

ACUTE DILATATION OF THE STOMACH—GASTRO-MESENTERIC ILEUS.

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In the study of 459 cases of hernia finished in January, 1899 (*The Johns Hopkins Hospital Reports*, vol. vii), I was not familiar with postoperative or primary acute dilatation of the stomach. For this reason I looked upon the only example of postoperative obstruction in this group of cases as a high intestinal partial occlusion. The patient was relieved, and recovered without a secondary operation.

In May, 1901, I operated for the first time for symptoms of intestinal obstruction following an operation under chloroform narcosis for reducible inguinal hernia. In this instance a loop of jejunum was caught in Treitz's fossa. The patient recovered. In the report of this case (*ANNALS OF SURGERY*, 1903, vol. xxxviii, p. 806) the possibility of an acute dilatation of the stomach is not discussed.

Later these two cases will be mentioned in the differential diagnosis between gastro-mesenteric ileus and high occlusion of the small intestine.

In March, 1904, my attention was first called to this postoperative complication in a case of appendicitis; here the clinical picture was distinct, the rapid epigastric distention associated with vomiting of large quantities of duodenal and gastric contents, and the rapid prostration differentiated the lesion from a high intestinal obstruction. The autopsy by Dr. Fisher confirmed the clinical diagnosis.

Such a distressing experience called my attention to the literature, which I should have been familiar with before. From now on I kept this postoperative complication in mind.

In September, 1904, when Dr. Sowers told me of the clinical picture of a patient who had died that day in the

surgical ward of the Johns Hopkins Hospital and on whom an autopsy was about to be performed, it was not difficult to conclude that death was due to a gastro-mesenteric ileus. The illustrations (Figs. 3 and 4) were made from my sketches taken at the autopsy. This case, operated upon by Dr. Finney, has been reported by him (Boston Medical and Surgical Journal, vol. cly, August 2, 1906, p. 107).

In July, 1906, I observed for the first time a lesion of the duodenum and the stomach which I think may be looked upon as a primary chronic gastro-mesenteric ileus, or chronic dilatation of the duodenum and stomach (see Fig. 6).

In March, 1907, I saw for the first time a case of primary acute gastro-mesenteric ileus. The complication had its onset during the convalescence from a very severe attack of acute articular rheumatism (see Figs. 1 and 2).

In discussing the cases which have come under my observation I feel that the subject can be presented best by first giving in detail the last observed case. Here the clinical picture was unusually distinct, and I was able at the operation and autopsy to investigate the pathologic anatomy.

CASE I, Path. No. 8025, J. D., patient of Drs. Pound and Seegar.

Primary acute gastro-mesenteric ileus, symptoms three and a half days; onset during convalescence from a severe attack of acute articular rheumatism. . . The symptoms began directly after the first solid meal, in a patient greatly emaciated and weakened by a long illness. The symptoms consisted of epigastric distention, gastric dilatation and peristalsis, vomiting of large quantities of gastric and duodenal contents, flatness of the lower abdomen and absolute constipation. The leucocytes were 40,000. There was no fever.

Operation; great dilatation of stomach and duodenum up to the mesentery, collapse of the small intestines; jejunostomy; death (Figs. 1 and 2).

I saw this patient on March 11th, 1907, with Drs. Pound and Seegar of Baltimore. The patient was a male, aged twenty-four. He was the only child of rather poorly developed parents and had always been delicate. About six weeks before he was taken

with an attack of high fever associated with great swelling of many joints. The condition was looked upon as acute articular rheumatism. He was critically ill. There was every evidence of some cardiac lesion. After the fever disappeared and the joint symptoms subsided the patient continued to have a very rapid pulse. His emaciation and weakness became extreme. During the beginning of the attack he was given large doses of salicylates.

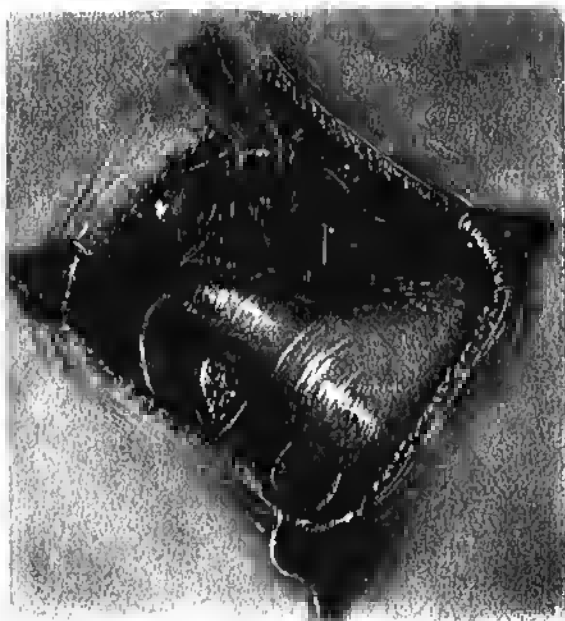
For about a week the patient had been better, the pulse rate had reached normal.

The onset of the acute attack began a few hours after his first meal of solids which included raw tomatoes. I saw the patient three and a half days after the beginning of the symptoms. The clinical picture from the onset had been the same—continuous epigastric distress, with intermittent colicky pains, fullness of the upper abdomen, flatness of the lower abdomen, vomiting of large quantities of a dark brown fluid at irregular intervals. The constipation was absolute; the quantity of urine diminished; there was no fever. The pulse had become more and more rapid. The treatment had consisted of high enemas and cathartics. The stomach tube had not been used.

Examination.—The patient is extremely emaciated; although the face is pale and sallow the lips have a bright red color. The pulse varied from 130 to 140; it was very weak. While I was looking at the patient he complained of a colicky pain and vomited about six ounces of a dark brown fluid, which contained mucus and a little blood. This fluid had a sweetish odor and did not remind me of normal gastric contents, but of the contents of the duodenum. As one looked at the abdomen there was distention in the epigastrium with an almost scaphoid abdomen below. When the patient complained of a colicky pain one could see a broad peristaltic wave pass from left to right in the area of distention between the costal margin. The distended area was tympanitic, and this tympany extended on the left to the nipple line above and the posterior axillary line behind; less to the right, partially obliterating the liver dullness. On palpation one could not positively make out the stomach because the muscles were held rigid and the patient complained of some tenderness. The lower abdomen however was soft, the rectal examination negative.

A diagnosis of gastro-mesenteric ileus was made and the possibility of a high intestinal occlusion considered.

FIG. 1



CASE 1.—Acute gastric and duodenal dilatation. Appearance of stomach and duodenum at operation. Author's case.

FIG. 2.



