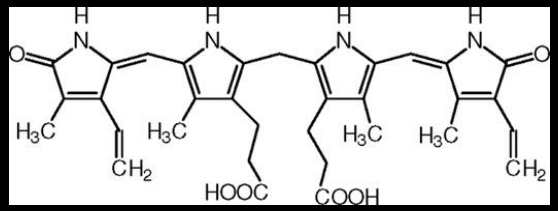

Neonatal Jaundice Hyperbilirubinemia

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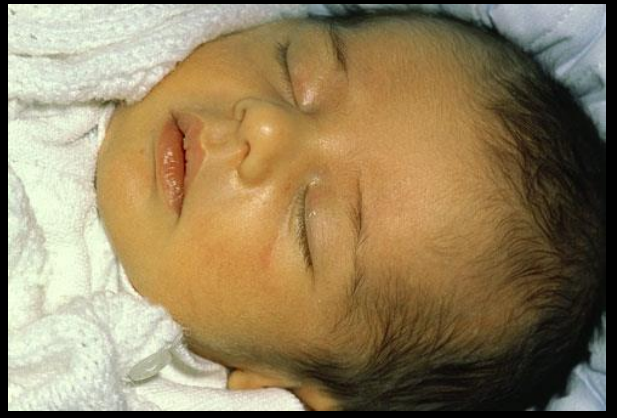
Professor of Pediatric
Head of NICU
Consultant Neonatologist

100 $ questions on Neonatal Jaundice (NJ)

Neonate : 28 days

1. What is the commonest cause of neonatal J ? physilogical
2. What is the frequently used therapy for neonatal J ? Phototherapy (Blue light)
3. Why do we need to study Neonatal J ? common 70%

Introduction



- Yellow-orange pigment Icterus –ikteros - bilirubin in the skin and sclerae
- It is one of the most common clinical pathology encountered in newborns (How common is it?)
- It may be a manifestation of another illness
- It is the cause of one catastrophic complication (kernicterus)

