walls of the tract become too rigid to collapse. In our patients the fistulous openings were 2 cm. in diameter or greater.

Surgical closure was considered in each of these patients. It would be difficult and hazardous and is seldom advisable.

Although gastrocutaneous fistulae are not likely to produce sepsis, control of casual infection can be important. Drainage is essential for correction of the fistula and prevention of secondary infection. Antibiotic agents should be employed as indicated by culture and sensitivity tests. Special attention must be given to fluid, electrolyte and nutritional support. Further, corticosteroid therapy must be continued.

Gastric suction has some advantages in collapsing the cavity between the stomach and the skin. This effectiveness disappears after a few days and nothing is gained by prolonged gastric suction in most instances. Regular diet as soon as possible seemed beneficial to our patients.

These patients are ideal candidates for stress ulceration. One patient bled severely and the cause of bleeding was never determined. Protection with antacids and vagal antagonists is essential in long-term management.

Some years ago Dr. James A. Kirtley stated that "everything begins healing about the fifth week." These defects apparently close between the fifth and seventh week.

Summary

The clinical courses of gastrocutaneous fistulae originating from an apparently devascularized area of intact gastric wall have been presented. In all the vasa brevia were divided during removal of the spleen. The possible additional ischemic effects of operative trauma or arteriosclerosis and corticosteroid arteriolitis of the left gastric artery have been discussed. Suggestions in the diagnosis and management of these fistulae have been made.

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Discussion

Dr. Charles H. Watt, Jr. (Thomasville): I enjoyed Dr. Byrd's paper very much. I would like to present this case because of its bizarre nature.

(Slide) In reviewing the literature, I have been unable to find a case similar to that of a 29-year-old man who had been told for years that he had a duodenal ulcer and had been medically treated for it. An acute episode of epigastric pain, leukocytosis, and fever had brought him into our hospital. He had lost weight and complained of intermittent diarrhea.

A GI series showed a large fistulous tract (slide) between the second portion of the duode-

num and the distal ascending colon and tip of the appendix. He had acute episodes of right lower quadrant pain for 20 years.

(Slide) Here again you see the rather large fistulous tract. Here is the appendiceal connection with the fistula at the tip of the appendix.

At operation there was a 1 cm. opening in the second portion of the duodenum, emptying into an 8 cm. receptacle. The ascending colon opening was 2½ cm. The fistula was lined with columnar epithelium.

Walker, Rhame, and Smith have stated: "The rupture of a peri-appendiceal abscess into a viscus is not rare, but the persistence of a fistula from the appendix is most unusual." It is rare to find two additional organs involved.

At operation, because of extreme narrowing of the upper ascending colon, we did a right colectomy and closed the duodenal fistula. The patient has done well for the past 5 years.

DR. JAMES D. HARDY (Jackson): Our interest in gastric remnant ischemia and fistula formation was stimulated some years ago by two patients, both of whom had had most of the major gastric arteries ligated at operation because of special circumstances in each instance.

Until that time it had been widely believed—I must say, somewhat loosely—that the blood supply to the stomach was somehow inexhaustible and, in fact, one method introduced a great many years ago for the management of peptic ulceration had been to reduce the blood supply to the stomach.

(Slide) In this slide is exhibited the necrosis of this part of the gastric pouch above the gastrojejunostomy, following ligation of the right and the left gastric arteries and both the right and the left gastroepiploic arteries, as well as the vasa brevia, by the surgeon at this operation.

(Slide) This drawing illustrates the substantial extent of severe ischemia and possible necrosis which may follow division of all major arterial supply to a large residual gastric pouch. As Dr. Byrd mentioned, the inferior branch of the left phrenic may be the only blood supply remaining, other than that which comes along the esophagus.

(Slide) In this slide is shown the blood supply of the stomach. I am duplicating slightly his presentation of the anatomy, since I did not know he would be showing it. However, this slide does illustrate that in the following experiment, which I will describe in a moment, the left gastric, the right gastric, the left gastroepiploic, and the right gastroepiploic were successively ligated. It may be seen further that the gastric remnant remained supplied by only the recurrent branch of the left inferior phrenic artery, in addition to a few branches along the esophagus.

(Slide) We took this problem to the laboratory (Surg. Gynec. Obstet. 118:1312, 1964) and measured rates of radiosodium clearance from the gastric wall in dogs following successive division of each of the four major arteries to the stomach. Just in broad terms, one may note that when only one artery was ligated, the rate of disappearance of the sodium, reflected in the counts per minute, from the gastric wall was still rapid. When two arteries were ligated—two of the four major arteries—there was a definite reduction in the rate of disappearance of radiosodium from the gastric wall. When three arteries were ligated there was a further decline in radiosodium clearance. But when all four major arteries to the stomach were ligated, one may see that there was a very, very low rate of clearance of radiosodium from the gastric wall, indicating, of course, a very low arterial blood flow.

DR. HERSCHEL A. GRAVES (Nashville, Tenn.): I would simply like to call attention to the fact that a significant influence on the formulation of this presentation came with the investigative work of Drs. Kilgore, Turner and Hardy at the University of Mississippi, the very work that Dr. Hardy just referred to, and that of Cate and Dawson at Vanderbilt. In their studies they not only reproduced total ischemic necrosis of the gastric remnant, but they also showed the effects of lesser degrees of insufficient blood supply to the remaining gastric remnant.

In their animal studies they noted latent areas of necrosis, including anastomotic breakdown in gastric remnants that appeared perfectly viable at the time the abdomen was closed.

