POST-OPERATIVE INTESTINAL OBSTRUCTION.

By Eugene Boise, M.D.,
Grand Rapids, Mich.

I shall, in this discussion, exclude all forms of intestinal obstruction that are not direct results of operation, and shall consider post-operative obstruction under two main forms—functional and organic. Under the term functional I shall include two opposite conditions: Tonic spasmodic contraction of the muscular coat of the intestine and that condition properly designated paralysis. Under the term organic I shall discuss only that form of obstruction caused by adhesions, omitting those that are accidental, as volvulus, incarceration, etc.

In the majority of cases, intestinal obstruction occurring after abdominal operations is due to adhesions, either existing prior to operation and remaining unrelieved, or those which form as the direct result of the conditions caused by the operation. The clear indication in pre-existing constricting adhesions is to separate them at the time of operation. The equally clear indication in the second group is to leave the abdominal and pelvic cavities in such condition as to reduce to the minimum the liability to post-operative adhesions. How to secure this end has been, still is, and will continue to be the object of discussion.

We have, then, post-operative intestinal obstruction occurring:

1. As tonic muscular spasm.
2. As true intestinal paralysis.

3. From the formation of new adhesions.

4. As a result of pre-existing constriction not discovered or relieved at the time of operation.

That we have post-operative obstruction as the result of tonic intestinal spasm is, I believe, not generally recognized, but this condition may, and often does, occur as the result of violent stimulation of the muscles of the intestine. Landois and Stirling[1] say: "All stimuli applied to the plexus mesentericus increase peristalsis . . . which may even produce spasmodic contraction of the musculature of the intestine." Murphy relates a case[2] of lead-poisoning with absolute obstruction, which had continued five days and in which operation revealed tetanic spasm of the muscular coat of the small intestine for a space of eight inches, so severe as to cause entire obliteration of the canal. This spasm, however caused, may persist till death. Ashton[3] relates two cases where this condition was revealed at the autopsy, one case occurring in his own practice, where death occurred on the fifth day. Autopsy revealed a tonic contraction of the small intestine for a space of about eight inches above the ileo-cæcal valve, the canal being rendered impermeable. There were no adhesions and no signs of peritonitis. His second case was reported by Dr. Baldy following hysterectomy. The abdomen was reopened on the sixth day, but death resulted twenty-four hours later. Autopsy revealed no peritonitis, no adhesions, but "the intestine was contracted in one or two places so as to feel like hard cords."

Intestinal peristalsis is directly under the control of the sympathetic system of nerves, and is influenced in two ways: 1. By stimuli applied to the motor nerves at their origin, during their course, or at the terminal ganglia in the muscles themselves. 2. By agencies which influence the vascular supply. Stimulation of the motor or vasomotor nerves

[1] Manual of Physiology, p. 318.
[2] Journal of American Medical Association, January 4, 11, 1896.
[3] Journal of American Medical Association, July, 1892.

causes increased peristalsis; overstimulation causes spasmodic contraction, which may become tetanic.

As shown in the case cited by Murphy, lead-poisoning may cause tetanic contraction of the intestinal muscles, because of the direct irritant action of the poison on the nerves controlling these muscles. Other poisons have a similar action. Excessive electrical stimulation will produce the same condition; and the cases reported by Dr. Ashton show that there are conditions resulting from operation that will cause the same result. In the consideration of the question as to what these conditions are we must bear in mind the nervous mechanism of the intestinal muscles; that the nerves belong to the sympathetic system and arise from the various abdominal plexuses; that these nerves terminate in a fine network of ganglionic cells, known as Auerbach's plexus, situated in the muscular tissue itself; and, finally, that an irritant applied to the mucous lining of the canal, to its serous covering, to the nerves in their continuity, or to their origin in the various plexuses, will cause muscular contraction, and that the severity of this contraction will be proportionate to the nature and severity of the irritant impulse. This, I may say, is the keynote of the pathology of intestinal obstruction from tonic spasm. This irritant impulse may be direct, it may be reflex, or it may act through the vasomotor nerves of the intestines.

