hospital stores all remaining bottles. We are reminded, of course, of the great importance of clearly and accurately recording in the patient's notes the batch numbers of all transfusion fluids given, for without a record of these numbers it is not possible to detect and withdraw from circulation the infected batches.

We are indebted to Dr. F. J. W. Lewis for the laboratory investigations.

HOMOLOGOUS SERUM JAUNDICE IN INFANCY

BY

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AND

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The indications for intravenous plasma therapy in paediatric practice are many. Since plasma possesses all the therapeutic properties of whole blood, except those conferred by the red cells, it is especially indicated in shock with haemoconcentration and in hypoproteinaemia. In practice it is widely employed in the treatment of such conditions as burns and severe gastro-intestinal affections.

Despite its widespread use, the complications and sequelae of plasma therapy in infancy are surprisingly uncommon; homologous serum jaundice, for example, appears to be extremely rare. We have been able to discover only one possible case in the literature (Scheinberg *et al.*, 1947), though another more fully substantiated case, following not plasma but blood transfusion in an infant, has recently been described (Bruyn, 1947). In later childhood, however, jaundice has followed the use of convalescent serum in several instances (Propert, 1938; Beeson *et al.*, 1944), while in adults it is a common complication of plasma therapy (*British Medical Journal*, 1946, 2, 423), occurring in 7.3% of cases in one large series (Spurling *et al.*, 1946). Various explanations may be advanced for the relatively low frequency of occurrence in infancy: a temporary passive immunity to the causative virus may be present at this period of life, hepatitis may occur but remain symptomless, or jaundice in infants may fail to be correlated with previous transfusion treatment. The history of the recognition of the condition in adults suggests that the last possibility is not unlikely to prove correct. For this reason two cases of fatal homologous serum jaundice occurring in infants are recorded, together with an inquiry to determine the incidence of this sequel after transfusion with reconstituted dried plasma.

Case 1

T.B., a male child who had never been inoculated or vaccinated, had had no illness until the age of 7 months. He was then admitted to hospital with bronchopneumonia. He was treated with sulphathiazole and recovered from the pneumonia, but before discharge developed diarrhoea and vomiting. With these he became so severely dehydrated that he was treated with intravenous fluids, including reconstituted pooled plasma (120 ml. on each of two occasions, with an interval of six days), and subsequently made an uneventful recovery.

Four months later, at the age of 1 year, he was admitted to another hospital. He had been sleepy and jaundiced for two days. He had not vomited, though his appetite was poor, and his motions had been pale. On examination he was jaundiced and comatose. The pupils were pin-point but reacted to light. There was no neck rigidity. The liver was palpable one finger-breadth below the costal margin. His temperature was 98.4° F.

(36.9° C.) on admission, but later in the illness it rose to 102° F. (38.9° C.). Coma continued for two days, and convulsive movements occurred occasionally. The child died after four days' illness.

The interval between the first infusion of plasma and the onset of jaundice was 122 days.

Necropsy Findings.—A post-mortem examination was carried out 60 hours after death. The child appeared plump and was moderately jaundiced. There were no haemorrhages on the body surface. Abnormal findings were as follows. *Lungs*: A small number of recent haemorrhages had occurred into the parenchyma posteriorly on both sides. The *spleen* was slightly enlarged (weight, 25 g.), and on section the pulp was firm and the surface flat. *Small intestine*: Peyer's patches appeared hyperaemic but were not ulcerated. The *kidneys* were very pale, but histological study showed no change in architecture. The *gall-bladder* contained some rather pale bile which was easily expressed into the duodenum. The *liver* showed some post-mortem hypostases. It was of normal size (weight, 350 g.), and the surface was smooth and pale yellow in colour. On section the liver tissue was firm, appeared extremely fatty, and was slightly jaundiced. No haemorrhages were seen. The bile ducts were not dilated.

Histology.—Unfortunately, post-mortem changes obscured the histological picture, but despite the short history of illness there was subacute hepatic necrosis with a lymphocytic cellular infiltration around the portal areas. The number of recognizable liver cells was greatly reduced, and most of the survivors contained fat vacuoles. The bile ducts were unaffected; they were not dilated, and contained no bile thrombi. A frozen section confirmed the impression that all the surviving liver cells were heavily laden with fat.