CASE 1.—Acute dilatation of the stomach and duodenum. Appearance of stomach, lower portion of duodenum, mesentery and collapsed jejunum; transverse colon filled up. Author's case.

After the patient reached the St. Agnes Hospital the leucocytes were found to be 40,000. During his transportation he continued to vomit and the pulse had increased to 150. On passing a stomach tube at least a litre of dark brown fluid was evacuated. It required at least ten minutes before the washings came away clear. This suggested that there was a dilatation at least of the duodenum as well as the stomach. The patient was given a subcutaneous salt infusion. The distention rapidly disappeared, his pulse improved and he said he was more comfortable. After an observation of one hour the distention returned. I now thought that perhaps the condition might be due to a high intestinal occlusion for which operation was indicated, and for this reason advised exploration of the abdomen. I am now of the opinion that this was a mistake, and that treatment by the stomach tube and salt solution infusion should have been continued. This treatment is the best for gastro-mesenteric ileus, and in this instance, if there had been an intestinal occlusion lower down, an operation three and a half days after the onset upon a patient so critically ill would promise too little to justify the procedure, especially as the symptoms favored gastro-mesenteric ileus. I consider the operation at this time in this case a mistake in judgment.

Operation Findings.—When the abdomen was opened through the right rectus under cocaine infiltration an enormously distended stomach presented itself. The lesser curvature was pushed against the liver, the greater curvature below the level of the umbilicus. As the stomach was full of gas it bulged out of the wound like a balloon and I had to push it back into the abdomen to examine the duodenum. The duodenum appeared as a dilated U-shaped tube situated high under the liver; the base of the U lay a little to the right of the gall bladder notch (Fig. 1). I wish to emphasize first that there is no gastropnoxis. Although we see adhesions between the duodenum and the gall bladder in the region of a normal mesenteric attachment, these have not produced the obstruction.

The transverse colon was collapsed, and when I lifted it with the omentum out of the wound I exposed the dilated stomach and duodenum (Fig. 2). The duodenum was tense and dilated up to the position of the mesentery. On the other side the jejunum was collapsed. The stomach and duodenum appeared like a

blown-up balloon, the duodenum fixed, the stomach movable. At the position of the junction of the duodenum and jejunum over which the mesentery and its vessels pass, the stomach pressed downwards with great force. There were no adhesions at the position of obstruction, nor could I by compression force anything from duodenum to jejunum. The obstruction was situated directly behind the mesenteric vessels. As shown in Fig. 2 the duodenum, before it passes beneath the mesenteric vessels through the mesentery, curves upwards and then, as it passes through the mesentery and becomes the jejunum, curves downwards, like the letter S. All the small intestines were collapsed, and it did not appear to me that their position was unusual. In many autopsy cases it is noted that the majority of the small intestines is in the pelvis. I could not demonstrate this in my case. It is very difficult to illustrate correctly the exact picture. The operator obtains his view of the duodenum first on the upper side of the colon, then on the lower by pulling the transverse colon up and down. He sees the contrast between the dilated duodenum and the collapsed jejunum by looking first to the left of the mesentery, and then to the right. The greater portion of the dilated stomach does not come into view. The diagnosis of an obstruction at the duodeno-jejunal junction is first suggested by the continuation of the dilatation of the stomach into the duodenum—an entirely different picture from that observed when the obstruction is at the pylorus. The diagnosis is confirmed the moment one lifts the transverse colon and exposes the duodenum and jejunum. In the case under discussion it required less time to see these things and to make the diagnosis, than to describe them.

Finding that I could not compress the contents of the duodenum into the jejunum, although I could pass my index finger through the mesenteric opening, invaginating either duodenum or jejunum, I decided to do a jejunostomy, and pass a tube into the duodenum. This was done with immediate relief. I considered that this procedure was more rapid and met the indications as well as a posterior gastroenterostomy. In addition, it evacuated the duodenum as well as the stomach.

The patient survived the operation about two hours. On opening the abdomen at the autopsy the tube was found in the duodenum which was empty, but still somewhat dilated, the stomach was very much smaller, but still large and practically empty.

Perhaps the duodenum was situated higher than usual in this case, but it seems to me that the high situation could be explained by its great distention and can be excluded as a factor in the obstruction.

Here is an uncontroverted observation of acute dilatation of the entire duodenum and stomach with no evidence of adhesions and no gross anatomical changes at the duodeno-jejunal junction different from normal. Whether dilatation was primary in the stomach or duodenum could not be established.

CASE II, Pathol. No. 8326, S. II., previously reported by Dr. Finney (loc. cit.).—*Acute postoperative gastro-mesenteric ileus. Death six days after operation (pyloroplasty, Finney's method). Continuous vomiting with rapid pulse after operation. Mucous excreted by pancreatic juice in the vomitus. Autopsy (illustrations, Figs. 3 and 4).*

The patient was a white female about thirty-five. Three years previously she had an acute attack of an abdominal lesion diagnosed acute gastritis, which had kept the patient in bed for three months. The details of this attack are not given in the history. The patient continued to suffer from epigastric distress and belching after eating. Two months before she was admitted to the medical clinic of the Johns Hopkins Hospital an operation had been performed on some pelvic organ. Since this time the patient has vomited almost daily.

From June 17 to September 7, a period of almost three months, she was under treatment in the medical clinic. The chief symptom was inability to retain food. In spite of attempts at careful feeding and rectal enemata with absolute rest in bed, the patient lost weight and strength, and the blood-count showed a progressive secondary anemia. At no time was it possible to make out a dilatation of the stomach, nor gastric peristalsis. The residuum in the stomach was never more than 75 c.c.; HCl was always absent; the total acidity was low—about 10; lactic acid was present. I emphasize this clinical picture, because it resembles my case of chronic dilatation of the stomach and duodenum.

At the operation by Dr. Finney the stomach was but moderately dilated. The duodenum was situated high under the liver, but there was no kink in the duodenum. The pylorus easily admitted the index finger. There was slight scar formation on

the anterior wall of the stomach near the pylorus. There is no note whether the duodenum was examined to the jejunal junction.

After operation the vomiting was worse. Before operation she had only vomited when given food. After operation she vomited at intervals of from three to six hours. The vomitus was green and then dark brown in color; it excoriated the mouth as if it contained pancreatic juice. Associated with this there was a very rapid pulse—128 to 140. At first there was moderate epigastric distention, but this with the vomiting ceased twenty-four hours before death. The patient had a number of stools, more than one would expect from the nutritive elements. The stomach tube was passed on the fourth day only: the result is not noted in the history.

Dr. Sowers told me about this patient the day I came on duty at the Hospital. The clinical picture impressed me as one of postoperative gastro-mesenteric ileus. As an autopsy was about to be performed, we immediately went to the pathological laboratory and found that the abdomen had been opened, and the small intestines handled. Dr. Francis who was performing the autopsy allowed me to examine the stomach and duodenum and to make sketches.

