given in Chart 2. The protein content of the ascitic fluid varied between 2 and 2.5 g. %.

By this time the patient was markedly emaciated and the abdomen was grossly distended by ascitic fluid and a large spleen, which reached right down into the pelvis. His liver, however, was only slightly enlarged.

On July 2 an estimation of the plasma protein gave a reading of 5.1 g. %, and a hippuric-acid test showed that the amount of hippuric acid passed in the four-hour period was only 2.7922 g., with the maximum excretion in the second hour. It was therefore decided to give 1 g. of choline chloride and 1 g. of cystine twice daily. This was done on the assumption that choline and cystine can help to prevent hepatic cirrhosis due to a very low protein intake.

The hippuric-acid tests were repeated, with the following results :

Date	Amount of Hippuric Acid Passed in 4-hourly Period	Maximum Excretion
12/7/46	2.7504 g.	3rd hour
19/7/46	3.1794 g.	3rd "
30/7/46	3.0220 g.	2nd "
6/8/46	3.0468 g.	2nd "
20/8/46	3.3484 g.	2nd "
29/8/46	3.5882 g.	2nd "

It was therefore concluded that there had been a great improvement in the liver function during this period.

Attempts were also made to raise the low plasma protein level by repeated plasma transfusions, high protein diet, and the addition of "casydrol" by mouth. Plasma protein estimations were as follows : July 2, 1946, 5.1 g. % ; July 12, 5.6 g. % ; July 15, 5.4 g. % ; July 19, 5.5 g. % ; July 29, 6.2 g. % ; Aug. 6, 6.2 g. % ; Aug. 20, 6.8 g. % ; Sept. 2, 6.85 g. % ; Oct. 1, 1947, 7.8 g. % ; Jan. 1, 1948, 8.8 g. %—i.e., 5.2 g. of albumin per 100 ml. of serum and 3.6 g. of globulin per 100 ml. of serum (albumin-globulin ratio = 1:1.5) ; March 9, 1948, 6.25 g. %—i.e., 4.5 g. of albumin per 100 ml. of serum and 1.75 g. of globulin per 100 ml. of serum ; albumin-globulin ratio = 2.6 : 1. It was, however, noticed that although the plasma protein level was 6.8 g. by Aug. 20, 1946, the ascites persisted.

The patient felt very much better, and it was then decided to try to keep the white blood cells within reasonable limits by the administration of urethane by mouth. He was given 1 g. daily for four weeks and the white blood count was recorded daily. After that he was placed on a maintenance dose of 1 g. thrice weekly and weekly blood counts were recorded.

The remarkable fact was that the ascites after tapping took much longer to recur, and when he was tapped on Nov. 12, 1946, 24 pints (13.6 litres) of ascitic fluid were removed. Since that date he has been receiving two "fersolate" tablets three times daily, 1 g. of choline chloride daily, 1 g. of cystine daily, and 1 g. of urethane thrice weekly, with occasional rest periods as determined by blood counts.

He was seen again on March 31, 1948, when he was admitted to hospital for another paracentesis—i.e., after an interval of 16 months. Fourteen pints (7.95 litres) were removed on this occasion, and he remained free from signs and symptoms of ascites until Sept. 4, when he was last seen. His liver was just palpable at the costal margin, whilst his spleen extended only a hand's breadth below the left costal margin. His weight has remained fairly stationary in spite of his excellent appetite, and he feels very fit though his haemoglobin is low.

Since May, 1948, I have administered, in addition to the other medicines, 4 ml. of "anahaemin" intramuscularly at weekly intervals in an attempt to raise the haemoglobin and red blood cells, but so far without success. It has also been necessary to administer the urethane in doses of 1 g. daily to keep the white blood count within reasonable limits.

