ILEUS FOLLOWING GYNECOLOGICAL LAPAROTOMIES.

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THE significance of inhibited peristalsis following laparotomy frequently is overlooked in postoperative treatment. Unless evident paresis or obstruction exists, postoperative meteorism is not, as a rule, a cause for alarm. Most surgeons agree that after every laparotomy there is always some paralysis of peristalsis; it is expected and, therefore, attracts little attention aside from administration, at times, of an opiate to relieve suffering. Bier emphatically states that postoperative ileus in some degree occurs during the first forty-eight hours after all laparotomies as a combined result of drying-out, cooling, mechanical or chemical irritation, or slight infection; and according to Krönig, it is never entirely absent. In regard to these conditions, there seems to be on the part of operators a tendency to assume the existence of an element of fatalism and they do not act until serious symptoms develop, as a result of which their patients suffer torture for from twenty-four to forty-eight hours. Finkelstein says that postoperative intestinal paralyses which follow major laparotomies are more frequent after gynecological operations, probably because of pelvic adhesions, the longer time required to operate, and the greater exposure to air.

The writer believes that intestinal distention coming on after operation should be regarded as a serious complication and treated as such from the beginning; that it should be classed with shock, hemorrhage, etc., as a condition demanding careful therapeusis. In his opinion, there is no doubt that many cases of severe ileus might have been prevented had the first signs of peristaltic interference been heeded and appropriate measures adopted to combat it.

"This ordinary immediate ileus," says Bier, "is, as a rule, harmless and passes away on the third day, but may persist and cause death." It is persistence that renders the condition dangerous. Although it usually is harmless, it is always the cause of much suffering and agony which must exert an unfavorable influence upon the nervous system of the patient and retard convalescence.

In a monograph upon his theory of anoci association, Crile says that postoperative distention can be explained as a biological adaptation to overcome infection; that in the course of evolution all abdominal penetrations are infective, but the peritoneum is able to overcome most infections if they can be localized. Nature, having no confidence in the surgeon, distends the coils of intestine against the abdominal walls, thereby shutting off any attempt of the infection to spread. This tenet, the writer believes, is unsubstantiated. In fact, farther on in the same article, Crile asserts that his nerve-blocking method does away with the abdominal distention and attendant pain and thus is a very great surgical improvement. He says: "The postoperative rise of temperature, the acceleration of the pulse, the pain, the nausea and distention are minimized or wholly prevented."

Postoperative ileus should be recognized as a disease entity, from the beginning, as G. Schubert points out. If the serious phenomena are to be recognized and guarded against, the milder phases cannot be overlooked or neglected.

Generally speaking, postoperative ileus is that which occurs within ten or fourteen days after operation. When it occurs later it is always due, as Elliott says, to obstruction and is so distinct in its manifestations and serious in its aspect that it readily is recognized. Early ileus frequently is so insidious in onset, so prone to combine one or more types that most writers in dealing with this condition draw the line rather sharply between it and the late form.

There are two main factors that enter into the etiology of ileus, which may be called the indirect and the direct causes. By indirect is meant the idiosyncrasies of the patient, her nervous make-up and her habits. It is a fact attested by de Francisco, Zadradincky,

Tansini, and others, that certain women seem to have an idiosyncrasy for the formation of adhesion and for ileus. Tansini describes a case in which he performed a nephropexy twice at different times and a paralytic ileus of an alarming degree appeared both times. Most operators have had the opportunity to observe the facility with which in some patients adhesions form after simple aseptic operations. The writer recalls a patient upon whom he operated four times for adhesions, each time in different parts of the abdomen, the primary operation being undertaken for the removal of an interval appendix. It is believed generally by medical men that the presence of a chronic lesion, such as tuberculosis, acting as a cause of faulty metabolism, predisposes to atypical peristalsis and renders the abdominal serosa susceptible to very slight trauma. Hysterical patients sometimes develop a spastic type of ileus which may cause grave concern to the surgeon. In these cases, however, there usually is no change in the pulse rate or cardiac activity, a dose or two of bromide relieving the condition.

