CASE IV.—Male, aged 6. Family history of rheumatism. Thin, neurotic. Catches cold easily, subject to skin eruptions. Complaints of suprapubic pain; dysuria; no calculus present. Urine acid, offensive odour; uric acid and urates.

CASE V.—A thin, neurotic boy, aged 4. Family history of rheumatism; has had tonsillitis. Suffers from constipation; pain on defecation; at times incontinence of faeces; micturition slow and delayed; occasional incontinence. Urine acid; uric acid and urates.

CASE VI.—A thin boy, aged 9. Family history of phthisis. Complaints of diarrhoea; abdomen distended; fulness and tenderness in right iliac fossa. Urine very acid; uric acid and urates; trace of albumen.

CASE VII.—A well nourished boy, aged 8. Family history of phthisis. Suffers from recurrent attacks of abdominal pain; dysuria; pain localised in right iliac fossa, with fulness and tenderness on pressure there. Urine acid; oxalates.

CASE VIII.—See previous page.

CASE IX.—See previous page.

CASE X.—See previous page.

HEPATIC CIRRHOSIS OCCURRING IN TWO CHILDREN OF THE SAME FAMILY.¹

By F. W. JOLLYE, M.R.C.S., L.R.C.P.LOND., D.P.H.ENG.,
Alresford.

THERE is nothing in the family history of these children bearing on the disease. All were free from any signs of rickets, syphilis, or scrofula. The father and mother are temperate, and Case I never touched any alcoholic drink for eight years previous to his death, and Case II was an abstainer for four years. There was nothing peculiar regarding the diet of these children with the exception of their extreme liking for vinegar, so that they would drink it out of the bottle when sent to buy some, and would drink it when at home if their mother was out of the way.

CASE I.—Male, 11 years. Had had measles when 2 years old. In the beginning of 1887 he complained frequently of the "belly-ache" and coldness, and often had diarrhoea. In March his legs and abdomen began to swell, and he was tapped by Mr. Shorland seven times between April and the following September, when he died. During his illness he had several attacks of hæmatemesis and epistaxis, and during the last fourteen days he was frequently delirious, and had occasional twitching of his hands and attacks of abdominal pain. There was never any jaundice, and no albumen was found in the urine; his temperature was not recorded. No *post-mortem* was obtained, but I think, if taken in conjunction with the next case, there can be no doubt as to the cause of the symptoms.

CASE II.—Sister of the above, aged 10 years, had measles in 1888 and influenza in 1890. The early symptoms were the same as Case I, and began in October, 1890. During the next month she had attacks of abdominal pain, was jaundiced for three days, and the legs and abdomen commenced to swell. She was admitted into the Warminster Cottage Hospital on November 22nd, 1890, with well-marked ascites, and oedema of the legs and sacral region. The epigastric region was especially prominent and rather tender; the liver dulness extended from the third rib downwards to the costal arch, but its edge could not be felt, and there was no enlargement of the spleen. The heart was displaced upwards and outwards, but there were no signs of valvular incompetence. The skin was dry and scaly, but no nevus marks were found; the motions were very offensive, and a little paler than normal; the urine, of which she passed 16 ounces in the first twelve hours, was high coloured, with a little albumen, but no bile or sugar, and the sediment contained some renal epithelium and blood corpuscles; temperature 98.6° morning and evening, pulse 98. There had been no hæmorrhages, and the fundus of each eye was normal. She was kept in bed and put on a milk diet, and given a pill of rhubarb and grey powder at night, followed by a scammony and jalapine draught in the morning, and during the day she took a mixture of digitalis and acetate of potash for the first fourteen days, and then quinine and iron, and by December 15th the ascites and oedema had quite gone, and she seemed practically well by January 17th, 1891, when she was discharged. She was then passing 40 to 50 ounces of urine a day with a specific gravity of 1020, containing a little albumen and a few blood corpuscles. The liver dulness measured 2 inches vertically in the nipple line. During her stay in the hospital her evening temperature was generally between 99° and 100°, and on December 22nd she had great muscular tenderness in the arms and legs, and her temperature was 100.6 and pulse 104 for two evenings. She had only been home a few days when the dropsy began to return, and she had attacks of abdominal pain and sickness, so she was readmitted on January 26th, 1891, when the dropsy was found to be more marked than when first admitted, and she was passing 8 ounces of urine in the day, containing a trace of albumen and 8.88 grammes of urea. For the next two days she was delirious at times, laughing and crying by turns; the temperature varied from 98.4 A.M. to 100.8 P.M., and she passed her motions and urine unconsciously. On January 29th she rather suddenly became comatose, with dilated pupils and rigidity of limbs, and died in a few hours without any convulsions.