The findings were almost identical with those described and illustrated in Case 1, except the stomach was but moderately dilated and was not pressing upon the mesentery; the duodenum was just as distended, but was not situated as high as in Case 1. This is easily explained by the fact that in the Finney pyloroplasty the duodenum is freed and pulled down in performing the suture.

In Fig. 3 the small intestines have been removed: the stomach is not dilated. The characteristic changes in the anatomy of the pylorus after a Finney pyloroplasty are beautifully shown: the dotted lines represent the suture. Just below this in the duodenum there is a kink always observed after this operation, but which has produced no obstruction. The suture had healed perfectly; it was covered with a thin fibrinous exudate and slightly adherent to the liver. The dilatation of the duodenum and its marked U-shape is well shown, and should be compared with Fig. 1. When I cut away the transverse colon and the small intestines the dilatation of the duodenum up to the mesenteric vessels, and the collapsed jejunum forming the S-curve were distinctly seen, and are shown in Fig. 4, which should be compared

FIG. 3



CASE II.—Acute dilation of stomach and duodenum after a Flapley gastrojejunostomy; drawing made from sketch by author of autopsy. Small intestine cut away.

FIG. 4.



CASE 11.—Same as Fig. 3: transverse colon cut away. Note relation of mesenteric vessels to position of obstruction.

with Fig. 2. When I cut the jejunum near the mesentery it was empty and nothing escaped. When I pressed upon the duodenum there was no leakage. But when I passed my index finger through the jejunum into the duodenum a large quantity of thin, brown fluid escaped, and the duodenum collapsed. At operation, therefore, in Case 1, and at autopsy in Case 2, compression of the duodenum could not force its contents past the obstruction behind the mesenteric vessels into the jejunum. It seemed to me there was a valve-like kink due to the tension of the distended duodenum, and not to traction on the jejunum by the weight of the small intestines—an explanation quite frequently given in the literature.

CASE III, Pathol. No. 7442, Mrs. R.—*Chronic gastro-mesenteric ileus, or chronic dilatation of duodenum and stomach. Exploratory laparotomy. Death in twenty-seven days. Partial autopsy. Illustration, Figs. 5 and 6, of findings at operation.*

This patient, a white female aged forty-two, was referred to me by Dr. Carr. She was a married woman and until a year ago had been comparatively well. She states her illness, however, to an attack of mumps from which she suffered about two years ago. During her convalescence she suffered from abdominal pain and nausea which have been present off and on since. During the last year she has had five acute attacks, the last five weeks ago. These attacks consist of pain in the left side of the abdomen with nausea and vomiting. The severe attacks last about three days and are relieved by calomel. Between the attacks the patient suffers with indigestion and belching. With the last attack, five weeks ago, there was diarrhoea.

Examination.—The patient is thin and not well nourished, restless, nervous and unusually apprehensive. Nothing is to be made out in the abdomen, except resistance in the epigastrium and a somewhat indistinct mass situated between the umbilicus and costal margin on the right side. This mass has somewhat the shape of a pigeon's egg and is about 5 by 2 cm. in diameter. It could be moved (Fig. 5) from side to side, but not from above down. It was slightly tender on pressure.

In view of the age of the patient and the palpable mass an exploratory laparotomy seemed justifiable.

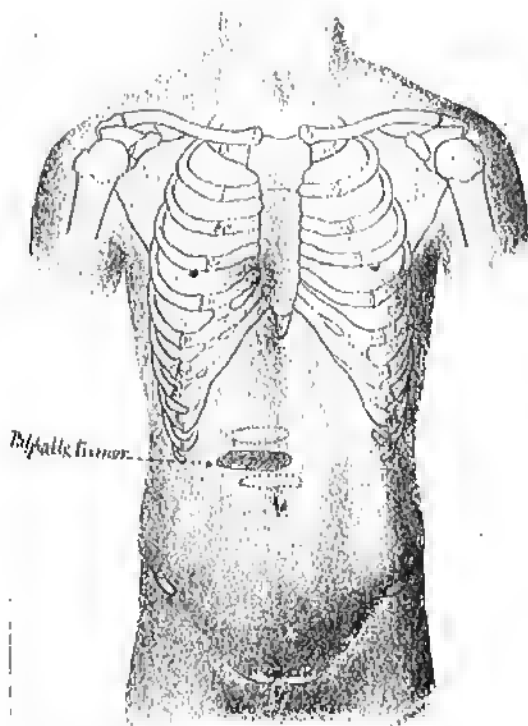
On opening the abdomen there was practically no subcutaneous fat and very little fat in the omentum. The stomach was but moderately dilated. There was no obstruction or scars

in the region of the pylorus. The condition that at once struck the operator as unusual was in the duodenum and pancreas (Fig. 6). The duodenum was a dilated, U-shaped organ extending high up under the liver and surrounded the pancreas. The duodenum and pancreas were unusually movable, the latter felt hard and indurated, the duodenum, although somewhat dilated, was soft and compressible. There were no adhesions. The third portion of the duodenum was less movable, and of about the same caliber. The entire small intestines were unusually small and ribbon-like—a condition which I have observed in a few instances when the abdomen had been opened in patients who had taken little food for a number of weeks (starvation atrophy). The enteroptosis of the stomach was but slight. The duodenum and pancreas, although quite movable from right to left, could not be pushed down, nor could I pull the duodenum out of the abdominal wound. In my only laparotomy for extreme gastropnoxis I found that I could lift the duodenum and pyloric end of the stomach out of the abdominal wound as easily as a loop of small intestine. In this case the duodenum, although dilated throughout, was compressible, and there was no evidence of any accumulation of its contents. Yet the contrast between the duodenum and the jejunum as to caliber was unusual.

I have never met with, nor read of, a similar condition, and as at this time I was somewhat prejudiced against the posterior gastroenterostomy without definite indications, I closed the abdomen. In the case of gastropnoxis just mentioned, the duodenum was almost as dilated and the contrast with the jejunum almost as great. As this patient had made a splendid recovery without posterior gastroenterostomy, after rest in bed and daily use of the stomach tube, I felt that the patient in Case 3, should do as well.

After operation nausea and vomiting were prominent symptoms, but there was absolutely no distention in the epigastrium, no dilatation of the stomach, no peristalsis of the stomach: the vomiting now and then contained bile; there was never any evidence of pancreatic juice; the bowels moved normally. For a few days the patient would take food, then there would be a recurrence of the inability to retain even water. Mentally, the patient was extremely depressed and apparently made up her mind to die. The intervals of slight improvement with ability to

FIG. 5.