Constipation, to which the majority of women seeking surgical treatment are subject, is a predisposing cause of ileus. The bowel torpidity may be due to the patient's failure to respond to the call of nature or to adhesive bands, kinks, membranes, and the like. A bowel, the musculature of which thus has become weakened and sluggish, very quickly responds to peristaltic inhibition.

The direct causes are such as arise from operative procedures. Nature resents any violation of the abdominal cavity. No matter how slight the unavoidable or avoidable trauma, the sensitive peritoneum reacts to the irritation when the abdomen is open. The usual postoperative reaction is caused by cooling and drying of exposed intestinal serosa by the air, by the more or less handling of the viscera, by the use of pads, sponges, etc., by slight infections from faulty technic, and by adherence of intestinal walls to abraded surfaces, ligature knots, or stumps. The rôle of narcosis in the production of ileus is a subject of much discussion, many writers giving this as one of the causes, others claiming that its effect has been greatly exaggerated. While clinical evidence seems to support the affirmative view, the manner in which narcosis acts in the causation of intestinal paralysis is not very clearly understood. In an exhaustive article on acute dilation of the stomach, G. A. Friedman claims that narcosis produces splanchnic paralysis by action upon the nucleus in the floor of the fourth ventricle. More advanced views, however, point to interference by narcosis with the interglandular action of the chromaffin system, especially that of

the adrenals, which seem to exert direct action upon the abdominal sympathetic nerves. As soon as the anesthetic is withdrawn interference ceases, which tends to prove that the more prolonged the narcosis the greater the adverse action of the controlling influences.

Hastie and Monat have pointed out that asphyxia causes intestinal anemia, lessening intestinal activity. With this view Nothnagel agrees and explains the phenomena by saying that the dyspneic blood increases the peristaltic action, thus tiring the intestinal musculature and favoring ileus. Crile says that ether immediately impairs the immunity of the patient by interfering with cellular oxidation.

The writer made a careful study of forty operative cases with the view to determine, if possible, what effect, if any, varying length of time of narcosis had on the presence of postoperative distention. The operations varied in time for from fifteen to ninety minutes, all possible care being taken to avoid intraabdominal trauma. The result of this study conclusively showed that the shorter the time of narcosis the less the distention, the comparative cases being carefully selected.

Whether the nerve shock, as described by Crile, has an inhibiting effect on peristalsis, is not universally acknowledged. In a series of twenty cases in which the anoci association method was used in conjunction with gas-oxygen and gas-oxygen-ether narcosis, little appreciable diminution in distention was noticed, but there was far less pain. Of course, this lack of result may have been due to faulty technic.

The usual or harmless form of ileus may be the forerunner of the more serious type. The latter may be ushered in very gradually, beginning immediately after operation. For this reason alone, it is wise to take precautions in the earliest stages, for the same reason that tonsillitis should be differentiated from diphtheria.

Severe ileus is not very uncommon. It occurs in about 1 per cent. of all laparotomies. Some writers give the percentage as high as 6 per cent. Thiemann reports 3 per cent. Klotz saw thirty-one cases in 569 gynecological operations. Döderlein and Krönig, in 2000 cases at the Tübingen Clinic, saw twenty-four cases. The writer, in 340 laparotomies, saw five. These percentages represent a very grave source of danger following laparotomies.

Severe ileus has been classified as dynamic or paralytic and mechanical, according as the cause is splanchnic paralysis or obstruction to emptying of the bowel. Vaccari claims that all cases

of ileus are due to either peritonitis or obstruction. He does not recognize the spastic type. The two types, as a rule, have distinct symptoms, but sometimes resemble each other so closely that differentiation is impossible. From the academic standpoint, the fine distinction of type is enlightening, but therapeutically it is apt to obscure the vital issue and lead to dangerous procrastination.

