Original Articles.

GASTRO-MESENTERIC ILEUS.

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IN a recent communication read before a joint meeting of the Chicago Medical and Surgical Societies, I presented a report of twenty-six cases of post-operative ileus which I had observed in my hospital and private practice. Of this number, one belongs to that extremely interesting group of obstructions occurring high up in the intestinal canal. This case has seemed to me to be of sufficient interest to warrant a more detailed report. Unfortunately, the true condition was not recognized during life, owing to unfamiliarity with the clinical picture presented, nor indeed were the pathological findings at the autopsy thoroughly understood until after my attention had been called by Dr. Bloodgood to Zade's article upon the subject which had just appeared. After a careful review of the literature, it has seemed to me that sufficient attention has not been directed to this condition, the existence of which has been known since the time of Rokitsansky and to which the name of "gastro-mesenteric ileus" has recently been given by Zade. That it is not of infrequent occurrence is shown by the increasing number of cases recorded in the literature. That it is a condition of great gravity, and one difficult to recognize, is amply attested by the fact that, with few exceptions, the condition has been recognized only at autopsy.

It is to a consideration of this form of obstruction, the conditions both anatomical and pathological which appear to be concerned in its production, and the phenomena by which it manifests itself, that I wish to direct your attention to-night.

Mrs. Susan H., aged forty-six years, married. Admitted to the Johns Hopkins Hospital, medical side, June 18, 1904, complaining of acute indigestion.

Family history: Negative.

Past history: Patient has always had trouble with stomach, and indigestion, appetite never very good, and of late has had a great deal of belching. Bowels usually constipated. In 1901, three years ago, she had a severe attack of gastritis in which she vomited a great deal, but no blood, was in bed for three months.

Present illness: In April, 1904, two months before admission, the patient was operated upon for "womb trouble." The exact nature of which is not known. Following the operation, she has been unable to keep anything on the stomach; even water causes her to vomit within five minutes. Patient says she is frequently nauseated and that fluid will regurgitate without any cause. Has had feeling of pain and distention in left side. Has lost in weight and strength, no edema of the extremities. Urine normal.

Physical examination: Color good, tongue slightly coated, pulse 92, small. The abdomen seems normal, slight epigastric pulsation, dermatographia marked. No visible peristalsis, no palpable mass, general abdominal tenderness, probably neurotic. Liver and spleen not palpable, no ascites, no jaundice. Area of stomach tympany normal. Red blood corpuscles, 4,416,000; white blood corpuscles, 5,200; hemoglobin, 81%.

July 9, three weeks after admission. The patient has continued in practically the same condition. Can

take nothing but albumen or whey by mouth and even these frequently cause vomiting. Looks badly, has lost in weight and strength. Ewald test meal removed in one hour. About 50 cc. of greenish watery fluid mixed with bread removed. No free HCl. Total acidity, 12%. Lactic acid present, no blood. Oppler-Boas bacilli present.

The patient's condition remained about the same except that tenderness was also complained of in both flanks, and it was thought that quite a definite thickening and resistance in the right half of the epigastrium could be made out. She was transferred to the surgical side, Sept. 6, 1904, and was operated upon the following day.

Operation: The abdomen was opened through the right rectus incision. The stomach was slightly dilated and on the anterior wall of the pylorus there was found a whitish scar-like thickening. The pylorus admitted the tip of the finger. The duodenum was slightly dilated and high up under the liver, but was not kinked. Gall bladder normal. The pelvis was not explored. Pyloroplasty was done in the usual manner.

Subsequent history: The patient vomited immediately upon recovery from ether, and continued to do so at intervals until death. At first the vomitus was green or yellow with some blood. After the second day, it became darker and more offensive, but not feculent. The pulse was rapid and weak throughout. There was slight elevation of temperature. Severe pain was complained of only during the first two days. The patient died on the sixth day in collapse. I was unfortunately called out of town immediately after the operation, and did not see the patient again. There was no note made in the history of any unusual distension of the abdomen.