CASE III.—Diagram of palpable rhinopharyngeal tumor.

FIG. 6.



CASE III.—Chronic dilatation of stomach and duodenum. Sketch from operation findings, Antile's case.

retain food for a few days encouraged the postponement of a posterior gastroenterostomy. The patient died suddenly the morning of the day selected for the secondary operation, five weeks after the primary operation. Dr. Carr was able to get an autopsy only after the body was embalmed, and the findings were the same as at the exploratory laparotomy. The embalming fluid prevented properly stained sections of the pancreas. There seemed to be, however, slight interstitial pancreatitis; the stomach and duodenum were practically normal.

These are the only three cases in which I have fairly complete operative and autopsy findings.

In all three cases the obstruction began abruptly at the junction of the duodenum and jejunum, beneath the mesenteric vessels. In Case 1 the duodenum and stomach were tremendously dilated, the duodenum fixed in a high position. In Case 2, at operation, the stomach and duodenum were moderately dilated; there was no positive evidence of pyloric obstruction; the duodenum was not examined in its third portion; at the autopsy in this case the duodenum was dilated down to the mesentery, the stomach was only moderately dilated. In Case 3 the duodenum and stomach were but moderately dilated, but the duodenum, except in its lower third, was abnormally movable. Case 1 is a typical example of primary acute dilatation. We know nothing about the condition of the stomach and duodenum before the attack. Cases 2 and 3 give a clinical history which resembles a gastric neurosis more than pyloric stenosis or ulcer. I am of the opinion that chronic dilatation of the duodenum was present before operation in both cases (2 and 3). In Case 2 the condition became acute after operation. There is no evidence that the pyloroplasty was a factor in its production, and there is every reason to believe that this pyloroplasty did not and could not relieve the original condition. In Case 3, the exploratory laparotomy had no effect whatever on the clinical picture. In this third case the stomach was washed out two and three times a day; this procedure may have prevented acute dilatation, but it had no curative effect upon the chronic condition.

Theoretically, posterior gastro-jejunostomy with a short loop may have been indicated in both cases. This procedure would have relieved gastric stasis, but might have increased the duodenal dilatation. Theoretically, also a second anastomosis between the duodenum and jejunum would have to be performed to drain both duodenum and stomach. Future experience may demonstrate that in cases of this character a simple duodeno-jejunostomy is indicated. This operation, as far as I know, has never been done, nor even suggested before. It certainly meets the indications better than gastro-enterostomy and is simpler than a combined gastro-enterostomy and duodeno-jejunostomy.

In all three cases the small intestines were collapsed, with absolute constipation in the first case, but some movement of the bowels in the other two cases. In none of these cases can it be positively said that the small intestines were in the pelvis. In the two cases which I observed at operation (1 and 3) the majority of the small intestines were not in the pelvis. Free fluid in the peritoneal cavity was absent in all, and there were no adhesions and no gross anatomical lesions to explain obstruction at the mesentery. Nor was I able to ascertain whether the dilatation of the stomach was primary or secondary.

As stated before, I am of the opinion that in Case 1 the stomach tube should have been used for a longer interval before the patient was subjected to operation; it also should have been employed by his attending physician from the onset of the disease. In Case 2 operation was indicated; the patient had been given the benefit of the best medical treatment, during which time she lost strength and flesh and became more anemic. After operation here, the stomach tube was not employed as it should have been, once or twice a day or more frequently. In Case 3 the palpable tumor was sufficient indication for exploration. In the postoperative treatment nothing was neglected. Whether this patient could have been cured by a duodeno-jejunostomy, remains to be answered by future experience in other cases.

FIG. 7.



CASE IV.—Acute postoperative dilatation of the stomach. Finney's case. Photograph from alcohol specimen.

CASE IV, Pathol. No. 7352.—*Acute postoperative gastric dilatation (following a Finney gastro-duodenostomy), associated with pregnancy. Death one month after operation (Fig. 7).*

This case I have published with the illustration in the *International Clinics* (April, 1907, page 285, Fig. 27). The patient was a white female, aged twenty; for six months she had suffered from indigestion consisting of epigastric pain and nausea. For two weeks the symptoms had been very severe, with vomiting. There had been and still was discomfort directly after taking food, and vomiting two to three hours later. On one occasion blood was in the vomitus. No improvement under rest and milk diet. On examining the abdomen nothing was made out, except slight resistance and tenderness in the epigastrium. In the gastric secretion there was no HCl, and a total acidity of 28. The patient did not improve after a month's medical treatment. Pregnancy was denied and no pelvic examination made. At the operation by Dr. Finney the stomach was found to be but moderately dilated, the pylorus was slightly constricted: there was no definite scar tissue, but a few adhesions about the pylorus. The pylorus seemed higher than normal, and it is noted that the duodenum was contracted.

A Finney gastro-duodenostomy was done. After operation the patient's condition on the whole was worse—continuous vomiting, which excoriated the mouth; slight jaundice and bile in the urine. The possibility of pregnancy was then admitted by the patient.

Autopsy.—The dilatation is confined to the stomach with a kink in the lesser curvature which, however, did not obstruct the pylorus (see Fig. 7). The pyloroplasty wound had healed; the small intestines were contracted; the adhesions between the pylorus and liver noted at the operation were still present.

The fetus in the uterus indicated a four months' pregnancy.

Evidently this is an example of acute gastric dilatation following operation, yet at autopsy there was no evidence of pyloric obstruction. The note, however, on the duodenum, both, at operation and autopsy, is not sufficiently explicit to exclude duodenal dilatation. In the specimen sent to the laboratory there is but a small piece of duodenum: this is dilated.

As the patient gave a history of six months' gastric discomfort, was under treatment one month before operation, and died one month after it, the gastric symptoms antedated the pregnancy four months. The marked nausea and vomiting were of but two weeks' duration and therefore coincident with the sixth week of pregnancy. At the operation there was sufficient pyloric stenosis to explain the gastric symptoms. My colleague, Dr. Williams, professor of obstetrics, tells me that he has never observed acute dilatation of the stomach in the vomiting of pregnancy. He recognizes two forms of vomiting in the gravid woman: the first—of neurotic origin—is usually cured by suggestion; the second—toxic—is only relieved by immediate evacuation of the uterus. The liver in this case, in gross and microscopic study, did not have the appearance of that observed in the toxic vomiting of pregnancy. For this reason we must look upon this case as an acute postoperative gastric dilatation, and in view of the large opening between the duodenum and stomach made by the anastomosis, we can exclude pyloric stenosis as a factor. Although unfortunately the autopsy record does not allow us to make positive statements with regard to the duodenum, I am inclined to the view that this case is identical in its etiology with the three already discussed.