bilirubin precipitate in the brain which may cause cerebral palsy

picked up if u examine in natural light

never ignore it it's serious

Clinical Physiology

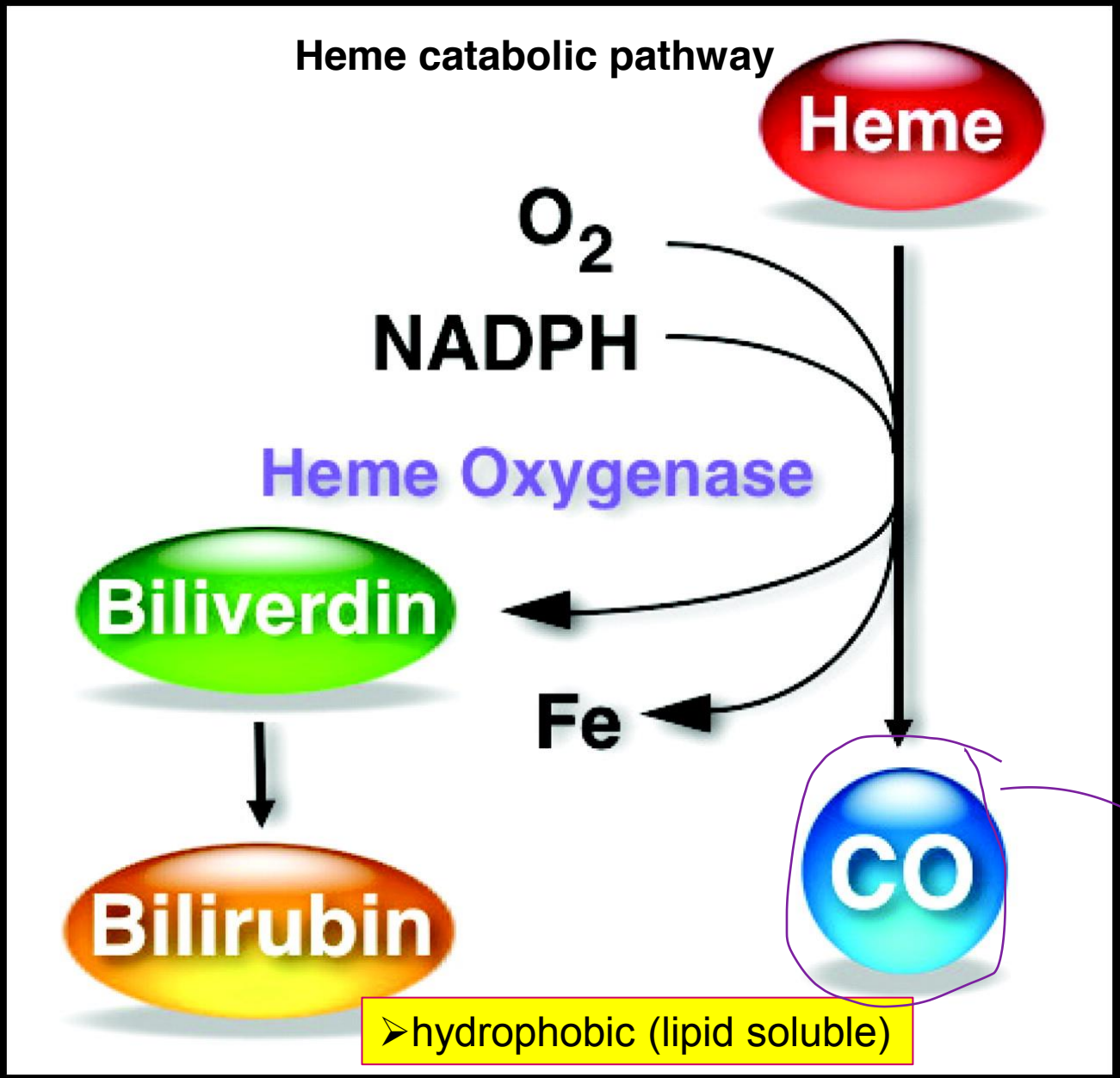
1- Bilirubin Production

➢ ***What is (are) the source(s) of Bilirubin ?***

1. Degrading heme from hemoglobin-containing RBCs (80%) the other 20% from
2. Ineffective erythropoiesis
3. Turnover of other hemoproteins (e.g., myoglobin, catalase, nitric oxide synthase, peroxidases, and cytochromes).

give iron , then by enzyme gives bilirubin

bilirubin : help in bile formation , and it's an anti-oxidant



MCQ : CO comes out of this process ,
Lab measure of CO is a good marker of hemeolysis

lipid soluble that's why it's dangerous , can cross BBB
what appear in urine is haemoglobin Not bilirubin

Wong, R. J. et al. Neoreviews 2007;8:e58-e67

2- Bilirubin Transport

albumin carry it

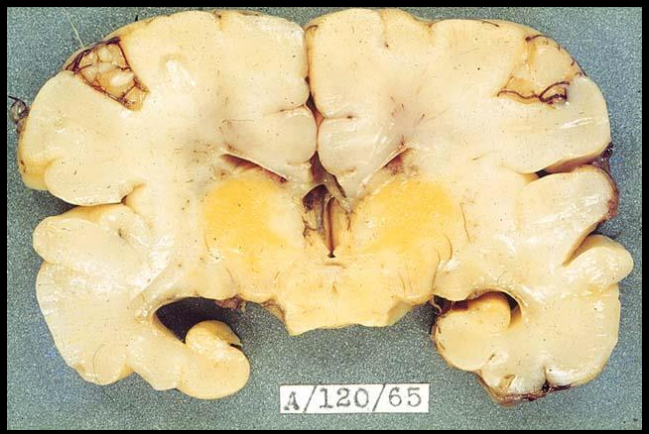
- It binds reversibly to albumin (bilirubin:albumin)
  (about 0.8 - 7 mg of bilirubin per gram of albumin)
- Low Albumin level and affinity binding sites
- Free" bilirubin is hydrophobic (lipid soluble)
- The movement of bilirubin from the circulation into tissue cross blood brain brayer

