JAUNDICE IN THE NEWBORN

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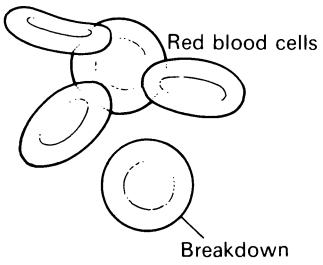
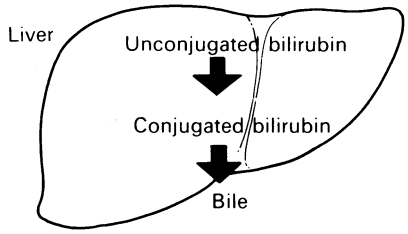
From the revised edition of "The First Year of Life," to be published next week



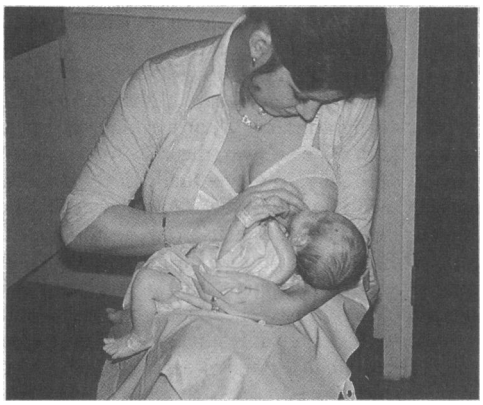


Jaundice is a yellow colour of the skin caused by high concentrations of bilirubin. Very severe jaundice may damage the cells of the basal ganglia and brain stem. This damage is produced by the fat soluble unconjugated bilirubin. If jaundice is severe high bilirubin concentrations may result in deafness, cerebral palsy, or death.

Neonatal jaundice is due to an increased bilirubin load with a transient inefficiency of hepatic excretion resulting from decreased activity of glucuronyl transferase in the liver. There are additional factors. Some of the conjugated bilirubin excreted in the bile is normally deconjugated in the small intestine and reabsorption is enhanced by the slower gut transit in the starved newborn. Bilirubin is absorbed from meconium and there is no intestinal flora to degrade bilirubin to urobilinogen.

Common causes of jaundice in the newborn

	Onset
Red cell incompatibility	Within 24 hours of birth
Physiological jaundice	After 24 hours
Septicaemia	Usually after 4th day



Common causes of jaundice include hepatic immaturity, red cell incompatibility, infection, and breast feeding.

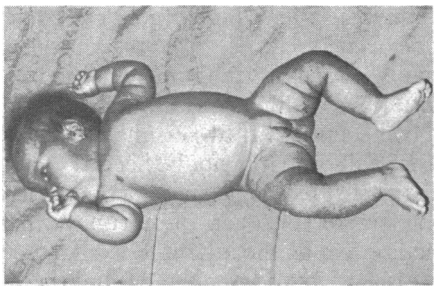
Jaundice due to hepatic immaturity, or "physiological" jaundice, is common both in preterm and in full term babies. A temporary deficiency of glucuronyl transferase enzymes reduces the rate of conjugation of bilirubin, with a consequent retention of unconjugated bilirubin. In full term infants the jaundice always appears after the first 24 hours of life and reaches a peak on the fourth or fifth day. In preterm infants it usually begins 48 hours after birth and may last up to two weeks.

In babies with red cell incompatibility jaundice appears within 24 hours of birth. The main causes are (*a*) incompatible rhesus grouping and (*b*) incompatible ABO grouping; the mother's blood is usually group O and the infant's group A or, less commonly, group B.

The common infective causes of jaundice are septicaemia and urinary tract infection. Septicaemia is especially likely to be present if the jaundice appears after the fourth day of life, but it is a possibility in any infant who seems ill. In urinary tract infections the jaundice is of hepatic origin.

In about 2·5% of infants who are breast fed the serum bilirubin rises to values between 260 and 360 μmol/l in the second or third week of life. These infants have no symptoms. If breast feeding continues the concentration remains constant for three or four weeks and falls to normal at 4-16 weeks. An abnormal progesterone concentration has been shown in the milk of some of the mothers.

Rare causes of jaundice in the newborn



- Hypothyroidism
- Galactosaemia
- Viral hepatitis
- Atresia of bile ducts
- G6PD deficiency

Rare causes of jaundice include hypothyroidism, galactosaemia, viral hepatitis, and atresia of the bile ducts. These cause prolonged jaundice lasting more than 10 days. Glucose-6-phosphate dehydrogenase deficiency is another cause of prolonged jaundice but it can also produce a clinical picture similar to blood group incompatibility.

In hypothyroidism physiological jaundice is prolonged, the plasma thyroxine (T4) concentration is reduced, and the thyroid stimulating hormone (TSH) concentration is increased.