CASE V, Pathol. No. 608, J. O.—*Postoperative acute dilatation of the stomach secondary to appendectomy, drainage, and enterostomy for perforating appendicitis with general peritonitis and intestinal obstruction. The patient died twelve and a half days after operation.*

This patient, referred to me by Dr. Murphy of Annapolis, a white male, aged twenty, was operated on in the fourth day of an attack of acute appendicitis: the appendix, situated in the pelvis, was perforated; there was pelvic peritonitis with a considerable collection of pus requiring pelvic drainage. Two and a half days later an enterostomy had to be performed for obstruction due to the pelvic peritonitis.

The patient did very well for a number of days. The upper tube drained well, but the loop seemed to be short, because the discharge consisted of material but slightly digested. As the

patient grew weak it was necessary to rather force the nourishment by mouth. On the fifth day after the second operation the patient had sudden epigastric distention, and in a few hours the pulse rose from 106 to 120; at the end of four hours he vomited 300 c.c. of fluid with immediate relief: the epigastric distention disappeared, the pulse improved, the vomiting ceased, and the tube began to drain again. In my notes made at that time I looked upon the condition as an acute gastric dilatation due perhaps to forced feeding in a patient weakened very much by his disease. On the ninth day, four days later, there was a second attack with syncope; as the vomiting had entirely relieved the distention the stomach tube was not used. After this attack the general condition of the patient became worse; we could not use the lower loop for feeding, as the obstruction was not completely relieved; the rectal enemas were not well retained; we were therefore forced to employ feeding by mouth. At five o'clock on the afternoon of the eleventh day there was no epigastric distention and no vomiting; this had been absent for forty-eight hours; the patient was taking moderate nourishing without nausea; the pulse, however, was rapid. Within a few hours he began to distend and vomit. Dr. Fisher, who was looking after the patient, attempted to pass a stomach tube, but it had such a serious effect upon the pulse that he considered it wiser to desist. The patient died in collapse a few hours later.

At the autopsy by Dr. Fisher the peritonitis had subsided. The enterostomy was about fifteen feet from the stomach; the intestines below this were adherent in the pelvis; there was no obstruction between the enterostomy and the duodenum; the stomach was enormously distended with fluid and gas. No special note was made upon the condition of the duodenum.

This case can be looked upon as one of acute postoperative gastric dilatation. There were no gross changes or adhesions in the region of the stomach to suggest pyloric stenosis. Whether the gastric dilatation was associated with a similar condition of the duodenum up to the mesentery cannot be positively stated.

CASE VI, Pr. Surg., No. 1483, J. P.—*Postoperative acute dilatation of the stomach following radical cure of left inguinal hernia under ether narcosis. Stomach tube. Recovery.*

White, male, aged fifty-eight. This patient gave no previous history suggesting any gastric trouble. He was, however, a thin,

delicate looking man, and as the left inguinal hernia was a large one, he was carefully prepared for operation. This preparation consisted of, first, a saline cathartic; then forty-eight hours' rest in bed on very light diet; no further catharsis, and effectual rectal enemata on the morning of the operation. The sac contained omentum which was reduced; the Trendelenburg position was employed for about twenty minutes.

After operation the patient very quickly had epigastric distention and discomfort; the zone of tympany indicated that the chief distention was due to the stomach; there was no vomiting; the pulse was good and the leucocytes were but 10,000 (in Case 1 the leucocytes were 40,000). This patient was given immediate relief when the stomach tube was passed, but nothing was obtained except gas; the stomach was washed out; the water came away clear. On washing out the stomach one evening Dr. Smead, in charge of the patient at the Union Protestant Hospital, introduced some castor oil. The next morning the oil was still in the stomach. During forty-eight hours the stomach tube was passed about four times. No cathartics were given except the castor oil mentioned. Sixty hours after operation the patient passed gas and fecal matter per rectum for the first time, after an enema. From now on there was very little epigastric distention or distress. The stomach tube was passed on only two occasions in the next five days.

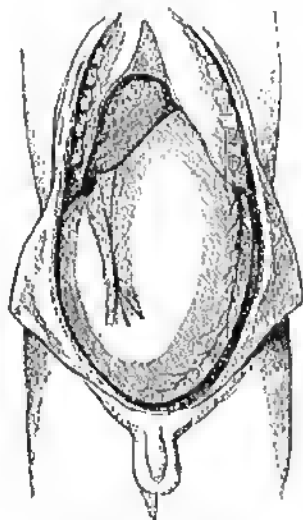
The stomach tube, after operation, is not only valuable in the treatment of postoperative gastric dilatation, but also in diagnosis. If one finds in the stomach intestinal contents, this may be looked upon as an almost positive sign of intestinal obstruction or gastro-mesenteric ileus.

Some six months later this patient was again operated on for a small right inguinal hernia associated with undescended testicle. In view of the previous complication local anesthesia with cocaine was employed. After this operation the same symptoms appeared, slighter in degree and of shorter duration, and were again relieved by the stomach tube. On both occasions there was no accumulation of fluid in the stomach. The distention was entirely due to gas.

It seems unnecessary to lengthen this article by a long discussion of the literature. Finney has presented the subject

in a very systematic manner in the Boston Med. & Surg. Journal, August 2, 1906. Halstead of Chicago (Surgery, Gynecology and Obstetrics, vol. ii, 1906, p. 13) reports an observation of his own in which the patient died after an operation consisting in fixation of the kidney. In this case the stomach and first portion of the duodenum were dilated; the obstruction was situated in the descending limb of the

FIG. 8.



Acute primary dilatation of stomach associated with retroperitoneal duodenal abscess.
Copied from Fagge.

duodenum as it passed beneath the peritoneum. Other cases with obstruction at this point have been reported; the huge stomach was somewhat V-shaped, like in Finney's case (Fig. 7).

In the *International Clinics* for April, 1906, p. 285, and April, 1907, p. 284, I have given a brief résumé of the more recent literature.

WICHMAN (Mittheilungen a. d. Grenzgeb. d. Med. u. Chir., 1906, vol. xvi, page 791) is the first to record this complication during the acute stage of typhoid. At the autopsy in his two cases there was no dilatation of the duodenum, differing, therefore, in the anatomical findings from my Case 1. This complication has been observed during the convalescence from typhoid and in the acute stage of pneumonia and scarlet fever.