Discussion

This case is most interesting because when the patient was first seen in April, 1946, it was thought that he would live only a few weeks. The remarkable change which took place in his general condition after the administration of the

cystine and choline chloride was astonishing. His plasma protein rose fairly rapidly, the ascites became less pronounced and required less frequent tapping, his liver became normal in size, and he rapidly put on weight. After the addition of urethane his white blood count was kept within reasonable limits and his spleen rapidly diminished in size.

It is impossible to draw any conclusions from only one case, but I consider that there is a very fruitful field for the treatment of such cases with cystine and choline chloride in combination with urethane.

As pointed out by Cheney (1934), the presence of ascites in cases of chronic myeloid leukaemia seems to be rare, and very few cases have been recorded in the literature. He gives a detailed description of two such cases, and mentions the close resemblance of this condition to Banti's disease. The importance of making a correct diagnosis is stressed, as also is the institution of the appropriate treatment. He considers that the only probable causes of the ascites in these cases are portal thrombosis and low colloidal osmotic pressure of the blood plasma.

I would like to express my deep appreciation to Dr. F. Young for her invaluable help in the pathological investigations in connexion with this case.

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RUPTURE OF THE LIVER ASSOCIATED WITH PARTURITION

BY

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A case is described in which an unusual complication developed during or after labour, giving rise to an acute abdominal condition. A rupture of the liver associated with haemoperitoneum was found at operation.

Case History

The patient, a 6-para aged 32, was admitted on Sept. 11, 1948, 17 hours after parturition, with severe upper abdominal pain. The onset was sudden, occurring one hour after expulsion of the placenta and after the midwife had left the house. The patient became collapsed and vomited several times. She described the pain as sharp, radiating to the back and right shoulder, and increasing in severity on movement and respiration. All six previous pregnancies and labours were normal. During the last month of the present pregnancy she had suffered from a persistent epigastric pain.

On examination the patient was pallid and weak and was prevented by the pain from moving in bed or sitting up. The temperature was 97° F. (36.1° C.), pulse 112, and blood pressure 110/85. There was generalized abdominal tenderness and guarding, and shifting dullness was detected. The liver was palpable and tender, projecting 1½ in. (3.75 cm.) below the right costal margin. Peristaltic sounds were active. The pelvic organs were normal, although considerable peritoneal tenderness was elicited by palpation of the lateral fornices. The haemoglobin was 51%. A catheter specimen of urine contained 100 mg. of protein per 100 ml.

Since the precise diagnosis was undetermined, it was decided to transfuse the patient with 4 pints (2.27 litres) of blood and keep her under close observation. Heroin in small doses was given periodically.

During the subsequent eight hours the pulse rate rose slowly to 120 and the blood pressure to 150/100. No further vomiting occurred, but as peristaltic sounds had almost ceased and the abdomen had become distended it was decided to perform a laparotomy. An intraperitoneal haemorrhage, with a large effusion of blood below the right diaphragm, was suspected.

Operation.—A right paramedian incision extending well above and below the umbilicus was made. The peritoneal cavity was full of fresh blood. The spleen and uterus were normal and the gut was distended. A large clot was found obscuring the surface of the liver; when this was removed a subcapsular haematoma was observed to extend over the superior surface of the right lobe. The left lobe was normal. The peritoneal coat had been stripped up, and there was a rent in it through which blood was oozing. The clots and free blood from the peritoneal cavity were removed, and "oxycel" gauze controlled the haemorrhage from the raw area of the liver. A biopsy of the liver was taken at the margin of the haemorrhagic area. The liver area was drained and the abdominal wall closed in three layers. A Ryle's tube which had been introduced before operation was kept *in situ*.

Progress.—The condition of the patient improved perceptibly; within 24 hours the pulse rate dropped to 100 and abdominal distension disappeared with the recurrence of peristalsis and the passing of flatus. Gastric aspiration was continued for 36 hours, after which the patient was able to take a light diet. Penicillin, 100,000 units at first, decreasing to 40,000 units, was given three-hourly for 15 days. The patient remained pyrexial for 25 days.