basal ganglia

hippocampus

Kernicterus

geniculate bodies



cranial nerve nuclei

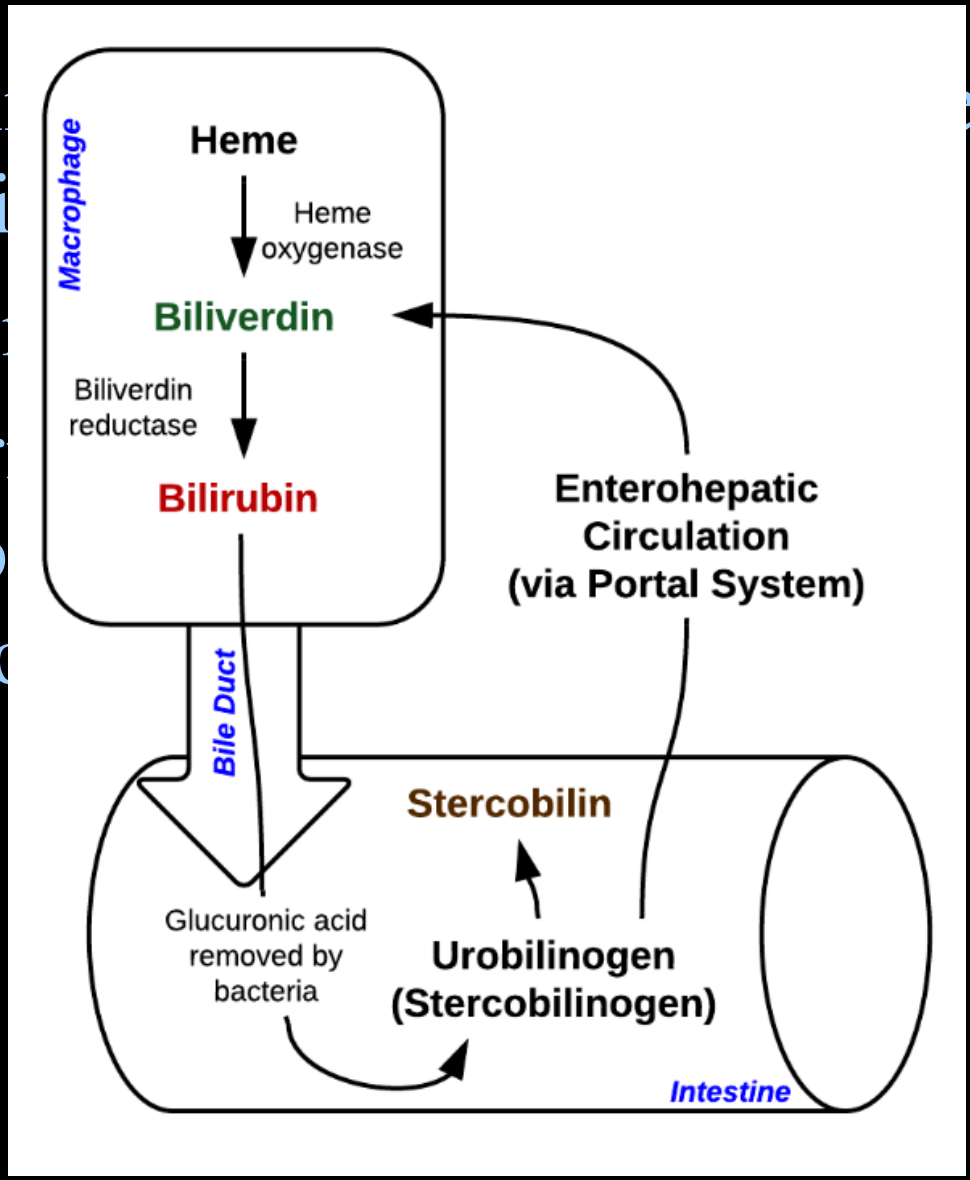
3- Conjugation

- Conjugation of bilirubin with glucuronic acid (water-soluble, non-neurotoxic bilirubin)
- ***Uridine diphospho- Glucuronosyl Transferase (UGT)*** remember it , very imp for conjugation
- Slower rate of hepatic uptake of free bilirubin from the blood
- Decreased concentrations ***of (UGT)*** ***?*** or maybe the function

crigler Najjar syndrome

4- Bilirubin Excretion

- Conjugated bilirubi
  water soluble (safe)
  excreted with the bi
- Mono or Diglucuro
- In the colon, bacter
  hydrogenate bilirub
  urobilins, and sterc



