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THE TREATMENT OF POSTOPERATIVE ADYNAMIC ILEUS

THERE are certain postoperative complications which are always potential dangers to a patient and a concern to the surgeon, no matter how simple the operation. Thus, there is no operation which does not entail a risk regardless of its seriousness or extent of the procedure. We all agree, I am sure, that this risk or potential danger varies with different procedures. I could name numerous complications, a number of which may be anticipated. Prevention of this is often possible by preoperative care, such as the diabetic patient, the obstructive jaundiced case, the bad cardiac, but not so with the patient unfortunate enough to develop an adynamic ileus.

This complication is most distressing and may be present from a very mild form to a severe type causing death. Much work has been done regarding this complication but very little has been given us regarding the etiology or method of prevention. There are, however, some facts of importance in the treatment of adynamic ileus.

As to the etiological factors much has been speculated, such as infection, trauma, type of operation, the cleansing of the gastro-intestinal tract preoperatively, anesthesia and the like. If, however, we study statistics on cases developing adynamic ileus none of these factors is present consistently in all cases, excepting perhaps one. That is the trauma of an operative procedure. Infection when present naturally must be accepted as a cause in the infected case.

It is a common observation that gallbladder and extraperitoneal operations (kidney) are more prone to cause it

than are operations on the gastro-intestinal tract, if, of course, we exclude acute appendicitis and peritonitis cases. I know of no way preoperatively to tell just which case may develop it or just how severe any one case will be once it develops.

As to the physiology of adynamic ileus little is known excepting that once it exists in a small way and not treated it progresses and finally simulates an intestinal obstruction, excepting that we do not have until late the obstruction of the blood supply to the bowel with its subsequent gangrene. Let us look for a moment at what happens in the paralytically obstructed bowel. The cause of this paralysis of the bowel musculature may be due to trauma at operation; injury and disease of the central or splanchnic nervous systems; poisons, bacterial or chemical; embolism and thrombosis of the mesenteric vessels, or nerve lesions. Pneumonia is also a causative agent of ileus probably by toxic inhibitory action on the nerves. The ileus often is transitory and after a few hours function returns and peristalsis begins. In the event it is longer than just a transitory state we have the progressing picture of obstruction.

Because the secretion of the stomach and bowel continues but does not pass on, there is an accumulation in the bowel with distention. The bacteria in the bowel contents increase very rapidly, as do the secretions. Some of the bacteria are gas producing and other sources of gas are transudation of CO_2 from venous blood and swallowed air. Ultimately this retained content of the bowel, because of chemical and bacterial action, becomes very toxic. As a result of the distention and toxicity the pressure increase in the bowel compromises the circulatory pressure and we have necrosis with digestion of mucosa and the liberation of another toxin. The distention may go on until relief is obtained by overflow into the stomach with vomiting. The distention increasing causes paralysis, thus a vicious circle is established. The amount of toxemia is in general proportionate to the amount of damage to the gut wall. The bacterial content is usually made up of the colon bacillus group and the anaerobes, particularly the Bacil-

lus welchii, depending on the amount of bowel involved. Stone[1] showed that the action of toxins in bowel is almost identical with that of histamine. Whipple[2] shows similarity of the action of the toxin to the reactions seen in conjunction with large overdoses of adrenalin and fatal anaphylactic shock.

What chemical changes take place in the body in adynamic ileus? The urea is markedly increased as it forms from the breaking down of the amino-acids, which in turn come from the splitting of the protein molecule. Thus, the high urea is an index of the rate at which the proteins are being broken down and this rate is faster than the kidneys can throw it off.

The low blood chloride must be considered in these cases. Work to explain the loss of chlorides has been done. Haden and Orr[3] tried to account for this loss in the urine and vomitus but could not account for all of the loss this way. Gatch[4] examined quantitatively the vomitus, urine, blood and tissues. He found that the tissue chlorides were reduced in the same proportion as the blood chlorides.

The acid-base regulating mechanism is disturbed as shown by the high CO_2 combining power of the blood. The H ion, as is well known, is the most easily regulated of all the factors in maintaining the *p*H of the blood. If there is a loss of acid-forming radicals from the body the CO_2 combining power of the blood rises and compensates: In vomiting we have just that, because of the depletion of the Cl ions. We may therefore assume that the CO_2 combining power of the blood goes up to compensate for this loss. If this loss of acid is so rapid that the increase in HCO_3 and other acid elements cannot keep up with it there is a resulting alkalosis. Gamble[5] believes that this disturbance of the acid-base adjusting mechanism is the big factor in the toxemia. Chlorine is lost in HCl and NaCl while sodium is lost only in the NaCl, therefore there is much more loss of Cl than the base Na.