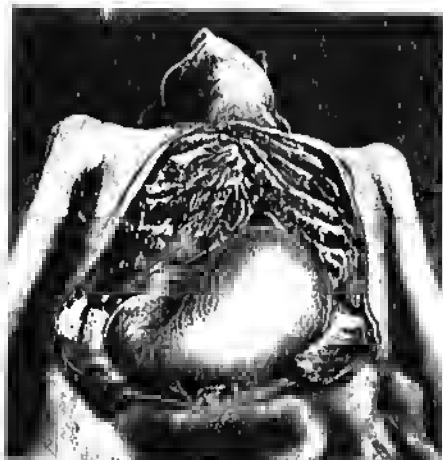
In C. HUYON Fagge's original communication (Guy's Hospital Reports, series iii, vol. xviii, 1872-3, p. 1), to which all writers refer as one of the earliest contributions to the subject, there is no note on dilatation of the duodenum. The V-shaped appearance of the hugely distended stomach is shown in Fig. 8, copied from Fagge. His two cases were not post-operative, but primary. In Fagge's Case 1 the cause of the obstruction was a retroperitoneal abscess communicating with the duodenum, probably due to perforating duodenal ulcer. In his second case there was no demonstrable cause for the obstruction.

Fig. 9 is copied from THOMSON (The Lancet, 1901, vol. ii, p. 1113). It illustrates the V-shape of the hugely dilated stomach, which was observed at autopsy, the patient also suffering from acute lobar pneumonia and pleurisy. In this case the dilated stomach reached the pubes. There is no note on the duodenum. The autopsy record simply states that the small intestines were collapsed.

Fig. 10 is copied from a second communication by THOMSON (The Lancet, 1902, vol. ii, page 287), and illustrates the autopsy of a case of acute dilatation of the stomach associated with dilatation of the upper eight feet of the small intestines. The complication began just before the birth of the sixth child of the patient and continued after it. The patient died about forty-eight hours after labor. The autopsy revealed peritonitis from a ruptured ovarian abscess.

Thomson uses this case as evidence against the view that in acute dilatation of the stomach and duodenum the distention begins at the mesentery, and that the lesion is a gastro-duodenal mesenteric ileus. There is no doubt that acute dilatation of the stomach has been observed to cease at the pylorus, in different portions of the duodenum, and, in a few instances, to extend below, into the jejunum. However, in the majority of cases the dilatation ends at the mesentery. I must agree with Thomson that this finding does not prove that the primary dilatation is in the duodenum, and that the mesentery is the etiological factor in the obstruction. Nevertheless, for practical purposes, it seems best to look upon this lesion as a gastro-duodenal dilatation, and if treatment by the stomach tube fails, not to neglect, at the operation, a careful exploration of the duodenum.

FIG. 9.



Acute gastric dilatation of the stomach associated with pneumonia. Copied from Thomson.

FIG. 10.



Acute dilatation of the stomach associated with the dilatation of the upper eight feet of small intestine and peritonitis. Copied from Thomson.

If the dilatation ends at the pylorus gastrostomy or gastro-jejunosomy should be performed. If the dilatation involves the duodenum, I would advocate jejunostomy as employed in my first case, or duodeno-jejunosomy.

The Differential Diagnosis between Acute Gastro-duodenal Dilatation and High Obstruction of the Small Intestines. In view of the fact that in the first lesion the consensus of opinion favors treatment by the stomach tube and position of the body (knee-chest, or left lateral with elevation of the pelvis), and that in the second lesion immediate operation is indicated, it seems important to ascertain, if a differential diagnosis is possible.

High intestinal obstruction is about as rare as acute dilatation of the stomach and duodenum. The following cases which I have observed apparently demonstrate that a differential diagnosis is possible.

CASE VII.—*Postoperative intestinal obstruction, loop of jejunum in Treitz's fossa. Operation. Recovery. (Previously reported in Annals of Surgery, vol. xxxviii, 1903, p. 807).*

As an etiological factor, it is of sufficient interest to know, that this patient—a physician, aged fifty—closed himself with cathartics for thirty-six hours before operation and then, a few hours before operation took paragon to check the diarrhoea. The operation was a simple one for an oblique, reducible inguinal hernia. The dissection was begun under cocaine and completed under chloroform. Vomiting with intense retching began the moment the patient came out of the narcosis and continued. The vomitus was at first bile-stained and later had the odor and contents of the jejunum. There was never acute pain, but general abdominal discomfort. Forty-eight hours after operation I noted that there was moderate asymmetrical distention of the right upper abdominal quadrant with increasing obliteration of the liver dullness. There was not the epigastric distention and distress observed in acute dilatation of the stomach. With the rectal tube some gas and fecal matter came away. The leucocytes rose from 12,000 to 16,000. There was no collapse. In Case I of acute dilatation of the stomach the leucocytes were 40,000 and collapse was marked. In view of the increasing obliteration of

the liver dullness and the vomiting of distinctly fecal matter the abdomen was opened fifty-six hours after the first operation. There was no free fluid. On manipulating the small intestines I found some distended and then I withdrew a loop with a definite constriction. The distention extended to, and abruptly ended at, the constriction; beyond this the intestines were collapsed. The constriction seemed to be in the lower portion of the jejunum and had the appearance as if the bowel had been impacted in one of the anatomical fossæ within the peritoneal cavity.

CASE VIII, Pathol. No. 7005, T. W. S.—*Acute intestinal obstruction due to the impaction of a bolus of food. Operation two and a half days. Death.* (Figs. 11 and 12.)

The clinical history in this case suggested acute dilatation, but the physical signs were those of high intestinal obstruction. The patient was a physician, aged sixty-nine, an unusually strong, robust, fat man. The symptoms began some hours after an unusually large Christmas dinner. He was first uncomfortable all over the abdomen, then there was localized pain on the left side between the umbilicus and the tenth rib. Twelve hours after this heavy meal the pain was so intense that he became nauseated; later vomiting, which within twenty-four hours became fecal in character. I saw the patient two and a half days after the onset. The pain had been less the last twenty-four hours, the vomiting of fecal matter continued. The patient did not look very sick; the pulse was 90°. There was no epigastric distention. The liver dullness was not obliterated. There was a distinct area of dullness and fullness in the left flank which was replaced by tympany when he turned on the right side. Although the character of the vomitus resembled that of Case 1 of acute dilatation of the stomach and duodenum, the physical signs of the abdomen were entirely different. When I washed the stomach out it contained but half a pint of distinctly fecal matter, and the washings were quickly clean. In my note made at that time I said: "This small quantity of fluid in the stomach would almost exclude acute gastro-duodenal dilatation and favor obstruction in the small intestine." Later I made out in the small intestine distinct peristaltic waves of the ladder pattern entirely different from the single broad peristaltic wave observed in Case 1. On opening the abdomen there were some clear fluid and distended small intestines; the stomach was contracted, the colon collapsed (Fig. 11).

FIG. 11.