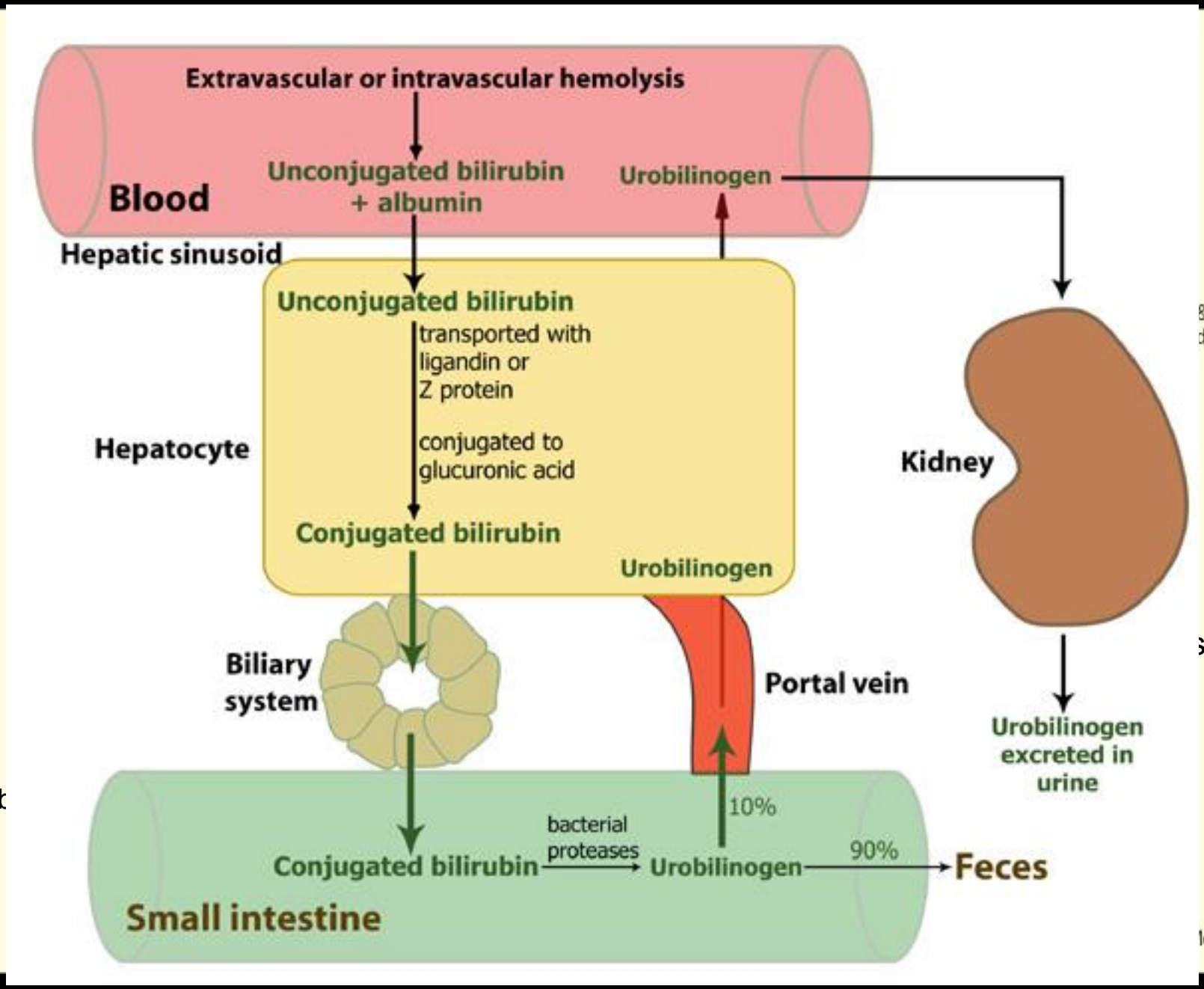

Extravascular or intravascular hemolysis
Blood
Unconjugated bilirubin
+ albumin
Urobilinogen
Hepatic sinusoid
Unconjugated bilirubin
transported with
ligandin or
Z protein
Hepatocyte
conjugated to
glucuronic acid
Conjugated bilirubin
Urobilinogen
Kidney
Biliary
system
Portal vein
Urobilinogen
excreted in
urine
10%
bacterial
proteases
Conjugated bilirubin
Urobilinogen
90%
Feces
Small intestine

Fig. 23-1

Etiological Classification

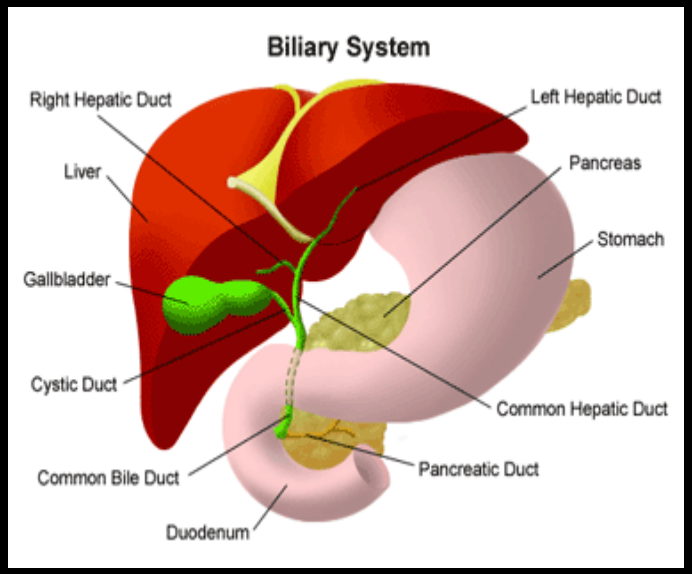
Bilirubin production

Binding

Transportation

Entero-hepatic conjugation

Hour-specific Bilirubin load



moves from hepatocyte then through the biliary system to the 2nd part of the dodnum

Bilirubin Elimination

pale stool , dark urine tea colour

What are the risk factors?

Risk Factors for Neonatal Hyperbilirubinemia

JAUNDICE

- **J**aundice visible on the 1st day of life
- **A** sibling with neonatal jaundice or anemia
- **U**nrecognized hemolysis (ABO, Rh incompatibility); **U**DP-glucoronyl transferace deficiency (Crigler-Najjar, Gilbert disease)
- **N**onoptimal feeding (formula or breast-feeding)
- **D**eficiency of G6PD
- **I**nfection, **I**nfant of diabetic mother, **I**mmaturity (prematurity)
- **C**ephalhematoma or bruising, **C**entral hematocrit >65% (polycythemia)
  - Causing extravascular hemolysis
- **E**ast Asian, Mediterranean, Native American heritage

Etiological Classification

- *Increased bilirubin load*

1. ***Haemolytic causes***

- Coombs' test positive: Examples ?
  hemolysis due to auto immune (ABO or Rh incompatibility)
- Coombs' test negative: Examples ?

(red blood cell membrane defects (spherocytosis, elliptocytosis), red blood cell enzyme defects (G6PD deficiency, pyruvate kinase deficiency)

autosomal dominant
causing severe hymolysis

x-linked recessive, affect males
rarely females if both parents or if she has Turner "X0"

- Why we do not include thalassemia or SCD ?

