

**REMARKS ON FATAL HEMORRHAGE FROM  
EROSION OF THE GASTRODUODENAL  
ARTERY BY DUODENAL ULCERS.\***

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IT has been my misfortune to have witnessed two fatal cases of bleeding from duodenal ulcers in spite of the operation of gastro-enterostomy. In both cases the ulcer was situated on the posterior surface of the first portion of the duodenum, and in both the hemorrhage, which proved fatal, came on at a time when it looked as if the patients would recover from the operation.

The first patient died 34 hours after the operation with all the symptoms of concealed hemorrhage, but as no autopsy was allowed we can only surmise, from the position of the ulcer, that the gastroduodenal vessel was eroded. For this reason I have appended brief notes only.

CASE I.—R. A., white, male, aged 28. He had suffered from symptoms of dyspepsia for over three years, characterized by epigastric pain appearing usually about two hours after meals, acid eructations, and the occasional appearance of blood in the stools. During the six months prior to operation he had suffered from several fainting spells, which had been followed by the passage of stools containing much blood. Three weeks before he came under observation an alarming hemorrhage had occurred, from which the patient had not entirely recovered. He was so weak and anæmic that he was confined to bed. Every stool was tarry.

Operation was performed on June 9, 1905. The stomach showed no evidence of ulceration. A thickened, ulcerated area was felt in the posterior wall of the first portion of the duodenum. A posterior gastro-enterostomy was performed. The patient

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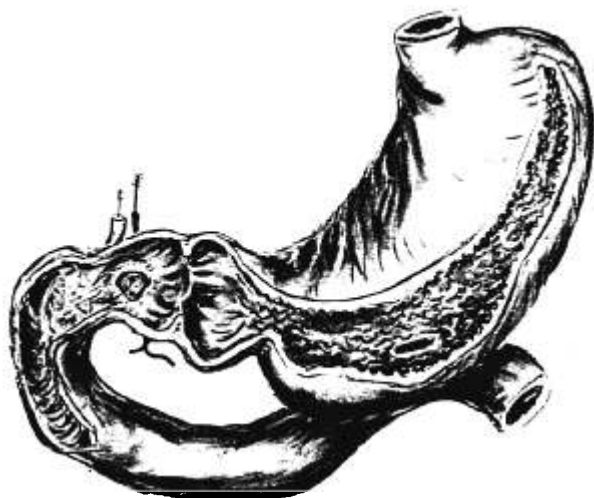
rallied from the operation poorly. Early next day he was a little better, but toward evening he became restless and faint. He grew gradually weaker and died at the end of 34 hours with all the symptoms of internal hemorrhage.

CASE II.—H. N. A., aged 35, white, male. In April, 1899, he began to suffer from pains in his stomach, which came and went without apparent connection with eating. They were relieved in a few minutes by bicarbonate of soda. No loss of weight or diarrhoea was noticed. The pains occurred at unequal intervals, and disappeared entirely at the end of two months.

In the fall of 1903 the same symptoms reappeared and were accompanied by vomiting. At first the vomited matter consisted of partially digested food only, but at the end of a month it was frequently dark or coffee-colored. At the end of three months the symptoms disappeared entirely. No notice was taken of the stools. In June, 1910, the pains returned again, and were relieved by bicarbonate of soda. In October of the same year he passed segments of a tapeworm and continued to pass them until November, when a vermifuge removed the worm. About the middle of November the pain reappeared. It was accompanied with gas, and vomiting soon after meals. There was gradual loss of weight. About Christmas the worms reappeared in the stools, and have been present ever since. From this time the stomach symptoms have continued without cessation up to the present. The pain comes on usually within an hour after meals and is intense for two or three hours, when it subsides somewhat. It never entirely disappears, however, and interferes with sleeping so much that he only rests on an average three or four hours each night. There are always intensely acid eructations and frequently vomiting of stomach contents. There has been marked constipation and a loss of weight of 44 pounds during the last four months.

On March 18, while undergoing a physical examination, he was handled rather roughly. On March 19 a test meal was taken, and on account of the failure to pass a stomach tube an injection of gr. 1/50 of apomorphia was given. About 20 minutes afterward, when the effects of the apomorphia had passed away, while sitting in a chair laughing and talking, he fainted without any warning. The condition appeared to be relieved promptly by the injection of strychnia. In a few hours the bowels

FIG. 1.



*Erosion of gastroduodenal artery by a duodenal ulcer.*

moved and the stool was saturated with blood. The same conditions were noticed on March 20 and 21. On March 22 he came under the care of my colleague, Dr. Graves. He was then suffering from the effects of severe hemorrhage. He was very blanched. A blood examination showed red cells 3,760,000, white cells 7800, hæmoglobin 75 per cent. The urine showed nothing abnormal. An examination of the stools showed blood present and segments of *Tania saginata*. On March 24 a rectal examination was made, but nothing abnormal found. A diagnosis of duodenal ulcer was made and operation suggested, but positively declined.