Paralytic or dynamic ileus results from sympathetic paralysis, induced by peritonitis, mechanical irritation of intestinal musculature or peritoneum, such as is caused by rough handling, traumatism, or chemicals, by improperly prepared or applied pads, by too much dry sponging and by eventration. Stumpf and Freund emphasize eventration as a cause of intestinal paresis. Circulatory engorgement caused by wounding a blood-vessel in the intestinal wall, and injury to the mesenteric vessels causing thrombosis, infarcts, etc., which may be caused by severe traction on the mesentery by rough handling, placing of pads and the long-continued Trendelenburg position are contributing causes. Kuskat, Craig, and Trendelenburg himself assert that the last mentioned may induce ileus.

Spastic ileus, which is dynamic in character, usually occurs in hysterical women and in cases of lead poisoning. It rarely occurs postoperatively. Some writers ignore this type completely in dealing with postoperative ileus. Döderlein states that spastic ileus is denied on good authority, but its occasional occurrence is still authenticated. It is known to occur in the predisposed and under certain irritations, such as gall-stones.

Sandos and Sterling, in their physiology, say: that all stimuli may even produce spasmodic contracture of the musculature of the intestine.

Baldy reports two cases after hysterectomy, the autopsies showing no peritonitis, but intestines contracted in one or two places. Experiments have shown that stimulation of the splanchnic plexuses of Auerbach and Meissner causes increased peristalsis, while overstimulation causes spasmodic contracture. Spastic ileus attacks, by preference, the colon, but may affect the ileum. Bunge, at the German Surgical Congress, 1908, reported two cases of spastic ileus. The first case occurred after an operation for appendicitis. Relaparotomy showed the entire colon in spastic contraction. An ileostomy was done and the patient recovered. In the second case, the contraction extended up the ileum for 30 or 40 centimeters. Spastic ileus is very hard to differentiate, but presents characteristic symptoms of intestinal paresis. The mechanical type of severe ileus is due to adhesions of gut to gut, of gut to the abraded surfaces,

such as a stump, a denuded area, such as is left after separating pus tubes, etc., ligature sites, or to a band of adhesions across the lumen. A loop of intestines caught in a rent of the omentum or mesentery, or between two constricting bands, if not released, is apt to produce the characteristic ileus.

Drainage devices may produce reflex disturbances of peristalsis, which promptly subside after removal of the drain. Other foreign bodies, such as gauze, tampons, etc., left in the cavity, may produce paralysis.

Arteriomesenteric ileus is one of the most dreaded postoperative complications. It has been called Riedel's syndrome or acute dilation of the stomach. As a rule, this is not described under postoperative ileus, it being a disease by itself. The etiology is obscure, although it is generally supposed that it is caused by closure of the duodenum by the constricting effect of the superior mesenteric artery and the mesentery between which the duodenum is caught. Some writers claim that the constricting of the duodenum is the result and not the cause of the dilation. While this accident is very serious, it lends itself usually to speedy relief, if recognition and treatment are early.

The etiological factors of severe ileus may be epitomized as follows:

1. Mechanical irritation of the peritoneum and intestine during operation.

2. Infections of the peritoneum.

3. Adhesions of intestinal loop to abraded surfaces or to adhesion strands.

4. Closure of mesenteric vessels.

The diagnosis of ileus is made by the presence of abdominal distention, local or general, by the failure of the bowel to expel gas or feces, and by vomiting. Pain, while always present in the first stages of intestinal occlusion, may be entirely absent in the severest forms, especially in the paralytic forms. In fact, if pain, usually present immediately after operation, disappears and the distention persists, it is good evidence of more or less severe paralysis. Even if the bowels have expelled gas or feces, or both, and distention persists, there is danger of grave sequelæ.

Every postoperative distention should be viewed with suspicion. Its course should be watched, hour by hour. It is only by so doing that the severity of the condition can be determined. In general, it may be said that the paralytic form, which includes the irritative, spastic, and septic types, appears in the first three days after operation and the mechanical form, after gas and feces have escaped.

Sometimes, however, both forms appear together, which makes differential diagnosis difficult.

