Neonatal jaundice

Neonatal jaundice eHandbook

Neonatal Jaundice

- Case
- Causes
- Effects
- Assessment
- Treatment

Case 1

- Caleb is an 8 day old infant referred to ED by the Child Maternal Health Nurse because of:
  - poor feeding & lethargic
  - weight loss of 800g
  - jaundice
  - His mother is of black West African ethnicity
  - *What are your concerns about this baby?*
  - *What else do you want to know?*

- Caleb was discharged home at 24 hours of life from hospital
- He is breast fed
- His mother thought he looked jaundiced on day 2 but as he appeared to feed well, she did not worry about it
- He is lethargic and floppy, has a dry mouth, and dry nappy. He is afebrile and feels cold. His sclera and palms and soles appear jaundiced
- *What is your management?*

An iv line is sited and bloods for FBE, BC, UEC, glucose and bilirubin

- Bolus of 20ml/kg 0.9% saline
- Results:
  - Hb 11 (13-21); WCC 12.5 (5-21); plts 320 (150-400)
  - Bilirubin 700 (<100)
  - Glucose 3.5
  - Urea 9.3 (1.1-4.3), Na 146 (135-145), K 4.6 (3.5-5.0)
  - *Comments? What other history/ assessments/ investigations and management would you recommend?*

History

Very worried about this patient
Call for senior help early and move patient to Resus

- Risk factors for sepsis
  - GBS, PROM, gestation, maternal fever/ health, antibiotics
- Risk factors for jaundice
  - Maternal blood group/ fhx; birth trauma; feeding history; urine and stool colour; duration/ onset of jaundice; polycythaemia
- Hydration assessment
  - Feeding history/ intake/ urine output

Examination

- Full examination
- Hydration/ perfusion assessment
- CVS/ RS/ obs including temperature
- Neurological status
- Abdominal - ? Liver
- Skin – degree of jaundice; bruising; haematoma

Investigations

- Haemolysis?
  - FBE, film, group, DAT (same as DCT)
  - ? G6PD etc
- Hepatic causes?
  - LFTs – SBR
  - ? Infective causes
- Hydration?
  - UEC; gas
- Sepsis?
  - BC, FBC, CRP, Urine, CSF (consider when patient stable)
  - BSL/ glucose

Impression

- Severe neonatal hyperbilirubinaemia
  - ? Kernicterus and risk of seizures and coma
- Dehydration
- ? Sepsis
- ? Haemolysis

Management

- Senior help
- A,B,C
- Iv access and fluids
  - Bolus (10-20ml/kg) & maintenance fluids
- Iv antibiotics
  - Cefotaxime; ben/fluclox/amox; gent
  - ? Aciclovir
- Early discussion with Seniors/ PIPER