Autopsy: Anatomical diagnosis. Operation, pyloroplasty. Partial obstruction of duodenum, bronchopneumonia in left lung. Cholelithiasis, uterine myomata. There is no leakage at the point of suture and no peritonitis. The stomach is but moderately dilated, the duodenum is much dilated up to the point where it passes through the mesentery beneath the superior mesenteric vessels, beyond this point it is contracted.

Upon introducing the finger into the lumen of the duodenum, an obstruction was met at the point where the mesenteric vessels cross. The examining finger met with considerable resistance in passing the obstruction, which was relieved upon lifting the intestine out of the pelvis. No lesion was found in the wall of the duodenum, at this point.

The remaining portion of the small intestine is collapsed and lying in the pelvis. The cecum and ascending colon up to the hepatic flexure are dilated. This dilatation is apparently due to adherent omentum at the hepatic flexure. The intestines appear normal, there is hyperemia of the mucosa of the stomach with some extravasation of blood.

It will be seen at once that the clinical picture presented by this case is that of acute dilatation of the stomach less the abdominal distension. Autopsy, however, did not reveal an extreme dilatation of the stomach. Ewart also noticed in one of his cases that the stomach which had presented all the phenomena of an acute dilatation during life, was found after death to have diminished considerably in size. It has been further noted that a stomach even when found extremely dilated at autopsy will frequently, when emptied, assume almost normal proportions after the distension has been relieved by withdrawal of the gas or fluid.

It is evident that the two conditions, the one

we recognize under the name of acute dilatation of the stomach, and the clinical picture presented by the case just reported, are either very closely related, or are one and the same thing. That they are very intimately associated and that the clinical picture of the one very closely simulates that of the other, cannot be gainsaid, but that they are two distinct entities, after a careful study of the histories and autopsy findings of the reported cases, I am convinced. I am equally convinced that a considerable number of the cases which have been reported in the literature as acute dilatation of the stomach, are really cases of gastro-mesenteric ileus. The prominent clinical feature in both instances is, as a rule,

the dilated stomach, therefore each may properly be classed as acute gastric dilatation. That the obstruction produced by the tense mesentery and superior mesenteric vessels is one cause of acute dilatation of the stomach, will, I think, be generally admitted, but that it is the sole cause, or even the primary factor in its production, has not been established.

There are many conflicting opinions as to the causation of acute dilatation of the stomach in general. Very little, really, has been added to our knowledge of the subject since the first description by Hilton Fagge in 1872. The pathology of it is not yet thoroughly understood. Many suggestions have been made to account for the dilatation, but no single one will explain every case. As pointed out by Thompson, the two

most prominent features demanding explanation are (1) the cause of the paralytic condition of the stomach walls whereby they are unable to expel their contents and (2) the mechanism which gives rise to the excessive secretion which in many instances plays such an important part in the disease.

Opinions differ as to which is the primary condition, the excessive secretion or the paralysis of the stomach walls, and whether the one is dependent upon the other, or whether they are two distinct processes. In the case of gastro-mesenteric ileus, also, the question at once arises as to the priority of the two conditions, the dilatation of the stomach or the duodenal obstruction. Does the obstruction of the duodenum, due to the superior mesenteric vessels, give rise to the gastric dilatation, or does the paralysis of the stomach walls and the subsequent hypersecretion by forcing the small intestine down into the pelvis, make traction upon the mesenteric vessels and thus produce the obstruction? Or, is the general enteroptosis so commonly found, with the displacement of the collapsed small intestine downward into the pelvis, the primary cause? These are perplexing questions which I confess I cannot answer to my own satisfaction. But whichever may be the primary condition, it is evident, as Albrecht points out, that once these conditions are present, we have established a "vicious circle."

