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INTESTINAL OBSTRUCTION.

SENIOR THESIS.

UNIVERSITY OF NEBRASKA COLLEGE OF MEDICINE.

PAULINE R. MCCONNELL. APRIL 21, 1933.

Intestinal Obstruction

The purpose of this paper is to bring together in condensed form the literature up to date on paralytic ileus and to set forth the views of some recent investigations along this line. The term ileus is used here to mean both paralytic and obstructive, as the difference between the two postoperatively is not always possible previous to the institution of treatment, and the emergency of the situation does not call for methods of accurate differential diagnosis with their accompanying delay. This article includes a discussion of the etiology, review of the anatomy and physiology of the autonomic nervous system and its relation to the condition, and a summary of the most efficient and recent methods of treatment.

Ileus may be defined as an interruption to the flow of intestinal contents through the gut together with perverted motor responses in the intestinal musculature and associated altered metabollic reactions.

From the etiologic standpoint ileus is of three types; 1. Delayed postoperative ileus-- having to do with adhesions. 2. The organic type-- having to do with such conditions as annular carcinoma. This type is not discussed here. 3. Immediate postoperative ileus-- in which infection and

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trauma are exciting causes.

According to Estrem (10), there are few emergencies that a surgeon has to meet that are more discouraging than cases of postoperative ileus following clean, surgical cases. In the first place, the keen disappointment that he suffers at the failure of the first operation to relieve the patient and then the necessity for subjecting the patient in a highly critical condition to a second ordeal far more hazardous than the first, together with the dismay and dread of the patient who has just passed through a difficult seige, produce a situation which requires real courage and prompt decisive action on the part of the surgeon.

In considering the etiological factors which produce this condition it is well to make a study of the anatomy and physiology of the autonomic nervous system.

The walls of the intestine are supplied by the sympathetic and parasympathetic systems terminating in the nerve plexuses of Auerbach and Meissner. In addition, the sympathetic nervous system with its plexuses act as the inhibitory motor nerve of the stomach and intestine, while the vagus nervr supplies exciting fibers to the digestive apparatus. It also contains some sensory conductors from spinal fibers. With the sympathetics we link the splanchnics, and with the parasympathetics the vagus nerve. Therefore, we have antagonistic dual innervation of the intestine by the vagus and splanchnic nerve fibers. Stimulation of the vagus nerve produces motor effect, whereas stimulation of the splanchnics produces inhibitory effect.From this we can conceive how paralytic ileus could be produced by trauma through reflex inhibition of bowel movements.

Starling(45) says, "stimulation of the splanchnic nerves causes complete relaxation of the intestine, while stimulation of the vagus increases contraction following a brief period of relaxation". He says that the relaxed condition of the intestines in many abdominal conditions is probably due to reflex stimulation of the splanchnic nerves which nullifies the motor action of the vagus.

The connection between the pre- and postganglionic fibers of the splanchnic nerves occurs in the celiac or semilunar ganglia. The celiac or semilunar ganglia, just mentioned, lie in the epigastric region of the abdomen behind the bursa omentalis and the pancreas, in the region of the origin of the celiac and superior mesenteric arteries. It is joined by the great and lesser splanchnic nerves of both sides, by celiac branches of the right vagus, and by filaments from the upper lumbar ganglia of the sympathetic trunks.

Intestinal obstruction or ileus may occur as a primary disease₂which sends the patient to the surgeon, or as a complication in the postoperative convalescence of patients who are subjected to operations for other reasons, especially those who have undergone previous laparotomies. Postoperative ileus is due in almost all cases either directly or indirectly to peritonitis. About one half of all cases of ileus are postoperative. Although occuring much less frequently than formerly, postoperative ileus (dynamic or mechanical) and (adynamic or non-mechanical) is still the cause of much of the present day mortality following abdominal surgery.

Cases of postoperative ileus occuring within a few days of an operative proceedure are usually directly dependent upon acute peritonitis, either localized or generalized, and obstruction caused by agglutination, plastic of one or more coils of intestine to other coils of intestine or other intra-abdominal viscera. Cases which occur later than this but still within the period of convalescence are usually due to residual abscess formation. Cases which occur after the period of convalescence, occasionally a number of years afterward, are due to strangulation of the intestines by bands of organized adhesions.

Moynihan (34) says that postoperative obstructions, coming shortly after operation, are almost invariably due to infection especially from pelvic peritonitis originating in appendicitis or in a septic condition of the pelvic genital organs. He emphasizes that limited peritonitis frequently causes complete paralytic obstruction of the segments of the intestine involved, which is so severe that unless dealt with promptly it will soon become fatal. He says there is definite proof that, where peritonitis is still localized in a single coil of the intestine with infection of the superficial blood vessels and loss of the normal luster, and the normal suppleness changes to a stiffening of the bowel, the paralytic type of ileus may result and produce just as fatal an obstruction as the true organic type.

Postoperative ileus, according to Grover (12) is very often found following clean cases of appendicitis. It rarely occurs in obstetrics following difficult delivery. It may follow injuries. There are a number of cases reported following a blow to the abdomen. It appears that any condition that disturbs the balance of the autonomic nervous system may be a causative factor.

The type produced by trauma cannot be explained except on the basis of nerve disturbance, according to Grover (12). The rough handling, actual injury, extensive manipulations, prolonged exposure to air, and irritations such as extremely hot sponges etc. are all causative factors. Many surgeons have observed that many postoperative obstructions are found near the ileo-cecal valve and in the pelvis, perhaps due to the fact that a relatively large proportion of abdominal operations are in the lower abdomen. High intestinal obstruction may occur after any abdominal operation but is probably more often found after operations on the stomach. These latter facts furnish evidence that trauma and exposure play an important role in the production of the condition.