Treatment threshold graph for babies with neonatal jaundice

NHS
National Institute for
Health and ClinicalExcellence

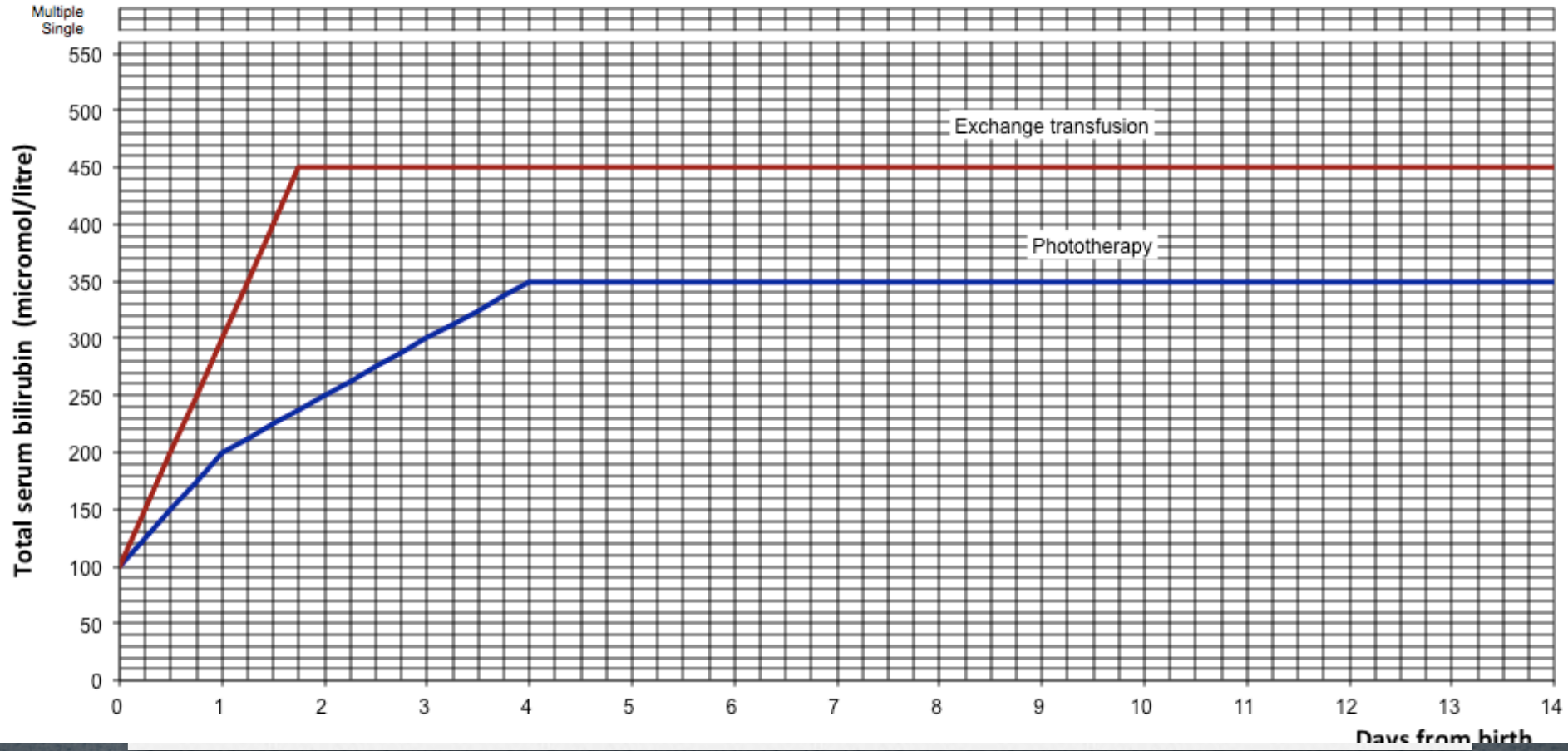
Baby's name _____ Date of birth _____

Hospital number _____ Time of birth _____ Direct Antiglobulin Test _____

Click below and choose gestation

>=38 weeks gestation

Shade for phototherapy Baby's blood group _____ Mother's blood group _____



Bilirubin Metabolism

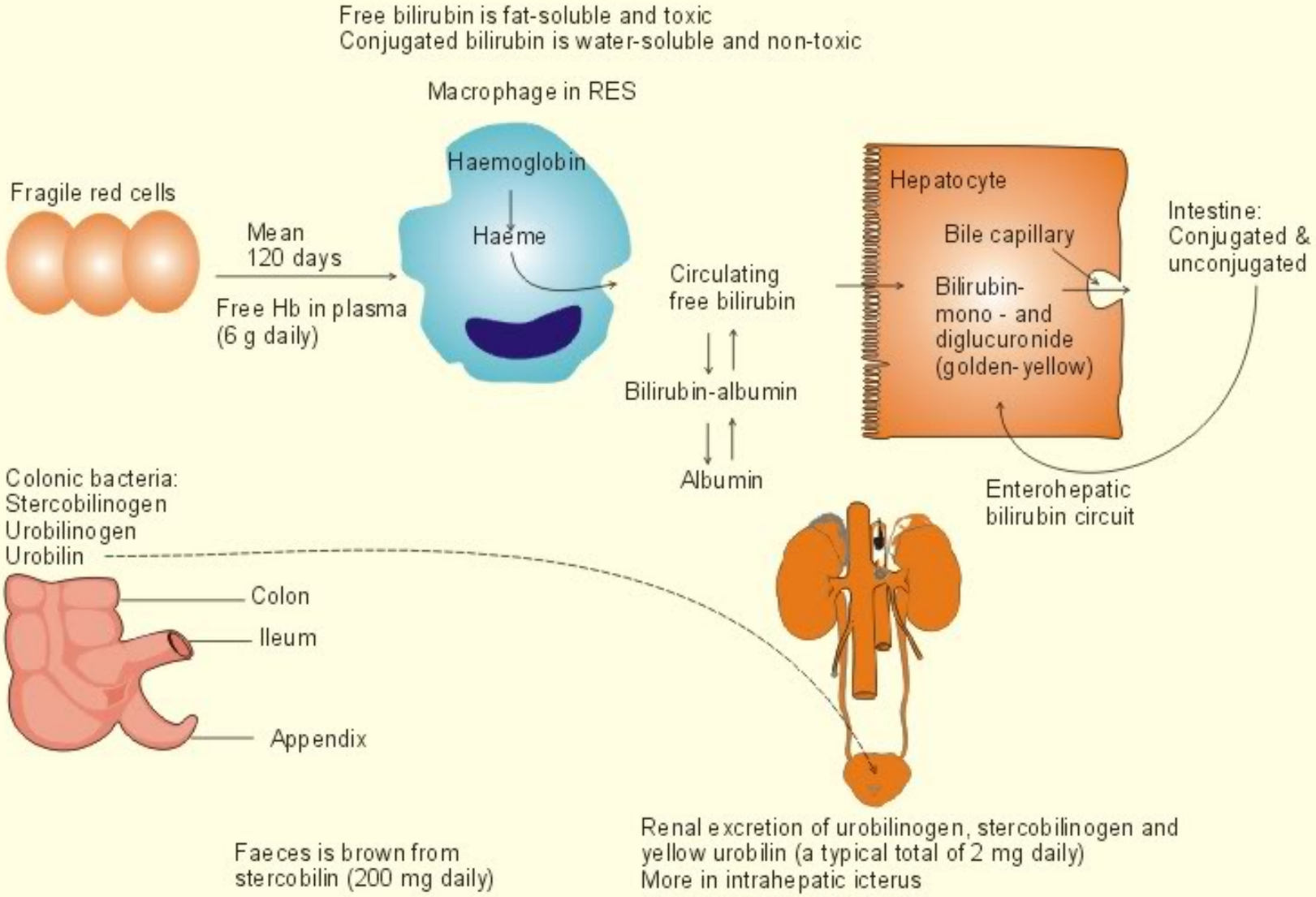


Fig. 23-1

KMc

Physiological Jaundice

- All babies develop increased SBR levels
  - 60% term and 80% preterm

  - Increased production(accelerated red cell breakdown)
  - Decreased removal (transient liver enzyme insufficiency)
  - Decreased Excretion(obstruction)
  - Increased reabsorption(entero-hepatic circulation)

Physiological jaundice is a diagnosis of exclusion

Treating jaundice

- Preterm (higher risk)?
- Is baby well or sick?
- Aetiology?
- Day 1 always pathological
  - Early(days 1-2)
  - ‘normal’(day3-10)
  - late(>14days) – also pathological
- Conjugated (indirect) and unconjugated (direct)

Care in preterm, babies with pigmented skin, unwell babies

The bilirubin range associated with each zone is:

Zone	1	2	3	4	5
SBR (micromol/L)	100	150	200	250	>250

Try and assess in natural light
Bilirubinometers can be useful if >24 hours and term

Haemolysis

- **Iso-immune** haemolysis or **blood group** incompatibility (ABO, Rh and minor bloodgoup antigens)

- Red cell **membrane** defect (eg spherocytosis)
- Red cell **enzyme defect** (eg G6PD deficiency)
  - G6PD deficiency is more common in Mediterranean, Asian and African ethnic groups. It is X linked and therefore more severe in affected males.
- Defects of haemoglobin – are and would be alpha thal (? Fatal in utero?)

- **Sepsis**
  - cause haemolysis, presumably through cell injury secondary to increased oxidative stress. Sepsis is an important cofactor in both early and prolonged jaundice and should be considered in all cases.

- **Polycythemia** or breakdown of sequestrated blood (**haematoma)**
  - Eg macrosomic GDM infants and cephalhaematoma/excessive bruising

Decreased clearance of Bilirubin

- **Inherited defects of the UDT enzyme**
  - Crigler Najjar syndrome, type 1(severe, lifelong) and type 2 (less severe)
  - Gilbert syndrome
- Commonest cause of **reduced UGT production or function.** Usually benign, but often a contributor to breast milk jaundice and G6PD related jaundice

- **Hypothyroidism**