The paralytic form is manifested by gradual increase in the meteorism and failure of the bowels to expel gas or feces. Vomiting soon supervenes and increases in frequency; there is an increase in the pulse rate, which soon loses its normal character and becomes weaker and thready, depending on the degree of toxemia. Döderlein and Krönig say that there may not be any rise in temperature, as the toxins may not affect the heat center. On the other hand, they do affect the vasomotor center, which they paralyze and cause the rapid thready pulse. As a rule, ileus is accompanied with some rise of temperature.

The typical symptom-complex would be as follows: After shock has disappeared and the patient feels better, with a fair pulse, the temperature and also the pulse rate rise, and the patient begins again to feel ill; the abdomen is tympanitic, the stomach feels distended, and gas does not pass from the bowel. Vomiting soon begins, and consists, at first, of stomach contents, then bile-tinged fluids, and, if the condition is not relieved, fecal-stained material. The condition becomes worse, especially as regards cardiac activity, due not only to toxemia but to pressing upward of the diaphragm by the distended bowels.

Obstructive ileus may occur as early as the third day, though, as a rule, it does not develop until the sixth to the tenth day. In the early stages, there is exaggerated peristalsis—which frequently can be seen if the abdominal wall is thin—resulting from an attempt of nature to force by the obstruction. Auscultation and also the increased borborygmi reveal this. A tumor at the site of the obstruction may be felt and there is generally tenderness over the wound. This last is particularly true of colonic obstruction, although the reverse is true with an obturation of the ileum. The clinical picture of cecal obstruction is identical with that low down in the ileum. Colonic obstruction sets in slowly and does not always cause vomiting at first. Meteorism may not develop for five or six days. Usually there is little circulatory or febrile reaction. On the other hand, obstruction of the small intestine in the upper ileum or jejunum shows early a decided effect on the pulse rate, with or without much fever, and vomiting is an early feature. There is not so much meteorism, as there is not so much intestine involved.

In obstruction of the lower ileum, the symptoms vary as the obstruction approaches the colon or jejunum.

The writer believes that a severe ileus should always be suspected

if, after recovery from the effects of the anesthetic, vomiting continues or again begins, accompanied by abdominal distention. There may be no change in the pulse rate, as that may have been increased by the effects of the operation or the general lowered vitality. Many writers emphasize the importance of differentiating the paralytic from the obstructive form, and it is not rare to see a case of undetermined character develop beyond the point where life may be saved.

The pathological conception of ileus, according to text-books and many writers, follows the natural road of clinical findings without the benefit of laboratory research or animal experimentation. In a great measure, autopsy findings have corroborated the clinical evidence. It is with this conception that many authors strongly assert the importance of making the differential diagnosis of the two forms, some writers declaring that surgical interference in paralytic ileus is unwarranted.

The classical pathological picture of obstructive ileus starts with occlusion of the lumen of the viscus, which is followed by increased peristalsis. Meanwhile, the contents of the bowel accumulate and, being prevented from escaping, increasingly distend the lumen until the obstruction is relieved or death ensues. This distention produces a thinning of the bowel wall with emigration of bacteria into the peritoneal cavity, causing a peritonitis from which toxins are absorbed. Sometimes ulcers form in the mucosa and break through into the abdominal cavity. In the distended loops, fecal material decomposes and toxic material is elaborated which, being absorbed, causes dangerous toxemia.

In the paralytic form, the putrefying feces or the presence of a septic peritonitis is believed to be the cause of death.

Krönig says that after fecal stasis, fermentative products of a toxic character are formed and absorbed, and that after prolonged operations or in weakened organisms these toxins are sufficient to cause death. All through the literature, absorption from septic peritonitis and fecal decomposition are advanced as the chief cause of death. G. Schubert says that it is doubtful if paralytic ileus itself ever causes death, that the death is due to complicating sepsis.