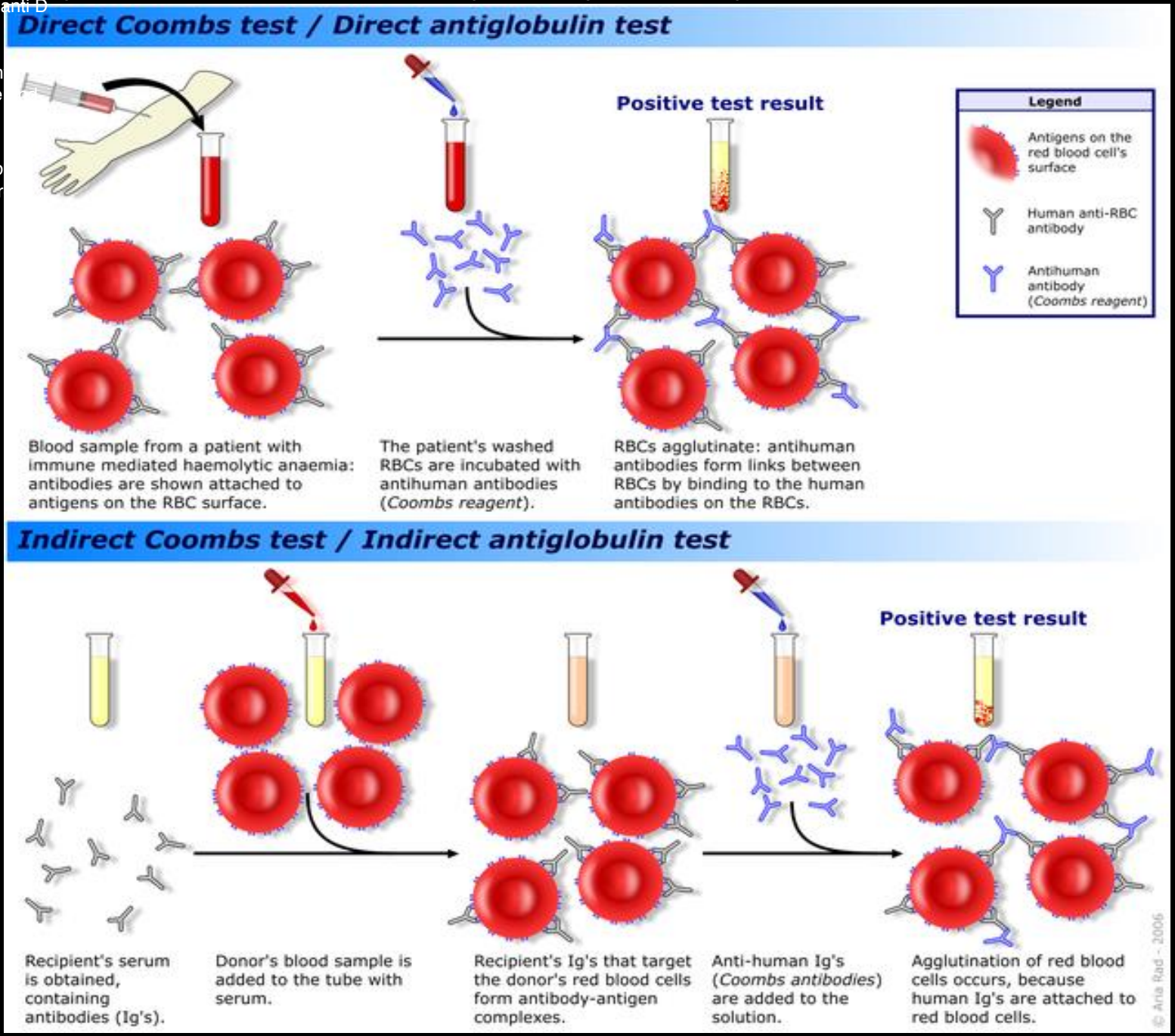
because these are Beta chain disease and the infants have only alpha and gamma , beta chain form when the baby is 4-6 months that's why manifistation of sickle cell anemia & thalassemia appears when child is 6 months

MCQ : all of the following causes neonatal hemolysis except : …… correct answer is SCD or Thalassemia

blood group incompatibility bt the mother and the baby e.g mother with group O (she has igG anti A & anti B) so if the fetus is A or B hemolysis will occur

we give Rhogam anti D to the mom after delivary to preven problems with the child