Case 2

S.J., a female child, had had no illnesses until she was 11 weeks old, when she developed diarrhoea and vomiting, and was admitted to hospital. She was severely dehydrated, and was at once given intravenous saline and dextrose with 200 ml. of reconstituted pooled plasma. She recovered and was discharged well after two weeks. Six weeks later she began to pass frequent stools. After five days her urine was noticed to be bright yellow and she started vomiting. Next day she became jaundiced and was readmitted to hospital. On admission she was slightly jaundiced, but well nourished and not dehydrated. No other abnormality was noted. Two days later she had two convulsions and vomited some blood. Next day the convulsions became more severe and frequent, and she died.

The interval between the infusion of plasma and the onset of jaundice was 63 days.

Necropsy Findings.—A post-mortem examination was carried out three hours after death. The infant appeared well nourished, and there was slight jaundice with some cyanosis. No superficial haemorrhages were seen. Abnormal findings were as follows. In the *lungs* a small number of subpleural haemorrhages were present, and there were some small foci of collapse but no consolidation. The *spleen* was dark red in colour, and was enlarged to three times the normal size; histologically it showed only venous congestion. The *liver* was smaller than normal, with a smooth mottled yellow surface.

Histology.—Sections from the left and right lobes of the liver showed a uniform degree of subacute necrosis. There was almost complete disappearance of the columns of liver cells, only a few islands of extremely fatty liver cells surviving in places. The liver sinusoids were intact and there did not seem to be any gross haemorrhages. The material between the sinusoids consisted of amorphous debris, with a cellular infiltration composed partly of polymorphonuclear leucocytes but chiefly of mononuclear cells. There was some early bile-duct proliferation, but no evidence of obstruction of any of the bile ducts.

Comment

The diagnosis of homologous serum jaundice in these two cases seems reasonably certain. The intervals between

the administration of plasma and the occurrence of jaundice (122 days and 63 days, respectively) fall within the accepted limits. There was no history of contact with patients suffering from infective hepatitis. Unfortunately, it proved impossible to trace other patients who had been treated with reconstituted plasma from the batches used in these two cases. Histologically the picture was the usual one of subacute hepatic necrosis.

An attempt was made to ascertain the incidence of homologous serum jaundice in babies treated with plasma intravenously. Of 39 babies under 18 months old treated with reconstituted dried pooled plasma 26 survived the initial illness; among these were the two cases described above. Questionnaires were sent to the parents of the remaining 24 after an interval of six months or more; 17 replies were received, and no further cases of jaundice were brought to light. Unfortunately, on the available material, only a small and entirely inadequate series of control cases could be investigated. Of 18 babies in the same age group as those previously described, and treated during the same period with whole blood or saline intravenously, 12 survived. Questionnaires were sent to their parents, but only five replies were received. Among these there was no report of the occurrence of jaundice.

Summary

Two fatal cases of homologous serum jaundice in infancy are described. One child developed jaundice 63 days after plasma infusion, and died at the age of 5 months, after a short illness characterized by vomiting and convulsions. The other child was 1 year old when jaundice developed 122 days after infusion; he died after four days' illness, having been comatose for two days.

No other cases of jaundice could be traced in 17 babies who had also been treated with intravenous plasma, or in five babies treated with intravenous saline or whole blood.

It seems possible that homologous serum jaundice in infancy may go unrecognized, just as it did until recently in adults, because of the long incubation period. The present investigation into incidence, though inadequate, is reported in the hope that it will stimulate further reviews based on the systematic follow-up of cases treated with plasma; by this means more substantial evidence of the real incidence of homologous serum jaundice in infancy may become available.

We are indebted to Prof. C. Bruce Perry for permission to record these cases and for his continued interest and encouragement. To Prof. T. F. Hewer our thanks are due for the necropsy reports.