The findings at autopsy in the reported cases have been quite constant, consisting of marked dilatation of the stomach and duodenum up to the point where the duodenum passes beneath the superior mesenteric vessels, although in a few cases, as has already been pointed out, this dilatation did not exist. The free mesenteric border, including these vessels, is found taut. The small intestine, as a rule, is collapsed and lying in the pelvis. In a few cases, the upper portion of the jejunum is not entirely empty. A dilatation of the cecum and ascending colon have been noted. On lifting the intestine out of the pelvis, gas and fluid are seen to pass the point of constriction at the superior mesenteric vessels.

The stomach walls are usually found thinned. In a few cases they have been normal. In one case of chronic pyloric stricture they were hypertrophied. Small capillary erosions of the mucous membrane have been noted, with hemorrhagic infarcts and necrotic patches. Perforation following necrosis has been observed in one case (Koerte's). Müller and Humby found atrophy of the musculature of the stomach wall, with separation of the muscle bundles so that the mucosa and serosa were in places in contact. Deep ulcers are not found. Adhesions about the pylorus have been noted in a few instances.

But few microscopic examinations have been recorded of the stomach walls. These show no special changes except thinning of the walls and separation of the muscle fibres with some granular degeneration, and here and there areas of hemorrhage, and round-cell infiltration. Rapid

dilatation of the stomach is suggested by the separation of the muscle bundles and thinning of the gastric walls.

Various explanations have been offered by different authors of the perplexing phenomena observed in these cases, but so far none explain satisfactorily all the conditions found present. Rokitansky in the first edition of his "Handbuch der Path. Anat.," 1842, states "that at one or more points of the intestinal canal, incarceration of one loop may be caused by pressure upon it by another part of the intestine or its mesentery," but that it "takes place only from before backwards against the stiff, unyielding abdominal wall." He says this occurs most frequently where there is a long slack mesentery in connection with prolapse of the pelvic organs, or in large herniæ. In the third edition of his book, 1861, he says "in this category belongs the compression of the lower portion of the duodenum by the mesentery of the small intestine, and the superior mesenteric vessels and nerves contained therein." He speaks of it as taking place more often in advanced age. He does not discuss the conditions concerned in the production of this form of ileus, nor does he report any cases in which he has observed this condition clinically.

Albrecht in an elaborate review of the subject calls attention to the fact that the lumen of the duodenum at the point where the mesenteric root with the superior mesenteric artery crosses it, has normally a lumen which is not round, but much flattened by the pressure of the mesentery and this vessel. He thinks that in order that the small intestine, as a whole, may find room in the pelvis, it is necessary that it should contain an unusually small amount of solid or liquid. If these conditions are fulfilled, the small intestine on account of its weight, sinks into the pelvis when the body is placed upon its back, thus producing closure of the duodenum by means of the traction on the superior mesenteric artery; the lumen of the duodenum being already relatively narrow. He thinks that the occurrence of this obstruction in no way depends upon an abnormal length of the mesentery or unusually pronounced lordosis of the lumbar vertebrae, or an abnormally low position of the duodenum, although he admits that the presence of these conditions may constitute favoring factors.

The sinking of the small intestine into the pelvis, he thinks, is especially brought about by means of pronounced gastric dilatation. He speaks of the favoring influence of tight bands about the waist in this connection. Transient closure of the duodenum in this way, he assumes as occurring quite often in susceptible persons when lying upon the back for any length of time. As to the causes which render permanent this condition of compression, he thinks there has, as yet, been no satisfactory explanation. *A priori* we have two possibilities: (1) either the *vis a tergo* is too weak to overcome the obstruction in which event there would be present a defect in the motility above the compressing point, or (2)

there are abnormal mechanical relations present, which increase the pull downwards and backwards of the small intestine and keep it permanent, so that the mechanism of the normal motor power is insufficient to force open the closure. The first explanation would presuppose a neuroparesis of the stomach or duodenum, or both. Such a condition has not up to the present time been demonstrated, although motor weakness of various grades is not infrequently met with. Explanation of these cases by means of motor insufficiency alone he considers unsatisfactory, after quoting the observations of Hanau, Kelyack, and others.

