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OBSTRUCTIVE JAUNDICE IN EARLY INFANCY

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Jaundice is common in newborn infants. It is probable that 40% of infants have a serum-bilirubin level of at least 4 mg. per 100 ml. during the first week of life. In the majority jaundice is mild and transient although not strictly 'physiological', but in others it is of serious import because of the underlying cause and/or the risk of kernicterus.

Jaundice in infancy is conveniently subdivided into non-obstructive and obstructive. In non-obstructive jaundice unconjugated bilirubin is raised and this is the substance which is toxic to the basal ganglia of the brain. It is twice as common as obstructive jaundice (Table I) and not of great concern to the surgeon apart from the assistance he can give with exchange transfusions.

TABLE I. JAUNDICE IN THE NEWBORN—144 CASES

Non-obstructive—93
- Functional immaturity of the liver.
- Haemolytic disease of the newborn.
- Congenital spherocytosis.
- Congenital familial non-haemolytic jaundice.

Obstructive—51
- Congenital atresia of bile ducts.
  - Extrahepatic.
  - Intrahepatic.
- Extrinsic obstruction.
- Cystic fibrosis of pancreas.
- Galactosaemia.
- Infections:
  - Bacterial.
  - Spirochaetal.
  - Herpetic.
  - Congenital hepatitis.
  - Cytomegalic inclusion disease.
  - Toxoplasmosis.
- Haemolytic disease with inspissated bile.

In obstructive jaundice conjugated bilirubin is raised. This substance is derived from bilirubin which has been conjugated by bilirubin glycuronyl transferase in the liver cells and is elevated in both bile-duct obstruction and parenchymal liver disease. The commonest cause of obstructive jaundice in young infants is biliary atresia (Fig. 1) which is of great concern to the surgeon. The other causes are mainly non-surgical (Table I), but difficulties arise in the differential diagnosis. This applies particularly to congenital or neonatal hepatitis and the inspissated-bile syndrome. Indeed, it has even been suggested by some that the 3 conditions, biliary atresia, neonatal hepatitis, and the inspissated-bile syndrome may be different manifestations of the same basic disorder. Jaundice prolonged beyond the second or third week is usually obstructive and caused by one of these 3 conditions.

Most workers still favour the viral nature of neonatal hepatitis. It is assumed that the virus can cross the placental barrier and that, although the mother has subclinical infection, the baby may have obvious clinical jaundice. In severe forms of this disease in the infant, total biliary obstruction may occur, and the empty, collapsed extrahepatic bile ducts are often so minute that they may not be recognized as such. It is in these patients that the differentiation from atresia becomes extremely difficult, and some workers have even suggested that the primary lesion is a congenital maldevelopment of the intercellular bile canaliculi leading from the liver cells to the bile channels with secondary atrophy of the ducts.

Since the original description of the inspissated-bile syndrome in 1935, the concept of this condition has gradually changed. There are, however, a very small number of infants with haemolytic disease who remain jaundiced for several weeks. The main pigment in their blood is bilirubin glucuronide which becomes elevated because of blockage of the intercellular bile channels by dark, inspissated bile. The portal tracts are normal, as are the main bile ducts, and the jaundice usually clears without treatment.

The generally accepted view of the aetiology of biliary atresia is that it is caused by an arrest of development during the solid stage of bile-duct formation. The possibility exists, however, that it may represent the end result of foetal hepatitis with sclerosis of the extrahepatic ducts, akin to cholangiohepatitis or cholidochitis found in adults. There is definite evidence that the biliary tree is still developing in the neonatal period and some grounds for believing that the atretic process may progress after birth. Many believe that intrahepatic atresia is a viral hepatitis which has occurred *in utero* or early in the neonatal period.

