Functional ileus: with special reference to its mechanism and treatment

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FUNCTIONAL ILEUS; WITH SPECIAL REFERENCE
TO ITS MECHANISM AND TREATMENT

BY PHILIP A. FUQUA

SENIOR THESIS
PRESENTED TO THE
COLLEGE OF MEDICINE
UNIVERSITY OF NEBRASKA
OMAHA 1940
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"Paralytic ileus" is the term that has been conventionally applied to a group of symptoms consisting of nausea and vomiting and a failure of the bowel contents to move forward. Some authors subdivide this term into adynamic and dynamic or (spastic) ileus. After surveying the literature it would seem that the term "paralytic ileus" is badly chosen, since the intestinal wall is not paralyzed but its activity appears to be inhibited by an overactive sympathetic nervous system, because it is found by blocking the splanchnic nerves or by spinal anesthesia, the paretic intestine of "paralytic ileus" can be made to contract. Probably Molivera (1) term "functional obstruction", better applies to this condition since there is a complete absence of propulsion of gas and fluid, a condition identical with that of complete organic obstruction. Gray (2) calls it "inertia distention."

Hay (3) considers this group of symptoms a syndrome and not a disease entity. Some writers dignify the common forty-eight hour post-operative distention, pain, vomiting, and temporary paresis as post-operative paralytic ileus, but cases which yield to hot fermentations, enemata and simple tubal drainage seem in the
opinion of most authors not to deserve this classification. While functional obstruction may represent progression from ordinary post-operative distention, it is the more advanced ileus, usually due to the added presence of infection as gross peritoneal irritation, injury, vascular changes, or extra abdominal causes that is considered for the most part in this paper.

Stout (4), however, states that ileus should be considered a definite pathological entity. The usual course he states is one of slow and insidious progress, the majority of cases never reaching the final textbook picture of ileus, but the speed with which ileus develops varies greatly and some cases rapidly reach the end stage of complete paresis.

Functional ileus may be defined as a disorganization and impairment of motor function of the gastro-intestinal tract with resulting toxemia and blood chemistry changes.
McIver (5) reviews the history of the subject in detail. He divides the progress made into three stages occurring since the beginning of the 19th century. In the first period which roughly covers the whole of the 19th century the development seems to have centered about the study of the anatomy and the physiology of the disease, and their relation to the clinical picture. It was in this period that different types of obstruction were distinguished and a start made on the study of the cause and mechanism. Ileus began to be distinguished from conditions such as typhoid fever, peritonitis, colic, asities and from other conditions.

McIver (5) culminates this stage of development in the last 25 years of the 19th century and the first decade of the 20th, when the development of abdominal surgery made possible a better knowledge of the pathological changes occurring early in the disease. He cites the works of Brenton, Leichtenstern, Bryant, Fitz, and Treves as outstanding in this period.

Treves' (6) work on 'Intestinal Obstruction' is interesting. The work deals mostly with mechanical obstruction. In considering 'paresis' of the gut he states that electricity as a cure for intestinal
obstruction, so far as he could ascertain were for the most part examples of ileus depending on fecal accumulation. In these cases electricity, he states, as a mode of treatment is "intelligible and may be expected to be of benefit."

McIver (5) begins the second period in the latter part of the 19th century and culminates it in the first quarter of the present century. This period seems to be characterized by a study of the toxic factors. He states in part: "With the increasing number of abdominal operations and autopsies the pathological picture presented by acute intestinal obstruction became clearer, and it was soon apparent that frequently these findings did not fully explain the acute symptoms and prostration manifested by the patient during the illness, nor adequately account for the death that so often ensued. The search for less obvious factors which might explain the observed factors has centered around the belief that the course of events following intestinal obstruction could best be explained by the action of some powerful toxin. The rise of bacteriology gave stimulus and direction to these investigations. The bacterial invasion of the peritoneal cavity and blood stream was first investigated, but it was found that while this occurred in certain instances it would not account for the death of the patient in a far larger number of instances where the findings were negative.
Attention was next turned to the contents of the obstructed bowel, which were obviously foul and grossly contaminated with bacteria; and it was easily proved by injecting this material into other animals that it was usually highly toxic. The possibility that some factor other than bacteria might be responsible for the acute illness has also been studied in length, notably that a toxic secretion is formed by the bowel mucosa or that certain of the digestive ferments become toxic under conditions of obstruction. A great deal of work has been done in an attempt to isolate and identify a single chemically pure toxin from the obstructed bowel; but while a number of substances have been isolated that proved to be highly toxic, there has been no general agreement as to their actual role in the disease."

Von Albeck, Clairmont, Roger, Murphy, Vincent and Drapper are the men of this period McIver considers of note.

Drapper (7) concludes at this time, "that the power of the liver to pair camphor and glycuronic acid is probably seriously impaired after duodenal obstruction and that toxemia in obstruction undoubtly arises from an interference with cellular reactions of the intestinal epithelium and in addition to the "penetrating of jejunal and iliac epithelium in

-5-
in the stomach of post-operative cases an emulsion of them should probably be used in colonic irrigations for the same indications and purpose."

In the third and present period McIver points out that the chief interest or point of advance has been in the less obvious bodily changes that come about as secondary results of obstruction, especially as shown by the chemical and physiological changes occurring in the body and in the body fluids, especially in the marked dehydration and lowering of the blood chlorides.

Hartwell and Noguet, Holden and Orr and others have done much work along these lines. McIver points out however that since these changes do not occur in all types of intestinal obstruction and when treatment is calibrated to correct them indiscriminately the results have naturally been disappointing. He goes on in part: "The investigations made during the third period concerning the changes that occur in the blood and body fluids with certain types of obstruction and the measures that may be taken to correct them are of first importance and can be understood only if one has a clear conception of the fundamentals of the altered physiology and pathology in different types of obstruction. Therapeutic measures have been influenced by all these findings but surgical treatment has rarely
Followed systematically on the advance of the theoretical knowledge of a disease, and in its early beginnings was empirical and designed to relieve certain symptoms. Attempts to treat by surgical measure, cases of intestinal obstruction have from very early times to the present proceeded along two main lines: First by the indirect method whose original object was to relieve certain symptoms, particularly distension by draining the intestine above the point of obstruction and secondly, operations intended to find and remove the cause of the obstruction. The most primitive of the indirect methods of tapping the distended coils of intestine by blind puncture through the abdominal wall goes back to very early times when this operation was used to relieve distension of the abdomen both from ascites and from accumulations in the intestine. The making of an external opening in the colon for the relief of distention has been practiced since the beginning of the 18th century at least, for Litter described this operation in 1710. A later modification by Amusatt consisted in opening the colon in the left lumbar region; over a hundred years later Dupuytren established drainage from the small intestine above the point of obstruction. It remained for Belaton in about the middle of the century, to establish this as an accepted surgical
procedure carefully thought out. The value of enterostomy was soon realized and it is in use today modified in technique and choice of location but not in principle. This procedure could usually relieve only cases of simple obstruction and did little or nothing to relieve the more frequent cases where in addition to the occlusion of the lumen there was interference with mesenteric circulation to the involved segment. Almost simultaneously with the employment of these indirect methods, direct attempts were made to relieve the condition by opening the abdomen and removing the obstruction. Before the days of asepsis and anesthesia these procedures were desperate and few in number."

The literature of the last 40 years reveals many new concepts both as to treatment and to mechanism. There has been a better understanding of the toxic factors and the secondary changes that occur in the body fluids. Much study has been done on the inorganic chemicals of the digestive juices especially sodium and chloride ions and their relation to dehydration. The roentgen ray has aided materially in the diagnosis. New drugs have been introduced along with improved methods of tubal drainage, so that the chances of a patient with 'ileus' have increased considerably.
It is the attempt of this paper then, to very briefly review the literature on the recent concepts of the disease with special reference to the mechanism and treatment of the functional variety of ileus.
DIAGNOSIS

The diagnosis of a functional obstruction will first be considered very briefly.

Most authors stress the necessity of making an early diagnosis. The real difficulty, however, seems to be not in diagnosing the presence or absence of an obstruction but in distinguishing between the organic and functional variety. Both types are found to have many basic signs and symptoms in common.

In reviewing the literature, few laboratory procedures are found to be of any benefit in the early diagnosis. Also it is found that the clinical signs and symptoms in the early stages are not at all constant. It is found that muscle spasm and tenderness may be absent. Abdominal distension may be absent or slight, the temperature and white blood count may be within normal limits. Usually the cardinal symptoms of abdominal pain, obstipation and vomiting are present but they are found to vary much in intensity and are also associated with diseases which may or may not require such careful treatment. Most authors state, as in any diagnosis, that the patient should be studied in general and with special reference also to information which does not deal particularly with the presenting symptoms, since the suspected obstruction may develop into lead colic or an abdominal
crisis of tabes or some other condition.

Since the pain, obstipation, and vomiting are most always present each one will here be considered in more detail.

Pain: Green (8) states that chills may be present in acyclic ileus but not in mechanical obstruction. He points out the pain is not colicky in type; also the complete absence of all peristaltic sounds in place of the turbulent gurgling bowel noises heard in the mechanical types. Malver (5) points out that where peritonitis complicates the picture or following serious operative procedure pain may be absent or if present may be not the outstanding, but only a very minor feature.

Donaldson (9) points out in his series of cases that in no instance was distention definitely noted in the first twenty-four hours. After forty-eight hours nearly all the cases presented clinical distention but in some was not marked in the earlier part of the fifth, twelve hour period.

Morgan and Hoffman (10) state that in a series of 100 cases, abdominal pain was present in 86%.

Obstipation: With complete intestinal obstruction it is evident that the intestinal stream is completely stopped; but with cessation of bowel movement even after appropriate stimulation, is by most authors not considered.
pathogogenic of the disease. It is found that obstrinate constipation and reflex inhibition of intestinal motility occur in other conditions in which abdominal pain occurs, as for example, renal colic. Stout (4) points out that even though the bowels have moved after the onset of the pain this is not conclusive evidence that obstruction does not exist, but he says, "failure to obtain even gas on the administration of subsequent enemas, however is a finding that cannot be disregarded, and in conjunction with abdominal pain should suggest complete obstruction." Abbott (11) states this should call for consultation.

Morgan and Hoffman (10) found constipation or obstipation present in 40% of the cases.

**Vomiting:** As before stated it is found that vomiting is most always present and is found to occur very promptly after the onset of the obstruction. The vomitus is found to present no characteristic feature early in the disease but later it is found to have characteristic and distinct qualities. McIver (5) describes it as a thin yellowish fluid containing small whitish particles that settle out on standing. The odor is foul and extreme. This fluid represents the contents of the small intestine and is a mixture of the secretions of the digestive glands and of the intestinal tract, which are poured out in great quantities and infected with bacteria. This is the material usually described as 'fecal' or 'stercoraceous'. The odor is from the colon bacilli as well as other nutre-
factive bacteria. It does not MoIver (5) makes clear, contain any feces since true vomiting of feces is found only where there is a fistulous communication between the stomach and colon.

So it is concluded that vomiting does not aid materially in the diagnosis since between the onset of obstruction and the appearance of the characteristic vomitus an interval elapses which in some cases may be days so that inspection of the vomitus may only confirm the diagnosis, and the most favorable time to initiate treatment may be found to have passed.

Physical Signs: No physical signs are found to be conclusive. Depending, on course, they may be found to be slight, absent or pronounced. Distention is the most common finding. Since, however, it takes an appreciable length of time for fluid and gas to accumulate in sufficient quantities to produce a demonstrable distention of the abdomen, this is not an early symptom unless there has been some chronic obstruction before the onset of the acute attack. Muscle spasm and tenderness are also found to be late signs.

Diagnosis by X-ray: Diagnosis of obstruction depends on noting the collections of fluid and gas in the intestine and the characteristic patterns they assume. In the small intestine small accumulations of gas may give the outline described by Case (12) as the "herringbone"
appearance; or a ladder arrangement of the dilated coils. The diameter of the dilated coils is also a point to note carefully according to Case (12). Ochsner and Granger (12) have found the detection of multiple fluid levels to be a very important point in diagnosis. They point out that to see this, the patient must be in the upright position, either sitting, standing, or lying on the side, the picture being taken anterio-posterior or a lateral view with the patient lying on back.

Smith (14) considers the roentgenologic examination very important and reliable being 92% positive in 78 cases. He recommends routine preoperative roentgenologic examination.

Differential Diagnosis: Diseases that may be confused with acute intestinal obstruction are in general those that give abdominal pain and at the same time cause interference with intestinal motility. The list of diseases having a symptom complex which may, at least in part resemble that of acute obstruction is long; a few outstanding examples of different types may be mentioned and briefly commented upon.