That motor insufficiency of the stomach may play indirectly an important role, he thinks likely. He considers, at some length, the forces which go to make constant the pull upon the mesenteric vessels. Adhesions, of course, suggest themselves as a means of holding the small intestine fixed in the pelvis, but these have not been generally observed. A great distension, which would increase their weight, has also not been observed. On the contrary, they are, with few exceptions, empty and contracted. Hanau first suggested the enormously distended stomach as the force concerned in rendering constant the position of the collapsed small intestines in the pelvis. This occupies the entire upper abdomen, preventing

their escape from the pelvis. He reports one case in which the stomach contained four liters of fluid. Other larger amounts have been reported by other observers. He calls attention

to the fact that enormous pressure must be exerted on the small intestine in the pelvis, and a corresponding pull upon the mesentery, by this superimposed weight.

Albrecht also carried out some experimental observations as to the effect of attaching small weights to the mesenteric vessels, and the amount of obstruction produced by these in the duodenum. He found that with a weight of 2 kg., a considerable water pressure would be necessary in order to force fluid through the constriction into the jejunum. He considers the mechanical effect of the distended stomach as a direct cause of the ileus symptoms, by holding the coils of small intestine in the pelvis once they have gotten there, and as an indirect cause in displacing them there primarily. He recognizes the existence of a chronic dilatation of the stomach which he believes of considerable etiological importance in the development of the acute form. He differentiates two varieties, those in which the wall of the stomach is found much thinned and those in which it is not. In the latter class, he places the chronic dilatations. He suggests also the possibility, in the case of chronic dilatation, that this may lead to a narrowing of the boundary point between duodenum and jejunum, which, under favoring circumstances, may become complete, and draws an analogy between this condition and over distension of the bladder.

I have quoted Albrecht's views at length because he was the first to give any adequate explanation of the conditions observed, and because they embody practically all that is known to-day as to the causation of the condition. It would lead us too far afield to discuss all the hypotheses that have been suggested by different writers, but reference to a few of the more important may not be out of place.

Glenard, whose classical work on enteroptosis has thrown so much light upon this and kindred conditions, believes that the dilatation of the stomach brings about an atony and sinking of the duodenum, and by means of the pull downwards, a narrowing at the duodeno-jejunal boundary. The emptying of the stomach is thus made more difficult. This increases its atony and may lead to dilatation. Following the sinking of the duodenum, a pressure upon the common duct is brought about. He also describes a physiological closure of the duodenum by the superior mesenteric vessels and mesentery, the function of which is to retain the chyme for a longer period in the duodenum.

Kussmaul and A. Fraenkel believe that the greatly dilated stomach produces kinking of the pylorus, but that this is not the case in the great majority of instances is shown by the fact that bile is present in the vomitus.

Kundrat and Schnitzler think that in order that such a condition may take place, there must be a change in the mesentery and also in the vertebræ. This change in the mesentery consists in a closure of the fan-shaped form, to that of a half-opened fan, and this can only take place when the intestines are contracted and lie in the

pelvis. By this means the direction of the pull on the mesenteric root is concentrated, and predisposes to incarceration. This condition, they think, is impossible in cases of stiff abdominal walls, and can take place only in those cases where the walls are very lax. They also think there is a relatively low position of the duodenum, bringing it nearer the prominence of the lumbar vertebræ, and thus making the compression of easier occurrence.

If the intestine contains no gas, there is a stronger pull exerted. Even with all these factors, they consider it necessary to have a slight twist of the mesentery, that is, not a true volvulus, but a position that is slightly diagonal. They have found from observation on the cadaver that compression of the duodenum occurs more easily when the mesentery is displaced slightly toward the right.