It is agreed that no single clinical feature or laboratory test, nor any combination of signs or battery of tests, will regularly distinguish between biliary atresia and neonatal hepatitis. All too often the final diagnosis has to be made at laparotomy or even at autopsy. In this report the clinical

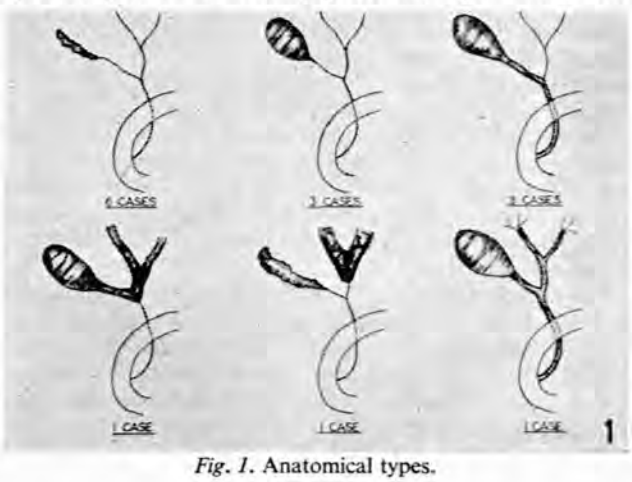


Fig. 1. Anatomical types.

features and laboratory data of 15 proved cases of biliary atresia are compared with the findings in 15 patients with hepatitis who developed jaundice before the age of 3 weeks. This analysis confirms earlier reports and our clinical impression that differentiation of prolonged obstructive jaundice in infancy may be exceedingly difficult at times. Points of particular value in differential diagnosis will be discussed.

The patterns of biliary atresia encountered in our patients are shown in Fig. 1. There was one patient who had intrahepatic atresia. He is still alive at the age of 2 years, but in poor condition, with moderate jaundice, xanthomatosis of the skin, a large irregular liver, and recurrent haematemesis. The average age at death of patients suffering from intrahepatic biliary atresia is 5 years. Only 2 of the 14 patients with extrahepatic atresia had an anomaly that was amenable to surgical correction. One of these died of progressive biliary cirrhosis 3 months after operation. The other is alive and well with no jaundice 6 months after surgery. The average survival in 'inoperable' cases was 5 months, and several lived for 7—8 months.

Of the 15 patients with neonatal hepatitis, 6 are known to have died and 4 to have recovered completely. Five have failed to report for follow-up. Two of the 6 deaths were caused by intercurrent disease, 1 by acute liver failure, and 3 by progressive cirrhosis. The infant who died of acute liver failure was operated upon and surgery no doubt hastened the end. Two of the patients who have survived were operated upon for suspected biliary atresia. The original operative diagnosis was 'inspissated-bile syndrome', but in retrospect these cases have now been classified as infective hepatitis.

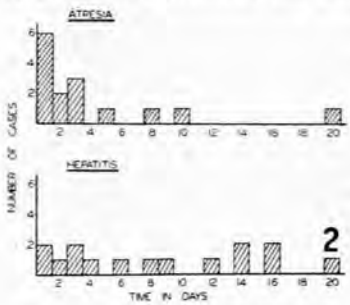


Fig. 2. Age at onset of jaundice.

Age at Onset of Jaundice (*Fig. 2*)

Although one would expect jaundice to be present from birth in biliary atresia, it has been stated that the diagnosis of atresia is unlikely in infants who become clinically icteric on the day of birth, and most paediatricians hold the viewpoint that recognizable jaundice on the first day of life is caused by haemolytic disease until disproved. However, a history of jaundice since birth may be obtained in about one-third of cases and this is so in our series (6 cases). Usually the jaundice is noticed during the first week, but not uncommonly 2 - 3 weeks elapse before it is dark enough to become conspicuous. The jaundice is usually persistent and progressive, although some parents have insisted that there are fluctuations in intensity.

It is usually said that the stools are always clay-coloured or white from birth, even though jaundice is noticed only later. We have not been so successful in obtaining an accurate history of acholia. Confusion often arises from staining of the stools by highly concentrated urine. Later the stool itself may be yellow from bile-stained succus entericus, saliva, and mucosal cells, and small amounts of stercobilin may be found on chemical testing. Nevertheless, persistent acholic, putty-like stools from birth are highly suggestive of biliary atresia, while dark stools rule out this diagnosis.

In the patients with neonatal hepatitis there was no significant difference in the age of onset and progression of jaundice. In two-thirds jaundice was noticed by the eighth day, and in all but 2 the stools were described as clay-coloured or white. However, fluctuations in the depth of jaundice and in the colour of the stools may occur and should be sought for as a differential point.

