position in the treatment of exophthalmic goiter. Patients seen in the early stages are sometimes wonderfully improved by the simple operation of double ligation. These cases are, however, comparatively few in number, and, since it is this type which may possibly abort, it is difficult to say that the improvement in all cases necessarily comes from the operation.

5. Serious risks are treated by a single ligation of the vessels at the upper left pole. The reaction is about three-fourths as severe as from a double ligation, but the missing one-fourth is an element of safety. If the reaction be very severe, a second ligation of the right upper pole is made a week later. As compared with the first ligation, the reaction following this one is slight. If the reaction be not severe, at the second operation the right lobe, isthmus and possibly a portion of the left lobe is removed. In some cases of extreme emaciation, yet with a temporary fair or improved condition, a double ligation is made at the one operation. In several hundred patients the average gain in those who were below weight at the time of ligation was 22 pounds within four months. At this time a thyroidectomy of one-half or three-fourths of the gland can be made with small risk.

Ligation of vessels should be made close to, or should include some of, the pole itself, so that a reversal of circulation in anastomotic branches with the inferior artery may not occur. Thyroidectomy of three-fifths or more of the gland is indicated in most of the chronic cases in which dilatation of the heart does not exceed one inch and there are not complications—ascites or mental changes.

The small percentage of patients with exophthalmic goiter who relapse in from one to several years after partial thyroidectomy, with an increase in the size of the remaining lobe, should have the benefit of further surgical aid. Unless medical treatment be rapidly beneficial in such cases, the primary procedure should be ligation of the vessels at the superior pole, leaving only the inferior artery and the lateral and inferior veins. Should this operation not restore these individuals to their former improved condition, a part of the gland can be excised at a later period with but little risk.

Ether, preceded by atropin and morphin, is the anesthetic of choice for double ligations and thyroidectomy. If there be extreme nervousness, scopolamin is used, and the worst type of cases with affections of the heart and kidneys are operated by local anesthesia or receive the benefit of Crile's anocia preparation—injecting local anesthesia into the field of operation in addition to the other preparations before ether is administered.

Ligations, both single and double, in the worst types of Graves' disease have been followed by a mortality which has fully equaled or exceeded the mortality of thyroidectomy. In the 278 operations there were 105 single and thirteen double ligations.

EXOPHTHALMIC GOITER

Single ligation	105
Double ligation	13
Single ligation followed by thyroidectomy.....	30
Two single ligations followed by thyroidectomy.....	21
Double ligation followed by thyroidectomy.....	26
Thyroidectomy	78
Thyroidectomy followed by ligation.....	4
Thyroidectomy followed by ligation followed by thyroidectomy	1
Total	278

SOME OBSERVATIONS ON CATHARSIS

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Observations on the movements of the gastro-intestinal contents by means of bismuth and x-ray were begun by Cannon about ten years ago.¹ For some unaccountable reason the subject did not receive the attention it deserved until several years later. Among important recent publications on the subject is that of Satterlee and Lewald,² who last year recorded the results of their observations on the passage of food from the stomach through the small and large intestine. Many other roentgenographers have verified their findings.^{3, 4, 5, 6}

In a normal individual the liquid stomach-contents begin to pass at once into the duodenum. The rate of outflow is governed by the pylorus, which relaxes and allows the gastric contents to pass through as fast as they are neutralized in the duodenum, but no faster.^{7, 8} Through the small intestine there is a steady forward flow toward the cecum. If a pint of bismuth soup be swallowed it takes ordinarily about six hours for all of it to pass into the cecum. But here rapid progress stops. In the cecum the mass remains for from twelve to twenty-four hours, or even longer, before there is much sign of passing on through the transverse colon. During the stay in the cecum and proximal part of the colon the mass becomes smaller and more dense from the absorption of water.

Peristalsis in the jejunum and ileum is continuous as long as there is anything solid touching and stimulating the mucous membrane. The peristaltic waves are short, worm-like and follow closely one after the other. The "rhythmic segmentation," described by Cannon,⁸ is a feature of intestinal digestion and absorption, and not a part of the propulsive force. In the large bowel, on the other hand, peristaltic movement is intermittent and powerful, and may involve a long segment at one time. In the transverse colon the contractions begin with a few short to-and-fro cradle-like movements,^{6, 9} which are probably caused by the longitudinal muscle-bands and are assumed to serve for mixing and lubricating purposes. These side to side movements are immediately followed by a series of ring-like contractions on which the fecal mass may pass rapidly over the splenic flexure and to the sigmoid. After such a functional effort the colon may rest for many hours before its next cycle of peristalsis begins.⁴ Reflex stimulation below the sigmoid causes evacuation.

