cholangiogram, the changes of bile ducts were investigated in the living body. In the experimental animals, cirrhosis was produced by CCl₄, T.A.A and choline deficiency. Also, subacute hepatitis was induced by ethionine feeding. Tritiated thymidine was injected before sacrifice and autoradiographs were prepared. The biliary system was injected with Neoprene latex and the same done with a differently colored material for the portal vein.

(Result) 1) The corrosion casts of normal liver showed the ducts ramified dichotomously into approximately equalized branches for five to six divisions, where the branches were about 150 u to 250 u in diameter. From that point on most branches were much smaller than half the parent branch, being on the order of 20 u to 100 u. They extended at right angles from the larger branch, although some dichotomous branching was noted even in the terminal branches. No communications were found between ducts either in casts or in thick frozen sections.

2) In the case of postnecrotic cirrhosis with large regenerating nodules, the inflammatory process around bile ductules was severe. The nodules compressed vascular beds, especially hepatic vein. The cast of bile duct appeared denser and more intricate than the normal. Closer inspection revealed an increase in the number of branches beyond the fourth dichotomous divisions, involving principally ducts of less than 250 u in diameter. Whereas in the central casts small straight branches arose at right angles from the larger branch, in these specimens they originated at random angles and were also tortuous changing direction two or three times. Thick frozen sections demonstrated occasional small communications between adjacent ducts. However, no direct compression by regenerative nodules against bile ducts, as case with vascular system, was found.

3) The cases of diffuse septal cirrhosis (nutritional or Laennec cirrhosis) were in all respects similar to those obtained in cases of the postnecrotic cirrhosis, but all variations from the control, such as tortuosity and increase in number of branches, were not so exaggerated. Clinically, the jaundice was less severe than the former case.

4) In the experimental cirrhosis, the course of bile ducts became irregular and independent from the vascular systems. Autoradiographs revealed many labeled epithelial cells of bile duct. The subacute hepatitis exhibited the changes between normal and cirrhosis on overall respect.

(Summary) The obstructive jaundice seen in cirrhosis is due to irregular proliferation of bile duct instead of the direct compression by regenerative nodule to the vascular bed.

4. A PEDIATRICAL STUDY OF CHOLESTASIS

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In this study, the authors and his co-workers classified cholestasis occurring in children into two major divisions, intrahepatic cholestasis and extrahepatic cholestasis which were further sub-divided as shown in Table 1.

The present paper deals in particular with the cholestasis in infancy, focussing stress on the study performed by the authors.

1) Intrahepatic cholestasis:

As used in this paper, the term "infantile hepatitis" is a general name for the incomplete obstructive jaundice which is found infants. Although the cause for the disease is yet to be elucidated, it is believed to be of multiple factors. At present, however, all the researchers do not necessarily share the same view of this cause.

The authors make it a rule to give the name "infantile hepatitis, Type I" to those with which no hepatic dysfunction other than jaundice is observed and the name "infantile hepatitis, Type II" to those with which any hepatic dysfunction is observed. With the infant hepatitis, Type II, particularly bilirubin-mono-glucuronide shows a high value in the serum bilirubin fraction, both the serum GOT and GPT values are elevated, and a slight obstruction is recognized in the colloid reactions.
As regards the prognosis, the infantile hepatitis, Typ I, is generally satisfactory, with the disappearance of recognizable symptom within 3 months on the average. By contrast, the infantile hepatitis, Type II, is divided into two groups, one with satisfactory prognosis and the other with inferior prognosis. At any rate, it is difficult to make a precise judgement as to the prognosis on Type II at an early stage of disease.

Among cases of infantile hepatitis, Type II, some are found to have giant cells and others to lack them through histopathological examination. As will be discussed later on, however, the giant cells cannot be concluded as being recognized peculiarly in the infantile hepatitis.

Among infantile hepatitis, Type II, whose prognosis is inferior, there are some which experience repeated occurrence of nephropelitis, which eventually transforms into biliary cirrhosis of the liver.

It is generally difficult to make a precise diagnosis of viral hepatitis in infancy, and there are rare cases which manifest typical symptoms. Although there are quite many cases which receive exchange transfusion because of incompatibility of blood types such as of Rh and ABO in the neonatal period, very few reports are available dealing with cases that suffer from occurrence of serum hepatitis due to such transfusion.

The authors have an experience of treating a newborn infant who, upon diagnosis for incompatibility of blood type of ABO immediately after birth, received exchange transfusion of 600 ml of preserved blood and, on the third month of birth, was observed to be suffering from swelled liver. In this particular case, the systemic conditions on the third month of birth did not show pyrexia, displeasure or similar abnormality, but a hart swelling of liver of a length about 5 cm could be felt, the serum GOT and GPT values was elevated, and the colloid reactions were positive. Thus, the authors diagnosed this case, with some doubt, to be a serum hepatitis. However, this case showed a total serum bilirubin value below 3 mg/dl and manifested an inconspicuous extent of jaundice.

The microscopic tissue image of this cases as observed for pathological study showed a remarkable infiltrations of lymphocytes, and potty necrosis of liver cells at Glisson's capsule and at the center of the lobes, but no giant cells could be detected. After about 3 months' treatment, this case achieved alleviation and was released from the hospital. It is now under a followup observation.

As an example of toxic hepatitis, the authors have an experience of treating a patient who, in the course of infantile hepatitis, Type II, was orally misapplied about 30 ml of 0.1% ethyl mercury sodium thiosalicylate (Mersonin) and, as a consequence, had her hepatitis aggravated. Aggravated symptoms were total serum bilirubin value which rose about twice as great, the serum GOT and GPT values which both rose about 4 times as great, and swelled liver which increased in size. These symptoms were alleviated invariably through treatment.

Furthermore, the authors have an experience of treating a pair of sisters suffering from Rotor's hyperbilirubinemia. The elder sister was 7 years old, and the younger sister 5 years old, and both were observed to have chronic latent jaundice. So far as findings of examination are concerned, no conspicuous abnormality was observed excepting a relative rise in the conjugated bilirubin values and a delay in the BSP excretion value. No conspicuous abnormality could be detected either from the pathological tissue investigation by many of liver functions test.

2) Extrahepatic cholestasis

The authors have an experience of treating a total of 15 cases of congenital atresia of the bile ducts. Of this many cases, 10 cases were of female patients. Where the time of occurrence of disease is concerned, 8 of the 15 cases were of jaundice which followed physiologic neonatal jaundice. However, the remaining 7 cases, although physiologic neonatal jaundice disappeared once completely, complained of jaundice which returned after an interval of from 7 days to 47 days. All these cases were reported to have feces yellow in color before the recurrence of jaundice.

This suggests that there is a possibility of some, but not all, bile ducts being closed after