Müller thinks that this form of compression can come to pass from a horizontal position on the back, and that this is the most important contributing cause. He thinks it is favored by the fact that in post-operative cases the purgation before operation leaves the intestine entirely empty, and that it is also favored by the weakening influence of the narcosis. In this condition, the empty small intestine needs only to sink into the pelvis, provided its mesentery is of sufficient length.

Müller is of the opinion also that compression of the duodenum is always the primary cause, and that dilatation of the stomach is of secondary origin. He does not believe in the theory of a latent gastrectasia.

Stieda thinks that the primary cause is an acutely increasing atony of the stomach and duodenum, and that the obstruction by the mesentery is secondary. The dilatation of the cecum and ascending colon sometimes observed, he thinks, is due to the fact that the dilated stomach produces a kink at the hepatic flexure by pushing down the transverse colon.

Kelling does not consider incarceration of the small intestine in the pelvis and a special length of the mesentery sufficient cause for the occurrence of this obstruction. He thinks there must be a primary over-filling of the stomach which compresses the small intestine into the pelvis. He gives the name of "combination ileus" to this condition. He thinks, to establish the diagnosis of arterio mesenteric closure, we must be able to demonstrate some factor which will place the small intestine in the pelvis, and prove that this took place at the same time as the occurrence of the symptoms of duodenal closure.

He reports the results of some experiments upon the cadaver which would tend to show that dilatation of the stomach took place only after the formation of a kink in the duodenum, together with a valve formation at the cardia.

Thompson thinks the primary cause the paralysis of the stomach walls, and discusses, at some length, the conditions giving rise to this. Later on distension by gas or excessive secretion takes place. That the latter is not the primary

cause is evidenced by the fact that it is not present in every case. Pepper and Stengel hold the view that there is some obstruction of the pylorus either spasmodic or permanent; but the presence of bile in the vomitus would exclude this as a possibility. Both Thompson and Wiesinger call attention to a distinct rotation at the pylorus, and Bennett has observed the same condition at the cardiac end. Thompson urges the absence of visible peristalsis as an argument against the theory of obstruction either at the pylorus or duodenum, in reply to which Mayo Robson points out that "peristalsis in any part of the intestinal canal is never seen in acute obstruction unless it supervenes upon a chronic impediment to the outflow of fluids." A rather too sweeping statement, I fancy.

Box and Wallace advocate the idea that the "Tense distension is due to actual pressure of the stomach upon the part of the duodenum which crosses the third, and ascends by the side of the second lumbar vertebra to end in the jejunum." They think that in order to explain the phenomena met with, there must be (1) a paralytic condition of the viscus which leads to distension, and then at a certain stage (2) the distended stomach actually produces obstruction by pressure of the duodenum against the front and left side of the spinal column.

Byron Robinson, of Chicago, who seems to be the only one in this country who has written upon this subject, insists upon the very important part played by enteroptosis. He believes that this factor, together with the relative fixation of the duodenum, which he explains on anatomical grounds, are the most important contributory causes in the development of this condition. He thinks the gastric dilatation is caused primarily by gas, and later by fluids.

It will be readily seen from this summary of the opinions of the various observers, that there is no unanimity of opinion as to the mode of production of the condition under discussion.

Etiology: Indiscretions in diet, enteroptosis, debility, whether caused by wasting diseases, acute infections, surgical operations, or narcosis, seem to be the most constant and potent etiological factors. To these might be added certain of the grave toxemias and severe injuries, examples of which have recently come to my attention. An example of the former occurred in a young woman with pernicious vomiting of pregnancy who, through false statements, had been operated upon for gastric ulcer. Typical symptoms developed shortly after the operation and continued unabated until death. At autopsy an enormous V-shaped dilatation of the stomach was found, with moderate distension of the intestine, and advanced fatty degeneration of the liver.