A so-called antiperistalsis has been noted in the transverse colon,^{6, 9} the peristaltic waves passing for a few moments from left to right. Bloch,³ who claims to have been the first to demonstrate it in the human, thinks it is of rare occurrence, but cannot say whether it is a physiologic or a pathologic phenomenon. Short, on the other hand, thinks antiperistalsis the rule in the first half of the colon.¹⁰

The complicated physiology of digestion and absorption is as yet far from final solution and comprehension. In the upper part of the digestive tract physiologic

1. Cannon: *Am. Jour. Physiol.*, 1902, vi, No. 5.2. Satterlee and Lewald: *THE JOURNAL A. M. A.*, Oct. 14, 1911, p. 1255.3. Bloch: *Fortschr. a. d. Geb. d. Roentgenstrahlen*, 1911, xvii, 121.4. Holzknecht: *München. med. Wehnschr.*, 1909, No. 47.5. Fischl: *München. med. Wehnschr.*, 1911, No. 36.6. Schwarz: *München. med. Wehnschr.*, 1911, No. 39.7. Abderhalden: *Physiol. Chemie.*8. Cannon: *The Mechanical Factors of Digestion.*9. Bergmann and Lentz: *Deutsch. med. Wehnschr.*, 1911, No. 31.10. Short: *The New Physiology in Surgical and General Practice.*

chemistry explains more or less successfully the processes involved in digestion. But the exact manner in which the intestinal mucous-membrane cell takes up many of the products of digestion from the intestine and passes them into the circulation is not well determined.^{11, 12} The mucous membrane seems to be a most wonderfully appointed living chemical laboratory. Some of its cells are capable of producing ferments which subdivide and chemically prepare food-substances so that other cells may absorb them, reunite elements and form new chemical compounds which pass on into the capillaries and lymphatics. The small intestine also produces a ferment which destroys bacteria, as shown by Fleiner.¹³

In the ileocecal region a most varied bacterial flora complicates still further these physiologic problems. The activity of these bacteria on the various food-elements, on the digestive ferments, on the cells of the mucous membranes and on the bacteria themselves is far from being fully understood. In the cecum certain varieties destroy the digestive ferments, thereby ending digestion, while others cause fermentation and putrefaction of unabsorbed products of carbohydrate and proteid foods, respectively.¹⁴ Still other bacteria seem to have a mission of regulating the number and life-history of the former varieties. The *Bacillus coli communis* group probably belongs to this class. After the fluids have been absorbed from the bowel-contents in the colon and the fecal masses are hardened, the bacteria are found to be mostly dead. Dead bacteria make up one-third of the volume of hard feces.¹⁵

The interrelationship between the various bacterial groups, and between them and the food-products and the physiologic elements of the intestine must be at a certain equilibrium.¹⁴ This interdependence between the elements involved may be quite individual. The life-history of the majority of these bacteria is short, lasting but a few hours. Before meals the small intestine is nearly if not quite sterile and in the large bowel the bacteria are dying off rapidly, owing to autotoxic activity. After a full meal the bacteria again proliferate with astonishing rapidity throughout the intestinal tract. When generations follow one another in such rapidity, evolutionary changes may be possible within the space of a few months or years. It is reasonable, therefore, to think that each individual may have his own peculiarly developed type of a given bacterium (*Bacillus coli communis*, for instance), which in the course of time and development has become necessary in this particular intestine to balance and check up the activity of the other bacteria and ferments. According to Kendall,¹⁴ the greatest interdependence among fecal bacteria exists between the fermentative and the putrefactive types—the former decomposing the carbohydrates and the latter the proteids. A marked disturbance of their relative activities will cause excessive gas formation and perhaps diarrhea, the symptoms of so-called “intestinal fermentation.”

An individual who eats habitually a varied diet, who secretes normal gastric, pancreatic and intestinal juices, who has a properly functioning neuromuscular apparatus for peristalsis, and who has an evenly balanced colonic bacterial flora—such an individual enjoys intestinal comfort. Whenever any one of these factors is deficient or excessive in its activity, the intestinal balance

is upset and the individual suffers discomfort and symptoms in proportion to the derangement.¹³

We are apt to think of the function of peristalsis as being limited to the propulsion of intestinal contents. It is easily conceived, however, that the successive contractions and relaxations of the intestines also cause alternately an ischemia and a hyperemia of the intestinal wall. These circulatory changes aid absorption from the mucosa and cause a more rapid dissemination of absorbed substances into blood-vessels and lymphatics.¹⁶

From this fact we should deduce a lesson in pathology. If pathologic products exist in or about the intestine, increased peristalsis may cause an increased toxemia. Increased peristalsis also means a mechanical spreading of infection on the peritoneum and a delay in the formation of protective adhesions.

