distended, a recital of these cases would be of no practical

value and of no interest to the experienced abdominal surgeon. Unfortunately the tendency with many of us is to report a long series of successful operations, often omitting the underlying basic principles on which we are enabled to philosophize correctly. The best surgeon is he who combines a knowledge of embryology, anatomy, physiology, bacteriology, pathology, diagnosis, and surgical technique, so as to enable him to reason logically as to what is best in the interest of the patient in any individual case; and, finally, he should be a surgical mechanic. There is no department of surgery to which this more fittingly applies than to the treatment of bowel obstruction from any cause. In true paralytic post-operative obstruction with no suppurative peritonitis, surgery is usually contra-indicated; hence the treatment should be rather preventive than curative. In some cases apparently severe, we may have a few days of discomfort, even with pain, nausea, vomiting, obstruction, and distention, followed by an uninterrupted recovery without treatment; but a severe toxemia or general peritonitis may develop, which may not improve either under medicinal or surgical treatment. Purgation is contra-indicated, and no food should be given in the stomach. If liquid and gas are regurgitated into the stomach and not easily expelled, they should be promptly removed by a stomach pump, and lavage frequently repeated. It must be remembered that repeated vomiting of small quantities indicates a dilated stomach, a condition similar to what is found in an overdistended bladder. At the same time, water through the rectum must be supplied to the tissues and thirst controlled by slow introduction of saline solution after the fashion recommended and described by Murphy. Finally, if gas does not pass from the colon, enemas may be given, care being taken never to distend the bowel too much. In some cases it is possible that the addition of small quantities of alum or carminatives may stimulate peristalsis in the large bowel and cause the expul-

sion of liquid and gas, leaving the cecum and colon relatively empty, thus encouraging the passage of liquid and gas from the small intestine into the large bowel.

In operations for paralytic or mechanical obstruction with peritonitis, drainage with a rubber tube introduced to the bottom of the pelvis is indicated, and may aid in the elimination of poisonous products. In such cases it may be better to put the patient in the semisitting or nearly sitting posture, so as to encourage gravitation of peritoneal liquids into the pelvic cavity, whereby the absorption of bacterial toxic products is reduced to a minimum. Proctoclysis should also be used, putting in the rectum from ten to fifteen pints of saline in each twenty-four hours. In such cases no food should be given by the stomach for several days or a week, and the patient should be given no solid food for ten days or two weeks. Milk in any form should not be considered a liquid food. After several days we may give nutritious enemas. Finally, when gas is passing freely and the bowels cannot be moved by enemas, castor oil may be given, but it is usually best not to give saline purgatives. The general belief that we should give remedies internally, subcutaneously, or intravenously, to establish intestinal peristalsis in paralytic obstruction, has no scientific basis. Such treatment is positively contra-indicated in cases in which there is peritonitis, inflammation of the intestinal walls, or injury to the nerve plexus or muscles in the bowel walls, and may cause death of a patient who would otherwise have recovered. In peritonitis, the intestines should be at rest to prevent the diffusion of infection, and in inflammation of or injury to any part of the bowel wall, rest is essential to encourage repair and a return to the normal anatomical and physiological condition; hence such remedies could be indicated only in cases in which the paralytic obstruction results from central inhibitory influences through the splanchnics, probably associated with control of the

vagi motor impulses, and in which there is but little injury to the nerve plexuses and muscles of the bowel. But we may expect such patients to recover without medicinal treatment if we observe the suggestions above mentioned as to stomach lavage and the use of saline solutions in the colon, as conditions may indicate.

While the experimental work of Meltzer and Auer and other laboratory research investigators has done much to show what medicines influence the motility and the inhibition of the gastro-intestinal tract, the results are far from complete, and from this work we have probably gained but little to aid us in the medicinal treatment of paralytic intestinal obstruction. Further research will probably give a clearer insight into the subject, and when we have developed a more correct knowledge of the etiology, the diagnosis, and the pathology of paralytic intestinal obstruction, we may then obtain better results from medicinal treatment. We know that ergot causes contraction of the involuntary muscle fibers, and it seems to increase peristalsis, but it is so erratic in its action that it cannot be depended on, and may sometimes, as shown by Meltzer and Auer, cause tetanic contractions similar to what may occur in the muscles of the uterus when given during labor or after the birth of the child. Atropine abolishes vagi motor impulses and impairs the spontaneous movements of the stomach and intestines, and should not be given.