CASE IX.—High intestinal obstruction; dilatation of upper portion of jejunum, not associated with dilatation of duodenum and stomach. A, B, C, D, E, F, G, H, I—dilated jejunum; N—position of foreign body behind this loop. Sketch at operation. Author's case. (Scheme copied from Roedel's illustration in Kelly's Operative Gynecology.)



CASE IX.—Position of foreign body and appearance of enterospasm, Author's case.

On pulling out the small intestines from the cecum up I encountered, first, a collapsed loop, then a movable foreign body 5.5 cm. long and 2.5 cm. in diameter (Fig. 12); just above this the intestine was constricted, and appeared as if in my manipulations the foreign body had been slightly dislodged; above the constriction the intestines were dilated. The enterolith was soft and could be easily crushed and pushed on. The constriction had the appearance of a local enterospasm and soon disappeared.

The patient died thirty-six hours after operation of anuria and symptoms of autointoxication. There were a number of stools after operation, but the patient continued to vomit some. The autopsy findings differed very little from those at operation. Apparently there had been complete small-intestinal paralysis. The point of obstruction could not be found, but as I had made an enterostomy to evacuate the distended small intestines above the constriction, I could locate its position. It was situated in the third portion of the jejunum. The latter and the duodenum were distended with fluid contents, and this distention with fluid was found eight feet below the point of obstruction. Beyond this the small intestines were somewhat contracted, demonstrating that intestinal contents had begun to move down. The stomach was not dilated. There was very little difference between the sizes of the jejunum and the duodenum on each side of the mesentery. There was no peritonitis.

CASE IX.—Intestinal obstruction from impacted gall-stone in the jejunum. Operation in forty-eight hours. Death.

R. J., white, female, aged eighty (patient of Dr. Hamburger). The symptoms of intestinal obstruction were distinct—initial pain and collapse, vomiting, later becoming fecal. As the patient became more toxic the pain became less. At my examination forty-eight hours after the onset, there was no epigastric distention or distress; there was very little abdominal distention; the vomiting was small in quantity and distinctly fecal. Peristaltic waves could not be made out. The leucocytes were 18,000; the patient's pulse was rapid and weak. On opening the abdomen a gall-stone was found impacted in the lower portion of the jejunum or the upper ileum. The stone was removed, the bowel evacuated, the enterostomy wound closed. The patient died a few hours later.

CASE X.—Acute intestinal obstruction by band. Operation on the third day with enterostomy. Recovery.

In this case acute dilatation of the stomach was not even suggested by the history, and the symptoms of intestinal obstruction were somewhat obscure. The patient was a healthy male, aged thirty-four, fat and a large eater. The symptoms began with abdominal discomfort and constipation. The initial acute pain and shock of high obstruction were absent. Later colicky pain characteristic of peristalsis were felt. Vomiting was present at rather long intervals. On the second day he vomited a pint of dark-brown colored matter. The distention of the abdomen was uniform and moderate. I was asked to see this patient on the third day by Drs. Thayer and Hammond. He was in excellent condition; the abdomen was but moderately distended; if peristalsis was taking place it could not be seen, as the patient was very fat. Obstruction indicated by the fecal vomiting was thought to be in the small intestine, as there was but little distention, although the history of constipation, colicky pain and late vomiting is that usually observed in obstruction of the large intestine. About the umbilicus there was an area of impaired resonance—a physical sign of fluid in the small intestine. The stomach tube brought away a large quantity of fluid of intestinal odor. On opening the abdomen, the upper portions of the small intestines were greatly distended; the obstruction was due a band in the pelvis below which the ileum was collapsed. As the band was divided the fluid contents passed. In view of the long duration of the obstruction the intestines were opened and evacuated, the opening sutured to the wound and closed for later drainage; this was again opened twenty-four hours after operation. The fecal fistula was sutured at the end of five weeks.

These cases demonstrate that the symptom-complex and the physical signs of high occlusion of the small intestine differ from acute gastro-duodenal dilatation. There are two symptoms in common—vomiting, and its fecal character. If my memory is correct, the odor of the vomitus which I carefully studied in Cases 1 and 6 (acute dilatation of the stomach) is less fecal in character than when the obstruction is in the small intestine. The character of the vomiting, however, is not sufficiently different to allow a differential diagnosis. In high intestinal occlusion initial pain accompanied, perhaps, by peritoneal shock which may later somewhat disappear, and

vomiting without marked distention are the symptoms which differentiate it from acute dilatation. In the latter the initial pain is absent; the patients suffer from epigastric distress; the collapse is gradual and progressive; the most characteristic feature is the abdominal distention beginning in the epigastrium and, in some cases, extending to the umb. This distention is immediately relieved by the passage of a stomach tube.

In high intestinal obstruction, in my experience, epigastric distention is a very late symptom, and so far in my cases at operation great dilatation of stomach and duodenum has not been found (see Fig. 11).

According to the most recent monograph by *Kayser* of which I have only the review (*Centralbl. f. Chir.*, 1907, xxxiv, p. 242), the mortality among about sixty cases of acute dilatation of the stomach has been 71 per cent.; recovery, with rare exceptions, has followed only the early and repeated use of the stomach tube. Among recent literature *Beck* (*Centralbl. f. Chir.*, 1907, xxxiv, p. 577) reports the recovery of a girl of fifteen, with symptoms forty-eight hours after appendectomy for appendicular abscess in the pelvis and drainage. These symptoms consisted of epigastric pain and distention. The stomach tube removed a liter of material, although the patient had had no food; in spite of nothing by mouth there was a reaccumulation of two liters in twenty-four hours; the stomach tube was used daily for four days. This observation of *Beck* demonstrates that there is a second factor in acute dilatation besides gastric atony or paralysis, and that is hypersecretion. In the literature, even in the most recent monograph by *Kayser* just mentioned, this factor is not dwelt upon with sufficient emphasis.

The observation of *Heile* (*Mittheilungen a. d. Grenzgeb. d. Med. u. Chir.*, 1907, v. Mikulicz Supplement, p. 707) is the best to demonstrate that hypersecretion is a definite factor. It is also of interest in that it illustrates that in chronic dilatation of the stomach with a history of hypersecretion one should be cautious not to select a pyloroplasty or Finney operation, or a Billroth (gastroduodenal end-to-end suture) after pyloroc-

tony, but perform a gastro-jejunostomy with a short loop. In two of the cases reported by me, in which acute dilatation up to the mesentery followed a Finney operation there was evidence of hypersecretion before and after operation in both cases. In my observation of chronic dilatation of stomach and duodenum there was no definite evidence of hypersecretion before, nor after operation. In Heile's case there was a history of pyloric obstruction in a woman of twenty-seven, of eighteen months' duration; vomiting of large quantities of food was the most marked clinical symptom; the contents of the stomach showed an acidity of 60, and free HCl acid; observation before operation demonstrated that the quantity of vomitus far exceeded the intake of food.