Among the more general constitutional diseases that may have abdominal symptoms of sufficient prominence to be confused with intestinal obstruction should be mentioned, lead colic, the gastric crisis of tabes, and uremia.
Müller (5) points out it should not be forgotten that pneumonia and cardiac thrombosis are capable of causing severe upper abdominal pain which may be accompanied by nausea and vomiting. Among the more rare medical conditions that have been mistaken for obstruction, Treves (6) mentions cholera and poisoning by arsenic. Oeler (15) states angio-neurotic edema may at times cause intense abdominal pain, nausea and vomiting.

The differential diagnosis then, depends upon finding in the history and physical examination, clear evidence that some disease other than intestinal obstruction is responsible for the symptoms.
GENERAL ETIOLOGICAL CAUSES

An outline of the general etiological causes as given by Ochsner and Gage (16) is here given. They divide the causes into intra and extra abdominal.

I. Intra Abdominal
   A. Peritoneal irritation
      1. Traumatic
         (a). Post operative
         (b). Penetrating wounds
      2. Bacteria
         (a). Peritonitis
      3. Chemical
         (a). Extravasation of blood
         (b). Perforated peptic ulcer
         (c). Bile Peritonitis
         (d). Acute pancreatitis
   B. Vascular Changes
      1. Strangulation
         (a). Intramural distention following mechanical ileus
         (b). Extramural compression of mesenteric vessels
      2. Mesenteric Thrombosis
   C. Extraperitoneal Irritation
      1. Hemorrhage
      2. Infection
      3. Renal

II. Extra Abdominal
   A. Toxic
      1. Pneumonia
      2. Uremia
      3. Empyema
      4. Systemic infection
   B. Neurogenic
      1. Injuries and diseases of spinal cord
      2. Lead Poisoning
      3. Fracture of lower ribs
         Irritation of splanchnic nerves
INCIDENCE AND MORTALITY

Van Buren and Smith (14) give a comparison of mortality of groups classified according to types in a series of 130 acute ileus cases.

<table>
<thead>
<tr>
<th>Series</th>
<th>Total No.</th>
<th>Late Case%</th>
<th>Recovered</th>
<th>Died</th>
<th>Mortality%</th>
</tr>
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<tr>
<td>Complete Obs.</td>
<td>91</td>
<td>62</td>
<td>78</td>
<td>12</td>
<td>14.2</td>
</tr>
<tr>
<td>Incomplete Obs.</td>
<td>26</td>
<td>88</td>
<td>12</td>
<td>11</td>
<td>42.2</td>
</tr>
<tr>
<td>Paralytic Ileus</td>
<td>12</td>
<td>69</td>
<td>1</td>
<td>12</td>
<td>92.7</td>
</tr>
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</table>

In this series of cases, a case was considered to be "paralytic ileus" if the intestines were distended uniformly, diffuse peritonitis was present and if no obstruction was found.

Ileus complicating all kinds of conditions are given in the literature. Gillespie and Rogers (17) report a case of a dynamic ileus in the newborn. Orr (18) cites several cases complicating skeletal injuries. Hoy (19) reports cases of intestinal paresis occurring from embolism and thrombosis of the superior mesenteric artery. Tucker (20) in a series of 89 cases reports a mortality of 28.21% in cases coming to surgery. Cases following appendectomy and gall bladder surgery were most frequent. Since division of different types of cases is not observed, the figures so compiled give in most cases, the gross mortality of a group of cases simply classified as "acute obstruction." So that no exact figures are available for the functional types of obstruction alone and since the functional type often complicates the organic condition figures are again confusing.
Suppose then, we have a case of functional obstruction let us consider why the obstruction has occurred.

The whole picture of the empty stomach and motionless intestine is that of a protective mechanism. It is to be regarded as a physiological response, to injury or infection as a mechanism designed to mobilize and rest the inflamed part; to prevent the ingestion of food and to localize the inflammatory process (16).

The presence of Pacinian (or touch) corpuscles in the mesentery, long recognized by Keith (21) and described as 'end organs for receiving pressure' are found to excite the reflex immobility of the abdominal walls, also a protective mechanism.

As shown in the preceding outline, the cause may be the result of local or intra abdominal pathology or may be a reflex response to general or distant body pathology.

MoIver (5) in discussing the complicated mechanism governing peristaltic states that it depends on the muscle structure of the gut wall, with its local nerve supply and influenced by an extrinsic nerve supply through the splanchnic and vagus nerves. Thus injury to the muscle or nerve elements in the gut wall might abolish
peristalsis; or reflex impulses transmitted over the extrinsic nerves might bring about the same result. The situation is further complicated by the fact that the bowel may respond to one and the same injury, at times by an atonic paralysis or inhibition of motility, and at other times, by a spasmotic tonic contraction of some segment of the intestine which interferes with the passage of the intestinal contents. Because of the fact that it has long been the custom to describe it as before stated, a functional obstruction as 'paralytic' or 'adynamic' and 'spastic' or 'dyskinesia.'

Angle(22) in his work on intestinal inhibitory reflexes presented evidence in 1926 showing that impulses from the urinary tract, rectum, peritoneum and certain areas reflexly diminish the tonus and movement of the small intestine. He concluded that the splanchnics contain the efferent paths of these reflexes, that the vagi are very little involved in these reactions and that some of the afferent paths involved are in the hypogastria.

In considering the mechanism of normal peristalsis there are generally described four types of activity in the small bowel. First, local swinging movements or rhythmic segmentations which knead the intestinal contents and mix the food taken in with the digest-
ive contents or juices; secondly slow changes in tone; and thirdly peristaltic rushes named by Metzer and Auer (23), which some times run from the stomach to the anus carrying more or less material before them. Brown and Workman (24) in 1924 described also a gentle swaying movement of the loop or 'pendulum movement'. Alvarez (25) points out that it is important to remember that all these activities continue after degenerative section of the vagi and splanchnics and that the first two and possibly the third continue even after removal of the bowel from the body. Bits of intestinal muscle cut out and suspended in oxygenated Locke's solution will contract rhythmically for hours at a time and the fact that they will sometimes beat more regularly on the third day after excision than on the first day, leads Alvarez (26) to believe that nervous ganglia have little to do with the phenomenon.

Then the anatomical intervention of the bowel is considered it is found that there is an intrinsic and extrinsic intervention. The intrinsic consists of a nerve plexus in the intestinal wall; the submucous plexus or Meissner's plexus and the more important myenteric plexus or Auerbeck's, which lies between the circular and longitudinal muscle layers and serves to conduct stimuli and to coordinate movements. Upon this reflex depends the local reflex or myenteric
reflex, (27) which according to Bayliss and Starling (28) governs the orderly process of the intestinal contents, a wave of contraction being preceded by a wave of relaxation. Alvarez (29) questions the importance of the myenteric reflex and the propagation of the peristaltic wave. According to his view, the downward passage of a wave is due to gradients of irritability, tone and metabolism between the upper and lower portions of the intestinal tract. He reasons that whatever the mechanism is that is responsible for the caudal progress of food, must be built into the structure of every inch of the bowel because when short segments are cut out, turned end for end, and the continuity of the gut restored, the direction of peristalsis in the reverse segment remains unchanged; liquids will pass but solids eventually kill the animal by blocking the lumen of the bowel. Every segment, then, he says in a normal bowel is so constructed that peristaltic waves tend to go over it more easily in one direction than in the other. The condition resembles that present in certain worms in which 'polarization' is so perfect that if the animal is cut into very small pieces they all crawl in the same direction toward the point where the head used to be.

Alvarez (29) further reasons that whatever causes this polarization must be graded from the duodenum to
the lower part of the ileum or it would not be demonstrable on every segment. Actually Alvarez and his associates, while studying the physiological properties of intestinal muscle found that many graded differences in irritability, rhythmicity, tone, latent period, muscular strength and metabolic rate exist. His studies show that just as in the heart, so in all the bowel, waves begin in the region of highest rhythmicity and run to the region of the lowest rhythmicity.

If these graded differences account for the direction taken by peristaltic waves, it follows he continues, that if the metabolic rate and the irritability of the ileum were to be raised in any way, as by the presence of inflammation, the gradient of forces might be so flattened or reversed that the bowel would transport material caudad no better than it ordinarily transports it crad. There may be active kneading movements in such a bowel but no more transportation of material than would be seen in a level ditch. Furthermore, if toxins of disease should injure the sensitive duodenum more than the hardy ileum they would upset the gradient of forces by weakening the pumping power at the upper end, according to Alvarez' own statement.

That irritation of the lower end of the bowel will slow the progress of the material coming toward it was shown by Cannon, when he irritated the cecum by injecting
the cecum with croton oil. It has been shown that in acute ileus the bowel for some distance above the obstructed and highly irritated segment is empty. Food residues are held back in the duodenum and jejunum and as Alvarez (29) states if enterostomy is to do any good the opening must be made far oral in the region of the gut were material has accumulated; and if on the other hand, the obstruction is produced slowly without irritation of the muscle, as by carcinomatous ring, the intestinal contents may pack up against the obstruction. In animals, Alvarez found the segment just above the block to be distended.

These observations are all explained, he states, on the basis of the gradient theory and the law that Alvarez associates with it, namely, that irritation of the bowel at any point tends to hold back material coming down from above and tends to hasten the progress of material that has passed. The rapid progress below an irritated segment they found will sometimes produce diarrhea, but that often, this hastening effect is obscured by the restraining action of the ileo-cecal and anal sphincters.

The work suggests, he concludes, that in the treatment of dynamic ileus attempt should be made first, to remove nervous inhibition, and, second, to disturb the normal dynamic gradient by giving food and avoiding
morphine and irritation to the lower bowel.

Experiments of the German workers, Notz and Arsl as reviewed by Alvarez led them to conclude that in some cases of ileus even with peritonitis the intestinal muscle is still capable of active contraction and is only held in leash by nervous inhibition. In addition, Alvarez points out, conduction down the bowel may be made difficult or impossible by flattening or reversal of 'gradients.'

Cannon (31) considers the myenteric reflex important but states that it does not govern the rhythmic contractions of the small bowel or the rhythmic peristalsis or antiperistalsis of the colon and probably not the rhythmic waves of the stomach.

Cannon states that although the intrinsic inter- vention is complete in itself and capable of carrying out in an orderly manner all types of peristalsis without connection with the central nervous system, it is influenced by impulses from the central nervous system transmitted over two opposed systems which together constitute the extrinsic intervention. The motor system is represented by the cranial (vagus) and sacral autonomies whose impulse tend to stimu- late intestinal movements and increased tone; and the inhibitory system consisting of sympathetic fibers (splanchnic) whose impulses tend to abolish intestinal
movement. The sympathetic also carries fibers to the pyloric and ileo-cecal sphincters. The cranial autonomic system, through the vagus, supplies fibers to the intestinal canal to the terminal ileum diminishing in its influence as it descends; the sacral system starts at the anal end of the canal and reaches upward along the colon with a diminishing influence as it ascends. Stimulation of these nerves are found to produce powerful contractions of the intestinal musculature. It is not known where the ganglion cells of these motor neurons to the stomach and intestines are situated, so it is not known with certainty that the vagus carries preganglionic fibers to these organs. Gaskell (32), however, thinks it probable that the motor cells are situated in Auerbach's plexus.

The sympathetic fibers are found to arise in the thoraco-lumbar region and reach the abdomen by way of the major and minor splanchnic nerves, the preganglionic fibers ending in ganglion cells in the celiac plexus. It has not been finally settled whether there also exist ganglion cells belonging to the sympathetic system in the intestinal wall; but the evidence as presented by Gaskell (32) seems against it, as stimulation of the splanchnic nerves is found to inhibit peristalsis and constricts the ileo-cecal and pyloric sphincters.
Intestinal peristalsis, then, is carried out by a neuro-muscular mechanism consisting of the smooth muscle of the intestinal musculature and the intrinsic nerve supply already described.

The respective parts played by nerve and muscle in the initiation and propagation of intestinal movements is not altogether clear. The views on this question have been embodied in the neurogenic and myogenic theories. There has been much investigative effort along this line and it has been found that the rhythmic segmentation of movement is not effected by the application of cocaine or nicotine and since these drugs paralyze nervous structures it has been concluded by many authorities that the rhythmic movements are myogenic in origin. This opinion is confirmed by the most recent work. McIver (5) explains it by saying, that rhythmic contractions and peristaltic waves may depend upon different mechanisms. The rhythmic contractions he states are simpler and more primitive and their function depends upon the well-known inherent ability of smooth muscle to contract in a rhythmic manner. The more complicated, highly developed peristaltic waves may depend upon the nervous elements for its initiation and propagation. This is a question of some practical importance he points out, in considering the functional disturbances of motility; for diverse types of injury may affect
different portions of the neuro-muscular mechanism. Cannon (23) and Gunn and Underhill (24) state that because of the intimate anatomical relations between muscle and nerve evidence has been hard to establish.

They state that the muscle bundles are surrounded by nerve filaments and even the smallest collection of muscle fibers show ganglion cells, so that methods tending to destroy one without the other may be inconclusive because of the resistance to anemia of this type of ganglion.