Wilkie, Hartwell, and Hoguet (15), Murphy and Brooks (36) and others have presented evidence to show that the essential factor in the production of ileus is not blockage of the lumen of the intestine but interference with the blood supply of the intestinal wall. Only when such vascular disturbance occurs do the various phenomena characteristic of ileus develop.

An attempt was made recently by Owings, McIntosh, Stone, and Weinberg (41) to explain the interference with the blood supply on the basis of increase in intra-intestinal pressure. Though these particular investigators found the pressure in the various parts of the intestinal tract to be similar regardless of the site of the obstruction and therefore were unable to explain certain characteristic differences between high and low obstruction, Morton (33) states that he has been able to demonstrate that the pressure in the duodenum is six or seven times as high in duodenal obstruction as the pressure in the ileum in ileal obstruction. This he believed he could explain on the basis of the fact that there is more material secreted into the lumen of the duodenum than is secreted into the ileum or jejunum and this he believed would account for the relative severity and suddenness of the onset of the symptoms in high intestinal obstruction. Nowhere else in the intestinal tract is so much secretion found as comes from the Ampulla of Vater in the form of biliary and pancreatic secretions.

Raine, and Perry (42) have introduced experimental evidence to show that the essential factor in ileus is the embarrassment of the blood supply due to increased intraintestinal pressure. They found that whereas administration of water to rabbits in which experimental obstruction had been performed did not appear to influence the course of the condition favorably, administration of liquid albolene caused very rapid development of serious symptoms and signs. The explanation is offered that the water is absorbed rather readily from the intestinal tract, such absorption keeping the intra-intestinal pressure relatively low, whereas liquid albolene, not being absorbed at all, produces rather prompt increase in intra-intestinal pressure due to its bulk.

Dragstedt, Lang, and Millet (8) believe the anatomical topography of the intramural blood supply is of direct etiological significance in the development of ileus. They believe that increase of intra-intestinal pressure is responsible for circulatory embarrassment, but that the degree of embarrassment caused by a given increase in intraintestinal pressure is dependent on the relative length of the veins coursing through the intestinal wall, the intramural portion of the veins is longer in the upper intestinal tract and is occluded much more readily than the intra-mural portion of the veins which occur more distally and are shorter. They present good evidence that an intra-intestinal pressure of thirty five or forty five millimeters of mercury is safe within the ileum and colon, whereas in the duodenum and upper jejunum such a pressure immediately produces venous collapse.

It is both clinically and experimentally true that obstruction high in the intestinal tract is very much more speedily followed by symptoms and signs of disease than intestinal obstruction in the lower portion of the small intestine or in the colon. In fact, it would be essentially true to state that the time, onset, and severity of the symptoms of intestinal obstruction vary, the former directly and the latter inversely, with the distance between the pylorus and the site of the obstruction. Possibly the foregoing considerations account in whole or in part for this difference in reaction.

Patients with an obstruction in the small intestine rarely survive more than six to eight days unless the obstruction is overcome by suitable means, and even then such patients may survive indefinitely or succumb depending largely on the duration of the obstruction at the time relieved. Obstruction in the duodenum or uppermost jejunum may prove very much more rapidly fatal, the patients usually surviving such an obstruction for only two to three days. Obstruction within the large intestine is not inconsistent with life unless it is prolonged for very much more considerable periods of time, occasionally several weeks or even two to three months. The only invariable signs of intestinal obstruction are vomiting and constipation.

Vomiting is the most characteristic sign of obstruction of the intestine. It is at first accompanied by retching and the vomited material initially expelled consists of gastric and duodenal contents. Later the vomiting becomes passive and the vomited material may spill out of the mouth without apparent effort on the part of the patient. As the condition progresses and in case the obstruction is low in the intestinal tract, the contents of the jejunum, ileum, and colon are ejected in the order named, and in cases of low obstruction the vomited material may eventually become fecal in character. Fecal vomiting, however, constitutes a Therald of approaching death" and is not an essential factor in a timely diagnosis of obstruction. The picture is clinically the same whether the condition is paralytic or obstructive.

Constipation is an invariable sign, however, it does not, of course, become absolute at the time the obstruction occurs. Inasmuch as the intestinal canal below the point of obstruction may contain products of digestion and may continue to function normally a variable period of time the patient may expell several apparently normal stools. Flatus may be passed as long as the bowel below the point of obstruction still continues to function.

The temperature of the patient is characteristically either normal or subnormal. The pulse, normal at first, becomes progressively weaker and more rapid. Associated with the weak and rapid pulse is an abnormally low blood pressure. The respirations become progressively more and more shallow.

Pain may or may not be a prominent symptom, depending on whether the ileus is the obstructive or paralytic type. In obstructive ileus there is characteristically acute, intermittent, colicky, abdominal pain, similar to that produced by obstruction of any other hollow abdominal viscus. Adynamic or paralytic ileus characteristically is painless in its onset and course.

In the early stages of ileus the general condition of the patient is relatively good. In the typical and fully developed case the patient appears acutely ill, the eyes are bright, the cheeks sunken, and he may present the typical Hippocratic facies. The extremities are cold and clammy and the hands and feet present fibrillary twitching. There are beads of cold perspiration on the forehead, the lips and nail beds are frequently cyanotic. In the terminal stages deep coma may supervene followed rather rapidly by death. Previous to this, however, the patient may be unusually alert mentally, and acutely aware of his surroundings. He usually tosses about in bed, he may talk of trivialities, and make plans for the future. He rarely recognizes the seriousness of his condition unless he is specifically warned.