In acute inflammations within the abdomen, Nature's first effort is to subdue peristalsis about the region of infection.¹⁷ Her next effort is to confine the infection within the smallest possible area by means of peritoneal adhesions.

Here we find another lesson. This is one in therapy and reads plainly as follows: In acute intra-abdominal infections do nothing to increase peristalsis.¹⁸

When we desire to evacuate the bowel rapidly it is a time-worn custom to give a saline purgative. In the small intestine a concentrated saline solution causes a profuse transudation of fluids into the intestine. The colon loses its absorptive power from the presence of the salt and its contents are expelled in liquid form. Peristalsis is increased most markedly in the large bowel. This transudative effect on the mucosa of the small intestine interferes with the formation of ferments. The zymogen-carrying cells become depleted of the necessary fluid. But the greatest factor in this derangement of intestinal balance is, perhaps, the destruction of the bactericidal principle.¹⁹ The food therefore passes into the cecum unabsorbed and in a state of but partial digestion. The fermentative and putrefactive bacteria find the best possible conditions for their existence and proliferation. That they improve their opportunity is shown not only by the fact that the diarrheal stool is teeming with living bacteria, but also by the gas distention of the colon experienced by most individuals as an after-effect of a dose of salts.

It may seem almost a sacrilege, but we cannot except even calomel from our criticism of salines in general. Calomel was formerly thought to be both a biliary stimulant and an efficient intestinal antiseptic. Schütz¹⁹ has shown it to be neither, but quite the contrary. According to this and other authorities,^{13, 20} the action of calomel on the intestinal mucosa is harmful and in favor of bacterial growth.

When a cathartic is necessary, oil is probably the least harmful to the intestinal mucosa. Oil does not destroy the disinfecting property of the succus entericus and it forms a good lubricant for the colon. Oil surrounds and impregnates the food particles. This retards bacterial activity, and thereby reduces gas formation.

A high rectal enema reaches and flushes the whole length of the colon. This can be proved to any doubter by giving it to a patient while the abdomen is opened so that the large bowel can be seen. A salt solution, stronger than normal saline, or practically any other

11. Howell: Text-Book of Physiology.

12. Hammersten: Lehrbuch. d. Physiol. Chemie.

13. Fleiner: Jahresh. f. Aerztl. Fortbild., 1911, No. 3.

14. Kendall: Jour. Med. Research, 1912, xxv, 117.

15. Strasburger: Ztschr. f. klin. Med., 1902, xiv, 415.

16. Mall: Johns Hopkins Hosp. Rep., 1896, No. 1.

17. Strümpell: Pathologie und Therapie.

18. Ochsner and Percy: A New Clinical Surgery.

19. Schütz: Arch. f. Verdauungskrankh., vii.

20. Nickels: Reference Handbook Medical Science.

irritant in an enema reaching beyond the splenic flexure will often cause immediate evacuation. Cannon observed increased peristalsis even in the ileum as a result of the stimulus from an enema of soapsuds.⁸ Should the enema fail, an irrigation of the colon is possible.

An internal secretion which stimulates peristalsis has been obtained from the gastric mucosa and from the spleen. The experiments of Dohrn, Marxer and Zuelzer^{21, 22} showed that this is a constant hormone in animal organisms regulating the function of peristalsis. Therapeutically it has been used to some extent, but will need further experimentation and use before definite indications for its employment can be established. When this is done it promises to become a physiologically ideal intestinal evacuant.

After this brief review over several facts concerned in the functions of the bowels, and over the action of some of the common cathartics, let us see what clinical observation teaches in regard to the use and non-use of cathartics, especially in certain surgical conditions.

I have searched my clinical records and experiences with respect to this question and found that the patients with acute appendicitis whom I have seen die from local extension of the disease, or from general sepsis, either with or without surgical intervention, had without an exception received and retained some active cathartic at the beginning of the disease. The patients with diseases of similar nature who have barely escaped death, but who have developed the worst postoperative complications, advancing peritonitis, bowel paresis, distention, fecal fistulas and miserere, had nearly all been given a dose of salts as a passport to prolonged suffering, tedious and expensive stay in the hospital, and finally months or years of reduced vitality and usefulness. On the other hand, the cases of acute intra-abdominal inflammation which had an equally stormy onset and which apparently promised fully as grave complications as those already mentioned, but into which the element of purgation had not entered—in these cases the patients had in comparison a surprisingly easy and quick recovery.