Thus in summing up the chemical changes taking place we have, first, the dehydration and the accompanying disturbance of the acid-base regulating mechanism; and secondly, a varying toxemia depending on the degree of ileus.

Barden, Thompson, Ravdin and Frank[6] have recently shown that low serum protein has a direct relationship to small bowel motility and that in the presence of low serum protein the motility is greatly reduced. This also must be considered not only from the standpoint of effect from ileus but must be considered as a possible etiological factor.

I have briefly tried to cover the highlights in the physiological pathology occurring in adynamic ileus in the hope it will explain the rationale of the treatment to be presented in the following paragraphs.

For clarity of discussion I would like to consider adynamic ileus under two separate headings and feel you will agree with the classification. The first type is the infective or septic type, and the second is the noninfective or nonseptic type. I will discuss the two types separately since the treatment should vary with the type.

Infective Type.—By the infective type I mean the case with a localized, diffusing or diffuse peritonitis whether bacterial, as from a ruptured appendix or bowel, or chemical as from a ruptured (traumatic) gallbladder, thus bile in the peritoneal cavity and blood in the peritoneal cavity. I prefer to discuss this chemical peritonitis group under the infected type since the treatment is more or less the same.

This infected group thus resolves itself to the adynamic ileus cases resulting from or associated with peritonitis. Descriptions have been made where in cases of pelvic peritonitis only the bowel lying in the pelvis, which might be several loops of small bowel, and the sigmoid were involved by adynamic ileus, the rest of the bowel being normal. This has been called ileus duplex by Handley.

It was on the basis of nature producing an ileus in peritonitis that caused the late John B. Murphy to advocate anatomical and physiological rest in the treatment of these cases to assist nature in what she was trying to do. As a rule, and we all have seen it, in the cases of peritonitis once an infection is controlled and begins to improve the ileus will disappear and the peristalsis improves. Recently in a study of cases of acute

ileus by van Beuren, Jr., and Smith[7] they cite 13 cases of adynamic ileus in their series with 1 recovery and 12 deaths, or a mortality of 92.3 per cent. If, however, we consider the 12 deaths—1 died of pneumonia and uremia, 1 died of adynamic ileus per se (age seventy-one), 1 of thrombosis and the rest of peritonitis. I am calling your attention to this citation because while all of these patients had an infective type adynamic ileus only one apparently died of ileus, the majority dying of peritonitis. This is mentioned so that you will see that in these cases it is most important to treat the peritonitis. The only conditions under which the bowel function will not return after peritonitis subsides are where we have a mechanical obstruction from adhesions or exceedingly low serum protein.

These cases of septic ileus show fever and rapid pulse as we have in peritonitis. The abdomen is distended and tense. At first there is muscle rigidity but as the peritonitis improves we have the distention without the muscle rigidity. The abdomen is usually silent all during this time. Tenderness on pressure is at first present over the abdomen, but again as the peritonitis improves the tenderness becomes less although the ileus still persists. Vomiting is present and consists of gastric and intestinal secretions. As long as we have ileus present there is a tendency for the pulse rate to be up a bit, but not as marked as the increase due to the peritonitis. From the standpoint of treatment it is very important to decide just when the peritonitis has cleared up and just the residual ileus remains. As a rule, the disappearance of rigidity, decrease of tenderness, lowering of temperature and lowering of pulse rate are all factors showing the subsidence of the peritonitis. The abdomen may even become a bit softer although still distended.

Treatment.—As to the treatment of the septic type of adynamic ileus, the most important discussion is the treatment of the septic condition causing it.

The method of treatment of the peritonitis must be modified and molded depending on the etiological factor responsible.

For example, operation for perforated ulcer must necessarily be done as early as possible, where a case of peritonitis due to pelvic inflammatory disease should not be operated on early but treated with conservative measures and later perhaps Elliott treatments. Thus the individual etiological factor presents its individual problem as to care. The surgeon's experience is the most important factor in guiding his care. I will not go into the operative procedure as we are more concerned about the treatment of ileus. The mortality in the septic ileus is high but this chiefly because of the septic condition and not so much the ileus.

After one has decided the peritonitis is improving, as shown by lower temperature, slowing pulse rate, diminished rigidity and decreased tenderness, active treatment of ileus should be started. Conservative measures are, of course, used all during the treatment of the peritonitis, such as Jutte tube, infusions, transfusions, check on serum protein and the like, but no active drug treatment should be used to overcome the ileus until the peritonitis has subsided. I will discuss the general treatment of this condition under nonseptic ileus to save repetition.

Nonseptic Type.—I would like next to discuss the nonseptic ileus which we more often encounter and which in its varying degrees has confronted the surgeon for some time. This condition is present from slight postoperative distention with gas pains to the markedly distended silent abdomen.

As to the etiological factors causing this type only a few are known. There are still many cases without a known cause.