Patients with intestinal obstruction characteristically have a scanty urine, which shows traces of albumin and frequently a few casts. The non protein nitrogen characteristically increases from two to seven times the normal amount. The urinary chlorides are proportionately reduced. The kidney function tests, such as the Rountree_Geraghty test, may show a slightly decreased or even a normal urinary output. The red blood cell count is normal or somewhat increased. There is characteristically a leucopenia. The blood chlorides are characteristically decidedly reduced. The increase in the non protein nitrogen of the blood is due almost entirely to an increase in the urea content of the blood. The creatinine may be increased. the carbon dioxide combining power characteristically is markedly increased.

The diagnosis of intestinal obstruction is made on the basis of the history, the symptoms as previously outlined and the laboratory findings. The physical examination of patients with intestinal obstruction, except for some inconstant finding such as the presence of an external hernia, the contents of which cannot be reduced, is of relatively little importance. Inasmuch as one of the most frequent causes of postoperative ileus is the presence of angulations and strictures caused by the presence of postoperative adhesions, the diagnosis of ileus should be considered in all cases in which suspicious signs and symptoms occur, and in patients who have undergone either recent or remote laparotomies. Loops of intestine frequently become adherent to abdominal wounds and the site of the abdominal incision may sometimes give a valuable clue to the site of the obstruction. The occurence of cramps or colicky pains in the abdomen always indicates active peristalsis, and by his sensations the patient is frequently able to localize, with some accuracy, the relative location of the obstruction. It is more important to remember that the small intestine is the most frequent site of mechanical obstruction and this is doubtless due to the length of its mesentery.

The area of maximum distention in an abdomen the seat of obstruction, is frequently a valuable indication of the part of the small intestine involved. Obstruction low in the ileum characteristically produces abdominal distention in the lower part of the abdomen; the upper part of the abdomen being relatively flat. Distention in the lower right quadrant usually indicates that the obstruction is in the low, or the terminal portion of the ileum, whereas distention in the left side of the abdomen characteristically indicates obstruction in the jejunum. These considerations apply only to the dynamic type of ileus, for in paralytic ileus the distention is characteristically more generalized over the entire abdomen. Obstruction high in the jejunum and in the duodenum characteristically produce no distention at all.

Bartlett, (quoted by Guthrie (13), recommends spinal

anesthesia, postoperatively, as the best means of differentiating between adynamic and dynamic ileus. The patient is given a spinal anesthetic and if the bowels do not move within fifteen minutes the patient is taken to the operating room and an enterostomy or more radical operation is carried out while the patient is still under the spinal.

A type of postoperative ileus seldom considered is that produced by previously existing but unrecognized intestinal carcinoma. Although ileus produced by carcinoma of the gut is insidious in onset in two-thirds of cases, the other third produce symptoms of intestinal obstruction. The possibility of the occurence of such an obstruction during the course of postoperative convalescence should not be forgotten especially in patients past forty years of age.

In both types of obstruction a diagnostic proceedure of utmost value is the skiagraphic examination of the patient in the upright position. Administration of a contrast substance to the patient with suspected ileus is a distinctly dangerous proceedure because of the possibility of converting a partial obstruction into a complete one as the result of the accumulation of the contrast material in the constricted portion of the intestinal lumen. Schwarz, in 1911, showed that in cases of ileus the intestine contains both fluid and air, and the ordinary flat plate with the patient in the upright position is capable of showing the presence of multiple fluid levels surmounted by accumulations of gas. Case (5) has brought this to the attention of American surgeons. In obstruction of the colon there is no contra-indication to a barium enema. Roentgen examination may be diagnostic within six hours of the onset and is a very valuable early diagnostic measure. The use of methods to stimulate the motility of the intestine and the elimination of a diagnosis on the basis of subsequent bowel movements is dangerous because the lower bowel may evacuate even though ileus is present in the proximal portion.

The gross or microscopic changes in the various organs of the body are remarkably absent in patients dying from ileus. Most of the characteristic changes are found in the intestine itself. The splanchnic area shows extreme vascular engorgement, the intestine above the site of obstruction if not ruptured is dilated, and the intestinal wall is dusky or a bluish red color. There may be petechial hemorrhages both beneath the serosa and on the mucosa. The lumen of the intestine is characteristically filled with foul-smelling brown or reddish-brown fluid and the bacterial content is increased over normal. Microscopically the Intestinal wall shows remarkable capillary engorgement and occasional areas of necrosis, especially above the site of the obstruction. Fatty changes in the liver and microscopical evidences of toxic nephritis are reported, but are not universally found. In experimental animals Cutting found the adrenal gland to be increased to three to six times the normal size, and on microscopic examination of the cortex he has found multiple areas of focal necrosis with degeneration of the cells and pycnosis of the nuclear material . In the medulla the chromaffin material is reduced and there is infiltration of lymphoid and plasma cells.

The physico-chemical changes of ileus with their symptoms are usually called the toxemia of intestinal obstruction. There are four definite and significant physicochemical disturbances sufficiently demonstrated. All of these changes increase progressively with the development of ileus, and all tend to be more rapid and profound when the obstruction is high in the digestive tube. They are respectively: dehydration, alkalosis, hypochloremia, and increase in the non protein nitrogen of the blood.

Hartwell (15) has shown rather clearly that the dehydration found in ileus is the direct result of vomiting. The normal intestine produces within twenty four hours an amount of secretion almost equivalent to the entire amount of blood and lymph contained within the body, Starling (45). The dilated and partially strangulated intestinal wall proximal to the site of the obstruction is incapable of reabsorbing secretions which are poured into the lumen, regurgitation and vomiting occur, and thus the normal process by which the continual and progressive dehydration is prevented is reversed. In the terminal stages the toxemia of ileus produces the characteristic picture of shock; the peripheral capillaries undergo extreme dilatation and the patient develops profuse coldesweating which still further depletes the body of its water content.