On the 24th day there was clinical evidence of right basal pulmonary collapse with pleural effusion. On the 34th day she developed a sharp attack of pleural pain associated with a swinging temperature of 102–98° F. (38.9–36.7° C.). A further course of parenteral penicillin was given, 60,000 units three-hourly and, later, 200,000 units twice daily, with penicillin inhalations for 18 days. Temperature and pulse rate became normal on the 43rd day. A pleural aspiration was carried out on the 39th day: the fluid so obtained was sterile.

On Nov. 11 (64th day) the patient was discharged from hospital. The blood pressure was 130/90. There was no albuminuria. Evidence of pleural effusion was still present, but her general condition appeared satisfactory.

Investigations.—The Wassermann reaction and Kahn test were negative; haemoglobin was 51%; and white blood cells numbered 22,000. A catheter specimen of urine contained 100 mg. of protein per 100 ml. The blood pressure was 114/85 to 155/100. The blood urea amounted to 124 mg. per 100 ml., the plasma bilirubin to 2 mg. per 100 ml.; and total proteins to 5.3 g. per 100 ml. (albumin, 3.1 g.; globulin, 2.01 g.; fibrinogen, 0.69 g.). A high vaginal swab produced no pathogenic organisms. An x-ray film of the chest showed the right diaphragm raised, with collapse of the base of the right lung and effusion, and, several days later, collapse of the middle lobe with effusion. Liver biopsy showed zonal necrosis with some cellular infiltration—an appearance compatible with that found in toxæmia.

On discharge the haemoglobin was 84%, and the white blood cells numbered 8,000. A catheter specimen of urine contained no protein. The blood pressure was 130/90. An x-ray film of the chest showed some improvement in the extent of effusion and pulmonary collapse. The E.S.R. was 33 mm. per hour (Westergren).

Discussion

Rupture of the liver has been described either as traumatic or as "spontaneous" rupture. The first is a well-recognized condition due to direct injury, and discussion of it is irrelevant in this article. The second—the "spontaneous" rupture—is the type to which the present case belongs. In this type the trauma is either slight or has passed unrecognized by the patient. This use of the term "spontaneous" is unfortunate, and may be misleading when applied to liver rupture, as it certainly is when used to describe a splenic rupture attributed to minimal or unrecorded traumata.

Under the heading "ruptures spontanées" Devic and Bériel (1906) have discussed liver rupture and made particular reference to the pathological condition of the organ at the time of its rupture and to the possible association with syphilis. Of the 21 cases reviewed by them only one—Abercrombie's case, to which further reference will be made—was associated with pregnancy.

Devic and Bériel suggested that when definite trauma could be excluded the term "apoplexie hépatique" might be used. They expressed a clear opinion that in traumatic rupture it is the rupture which causes the haemorrhage, but that in spontaneous rupture the haemorrhage is the cause. The sequence of events leading to spontaneous rupture is, they suggest, as follows. Rupture of a blood vessel leads to intrahepatic haemorrhage; this in turn causes local laceration of liver tissue and the formation of a subcapsular haematoma; finally, the capsule of the liver may rupture and blood escape into the peritoneal cavity.

Such intrahepatic haemorrhage, they suggest, may be caused by a variety of conditions—for example, vascular disease, necrosis of the liver associated with septicaemia, or hepatic malignancy. In some cases minimal trauma may cause haemorrhage in diseased liver comparable with the haemorrhage that is known to occur in the diseased spleen.