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The 1946 Year Book of General Therapeutics, November, 1945, to August, 1946, edited by Oscar W. Bethea (Year Book Publishers, Chicago, \$3.75; H. K. Lewis, London, 21s.) is a well-selected summary of current medical literature and gives a concise and balanced review of recent progress in therapeutics. The summaries include accounts of some of the newer sulphonamides likely to be useful in diseases of the colon, advances in the use of penicillin and streptomycin, the treatment of barbiturate poisoning with amphetamine, and recent experience with "benadryl" in allergic disorders. It is interesting to note that with the advent of newer influenza vaccines (A and B viruses) patients no longer prefer the risk of disease to the unpleasantness of prophylaxis. The Year Book is easy to read, well indexed, and enlivened by pithy editorial footnotes.

Medical Memoranda

Traumatic Delayed Rupture of the Gall-bladder in a Child Aged 9

Traumatic rupture of the gall-bladder is an injury of considerable rarity. Usually it is found in association with other abdominal injuries, these being of such a nature as to overshadow the biliary lesion. The following case, though not unique, is thought to be of sufficient interest to warrant recording.

CASE REPORT

A boy aged 9 years was admitted to hospital on July 29, 1946. Two days before admission he fell while attempting to jump from a stationary railway truck on to the embankment. He was unconscious for about half an hour, after which he was removed to a local hospital, where he regained consciousness. His condition was then one of mild shock, accompanied by vomiting. He was complaining of vague abdominal pain, and slight abdominal rigidity was present. The usual measures to combat shock resulted in marked improvement in his general condition, which remained satisfactory until the following evening, when he again complained of vague abdominal pain. On this account, and in view of the probability of laparotomy being necessary, he was transferred to a larger hospital.

On examination the child did not appear shocked and lay quite at ease while being examined. Abdominal distension was not present, but on palpating the abdomen there was generalized tenderness, maximal over the upper part of the right rectus muscle. Rigidity and "rebound tenderness" were absent, and there was no evidence of free fluid in the abdomen. On rectal examination the boy complained of pain about the umbilicus; otherwise nothing abnormal was detected. The pulse rate was 110 and the temperature 98° F. (36.7° C.). A tentative diagnosis of retroperitoneal haematoma was made.

Throughout the next two days his general condition remained excellent, the pulse being about 100 and the temperature varying between 98° and 100° F. (36.7° and 37.8° C.). This low-grade pyrexia was taken to indicate absorption from a retroperitoneal haematoma, and signs of peritonitis were still absent.

On July 31 the boy's condition suddenly deteriorated. He complained of severe abdominal pain and signs of shock were evident. He vomited three times within half an hour, the vomitus consisting of white frothy material. Abdominal examination revealed rigidity of the right rectus muscle and dullness of the right flank. Laparotomy was now considered necessary.

Operation.—The abdomen was entered by the right paramedian route. About 17 oz. (480 ml.) of bile-stained fluid was present in the peritoneal cavity. This fluid was removed, and on exploring the abdomen a hole measuring about 1/4 in. (6 mm.) in length was found in the fundus of the gall-bladder. Signs of inflammation were not evident in the organ, and although the edges of the hole were discoloured the perforation appeared to be of recent origin. There was no lymph exudate or any evidence of adhesions to surrounding structures. The small bowel was slightly dilated but otherwise normal. Drainage of the gall-bladder was effected through the hole by means of a fine rubber tube, and that of the associated area by a "cigarette" drain. The abdomen was closed around these drains.

No organisms were seen on microscopical examination of smear preparations, and culture of the bile-stained fluid from the abdomen showed no bacterial growth.

Routine post-operative measures were employed to combat shock, and within twelve hours the patient's general condition was satisfactory. Convalescence was uninterrupted and the child was discharged well after twenty-one days.

In the opening remarks I inferred that this case was not unique. Two similar cases are recorded—one by Siegel (1909) and a second by Hicks (1944). The latter case was one of perforation of the gall-bladder in a child aged 3 years; in the former, perforation occurred eight days after injury and was explained as being a distension gangrene due to secondary infection when the cystic duct became occluded with blood clot.

I should like to express my thanks to Mr. Matthew White for his permission to record this case.

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