There are various explanations of the hypochloremia of ileus. The fasting usually incident thereto, the accompanying increase in body temperature, the effect of alkali therapy, the altered renal threshold, vomiting, and detoxication retention are some of the factors considered. Most of these are unimportant. In the normal individual, feeding or with-holding of the chlorides affects the concentration of the chlorides in the blood little or not at all. Increases of body temperature do not regularly or extensively lower the renal threshold for chlorides and in any case fever rarely occurs as a symptom of uncomplicated ileus. Hypochloremia occurs even in those cases in which alkali therapy is not adopted and therefore failure

to provide alkali therapy is not explanatory. Haden and Orr (18) do not believe that vomiting accounts for the decrease of the chloridescontent of the blood, because they found consistently that such changes occurred in rabbits and monkeys, both of which do not vomit. As suggested by Cooper (6) however, these observations fail to take into account the considerable quantities of fluid which can accumulate in the dilated portion of the intestine proximal to the obstruction. Haden and Orr (19) have offered the explanation that the chlorides are removed from the blood and become fixed within the tissues in combination with toxic bodies. They believe these taxic bodies are neutralized by this process and become innocuous. The administration of chlorides in forms other than sodium chloride is apparently of no value. Gatch(11) Trussler, and Ayers found that tissue chlorides, as well as blood chlorides are reduced and in similar proportions.

Hastings, Murray, and Murray (17) were apparently the first to demonstrate that the carbon dioxide combining power of the blood is unusually high in intestinal obstruction. It is said that the chlorine ion accounts for fifty per cent of the acid forming elements in normal blood. The gastric mucosa secretes hydrochloric acid which contains the chlorine iop, and the liver and pancreas both secrete quantities of chlorine in the form of sodium or other chlorides. Whether these chloride ions are vomited and thus actually discharged from the body or whether they merely accumulate in the dilated intestine as in the cas e of animals like the rabbit which cannot vomit, the effect is the same. If the loss of the acid radical, chlorine, occurs so rapidly and extensively that the increase in the acid carbonate radical cannot compensate for it, alkalosis results. MacCallum, Lintz, Vermilye, Legget, and Boas (26) have formulated the idea that the hyperexcitability and tremor frequently shown by patients with excessive vomiting are actually manifestations of alkalosis, and on this basis they have suggested alkalosis as a cause of gastric tetany.

The blood non protein nitrogen increase which is observed characteristically in intestinal obstruction is very largely due to an increase in the urea concentration. This phenomenon was first described by Tileston, and Comfort in 1914 (46). It suggests impaired kidney function, but impairment of kidney function is not the cause of the condition, for the increase in urea concentration can be shown to be due, not to retention, but actually to increased formation of urea. Haden, and Orr (20) and Whipple, Cook, and Stearns (49) have shown that nitrogen excretion in the urine is not diminished in cases of ileus but is actually increased to four to five times the normal value. McQuarrie, and Whipple (28) have shown that the kidney may be damaged slightly, but that the amount of damage is inconsiderable.

The fact that the kidneys in ileus are capable of secreting so much urea serves as a good indication that kidney damage is no essential part of the picture. The finding of excessive amounts of urea in the urine disposes of the contention that the increase in blood urea is secondary to the dehydration and concentration of the blood. Since urea can be derived in no other way than from the destruction of proteins or amino acids, one is forced to conclude that in intestinal obstruction there occurs an exaggerated catabolism of the protein molecule somewhere in the body. Whipple and his co-workers (50) explained this on the basis of the action of some toxic body on the tissues, and believe that the administration of quantities of fluid dilutes the toxin and produces less protein cleavage. Hartman, and Smyth (16) suggest that the urea appearsiin the blood to compensate for the loss of chlorides. They say it has some action either in stabilizing the acidbase balance or in maintaining the proper osmotic relations. Bacon, Anslow, and Eppler (2) believe that in dehydration

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the water of dehydration is removed from the protein molecule to stabilize the water metabolism; the dehydrated protein molecules then disintegrate with resultant production of excessive quantities of urea.

The treatment of ileus may be considered under two main headings: prophylactic, and active treatment.

Prophylactic treatment consists of the avoidance of undue manipulation, care in the reperitonealization of denuded areas which prevents the development of extensive adhesions. Careful suturing of the abdominal wall prevents postoperative herniation and wound rupture, and the avoidance of any condition which might produce or predispose to angulation, kinking, or other factors producing mechanical occlusion of the intestinal canal all help in the prevention of the condition. Certain patients have a tendency to excessive connective tissue formation within the abdomen after laparotomy; the plastic exudate which always forms on the loops or intestine which are subjected to trauma, and which in most patients is subsequently liquified and absorbed, in patients showing this "keloid tendency" undergoes organization and contraction with age. Such patients are especially to postoperative adhesions. This of course cannot be recognized at the first operation but should be considered in subsequent ones for the relief

of adhesions. Attempts to combat the re-formation of adhesions by the introduction of various substances into the abdominal cavity at the time of operation have been relatively unsuccessful. The use of vegetable or animal digestants seems to hold some promise of success, papain, and possibly trypsin and also the use of amniotis fluid, or some extract of such fluid have been advocated.

The active treatment consists of two parts; 1. The re-establishment of the intestinal flow. Although no treatment of intestinal obstruction can possibly avail which does not include the restitution of the normal flow of the intestinal contents toward the anus, the mere reestablishment of the flow may or may not insure the patient's recovery, depending on the degree of the associated toxemia. 2.Clinically, the evacuation of the obstructed loop or segment of intestine, however accomplished, may either establish the process of recovery or may very soon precipitate the death of the patient.