On March 25, in the afternoon, the patient became faint, was covered with cold sweat, and showed signs of severe internal hemorrhage. I saw him on March 26, and although he had recovered somewhat, his condition was still desperate. During the day he improved somewhat and on March 27 he consented to the operation. At 2.30 P.M. the operation was performed. The stomach was large, flabby, and anæmic. The first part of the duodenum at first appeared healthy, but on palpation a large indurated area was felt occupying its posterior wall and the adjacent part of the pancreas. The coils of jejunum were distended with blood. A gastro-enterostomy was performed, but the duodenum was not folded in.

The patient endured the operation fairly well and was carried to bed in fair condition. After the operation the patient continued to improve steadily. His condition when I saw him late at night on the 28th was excellent. At 7 A.M. on the morning of the 29th he showed signs of a return of the hemorrhage. In half an hour he was almost pulseless, and he died a most tragic death as we were preparing to transfuse him directly from the radial artery of his wife. He lived 42 hours after the operation.

I conducted the post-mortem examination personally within an hour of his death. The stomach, pancreas, and duodenum were removed "en masse." The stomach, duodenum, and the whole length of intestinal canal were full of blood. On slitting up the duodenum an ulcer 17 mm. long and 17 mm. broad was found occupying its posterior wall. It was situated almost midway between the pyloric ring and the bile papilla. The edges were rolled out and indurated. At the bottom of the ulcer a small aperture was seen (Fig. 1). A probe passed into it travelled easily in either direction upward or downward. A search was made for the gastro-

duodenal artery. After tedious dissection through dense inflamed tissue it was found. A stream of water was passed downward along it and it escaped almost at full bore through the opening in the base of the ulcer. A similar stream was forced upward along the right gastro-epiploic artery with the same result.

Bleeding from duodenal ulcers may vary to any extent both as to quantity and periodicity. It is probable that most duodenal ulcers bleed periodically and that the slighter hemorrhages are overlooked, only the more massive ones being recognized. Where careful examinations of the stools are made systematically, occult blood is found in a very large proportion of cases, for it is a matter of daily observation that patients do not recognize coffee-ground vomit and tarry stools as positive signs of hemorrhage. Ocular manifestation of bleeding is usually registered as "hæmatemesis" or "melæna," and in taking the history of a patient we have usually to rely on their conception of hemorrhage. From a clinical stand-point we may divide the bleeding into two varieties: (1) *chronic hemorrhage*, in which the blood is lost slowly, and usually in a moderate quantity; and (2) *acute hemorrhage*, often profuse and so abundant as to endanger the patient's life. Either variety of hemorrhage may show periodicity, but the chronic form is more likely to be constant than the acute.

*Frequency of Hemorrhage.*—It is interesting to note the frequency and variety of bleeding. In Moynihan's first series of cases he noticed that 71 patients (38 per cent.) gave a history of bleeding at one time or another; of these, 17 had hæmatemesis alone, 24 had melæna alone, and 30 had both hæmatemesis and melæna. In his second series of cases (101) hemorrhage occurred in 49 (almost 50 per cent.); of these, 9 suffered from hæmatemesis alone, 10 from melæna only, and in 21 both hæmatemesis and melæna had been observed.

The figures of the older writers were not based on such close clinical observation, but dealt with severe hemorrhage only. Perry and Shaw estimated that 13 per cent. of the bleeding cases end fatally.

*Source of Hemorrhage.*—There is not sufficient evidence either of a clinical or pathological nature to enable us to state accurately the source of hemorrhage even in the cases where bleeding has been a prominent symptom. All of Moynihan's cases recovered, so, of course, no pathological proof is possible. He noticed unusually severe hemorrhage in five cases (Nos. 14, 19, 28, 43, 138). In three of them the position of the ulcer is stated as being on the posterior wall; in the remaining two the infiltration of the duodenum by inflammatory products was so massive that its exact position could not be determined. The statement is probably true that ulcers occupying the anterior wall rarely cause severe hemorrhage, whereas those on the posterior surface are extremely likely to erode important vessels such as the hepatic and gastroduodenal arteries. Still it must not be forgotten that in the duodenum, as in the stomach, superficial insignificant ulcers may bleed profusely. Thus Quénu reported a case of extreme anæmia due to hæmatemesis, where at operation a small ulcer was found occupying the anterior wall of the first part of the duodenum. Bleeding ceased after obliteration of the pylorus followed by gastro-enterostomy. Also Moynihan reports a case (No. 114) where operation was performed on a patient who had suffered from a severe hemorrhage followed by profuse mælena, in which he excised a small round ulcer on the anterior wall of the duodenum, and sutured a precisely similar ulcer on the posterior wall. In the cases collected by Collin, where the source of the fatal hemorrhage was determined by a post-mortem examination, the blood-vessels eroded were as follows:

Arteria pancreaticoduodenalis .....	12 times.
Arteria gastro-epiploica dextra .....	2 times.
Arteria pancreaticoduodenalis superior..	3 times.
Arteria abdominalis .....	2 times.
Vena portæ .....	2 times.
Arteria hepatica .....	1 time.
Arteria mesenterica superior .....	1 time.

