TRAUMATIC RUPTURE OF THE STOMACH

BY

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In the *Glasgow Medical Journal* of 1894, J. Grant Andrew, in a paper read before the Glasgow Pathological and Clinical Society in December, 1893, reports a fatal case of traumatic rupture of the stomach due to a crush, for which no operation was performed. In his review of the literature, he states that up to 1858 three cases of complete rupture of the stomach had been reported, and, including the one he related, there were eight further ones up to 1893. All these cases were fatal, some immediately following the injury, two being five days after the injury. He makes a note that the rupture was generally near the pylorus, and that vomiting was frequent in most of the cases.

In *Acute Abdominal Diseases*, published by Adams in 1923, it is stated that rupture of a normal stomach may be caused by violent contraction of the abdominal muscles, by blows, or by crushes. He states that the rupture is usually longitudinal, and near the pyloric portion of the lesser curvature of the stomach, and the only pathognomonic signs are fresh blood in the vomit and obliteration of the liver dullness—in addition, of course, to the usual signs of rupture of any abdominal viscus.

Sherren states that between the years 1899 and 1919 eleven cases of ruptured stomachs were admitted to the London Hospital, but in only three was it the sole viscus ruptured, and all these cases treated in the London Hospital died. He says that ruptures from external violence are usually situated in the region of the greater curvature. He also quotes Deaver and Ashurst as stating that only four operations for traumatic rupture of the stomach have been recorded, and all resulted in death.

Walton, in his *Surgical Dyspepsias*, states that traumatic rupture of the stomach is rare, and generally other viscera are affected. He quotes Sherren as saying that of 270 cases of abdominal contusion admitted to the London Hospital in ten years the stomach was ruptured in five, and in only one of these cases was no other viscus affected. He also quotes Battle, who published 165 cases of intestinal injury, but no example of injury to the stomach. Walton states that practically all reported cases of rupture have occurred when the stomach is full, and that rupture takes place at the weakest part of the wall—namely, the greater curvature. Owing to the severity of the injury and the frequency of the accompanying lesions these injuries are nearly always fatal.

I thought it would be interesting for me to publish (with the kind permission of Professor Burgess and Mr. Wrigley, under whose care these patients were while at the Manchester Royal Infirmary) two cases which I operated on, and in which the rupture of the stomach was the only abdominal injury present, and both of which made uninterrupted recoveries, each patient being discharged from the Infirmary in a fortnight.

CASE I.

Mrs. J. G. was admitted to the hospital on July 14th, 1928, following the injuries she received by her car colliding with another one, the impact doubling her up and throwing her under the dashboard. When I examined her she was pale, she had vomited once following the accident, the abdomen was rigid, she was suffering severe abdominal pain, and there was diffuse tenderness and guarding all over the abdomen, more especially at the right side. There was no shifting dullness to be detected in the flanks. Her pulse rate was 92, and temperature 99° F. There were no other signs of injury anywhere to be made out. On the diagnosis of the rupture of an intestinal viscus, I made a right rectus slide incision, and on opening the peritoneum I found blood and extravasated contents in the abdominal cavity. These were swabbed out, and rupture about the size of a threepenny piece was found at the pyloric end of the stomach near the lesser curvature. The rent was sutured with interrupted Lembert sutures of catgut, and the abdomen was then closed, with drainage to the abdominal wall. She was discharged home on July 28th.

CASE II.

Mr. A. F. was admitted to the Manchester Royal Infirmary on December 18th. He could not give any account of the accident, as he was suffering from some slight degree of concussion of the

brain. I learnt from witnesses that he had been knocked off a pedal cycle by a motor bus. A neighbouring doctor, who saw him immediately after the accident, gave him 1/4 grain of morphine. When I saw him he was complaining of abdominal pain, and also the abdomen was generally rigid and tender. His pulse was 70, strong and steady, and the temperature 97° F. At operation I found much free blood, gas, and stomach contents in the abdominal cavity. These latter were swabbed out. The stomach was bruised and oedematous, and there was a rent about an inch long high up on the lesser curvature of the stomach, which was sutured, as in the previous case. The abdominal cavity was then filled with hot saline solution and the abdomen closed. The wound healed by first intention, and he was discharged to the convalescent home on December 31st.

Both these cases were caused by injuries when the stomach was full, and both were operated on within eight hours of the accident. In one case the upper part of the lesser curvature of the stomach was affected, and in the other the pyloric portion of the stomach. In neither case were there any other abdominal injuries, either internal or external. The immediate after-treatment was similar to that employed in the case of rupture of a gastric or duodenal ulcer.

I have examined both patients about four months after the injuries, and neither complains of any disability.

TOXIC JAUNDICE WITH ASCITES.

BY

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ALTHOUGH the occurrence of ascites in cases of toxic jaundice is a relatively uncommon happening, it is probable that its frequency is considerably greater than the very sparse literature would suggest. I have given much attention to certain aspects of liver disease, and naturally a fair concentration of liver cases has occurred in my practice; it was not until recently, however, that an example of the syndrome under consideration came under my care. A search of the literature disclosed only four references to it, and I felt that a brief review of the subject would not be devoid of interest.

Fiessinger and Brodin¹ and Fiessinger and Walter² give a very clear account of their conception of what they have termed the "syndrome ictéro-ascitique," and their description, both of the clinical features and of the pathology, cannot be bettered. During the course of a multilobular cirrhosis of the liver the clinical picture occasionally becomes dramatically altered by the sudden onset of ascites and jaundice. This jaundice, they say, is not the usual subicteric tint of the cirrhotic, but a frank icterus with deep coloration of the integuments, bile in the urine, and clay-coloured stools. Ascites develops rapidly, but the amount of the effusion, and the necessity for frequent tapping, vary widely in different cases. At the same time the patient's general condition degenerates rapidly, emaciation and marked weakness ensue, and there are usually digestive troubles; fever may appear, and its explanation is a matter of some difficulty.

The progress of the malady is variable; usually the condition is rapidly fatal, and the presence of the syndrome may be taken to mean the presence of an acute or sub-acute hepatitis—cholaemia may carry off the patient with its familiar train of symptoms. Some patients recover, however, and these are the ones in whom some definite cause can be found for the onset of toxic jaundice—the transitory toxæmia of surgical anaesthesia, the action of novarsenobillon, etc. Where no cause can be found the outlook is grave.

With regard to the pathology of the condition these authors describe a widespread fatty degeneration of the parenchymal cells of the liver, together with the usual findings of atrophic cirrhosis. Their explanation of the syndrome is ingenious; it is obvious that acute hepatitis in a normal liver would produce jaundice, and in such a liver the swelling of individual cells would result in an increase in the volume of the organ. If such a hepatitis arose in a cirrhotic liver jaundice would naturally ensue, but the fibrosis of the organ would prevent, in certain