In adynamic ileus one must attempt to bring about the restoration of the normal tone and motility to the paralyzed intestinal wall. The reason for the paralysis of the musculature of the intestine in adynamic ileus is by no means clear. Therefore the treatment of this variety is not as obvious as one could wish. Granting that the derangement consists in interference with the blood supply, then the local tissue anoxemia and the degenerative changes incidental to the vascular insufficiency may play an essential part in the etiology of the condition. If this is so, it would be rational to attempt to re-establish the normal blood supply. Assuming that the fundamental derangement is one of increased intra-intestinal pressure, the indication would be for the evacuation of the intestinal contents, which may be achieved to some extent, at least, by the performance of one or more enterostomies.

It must be admitted that this is not the view of the function of enterostomy most commonly held. Most of those who have written on the subject assume that the drainage of toxic material rather than the relief of intra-intestinal pressure is the important function of an enterostomy, and doubtless this toxic drainage is quite as important as the relief of stasis due to intra-intestinal pressure. In either case, however, the muscle cell profits directly by the proceedure of enterostomy.

Enterostomy is a formal operative proceedure and one which, though of minor rank in the normal individual, may assume major proportions because of the associated toxemia in patients with adynamic ileus. Certain patients with frank adynamic ileus undoubtedly develop spontaneous in-

intestinal movement under suitable accessory therapy and recover without enterostomy: consequently, the question of whether to enterostomize, and if so when, often gives for considerable anxiety.

According to lahey (24) in postoperative cases in which non-mechanical obstruction is diagnosed with reasonable certainty, non-operative measures may usually be employed for about four days with safety. Those obstructions which do not manifest themselves as mechanical will often be subsequently found to have been resultant upon local inflammatory processes which eventually become localized intra-peritoneal abscesses. To attempt to drain such an inflammatory accumulation early, especially when such a condition is diagnosed merely on suspicion, is distinctly dangerous. A delay until the process has walled itself off and a definite mass becomes palpable is usually entirely safe and the treatment of such a walled off process, e.g., in the cul-de-sac of Douglas, is thoroughly satisfactory.

The preoperative and postoperative care of the patient about to undergo an enterostomy is important. Special care is taken to withhold all the food and fluid by mouth and administration of all cathartic drugs is avoided. Preoperative gastric lavage is of particular importance and

should be performed routinely, especially immediately preceeding the operation. This removes any toxic material that is in the stomach and relieves the stomach and the upper part of the intestinal tract of the pressure incident to the presence of this material and by the relief of distention places this portion of the alimentary canal in a favorable condition for the re-establishment of the normal intestinal movement. Lavage also tends to prevent the regurgitation of the stomach contents into the mouth and pharynx during the operation and thus prevents the possibility of aspiration pneumonia.

The selection of the anesthetic is important. Ordinarily the inhalation anesthetics are contra-indicated. The ordinary local analgesia is the method of choice in some cases, but spinal analgesia and especially splanchnic analgesia are good inasmuch as both have a tendency to restore normal motility to the intestinal tube.

Cecostomy is of very little value for this purpose in most cases, because (1) in the primary paralytic variety of ileus there is only local drainage of toxic products, there being no peristalsis to clear the proximal coils, and (2) in paralytic ileus engratted upon the obstructive variety the obstruction is usually higher in the small intestine. Enterostomy through an incision in the left hypocho hypochondrium into the upper jejunum by the method of Witzel, bringing the enterostomy tube out through a small rent in the omentum after the method of Mayo, has much to recommend it, since by this means the gut is usually drained proximal to the mechanical obstruction if such be the variety, and if the ileus be primarily adynamic the upper portion of the jejunum contains the most virulent toxic material and it is in this variety the logical place to perform drainage.

Jejunostomy was advocated for this purpose by Heidenhain as early as 1897. Lee and Downs (25), proceeding on the assumption that toxic products were primarily absorbed from the upper part of the intestinal tract regardless of the site of the obstruction have recently emphasized the value of the method. Haden and Orr (21), however, found experimentally that, if a high intestinal obstruction and a jejunostomy are performed in experimental animals both at the same time, the period of survival of these animals is considerably shorter than if no jejunostomy is performed. They found, however, that when animals with a simple enterostomy received quantities of 1% NaCl solution hypodermically the period of survival was much increased, and as the administration of a 1% NaCl solution is a relatively simple matter they state that "when any doubt exists concerning the value of an enterostomy it should be performed". Van Beuren (47), after a study af the cases of acute ileus operated in one hospital during the past twelve years, found a progressive reduction of the mortality rate in the more recent years, which he attribites, first, to the increasing prevalence of early diagnosis, and second, to the use of early enterostomy. He believes that enterostomy performed properly and at the proper time is of great value. Guthrie (13) states that it would be just as hard to attempt to do goiter surgery without the use of Lugol's solution, or treat diabetes without insulin as to attempt abdominal surgery in these cases without the use of enterostomy.

The second possible line of attack on the motor function of the tissue cells of the intestinal musculature consists of stimulation by drugs, the function of which may be either to increase the irritability of the muscle cell or decrease the threshold for normal stimuli conveyed to the cell by the nervous regulatory system. Among the drugs used for this purpose are serine, pituitrin, pitocin, choline, acetyl choline, and peristaltin. As Ochsner, Gage, and Cutting (37) have shown, all these drugs produce a more profound effect on the intestine which has previously undergone experimental obstruction than on the normal intestine. However, with the

single exception of eserine, none of these therapeutic agents are active enough to be of any value in the restitution of normal motility, or any degree of motility approaching normal, to a gut the seat of ileus. The drug therapy of ileus would therefore seem very unimportant.