Abercrombie's (1843–4) case, to which reference has already been made, was associated with pregnancy. He gives a vivid account of a woman aged 35 who suffered from epigastric pain, abdominal distension, and belching at the eighth month of pregnancy. He says of the patient: "Having had frequent attacks of gastrodynia on former occasions, she, of her own accord, had immediate recourse to pressure for relief; and placing a silk handkerchief round her body she desired one of her servants to pull it as tight as she possibly could. The wish was complied with, but to an extent that made me fear some injury under existing circumstances. I therefore begged it might be removed. I then prescribed for her a draught composed of Calced Magnesia, Liquor Opii Sedativus, Spt. Ether, Sulph., and Aq. Cinnamomi, to be taken immediately, and ordered hot fomentations to the epigastrium. By these means the pain gradually abated." A few hours later labour began and was soon followed by the successful breech delivery of the foetus. An hour after the expulsion of the placenta the patient collapsed. Haemorrhage was suspected, but there was no evidence of its coming from the birth canal. Two days later the patient died, and a post-mortem examination revealed a large unruptured subcapsular haematoma on the superior and anterior surfaces of the liver.

Abercrombie was of the opinion that the damage to the liver might have been caused by the tight bandaging, but that haemorrhage did not occur until the pressure of the gravid uterus on the upper abdomen had been removed. He also put forward an alternative suggestion that haemorrhage took place in a liver that was already so diseased that it was readily injured by the muscular compressions of labour.

Alessandri (quoted by Rademaker, 1943), writing on this subject, agrees that rupture of the liver may occur as the result of violent muscular activity during parturition or during epileptic seizures, but he gives no case report or reference to support his views.

Rademaker (1943) describes the case of a woman aged 32 admitted to hospital in the eighth month of pregnancy with a blood pressure of 260/160 and albuminuria. After a severe bout of vomiting she became collapsed and the diagnosis of a ruptured uterus was made. At operation a rupture of the liver (the exact site is not specified), with blood in the peritoneal cavity, was found. The haemor-

rhage was controlled by a pack, and after a stormy convalescence the patient recovered. In this case hypertension was thought to be the cause.

Rademaker submits the interesting suggestion that sudden death during eclampsia may sometimes be due to a ruptured liver. We doubt, however, whether this speculation can be sustained, for, although scattered areas of necrosis and small subcapsular haemorrhages are not infrequently found in eclampsia, severe haemorrhages of the liver are not a feature of this disease.

Links (1946) describes a case of rupture of the liver during the fourth month of pregnancy. The patient developed acute abdominal pain with signs of blood in the peritoneal cavity. At operation the blood was found to be coming from the foramen of Winslow. On exploring the lesser sac a laceration of the caudate lobe of the liver was seen; the liver tissue itself appeared to be normal. Haemostasis was effected by covering the site of bleeding with muscle tissue. The patient made a good recovery. The cause of the liver damage was obscure, but Links suggests that it may have been due to a transient hypertension.

Conclusion

We have been able to trace three cases of ruptured liver associated with pregnancy, two of which survived as the result of operative intervention. To these three cases we have added a fourth, in which also the patient recovered after surgical treatment.

We believe that in our case the injury was the result of trauma produced by violent contraction of the diaphragm and abdominal muscles during labour. But it is interesting to note that there was also an element of toxæmia, evidenced by the raised blood pressure (found after the initial shock had been overcome) and by the presence of albuminuria. It is probable that this toxæmia produced changes in the liver, making it particularly liable to injury. (The complaint of epigastric pain for one month before parturition and the report on the liver biopsy obtained at the time of operation strengthened this suggestion.) There was, in our case, no evidence of syphilis or other constitutional disease.

Regarding operative treatment, we believe that the application of "oxycel" gauze, fibrin foam, or muscle tissue is preferable to suture. It is also obvious that blood transfusion is an indispensable part of the treatment. Our patient's satisfactory recovery bears out the statement of Rademaker that in cases of liver rupture operation affords a good chance, and probably the only chance, of survival.

Summary

A case is reported of rupture of the liver, with haemoperitoneum, occurring in association with parturition. Only one comparable case has been found in the literature.

The immediate cause of the rupture was probably the muscular compression that occurs with the expulsive efforts of labour. A predisposing factor may have been the presence of liver damage brought about by pregnancy toxæmia.