It would appear, then, that the gastroduodenal artery is more likely to be opened than any other vessel, and this we should expect from the anatomical relationship of this vessel to the first part of the duodenum. Deep eroding ulcers occupying the posterior wall are sure to extend to this vessel sooner or later, and if thrombosis fails to precede the erosion, severe hemorrhage is inevitable.

*Treatment.*—It is proven beyond doubt that in the majority of cases of duodenal ulcer associated with hemorrhage, gastro-enterostomy will check the bleeding. This is shown conclusively in Moynihan's series of cases of severe hemorrhage, 18 in number, where the operation was unattended by any mortality. Of course, there is no definite proof that such a large vessel as the gastroduodenal artery was bleeding in any of his cases, but the severe symptoms in three of them where the ulcer occupied the posterior wall (Nos. 14, 28, 138) point strongly to such a condition. I have found one clinical observation noticed by Mayo Robson that proves conclusively that the rest imposed on the duodenum following an anastomosis favors closure of the artery by a clot. A patient upon whom a gastro-enterostomy had been performed for bleeding supposed to be from a gastric ulcer died at the end of ten days from perforation at the site of anastomosis. The post-mortem examination revealed no evidence of a gastric ulcer, but a large deep duodenal ulcer, at the bottom of which lay the gastroduodenal artery with an ulcerated hole in its side completely closed by a firm thrombus.

The essential principle involved in this method of treatment lies in the enforced rest of the duodenum, which favors clotting at the bleeding points. It is common knowledge that better results follow the operation of anastomosis in cases where the pyloric orifice is obstructed than in those where it is free, and that equally good results can be obtained in the latter cases if the pyloric orifice is obstructed by infolding or is obliterated. Therefore it would be rational in these cases to obliterate, temporarily at least, the pyloric orifice by massive infolding, so as to prevent any particle of food or fluids

passing along the duodenum. In addition to this, infolding of the ulcer is practised if it is situated on the anterior wall, or in case of severe hemorrhage the ulcer might be excised or surrounded with a deep purse-string suture before infolding it. In ulcers of the posterior wall, infolding of the ulcer is not practicable, but it would be feasible to infold the anterior wall so completely that it would form a plug capable of closing the ulcer and blocking up the bleeding vessel mechanically.

I am afraid that in my two cases I allowed the patients to slip through my fingers by trusting too much in the value of gastro-enterostomy alone. It is possible that massive infolding might have prevented a repetition of the bleeding.

Of course direct attack of the bleeding point with suture of the vessel would be the most scientific procedure, but it is more than doubtful if it could be done safely. The situation of the ulcer, its depth, immobility, and the dangers of hemorrhage, all would render such an operation unusually hazardous. Personally I was glad enough to get my patients off the table alive after the anastomosis had been completed.

In a letter recently received from Sir Berkeley Moynihan, he outlined the plan of treatment that has been so uniformly successful in his hands:

"I have found that the gastroduodenal artery is rather apt to be opened if the ulcer occupies any portion of the posterior surface, and especially when the ulcer occupies the upper border and posterior surface. I have two ways of meeting the difficulty: If the artery is seen running into the ulcer, I pass a needle underneath it, and ligature it; and, secondly, close down the anterior wall of the duodenum by infolding sutures on to the ulcer, and make sure of closing the pylorus or the stomach on the near side of the ulcer. In either case, of course, gastro-enterostomy is done. I feel quite sure that in all cases of duodenal ulcer local treatment is necessary in addition to gastro-enterostomy, for many cases of hemorrhage, after an apparently successful gastro-enterostomy, have been recorded."

My interpretation of this letter is that no attempt is made



to open the duodenum but that all the work is accomplished from the outside as outlined. Referring to the possibility of passing a ligature under the gastroduodenal artery before it reaches the ulcer, a careful dissection of our specimen showed that it would hardly have been possible. Both the hepatic artery and its gastroduodenal branch were embedded in inflamed tissue of such cartilaginous density that the vessel was dissected post mortem with the greatest difficulty.

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