Brunton and others, by accurate experiment, have determined the fact that excess of venous blood in the intestine excites peristalsis. Therefore, any impulse that causes contraction of the arterioles and consequent arterial anæmia causes increased peristalsis. It therefore follows that tonic spasm of the intestine may be brought about by excessive irritation of the motor nerves governing the intestinal muscles, and that this spasmodic impulse may be aggravated or exaggerated if, at the same time, the vasomotor nerves of the intestinal vessels are irritated.

There is always more or less violence to the motor mech-

anism of the intestinal canal during severe operations within the abdominal cavity. This violence necessarily acts as an irritant to the nerves and muscles of the intestines and causes exaggerated and irregular peristaltic action, which may be so severe as to cause intestinal obstruction by tonic spasm. This condition has been often mistaken for paralysis, but the term intestinal paralysis should be restricted to that condition of the intestinal muscle where its contraction is destroyed, the tube being dilated rather than contracted. It may occur as a result of "muscle-tire"[1] following the excessive muscular action described above, as a result of any factor that causes pathological infiltration of the intestinal wall or as the direct result of section of the nerves.

The first condition, muscular exhaustion, need not be dwelt upon, as it is properly an extension or sequel of excessive muscular action. The second condition may arise: (1) From peritonitis, sepsis, etc., and (2) from any cause that cuts off the arterial blood-supply, as thrombus, volvulus, etc. Maas[2] relates a case (not following operation) where the intestinal wall for a space of several feet was thickened and infiltrated with pus, causing fatal obstruction. The direct cause of the infiltration was not demonstrated, but in the absence of signs of peritonitis was thought to be due to thrombus of an artery.

Stumpf, of Munich, in a paper read before the sixth session of the Deutsche Gesellschaft für Gynäkologie, at Vienna,[3] mentions expressly as one of the factors of "Darmlahmung" eventration of the intestines. Freund, in the course of the discussion of this paper, laid special stress on eventration as a cause of intestinal paresis, but stated that, in his opinion, the cause of ileus was due, in the majority of cases, to general or circumscribed peritonitis. Some writer has stated that eventration causes a serous infiltration of the walls of

[1] Landois and Stirling, p. 318.
[2] Annals of Gynecology, February, 1895.
[3] Centralblatt für Gynäkologie, June, 1895.

the intestine, and consequent muscular paralysis. However that may be, it is probable that eventration, or long exposure of the intestines to the air, may cause true paresis in many cases, and that the effect of such exposure tends in all cases toward paresis rather than toward spasm. We are confirmed in this belief by the combined testimony of operators, by the case of Dr. Murphy, above cited, where a spasmodically contracted intestine, which had caused total obstruction for several days, rapidly relaxed when exposed to the air, and by the fact that intestines exposed to the air for any considerable length of time become distended with gas. Again, I can conceive that bruising of the intestinal wall, as by violent and injudicious effort to release adhesions, may cause direct muscular paralysis. However this may be, we still have as operative causes of functional intestinal obstruction severe irritation of the nerve-supply, causing persistent tonic spasm and local or general peritonitis, obstruction of arterial or nervous supply, and eventration, causing true paralysis.

Organic obstruction may be caused by pre-existing bands or by post-operative adhesions. Those adhesions that exist prior to operation are due to former peritonitis, and should be released at the time of operation. They, therefore, merit no further discussion in this connection.

But it is a very pertinent question, How shall we reduce to the minimum the liability to the formation of adhesions after operation? A tendency to adhesion is a peculiarity of serous membrane; and yet, as long as the integrity of its epithelium is preserved, it will remain in close contact with an opposite, intact serous surface with no tendency to adherence. It would, therefore, appear that the one important indication is to preserve the epithelium intact to as great an extent as possible. In making a diagnosis in cases of post-operative intestinal obstruction it is practically only necessary to differentiate between functional obstruction and organic, inasmuch as such decision indicates the line of

treatment. In making this diagnosis we shall be obliged to rely mainly upon the history of the case, inasmuch as the symptoms of obstruction vary very little, whatever the cause.