It is interesting to consider here what happens when the nervous inhibition of the intestine is removed. Alvarez (35) states that since the gastro-intestinal tract is so highly automatic it seems to him unlikely that a withdrawal of nervous stimuli could produce paralytic ileus. It is more probable Alvarez states "that post-operative ileus would be produced by a stream of inhibitory stimuli arising in the brain or cord or in the tissues, cut or injured during an operation. In animals with the abdomen open under salt solution, the bowel usually remains quiet until the distal two-thirds of dorsal cord is destroyed or the splanchnic nerves are cut. Rabbits with the vagi or splanchnics or both cut and most of Auerbach's plexus degenerated have such overly active intestines that
many develop diarrhea and apparently die of inanition. The animals eat, but the stomach and cecum empty so rapidly that sufficient time is not allowed for digestion and absorption. But even when the intestinal muscle is paralyzed it is hard to understand why it shall completely fail to pass onward its contents." Alvarez has published records showing the seepage of fluids through the bowel in the absence of rush waves. He has found that dogs will go on living and digesting after the removal of all the muscle from the long segments of the bowel.(25).

Alvarez (25) states that the behaviour of the excised digestive tract of rabbits might be due at times to a tendency of denervated bowel to cramp down and resist the passage of liquid and gas. This cramp-like action might be brought about, he states, not by an increase in the nervous activity but by a decrease, a decrease similar to that responsible for spastic paralysis in the arm or leg. At other times, Alvarez explains the obstruction seems to be produced by gas which causes the bowel to become packed tightly in the abdominal cavity. Normally, the innumerable kinks do not produce obstruction since rush waves travel rapidly, push material ahead of them with such force so as to distend the loops and straighten them, but Alvarez found in sickly animals with an abscess about the ileo-
cecal sphincter, the rushes travel so slowly and hesitatingly that it does not take much to stop them.

Having then discussed the normal mechanisms of intestinal movement, the mechanisms coming into play when the intestine is functionally obstructed will now be considered.

The sequence of events seem, according to most authors to occur about as follows: (a) Loss of tonicity of the gut through splanchic stimulation such as occurs most frequently in peritonitis or operative interventions involving the peritoneum or intestinal musculature. (b) This primary inhibition of peristalsis results in gaseous distension, mostly, it is thought because of decreased absorption of gases from the bowel by the blood stream. The decreased rate of absorption is usually explained as being due to a decreased rate of local blood circulation, because of the lost tone of the bowel, thus slowing gaseous absorption. (c) Dehydration which causes a more sluggish circulation in the intestinal mucosa due to an increase in viscosity, thus aggravating the tympanies by disturbing the normal gaseous exchange. This distention, then, (d) acts to lessen absorption of gases also, by impairing the mesenteric circulation on the venous side and in addition diffusion of gases from the blood into the bowel lumen is much increased. It is generally believed
that as distention increases the pendulum movements of the gut stop first, then, the rhythmic contractions and the true peristaltic wave last. Then, when the pressure within the intestine equals the venous pressure, necrosis results (36). (e) The partial ileus resulting from these factors in some manner brings about the elaboration of a toxin which reduces the contractility of smooth muscle and has a paralyzing action on Auerbach's plexus. This last action completes the picture of complete motor inhibition, the result being a functional ileus as deadly in its consequences as that produced by an organic obstruction.(36).

Each one of these events will now be considered in more detail.

The occurrence of these types of obstruction have been recognized since the time of Henrot (27) but the exact way in which they come about is not generally agreed upon in all respects. Cannon and Murphy (38) showed experimentally in 1936 that distant pathologic processes were capable of inhibiting intestinal movements. They noted that authentic states occurring in the course of general infection tended to abolish or inhibit intestinal peristalsis. Next, they studied the effects of powerful sensory stimuli which they produced by injury to the testicles,
of the anesthetized cat. They found that such sensory stimulation also caused an inhibition of intestinal movements. These authors, further showed that the stoppage of intestinal movements under the foregoing conditions was due to inhibitory impulses from the spinal cord transmitted over the splanchnic nerve; for when they sectioned this nerve the inhibition was abolished. Following section of the splanchnic nerve intestinal peristalsis was observed even in the morbid animals. This reflex mechanism whereby distant lesions are able to bring about an inhibition of intestinal movements is now generally accepted.

This original work by Cannon and Murphy was confirmed in 1924 by King (29).

Only two types of obstruction produced by local injury seemed to have been studied in any detail. They are, those following trauma to the gut by rough handling and those following bacterial peritonitis. Cannon and Murphy (35) investigated the intestinal inhibition of peristalsis that follows trauma to the gut. They found that they were able to produce strong inhibition of intestinal movement by rough handling of the intestines of a cat and considered that the inhibition of movements following this type of injury was probably due to direct injury to the neuro-muscular mechanism of the intestinal wall. They were of the opinion that
inhibitory impulses from the spinal cord did not play any role in this instance, for the inhibition was not removed by section of the splanchnic nerves.

These findings have been confirmed by Olivecrona (40). This author considered another possibility, aside from the injury to the neuro-muscular mechanism of the bowel, or inhibiting impulses from the spinal cord, might explain the inhibition: namely, that there might be a local reflex by way of the celiac plexus. Having first confirmed the observation of Cannon and Murphy that section of the splanchnic nerves did not abolish the inhibition of intestinal movements produced by trauma, he removed the celiac plexus as thoroughly as possible; and after allowing time for the degeneration of the nerve fibers, traumatized the intestine and found that the usual post-traumatic inhibition was almost completely absent. Olivecrona considered that these experiments proved that trauma to the gut may produce inhibition by local reflexes through the celiac plexus, rather than as a result of injury to the neuro-muscular structure of the gut wall itself.

The various views as to the mechanism by which peritonitis brings about functional inhibition of intestinal movements are vague. Eims (41) assumes that both muscle and nerve plexuses are affected by the edema and inflammation resulting from the action of bacteria and their toxins. Cannon and Murphy (28) assumed that
the mechanism was the same as in the case of mechanical trauma by direct injury to the intestinal musculature and its intrinsic nerve supply and that section of the splanchnics, therefore should not abolish the inhibition. Arai (42), however, found that when he produced peritonitis by intraperitoneal of sub-lethal doses of bacteria, inhibition of movement occurred; if, however, the splanchnic nerves were first cut, no inhibition followed the peritonitis. This was also true when he produced a chemical peritonitis by injection of an irritating solution (5cc per kg. body weight of Lugol's solution). He concluded, that in peritonitis, inhibition of the intestinal movements was produced by inhibiting influences from the spinal cord. Olivecrona (40) was not able to confirm Arai's observation that section of the splanchnic nerve abolished the inhibition of intestinal movements, caused by peritonitis of bacterial origin. He explained the difference between his own findings and Arai's by the fact that he had produced a rapidly advancing, fatal peritonitis; and drew the conclusion that in the milder grades, paresis of the intestinal movements was brought on by inhibitory influences from the spinal cord, while in the more severe forms the disturbances of intestinal motility were caused by injury to the neuro-muscular mechanism in the wall, agreeing with Cannon and Murphy (28)
in this. But since he found that neither mechanical trauma or peritonitis abolished rhythmic contractions he was inclined to the belief that the chief injury is sustained by the nerve elements rather than the muscular structure.

It is found that the late stages of most cases of mechanical obstruction are complicated by functional disturbances of the motility of that portion of the bowel lying above the obstruction and not infrequently when patients are operated on late in the disease, according to Moller (5) the bowel does not regain its motility even when the mechanical difficulty has been relieved, the normal tone and peristalsis having been completely abolished by the long continued distention and resultant injury to the capillary circulation of the intestinal wall.

In connection with these functional disturbances Richards, Fraser and Wallace (43) have pointed out that resections and anastomoses for gunshot wounds of the intestines are often followed by functional obstruction, the segment above the anastomosis becoming distended and paralyzed. This result may follow even though the operation take place relatively soon after injury, although localized peritonitis frequently plays a part in this picture, these authors consider that the functional element due to trauma of the gut.
is an important factor.

Functional disturbances caused by distant lesions have been reported as occurring often and most frequently in connection with renal pathology, tumors, infection, operations or colic. They have also been observed following retroperitoneal hemorrhage or infection (44). Esenbrath (45) stresses the intimate relationship between the splanchnic nerve supply to the kidneys and to the intestine. Texier and Clavel (46) have also discussed the reflex mechanism whereby pathological processes in the kidneys or retroperitoneal tissue produce serious gastro-intestinal disturbances.

It is interesting to note that obstruction can occur without any narrowing of the lumen as shown by Alvarez (29). He opened the abdomen of a rabbit under normal saline and bruised the intestine with a hemostat, closed the abdomen, and reopened several hours later and observed that no food had passed the site of injury, although ample peristaltic waves passed downward, they broke up on approaching the ring of injured tissue and the proximal loops were found distended with gas.

Having a motionless gut, then we are now ready to consider the process of gaseous distention.

Gases in the intestine were first systematically studied by Planer in 1860, as recorded by Kantor (47), who has collected an extensive bibliography on the
subject.

Normally gas is present in the intestine in small amounts. The origin of this gas according to Kantor (48) is from two sources, from the atmospheric air and from the food stuffs.

Atmospheric air, he explains is swallowed during eating, and especially while drinking. By far the greatest part of injected air is quickly belched. What little is left in the stomach assumes significance because it is composed of 80% nitrogen which is practically unabsorbable by the body and 20% oxygen which is absorbed in part only. This residue Kantor points out, is forced to make the transit of the entire digestive tract, and appears almost quantitatively in the flatus. Enormous quantities of gas are produced during the intestinal phase of digestion, the exact amount depending on the composition of the diet. Most of this gas, however, has been shown to readily absorb into the blood stream and to be fixed there or exhaled through the expired air. By far the greatest part of the gas produced by food digestion is carbon dioxide. Kantor gives the following sources. (a) In the upper small intestine from a mixture of the acid gastric contents with the carbonates present in the alkaline biliary and pancreatic secretions. This amount thus
manufactured has been estimated at six liters daily.

(b) In the lower small intestine from the possible bacterial decomposition of sugars and starches which are normally broken down by enzymatic action to monosaccharides and absorbed as such, so little monosaccharide, is thought to be available for decomposition to carbon dioxide.

Other gases normally present in small amounts that Kantor (48) gives are, methane and hydrogen from cellulose and occasionally indol, skatol, hydrogen sulfide and ammonia from the putrefaction of proteins. Carbon dioxide is by far the most readily absorbable gas, nitrogen the least; the other gases in order of their solubility, hydrogen sulphide, oxygen, hydrogen, and methane.

Gas absorption has been shown to follow the physical laws of diffusion by Dunn and Thomsen (49) and McIver (50). It has been definitely demonstrated that the walls of the intestine are permeable to gasses and that an active interchange takes place, tending to keep the gases on the two sides of the mucosa in equilibrium. Alvarez (29) points out in this connection that when nitrogen is diffused into the bowel, it is not reabsorbed, not only because of its low diffusion constant but also because the blood and tissues are already saturated.
with this gas.

Intestinal flatus then, is to be regarded as a resultant of gas production from food digestion, gas absorption from the intestine, and possible gas diffusion into the intestine, the last named factor according to most authors being probably unimportant in health. Since nitrogen is produced during digestion all of this gas is assumed to result either from the air originally swallowed or from the gas secreted from the blood in the process of equilibration. The amount of gas passed per rectum is estimated by Fries (51) at one liter per day. This is to be contrasted with the much greater amount absorbed. Tache (52) working with rabbits found that ten to twenty as much intestinal gas escapes by the lungs as by direct expulsion from the lower bowel. The figures for man are unavailable.

In order to produce distention then, there must be a breakdown in the gas balancing mechanism, either in the direction of increased production or diminished removal or of the two together.

Victor Bonney (53) in considering increased production states that there is no reason to suppose that an abdominal operation increases the amount of air swallowed, since for the first 24 hours the patient not only swallows very little but is often vomiting
what is swallowed. Post-operative distention has been said to be due to accumulation of swallowed air during the period when intestinal movement is reduced or absent. This theory Bonney reasons is not in accord with the fact that the distention rarely appears before the second day, by which time the stomach and upper intestine have recovered their mobility. Also if the distention was solely or chiefly the result of swallowing air it should be less marked the more the patient vomits, but the reverse is found to be the case.

In health the amount of gas introduced into the intestinal tract is found to be balanced by a mechanism of removal which is made up of two factors, absorption and peristaltic transport. "Absorption occurs when the pressure of any particular gas in the lumen exceeds its tension in the blood, but the amount absorbed depends upon the rate at which the blood can carry it away, that is, on the activity of the circulation thru the intestinal vessels. That the removal of gas by absorption can be rapid has been proven by experiments on animals and is also demonstrated by the fact that during the period of an abdominal operation the intestines may markedly collapse." (53). Bonney gives peristalsis credit of being an important aid to absorption by passing a locally produced gas to
other parts of the intestine, thereby making available for absorption a much larger area of surface than would otherwise be the case.