In all the patients of both groups the urine contained large quantities of bilirubin, but no urobilin. Absence of urobilin is to be expected in atresia, but it may occasionally be present in patients with hepatitis. Others have found urobilinogen in about one-third of infants suffering from atresia.

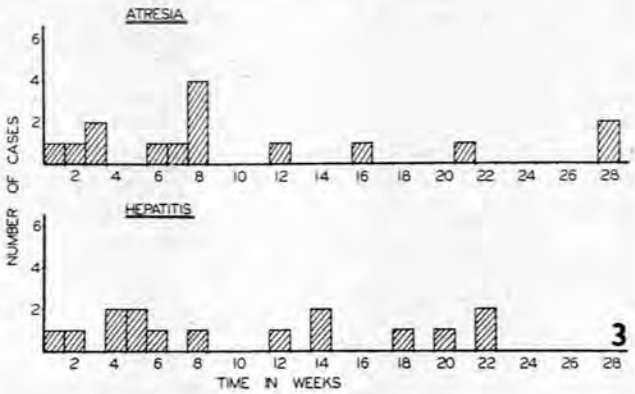


Fig. 3. Age on admission.

Age on Admission (*Fig. 3*)

There was no significant difference in the 2 groups, although in general the babies with biliary atresia tended to be in better condition and well nourished during the first 2 - 3 months. The older infants with biliary atresia tended to be below par, while some of those with hepatitis who were improving were in good condition. It should be remembered that the average duration of life of infants with extrahepatic biliary atresia is 5 months, and many may survive for 7 - 8 months. (In patients with intrahepatic atresia the children may live? or several years.)

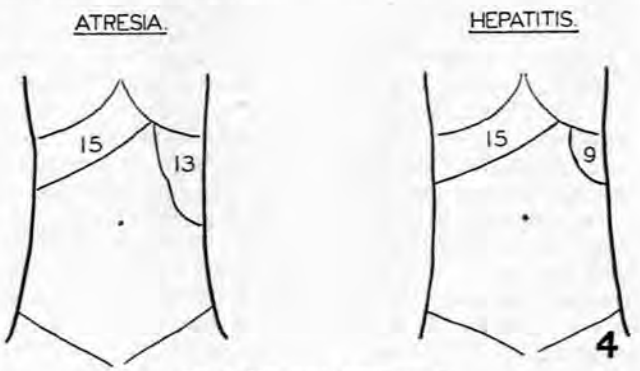


Fig. 4. Hepato-splenomegaly.

Hepato-splenomegaly (*Fig. 4*)

Enlargement of the liver was present in all the patients, but tended to be grosser in those with biliary atresia. Splenomegaly, often of considerable degree, was present in 13 of

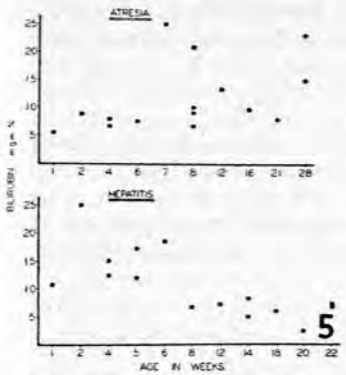


Fig. 5. Bilirubin on admission.

the infants with atresia. Among those with hepatitis the spleen was palpable in 9 only, often only just the tip being felt. This feature may thus be of some significance.

Serum Bilirubin

The total bilirubin levels at various ages on admission are shown in Fig. 5.

In biliary atresia values ranged from 8 to 25 mg. per 100 ml. There appears to be a slight rise in the mean values with age, but individual examples are glaringly out of line. In the hepatitis group the values were very similar, but showed a clear tendency to lower values with increasing age.