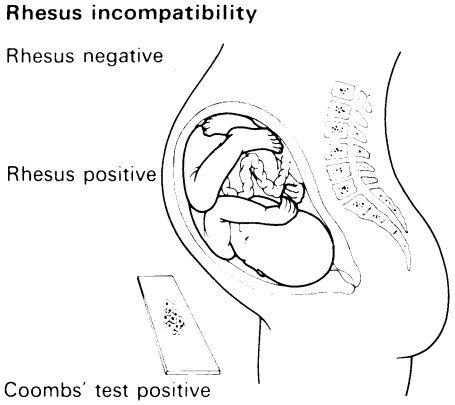
In infants with viral hepatitis, which is usually due to intrauterine infection, the stools are pale, the urine dark owing to bile, and there is a high concentration of conjugated bilirubin in the plasma.

It is difficult to differentiate between hepatitis and atresia of the bile ducts clinically, and they may represent the two ends of a range of disease. If jaundice persists more than 10 days the advice of a paediatrician should be sought.

In galactosaemia the urine gives a positive result on testing with Clinitest for reducing substances, but the Clinistix test may be negative. The infant needs to be referred to a paediatrician immediately for special investigations.

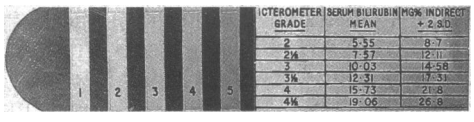
Glucose-6-phosphate dehydrogenase deficiency occurs in infants of Mediterranean, African, or Chinese stock. This hereditary red cell enzyme defect is found in babies with haemolytic episodes that often occur without the usual precipitating factors of drugs or infection. The enzyme is necessary for maintaining the stability of the red cell membrane.

Management of jaundice starting in first 24 hours



If jaundice appears within 24 hours of birth it must be considered to be due to blood group incompatibility, with a high risk of cerebral palsy, until proved otherwise. Most rhesus problems should be anticipated before the neonatal period. Urgent exchange transfusion may be indicated in infants severely affected by haemolytic disease of the newborn, and it is advisable for the infant to be admitted to hospital immediately for investigation. If the mother is rhesus negative, the infant rhesus positive, and the Coombs' test positive jaundice is due to rhesus incompatibility. The plasma bilirubin concentration should be measured every five to eight hours and the results plotted on a special chart (see next page). Once the second estimation has been performed the maximum concentration can be predicted, as the rate of increase is linear. When the serial concentrations fall below the printed line the infant is unlikely to need any treatment.

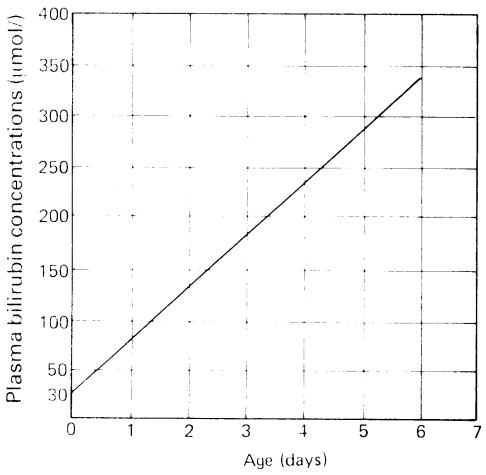
Management of jaundice starting after first 24 hours



ICTEROMETER GRADE	SERUM BILIRUBIN MEAN	mg% INDIRECT ± 2 SD
2	5·55	8·7
2½	7·57	12·11
3	10·03	14·58
3½	12·31	17·31
4	15·73	21·8
4½	19·06	26·8

The possibility of septicaemia or urinary tract infection should be considered in any ill baby who develops jaundice after the first 24 hours of life. If there is any doubt about when the jaundice first appeared the possibility of blood group incompatibility should be investigated.

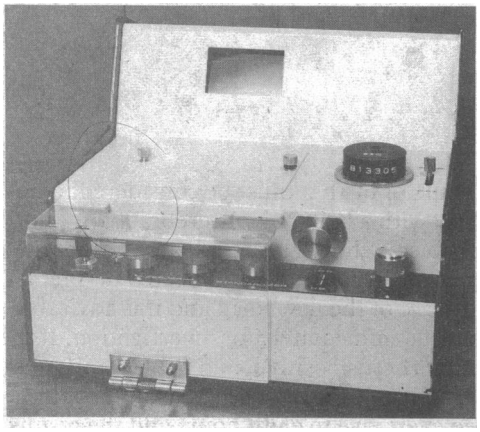
When a doctor visits the infant at home a guide to the plasma bilirubin concentration can be provided by the dermal icterometer. This is a piece of transparent perspex with yellow lines of various shades which correspond to plasma bilirubin concentrations. It should be compressed gently on the infant's nose to indicate the approximate plasma bilirubin concentrations. The dermal icterometer is not accurate in artificial light, when bilirubin values are rising rapidly, or when phototherapy has been given. In infants with pigmented skin the dermal icterometer should be compressed against the gums, not the nose.