ABO incomptab lity is less sever than D (Rh)



how do we know this Hgb is from hemolysis or other causes as criggler najar syndrome ? the bilirubin will be indirect ,low hemoglobin , high CO , high reticolocyte (Triad of hemolysis)

1. Haemolytic Disease

- Jaundice in the first 24 hours of age
- Blood group incompatibility (ABO, Rhesus Less common Kell and Duffy
- Red cell enzyme deficiency
- Red blood cell membrane defect
- + ve family history
- Sepsis (... *poor intake rduce hepatic function and an increase EHC*)

if u see it after 1 or 2 hrs don't say this is physiological this is pathlogical

child come to ER yellow , be carful and make sure it's not sepsis as it can cause hemolysis or stasis in the hepatocyte and give combination of direct and indirect bilirubin

causes of indirect hyper bilirubinemia with no evidnce of hemolysis :
physiological jaundice is the most common
criggle najar syndrome (enzyme deficincy) but that's rare
Gilbert
extra vascular hemolysis
polycythemia

2. Non-hemolytic N jaundice problem with conjugation

- Increased un-conjugated bilirubin level, normal percentage of reticulocytes or Co

1. Physiologic jaundice
2. Extra vascular sources
3. Polycythemia
4. Exaggerated Entero- hepatic circulation

3- Decreased bilirubin conjugation

1. Physiologic jaundice
2. Crigler-Najjar syndrome
3. Gilbert syndrome
4. Hypothyroidism
5. Breast milk jaundice

more than 3wk

means when the baby gets enough milk , sth in breast milk causing jaundice
breast feeding jaundice means problem in the child occurs early in primy mother usually