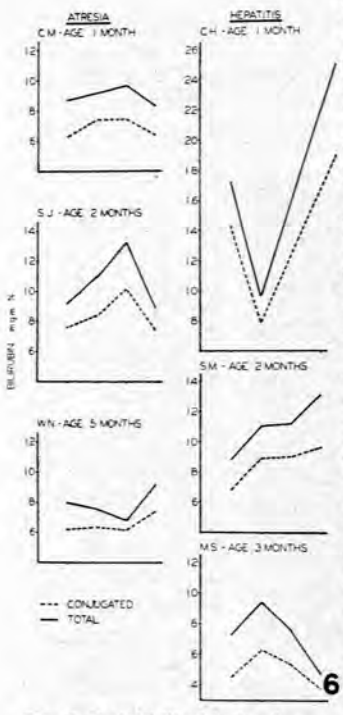


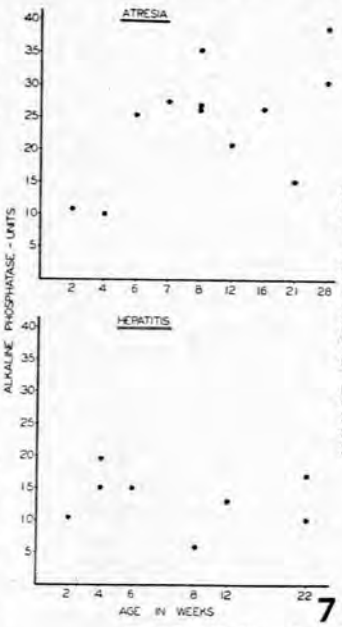
Fig. 6. Serial bilirubin levels.

Serial determinations of the conjugated and total bilirubin of 3 patients in each group are shown in Fig. 6. It is apparent from these graphs that no single pattern can be considered characteristic. However, in biliary atresia, over a period of 4 weeks, the bilirubin level shows no gross fluctuations and there is a general tendency to increase. Others have found an upward trend in two-thirds of the cases suffering from atresia. In hepatitis 3 patterns may be found, viz. constant over a period of 4 weeks, rapid deterioration, or rapid improvement—depending on the progress of the disease. The key point of differentiating hepatitis from atresia is the continued decline in bilirubin levels that occurs in favourable cases, but it requires a period of observation of at least 4 weeks to determine this, and the test is only a *fair* method of differentiation.

Liver-function Tests

The zinc-turbidity, thymol-turbidity and thymol-flocculation tests did not show any significant difference between the 2 groups. Grossly abnormal values were found in 3 of the patients with atresia and 5 of those with hepatitis. (They were all late cases.) Most of the high values occurred after the age of 3 months. Others have found these tests *fair to poor*, and the bromsulphthalein (BSP) tests have been of little value. Serum-cholesterol levels tend to be high in atresia, especially in the intrahepatic variety, but only in the late stages.

High alkaline phosphatase values, i.e. over 20 Bodansky units, were found in 7 patients with atresia and in none of those with hepatitis (Fig. 7). Levels above 30 occur almost



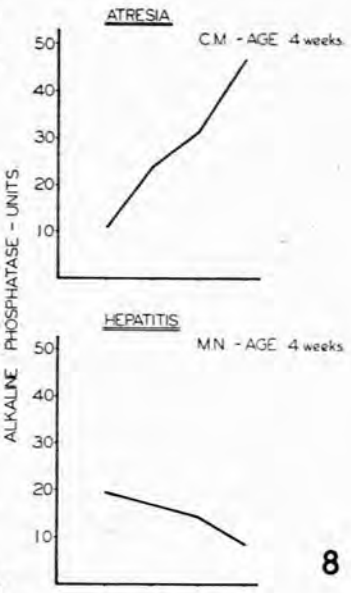


Fig. 7. Alkaline phosphatase on admission.

Fig. 8. Serial alkaline phosphatase.

exclusively in atresia, but in many cases the level is below 30. Furthermore, the patients with atresia tended to show a steady increase (Fig. 8), while many of those with hepatitis showed a significant decline over a period of 4 weeks. This test may, therefore, be of some value in selecting patients over the age of 2 months or after a period of observation of 4 weeks.

Serum-transaminase Levels

Transaminase studies (glutamic oxaloacetic transaminase and glutamic pyruvic transaminase) are of value in determining not only the extent of liver injury, but also the degree of parenchymal destruction, and may be of value in differentiating atresia from hepatitis.