After nearly all abdominal operations, even of moderate severity, there is transient obstruction, the evidence of which is intra-abdominal pain, more or less spasmodic in its nature, and generally referred to a point at or near the seat of the greatest violence. This may be caused, in part, by the constricting ligature about the stump, but is largely caused by irregular, exaggerated (or even spasmodic) peristaltic action, an effort on the part of normal intestine to overcome obstruction at this point. This obstruction, in the absence of any history of prolonged exposure of the intestines or of partial pre-existing obstruction, may fairly be attributed to intestinal spasm, which, undisturbed, always tends to speedy relaxation, with re-establishment of normal peristalsis. If, however, this condition be aggravated by injudicious treatment, tetanic contraction may occur, which may persist till death, or it may give rise to actual, fatal intestinal paralysis, the result of muscular exhaustion. If there has been eventration of the intestines or prolonged exposure to the air, the condition may well be, from the first, paresis of that portion of the bowel so exposed, with exaggerated peristalsis of the healthy intestine. Or if the symptoms only show themselves with the onset of peritonitis the condition is one of true paralysis, due to involvement of the muscular coat in the inflammatory process. In intestinal paralysis we should expect more or less gaseous distention. In tonic spasm this would probably be absent till a condition of muscular exhaustion ensued.

Again, if several days have elapsed before the symptoms of obstruction show themselves, especially if for a longer or shorter period flatus has been passed, and if there is an absence of signs of peritonitis or sepsis, we may be confident that we have to do with organic obstruction due to post-operative adhesions. Like the occurrence of post-opera-

tive hemorrhage, the question as to obstruction is largely decided at the time of operation, and our discussion of the treatment must involve to a certain extent a discussion of techniqne. It is important, therefore, in making an abdominal operation, that we continually keep before our minds not only those conditions that predispose to hemorrhage and sepsis, but equally those that tend to impairment of function of the intestinal canal.

Inasmuch as irritation of the motor nerves at their origin, in their continuity, or at the terminal intermuscular plexus tends to promote excessive contraction of the muscles; and inasmuch as *severe* irritation may cause tetanic contraction, it follows that we should, during the operation, use all possible precaution that we may reduce to the minimum such irritation. This suggests the use of the smallest abdominal incision compatible with skilful work; the early and free use of the Trendelenburg position in cases where it is not absolutely contraindicated, and tender, skilful, delicate manipulation of the intestines themselves in the separation of adhesions. That we should avoid eventration of the intestines or their prolonged exposure to the air, goes without saying. But it is probable that in the majority of cases post-operative intestinal obstruction is due directly to adhesions, which form as a result of conditions caused by the operation. This has long been recognized, and many efforts have been made to devise some means by which it can be avoided. Martin has used sterilized olive oil; Morris has used and still uses aristol to cover the denuded surfaces. Other measures have been suggested, but have proved ineffective.

There are several general principles to be kept in mind: (1) That while intestines whose peritoneal epithelium is intact will adhere to raw surfaces, much more quickly and readily will this occur if their peritoneum is abraded. (2) While they will form adhesions to an opposite abraded peritoneal surface, much more readily will they adhere to a raw

surface; and (3) that two surfaces denuded of their peritoneum will almost certainly adhere.

What steps can we then take to reduce the formation of adhesions to a minimum? First, as I have remarked, avoid all unnecesary handling of the intestines. This is extremely important, and to this end the aid of posture must be invoked. Every foreign body that touches an intestine robs it correspondingly of its epithelium. It often becomes necessary to wall off the abdominal cavity or to hold the intestine away by the interposition of gauze sponges or towels. This is an evil that should be absolutely avoided when possible, but when they must be used let it be with the least possible violence and manipulation. Sänger has urged that while dry gauze will, without question, remove the epithelial covering of the peritoneum, gauze saturated with a warm soda-salt solution will do much less violence, and that therefore such solution should be used. Other operators have, after trial, denied this. I believe it to be advantageous. The use of gauze packing to check excessive oozing predisposes to the formation of adhesions. It has been suggested that it might be avoided by the use of steam, as advocated by Snegeriff, to check the bleeding, drainage being secured by the use of a glass tube instead of gauze.