In considering the adaptive power of the intestinal wall and the abdominal wall, the intestinal gas is found not to constitute a single gaseous column, but a series of columns which is considered why the intestine, when opened, does not collapse through out its whole length like a punctured tire. Each column of gas is bounded not only by the walls of its compartment, but by the coils of intestine that surround it. If the amount of gas in any particular column is quickly increased the pressure in that compartment, will rise and adjacent compartments will be compressed, Bonney (53) explains, until the pressure in them rises to somewhere about that in the first compartment, and compartments further away will be less and less compressed as the force generated by the increased gas content of the first compartment is gradually used up in overcoming the resistance of the walls of the other compartments. Local distention of any segment of the intestine, unless compensated for by the withdrawal of gas elsewhere compresses and raises the pressure in neighboring coils, so that additional force is required to pass anything into them. It may be surmised, then, Bonney concludes that the colic often seen in post-operative
intestinal derangement, is due to the efforts of the intestinal muscle to force gas and fluid into the compressed coils.

Mclver (50) observed that when either oxygen or hydrogen was injected into isolated loops of small intestine, there was a progressive displacement of the absorbed gases, but in each case the greatest amount of new gas found was not oxygen or carbon dioxide, but nitrogen. He explains this phenomenon due not only to the fact that nitrogen occurs in the blood under a partial pressure of about four-fifths of an atmosphere (that is under a high tension as compared with that of oxygen which is about one-fifth of an atmosphere); but also because their entire amount is held only in the form of a loose solution in the blood plasma, whereas both the oxygen and carbon dioxide exist almost wholly in chemical combination with the hemoglobin of the red cell. In other words, the nitrogen is free to come out of the blood when conditions are proper. He then lists injuries or diseases of the spinal cord with involvement of the upper motor neuron fibers or the so-called neurologic ileus, toxic or post-operative ileus, severe intoxications as pneumonia and typhoid fever and some hysterical states regarded as being associated with auto inflation. He assumes that in the above named conditions that there is a sudden drop in intra-intes-
tinal pressure, due to some sort of interference with the normal nerve control of intestinal tonicity.

Kantor (48) draws an analogy between the sudden local drop in pressure in the lumen of the intestine and the phenomenon associated with a sudden general drop in atmospheric pressure as in cession workers and divers. He states that in a fatal case of the 'bends' it has been found in an analysis of gas from blood of the right heart that 80% was nitrogen and 20% carbon dioxide. He then reasons that intestinal flatulence may be regarded as a regular accompaniment of suddenly diminished intra-intestinal pressure, or in other words a kind of intestinal 'bends'. The recovery of gas resembling the composition of atmospheric air could, he states, be well explained on this basis since its chief constituent, nitrogen, would naturally predominate in any effort at striking a balance in gas tension between the blood already in equilibrium with the nitrogen of the outside air, and to the suddenly created partial vacuum within the intestinal lumen. He does not claim originality in this theory but a 'new-dress' of the ancient notion of 'pneumatosis' which he states was advocated a hundred years ago by Graves. A generation ago, he points out, Evans based his opinion on Ewald's observation that the gas of 'thyroid meteorism was over 90% nitrogen and concluded that the presence
of large proportions of this gas in the mixture of gases contained in any viscus does not necessarily imply that the origin of the gas is the atmospheric air.

Kantor (48) has shown that when for any reason the intestinal muscular tone is impaired, the lumen of the gut enlarges. This, he explains immediately lowers the intraintestinal pressure so that not only is gas absorption diminished but the possibility of gas excretion from the blood is enhanced. Soon then, a vicious cycle develops since the greater the accumulation of gas the greater the depression of muscular tone due to stretching. This is particularly true if some of the gas locally produced is neuro- or myo-toxic in nature. Kantor mentions the extreme depressing effect of certain amines derived from intestinal digestion. McIver (50) reports a marked but transient drop in blood pressure from the intestinal injection of highly absorbable hydrogen sulphide. The mechanism of intestinal atony, as has been described is best explained on a neurogenic basis, and accordingly when ever the intestinal distribution is involved, ileus with meteorism develops.

The part played in the production of distention by chemical changes is difficult to evaluate because
little knowledge seems to be available in the literature. The material obtained from an obstructed loop besides the gas has been described as a more or less corrupt substance identical with that material the patient is vomiting. It is usually ascribed to bacterial action, but Bonney believes, that in a large part it is the result of obscure bio-chemical actions since the involvement of organismal activity within the intestine is generally accompanied by signs of enteritis whereas in the case of obstruction the intestinal mucosa presents no such signs. However, both may play a part. The amount of gas that could be produced by bio-chemical or bacterial action would not be subject to the limitations, like gas swallowed or from gas derived from diffusion and could be continued after persistent vomiting and great pressure in the intestine had stopped the two latter sources of production.

In summary then it may be concluded that in the creation of distention, diminished removal of gas plays a greater part than does increased production; and of the causes of diminishment, deficient absorption is the most important.
The third mentioned factor that occurs as a result of an obstructed gut, will now be considered namely, the important factor of dehydration and associated blood changes, which as before cited, aggravates the distention further by disturbing the normal gaseous exchange.

The fact that patients with intestinal obstruction frequently give evidence of extreme loss of water was familiar to the early writers, Barlow (54), Brinton (55), and Leichtenstern (56). The last named author mentions dryness of the mucous membranes and skin, intense thirst, choleraic face and voice, sunken eyes, cramps in the calves of the legs and anuria. All these symptoms he cited as pointing to rapid withdrawal of water from the blood; and this he considers as due to vomiting and false sweating.

In 1920, MacCallum (57) showed that the blood chlorides are lowered and the alkali reserve increased. In 1922, Haden and Orr (58) showed that the blood chlorides were lowered also, and confirmed Hartwell's (59) experiments demonstrating the value of salt solution in prolonging the life of the animal. Haden and Orr in their earlier experiments advanced the theory that the reason the chlorides were lowered in the blood was that they are withdrawn from the plasma to neutralize some of the toxin absorbed from the obstruct-
ed intestine. They later modified their opinion to agree with Gamble and McIver (60) who believe the decrease in chlorides is due to their loss in the digestive secretions by vomiting, and which is the general opinion now held.

Gamble and McIver consider dehydration the result of the loss of sodium and chlorine accepting the general physiological concept that the volume of a body of fluid is sustained by its total ionic content. According to the conception which they use, the consequent depletion of the total ionic content of the plasma and of the interstitial body fluids, will be accompanied by an approximately parallel loss of water, with the result that a normal total ionic concentration tends to be sustained at the expense of the reduction of volume. Therefore it is the general concept now that dehydration cannot be repaired by the administration of fluid alone.

The extent of loss of water and electrolytes in the digestive secretions as given by Howntree indicates that, for an adult, the secretion of digestive fluids, taken together amounts to between 5 and 7 liters per day. This is two or three times the volume of the blood plasma, which is the immediate source of these secretions. Under normal conditions these secretions are absorbed at lower levels. Keith (62) has shown
that circumstances causing severe dehydration may reduce the blood volume by one third. The intracellular fluids are found to be drawn on, only to a slight extent since they contain relatively small amounts of electrolytes, so that the cell volume is defended.

The changes in the acid-base structure is thought by most authors to be as follows. First the most important change is a change in the reaction of the plasma, due to the alteration of the bicarbonate concentration and this in turn is referable to the relative amounts of the sodium and chloride ion withdrawn from the plasma. The sum of the acid factors in the plasma is found to be exactly equivalent to the total of fixed base. This equilibrium is maintained in the presence of changes in the individual factors, but the adjustibility of the bicarbonate ion concentration in the gastric juice, is found to be in large excess of the fixed base. So that withdrawal of these substances from the plasma may be expected to greatly increase the extent of which fixed base in the plasma is lowered by the chloride ion, and to this extent the bicarbonate ion concentration will be increased.

Camble and McIver (64) have shown that in pancreatic juice and in gall-bladder bile, in contrast to the gastric juice, that these secretions contain fixed base
in much greater amounts than chloride ion, so it is that loss of these secretions will tend to reduce the plasma bicarbonate. Gamble and McIver (65) verified this by finding a marked degree of acidosis as measured by bicarbonate reduction, in the blood plasma of a dog following construction of a pancreatic fistula which completely drained away the external secretion. In conclusion then, this variability of the plasma carbonate is understandable as the result of differences in the relative amounts of duodenal digestive secretions which have been lost; loss of the gastric secretions tending to produce alkalosis and loss of the duodenal secretions tending to produce acidosis.

These workers believe that beside the loss of gastric juice, pancreatic juice, and of bile, that it is quite likely that another factor is present: That is, the fluid derived from the intestinal mucosa. There is some doubt as to whether this fluid should be regarded as an irritatively stimulated digestive secretion or as an exudate; but it is found to contain about the same amount of fixed base and of chloride ion as does blood plasma and loss of this fluid causes reduction of the volume of blood plasma and of the interstitial body fluids but the plasma chloride is not altered. In considering the concentration of plasma chlorides, they point out that in obstructions, they remain at or near
the usual value, although the absolute loss of chloride ion from the plasma and interstitial fluids may have been extremely large. This, they illustrate by stating that when a large part of the contents of a flask of salt solution is poured out, the chloride concentration in the flask remains the same; so that even when the plasma chloride is found greatly reduced, it is not a measure of chloride loss.

The recent work of Gandel and Fine (66) shows that distention of the obstructed intestine in dogs causes and early and progressive loss of blood plasma. The average loss of blood plasma volume they give as 36.4% within four to six hours and 53% within twenty-four hours. A 55% loss of plasma they give as equivalent of 3.1% of the body weight, of this amount 0.7% they attribute to dehydration. The remainder or 2.38% as due, solely to the deleterious influence of the distention on the general circulation. A loss of 2.38% of the body weight in terms of plasma is sufficient to cause death in dogs. They believe that the magnitude of the plasma loss due to distention alone is sufficient to indicate that the need for immediate decompression of the gut must be accompanied by simultaneous administration of adequate quantities of plasma. The volume of plasma necessary to restore
the normal plasma volume is found to far exceed the amount commonly given to obstructed patients in clinical practice. The use of whole blood they state, has disadvantages which makes the use of plasma preferable. The administration of this plasma is necessary, long before evidence of shock is indicated by the level of the blood pressure, which they consider a misleading guide to patients condition and in its place recommend the hematocrit reading as dependable. The use of intravenous fluids and electrolytes, they consider necessary but not so vital as that of plasma. They do not explain the mechanism by which distention causes a loss of plasma, but are certain that it is not because of a loss of fluids into the intestines and peritoneum, unless strangulation supervenes.

In summary, then, of the blood changes, it is found that the disturbance in the acid-base balance, the change in plasma chlorides as well as volume is intimately associated and related to the loss of digestive secretions by vomiting. Other changes that are found include (a) an increase in the concentration of the plasma protein, so that the red blood count, hematocrit, and viscosity readings increase, (b) an increase in the non-protein-nitrogen content; the factors responsible for this change seem to be an increase in tissue destruction, due to the effects of
dehydration, and decreased kidney function and possibly due to the action of some circulating toxin. \((\text{67})\)

(c) Large reduction in the oxygen content in venous blood, increase in fibrin, decreased sedimentation rate and occurrence of hemolysis.

The fourth factor, that of mesenteric circulation will now be considered. It will now be recalled, as before stated, that distention acts to lessen absorption of gases by impairing the mesenteric circulation on the venous side and in addition, diffusion of gases from the blood into the bowel lumen is much increased. Then as distention increases, the pendulum movements of the gut stop first, then, the rhythmic contraction and the true peristaltic waves last, so that when the intra-intestinal pressure equals the venous pressure necrosis results \((\text{56})\). Gray \((\text{2})\) has demonstrated that distention of the intestine stretches the mesentery with consequent impeding of the flow through its veins, but as considerable distention already exists before this mechanism can operate, it is contributory to the final state only. This congestion of the mesenteric veins is present in all cases of mesenteric derangement in which the abdomen is reopened, and probably in those which recover without a second operation, according to Bonney \((\text{53})\). This congestion, he states, may be of

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vasomotor origin and later on, it is increased by traction on the mesentery. When distention reaches a sufficiently high grade, two other causes of retarded absorption become operative, namely, the effects of the intestinal gas pressure on the capillaries of the intestinal wall and the effect of the intraperitoneal pressure on the veins of the portal system. As the normal pressure in the capillaries is estimated at somewhere between ten and thirty millimeters mercury above atmospheric pressure and that in the mesenteric veins as being lower still, it is found that no very great increase in either the intestinal or intraperitoneal pressure would be required to slow the blood flow through either of them. It has been shown that not very marked interference with the venous return produces an outpouring of fluid into the lumen of the intestine.