Recently various investigators have reported favorably on the use of hypertonic NaCl solution for its effect on intestinal motility. Hughson and Scarff (22) found that the intravenous administration of hypertonic NaCl solution produced violent peristalsis in animals. This observation was also corroborated by Ross (43). Ochsner, Gage, and Cutting (37) have found that the administration of 20% NaCl solution is rather effective in producing increased tone and increased amplitude of intestinal movements. If NaCl canbbe shown to be an efficient intestinal stimulant it can serve a double purpose, since the blood chlorides are characteristically decreased and the administration of hypertonic NaCl solution would not only increase the tone of intestinal movement but would be of value in restoring the normal chloride concentration to the blood.

Because of the inefficacy of drug therapy designed to stimulate the individual muscle cells or neuromuscular mechanism within the wall of the intestine, various attempts have been made to attack the problem from the extrinsic regulatory nervous system point of view. It is generally accepted that the small intestine, at least, is controlled by a dual and antagonistic nerve supply. Domenech and others have assumed that in paralytic ileus there is an irritative effect on the splanchnic nerves. It is on this theory that the use of splanchnic and spinal analgesia is based. In spinal analgesia the splanchnic fibers are anesthetized at their source as they make their exit from the anterior horn of the various spinal cord segments. In splanchnic analgesia the reflex are is interrupted after the splanchnic nerves have become fully formed and as they enter into the formation of the great and lesser splanchnic plexuses in the retroperitoneal space in front of the bodies of the last thoracic and the first lumbar vertebrae.

Theoretically spinal and splanchnic analgesia should be equally efficient in relieving adynamic ileus, as both proceedures produce chemical section of the splanchnic nerves. Clinically, spinal analgesia has been used more frequently for this purpose than splanchnic analgesia. Wagner, in 1922, gave the first report of the use of spinal analgesia for the relief of ileus. Since this numerous observations have been made on the use of the proceedure. Markowitz, and Campbell (27) have successfully employed spinal analgesia

in treating experimentally produced ileus in dogs. Splanchnic analgesia was suggested by Ochsner, Gage and Cutting (38) as a preferable method of producing a similar effect, and recently, they have presented evidence to show that, at least in experimental animals, splanchnic analgesia is actually more efficient. Rosenstein and Kohler, also Alvarez (1) recognizing the efficiency of splanchnic analgesia, have advocated the use of nicotine by injection into the splanchnic area. They believe that nicotine should be more efficient than novocaine because nicotine has a specific action on the sympathetic ganglia. In their experimental investigation they injected nicotine solution directly into the semilunar ganglion and believed they accomplished by this method a more complete chemical section of the splanchnic nerves than could be achieved by ordinary novocaine solution. Injection of any solution, however, directly into the semilunar ganglion in the human being is quite impossible, the methods available for induction of splanchnic analgesia being hardly equal to such an accurate localization of drugs. Furthermore, Ochsner, Gage, and Cutting (38) found that nicotine solution injected in the ordinary way not only produces less adequate effects than novocaine, but the blood pressure of experimental animals is reduced

to a dangerous degree by nicotine.

The explanation offered for the superiority of splanchnic over spinal analgesia is that the reflex arc concerned in the production of ileus is entirely within the splanchnic system. Splanchnic analgesia interrupts this arc in its entirety, presumably producing both sensory and motor effects. Spinal analgesia interrupts only such portions of the arc as pursue a course within the spinal cord.

More clinical data than is at present available must be collected before either relative or absolute value of these methods can be definitely established. In using splanchnic or spinal analgesia it is essential to realize that success can be expected only in cases in which the intestinal lumen is patent and only in case the therapy is used early. These methods are intended to re-enforce the treatment of the associated toxemia, and, inasmuch as neither of these methods affect the large intestine such treatment should always be supplemented by the administration of an enema. A point of interest made by Ochsner, Gage and Cutting (38) is that the effect of splanchnic and spinal analgesia can be absolutely overcome by epinephrine or ephedrine. Consequently neither of these should be used in conjunction with either method when used for the relief of ileus. Furthermore, since the action of the splanchnic and spinal analgesia is rather fleeting, no hesitation should be felt in repeating either proceedure as many times as may be deemed desirable at intervals of an hour or so.

Unfortunately the treatment of the toxemia of ileus is almost entirely symptomatic. Undoubtedly a clearer understanding of the source and method of production of the toxemia would simplify the treatment considerably, but until such an understanding is gained the best that can be done is to attempt to combat the manifestations of perverted metabolism insofar as they are known.

The dehydration and hypochloremia are conveniently treated together. The obvious treatment is the administration of quantities of NaCl solution. In as much as the administration of anything by mouth is strictly contraindicated in ileus, fluids must be given either rectally, subcutaneously or intravenously. Orr and Haden recommend the intravenous administration of 500 cc. of 5% NaCl solution in all cases subjected to operative proceedures. Eliason (9) recommends a 5% glucose solution in normal salt solution, McVicar and Weir(29) recommend a 10% glucose solution in 1% NaCl solution.

The addition of hypertonic dextrose solution to sol-

utions for intravenous administrations is recommended because of the effect on diuresis. This is valuable, there, fore, in removing the excess nitrogenous waste products from the blood stream. Postoperatively, and at all times, patients with ileus should be subjected to blood chemistry examination at frequent intervals, at least two times a day, to determine the efficiency of treatment.

Careful hemoglobin estimations should be performed to determine whether or not the blood concentration is being sufficiently combated. Plasma chloride determinations should be made to determine the presence or absence of hypochloremia; Non Protein Nitrogen determinations to determine the presence or absence of Nitrogen retention, and estimations of the carbon dioxide combining power of the plasma to determine the status of the acid-base balance. It would be futile to attempt to state either the amount or the composition of fluid suitable Most cases require at least from three to five for all cases. liters of fluid every twenty-four hours. All patients need NaCl either in normal hypertonic solution, and probably most patients need dextrose in from five to ten per cent solution. Requirements of the individual patients with regard to these substances vary and it is only by the aid of accurate and frequent laboratory findings that the proper amount of the

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substances can be determined with accuracy.