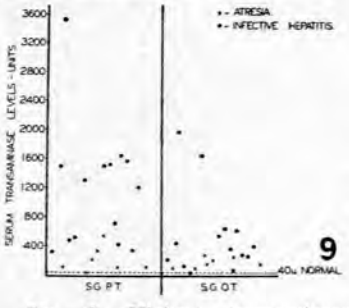


Fig. 9. Highest transaminase values—21 cases.

In Fig. 9 the levels in early cases of atresia and hepatitis are depicted. It should be noted that in hepatitis the levels are considerably elevated at the onset of the illness and then fall, while in atresia they are normal at the beginning and then rise to moderate heights only (Fig. 10). In general the levels in hepatitis are considerably higher than in atresia, especially in regard to SGOT. However, individual exceptions are common, and at about 2 months, when many patients are first referred to hospital, there may be no significant difference.

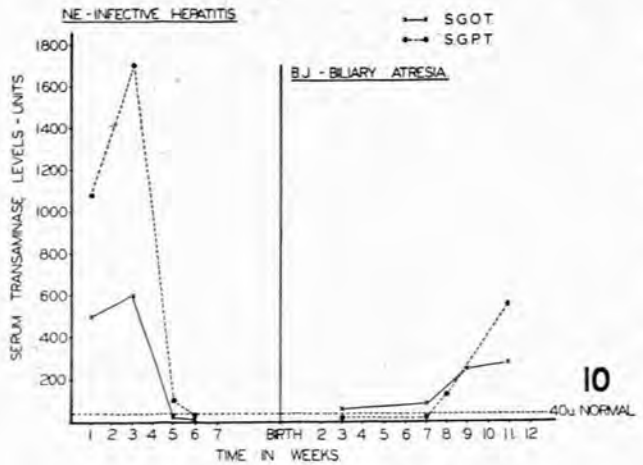


Fig. 10. Transaminases in jaundice.

Lathner and Smith, from Durham University, report that the *transaminase/alkaline phosphatase ratios* show than in adults nearly ¾ of patients with hepato-cellular disease have a ratio of 7 or above, whereas only 7% with obstruction have ratios of 7 or above, and more than 50% have ratios of 3 or below. Among the latter higher ratios occur when biliary cirrhosis develops. These authors claim that with this method of assessment the diagnostic error is only 8%. We are still investigating the value of this ratio in newborn infants suffering from jaundice.

Bile in Duodenal Aspirate

We have performed this test in only a few cases. It may be helpful, especially if done under fluoroscopic control. If consistently negative, atresia is likely and exploratory surgery indicated. Duodenal intubation combined with BSP excretion and the use of magnesium sulphate to dislodge 'inspissated bile' may be of more value.

Radioactive Rose-Bengal Test

Robert Brent, of Jefferson Medical College, Philadelphia, has drawn attention to the use of radioactive Rose Bengal in the evaluation of infantile jaundice, but we have not yet had the opportunity of using this test. In biliary atresia stool excretion is less than 2% of I^{131}, administered while urinary excretion is high. In hepatitis, on the other hand, stool excretion is usually well above 4% while urinary excretion is low.

Liver Biopsy

To differentiate histologically between biliary atresia and neonatal infective hepatitis is by no means easy in the individual case. All the features which have been stressed by various authors as favouring a diagnosis of neonatal hepatitis may be found in patients with proved biliary atresia. However,