On *post-mortem* examination three hours after death the abdomen contained 10 pints of fluid; there was no peritonitis or ulceration of the intestines, but the mesenteric glands were a little enlarged but not caseous. The liver was adherent to the diaphragm, yellowish-red in colour, covered with granules varying from the size of a pin's head to half a pea, very tough on section, not bile-stained, and weighed 20 ounces; the spleen weighed 2½ ounces, and was normal in appearance and consistence; the pancreas was very hard and tough; the thoracic organs were normal; the

brain weighed 32 ounces, and appeared normal to the naked eye; but, unfortunately, not having read Dr. Ormerod's² paper at that time, no microscopical examination was made. Sections of the liver, kidneys, and pancreas were made after hardening, and both the latter were found to be normal, but the liver showed well-marked multilobular cirrhosis, the fibrous tissue showed a great excess of small bile ducts, and staining by osmic acid demonstrated excess of fat in the cells of the periphery of the lobule. Dr. Duffin, Professor of Pathology in King's College, kindly examined my sections, and confirmed what I have already stated, and added that he was struck with the size of the remaining lobules and the good condition of their cells, so that there must have been plenty of good bile-secreting power at the time of death.

The chief points in the history of these cases are that measles was the only illness common to both, and that both very largely indulged in vinegar. Dr. Lauder Brunton has pointed out that vinegar taken for some time can lead to fatal emaciation; and I suppose by lessening the gastric secretion it so interferes with digestion as to cause the formation of digestive irritants (albumoses and allied bodies), which, after absorption by the portal system, are capable of setting up a hyperplasia of the connective tissue in connection with hepatic portal system. The symptoms which are noteworthy in these cases are the gradual onset of the disease in an indefinite manner; the various hæmorrhages, the absence of jaundice due to the stress of the disease falling on the portal system; the various nervous phenomena, namely, muscular twitchings in the boy, and delirium and coma towards the close of both cases; slight pyrexia in the evening.

On analysis of 112 cases under 18 years of age, it is seen that the part taken by alcohol and syphilis respectively as a cause of biliary cirrhosis is a much smaller one than was at one time supposed. Alcohol can be excluded in 46.4 per cent., and of the remainder it accounts for 18.7 per cent. Syphilis might be a possible cause in 16.3 per cent.; so that both taken together only account for 35 per cent., leaving nearly two-thirds of the cases attributable to other causes. Besides alcohol, other toxic substances after absorption by the hepatic circulation have been assigned as causes, namely, the malaria miasm found as a cause on the West Coast of Africa, the products of faulty digestion, and the use of too stimulating articles of diet. Dr. Eustace Smith³ mentions two cases recorded by Wettergreen and S. West, in which drinking largely of coffee might have been the cause. Dr. Gibbons⁴ has called the attention of the profession to the chief features of the disease (both inter- and intra-lobular) as seen in Calcutta, where it is practically limited to children of well-to-do Hindus, and as many as three or four children die in the same family before they reach the age of 2½ years, whilst the Mohammedan children nearly always escape. The reason of this fatality amongst the Hindus is obscure, and Dr. Alex. Crombie suggests that its prevalence being concomitant with the extension of the underground drainage system and the direct connection of this with the better class of houses, especially of the well-to-do Hindus, might offer the most likely clue to the etiology of this most remarkable disease.