Gastric lavage has been mentioned as a valuable preoperative proceedure, but frequent lavage of the stomach postoperatively is almost equally important both in the relief of toxemia and in the promotion of the patient's comfort. Muller (35) has ventured the hypothesis that in paralytic ileus decrease of movement in the intestinal tract is associated with increase in secretion. He believes that the application of heat to the abdomen produces a dilatation of the peripheral blood vessels in the abdomen which is associated with contraction of the splanchnic vessels by the mechanism of visceral splanchnic balance. Thus contraction of splanchnic vessels not only diminishes intestinal secretion but also increases intestinal motility.

A number of attempts have been made recently to incriminate the B. Welchii group of organisms in the production of the toxemia of intestinal obstruction. Williams (53), in 1926, fostered this theory. He found that B. Welchii grew best in slightly acid or neutral medium, and considered that such medium was found in the upper, rather than the lower, portion of the intestinal tract. He made use of immune serum in the clinical treatment of certain cases and reported favorable results. Experimentally, McIver, White and Lawson (30), and Stabius and Kennedy (44) found that whereas B. Welchii was present in small numbers in the normal intestinal canal, they increased remarkably in the presence of intestinal obstruction. Morton, and Scabins (32), and Bower, and Clark (3) following the lead of the previously named authors, have reported favorable results with antitoxin, the former in experimental and the latter in clinical cases. Oughterson, and Powers (39) and Owings, and McIntosh (40) have, however, been unable to confirm these findings, the former making use of antitoxin and serum obtained by active immunization of animals against B. Welchii, the latter employing B. perfringeus antitoxin.

The prognosis of a well established case of intestinal obstruction is invariably grave. When associated with peritonitis, the prognosis is even more grave. The time elapsing between the onset of the obstruction and the performance of the operative proceedures is of chief prognostic importance. Van Beuren (47) says that the longer a patient with intestinal obstruction lives prior to operation the sooner he dies afterward.

Miller (31) states that as a rule the mortality rises about one per cent per hour of delay in the relief of the obstruction. He found in reviewing 343 cases, that the mortality rate, when the patient was subjected to operative proceedures within twelve hours of the onset of symptoms, was only 29.4 %. Within 24 hours this percentage was increased to 42.9 %; within 36 hours to 50 %; within 48 hours to 59.6 %; within 72 hours to 63.4 %; within 96 hours an 84 % mortality was observed.

Brill (4), in a series of 83 cases, found the mortality rate nil when the patients were subjected to operation within twelve hours of the onset of symptoms. When such operations were performed in periods between twelve and twenty-four hours, there was a 12.5 % mortality, and between twenty-four and fortyeight hours a 61.1 % mortality.

Altogether the mortality is high. However this cannot be improved upon until the cause of the condition is determined more definitely. Then the treatment of the condition can be placed on a sound and logical basis.

BIBLIOGRAPHY.

- 1. Alvarez, W. C. Mechanics of the Digestive Tract, pg. 11, 1928.
- 2. Bacon, D. K., Anslow, R. E., and Eppler, H. H. Intestinal obstruction. Arch. Surg., 3: 641, 1921.
- 3. Bower, J. O., and Clark, I. Intestinal obstruction; B. welchii (perfringens) antitoxin; therapeutic value; preliminary report based on twenty-five cases. Am. J. M. Sc., 176: 97, 1928.
- 4. Brill, S. Mortality of Intestinal obstruction. Ann. Surg., 89: 541, 1929.
- 5. Case, I. I. Value of roentgen examination in early diagnosis of post-operative ileus. Ann. Surg. 79: 715, 1924.
- 6. Cooper, H. S. F. Cause of death in high obstruction. Arch. Surg., 17: 918, 1928.
- Delprat, G. D. and Weeks, A. Postoperative ileus.
 Am. J. Surg. 8: 1189-1193, June '30.
- B. Dragstedt, C. A., Lang, V. F., and Millet, R. T. Relative effects of distention on different portions of intestine. Arch. Surg., 18: 2257, 1929.
- 9. Eliason, E.L. Treatment of acute intestinal obstruction. Pennsylvania M. J., 32: 349, 1929.

- 10. Estrem, C. O. Postoperative ileus. Minn. Med., 11: 83-86, Feb., 1928.
- 11. Gatch, W. D., Trussler, H. M., and Ayers, K. D. Acute intestinal obstruction: mechanism and significance of hypochloremia and other blood chemical changes. Am. J. M. Sc., 173: 649, 1927.
- 12. Grover, G. G. Paralytic ileus. Northwest Med., 30: 178-181, April '31.
- 13. Guthrie, D. Postoperative ileus; early recognition and control. N. Y. State J. Med. 31: 1021-1024, Aug. 15, '31.
- 14. Handley, Sir S., Wilkie, D. P. D., and Taylor, W. Acute intestinal obstruction. Brit. M. J. 2: 993-1004, 1925.
- 15. Hartwell, J. A., and Hoguet, J. P. Experimental intestinal obstruction in dogs with especial reference to the cause of death and treatment by large amounts of normal saline solution. J. A. M. A., 59: 82, 1912.
- 16. Hartman, A. F., and Smyth, F. S. Chemical changes in body occuring as result of vomiting. Am. J. Dis. Child., 32: 1, 1926.