In our cases, each case had a significant element which called for caution at the begining of the operation; extreme obesity, pathology of the enlarged spleen and the perisplenitis, and the previous surgery. We conscientiously tried to recall unusual trauma to the stomach, but we sincerely did not believe that such occurred, and accordingly, in the light of this investigative work we came to the conclusion that arterial insufficiency in truth did cause these difficulties with gastric healing.

One further comment I would like to make that was impressive to us in the management of these patients: After the initial period of combined suction, both by Levin tube in the stomach and by suction through the drainage site has accomplished emptying of the fistula and control of infection, continued use of this suction and temporizing liquid diets serve no useful purpose. In fact, it seems to promote detrimental effects: more fluid loss, greater drainage, poor nutritional state.

After this initial period of fever and toxicity is over, we feel that the institution of solid alimentation will be of great benefit to the patient and result in quicker healing of the fistula.

DR. RICHARD T. SHACKELFORD (Baltimore): The remarks I am going to make I had originally thought to direct to Dr. Dunphy's paper, but the gist of my remarks is a warning about the use of sump drainage. It has certain dangers, I think, as well as assets, or virtues.

As an illustration, on the 28th of September a

prep school football player tackled a man who was enraged because he had been thrown. As he got up he stamped on his stomach with his shoe. The boy suffered a complete transection of the duodenum.

This patient was seen in another hospital. I did not see him in consultation until last Wednesday. He was operated on the night of injury and an end-to-end anastomosis of the two cut ends of the duodenum was done. Within a week the anastomosis had dehisced, after which they did a gastroenterostomy.

A short while later he developed intestinal obstruction. At operation Dr. Baker's tube was used at the point of ileal obstruction to empty the bowel of its contents. The stab wound in the bowel was closed with two concentric purse-string sutures. Shortly after, that opening opened to form an ileal fistula.

A sump was inserted after the duodenum dehisced. At reoperation the ileal fistula was closed and another sump was inserted in the left quadrant. He continued to lose about 7,000 cc. of fluid per day—and when I saw him that Wednesday morning at 6 o'clock, blood pressure was a palpable 40. You could not hear the blood pressure. The pulse rate was somewhere between 180 and 200, and he was bleeding heavily through both sumps. It seemed essential to reoperate to control the bleeding and defunctionalize these fistulas in some way.

For the first time in my experience, the anesthetist refused to put the patient to sleep because he thought he was too sick, and I called Hopkins to send an anesthetist who was willing to do it.

At operation we found 1,500 cc. in measured blood in the abdominal cavity and all the intestines matted together. Around the sump which drained the duodenal fistula the entire posterior wall of the stomach had necrosed. There was a hole that was big enough to put your whole hand into; I had to do practically a 60% gastrectomy to get something to work with.

At the site of the other sump in the left lower quadrant, which had only been in for 7 weeks—whereas the other had been in for 9 weeks—we counted nine fistulous openings in various loops of intestine. Since there had only been one at the beginning, it was apparent that the sump had eroded its way through these various loops of intestine.

I wish to emphasize that if sumps are used, I believe consideration should be given to the length of time that they remain in contact with the bowel.

In the past 2 years I have seen two other patients, both following gallbladder operations, whom I saw in consultation when they developed complications. Both developed duodenal fistulas at the point where the sump was in contact, or appeared to be in contact with the duodenum.

DR. WILLIAM S. MCCUNE (Washington, D. C.): I think this subject is important and one that has involved all who have done much gastrointestinal surgery. In the only similar instance that we encountered we inadvertently put a clamp across the fundus of the stomach in removing the spleen. This certainly is one of the commonest causes of gastric fistula.

I wish to discuss briefly the development of the breakdown of intestinal anastomoses and its etiology. I don't believe that anyone in this room who has done much of this type of surgery has not had an intestinal anastomosis breakdown, even one that he thought was perfectly all right, in which the intestine was perfectly normal.

A few years ago an elderly lady was admitted to George Washington Hospital with obviously advanced intestinal obstruction. She hated all doctors, including myself, but was convinced that she should have an operation. At operation she had a twisted loop of bowel, gangrenous, which was resected. The arterial supply seemed perfectly adequate. There was no evidence of venous thrombosis, and an end-to-end anastomosis was performed.

About the 7th postoperative day the anastomosis broke down and she developed a large fistula which drained between 900 and 1,000 cc. a day. It soon became obvious that reoperation was required if she was to survive.

At second operation the fistula was resected and again the arterial pulsations were perfectly normal with no evidence of venous thrombosis. After an end-to-end anastomosis was performed it appeared that the distal loop was slightly dusky for about 2 inches, so it was resected and another anastomosis was performed. Postoperatively she developed another fistula and died.

In a study of these sections there were multiple small thrombi found in the veins of the bowel wall itself. There was no evidence of thrombosis of the major veins or arteries or in the sections of the original suture line.

I am beginning to believe that there may be some connection between long-stand obstruction and development of minute occult thrombosis with interference of circulation, and subsequent poor healing.

DR. BENJAMIN F. BYRD, JR. (Closing): I'd like to thank Dr. Hardy for letting us observe his fine experimental work which led to the clinical observations in the three patients we reported. There is nothing particularly new in American medicine about gastro-cutaneous fistulas. We are all familiar with the work of Major Beaumont and Alexis St. Martin over a period of several years, which can hardly be termed a surgical triumph. However, there were times when we were willing to write a 7-year protocol for the study of gastric physiology with the expectation that this might be the solution to our problem. We thought we were going to have a persistent fistula for that long. However, Dr. James Kirtley, of this Association, reassured me. He said that everything begins to heal about the 5th week; and, sure enough, it did.