The other conditions under which hepatic cirrhosis in the young occur are: (1) Congenital obstruction or deficiency of the bile duct, which by blocking back the bile causes a primary ectasis of the smaller canals, followed by extravasation of bile among the hepatic cells, and consequently their necrosis, with secondary formation of connective tissue. Drs. W. Legg⁵ and H. Gibbs⁶ have recorded cases which lived 5½ months and 7 months respectively. (2) Chronic venous congestion of the liver due to pulmonary or heart disease, although a frequent cause amongst adults, is very rare in childhood. (3) Pyle-phlebitis of syphilitic origin. (4) As part of a general overgrowth of fibrous tissue in several different organs due to a "fibroid diathesis." Dr. Pye-Smith⁷ and Dr. Cayley⁸ have recorded cases, and Dr. Burdon Sanderson suggests that the finding of pericarditis, pleurisy, and cirrhosis together points to their being the expression of some general disease of the lymphatic tissue throughout the body, inasmuch as the pleuræ and serous membranes generally, with the sheath of the biliary canals, are part of the same lymphatic system. (5) Combined with various forms of tuberculous disease. The liver may be either infiltrated with tuberculous

² St. Barth. Hosp. Rep., vol. xxvi.

³ Diseases of Children, p. 273.

⁴ Medical Annual, 1891.

⁵ Trans. Path. Soc., vol. 27, p. 178.

⁶ Ibid., vol. 34, p. 129.

⁷ Ibid., vol. 33, p. 172.

⁸ Ibid., vol. 27, p. 194.

¹ For some of the notes of Case I I am indebted to Mr. Shorland of Westbury; Case II I saw when under Mr. Willcox's care, and to him I am indebted for permission to publish my notes.

deposit or a child dying of tuberculous disease elsewhere may be found *post mortem* to have a cirrhotic liver without the deposition of tubercle, as in a case recorded by Legg.⁹ (6) Extension of the inflammation to the liver from the peritoneum or capsule of the liver. (7) Exposure to bad living. Dr. Cazalis has recorded two cases due, he thinks, to this cause. (8) Following the exanthemata. Botkin advanced this hypothesis in 1872, and since then French observers have studied the liver changes in scarlet fever, variola, diphtheria, typhoid, and especially in measles. From a large number of *post-mortem* examinations they were able to study all stages from simple lymphatic infiltration to confirmed cirrhosis. Klein has examined eight cases of acute interstitial hepatitis in scarlet fever patients. The only case in which symptoms referable to the liver came on during the infectious illness is one recorded by Pepper,¹⁰ and is that of a child who developed jaundice during an attack of measles, subsequently symptoms of hepatic cirrhosis, and death by coma ensued in less than a year; at the *post-mortem* examination a typical hob-nailed liver was found. Whether alcohol was administered during this child's illness is not mentioned.

The early symptoms in hepatic cirrhosis of children are often very obscure, there being nothing more definite than occasional "bilious attacks," epistaxis, or general debility for a year or more, and no more definite symptoms occur until one or both of the canal systems of the liver become implicated; if the parenchyma of the organ is the seat of the chief pathological change, the disease may run its course with symptoms very indefinite and anything but alarming until quite the close of the case. Several cases with very obscure nervous symptoms as the only ones have been recorded.

Frerichs narrates the case of a boy who suffered from general muscular weakness, difficulty of speech, and facial paralysis, but without mental symptoms until shortly before death. Dr. Ormerod¹¹ has described the case of a boy who gradually developed hemiplegia, with contractions of the side followed by similar symptoms on the other side, and later on by cystitis and bedsores, and a temperature of 105°. On *post-mortem* examination the liver weighed 10 ounces, and there were patches of softening in the outer layer of the lenticular nuclei, as well as in the pons. During life there were no symptoms pointing to the liver as the seat of cirrhosis. Dr. Buzzard has had one, and Dr. Gowers three cases in the same family, with symptoms very similar to the case of Dr. Ormerod, and Dr. Gowers¹² classifies his cases as tetanoid chorea. Dr. W. Legg recorded a case in which the early symptoms resembled typhoid fever or tuberculosis, and it was not until the case had been under observation for fourteen days that enlargement of the liver was detected and purpuric spots developed on the skin.

From the above short sketch of some of the early symptoms, it will be seen how very obscure some of the early, and even the late ones, may be, and how easily cases of this kind may perplex us, unless we have had the good fortune to come across a similar one.