- 17. Hastings, H. B., Murray, C. D., and Murray, H. A. Certain chemical changes in blood after pyloric obstruction in dogs. J. Biol. Chem., 46: 223, 1921.
- Haden, R. L., and Orr, T. G. Chemical changes in blood of dog after pyloric obstruction. J. Exper. Med., 37: 365, 1923.
- 19. Haden, R. L., and Orr. T. G. Effect of NaCl on chemical changes in blood of dog after pyloric and intestinal obstruction. J. Exper. Med., 38: 55, 1923.
- 20. Haden, R. L., and Orr, T. G. Cause of certain acute symptoms following gastroenterostomy. Bull. Johns Hopkins Hosp., 34: 26, 1923.
- 21. Haden, R. L., and Orr, T. G. High jejunostomy in intestinal obstruction. J. A. M. A. 87: 632, 1926.
- 22. Hughson, W., and Scarff, J. E. Influence of intravenous NaCl on intestinal absorption and peristalsis. Bull. Johns Hopkins Hosp. 35: 197, 1924.
- 23. Kappis, cited by Ochsner, A., Gage, I. M., and Cutting, R. A. The treatment of ileus with splanchnic and spinal anesthesia. Anesthesia and Anal., 9: 91-94, Mar: Apr., 1930.

- 24. Lahey, F. H. Management of some complications following abdominal operations. J. A. M. A., 89: 1735, 1927.
- 25. Lee, W. E., and Downs, T. Treatment of acute mechanical intestinal obstruction by high temporary jejunostomy. Ann. Surg., 80: 45, 1924.
- 26. MacCallum, W. G., Lintz, J., Vermilye, H. N., Legget, T. H., and Boas, E. Effect of pyloric obstruction in relation to gastric tetany. Bull. Johns Hopkins Hosp., 31: 1, 1920.
- 27. Markowitz, J., and Campbell, W. R. Relief of experimental ileus by spinal anesthesia. Am. J. Physiol., 81: 101, 1927.
- 28. McQuarrie, I., and Whipple, G. H. Renal function influenced by intestinal obstruction. J. Exper. Med., 29: 397, 1919.
- 29. McVicar, C. S., Weir, J. E. Nature and treatment of intestinal obstruction and ileus. J. A. M. A., 92: 887, 1929.
- 30. McIver, M. A., White, M. A., Lawson, J. C. The role of B. Welchii in acute intestinal obstruction. Ann. Surg., 89: 647, 1929.

- 31. Miller, C. J. A study of 343 surgical cases of intestinal obstruction. Ann. Surg., 89: 91, 1929.
- 32. Morton, J. J., and Scabins, S. J. Relation of B. welchii antitoxin to toxemia in intestinal obstruction. Arch. Surg., 17: 860, 1928.
- 33. Morton, J. J. The difference between high and low intestinal obstruction in dog. Arch. Surg., 18: 1119, 1929.
- 34. Moynihan, Berkeley. Textbook of abdominal surgery, pg. 461, 1906.
- 35. Muller, E. F., and Peterson, W. F. Lymph production and heat regulation. Proc. Exper. Biol. & Med. 26: 169, 1928.
- 36. Murphy, F. J., and Brooks, B. Intestinal obstruction: an experimental study of the causes of the symptoms and death. Arch. Int. Med., 15: 392, 1915.
- 37. Ochsner, A., Gage, I. M., and Cutting, R. A. Value of drugs in relief of ileus. Arch. Surg. 21: 924, 1930.
- 38. Ochsner, A., Gage, I. M., and Cutting, R. A. The treatment of ileus with splanchnic analgesia. J. A. M. A. 90: 1847, 1928.
- 39. Oughterson, A. W., and Powers, J. H. Relationship of toxin of B. welchii to toxemia of intestinal obstruction. Arch. Surg., 18: 2019, 1929.

- 40. Owings, J. C., and McIntosh, C. A. Perfringens antitoxin and experimental intestinal obstruction. Arch. Surg., 18: 2237, 1929.
- 41. Owings, J. C., McIntosh, C. A., Stone, H. H., and Weinberg, J. A. Intestinal pressure in obstruction. Arch. Surg., 17: 507, 1928.
- 42. Raine, F., and Perry, M. C. Intestinal Obstruction experimental studies on toxicity, intra-intestinal pressure and chloride therapy. Arch Surg., 19: 478, 1929.
- 43. Ross, J. W. Hypertonic saline in adynamic ileus. Canad. M. A. J. 16: 241, 1926.
- 44. Stabins, S. J., and Kennedy, J. A. The occurence of B. welchii in experimental high obstruction. Arch. Surg., 18: 753, 1929.
- 45. Starling, E. H. Textbook of Physiology, pg. 584, 1930.
- 46. Tileson, W., and Comfort, E. W. Total N P N of urea of blood in health and disease as estimated by Folin's method. Arch. Int. Med., 14: 620, 1914.
- 47. Van Beuren, F. T. Jr. Relation between intestinal damage and delayed operation in acute mechanical ileus. Ann. Surg., 72: 610, 1920.

- 48. Ward, Robertson. Acute general peritonitis. Calif. and West Med., 395, Dec., 1929.
- 49. Whipple, G. H., Cook, J. V., and Stearns, I. Proteose intoxixation and injury to body protein: metabolism of dogs with duodenal obstruction and isolated loops. J. Exper. Med., 25: 479, 1917.
- 50. Whipple, G. H., Stone, H. B., and Bernheim, B. M. Intestinal obstruction. J. Exper. Med., 17: 307, 1913.
- 51. Wilkie, D. P. D. Acute intestinal obstruction. Lancet, 1: 1135, 1922.
- 52. Wilkie, D. P. D. Intestinal Obstruction. Brit. Med. J. 3: 993-1004, 1925.
- 53. Williams, B. W. Importance of toxemia due to anaerobic organisms in intestinal obstruction and peritonitis. British J. Surg., 14: 295, 1926.