If the main vein of a portion of the mesentery of an animal be experimentally ligated, the segment of intestine which it drains becomes distended with fluid, not with gas, according to Bonney (52).

Ochsner and Gage (67) in discussing the pathological changes with venous obstruction describe within the walls of the intestine edema and even infarction on the anti-mesenteric border and they cite Muller's finding in animals with a dynamic ileus, that
muscle cells of the intestinal muscle were hydric, elongated, enlarged and swollen.

Gray (2) in a clinical analogy to the experimental ligature of the intestinal veins, with arteries intact, describes the following, (a) severe stimulation of the Pacinian corpuscles to lead to shock and initial immobility, (b) as soon as the pressure in the obstructed vein exceeds that in the intestinal lumen, gases pass from the blood into the viscous by osmotic pressure and progressive inertia and distention begin. (c) When the pressure of the bowel exceeds that in the veins, gases will pass by osmosis into the blood stream and these exhaled by the lungs will give a fecal odor to the breath. The progressive extension of mesenteric tension so induced leads to a proportionate spread of venous engorgement. (d) Then fluid from the veins passes not only into the peritoneum but into the intestinal lumen. In time then, absorption of fluids and gases from the bowel accentuates the initial shock with a profound toxemia. Such a process if unrelied is essentially progressive; so that when the gastric musculature is involved and failure of the pyloric sphincter occurs the intestinal contents obtain access to the stomach and the result is stercoral vomiting, condition is now analogous to that seen in acute dilatation of the stomach. (e) Normally the large bowel is adapted to tolerate
a degree of intermittent obstruction, created by
the voluntary control of the anus. The small in-
testine is protected from this by the ilio-colic
sphincter. This has been shown by Elliott to con-
tract as the result of sympathetic stimulation,
anemia and adrenaline. That this function of the
sphincter is important is shown by the changes in
the ileum following colectomy, changes which are
hardly noticeable after a year, when the same oper-
ation is performed by implanting the ilio-colic sphincter into the sigmoid and so preserving its func-
tion. Then, then the ilio-colic sphincter is closed
in acute ileus from sympathetic stimulation, it will
in common with the rest of the intestine, be paralized
by venous engorgement. The free access of the infec-
tive contents of the colon to the small bowel may be
one of the reasons for the highly increased 'infectivity'
of the contents when intestinal paralysis follows venous
obstruction. (f) Increase in intra-abdominal pressure
by further compression of the large veins accentuates
venous stasis. Accordingly paralytic ileus tends to oc-
cur sooner in subjects who are stout or have firm abdomi-
inal walls than in those with flabby muscles. The marked
distention which sometimes follows hysterotomy is many
times, without distress or constitutional symptoms.

Scott and Wangensteen (69) observed that loss of
blood into the infected gut constituted an important factor in causing shock. They then ran experiments to determine whether intestinal distention per se affected the circulation (70). They studied the effect of intestinal distention upon venous pressure in the lower extremities, the circulation time, and the inferior vena cava as well as the portal venous pressure were determined. They found that experimental intestinal distention of anesthetized dogs produced an elevation of intra-peritoneal pressure, which did not increase at the same rate as the intra-luminal pressure. Also venous pressure in the lower extremities increased with increasing intra-peritoneal pressure. At the same time the rate of blood flow from the lower extremities to the carotid was slower. The shock eventually produced by prolonged intestinal distention was relieved by deflation, blood transfusion and steep Trendelenburg position. They also found that opening the abdomen or exteriorization of segments of bowel in these animals caused a fall in the intra-luminal pressure.

So in summary, the obstruction of the mesenteric circulation is found to be a vital factor in the results of ileus.

The fifth and final factor now comes into play, that of the elaborated toxin. A discussion of this factor
leads into the mechanism of the cause of death. It is found that there has been many theories to explain the death that follows intestinal obstruction.

The belief that a vaso-motor reflex was responsible for the symptom complex of obstruction was among the older explanations. The advocates of this theory were impressed by the pain and other symptoms of nervous irritation shown by the patient and they attributed these symptoms and the collapse to a reflex action upon the vital centers in the central nervous system resulting from injury to the nerve endings and important plexuses of the splanchnic area (70). Another old theory was the belief that a bacteremia was responsible for the illness and death. This was disproved by McClure (71) in 1907.

Of the theories that are advanced at the present time, the belief that the disease is a result of absorption of a toxin from the bowel will be discussed first. Cooper (72) gives a comprehensive analysis and bibliography on this theory. The efforts of most authors have been directed first toward proving that the disease is caused by a toxin and next toward determining the chemical nature of this toxin, its source and mode of absorption. The literature is found to be complicated on the subject since the cause and symptoms for the various types of obstruction have not been clearly separated.
Murphy and Brooks (73) have drawn off the intestinal contents that collect above an obstruction and injected it intravenously into normal animals. These animals were found to die a death resembling that of intestinal obstruction. This evidence seems to constitute the major direct evidence that the absorption of toxin is responsible. Wangensteen and Church (74) have shown that at times even the normal contents of the small gut are toxic. Most authors agree that a toxin is formed but the nature of this toxin has not been clearly worked out.

Nesbitt (75) in 1916 reported the finding of choline and neurine. Murphy and Brooks thought the toxic substances resembled a promaine, (4). Whipple (76) and co-workers tried to determine the chemical nature of toxic substances found in their closed loop experiments. They concluded that it was a primary heteroproleose, having the following characteristics: it resists autolysis and pancreatic and tryptic digestion, it is thrown out of solution by five volumes of alcohol or by half saturation with ammonium sulphate, it is readily soluble in water, it is not injured by boiling, and it is not removed by dialysis. In 1919 Dragstedt (77) and his co-workers came to the conclusion that the toxic amines were formed by the action of
bacteria on amino acids.

Wale (78) in studies of the pharmacological action of histimine, have noted particularly its depressor action on the systemic blood pressure due to its effect on the capillaries. Cannon (79) has considered that some histimine like substance is the cause of traumatic shock. The presence of histimine then, since shock like symptoms in the terminal stage are met with, was considered to be a cause. Neakins and Harrington (80) after studying its absorption from the gut decided that it was probably not an active agent in causing intestinal intoxication. Gerard (81) found that histimine was present in the fluid from seven out of eight intestinal loops and that the contents of the one loop that did not show histimine did not produce toxic symptoms on injection. This author also showed the presence of a combined histimine derivative in obstructed loop contents. Wangensteen and Souchs (82) did not find any absorption of histimine from a simple obstruction of the small intestine of two days standing; they considered that absorption of histimine was slight even from strangulated but still viable segments. So the question of the exact nature of the toxin is not yet clear.

Where the toxin comes from has been the cause of much experimental work. Harper (7) in 1914 showed that
the presence of bile and pancreatic juice did not alter the picture in cases of obstruction. Hartwell and Sognet (83) showed that if animals are starved for a considerable time before the production of ileus, the course and duration of the disease was not essentially changed, thus proving that food does not play an important part.

Stone, Bernheim and Whipple (84) believed that under normal conditions of obstruction the mucosa of the duodenum produces a toxic secretion. They found that the contents of an obstructed loop were not toxic if they destroyed the mucosa of the duodenum with sodium floride, before producing the obstruction. All work lead them to believe that the toxin was formed in the mucosa of the duodenum. They concluded that it was a protease and that the animal could be partially immunized against it.

It is known that the intestinal fluid contains a large number and variety of bacteria. That bacteria produced the toxic material has occupied a large space in the literature. Murphy and Vincent (85) concluded from their studies that bacteria and exotoxins were responsible for the toxemia, because they found that the small quantities of the intestinal contents, which had been passed thru a Berkfeld filter and injected intravenously, were not toxic.
Dragstedt (86) and coworkers found that if a loop in the upper intestinal tract were isolated with its blood supply intact, and were then washed, the continuity of the gastro-intestinal tract reestablished, and the loop dropped back into the peritoneal cavity, half the animals so operated upon died of general peritonitis. The other half, after a lapse of number of days, it was found, that the loop had been sterilized by the action of the peritoneal fluid. If, at this time, the ends of the loops were closed, the animals lived almost indefinitely and death did not follow even when obstruction to the mesenteric circulation was produced. These authors consider these experiments, proof that bacteria were responsible for the disease.

Much attention has been focused on the kind of bacteria present. Cannon (87) concluded that it was always associated with a proteolytic flora. It is generally agreed, according to McIver (86) that the normal intestinal flora include a large number of anaerobic organisms. In 1926, Williams (89) advanced the theory that the Welch bacillus was the organism particularly responsible for the toxemia. In support of this theory he cited certain points of similarity between the clinical picture of intestinal ileus and that of gas bacillus infection, also the fact that he had found 3. Welch in large numbers in the contents of
obstructed intestines and in the vomitus from such cases. On the basis of this theory he gave B. welchi antitoxin to two heterogenous groups of patients suffering from intestinal obstruction or peritonitis with an apparently favorable effect upon the course of the disease and with a lowering of the mortality rate.

Bower and Clark (90) reported a series of twenty-five cases of intestinal obstruction or peritonitis treated with B. welchi antitoxin. They believed that its administration had favorably influenced the course of the two diseases. However, Molver (88) and coworkers were not able to find sufficient evidence that B. welchi played an important role in the production of toxemia. They state that B. welchi normally found in the gastrointestinal tract are of low virulence. They base this on the fact that of a large number of intestines, in which the intestine is drained, through an abdominal wound and the rarity with which gas bacillus infection of the abdominal wound takes place, in spite of the fact that B. welchi is frequently cultured from the draining wound. While the question as to whether anaerobic or aerobic are responsible for the toxemia has not been settled, the experiments of Murphy and Brooks (73) furnish evidence that the toxemia in experimentally obstructed loops is due to bacterial action and not to the toxic secretion of the mucosa of the intestine.
In considering the absorption of toxin, it may be considered an established fact that no absorption of a toxic substance can take place through normal mucosa. If the most virulent toxic fluid from an obstructed loop be placed in the normal intestine of another animal, it is found that no symptoms follow. The question is, then, what changes take place under conditions of intestinal obstruction which permit the poison to be absorbed. The most reasonable explanation, according to McIver (88) is that the mucosa is so damaged or destroyed, that it is permeable to the toxin from the lumen, or that after the barrier of epithelial cells is broken down the toxin may be actually formed in the intestinal wall itself by the action of bacteria or autolytic changes. The degenerative changes taking place are usually attributed to the fact that the circulation of the bowel is damaged by distention as has been shown. Hartwell (91) and coworkers state that the intraintestinal pressure influences absorption in two ways: indirectly, by interfering with the circulation, and, directly by forcing the toxin into the absorbing channels. Burget (92) found that animals with closed intestinal loops could be kept alive almost indefinitely if over-distention of the loops were prevented by aspirating the contents.

It is found, provided the toxin has passed the barrier imposed by the mucosa, that there are three ways in which general dissemination may take place, by blood stream,
by lymphatics, or by diffusing into the general peritoneal cavity and be absorbed from that source. (88) If, via the blood stream, their seems to be no important evidence either for or against it. Carlson, Lynch and Wangensteen (92) showed that the blood of animals, dying from intestinal obstruction, is not toxic when injected into another animal. Whipple (94), however, states that the blood of animals poisoned in another manner, may also be non-toxic when transferred into another laboratory animal. If via the lymph channels, the adherents of the theory believe that their is an absorption of toxin and that absorption occurs by way of the blood stream and by way of the lymphatics. Murphy and Vincent (85) have stressed the importance of the latter. Morton (95) using colloidal silver, which has a selective staining action for lymphatics, was not able to demonstrate by staining of the thoracic lymphatics, any evidence of absorption unless an actual perforation of a loop had occurred.

Wilkie (96) in studying the peritoneal cavity, as a source of absorption, found no toxic substance in the peritoneal fluid by injecting it intravenously into normal animals, even with large quantities. Foster and Hauser (97) concluded that placing short strangulated loops in a rubber bag in order to isolate them from the peritoneal cavity prolonged the life of the animal.
So in summary of the theory that a toxin is the cause of death, it may be said that there is no conclusive evidence on the mechanism of absorption, or on the pathways by which dissemination of the toxin occurs; but even so, the theory is considered by many authorities to be one of the best explanations.

Since dehydration appears as a frequent and severe complication, it has been considered as the cause of death. The adherents of this theory do not believe that death in all types of intestinal obstruction can be explained on this basis, however. Hartwell and Hoguet (91) showed that animals dying from simple obstruction seldom showed any change in the intestine above the obstruction. This led them to doubt that death resulted from the absorption of a toxin and because of the large amounts of fluid lost by animals in vomiting, and the rapid restoration of fluids by using physiological saline, caused them to advance this theory. Opponents of this theory cite Harper's (7) experiments in 1914, who measured the water content of liver, kidney and other tissues under normal conditions and following fasting, salivation by pilocarpine, and duodenal obstruction, he found the water loss to be about 10% in all three conditions. The fasting and salivated animals showed no signs of disability while the animals with duodenal obstruction died. This experiment
may be criticized on the basis of the fact that the water content of parenchymatous tissues do not furnish an accurate estimate of the degree of dehydration.