The use of the term "spontaneous" rupture is deplored. Liver ruptures may be divided into two main groups: traumatic rupture and hepatic apoplexy—the first usually affecting a healthy liver and the second occurring because of haemorrhage in a pathological liver. A subgroup may be added in which minimal trauma causes rupture of a pathological organ. The case reported may be regarded as an example of this subgroup. This classification of liver rupture might usefully be applied to ruptures of the spleen.

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Medical Memorandum

Treatment of a Chronic Typhoid Carrier with Chloromycetin

Good clinical reports upon the use of chloromycetin in the treatment of typhoid fever have naturally aroused interest in the possibility of dealing with the chronic typhoid carrier by similar means. Unfortunately there is no literature available on the efficacy of this form of treatment; nevertheless as a suitable case had recently come under our care it was decided to try the effect of the substance in doses which had been successful in dealing with the acute disease.

CASE REPORT

A child aged 9 years, son of a flight sergeant in the Royal Air Force, contracted typhoid fever in Egypt in June, 1948, from which he made a good clinical recovery, though he was left a chronic faecal carrier. He came under our care in the spring of this year. He was a well-nourished healthy-looking boy. His stools consistently yielded a heavy growth of *Salmonella typhi*, though over 40 specimens of urine were sterile. *In vitro* sensitivity tests with the isolated organism showed it to be very susceptible to traces of chloromycetin: dilutions in excess of 2.5 µg. per ml. inhibited bacterial growth. It was decided to follow a therapeutic course which had been successful in treatment of the acute disease in the adult, using 19.1 g. of chloromycetin in 8.1 days. As the child weighed 4 st. 7 lb. (28.5 kg.) it seemed appropriate to give half the adult dose, which was administered orally in 0.25-g. capsules four-hourly day and night for seven days—a total of 10.5 g. Stools were cultured on alternate days while undergoing treatment, and we were disappointed to find the organism present in all of them: failure was evident before treatment was completed. In view of this finding, a second course, using three times the dose, was instituted—viz., 0.75 g. for seven days (total, 32.25 g.). Stools became negative almost immediately after starting treatment and remained negative until two days after withdrawing treatment, after which he continued to excrete the organism daily in his stools.

While undergoing the course of treatment his blood serum inhibited growth of the isolated organism in dilutions rising from 1 in 2 on the first day to 1 in 32 by the third day. This inhibition of growth disappeared 16 hours after the last dose of chloromycetin was given.

COMMENT

Chloromycetin in doses of 30 mg. per kg. of body weight for seven days has failed to eliminate the organism in a chronic faecal typhoid carrier. This dose is about three times that which has been successful in treatment of the acute disease. The cause of the failure is not clear, especially in view of the marked sensitivity of the organism to chloromycetin *in vitro*. It is possible that much heavier doses for longer periods are advisable. Further clinical trials are necessary before any conclusions can be drawn regarding the efficacy of the substance in treating the chronic faecal carrier.

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In a referendum the Swiss electorate has rejected by a proportionate majority of three to one a controversial law to make x-ray examinations and tuberculosis treatment compulsory. The draft law (according to a B.U.P. report) provided for the periodic examination of the population for tuberculous infection, for the compulsory insurance of the poorer classes against the economic effects of tuberculosis, and for removal to an isolation hospital of sufferers who might endanger others. The law would have been costly: besides its insurance provisions, of which part of the cost would have been borne by the Government and employers, the law required the cantons to contribute to the support of poor families when the wage-earner was suffering from tuberculosis and to pay the costs of x rays when the family concerned had too low an income to afford them. There would also have been the difficulty that many Swiss peasants have never been, and do not want to be, treated by a doctor. A number of Swiss doctors also criticized the draft law, pointing out that people can hardly be compelled to undergo examination and treatment when there is no acknowledged method of either.