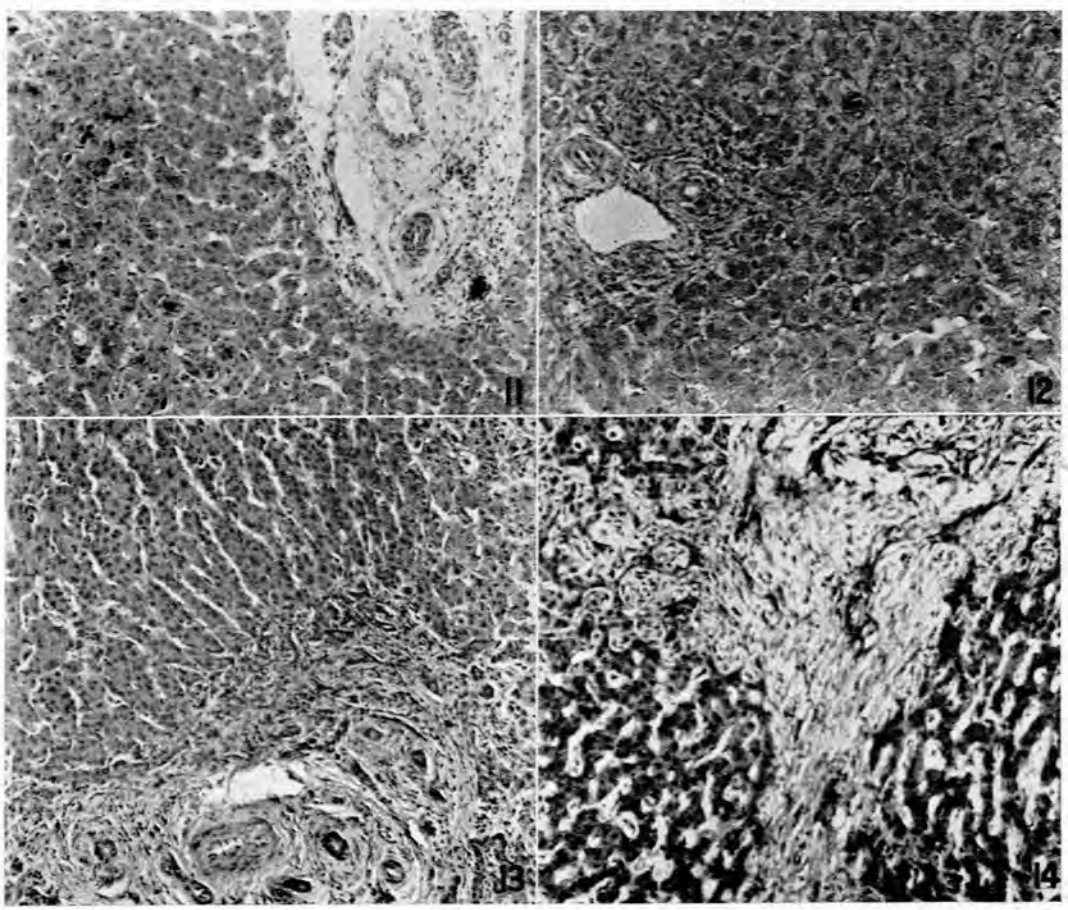


Fig. 11. Formal biopsy from a 4-week-old infant. Portal tracts are prominent for this age and signs of obstructions are far in excess of parenchymal damage. An early case of atresia.

Fig. 12. Formal biopsy from a child of 6 weeks with neonatal hepatitis. Note the extensive parenchymal damage in comparison to the relatively normal portal tracts. Obstructive signs are minimal.

Fig. 13. A proved case of atresia—here the position is more difficult. There is some parenchymal damage and also periportal fibrosis and bile-duct proliferation— the latter more marked, however, and histologically favouring atresia.

Fig. 14. Histological pattern in a late case of biliary atresia. In this case a well-established biliary cirrhosis is present.

some features are rather more common in hepatitis than in atresia, and *vice versa*, and it is our opinion that the relative preponderance of these features, weighed in conjunction with the duration of jaundice, provides the only feasible means of assessing the histology in the individual case.

Giant-cell formation, evidence of cell necrosis, and hydropic and eosinophilic degeneration may be present to the same degree in either condition, but it is our experience that these histological changes are present much earlier in the course of neonatal hepatitis than they are in atresia, and this may well be an argument in favour of early biopsy. The impression is also formed that these changes *ab initio* affect the parenchyma in a more haphazard and diffuse fashion in neonatal hepatitis than is the case in atresia, and that in atresia they are first seen in those parenchymal cells abutting on the portal radicles.

Perilobular fibrosis and bile-duct proliferation occur in both conditions, but seem to be established much earlier in patients with atresia, and are usually reasonably marked by the time that parenchymal damage of the types outlined above are established. In neonatal hepatitis, however, it is our experience that parenchymal damage usually precedes periportal fibrosis and bile-duct proliferation for some appreciable time. This does not make differentiation any easier, though, in patients presenting after jaundice, parenchymal damage has been of some duration.