Again, it is an established principle of abdominal surgery that, where it is possible, raw surfaces should be covered with peritoneum. Much can be done in this line in the treatment of the stump left after removal of an ovary and tube. In a case uncomplicated by adhesions the appendages can be enucleated according to the method employed by Dr. Pratt, of Chicago, with almost no bleeding, and the edges of the peritoneal surfaces of the broad ligament be brought together by a continuous catgut suture, leaving only a linear denudation; or a chain ligature may be used and the peritoneal surfaces united. A broad, thick stump is undesirable, as neither oil, aristol, nor the cautery will prevent adhesions.

After all serious abdominal operations, especially when

complicated by adhesions necessitating more or less violence to the intestines, there is irregular, painful peristalsis during the first twenty-four hours or more. It is then that tonic spasmodic contraction of the intestinal muscles occurs. In support of this view let me again mention the two cases reported by Dr. Ashton, where autopsy revealed this condition. Another case was reported to me by Dr. O. E. Herrick, of obstruction following cœliotomy, which resisted all measures to provoke a fecal movement, or even the escape of flatus, till finally, in desperation, a quantity of tobacco smoke was thrown into the rectum. In a very short time free fecal evacuations occurred. In my own practice, after cœliotomy for the removal of pus-tubes, efforts to produce catharsis resulted only in the production of nausea and vomiting, which rapidly became fecal. There was only moderate abdominal distention. The pulse and temperature did indicate sepsis, and yet the most faithful and persistent effort failed to even provoke an escape of flatus. To give the patient temporary relief from the persistent vomiting I gave a large hypodermic injection of morphine. Within one-half hour her bowels moved *copiously*.

The condition in these cases was, I am confident, tonic obstructive spasm of the intestines, aggravated by injudicious treatment. The indication in such cases is to cause relaxation of the spasm, and thus relieve the obstruction. For this purpose no drug is equal to opium in some form, preferably codein, because of its freedom from nauseating tendencies. In saying this I am aware that I am advocating that which, in the opinion of many, is heresy. There are those who, in the strongest possible terms, condemn the use of an opiate after abdominal section; but I contend that by the free use of codein during the twenty-four or thirty-six hours immediately following an abdominal operation, the best interests of the patient are conserved. It not only relieves the pain (often severe), but, by its sedative action on the sympathetic nervous system, allays irritation and converts irregular

spasmodic intestinal contraction into normal, painless, though active, peristalsis. I always use it freely in this manner, and have never had occasion to regret it. Under its use normal peristalsis, as evidenced by the escape of flatus, is more quickly established, and instead of delaying catharsis it makes it more easily possible.

The very early use of cathartics in cases of spasmodic abdominal pain after section I cannot sanction. This pain is, as I have said, caused by irregular obstructive intestinal contractions, and the use of a cathartic before normal peristalsis has been restored aggravates the existing irritation. Sedatives are indicated. The condition is parallel to that existing in lead-colic, where no remedy will overcome the existing constipation and promote an evacuation of the bowels as readily or certainly as a free use of opium, or to those cases cited by Brunton of obstinate constipation complicating ovarian neuralgia, where opium not only relieved the neuralgia, but produced catharsis.

It may be urged that the use of codein after abdominal section will tend to promote the formation of adhesions by preventing peristalsis, but I cannot think so. The conditions are abnormal. It must be remembered that the intestines are no longer quiet. In addition to the stimulus of irritated nerves, there is the normal stimulus of gas in the intestinal canal. The intestines are no longer empty and flattened as at the beginning of the operation, but by the processes incident to the operation there has been a formation of more or less gas, supplying a normal stimulus to peristalsis within the canal itself. Moreover, the peristalsis is not normal nor regular. It is more or less spasmodic, even obstructive. Codein, properly used, instead of quieting normal peristalsis and allowing adhesions to form, quiets excessive peristalsis, regulates the action of the intestines, and renders the production of catharsis possible at an earlier period than by any other means. I believe that in serious abdominal operations the exhibition of cathartics by the

stomach should be avoided till it is shown by the escape of flatus that normal peristalsis has been re-established, but that we should rely on the free use of codein, supplemented, if necessary, by the use of high enemata.

If, after judicious trial of the measures above detailed the symptoms of obstruction persist, the abdomen should be re-opened and the cause of the obstruction sought. Nor should we hesitate to incise the intestines, if necessary, to reduce their volume. For organic obstruction there is no other remedy.