The following conclusions may be drawn from the analysis of the above cases:

1. That alcohol, syphilis, tuberculosis, and malaria account for 50 per cent. of them, the other most frequent causes being probably the exanthemata and errors in diet.
2. That acute interstitial hepatitis is frequently found microscopically after the infectious fevers, especially after measles and scarlet fever, but the part played by the disease, alcohol, and diet respectively in those cases which afterwards become examples of cirrhosis, is an open question, as is also the reason why some livers are affected with the hypertrophic and others with the simple form.
3. That the symptoms may be wholly referable to the nervous system, the relation of the pathological changes in the liver to those in the brain being undetermined.
4. That severe pyrexia, quick pulse, and increased frequency of the respirations, are frequent symptoms, and may make the diagnosis difficult from tuberculosis, typhoid, and other fevers.
5. That the symptoms of failing health in children, with no

marked adequate cause, especially if associated with epistaxis or other hæmorrhages, the development of nœvoid growths, or the occasional presence of jaundice, should lead us to examine the liver for signs of cirrhosis.

6. That the later symptoms depend upon the canal system of the liver chiefly involved, or whether the parenchyma chiefly suffers.

7. That nearly half the cases occur between the 7th and 13th years, and that males are nearly twice as frequently attacked as females.

8. That if all severe symptoms disappear under treatment, they will certainly reappear and end fatally within, at the outside, as far as we know at present, a period of three years.

9. That the best treatment appears to be a tonic one, combined with special treatment for special symptoms.

10. That some cases are part of a general disease due to some poison getting admission to the general circulation and especially attacking the liver, owing to the slow circulation in the hepatic capillaries, just as, no doubt, acute yellow atrophy is a general disease, the chief pathological change found *post mortem* having caused it to be classified amongst the diseases of the liver.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

PROLAPSE OF PREGNANT UTERUS: "EXTRA-ABDOMINAL DELIVERY" AT TERM.

IN the EPILOGUE of the BRITISH MEDICAL JOURNAL of February 13th, p. 27, I read an extract of a report by F. Stein of a case of prolapse of the pregnant uterus and extra-abdominal delivery at term. I should like to record a case, similar in nearly all the details given, which occurred in my practice last year.

In February, 1891, I was called to a young woman, aged about 22, who had been in labour several hours, during which a tumour about the size of a child's head formed between the thighs. The condition was at once recognised as a threatened delivery of both uterus and fœtus. The uterus was now in a state of inertia, and any straining seemed to aggravate the condition. I found the external os rather rigid. It could not be easily dilated by the fingers, so I applied forceps to the head, and, with slight laceration of the cervix, I succeeded in delivering the child, which was to term, living, and fully developed.

The mother was in a very weak state, and was subsequently threatened with septicæmia, but eventually made a good recovery under rigid antiseptic treatment. I advised the use of a pessary, but, although I saw her afterwards, so slight must have been the inconvenience, if any, that the patient never returned for it.

STEPHEN M. LAURENCE, M.B., C.M.Ed.

Trinidad, Port of Spain.

GUNSHOT INJURY OF THE LEG.

IN view of a recent article in the BRITISH MEDICAL JOURNAL entitled "Weapons and Wounds in Future Wars," a description of the injuries inflicted by a Snider bullet, in a case I had lately charge of, may have some interest.

The projectile entered the leg about 3 inches below the upper extremity of the shaft of the tibia on its outer aspect, and passing upwards and inwards effected its exit by three openings in the thigh, one being on the inner and two on the anterior aspect of the limb. From one of the latter the bullet was removed, having just broken the skin. The projectile thus travelled from below upwards, the shot having been fired from a deep ravine, on the side of which the wounded man was.

On examination of the limb the upper extremity of the tibia was found to be comminuted. The condyles of the femur were widely separated, and the bullet passing upwards had splintered the shaft of that bone into numerous fragments for more than half its length. In its path the bullet, or more probably that splinter of it which made the wound of exit on

⁹ St. Bart. Hosp. Rep., vol. xiii.

¹⁰ Lancet, 1887, vol. ii, p. 226.

¹¹ St. Bart. Hosp. Rep., vol. xxvi.

¹² Quoted by Ormerod, loc. cit.