The second case, related to me by Dr. W. S. Watson, was a feeble old lady, aged sixty-five years, the mother of twelve children, with very relaxed abdominal walls, suffering from diabetes, who fell and sustained a fracture of two ribs. She progressed favorably until the fourth day, when, without apparent cause, she began

to vomit frequently, and in considerable quantity. The abdomen began to swell, and a large-sized tumor rapidly developed. The pulse became quick and feeble, temperature normal. The vomitus was very dark like coffee, the odor was disagreeable, but not feculent. Outlines of the greatly distended stomach could be seen lying diagonally across the abdomen, from left to right, and extending almost to the symphysis, it is tympanitic and very tense, no visible peristalsis. The abdomen was opened by another surgeon, and the greatly distended stomach was punctured and sutured to the parietal peritoneum. The duodenum was not seen, transverse colon and small intestine reported as normal. No recurrence of vomiting since the operation. Moderate drainage from gastric fistula. After a day or two the patient became drowsy, and later lapsed into unconsciousness, and died on the seventh day from apparently a diabetic coma and general debility. Whether the effect of the fall and the broken ribs, or the toxemia from the diabetes exercised the greater influence in this case is a question.

Of the surgical causes, narcosis comes first, chloroform being the anesthetic most at fault. In some experiments on dogs, Kelling showed that without anesthesia, he could dilate the stomach only up to a pressure of 25 cc. of water, and at this point it was released. Under narcosis, however, the stomach could be blown up to a pressure of 70 cc. of water.

Next in importance are those cases following operations upon the bile passages. Kelling considers that the obstruction in the duodenum in these cases is not due to any pulling on the duct, but either from a local peritonitis or by pressure of dry gauze packing which expands as it becomes moistened.

Kelling states that there is a certain amount of chronic dilatation of the stomach in cases which have had a number of gallstone attacks. The application of plaster jackets for kyphosis and scoliosis has been noted in a few cases. In addition to operations upon the stomach and bile passages, it has developed after surgical operations upon the trunk and extremities.

Contrary to Rokitsansky's original statement, the condition seems to be more common in middle life than in old age. Sex seems to have no etiological bearing.

Symptoms: The symptoms of this condition are vomiting at frequent intervals of large quantities of fluid, which may be greenish or yellowish at first, but which later becomes very dark brown or black. The quantity at times may be enormous. As much as a liter at one time has been observed. It is usually ejected with little or no effort. The vomitus is at first odorless, later becoming more offensive, never feculent. Müller reports a case in which following drainage of the bile ducts, the same fluid escaped from the biliary fistula that was vomited from the stomach, pointing to an obstruction below the opening of the common duct. Meyer and Hochhaus report similar observations.

Abdominal pain is frequently observed in the early stages, but it is usually not pronounced later. Distension, beginning in the epigastrium and some times extending much lower, is usually

present. Tenderness is not marked, constipation is usually present, but frequent watery stools have been occasionally observed. The great amount of fluid vomited gives rise to intense thirst, which is one of the prominent features. The pulse is usually rapid, temperature normal or subnormal. Collapse is a late development. The respirations are shallow and rapid. The amount of urine is much reduced, amounting, at times, to almost complete anuria.

Diagnosis: The diagnosis would appear to be easy, past experience has proved otherwise. The appearance in debilitated persons of frequent and copious vomiting of dark colored fluid, either associated with disease or following surgical operation, or injury, later the development of abdominal tumor, characteristic pain, elevation of pulse and normal or subnormal temperature, present a picture which, when seen once, one will not fail to recognize again. In cases where the clinical picture is not so pronounced, difficulty may be experienced in arriving at a correct diagnosis. Curiously enough, the large majority of cases have not been recognized, but have been mistaken for intestinal obstruction or peritonitis, which are the chief conditions one has to differentiate. Uremia, pancreatic cyst, encysted abscess of the peritoneum, chloroform toxemia, ovarian cyst, have all been mistaken for acute dilatation. Peritonitis is the condition for which the trouble is most often mistaken. In the post-operative cases where the element of infection is present, special difficulty will be experienced. The patient presents a picture not unlike that of a general peritonitis, an anxious, pinched expression of countenance, abdominal distension, which is apt, however, to be more uniform in peritonitis: the abdominal pain and tenderness which is also usually more marked in peritonitis; a rapid pulse and great thirst, all are frequently observed in both conditions. The temperature, notoriously unreliable in peritoneal inflammation, may or may not be elevated. In doubtful cases one must seek help from special aids in diagnosis. The stomach tube will be of service, frequently distension is markedly relieved by its introduction. The presence of succussion is strongly suggestive. A leucocyte count, though frequently unreliable, is of some assistance.