Descriptions in detail of illustrative cases as well as a classification of all such patients would be of interest, but time-limit forbids. I feel, however, that my experience along this line has been great enough to warrant the greatest possible attention to the facts stated. The country practitioner has here a grave responsibility. Beyond his duty to each individual patient, he should be a missionary among the uninformed. It has become a custom whenever a patient with acute appendicitis or other acute inflammation about the movable viscera is brought to me, to inquire at once: "Have you taken a physic?" If this question is answered in the affirmative, the next question is whether the stomach resented the insult with free emesis, or whether the cathartic were retained to perform its malicious work. This knowledge is of importance in prognosis. Sad experience has been my teacher.

About two years ago my attention was drawn to the fact that most patients who were subjected to abdominal operations immediately on their admittance to the hospital, on account of strangulated hernia, acute appendicitis or other abdominal emergency, recovered with but slight discomfort from gas distention. During the same time other patients who had been deliberately prepared for operation and who had received fully as careful and

delicate handling of peritoneum and viscera as the former patients, suffered relatively much more from meteorism and pain and had a more serious time in getting the bowels opened after the operation, than had the emergency cases. The relation between operative trauma and postoperative gas pain was decidedly in favor of the "unprepared cases."

A closer study about these circumstances led me to believe that this difference in results must have its cause in some step in the preparation of the patient before the operation. That the operative technic was a radical factor could not be assumed, for several reasons. In a strangulated hernia, for instance, the intestine would be handled and rubbed with fingers, gauze and instruments, and perhaps resected, and yet the intestinal contents would not be materially hindered in their transit. Comparatively little gas would be formed in the bowel. In immediate operations for acute appendicitis, the peritoneum would be opened freely, gauze pads would be laid over omentum and numerous loops of intestine and held firmly against the peritoneum by intra-abdominal pressure. Many such patients recovered with a surprisingly small amount of meteorism. On the other hand, an interval appendectomy, a gall-bladder incision, a pelvic operation would at times be followed by two or three days of distention and distress before the bowel could be induced to functionate properly. And this would sometimes happen after the most painstaking preparation of the patient, including a liberal use of cathartics a day or two before the operation with the erroneous idea that this would remove all gas-producing materials.

It was evident that the greatest difference in the preparation of the two classes of patients lay in the non-administration of cathartics in the emergency cases. I concluded therefore to leave out, by way of experiment, this feature of the preliminaries in ordinary laparotomies. This was done tentatively at first, but as experience made me bolder and more confident I discontinued all use of purgatives before operations, except in rare instances and for special reasons. Instead of the classical dose of calomel and salts, two or three high enemas were given to flush out the colon.

I have followed this practice now for over a year to the great satisfaction of patients, nurses and surgeons. It includes operations on the stomach, gall-bladder and ducts, intestine, uterus, tubes and ovaries.

A bowel that has not been vitiated by a saline scouring is in condition, physiologically and bacteriologically, to prevent the excess formation of gases, and to expel those that do form. It is the gas distention which causes the bulk of the postoperative distress. This complication being reduced to a minimum, fecal contents are taken care of by normal peristalsis or by enemas if required.

The following are the most important conclusions which I have reached after a study of this subject:

The food normally passes rapidly from the stomach and through the small intestine. The small intestine is practically empty six hours after an average meal. In the cecum and colon the bowel-contents remain for from twenty-four to forty-eight hours.

The maintenance of a certain equilibrium between the physiologic and bacterial processes in the intestine is necessary for normal bowel function.

Saline cathartics disturb this poise to a marked degree, making their use a matter of grave consideration. After the intestinal mucosa has been depleted and exhausted by any powerful cathartic, it takes some time

21. Dohrn, Marxer and Zuelzer: Berl. klin. Wchnschr., 1908, No. 40.

22. Zuelzer: Internat. Zentralorgan f. Blut u. Serumforsch., 1910, vi.

to recover its energy. During this time bacteria flourish unmolested by intestinal ferments.

Intestinal antiseptics is at best of unknown and doubtful efficacy; a comparative asepsis is practical.

An artificial evacuation of the large bowel, satisfactory for most surgical and many other purposes, does not require the ingestion of any drug acting on the small intestine. High rectal injections are efficient and cause less damage and discomfort.

A cathartic given in the beginning of an acute intra-abdominal infection is a dangerous practice and contrary to modern scientific knowledge and experience. Numberless gravestones mark the disregard of this principle.