If a paralytic condition of the bowel is due solely to inhibitory impulses, then hypodermic administration of morphine may be indicated, but we have no means to decide this question. Eserine (the physostigmine), hypodermically administered, will increase peristalsis in the healthy animal, but its effect is not lasting, and, while some surgeons have reported successful results from its use in paralytic obstruction, it has no claim for general acceptance; it may be given in doses of  $\frac{1}{40}$  to  $\frac{1}{80}$  grain, repeated when necessary to get its therapeutic effect, but never used to excess.

Resection of the devitalized intestine in paralytic obstruction, with the walls filled with bacteria and bacterial toxic products, is generally fatal, and is especially likely to be so if the involvement is extensive. If undertaken, the operation must be performed quickly with lateral anastomosis. The bowel should be crushed well above and below the dead part in healthy tissue and ligated with catgut in the crushed groove, the ends inverted and closed with a purse-string linen suture. The anastomotic opening should be three inches in length, and sutured as in gastro-enterostomy, the inner suture of catgut introduced through all the walls, linen being used for the outer seroserous suture. While it is theoretically preferable that all intestinal anastomosis should be made isoperistaltic, experience has taught that if this method is not observed, peristalsis will be established, but will be delayed in the anastomotic region. In obstruction from hernia or mesenteric thrombosis, where the devitalization of the bowel is not too extensive or shock not too profound, resection with lateral anastomosis is indicated. This does not mean that in all intestinal resections the union must be made by lateral anastomosis, for in many cases in which the trouble is not acute and the resected ends are in a healthy condition, it may be better to make an end-to-end union, using the clamps for suturing, as we do in lateral anastomosis or in gastro-enterostomy. Fortunately, postoperative paralytic or mechanical intestinal obstruction is now comparatively rare in the practice of a careful abdominal surgeon who rigidly observes asepsis in himself, his patients, assistants, nurses, and everything that could convey infection, and who properly prepares his patients for operation. In cases in which immediate operation is not imperative, the bowels should be thoroughly moved by a large dose of castor oil, and nothing but liquid food given for two days before the operation, and no food the day of the operation. The intestines should be exposed and handled as little as possible,

and the mesentery should not be put on the stretch; otherwise violent inhibitory impulses may be caused through the splanchnics, or peristaltic inhibition, because of traumatism to the nerve plexus or muscles in the bowel walls; such exposure and handling may also destroy much of the resistance in the endothelium of the peritoneum to the invasion of bacteria or bacterial toxic products, which causes peritonitis, local or general toxemia, or septicemia. Drainage should be avoided, if possible, and when necessary a perforated or split rubber tube should be used, for bad post-operative results have often been caused by the use of the glass tube. Gauze ceases to drain after a few hours, and then is positively harmful; it should never be used in the peritoneal cavity except to control dangerous oozing of blood or to isolate an infected area. I have often seen in my own experience bad results from the use of gauze, and even when it is used to isolate an infected area the rubber tube may be also used.

I have recently had two cases of perforation of the stomach, in the first of which drainage by the tube alone was employed; in the second, drainage by the tube and gauze. The first patient made an uninterrupted recovery with practically a normal temperature and pulse, though the operation was performed nearly twenty-four hours after the perforation occurred. The second operation was performed seven hours after perforation, and, while the patient recovered and is now in vigorous health, there was decided suppuration, elevation of temperature, and acceleration of pulse, which I believe, would have been avoided had I not used gauze with the tube.

In conclusion, I beg to suggest that surgeons who are contributing something of value for the betterment of abdominal surgery should know the results of the research laboratory investigators of the gastro-intestinal tract and the peritoneal cavity. They should be familiar with the results

of the work of Pawlow and Starling and Bayless on the physiology, Metchnikoff and Herter on the bacteriology, Cannon, Murphy, Meltzer, and Auer on the motility, Buxton on peritoneal absorption, Lennander on the sensibility of the intraperitoneal structures, and the laboratory research work of many other men who have done much in these related fields of science.