Recent investigations upon the opsonic content of the blood made by Dr. Charles E. Simon, in a series of abdominal operations occurring in my service at the Union Protestant Infirmary, show very high values in certain cases of peritoneal inflammation. In a small number of cases of intestinal obstruction, on the other hand, values were obtained which were essentially normal. This suggested a series of experiments to ascertain to what extent the determination of the opsonic content of the blood might be of service in distinguishing between infective and obstructive cases. In the latter, there was found to be very little deviation from the normal, while the infective cases showed comparatively high values. Our experiments as yet are too few to warrant any definite conclusions, but the results

obtained are suggestive and worthy of further study.

Careful examination of the urine will usually establish the diagnosis of uremia. In many instances, the diagnosis can only be established by an exploratory incision or at autopsy.

Treatment: In the majority of cases, treatment has been of no avail. If the diagnosis is established early, the frequent use of the stomach tube will be found of great service. The position of the patient in bed is of great importance. Dorsal decubitus should be avoided. The patient should lie on the right side or on the abdomen, with the hips elevated. In refractory cases, the knee-chest position should be tried. Large enemata have been recommended with the idea of distending the large intestine.

My associate, Dr. W. A. Fisher, has suggested the dilatation of the sphincter, and in the knee-chest position in order to allow inflation of the rectum with air and thus push up the small intestine from the pelvis, in the effort to relieve the tension upon the superior mesenteric vessels, a suggestion, possibly of some value.

Strychnine and atropine in large doses hypodermically have been recommended. Abdominal pads and binders of one sort or another have also been applied. Where these measures have been tried and failed, recourse must be made to further surgical measures. Manual replacement of misplaced viscera should be carried out. As a last resort, gastro-enterostomy has been advised. It was first successfully performed by Robinson, of Chicago. Gastro-mesenteric ileus developed, however, in Zade's case subsequent to a gastro-enterostomy for carcinomatous stricture of the pylorus.

It would appear, therefore, that too much reliance should not be placed in this procedure. Robinson has proposed the section of the duodenum and its reunion again by suture in front of the mesenteric vessels, a thoroughly impractical procedure.

Prognosis: The vast majority of cases have resulted fatally in spite of all measures instituted for their relief. Recently, however, a few cases of recovery have been reported. Schnitzler and others have been able to save cases treated by postural methods and Mayo Robson in his book reports three recoveries. If the diagnosis is made early and frequent lavage through the stomach tube instituted at once, we may hope that an increasing number of recoveries will be secured. Unfortunately, one cannot expect much from secondary operations on account of the condition of the patient and the necessity for a second anesthetic. Kerr reports one case successfully operated upon under Schleich's solution.

CONCLUSIONS.

- (1) Acute dilatation of the stomach and gastromesenteric ileus cannot be differentiated clinically.
- (2) Obstruction to the lumen of the duodenum by the root of the mesentery and the contained superior mesenteric vessels, has been demon-

strated, and is probably of more frequent occurrence than has been supposed.

(3) Whether this is primary or secondary to the gastric dilatation, or whether this relationship is a constant one, has not been determined.

(4) The diagnosis would appear to be more easy than past failures seem to indicate.

(5) The use of the stomach tube and avoidance of dorsal decubitus offer better results probably than secondary operation, owing to the unsatisfactory conditions existing.