Foster (97) considered that the time elapsing before death was too short for the development of a toxemia and Wilke (96) considered that the most outstanding finding in patients dying of obstruction was the accumulation of fluid throughout the splanchnic area, in the engorged blood vessels, in the intestines above the obstruction and in the peritoneal cavity itself. This drainage of fluid into the splanchnic area then, decreases the circulatory blood volume, thus lowering the efficiency of the circulation in general, and of the cerebral circulation in particular.

Herrin and Meek (98) and Taylor (99) report that they have prolonged the survival time of dogs dying from distention of the intestinal loop, by denervation of the adjoining mesentery. The nervous element in traumatic shock has been studied by Freeman(100) and since Moon (101) has compared the prelethal stage of intestinal obstruction to shock, Fine, Rosenfeld and Genasel (102) have studied the possibilities. They found that the survival time in cats, with obstruction and gaseous distention of the small intestine, to be inversely proportional to the level of the pressure in the lumen of the bowel and preliminary exclusion of the extrinsic nerve supply of the gastro-intestinal tract did not influence the
survival time of such animals. Also, the fluid accum­ulation in the intestinal lumen, bowel wall, and peri­toneal cavity in these animals, was not sufficient to account for their rapid death.

In summary then, it is seen that all the true factors as to the cause of death are not known; but, it is by the study of these factors, that a rational basis of treatment may be worked out.
TREATMENT

Since the decline in the incidence of typhoid fever, intestinal obstruction according to Wise (103) has become the most lethal affection of the small bowel. Its local treatment he states, consists in reducing the distention, proximal to the lesion, then locating and relieving the obstruction and excising any gangrenous tissue devitalized by disturbance of its blood supply.

Morgan (104) bases the intelligent treatment of ileus on two factors. First, the bowel damage and complication for which the ileus was directly responsible and secondly, the body chemical changes known to take place, Continued vomiting, dehydration, alkalosis and in late cases uremia, must be dealt with.

Ten essentials necessary to attain a logical treatment of the underlying pathology rather than treating the symptomatology presented, as given by Stout (4) are: (a) proper preoperative preparation, (b) early recognition of the signs and symptoms, and the immediate beginning of therapy, (c) proper sedation, (d) drugs inhibiting the toxic process, (e) prevention of toxic absorption, (f) facilities for the control of intraluminal pressure, (g) improvement of gastro-intestinal tonicity by drugs, (h) the improvement of circulation in the splanchnic area, (i) and the prevention of dehydration. These
measures will now be considered in more detail.

In considering the proper preoperative preparation Stout (4) considers the mental preparation important. The many fears of anticipation harbored by most patients, he believes, should be carefully dealt with as functional ileus is more often seen in the highly nervous types. Engle (22) states that the characteristics of the individual patient, unquestionably have something to do with post-operative distention. This factor, he cites as almost impossible to remedy and must be accepted as a case of distention diathesis. He considers the preoperative care of patients a deciding factor, however, he goes on to point out that the emergencies, as a rule, providing no peritonitis is present, do better than long prepared cases, except in preparation of cases for resection of the bowel. He recommends stopping all fluids by mouth ten to twelve hours before operation, as a preventive measure.

As to the purging of the gut, pre-operatively, Alvarez (106) has shown that after purging, there is a refractory period during which time there is an inhibition to peristalsis, so that the use of cathartics preceding laparotomy should be avoided. Also, he points out that some of the purgatives owe their effects to the fact that they are irritant poisons that must be removed quickly from the body, an others act by inter-
ferring with intestinal absorption and upsetting the balance of salts. In either case they bring out pathological conditions and the body is weakened, not strengthened.

Wilke (96) believes that there will be no improvement in the pre and post operative treatment that will ever make good the damage that is done in early management of the cases. The discovery of any degree of peritonitis, whatever in the performance of an abdominal section should, he believes, be the signal to anticipate ileus by starting treatment at once. Potter (107) uses successfully routine prophylactic doses of pitressin and reports a marked decrease in the incidence. His findings will be subsequently discussed in more detail, when considering the merits of pitressin.

As to the preoperative use of dextrose, there is some division of opinion. Gechner (108) states as follows: "we do not know why glucose inhibits intestinal activity. We were given this lead largely by Carlson's work, who some years ago showed that glucose inhibited action of the stomach and we wondered whether this was true, also, of the intestine, and found it was. Probably the effect is directly on the muscle. This may be of extreme importance, clinically. However, we have not been able to observe many of these cases clinically, and it takes thousands of cases to prove anything. We
had noticed that patients with gallbladder disease had more postoperative distention than other cases, but that it was probably due to handling of the viscera in the upper abdomen. In these cases, we had been using large doses of dextrose, because of liver damage. Recently, we have fortified the dextrose with insulin and have not had this distention, so we believe that the former effect was due to dextrose." According to Quigley (109) dextrose does not exert an inhibitory effect upon bowel motility. Hughson and Scarff (110) state that hypertonic dextrose like hypertonic sodium chloride augments motility.

In considering, proper sedation, Stout (4) points out that these patients are apprehensive, irritable, and later excitable from their toxemia and that they suffer from insomnia, restlessness and distress from distention. If the latter is relieved, they have very little pain and he believes there is no occasion for the promiscuous use of morphia. Rest, he states, is imperative and may usually be provided by the use of bromides, as a basic sedative and the more potent barbituric acid salts as are required to handle the individual case.

The widespread use and general employment of morphine seems to warrant special attention. Alonzo Clark in 1879, according to Dragsteat (111), was an advocate of deep morphinization in the treatment of peritonitis, and advised a semi-narcoosis to insure, as he thought, a complete bowel
rest. He gives credit to Graves in 1821, as the first
to use clinically large doses of opiate in the treatment
of peritonitis, and to Stokes in 1822, for the first
publication on the subject. It came to be called the
Alonzo Clark treatment, however, and modifications of
his regime constitutes the accepted therapy, for many
years as shown by the textbooks of Austin, Kelly and
others. Dragstedt states this type of therapy came in
time to be challenged by a number of clinicians not on
the basis of observed therapeutic results but rather on
the notion that the drug induced ileus, was a two-edged
sword, splinting the bowel to restrict the spread of in-
feccion on one hand, but intensifying the toxemia spec-
ifically related to the ileus on the other. Although
numerous experimental studies on the action of morphine
upon the intestine has been made, Dragstedt continues,
conflicting results had been obtained, so that in spite
of the fact, that Poe's studies in 1900, indicated that
morphine had a stimulating action on the intestine, the
well-recognized constipating action of morphine served
largely to nullify such findings and to strengthen the
belief that morphine was essentially depressant. It wasn't
found until the work of Plant and Miller in 1926, that
both in man and animals, the characteristic effect is to
increase motility. That the action of morphine is stim-
ulat rather than depressant to the intestinal musculature,
Dragstedt (111) points out, is now generally recognized as Plant and Miller's observations, have been confirmed by many workers, both for the normal intestine and when obstruction and peritonitis are present.

Assuming a non-specific effect of the morphine regime, it is interesting to note the changing conceptions of the mechanism whereby this is brought about. With the recognition that morphine does not splint the bowel, came the suggestion, according to Dragstedt, that it helped to overcome the effects of the ileus, secondary, to the peritonitis, by encouraging transport of a toxic bowel content from the highly absorptive small intestine to the less absorptive colon. This is not the only way in which it could act, Dragstedt continues, and there is also some evidence that the motility induced by morphine is not effective in the transport of the bowel content. Circulation through the bowel is greatly facilitated by the contractions and motility of the intestines. Conversely it is decreased in the parietic intestine and still further decreased by the distention. The indirect effect of morphine by improving the circulation through the bowel and thus promoting tone and motility in the intestine is the probable reason for whatever beneficial effect it may have in peritonitis, according to Dragstedt.

Kensing (112) states that morphine apparently diminishes the secretion of digestive juices. This decrease
in the amount of intestinal secretion, he explains, is most likely due to an inhibitory action of morphine upon the secretory demands, as to its stimulating action upon the tonus and segmentation movements, an effect he believes, favors the reabsorption of secreted fluids. Any deleterious effect that might result from the tendency of morphine to present hypertonus of the gastro-intestinal sphincters should, Mensing believes, be overcome by tubal deflations. Atropine, he warns, should not be added to the morphine because it depresses the accelerator vagus and pelvic nerves.

The prevention of toxic absorption from the bowel Stout (4) believes, is theoretically accomplished by the introduction of hypertonic saline into the gut to hinder water absorption and administration of sufficient fluids parentally, to cause an active secretion into the intestinal lumen and at the same time, providing tubal drainage, of the gastro-intestinal contents.

Hughson and Scarff (113) have shown that the injection of hypertonic salt, intravenously, decreases the absorption rate of water from an isolated loop of intestine. Carlson and Wangensteen (114), Ochsner, Gage and Cutlin (115) have shown that the administration of salt stimulates both the intestine tone and peristalsis.
They believe that sodium chloride acts directly on the bowel. Dreyer and Tsung (116) have also noted in experimental animals that hypertonic solutions of sodium chloride cause an increase in intestinal movements. No effect was noted by these authors when an isotonic solution was used.

Facilities for the control of intra-intestinal pressure: Wood (117) states that the evolution of the successful method of tubal drainage by Wangensteen has probably saved more lives than the introduction of liver therapy in anemia. The most recent improvement of this method is the use of a double lumen tube which may be passed by mouth to the ileo-cecal valve. Patients seriously ill from toxemia and dehydration are found not to stand exploration and a simple enterostomy may fail to alleviate the condition and is found to have many disadvantages.

The double lumen tube was first devised by Miller and Abbott (118, 119). These men worked with the tube studying the secretion and absorption of the normal small intestine and with Randim first suggested its use in the treatment and diagnosis in 1938. The tube as described by these authors, is ten feet in length and 16 French in diameter. A rubber septum extends throughout its length making it into a double lumen tube. The inflation tube opens into a soft rubber balloon; the suction tube has
several openings at its distal end terminates in a metal tip. When the tube has passed the pylorus and the balloon is inflated it will be carried, by peristalsis down the entire length of the intestine. As it traverses the intestinal tract, suction is applied which removes fluid and gas from each distended loop. It is possible for this tube to deflate the entire gastro-intestinal tract in patients with intestinal obstruction from whatever cause, mechanical or paralytic according to Miller and Abbott (118, 119). Every effort should be made to get the tube into the duodenum by natural means according to Wise (120) for it is often impossible to move a sick patient to the fluoroscope and manipulation, with a distended abdomen is by no means easy. Once the tube is in the duodenum, thirty cubic centimeters of air is injected into the balloon, the balloon tube clamped, and constant Wangansteen suction applied to the suction tube. Each hour six inches more of tube is inserted until the eight foot mark is reached. The suction tube is irrigated each hour with 20cc. of water.

In cases of 'ileus duplex' the localized inhibition of function of pelvic colon results in a localized obstruction which can be overcome by passing a small colon tube through a proctoscope, well into the sigmoid. Wangansteen suction may also be used here.
Mensing (112) points out that suction siphonage unless accompanied by the performance of a laparotomy apparently does not materially lower the intra-abdominal pressure even though it deflates the small bowel because the tone of the diaphragm and abdominal muscles compensates for the diminution in the size of the intestine.

Disadvantages of the nasal tube as given by Van Bueren and Smith (121), are: (a) A few patients will not tolerate it. (b) Some patients vomit the tube repeatedly. (c) Ulceration in the esophagus has been noted at post-mortem in some cases (d) one case with a deviated septum, had an ulceration of the mucous membrane on the septum and an adhesion to the lateral nasal wall formed. Nose and throat consultants considered the condition not curable without a septal resection. (e) Cases of alkalosis from prolonged use of the tube have been reported. (f) It may relieve symptoms preoperatively temporarily, while a loop of gut with damaged blood supply remains in the abdomen.

In considering enterostomy, Mensing (112) states it is never indicated in the treatment of paralytic ileus of general peritonitis. Van Bueren and Smith (121) state that the nasal tube has replaced enterostomy in many of their cases. They noted, however, in their series of cases that a primary enterostomy was
more effective than a secondary one, and an ostomy in the more viable jejunum seems to function better than one in the more involved ileum. They stress that the pathologic indications for enterostomy require astute judgement and that enterostomy in paralytic ileus is relatively ineffective. They give the mortality following enterostomies performed upon thirteen cases of paralytic ileus as 90%. Stout (4) states that the operation of enterostomy is very rarely necessary and that tubal drainage gives better results and is without the inherent disadvantages of the operation.

Van Beuren and Smith (121) conclude that enterostomy as a treatment for acute ileus has been used in only a small percent of the total number of cases. It seems to have been reserved as a last resort in the cases in which the patient is sickest and the condition most advanced. They state there is evidence suggesting that this procedure has been effective in lowering the mortality rate but it has had apparently little influence on the general mortality rate.

