The response was encouraging, and after 90 minutes she was breathing without assistance and showed signs of returning consciousness. The endotracheal tube was no longer tolerated and was removed. After a further 30 minutes, however, when the patient was still apyrexic, she suddenly became extremely cyanosed with signs of gross pulmonary oedema. She was transferred to the Royal Devon and Exeter Hospital.

On arrival at the hospital she was barely conscious, and had extreme central cyanosis and severe pulmonary oedema. She was hypothermic, her heart rate was 90 per minute, and her blood pressure could not be measured. Endotracheal intubation was again performed, the air passages cleared by suction, and oxygen administered. She was given ampicillin, hydrocortisone, frusemide, and aminoophylline intravenously and morphine intramuscularly, and transferred to the intensive care unit, where intermittent positive pressure ventilation was commenced. A Capnscope respirator was used (stroke volume 250 ml, pressure 20 mm Hg, rate 20/minute). Examination of the blood at this time showed haemoconcentration (Hb 12.0%, P.C.V. 55, W.B.C. 4,500, N 152, K 4-0, Cl 110, urea 37). It was necessary to continue intermittent positive pressure ventilation for 54 hours, since, on two occasions it was discontinued and rapidly became uncontrollable.

Sedation was provided by the administration of morphine or phenoxymer. An endotracheal tube (nasal, Portex 9 mm) was left for a further 11 days to facilitate bronchial suction and efficient oxygen therapy. During this time asystole revealed a steady decrease in pulmonary oedema, which was confirmed by radiological examination.

After eight days her condition allowed her transfer to the general medical ward. Her continued progress was interrupted by repeated melena stools as a result of bleeding from a large hiatus hernia which required blood transfusion. She was finally discharged six weeks after her admission.

We are grateful to Dr. A. J. Daly for permission to report this case.

Jaundice and Methyldopa

Sir,—Jaundice has been reported as a rare complication of methyldopa therapy, and abnormalities in liver function tests are occasionally found in patients taking the drug. Only three such cases have been reported and one case with abnormal liver function tests but without frank jaundice has also been reported.

A 74-year-old man was first seen in 1961, when he was found to be hypotensive. He was a publican and took large amounts of alcohol. His liver function tests were within normal limits at that time. They were repeated two years later and were again normal. His blood pressure was controlled on reserpine and hydrodiamethazide.

He had not been seen again until his admission to Hounsdown Hospital on 29 October 1968. At the end of August 1968, for the first time, methyldopa 250 mg. per day had been prescribed. He was taking no other drugs with the methyldopa. Prior to that he was taking guanethidine. In mid-October 1968 he noticed that his right upper quadrant was tender. The liver was soft and not tender. He continued to take the methyldopa until he was admitted to hospital. Meanwhile the jaundice was becoming progressively deeper and his urine dark. He had had no symptoms prior to the onset of the jaundice; he was apyrexic and he remained symptom free during the whole period of his illness. He had not been in contact with any known case of hepatitis and had not received any transfusions or injections in the past.

On examination he was deeply jaundiced. His liver and spleen were not palpable and there was no abdominal tenderness. Blood pressure was 190/110, Haemoglobin 117%, W.B.C. 3,500/mm. (differential count normal), E.S.R. less than 1 mm./hr., serum bilirubin 17-6 mg./100 ml., alkaline phosphatase 16-6 King-Armstrong units/100 ml., thymol turbidity 0-5 units; total protein 6-3 mg./100 ml.—albumin 3-8 mg./100 ml., globulin 2-5 mg./100 ml. Unfortunately the alamine transaminase was not estimated at that time. His urine showed increased urobilinogen and bilirubin was present.

The methyldopa was discontinued immediately and the serum bilirubin level dropped to 8-9 mg./100 ml. in seven days, to 5-1 mg./100 ml. in 13 days, and 4-5 mg./100 ml. in 16 days. Alkaline phosphatase was 9 King-Armstrong units/100 ml. in 13 days, and aspartate transaminase level at that time was 71 units/ml. The jaundice began to fade 1 month after the onset of the jaundice was 79 units. Liver function tests returned to normal on 2 December 1968—that is, about four and a half weeks after discontinuance of the methyldopa.

In the absence of any constitutional disturbance or other known causes of hepatitis, injections, or blood transfusions and the rapid improvement on discontinuing the drug, no explanation for his jaundice could be found other than a reaction to methyldopa. The fact that the patient developed jaundice on very small dose of methyldopa (25 mg. daily), while other patients may take up to 3 g. daily for long periods and never develop jaundice, suggests a hypersensitivity reaction. One could suggest that, being a heavy drinker, the past alcohol might have caused liver damage and this predisposed to the onset of the hepato-cellular jaundice. However, his liver function tests previously were within normal limits.

We should like to thank Mr. S. A. Taylor, F.P.S., and Dr. D. C. P. Brown, of Merck Sharp & Dohme Ltd., for their kind assistance in preparing this report.

We are, etc.,
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Haemophilus influenzae Type B Septicaemia

Sir,—We would like to report another case which emphasizes the point made by Dr. R. J. Farrand (18 January, p. 150) that this condition may not be as rare as previously believed.

A 6-month-old girl was admitted in Feb-

BOND-TAYLOR.

REFERENCES

Horsenorthrop

Sir,—Mr. O. K. Gibbon (18 January, p. 187) writes that the results of a widespread return to basic principles in the teaching and practice of inguinal hernia repair would show that there is no need for the introduction of yet more new operations, as he condemns the operation recommended by Mr. L. F. Tinkler (28 December, p. 832). It has been the return to basic principles, however, which has allowed more and more surgeons to take advantage of the preperitoneal approach in curing groin hernias.

The preperitoneal muscle-splitting approach for the repair of inguinal hernias was first described by Mr. L. F. Tinkler, and Mr. Tinkler is right (in my opinion at least) in suggesting that the insertion of a prosthesis in the preperitoneal plane is a logical solution for some inguinal hernias. The approach is no innovation, having been used as long ago as 1943 for femoral hernias by the late Mr. F. S. Tinkler. However, the method was not taken up again until 1969 at the request of Mr. L. F. Tinkler, the present Mr. L. F. Tinkler. The use of artificial tissues in the preperitoneal plane has been shown by alimentation repair to be a sound procedure for repair of inguinal hernias, but it has been slow to be accepted by all surgeons. The method has its advocates, however, and it is to be hoped that it will be more widely used in the future.