While agreeing with the pathological conditions described above, the writer believes that the most potent factor in the production of toxemia is not the septic process or the emanation from putrefying feces, but an autogenous toxemia resulting from toxins far more poisonous than the bacterial or fermentative toxins, elaborated from the blood by the cells of the mucosa of the upper segments of the

small intestine. In every serious or fatal case, the upper bowel is always involved, and it is here, experiments have shown, that a very toxic material is elaborated if distal drainage is occluded. This toxin secretion is made possible by dehydration from vomiting and, perhaps, the lack of internal secretion control.

Whipple, Stone, and Bernheim (*Jour. Exp. Med.*, xix, 144, 1914) in an article on intestinal obstruction, speaking of the defensive mechanism against duodenal loop poisoning, say: "Intoxication is evident in a drained duodenal loop, whether it opens externally or into the jejunum and may be associated with more or less immunity which can be demonstrated after a period of days. Intoxication with a closed loop is identical whether the loop is left empty at operation or filled with a lethal dose of loop fluid. This emphasizes the fact that absorption of the poison is essentially from the mucous membrane rather than from the contents of the closed loop. Cessation of the normal flow of intestinal fluids which bathe the mucous membrane may be essentially responsible for the perverted activity of the mucosa and the secretion of a poisonous material."

Hartwell, Hoguet, and Beekman (*Arch. of Int. Med.*, xiii, 701, 1914) say that if the upper segment (of the bowel) has become damaged, poisonous absorption is threatened, and a drainage enterostomy should at once be performed, in advance of the relief of the ileus.

Clairmont and Rainizi, quoted by Finney, injected filtrates from normal unobstructed intestines into guinea-pigs, and perceived no toxic results. They then injected pathological fluids from obstructed intestines and found they were profoundly toxic and lethal. The fluids from the colon were not as poisonous as those from the small intestine. Hartwell, in some experiments on dogs with bowels artificially obstructed, found that by subcutaneous injection of saline these dogs were kept alive and well for twenty-four days. When they were killed, autopsy showed no pathological lesion except dilation above the ligature. Draper (Maury) found, experimentally, that by feeding normal mucosa cells from the intestines of a well dog to dogs with obstructed bowels he was able to keep them alive and well.

Moraselis, Ruis, and Natale, quoted by Draper, showed conclusively by injection into rabbits that intestinal contents from an obstructed segment of intestine become less toxic directly the obstruction neared the rectum. Duodenal contents were most toxic, and the sigmoid contents least so. This is attributed to the symbiotic action of the bacteria in the lower bowel.

Cases of fecal vomiting frequently are seen which, as soon as the

obstruction is removed and drainage established, promptly recover (F. Wood). This is well illustrated by the following case:

Mrs. P., aged forty-eight, was operated on by the writer at the Woman's Hospital, October 3, 1914, for pelvic adhesions and partially obstructed sigmoid, as shown by the röentgenograph. She made a good recovery from the anesthetic, her temperature at 8.00 P. M., being 99.3°, pulse 82, respiration 22. During the night she was somewhat restless. There was only slight abdominal distention. Morphine, grain ¼ by hypo. was given and she slept well until 2.30 A. M., when she awoke and complained of pain in the abdomen. The rest of the night she slept at short intervals but was more or less restless. On the morning of the 4th, she felt fairly well, but complained of nausea. She was given water by the mouth, which did not increase the nausea nor cause vomiting. Her temperature at 8.00 P. M. was 100°, pulse 92, respiration 22. On the 5th, the temperature dropped to normal, pulse 88, respiration 22. There was quite marked meteorism, accompanied with more or less discomfort. The urine was negative At one o'clock of the 6th, the pain was worse, a little watery fluid was vomited, after which some relief was felt. An enema given was expelled without gas. The rectal tube was inserted for twenty minutes, with relief. At 6.00 A. M., enemata of oil and turpentine followed by soapsuds were given, which she expelled immediately. At 8.30, vomiting began and continued about every half hour. Hot water was given by mouth and retained. The abdominal distention was apparently increasing. The emesis continued; a large, hot flaxseed poultice was placed over the abdomen. The pulse at 8.00 A. M. was 120; at 12.00, it was 130. The temperature showed only a slight rise to 101°. Vomiting of greenish fluid continued about every fifteen minutes. An enema of milk and molasses, of each a half pint, was given without relief. The rectal tube brought away no gas. At midnight, the pulse was 145, temperature 101°, respiration 28. A diagnosis of ileus was made and the patient was taken to the operating room and given a quarter of a grain of morphine and a hundredth of hyoscin. The abdomen was opened through the operative wound and a diffuse peritonitis was found extending all through the pelvis and the lower part of the abdomen. The patient's condition was not good, so a loop of upper ileum or jejunum was brought up and sutured into the wound, then opened and a No. 30 rubber tube inserted into the proximal segment. Two gauze drains were inserted down into the pelvis. The time of operation was fifteen minutes. At 8.00 A. M., the morning of the 7th, the patient's temperature was 98.3°, pulse 130, respiration 24. Vomiting and pain had ceased. At 8.00 P. M., same day, the temperature was 98.2°, pulse 100, respiration 24. Recovery was uninterrupted, the enterostomy wound being closed later.