Trauma is a common cause and most of this is due to operative technic. One must remember that tissue cells are delicate structures. This is particularly true of the contents of the abdominal cavity. One must handle these tissues with great delicacy. The late John B. Deaver spoke of it as "handling the tissues lovingly as though with a touch of a woman." While this may be exaggerating the point a bit nevertheless it is true. If I can impress surgeons with this one point it will

be worth while, no so much to the surgeon perhaps but certainly to the patient and his comfort.

Another etiological factor seems to be certain types of operations. We rarely see it after operations on the small bowel, large bowel or stomach and yet it is fairly common after pelvic operations such as hysterectomies and also after cholecystectomies. The answer to the above might be that we defer the giving of fluids longer in gastric and intestinal operations than we do in operations on the pelvic or the biliary tract. Another possibility is that the cases for operation on the gastro-intestinal tract are prepared a bit more thoroughly.

Too much morphine to a postoperative case might be a factor since morphine has been shown to cause spasm of the common duct with increase in the biliary pressure, thus imperfect digestion with distention.

The nervous origin has been much discussed and yet there are no facts to guide us other than that the nervous, apprehensive patient is more prone perhaps to develop some degree of ileus.

The low serum protein may be a cause of ileus as shown recently by Barden, Thompson, Ravdin and Frank. They demonstrated that with a low serum protein there was marked retardation in the motility of the small bowel.

Hypochloremia may be a factor and proved to be in a case recently on the service of Dr. George P. Muller. With the correction of the hypochloremia the ileus disappeared. The serum protein was normal in this patient throughout.

As to the physiology in ileus it is similar to intestinal obstruction and has been discussed earlier in this paper. It is important to keep this in mind as it is always a guide to the treatment in the particular case.

The symptoms of nonseptic adynamic ileus are simple and clear. The patient, usually a postoperative case, will complain of distention, rarely pain but discomfort. There is nausea always later if not present at first. The temperature will be normal or only slightly elevated at first; later as the toxemia increases the temperature may increase. Pulse rate at first is

slow, later becomes rapid. Examination reveals a distended abdomen which may become tense, a few peristaltic waves may be heard at first or it may be a silent abdomen. There is no rigidity. As the ileus increases there may be embarrassment to respiration because of diaphragmatic pressure. There will be no flatus or feces passed by rectum or perhaps only a small amount.

All of these symptoms and findings become worse without treatment, and a case which seems at first so simple, if neglected may become so severe that death results. Thus the importance of immediate treatment no matter how mild the condition appears.

Treatment.—In considering treatment I will outline and discuss the different weapons we have. At times it will be important to remember the use of certain of them depending on the etiological factor causing the ileus.

As soon as the diagnosis of adynamic ileus is made, whether nonseptic or the septic type, when peritonitis has subsided start treatment at once. First, of course, it is important to have blood chemistry done. Find out the plasma CO_2, serum protein and blood chlorides. These three chemical tests are important in guiding your treatment.

If one uses common sense in treatment the first thing we think of is to empty the contents of the stomach and bowel. There are several tubes which will serve this purpose.

The Jutte and Levine tubes you are all familiar with and I need not go into detail. I believe that either of these tubes should be passed at once and a continuous suction employed after the method of Wangensteen[8] or the method of Welch.[9] This continuous suction is important and should the drainage stop the tube should be immediately irrigated with tap water or saline to insure a clear lumen for good drainage.

Another tube which I have used on several occasions is the Miller-Abbott double lumen tube.[10] This tube can be passed all the way to the large bowel. The continuous suction also connects on this tube. It is perfect in a mechanical obstruction and will go down to the point of obstruction.

Thus we must accept that a gastric or duodenal tube with suction is the first important step in any ileus.

Next we must consider the nourishment and fluid into the patient. One must keep the fluid and chloride intake going, also some glucose for the liver. This is done by continuous infusion and we make a practice of alternating the fluid in the infusion. The first 500 cc. made up of normal saline solution; the next 500 cc. made up of distilled water and 5 per cent glucose. By alternating this way one runs less chance of causing a hyperchloremia. We usually try to get about 3000 cc. of fluid into the patient in twenty-four hours. The amount of glucose pushed is usually 100 Gm. or better. This fluid and chlorides must be varied with the patient's needs.

Should the case be one of low serum protein per se, transfusions with lyophilized plasma are of assistance in restoring serum protein. Plain transfusion is also of value. One should always therefore keep in mind the possibility of the hypoproteinemic state.

Enemas may be of assistance in emptying the colon and stimulating reflexly small bowel activity. For this purpose we like to use a combination of glycerin and water, about 90 cc. of each, given warm and slowly and retained for a while if possible.

Turning the patient at short intervals from side to side is also at times of assistance and should be kept in mind.