Another method recently advocated is that of breathing 95% oxygen for the relief of gaseous distention. The laboratory studies of Rosenfield and Fine (122) which provided the basis for clinical application of the method, demonstrated the capacity of 95% oxygen to dilate a closed loop of small intestine distended with
nitrogen, which in man as we have seen, forms a major constituent of the distending gases. The mechanism by which the oxygen accomplishes this result consists in the exclusion of nitrogen from the inspired air. According to the law of gases, the diffusion of any gas through a semi-permeable membrane is found to be proportional to the difference between its partial pressure on the two sides of the membrane. Inhalation of pure oxygen necessarily, Rosenfield explains, reduces the pressure of nitrogen in the lungs towards zero, so that the nitrogen in the blood diffuses into the expired air; and by the same mechanism the resulting reduced partial pressure of nitrogen in the blood allows this gas to diffuse more rapidly from any body cavity or tissue space into the blood and so expelled through the lungs. Rosenfield makes it clear that oxygen per se has no direct effect on the diffusion process. Its virtue, he states, lies only in the fact that, when properly used, it is a convenient responsible gas which prevents nitrogen from being inhaled.

Rosenfield showed that by causing an animal to breathe pure oxygen instead of air, the volume of nitrogen in a closed loop of small intestine distended with gas can be reduced in twenty-four hours to about 40% of its original volume. This is in contrast to an average variation of only 10% of the original gas volume.
when the animal breathes room air for the same period of time. The pathologic changes in ileus were found primarily referable to the effect of increased intra-luminal tension rather than gas volume.

Fine, Hermanson and Trehling (123) cite clinical cases where the benefit of breathing oxygen is observed. The corresponding satisfactory results lead them to believe that a high average of successes can be obtained with the proper technique.

Binger (124) and others have reported toxic effects from the prolonged breathing of pure oxygen. But Fine (123) points out that this does not constitute a valid objection for the following reasons: (a) The nursing requirements of patients make interruptions in the administration of oxygen necessary and such interruptions average one-half hour in each four to eight hours and constitute a sufficient factor of safety. Oxygen has been given in this manner as long as thirty-five hours in a number of cases without any suggestion of oxygen poisoning. (b) Should future experience fail to sustain this belief, an oxygen-helium mixture could be supplied which would eliminate this objection.

The simplest type of distention for which the method may be used, Fine explains, are the post-operative functional types and those associated with pneumonia or cardiac disease. In addition the distention of peritonitis, he
believes, is a particularly appropriate type for its application. Brown (125) considers the disturbed function of the gastro-intestinal tract in peritonitis to be as inimical to recovery as the peritonitis itself and if overdistention can be avoided or minimized by an agent which is more directly effective than those in current use, we may be in a position to treat peritonitis with much more success.

Fine (123) et al use the intra-gastric catheter routinely during the administration of oxygen in order to prevent the entrance of air or oxygen into the duodenum. They found that in no instance was it possible to attribute any substantial portion of the deflation accomplished to the amount of gas discharged to the catheter. They state that a suction tube cannot answer the needs of a case in which more than one level of obstruction exists, such as may occur with peritonitis. In this instance they state, as in any closed loop obstruction, the commonest example of which is obstruction of the colon, the choice rests between an enterostomy or 95% oxygen administered through a helmet. They admit that, to the extent to which a suction tube, like an enterostomy tube removes fluid directly, it is probably superior to the oxygen method, which can only do so indirectly by improving the absorptive capacity of the bowel after partial deflation. A combination of both methods then applied simultaneously should provide maximum
benefit which each has to offer.

In the long standing obstruction the bowel musculature is occasionally so paralyzed from over distension that enterostomy fails to result in prompt evacuation. Cheever (126) has eviscerated the gut and expressed the retained fluids and gas at the time of enterostomy. This, many authorities believe, is a heroic procedure and accompanied with danger. Fine (123) concludes concerning the relative merits of rapid, versus slow decompression, a middle ground may be taken by performing the enterostomy and supplementing it with the administration of 95% oxygen. If the enterostomy fails to function, then the oxygen tent will at once initiate gradual deflation and permit a more rapid recovery of muscle tone, than might occur spontaneously. If the enterostomy functions too well, so that the effects of too sudden decompression, Fine states that the tube can be clamped and gradual deflation, accomplished more gradually by the oxygen tent, and the tube opened at a less critical period thereafter.

To sum up then, decompression of the distended bowel that has not been stretched beyond its capacity to recover, results in the following physiological effects according to Mensing (112). (a) Improvement in the splanchnic circulation and increase in the circulating blood volume. (b) Restoration of the intestinal tone,
mobility and gradients. (c) Acceleration of absorption of intra-intestinal gases. (d) Acceleration of absorption of intestinal fluids. (e) Cessation of the excessive 'paralytic secretion' of intestinal juices, and (f) Diminution in the amount of edema and spasm at the site of obstruction.

It is the opinion of most authors that the post-operative hypodermic administration of such peristaltic drugs as pitressin and prostigmine combined with the employment of colon irrigations, lavages, hot packs or stupes and enemata have clinically improved peristalsis, and that their use should be persisted in until deflation and successful response are consistently obtained.

Kirklin and Seedorf (136) of the Mayo foundation state that their search up to date has revealed no other drug comparable to pitressin in its dependable results.

The separation of pituitrin into pitressin, the pressor factor and pitosin the excitotoxic factor, was announced by Kamm et al (127) in 1927. Pitressin is now much used for the control of intestinal distention and ileus, prophylactically and therapeutically. Potter and Mueller (126) found pitressin specific for their purpose and adopted it for routine use and further clinical study. One hundred patients, operated on for acute appendicitis or for biliary tract disease, where significant distention might be antici-
pated in 49 per-cent, were given pitressin every four hours for eight to twelve injections. Where general anesthesia was used, the initial dose of pitressin was given before operation to prevent ballooning of the intestine and to facilitate operative manipulation. When spinal anesthesia was used, the anesthetic prevented distention during operation and pitressin was started directly following operation. Commenting on the results of this clinical investigation, Potter and Mueller stated: "In 92 out of 100 cases there was no evident distention...In no case, save that which there existed a mechanical obstruction, was there any outward evidence of increased peristalsis. The action of the drug, as in our previous series, appeared to be that of merely maintaining tone."

Early paralytic ileus may respond to nonsurgical treatment based on intubation, pitressin therapy and supportive measures. Rensing (112) pointed out that pitressin augments intestinal tone and increases retention of fluids by the tissues in these cases. Prophylactic use of pitressin at the time of operation and repeated every two to four hours as indicated, in 200 cases of acute appendicitis was reported by Potter (129). In this series there was no death from functional ileus and distention was a serious problem in only one instance; mortality was 4.5 per cent. Acute diffuse peritonitis was present in 25 patients and mortality in this group was 12 per-cent. This may be
constrasted to a mortality rate of 35 per-cent in previous cases of acute diffuse peritonitis in which pitressin prophylaxis of distention had not been employed.

Administration of pitressin at the time general anesthesia was started, with repetition of injections at four hour intervals as indicated, was employed by Black (130) in 60 abdominal surgical cases to prevent distention. Results were favorable and post-operative gas pains were less frequent than usual. When pitressin was given, the small gut as seen at operation appeared to become squeezed dry and hung in a small, limp cluster that could be easily packed out of the operative field and did not interfere with abdominal closure. There was almost no change in the large bowel.

Potter (107) states that a method which has proved a safe and efficient means of minimizing the incidence of paralytic ileus is the prophylactic use of pitressin. The initial dose must be given in the presence of a nondistended intestine and should be continued at regular intervals. In early appendicitis, without peritonitis, eight doses of pitressin he believes, sufficient. In biliary cases 12 doses may be required. No cathartics or enemas are given until pitressin is discontinued; following the final dose a colon irrigation may be ordered.

Discussing surgery of the stomach Kline (131) points out the usefulness of pitressin as a post-operative measure
in the management of perforated gastric ulcer and sub-total gastric resection. In addition to routine post-operative measures for perforated ulcer, pitressin is given every two hours for three or more doses. "This restablishes normal peristalsis, does not of itself spread infection, but empties the gut, promotes necessary circulation, prevents the more deadly ileus, absorption of toxic materials, necrosis, and vomiting."

The use of pitressin in laparotomies was studied by Seld (132). He was also of the opinion that the routine use after operation tends to reduce the incidence of distention and discomfort following operation.

Frazier (133) concluded that pitressin exerts a stimulating effect on gastric and intestinal musculature under normal conditions. Since review of thirty years worldwide clinical experience with pituitary preparations for the relief of distention revealed a controversial issue, Frazier studied the problem on the surgical service at the hospital of the university of Pennsylvania. Pitressin was given in doses of 0.5-1.0 cc. every four hours for as long as 86 hours after operation. The series included 24 patients to whom pitressin was given routinely after operation, without waiting for the symptoms of distention to develop; 66 to whom it was given post-operatively; 47 to whom it was given after operation for relief of gas pains; 4 to whom it was
administered to control the obstinate distention resulting from ileus unrelated to operative trauma or infection; and 15 with widespread peritonitis. The results were as follows:

<table>
<thead>
<tr>
<th>Degree of Distention</th>
<th>No.</th>
<th>Improved</th>
<th>Unimproved</th>
<th>Made Worse</th>
<th>Patients who passed gas</th>
<th>Patients who passed feces</th>
<th>Inc. in Peristalsis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor</td>
<td>9</td>
<td>6</td>
<td>3</td>
<td>0</td>
<td>5</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Moderate</td>
<td>38</td>
<td>37</td>
<td>1</td>
<td>0</td>
<td>26</td>
<td>10</td>
<td>10</td>
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<tr>
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<td>19</td>
<td>14</td>
<td>5</td>
<td>0</td>
<td>13</td>
<td>6</td>
<td>8</td>
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For Gas Pains

<table>
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<tr>
<th>Degree of Distention</th>
<th>No.</th>
<th>Improved</th>
<th>Unimproved</th>
<th>Made Worse</th>
<th>Patients who passed gas</th>
<th>Patients who passed feces</th>
<th>Inc. in Peristalsis</th>
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</thead>
<tbody>
<tr>
<td>Minor</td>
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<td>7</td>
<td>0</td>
<td>0</td>
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<td>not recorded</td>
<td>not recorded</td>
</tr>
<tr>
<td>Moderate</td>
<td>25</td>
<td>21</td>
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<td>0</td>
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<tr>
<td>Marked</td>
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<td>14</td>
<td>1</td>
<td>0</td>
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<td>not recorded</td>
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</table>

Cases With Widespread Peritonitis

<table>
<thead>
<tr>
<th>Outcome</th>
<th>No.</th>
<th>Improved</th>
<th>Unimproved</th>
<th>Made Worse</th>
<th>Patients who passed gas</th>
<th>Patients who passed feces</th>
<th>Inc. in Peristalsis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recovered</td>
<td>10</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Died</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Relief from symptoms was obtained in the majority of cases. In the entire serries only three patients experienced reactions characterized by pallor, headache, shallow respiration, and thready pulse. All reactions were transiet lasting ten minutes to one hour after which spontaneous recovery occurred. Frazier (133) concluded:
"In our experience, pitressin has proved a valuable agent in the prevention and relief of abdominal distention due to adynamic ileus resulting from operative trauma and peritonitis infection. Reactions are few, mild, and transient and associated with no danger to the patient. Blood pressure is apparently not affected. We, therefore do not hesitate to recommend its use in combating this distressing post-operative condition."

A series of 350 cases operated upon for appendicitis including perforations and peritonitis with no deaths from functional ileus was reported by Ferguson (134). He attributed a fair share of this success to the routine use of Pitressin as a prophylactic against distention. This surgeon suggests use of pitressin early in the surgical procedure if there is marked distention in the intestine. Post-operative management as described by Ferguson, may be divided into three stages: physiologic ileus; intermediate stage; true paralytic ileus. Therapy of distention caused largely by swallowed air usually results in uneventful recovery. Intravenous saline or glucose is given. If pitressin has not been given before or during operation it is injected immediately afterwards and repeated every four hours for eight to twelve doses as indicated. If the patient remains comfortable an enema is not given until the third day; if
this is effective, pitressin is discontinued. During the intermediate stage, when early functional ileus is suspected, attention is given to fluid intake and three injections of pitressin may be given, one hour apart, followed by an enema. Tubal drainage is used if necessary. If true functional ileus is present, Ferguson discontinues pitressin and says, in this situation, there is usually no functionally active intestinal muscle to react by contraction. Morphine, tubal drainage, intravenous fluids and transfusions are continued. As Ferguson points out, the best treatment for functional ileus is its prevention.