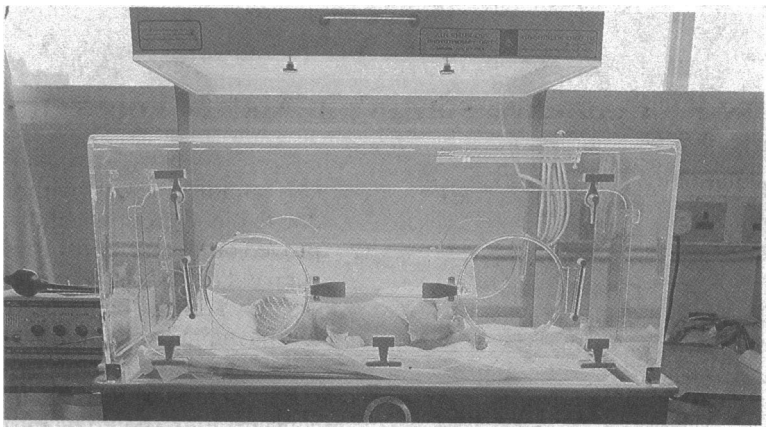


400 μmol/l = 23·4 mg/100 ml
300 μmol/l = 17·5 mg/100 ml
200 μmol/l = 11·7 mg/100 ml
100 μmol/l = 5·8 mg/100 ml
17·1 μmol/l = 1·0 mg/100 ml

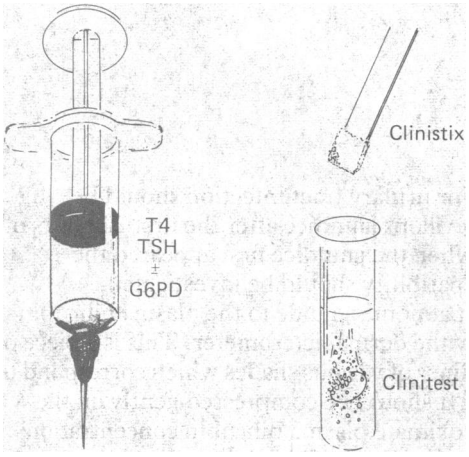
In full term infants if the dermal icterometer suggests that the plasma bilirubin concentration is about 250 μmol/l (15 mg/100 ml) or the result lies above the line on the chart the infant should be transferred to hospital immediately. If the plasma bilirubin value is very high urgent exchange transfusion or phototherapy may be needed.

In special care units there is usually a ward bilirubinometer which measures plasma bilirubin concentrations within a few minutes, using a small specimen of blood obtained by heel prick. If two estimations fall below the line on the chart treatment will probably not be needed. Those with values above the line may need exposure to light under a phototherapy unit. Phototherapy produces geometric stereoisomers of bilirubin which have no known long term deleterious effect on the infant. The infant's eyes are shielded with eye pads held in place with Micropore tape, but the mother should have the procedure explained first. Although phototherapy units have a shield to reduce transfer of heat from the lamp to the infant, monitoring of the temperature of the infant is essential. Extra fluids may be needed to compensate for the additional evaporative loss, and this can be given as plain water to breast fed infants or additional milk to bottle fed babies. Oral fluid decreases the gut transit time and improves the excretion of bilirubin and the associated compounds. The indications for phototherapy are controversial but many units give phototherapy if the plasma bilirubin value is above the line on the chart, but other units start treatment at lower values in preterm infants. Despite phototherapy an exchange transfusion may still be needed but the critical level varies with the unit and the gestational age of the infant. Exchange transfusion should be considered if the plasma bilirubin value is approaching 340 μmol/l in a full term infant and 300 μmol/l in a preterm infant.





Prolonged jaundice



If jaundice persists longer than 10 days in a full term infant blood should be taken for plasma thyroxine and thyroid stimulating hormone estimations and a specimen of urine collected to measure reducing substances by Clinitest and glucose by Clinistix. The urine should be examined in the laboratory for the presence of infection. If the parents are of Mediterranean, African, or Chinese origin the screening test for red cell glucose-6-phosphate dehydrogenase activity should also be performed.

Pale stools and a plasma conjugated bilirubin level greater than 30 μmol/l suggest the possibility of hepatitis or atresia of the bile ducts, and the advice of a paediatrician is needed within a week.

If there is a suspicion that the jaundice is related to breast feeding the other conditions causing jaundice should be excluded and the mother advised to continue breast feeding. If the plasma bilirubin concentration is rising rapidly and breast feeding is stopped for 48 hours the infant's plasma bilirubin concentration will fall abruptly and will not usually rise on return to breast feeding. Although the mother can continue lactation by expressing her milk during this diagnostic test, there is a risk that breast feeding will not be resumed.

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