While the enterostomy wound was open the patient rapidly lost flesh and strength through starvation, though rectal and tube feeding, carried out carefully at two-hour intervals and in small

quantities, arrested this somewhat. After the fistula was closed the lost strength and flesh were rapidly regained.

W. W. Grant (*Surg., Gyn. & Obst.*, April, 1915) describes two cases in which the same procedure was followed, namely, enterostomy, with immediate relief but a fatal outcome. He operated on the third and fifth day after obstruction, before which time lethal doses of toxin may have been absorbed from the upper intestine.

Gurb, of Montreal, believes that since the mucosa of the upper bowel contains a greater content of toxic principle than do the contents of the lumen, it would seem that the poison is produced by autolysis rather than by bacteria. Roger believes in the autogenous origin of obstructive toxemias, and says: "Emancipation from the text-book dogmas of nervous influences, shock, stagnation of bowel contents, passage of bacteria and bacterial products through the stretched wall as the cause of toxemia" is very necessary. He says these should be set aside in favor of modern conceptions of glandular interaction, interference with which probably is the fundamental cause of obstructive death.

Draper (Maury), in another article, says that dogs will live for weeks with iliac obstruction, but die in a few hours with duodenal obstruction, and the same dog will live for weeks if there is the slightest drainage of the duodenum. In ileocolic obstruction, the danger lies in the sequence of peritonitis, which produces not only its own toxins but, by paralyzing the whole gut, induces the vicious secretion of the upper coils.

Granting that any postoperative distention is pathological and as such should be considered worthy of every attention, the prophylaxis assumes great importance. The preoperative preparation should consist of careful regulation of the bowels, without purges, etc., some days before operation. Purging, or even milder catharsis, just before operation is to be condemned. An enema or two the night before is sufficient. Some operators advise against even an enema twenty-four hours before operation. The writer's method of giving a laxative two or three days before operation and an enema the night before has acted very satisfactorily. In emergency cases, no enemata are given. The nervous or hysterical patient should, as far as possible, be guarded against the worry of distressing anticipation by the judicious use of bromides for a few days. A little morphine or morphine and hyoscine, given just before she is taken to the operating room, not only frees her from the horror of the last few moments before operation, but minimizes the amount of anesthetic needed. Crile lays great stress on the effect of emo-

tional stimuli on the brain. The less anesthetic employed, the less the liability to narcosis depression. There is no doubt that the shorter the time of operation, the fewer bad effects will follow. This applies not only to the narcosis and the natural surgical shock, but to the length of time the abdominal contents are exposed to the air. Many writers emphasize the necessity of avoidance, as far as possible, of contact by the intestines and peritoneum with the air and chemical substances. Overlarge incisions always invite splanchnic irritation. The smaller the incision, the less the exposure. Many operators scoff at the small incision, but the writer believes that operative technic will never approach the ideal until the smallest possible incision through which the work readily and safely can be done, is universally adopted. The whole abdominal cavity can be explored through a three- or four-inch incision. The writer saw one of the most noted and publicly acclaimed surgeons make an incision for the removal of the uterus and two ovarian cysts, the larger of which was about the size of a grapefruit, which extended from ½ inch below the ensiform cartilage to the pubes. When asked why such a large incision was made, he replied he wanted plenty of room. The patient died of shock. The same surgeon, operating in the upper abdomen, where he was most adept, never made an incision over 3 or 4 inches long.