Patients who are prepared for laparotomies by a restricted diet and rectal injections have a much more pleasant postoperative recovery than have the patients who have been purged.

INTRA-ABDOMINAL PRESSURE

ITS IMPORTANCE IN MAINTAINING STATIC EQUILIBRIUM
AND THE NECESSITY OF CONFORMING TO ITS
LAWS IN THE RESTORATION OF ORGANS
TO THEIR NORMAL POSITIONS*

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As the Roman gladiators in the days of old entered the arena to meet each other in deadly combat or to be torn to pieces by the fierce Numidian lions, it was their custom to greet their audience with the expression *Morituri salutamus!* "We who are about to die salute you!" Conscious of the wide divergence of opinion on this subject of intra-abdominal pressure and knowing the powers and skill of the many gladiators who enter this arena, to whom the Numidian lion is but a toy, I can only exclaim in the language of the ancients, "I who am about to die salute you." Despite this depression of the hour I am buoyed up by the thought that out of the contest there may perhaps come some slight illumination on the subject or at least some inspiration for further investigation.

The universal application of abdominal section which has come into vogue during the past quarter century for the relief of positive pathologic conditions in the abdominal cavity has brought to light a startling frequency of ptosis, sometimes of one organ, sometimes of another, and occasionally of all. Many and various surgical procedures have been instituted for relief of these conditions but with varying and uncertain success. Failure to obtain satisfactory results has led certain investigators to inquire into the causes of ptosis. And these wise men tell us that ptosis is due to "abdominal static conditions," that these conditions are produced by "aberrant intra-abdominal pressure," which, in turn, is produced by "faulty postures;" that "faulty postures" of the body come from "lack of physical vigor and vitality," "that ptosis interferes with digestion and assimilation" and hence the lack of vitality. And so we go around in this vicious circle, like a dog chasing his own tail.

We can understand faulty postures; lack of vitality is comprehensible; ptosis we recognize when we see it; static conditions are explicable, but the one link in the chain that eludes us is intra-abdominal pressure. What is intra-abdominal pressure? Breathe softly now, for we are on sacred ground. Intra-abdominal pressure is a great mystery.

At the meeting of the British Medical Association, Sheffield, England, in 1909, I had the honor of being invited, as the American representative, to open the discussion on displacements of the uterus and operative procedures for their relief. In these remarks I presented my conviction regarding the importance of ligaments in maintaining the normal position of the uterus. In the general discussion which followed I was informed by one of my British confrères that this matter had all been settled in previous discussions on this subject; that ligaments had nothing to do with the case; that abdominal and pelvic organs, as was well known, were held in place by intra-abdominal pressure and the floor of the pelvis; that the liver, which had many square inches of space on the lower surface, was lifted up by an intra-abdominal pressure many times its weight.

It has interested me to discover what was being taught the present generation of medical students in regard to this matter, by putting to the applicants who apply for positions as interns in the various hospitals with which I am connected the question: "How are the kidneys and the liver kept in place?" The common reply is, "The ligaments may have something to do with it but the chief support is negative pressure." When I inquire, "What is negative pressure?" the universal response is, "I don't know what it means, but that is what I was taught."

Let us revert again to the question, What is abdominal pressure? Intra-abdominal pressure may be defined as pressure within the abdomen due to external atmospheric pressure, to gravity, to muscular contraction of its walls and to intravisceral pressure. The first two, atmospheric pressure and gravity, are constant; the latter two, i. e., muscular contraction and intravisceral pressure, are variable and therefore constantly modifying the intensity of the pressure. The intensity of intra-abdominal pressure at any time and in any place is the resultant of all these forces working together at the specified place and specified instant of time. Muscular contraction has reference not only to the abdominal walls, but also to the diaphragm, which forms the upper wall of the abdomen and modifies intra-abdominal pressure with every breath. By intravisceral pressure is meant the pressure produced by a full stomach in filling the abdominal cavity and thereby increasing the pressure. The stomach contents may be solid, liquid or gas. This applies equally to the intestines. To a minor degree a full gall-bladder or an enlarged liver may increase intra-abdominal pressure, and likewise any pathologic tumor.

We all recognize that the specific gravity of the organs and other contents of the abdomen do not vary much from that of fluids, and consequently, intra-abdominal pressure corresponds closely but not absolutely to hydrostatic laws. If we press the abdominal wall with the hand, the pressure is communicated in all directions till it meets with organs or tissues that are of greater density. These cannot transmit the force with equal facility and consequently they reflect what they cannot readily transmit, but finally are pushed before it till the force has been equally distributed and equilibrium has been attained. This moving force would be felt least by the

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