(6) With earlier diagnosis and the early institution of the measures just suggested, an improvement in the very high rate of mortality can be confidently expected.

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SPONTANEOUS RUPTURE OF THE HEART WITH REPORT OF A CASE.

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WHILE death from spontaneous heart rupture is not an extremely common occurrence, yet it is by no means as rare an accident as was formerly supposed. The increasing number of autopsies in cases of sudden death proves that the condition referred to is substantiated more frequently each succeeding year. Devergie (quoted by Councilman), in 1840, reckoned that 2½% of all cases of sudden death were caused by this accident. Aran (also quoted by Councilman) in analyzing 202 cases of sudden death attended by pathological changes in the circulatory system, found 88 to be due to spontaneous heart rupture. Since the time of these communications, there has been a steady increase in the number of cases reported. Councilman and Somers both emphasize the fact that spontaneous cardiac rupture is not extremely rare. Statistics collected from the records of institutions are very contradictory as to this point — Meyer found only 7 cases among the records of between 12,000 and 13,000 autopsies performed at the Munich Pathological Institute, while Hamilton found an equal number of cases among the comparatively small number of autopsies recorded at the Independence State Hospital for the Insane (Iowa). It is apparently true, however, that this accident is much more often met with among the insane than in any other class of patients.

Quain's assertion that spontaneous rupture never occurs in a normal heart, has been confirmed by the autopsies in which a careful micro-

scopical examination has been made. We have, then, in this myomalacia cordis, the predisposing etiological factor. Fatty degeneration of the myocardium, atheroma and thrombosis of the coronary arteries, myocarditis, cardio-valvular disease, ulcerative endocarditis, syphilitic changes in the myocardium, cysts and neoplasms of the heart wall, are all capable of acting as direct causes. Segmentation of the heart muscle has been found in a few cases, but it is questionable whether this condition is pathological or an artifact produced by the microtome.

There is generally some mental or motor excitement which acts as a determining cause in the production of the rupture. Osler, Quain, Bruce and others state that mental or motor excitement usually precedes the accident. W. J. Greig has described a case where the rupture occurred while a stomach tube was being passed. Moore reports a case seen by T. A. Dixon, where a rupture was caused by strangulation in a boy of eight years. Morgagni tells of a case of heart rupture which occurred during coitus, and the accident occurs strikingly often in stallions during the sexual act. It is a question whether the cases following non-penetrating wounds of the thorax should be considered spontaneous or traumatic; rupture of the papillary muscles, aortic and mitral valves have occurred after slight contusions in the cardiac region, falls, etc. Experimental increase of intra-ventricular pressure does not bring about rupture in the normal heart, but increased blood pressure serves as an accelerating cause when the myocardium is diseased.

On the other hand, many cases occur when the patient is either asleep or in repose. Foote, McIntosh, Mickle, Williams, Hamilton and others have described cases where no excitement or exertion preceded the rupture. Grief and intense mental disturbance have been enumerated as causes. It would be interesting to trace the genesis of the traditional term "broken heart," although its present meaning does not imply any pathological condition of that organ. The death of Philipp V of Spain, following so soon after the defeat of the combined French and Spanish forces by the Imperialists at Piacenza, has been ascribed to grief, and there is no reason why the mental strain could not have hastened the rupture which caused his death.

By far the greater number of cases occur in persons over sixty years of age. In Quain's analysis of 100 cases, 63 were over sixty years old and, out of this number, 33 were between sixty and seventy years, while 24 ranged between seventy and eighty years. In those cases of Meyer, where the age was recorded, over 60% were sixty years or older, only 3 cases being under forty years of age. Very rarely does this accident affect persons in early life. The rupture cases occurring in people not far advanced in life include Elleaume's case of a woman aged twenty-two, Groom's case of a boy of sixteen years, Moore's report of Dixon's case of a boy aged eight, and Green's description of a case occurring in a child of only nine and one-half months.