Except for cauterizing, chemicals are never used in contact with the peritoneum, but the prevalent use of iodine in the preparation of the abdomen has brought the danger of intestinal irritation when the gut comes in contact with the skin. Rehn, quoted by Bier, first drew attention to the irritative effect of iodine on serous surfaces. It is, therefore, important to cover the skin by pads or towels held in place by clamps or pins.

Rough handling of the bowel is to be avoided at all times. There is no condition which demands maltreatment of the intestines. This applies especially to the preparation and use of abdominal pads. Dry pads should never be used; thin pads are bad because the water evaporates quickly and they are liable to chill the gut. Thick, warm pads, moistened in saline, are best. Pads roughly shoved in through the incision to keep back the intestines, not only irritate the sensitive serosa, but may produce injury to the mesentery, resulting in venous or arterial occlusion.

One of the most difficult problems before the surgeon to-day is to prevent adhesions. Thiemann says: "We know of one resource only for preventing adhesions, to wit, keeping up the peristalsis from the earliest moment." He also says that if the various substances which

have been applied to the peritoneum, prevent adhesions, it is very likely because they stimulate peristalsis. He goes so far as to advocate opening the abdomen after all postoperative inflammatory disturbances have subsided and dividing all bands.

The covering of all raw surfaces, stumps, ligature-knots, and suture lines, were it possible, would go far to prevent the formation of adhesions. But this is not always possible, so the problem really resolves itself into the necessity of keeping up peristalsis from the very first.

The use of olive or cotton-seed oil, liquid vaseline, animal oils, and the like, have proved rather unsatisfactory in seventy-five of the writer's cases. Some operators use a Cargile membrane with good results, but it is very hard to apply and many surfaces do not lend themselves to complete covering. Transplanting of omentum to cover raw places, as recommended by A. T. Mann, of Minneapolis, and others, has proved of value in some hands.

The stimulation of peristalsis immediately after operation has received a great deal of attention for a number of years. Of the drugs most highly acclaimed, atropine and physostigmine lead. Vogel, Bier, Zadradnicky, and others, praise physostigmine above all drugs. While there is no doubt that this drug and atropine do act well in some cases, their action is uncertain and bad after-effects have been reported.

Simoncelli and others have used the preparation called "Peristaltic Hormonal," introduced by Zuelzer, with marked effect. This preparation, however, has a pronounced effect in lowering the blood pressure and must be used with great care. Deaths after its use have been reported.

The direct injection of castor oil and Epsom salts into the bowel has few advocates now.

The writer has been using a water-soluble glucoside from cascara sagrada, recommended by K. Finkelstein (*Deutsch. Med. Wochenschr.*, December 17, 1914) in twelve cases. These cases included three pus tubes with exudates, four supravaginal hysterectomies, one case of adeno-carcinoma of the uterine body, two cases of retroflexion of the uterus, and two of appendicitis. With the exception of one case, all had more or less adhesions. The result of the treatment has been very startling. None of the cases had marked distention. Two of the pus tube cases had some distention, but passed gas naturally at the end of twenty-four hours. The drug was administered hypodermatically before the patient left the operating table, the dose being 1 ampule of 0.5 gm. This dose was repeated

every six hours, until gas was passed. Of course, twelve cases are insufficient from which to draw any definite conclusions, but the decided results obtained warrant continued trial.

The treatment of ileus after it has once set in consists of, first, an attempt to excite peristalsis strong enough to produce an evacuation by medical means; and then, if failure ensues, to relaparotomize for drainage and, if possible, the relief of the obstruction.