The maximal situation of the bile thrombi and the situation of foci of inflammatory cells have not proved particularly helpful, nor has the presence of intracellular bile stasis.

In late cases where well-marked cirrhosis is already present, the histological differentiation would seem to be wellnigh impossible unless the cirrhosis is frankly biliary in type. Figs. 11-14, taken from our cases, illustrate some of these features.

Summing up from the histological standpoint and for the reasons stated, there is in our opinion little to lose and a great deal to be gained from early and adequate biopsy and, whereas we feel that both parenchyma and portal radicles must be adequately visualized, needle biopsy of the liver falls far short of a formal biopsy taken at laparotomy. Some claim that the biopsy specimen may be examined by frozen section, but most authors agree that this is expecting too much from the pathologist. It is our practice to perform a formal biopsy and to examine the specimen by paraffin section.

THE THERAPEUTIC PROBLEM

From the data given above it should be obvious that the differential diagnosis between biliary atresia and neonatal hepatitis during the first 2 - 3 months of life is often impossible on clinical and biochemical grounds. The principal rôle of the surgeon is to recognize and treat the children affected with atresia before the damage from obstruction has become irreversible. Numerous attempts have been made to decide the 'point of no return' as far as obstructive jaundice is concerned, but there is, as yet, no unanimity. It is agreed, however, that moderately advanced cirrhosis may be present at 2 months, and certainly at 3 months, and it has repeatedly been emphasized that prolonged delay before operation will prevent salvage of those infants with surgically correctible lesions because of progressive biliary cirrhosis. It is also felt by some that 'disuse atrophy' or chemical cholangitis over a period of 2 - 3 months may render a previously 'operable' condition 'inoperable', particularly if biliary atresia is in fact caused by some form of sclerosing cholangitis. Such a course of events in these tiny infants makes it evident that definitive therapy for atresia of the ducts must be carried out, preferably before 2 months of age and certainly before 3 months. Most writers recommend exploration at 4 - 6 weeks.

It should be remembered, however, that the diagnosis of biliary atresia at operation is a time-consuming performance which involves complete exploration of the lesser omentum and porta hepatis. This entails prolonged anaesthesia and inevitable trauma which, in a case of hepatitis, might tip the scale against survival. Indeed, it has often been reported that morbidity and mortality are greatly increased in patients with infective hepatitis who have undergone surgical exploration. In at least 1 of our patients death was precipitated by surgery, and postoperative intestinal obstruction is a common complication.

Another factor which must be seriously considered is that the collapsed, bile-free ducts of hepatitis may not be recognized at operation. This may result in an erroneous diagnosis of inoperable atresia or, worse still, in irreparable damage to the minute ducts during dissection. The surgical diagnosis of inoperability is far from easy and often inaccurate. This is supported by the fact that a number of spontaneous recoveries following negative exploration have been reported in the literature. Indeed, there are now about one-fourth as many reports of recovery following simple exploratory laparotomy as there have been following accepted corrective procedures. In this connection, it seems fairly clear that many cases of 'inspissated-bile syndrome' will clear up without surgery, and that the risks of anaesthesia and complete biliary exploration are not justified. Furthermore, it has been suggested that, because of the mortality and morbidity of surgical exploration, operation should not be carried out before the age of 5 or 6 months to allow those cases that would clear up spontaneously to do so and those that had undoubted atresias to manifest themselves by progressive signs of obstruction. Needless to say, this has not been met with general approval.

It is obvious that an accurate estimate of cure rate in biliary atresia is of fundamental importance. If an appreciable number of these infants can be cured by surgery, then the risks of a few unnecessary early explorations in patients who prove not to have biliary atresia are justified. A review of the literature, however, shows that only 20% have 'operable' lesions and only 8% are 'cured'.

Cholangiography

In response to the suggestion of 'the waiting game,' various authors have introduced the more expedient and briefer surgical procedure of liver biopsy and cholangiography at an early age to determine which patients are surgical candidates as opposed to those who have medical disease. This method has now been widely accepted and is used by us. The main advantage is that it involves minimal anaesthesia and surgical trauma and yet provides accurate information at a stage when liver damage is still minimal. It can be carried out with safety in infants of less than a month old, which is the optimum time to arrive at a definitive diagnosis.