- **Galactosaemia** (usually conjugated hyperbilirubinaemia)

The latter two conditions usually present as prolonged jaundice and are usually identified on the Newborn Screening Test. The NST result for all babies with prolonged neonatal jaundice should be checked (ring Newborn Screening laboratory at RCH)

Increased enterohepatic circulation

- **Breast milk jaundice**

- A benign condition defined as the persistence of physiological jaundice beyond the first week of life. It usually peaks within 2 weeks of life and then normalises over 3 – 12 weeks. It is a diagnosis of exclusion in an otherwise healthy, breastfed infant with prolonged jaundice

- **Breast feeding failure jaundice**

- Typically occurs in the first week of life as lactation failure leads to inadequate intake with significant weight and fluid loss. Decreased elimination and increased enterohepatic circulation also play a role. Adequate breast feeding support is crucial, particularly for first-time mothers and with early discharge.

Neonatal liver conditions

These conditions usually present with a significant conjugated fraction (>15% of the total bilirubin)

- Hepatitis (TORCH, Hep A, B, C and others)
- Anatomical abnormalities
- Complication of TPN (total parenteral nutrition)
- Billiary atresia (progressive condition with conjugated hyperbilirubinaemia and pale (acholic) stools)
- Alpha -1-antitripsin deficiency

Phototherapy

- SBR and investigate for causes
- Appropriate gestation jaundice chart
- Consultant/ PIPER involvement if at exchange level
- Recheck every 4 hours if exchange/ haemolysis expected
- Check every 12-24 hours for other cause

Works by photoisomerization and photooxidation of bilirubin, forming more soluble bilirubin products, excreted in bile and urine=irreversible. Most important is lumirubin

Bilirubin toxicity

- Free bilirubin presumed to cross into brain cells due to its lipophilic characteristics
- Worse with disruption of blood-brain barrier:

infection	sepsis
acidosis	prematurity
hyperoxia	hyperosmolarity

- Kernicterus by definition is the yellow staining of basal ganglia, pons, cerebellum at pathology

- Bilirubin encephalopathy is a broad spectrum of neurological signs attributed to raised bilirubin.

Summary

- Red flags:
  - <24 hours/ >2 weeks
  - Neurological features/ systemic features

- Assess and investigate for causes of jaundice
- Plot on appropriate gestation jaundice chart
- Early senior involvement if near/ above exchange level