The writer believes that measures to relieve distention should be instituted at the end of twenty-four hours if gas has not passed the anus. The usual custom of allowing a distended abdomen to persist for two to four days is very hazardous. If there is no meteorism, it does no harm to wait two or three days before moving the bowels. Cathartics and the like are not demanded, but enemata consisting of glycerine, soapsuds preceded by oil, and turpentine, should be tried first. Other mixtures, such as milk and molasses, equal parts, have been used successfully. Oxygen gas, introduced per rectum, has, in the writer's hands, proved very valuable, acting by stimulation of the nerve endings and also by the mechanical effect of straightening agglutinated coils. The abdominal application of hot poultices and Gelinsky's hot-air baths, have proved of great aid, especially if peritonitis be present. The writer has used the cluster of electric light bulbs with very satisfactory results in certain cases. Pettenkofer uses galvanic electrical enemata, as recommended by French authors. As slowed peristalsis is favored by the recumbent position, it is important to insist on the frequent change in position of the patient, by turning her from side to side, or tipping the bed. The knee-chest position has been used to relieve a kink or a twist in the intestines. These body movements also tend to prevent adhesions. Gentle massage to the abdomen has been recommended, but is of doubtful value on account of the discomfort to the patient.

If, in spite of all efforts, the ileus is not relieved, relaparotomy must be considered, some authors say as early as two days, others say, by the fifth day. Relaparotomy is a very grave procedure and becomes graver the longer the ileus has persisted. It is a question whether the seriousness of the reoperation is not brought about by the tendency of the surgeon to wait too long.

The writer believes that if during the first forty-eight hours the patient's bowels become distended, causing pain, no gas passes the anus and vomiting is present, with an increasing pulse rate, the diagnosis of ileus should be made at once and an attempt made to open the bowels. If ineffectual after twelve to twenty-four hours, relaparotomy should be done. Increasing vomiting and pulse rate

are signals that should be obeyed at once. If the ileus is relieved, before the so-called fecal vomiting is noticed, the chances of recovery are very excellent. After fecal vomiting has begun, toxic absorption also is going on, and the prognosis grows graver as the time slips by.

After the abdomen has been opened, the type of ileus reveals itself. If it is septic, an ileostomy or low jejunostomy should be done at the same time the septic condition is treated. The sooner out of such an abdomen, the better the chances of the patient. Simple puncture of the bowel does no good, for the gas is not the offending agent. Spastic ileus is always treated by ileostomy. If the ileus is obstructive and the patient's condition permits the time, a search for the obstruction should be made and the obturation relieved. As a rule, these cases are in a pretty low state of vitality, not only from the effects of the ileus, but from the primary operation and it is a grave question whether it is good surgery to expose the patient to the shock of handling the intestines in searching for the lesion, or of a resection, however cleverly done. Far better is it to drain off the toxic products and wait until later for the radical work.

In the light of the autogenetic hypothesis, the efficacy of an early ileostomy must be recognized. It is an axiom of surgery that the earlier a lesion is attacked the more favorable the prognosis. Ileus is the germinal bud which, if allowed to grow and blossom bears a very deadly fruit. It is the bud of the early manifestation of intestinal paresis that must be attacked, not necessarily by operation, to ensure the patient's safety. If operation is the only resource, is should be aimed at the root and done in the quickest possible time.

In conclusion, the writer would summarize as follows:

1. That the postoperative distention which is nearly always present is likely to be a signal of danger and not a condition to be ignored.

2. That differential diagnosis is not sufficiently important to demand even one hour of delay in therapeusis.

3. That the greatest source of danger in ileus is the vicious secretion of the upper small intestine caused by the failure of glandular interaction and dehydration by vomiting.

4. That a strict prophylaxis includes preoperative care as well as most careful attention to perfect technic at operation.

5. That early treatment at the end of twenty-four hours is indicated and early operation if nonoperative measures fail.

6. That the upper bowel should always be drained and not merely punctured, the radical cure being postponed unless the patient's condition allows an extended operation.

40 EAST FORTY-FIRST STREET.

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