The procedure may be carried out under local or general anaesthesia. A short transverse or muscle-splitting subcostal incision is made about an inch above the liver edge. This affords adequate exposure and protects against subsequent disruption. The subhepatic area and porta hepatis are in-

spected, but no dissection is done for fear of damaging minute bile ducts. If the gall bladder is absent or represented by a fibrous cord, it is probable that the child also has obliteration of the extrahepatic bile ducts. If a gall bladder is present, a small rubber or polythene tube is inserted into it and an immediate cholangiogram is taken, using 35% urodione. Only small quantities of dye should be used—we inject ½ ml. for the first plate and then ½ ml. more for each of 3 or 4 more plates. In either instance a deep wedge of liver is resected and the biopsy site oversewn. The subsequent management depends on the findings, viz:

Fig. 15. Specimen of a case of complete biliary atresia with secondary biliary cirrhosis.

1. No gall bladder present (Fig. 15).

If a frozen-section report on the liver biopsy can be obtained proceed immediately with exploration. If not, await result of report and then explore.

2. Gall bladder present.

(*a*) Dilated bladder, cystic duct and common hepatic duct, but no common duct. Explore immediately.

(*b*) Bladder and all ducts patent and not dilated. Remove the catheter. No further surgery required.

(*c*) Other.

Do not remove the tube, but bring it out through a small stab at the liver's edge and place it on drainage. Repeat the X-rays after 24 hours to see if any further ducts have filled and repeat the cholangiogram through the cholecystostomy tube if necessary. Await result of the liver biopsy before proceeding further.

It should be noted that cholangiography *via* the gall bladder is possible in only about half of the patients with atresia, and in about a third of these the true anatomical pattern may not be visualized because of the diminutive size of the ducts. It is for this reason that the tube is left *in situ* and further plates may be necessary. Furthermore, cholangiography in cases of hepatitis may fail to show up the ducts owing to their minute calibre and the presence of inspissated bile. It is, therefore, advisable to irrigate the ducts before injecting the dye and to take further plates if no ducts are visualized at operation.

It should be obvious that in many cases the final decision about exploration will depend on the pathologist.

SURGICAL TREATMENT

We do not propose discussing the details of treatment. Suffice it to say that exploration should be preceded by irrigation through the gall bladder with warm saline followed by injection of methylene blue to outline ducts that may be present. Adequate exposure (some even use a thoraco-abdominal approach) and extreme gentleness are of the greatest importance. Any bulging structure which may be a dilated bile duct should be aspirated with a fine needle—it usually turns out to be a vein! If no ducts are found in the lesser omentum or portal fissure, any depression in the hilus of the liver must be explored with a probe.

If an 'operable' condition is found, the duct or gall bladder should be anastomosed to the duodenum or to the jejunum Roux-en-Y. The anastomosis is usually best performed over a small tube which will dislodge and be passed in due course.

From time to time reports appear of ingenious methods of dealing with 'inoperable' cases. These include multiple punctures of the hilum of the liver to establish a biliary fistula; anastomosis of small bowel to a scarified area of liver; 'opening up' of the hilus of the liver with a valvulotome followed by hepatico-enterostomy; and Longmire's procedure. In some cases successes have been claimed, but it seems altogether reasonable to assume that, coincidental with such an operation, an overlooked common duct spontaneously opened and took over its normal function. None of these procedures have proved to be worth anything at all and the procedure of choice if no correctable atresia can be found, would appear to be to resist the therapeutic urge by leaving well alone and getting out. It should be remembered that the operative diagnosis is not infallible and that there are reports of at least 14 patients who were thought to be inoperable and given a hopeless prognosis, who have subsequently cleared their jaundice.

CONCLUSION

Congenital atresia of the bile ducts remains the darkest chapter in paediatric surgery. The aetiology is unknown, clinical diagnosis is at the best a good guess, laboratory tests often fail, the histopathology is variable, radiology is of limited value, and surgical exploration far from satisfactory. Above all, the results of treatment certainly leave a great deal to be desired. The correct management of the jaundiced infant calls for careful clinical assessment, integrated teamwork, expert surgical judgement, and a gentle surgeon.

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