The palpable tumor proved to be, at operation, an infiltrating carcinoma in an old ulcer. After resection the duodenum was sutured to the stomach. After operation, in spite of no food by mouth, the patient began to vomit within forty-eight hours and continued to do so for sixteen days. The daily quantity averaged about three liters; it resembled gastric secretion unmixd with duodenal contents; the condition was not relieved by the stomach tube. The patient recovered after a posterior gastroenterostomy. The duodenum was not dilated; there was no mechanical obstruction at the suture; the remaining portion of the stomach which at the operation had been reduced to less than one half, was found, at the second operation, to be as large as the dilated stomach before the first.

This case of Heile, I believe, belongs to a group of acute dilatations in which further experience will demonstrate that after a sufficient trial of the stomach tube and irrigation without relief, operation is indicated: when the dilatation ends at the pylorus—posterior gastro-jejunostomy; when it is a true gastro-mesenteric ileus—gastro-jejunostomy with jejuno-duodenostomy, or the latter alone, should be preferred.

Acute dilatation of the stomach is a lesion which we must be constantly on the alert to recognize, both, as a primary and a postoperative disease. Its recognition should not be difficult. Prompt treatment with the stomach tube and irrigation, re-

peatedly performed, is the first requirement. [In some cases the tube may have to be passed through the nose.] This should be associated with change in position—knee-chest, and left lateral with elevation of pelvis. When the dilatation of the stomach persists and the accumulated fluid fails to pass, and the patient becomes weaker from loss of food, operation should be performed.

The chief etiological factor seems to be some toxic agent in a patient weakened, perhaps, by previous disease and suffering from some chronic gastric lesion. In some cases the toxic factor may be sufficiently great to produce paralysis with hypersecretion in a stomach previously dilated. This was apparently so in my first case. In the observations during and after typhoid, in pneumonia and scarlet fever, the patient is first weakened by the disease; then, later, the acute dilatation has its onset. It seems strange that acute dilatation of the stomach has only recently been observed in eclampsia (Andebert et Pournier: *Comptes Rendus de la Soc. d'Obst., de Gynec. et de Paed.*, 1907, vol. ix, p. 116). My attention was called to this article by my colleague, Dr. Williams. In the first reported case, there were two factors—the toxins of eclampsia and the chloroform used at the delivery; the condition was recognized and properly treated with the stomach tube, and although the patient was critically ill, she recovered. In the second case, there was a third factor—a general streptococcus septicemia from a uterine infection; in spite of early recognition and treatment, the patient died within eighteen hours after the first symptom; there was huge dilatation of the stomach, and, in addition, great dilatation of the cecum, ascending and transverse colon; there is no note on the ileo-caecum. In my Case 2 (Fig. 4) the cecum was dilated, and I found an adhesion kinking the ascending colon just above the cecum, not shown in the illustration. In this reported case, there was an adhesion kinking the transverse colon.

In regard to postoperative acute dilatation of the stomach, surgeons should give more care to the preparation of the patient before operation, especially in laparotomy cases. For at least

forty-eight hours food should be very much restricted; cathartics should not be used just before operation; when the operation is for a lesion situated some distance from the stomach considerable attention should be given to this organ in the history and examination. I have already used Heile's case as an observation of the importance of properly selecting the operation for relief of gastric dilatation with hypersecretion associated with stenosis or tumor. When this dilatation follows operations upon the gall-bladder or the duets, or any operation in which drainage has been introduced in the region of the duodenum, the drains should be loosened or removed immediately if there are any symptoms of acute dilatation. I now remember that Dr. Halsted had such a case in which the symptoms began with persistent vomiting of large quantities of gastric material twenty-four hours after cholecystectomy and drainage. I have had one such observation, in which the symptoms were of a lighter degree. Both recovered after loosening of the drain and the stomach tube.

Orthopedic surgeons must bear in mind the possibility of acute dilatation in their cases of kyphosis. These patients should be cautioned against over-eating, especially after a plaster jacket is applied. NECK (*Zentralbl. f. Chir.*, 1907, xxxiv, p. 58) in his most recent contribution since his collective review on this subject, reports four primary cases, with three recoveries after the use of the stomach tube; in two the patients were kyphotic and gave a history of over-indulgence in food; in one patient there was a history of only an excess of food and liquid; in the fourth an overdose of *veronal* seemed to be an etiological factor. All cases had the characteristic symptoms and physical signs.

In an extensive clinical and experimental study recently brought out by BRAUN AND SEIDEL (*Mittheilungen a. d. Grenzgeb. d. Med. u. Ch.*, 1907, xvii, p. 533) and the monograph of Kayser (*loc. cit.*) there is nothing especially new. All the etiological factors are by no means established. These two most recent communications advocate the view that the dilatation is primary in the stomach; that duodenal dilatation,

if present, is secondary, and this is brought about by a kinking at the mesentery. The latter is due to pressure of the stomach on the mesenteric vessels and traction of the mesentery of the small intestine. The primary acute dilatation of the stomach is brought about by the action of some toxins on its nervous mechanism.

In the observations of my own cases and now, after a careful reading of the literature extending over a period of three years, I am not at all convinced that we have settled either the etiology, or the pathological anatomy of this affection. To me the more interesting group are those cases in which the dilatation extends to the mesentery and in which there is no evidence of a pathologic condition to explain the obstruction other than the normal anatomical factors at the mesenteric junction of the duodenum and jejunum.

I was also impressed by the fact that in two of my observations relief of tension on the mesentery and jejunum, pressure on the duodenum, and pushing up of the dilated stomach did not relieve the obstruction nor allow the duodenal contents to flow into the jejunum. In my first case the presence of stomach peristalsis three and a half days after the onset of the symptoms is evidence against complete paralysis, and I wish to emphasize again the importance of hypersecretion.

In conclusion I again call especial attention to Case 3, which is undoubtedly an example of chronic gastro-duodenal dilatation or gastro-mesenteric ileus. Further experience may demonstrate that this is the common pathological condition of many cases of gastric neuroses, which have not been relieved by posterior gastroenterostomy, and which may be relieved by duodeno-jejunosomy.

I find that I have failed to mention the very comprehensive review of Lewis A. Conner, of New York (*Amer. Jour. of the Med. Sciences*, March, 1907, vol. cxxxiii, p. 315), who reports an observation of his own and makes a critical analysis of 102 cases recorded in the literature. In this series there was no example of the complication following operations on the stomach itself. Two are recorded among my observations,—Finney's pyloroplasty, and one from the literature. Stomach peristalsis as observed in my first case, according to Conner, has been noted in only three of the 102 observations. Conner's bibliography is complete up to date.

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