The concentrated oxygen therapy after Fine[11] may be of assistance in removing the gas from the bowel. The inhalation of pure oxygen prevents the entrance of atmospheric nitrogen into the lungs. Because of this the nitrogen in the tissues and the blood is rapidly exhaled. Hydrogen is also rapidly absorbed and removed from the bowel in concentrated oxygen.

We also have evidence to believe that histamine is one of the toxic products formed in the bowel during ileus, and McHenry and Gavin, at the University of Toronto, showed that the histamine-histaminase reaction proceeds slowly in an atmosphere of nitrogen but is accelerated by oxygen, and therefore is an oxidative reaction. It seems therefore that Fine's

95 per cent oxygen therapy not only removes the nitrogen and hydrogen from the distended bowel but also increases the oxidation of histamine to a nontoxic product.

Heat is at times of value and a heat cradle over the abdomen or a heat pad has at times been of value and should be tried.

Last but not least, drugs are to be considered. The use of drugs has not measured up to our expectations except in a few instances. Pituitary preparations, while they occasionally work, have been on the whole disappointing.

The one drug which has been of more benefit than any other has been prostigmine. Harger and Wilkey[12] have written conclusively on this and cite numerous methods of administration. This drug may be used to prevent ileus. A dose (1 cc. of 1:4000 solution of prostigmine) is given four to six hours after operation and repeated every six to eight hours until peristalsis is going nicely; then an enema is given. In cases where ileus has developed the 1-cc. dose may be repeated every two to three hours until peristalsis starts. We then like to continue this drug for about three more doses after peristalsis starts, and give it at eight-hour intervals. This drug has given us by far the most satisfactory results from the standpoint of prevention and treatment of adynamic ileus.

I will just mention in passing the use of spinal anesthesia as a treatment for adynamic ileus. This has not been satisfactory in our hands. It works occasionally.

Enterostomy in the form of jejunostomy we never use as this will drain only a single segment of bowel, and we have abandoned it.

Some of the severe ileus cases still die in spite of all the above possible treatments, and therefore we have not reached perfection in the treatment of this distressing complication. There is more work to be done.

One must also remember that the treatment given here must be varied and modified for the individual case.

Only when we realize we are treating a particular condition in a particular individual can we do our best.

BIBLIOGRAPHY

1. Stone, H. B.: The Toxic Agents Developed in the Course of Acute Intestinal Obstruction and Their Action, Surg., Gynec. and Obst., **32:** 415–419, 1921.

2. Whipple, G. H., Rodenbaugh, F. H., and Kilgore, A. R.: Intestinal Obstruction; Proteose Intoxication, Jour. Exper. Med., **25:** 479–494, 1917.

3. Haden, R. L., and Orr, T. G.: Experimental High Intestinal Obstruction in the Monkey, Jour. Exper. Med., **41:** 107–111, 1925.

4. Gatch, W. D., Trusler, H. M., and Ayers, K. D.: Acute Intestinal Obstruction; Mechanism and Significance of Hypochloremia and Other Blood Chemical Changes, Amer. Jour. Med. Sc., **173:** 649–667, 1927.

5. Gamble, J. L., Blackfan, K. D., and Hamilton, B.: A Study of the Diuretic Action of Acid-Producing Salts, Jour. Clin. Invest., **1:** 531–545, 1924–1925.

6. Barden, R. P., Thompson, W. D., Ravdin, I. S., and Frank, I. L.: The Influence of the Serum Protein on the Motility of the Small Intestine, Surg., Gynec. and Obst., **66:** 819–821, 1938.

7. van Beuren, F. T., Jr., and Smith, B. C.: Acute Ileus, Annals of Surgery, **107:** 321–339, 1938.

8. Wangensteen, O. H., and Paine, J. R.: Treatment of Acute Intestinal Obstruction by Suction with the Duodenal Tube, Jour. Amer. Med. Assoc., **101:** 1532, 1933.

9. Welch, Claude E.: A Method of Constant Suction Applied to the Levine Tube, Surgery, **3:** 693–695, 1938.

10. Abbott, W. Osler, and Johnson, Charles G.: Intubation Studies of the Human Small Intestine. A Non-Surgical Method of Treating, Localizing and Diagnosing the Nature of Obstructive Lesions, Surg., Gynec. and Obst., **66:** 691–697, 1938.

11. Fine, J., Banks, B. M., and Hermanson, L.: The Treatment of Gaseous Distention of the Intestine by the Inhalation of Ninety-five Percent Oxygen, Annals of Surgery, **103:** 375–387, 1936.

12. Harger, J. R., Wilkey, J. L.: Management of Postoperative Distention and Ileus, Jour. Amer. Med. Assoc., **110:** 1165, 1938.