Since 1932, pitressin has been used routinely on the wards of the First Surgical Division of Bellevue Hospital in 2500 abdominal cases, with the aim of preventing paralytic ileus. Potter and Miller (135) in 1939 re-emphasize the principle of prophylaxis in regard to functional ileus and outline in detail their present technique in attaining this aim. The prophylactic use of pitressin in abdominal surgery is based on the supposition that, if a condition of hypertonia of the small intestine can be produced pre-operatively and maintained during the post-operative period, the incidence of functional ileus should be materially decreased. According to the extended observations of Potter and Mueller, where pitressin is given early in
the presence of nondistended intestine and continued at regular intervals there is seldom any evidence of increased peristalsis. When Pitressin is given before operation and the effect in the open abdomen is noted, there is seen a gradual shrinking of the small intestine, coming on about twenty minutes after the first injection. While this shrinking is at times uniform, it may be segmental but has never produced anything resembling violent peristalsis. Routine use of Pitressin as taught at Bellevue Hospital is summarized, as follows: A dose of 1 cc. containing 20 pressor units is recommended by Potter (125) for patients over 12 years of age (although many prefer a 10-unit dose). If general or local anaesthesia is to be employed, the first dose is given as the patient is placed on the operating table. Where spinal anaesthesia is used, the initial dose is given at the end of the operation, since the constricting action of the anesthetic is operative up to that point. Postoperatively, Pitressin is given every four hours in uncomplicated cases for 10 or 12 doses. If any degree of peritonitis is present, the interval is shortened to two hours and if pneumonia develops, the two-hour schedule is adopted. Patients with ventral hernias are also placed on a two-hour schedule.

Pitressin should be given intramuscularly since this not only ensures prompt action but is a means of avoiding accidents. In most patients, receiving Pitressin as des-
scribed, there develops a blanching of the skin which is of no clinical significance and results from the normal action of the hormone on skin vessels. Vomiting and abdominal cramps occasionally occur. Shock characterized by fall in blood pressure, pallor, a moist skin, and thready pulse lasting from 20 to 30 minutes may occur; Potter and Mueller (125) have observed this in three instances in their series of 2500 cases treated with Pitressin. A second form of shock, apparently a result of accidental intravenous administration of Pitressin, was observed in 10 instances. None of these patients died nor were the authors aware of any report in the literature to the effect that a patient has ever died of "pituitary shock." Therapy of "shock" following injection of pituitary extracts consists of intramuscular injection of adrenalin chloride solution 1:1000.

While the prophylactic use of Pitressin is not always sufficient to prevent distention, these authors believe it does definitely lower the incidence of this complication of abdominal surgery.

Prostigmine has been used in many clinics and reports covering the use of the drug running well over a 1000 cases indicate that it is harmless, clinically effective and has definite advantages over the formerly used drug, physostigmine. Lewis and Axelmann (137) use prostigmine to prevent
post-operative distention. They began the administration of prostigmine shortly after, or at the time of, operation and continued medication as necessary. Prostigmine, 1 cc. of 1:4000 solution is given at intervals of four to six hours until the condition of the patient warrants discontinuance of the medication. Prior to the use of this agent, from 60 to 75% of their patients complained of subjective symptoms or exhibited objective signs of intestinal atony; the administration of prostigmine reduced the percentage to a negligible minimum.

Schlaepfer (138) begins prostigmine injections, 1 cc. of a 1:2000 solution from eight to ten hours following the operation and repeats each eight hours until normal peristalsis is evidenced. He injects prostigmine intramuscularly and considers it a very reliable agent to restore early normal peristalsis following laparotomy.

Uznamski (139) applied prostigmine both before and after operation. He found prostigmine valuable both as a prophylactic agent against and as a treatment for functional ileus and distention. Harger and Wilkey use the 1:4000 solution of prostigmine methyl sulfate administered subcutaneously (140).

Mensing (141) states that hypertonic saline is particularly valuable in stimulating the propulsive motility of the intestine following operation for the relief of a mechanical obstruction but should not be used in the early
stages of paralytic ileus of peritonitis because inhibition of intestinal function appears to be a defensive mechanism during this stage. In the later stages of peritonitis, hypertonic saline solution does not have any effect on the propulsive motility of the bowel, and its use is contraindicated in his opinion.

In considering means to improve the splanchnic circulation Mensing (141) states that the importance in restoring the circulating blood volume is the improvement that occurs in the splanchnic circulation, when the stretching of the gut and the increased intraintestinal pressure of distention is relieved by decompressive measures. The return of intestinal motility following decompression, he says, restores the function of the 'peripheral heart,' contracting intestine, that is, it aids the venous return from the intestine to the second set of capillaries in the liver. Mensing states, that hot applications to the abdomen alleviate the symptoms of distention and in all probability they improve the splanchnic circulation. Skelton (142) makes clear that dehydration contributes to the impairment of the splanchnic circulation. He cites, Muller and Haast's statement, that the application of heat to the abdomen alleviates distention by producing a peripheral vasodilation and a splanchnic vaso-constriction, which they call the "splanchoperipheral balance."
Splanchnic and spinal anesthesia have both been used in the treatment of ileus. From a theoretical point of view, they both accomplish the same end, in that they both produce a block of the splanchnic nerves. While the technique of splanchnic anesthesia is not particularly difficult, only one who has performed the procedure under close supervision or who has taken the occasion to practice the technique on the cadaver, and who is familiar with the anatomy involved should be considered competent to perform it clinically, according to Cohaner and Gage (143). They believe that the method of Kappis, which involves introduction of a needle posteriorly through the flank, is almost invariably the method of choice for treatment of ileus.

There are numerous reports in the literature stating the good results of spinal and splanchnic anesthesia in functional ileus. It is well-known that spinal anesthesia causes violent peristaltic movement. This action begins according to Brown (144), in about fifteen minutes after the injection and gradually wears off within a few days. Since in ileus, the splanchnic system overfunctions, it seems desirable to numb the paralyzer. One may visualize Brown states, how spinal anesthesia or analgesia inhibits the undesirable overfunction of the splanchnics and how this allows the cranio-sacral system to overfunction by releasing the latter's check rein. The cranio-sacral system
escapes the procaine. From clinical observation, it has been found this overfunction of the cranio-sacrae continues for several days and it is this sustained action that accounts for the relief of functional ileus.

Much has been written by German and French authors on this subject. Duval (145) reported 99 cases of mechanical obstruction, in sixteen of which, evacuation was produced by spinal anesthesia.

The subject of dehydration is large and a comprehensive review of the vast literature will not here be attempted. Only a few factors will be stressed.

Mensing (112) states that excessive amounts of saline solution should not be administered after the urinary output has been brought up to 1500 cc. in 24 hours, because they are liable to cause a reduction, in the colloid osmotic tension of the blood plasma with a resulting depletion of the blood.

Van Beuren and Smith (121) point out that the size and weight of the patient, the functional capacity of the myocardium and kidneys, the degree of shock, and the insensible fluid loss should be carefully estimated, and the plasma chlorides, and serum protein and blood specific gravity should be followed to maintain a patient in water, electrolyte and protein equilibrium. They give as an average total parenteral intake, 4 to 6 liters of normal saline.
for the first few days as being indicated in the average case. Stout (4) points out, it is well to remember when giving large quantities of liquids, that in the presence of fevers there is a tendency toward retention by the tissues of both water and chlorides.

Mensing (112) cautions that the administration of fluids per rectum is to be avoided especially in obstruction of the lower ileum and colon and in the various forms of functional ileus. He believes that by introducing fluids into the cecum and ascending colon, where normally the intestinal contents are being dehydrated, one is encouraging fermentation, with a result that there occurs besides an increased production in gas, a flattening or reversal of intestinal gradient of forces. Fluids are considered, best given, by the intravenous route and the most recent work seems to indicate that the tendency has been to give too much sodium chloride. Glucose in distilled water should be given plus the calculated estimate of the sodium chloride required depending upon the amount of gastro-intestinal secretions being lost. Skelton (142) points out that the intestine is one of the first tissues in the body to give up water when anhydremia exists.

As previously mentioned, there is an unusually large amount of liquid content in the gastro-intestinal canal in these cases. Stout (4) states the amount present depends directly upon the severity of the ileus, and it
usually exceeds one liter. He cites instances in which the liquid contents of the intestine approached the total volume capacity of the normal size gut, and the amount of intestinal liquid found in patients who experienced sudden cardiac deaths and in those dead from causes other than ileus, is usually less than 300 cc. and is frequently so small in amount that it is not easily measured.

To more clearly present the evidence in disturbance of water metabolism, Stout gives evidence of these illustrations, which have been obtained from water exchange records routinely kept in cases of ileus and arranged for this purpose. The column headings are, from left to right, post-operative day; parenteral intake; gastro-intestinal balance; tissue balance; total intake; urine output and fluid balance. The parenteral intake as here used represents the fluid given by hypodermoclysis and venoclysis; the gastro-intestinal balance is the difference between the quantity introduced into that tract and the amount recovered therefrom. The tissue balance is the difference between the quantity of liquid introduced into the tissues by injection and that directly moved from the tissues, namely, the water of the urine and the water losses by way of the skin and lungs. For simplicity Stout states, the last mentioned losses of skin and lungs has been considered to be one liter and for clinical purposes this figure may prove sufficiently accurate. Stout considers the error in
fluid balance as here shown rather large and a more accurate balance, he explains, may be obtained by a more careful consideration of such factors as the preformed water, water of oxidation, sensible and insensible perspiration, environmental temperature, relative humidity etc. The explanation as related to water balance is complicated and involves a knowledge of biochemistry. Such accuracy Stout points out in this type of patient is impracticable and not essential to successful clinical use of a water metabolism record in these cases.

<table>
<thead>
<tr>
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<th></th>
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<td>1</td>
<td>2000</td>
<td>-50% or -1826cc.</td>
<td>+33% or +675 cc.</td>
<td>325</td>
<td>-1150</td>
<td></td>
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<tr>
<td>2</td>
<td>2650</td>
<td>+0.9% or +80 cc.</td>
<td>+31% or +625 cc.</td>
<td>1025</td>
<td>+685</td>
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<tr>
<td>3</td>
<td>2000</td>
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<td>+2279</td>
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<td>-100% or -3100cc.</td>
<td>2100</td>
<td>+600</td>
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It will be noticed, Stout makes clear, that early in the case when the gastro-intestinal balances are negative, that the tissue balances are positive, and later when the gastro-intestinal balances are positive, the tissue balances
are negative. The two sets of figures offset each other with considerable accuracy. Also, the urine output does not follow the parenteral intake, the urine output being least, when the parenteral intake is greatest.

He concludes from this study that, from proper water exchange records, one may determine the severity of the case of ileus and the daily progress that is being made toward recovery. Also that one may determine when it is safe to administer liquid nourishment and to remove the tubal drainage used, and place the patient on his own. Stout claims to be able to predict the development of edema even before it is determinable clinically.
CONCLUSIONS

(1). Functional ileus may be defined as a disorganization and impairment of the motor functions of the gastro-intestinal tract, with resulting toxemia and blood chemistry changes, and should be considered a definite pathological entity.

(2). The functional ileus that almost immediately follows a laparotomy is due to a reflex splanchnic nerve inhibition of intestinal motility. Functional ileus, that is secondary to a mechanical obstruction is usually due to an excessive stretching of the intestinal musculature. The cause of the functional ileus of peritonitis is complex, and such factors as reflex splanchnic nerve inhibition, distention, plastic exudate between the loops of bowel, dysfunction of the myenteric plexus and toxic paralysis of the splanchnic vaso-constrictor fibers seem to enter into its production.

(3). The treatments of all three forms of functional ileus is, essentially non-surgical, and consists according to most authorities, not only in the treatment of distention and its secondary effects, but also, in the prophylaxis of distention. Most important measures are; morphine and pitressin to increase intestinal tone; tubal drainage with suction deflation; heat to the abdomen;
replacement therapy, consisting of intravenous fluids on a calculated basis by routine water-balanced charts and a calculated estimate of the grams of sodium chloride needed, depending on the loss of gastro-intestinal secretions. Transfusions may be necessary also for satisfactory replacement therapy; intra-dural injections of novacaine in the non-peritonitic types of functional ileus, that is, the functional ileus following surgical trauma and cases of advanced mechanical obstruction, since they are found to be quite effective in stimulating the proplusive motility of the intestine.

(4). When the functional ileus of peritonitis occurs, it is found to be unusually resistant and it should be tried to take the load off the gut by decreasing, in every way, its need for transporting fluids and gases. This is accomplished by immediate avoidance of all food; by early tubal drainage, which includes colonic intubation and early use of morphine. It is found that once extensive distention has occurred in peritonitis, nothing seems to be able to overcome it. Under these conditions, attempts to stimulate the proplusive motility of the gut by the above methods are futile and may be harmful.

(5). The ideal and successful treatment then, resolves itself in the prophylaxis, early diagnosis, and early initiation of therapy, so that the generalized excessive
distention, that usually results in a fatal outcome is avoided.

The End
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