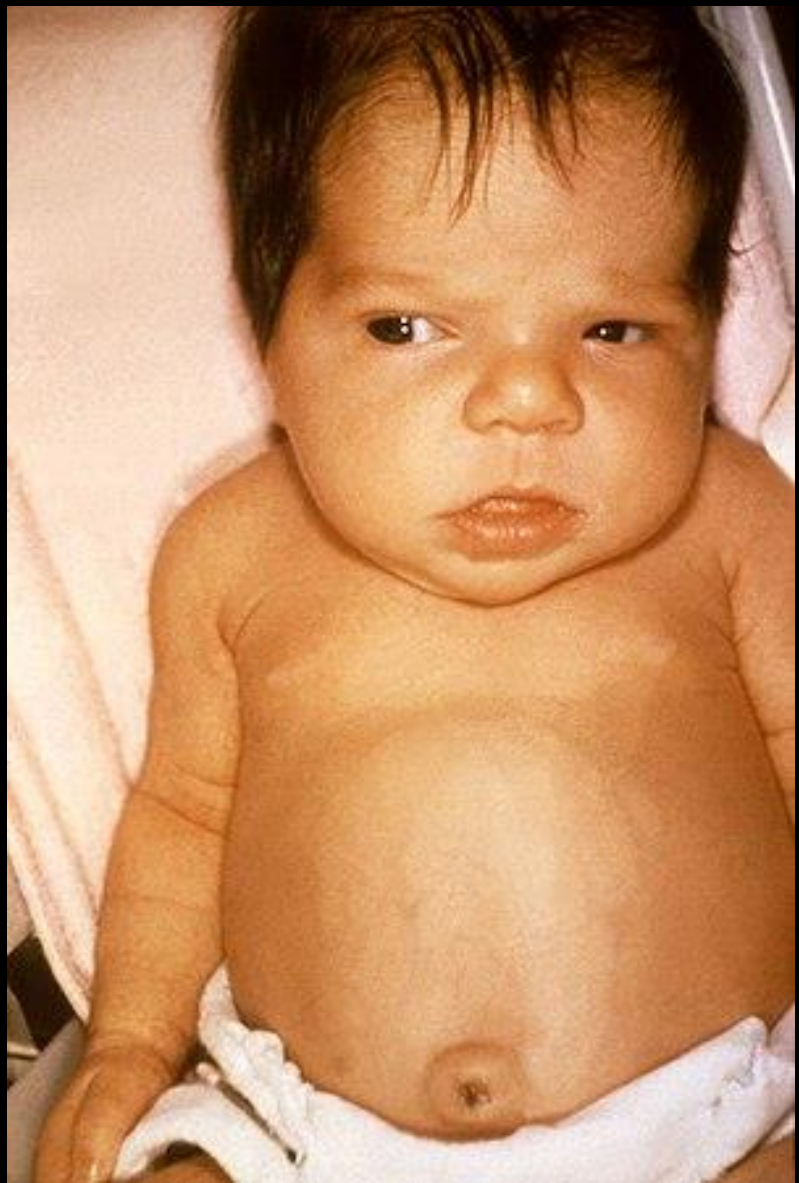
433 female MCQ

6week male infant with prolonged N.J

beyond 2 wks called neonatal jaundice

What is your diagnosis?

How do manage ?



DDx: liver disease,high direct bilirubin b.c ascites , umbilicus inverted , tongue is protruded , hypotonic

Dx:(prolonged jaundice , hypotonia , protruded tongue , umbilical hernia choppy babby) this is conjenital hypothyroidism

it causes combined conjugated and un conjugated

down syndrome may have jaundice due to polycythemia

anything affect the liver can cause early conjugated prolonged jaundice

Text

5- ***Impaired bilirubin excretion***
Direct hyperbilirubinemia

- Conjugated bilirubin level of >2 mg /dL (34 μmol/ L) or >20% of total serum bilirubin level
- Baby passing dark urine and pale stools

1. Biliary obstruction ***(need early promptly DX)***

biliary atresia child born with jaundice , high direct bilirubin (this is not physiological) don't reassure and send home you need to investigate do "Ultrasound , MRCP" and pick it up early if not the child will present after 3 wks with hepatic failure , if u Dx it early > surgery > u saved the liver

2. Infection (Hepatitis)
3. Metabolic disorder

what causes of prolonged jaundice , hypoglycemia with reducing substance-glucose like substance in the urine , Gram -ve sepsis? Galactocemia

4. Chromosomal abnormality

dubin jhonson

Case scenario

OSCE

Term male newborn, presented on the second day of life with jaundice

- What farther questions do you want to obtain?

  blood group ? fever? -looking for sepsis- , stool and feed normally, passing muconium

- Clinical signs you want to elicit?

  vital signs,power , look for lethargy -looking for features of sepsis- or green yellow if hemolysis , examin abdomen for hepatomegaly

- How do you manage such neonate ?

  total serum bilirubin , is it direct or indirect if indirect we can do phototherapy (physiological)
  see bilirubin chart to see if mild , mod ,or severe and manage accordingly

MCQ : commonest cause of nonhemolytic hyperbilirubinemia ? physiological

Infants with multiple risk factors may develop an exaggerated form of physiologic jaundice in which the total serum bilirubin level may rise as high as 17 mg per dL (291 μ mol per L)

What is the commonest cause of non hemolytic hyperbilirubineamia?

Criteria for Physiological Jaundice

child is feeding well , active , playing

1. Onset — presenting after 24 hrs (2nd day)
2. Rate of TSB increment *(5mg/dl/day)* — total serum bilirubinemia
3. Level of TSB — below 300
4. Type of Bili — should be indirect
5. Duration (Less *than 2wks in term and 3wks in preterm Neonates)*

JAUNDICE AND BREAST FEEDING

- Early-Onset Breast feeding associated Jaundice or Breast feeding failure.
- Breast milk jaundice occurs later in the newborn period usually peaking in the sixth to 14th days of life. Why?

PATHOLOGIC JAUNDICE

All etiologies of jaundice beyond

1) Physiologic
2) breastfeeding or
3) breast milk jaundice

are considered pathologic.

either secondry to sepsis . congenital anomalies

Classification of neonatal jaundice

Physiologic jaundice

- Appears after 24 hours
- Maximum intensity by 4th-5th day in term & 7th day in preterm
- TSB levels within normal centiles for age in hours based on normogram.
- Clinically not detectable after 14 days
- Disappears without any treatment.

Pathologic jaundice

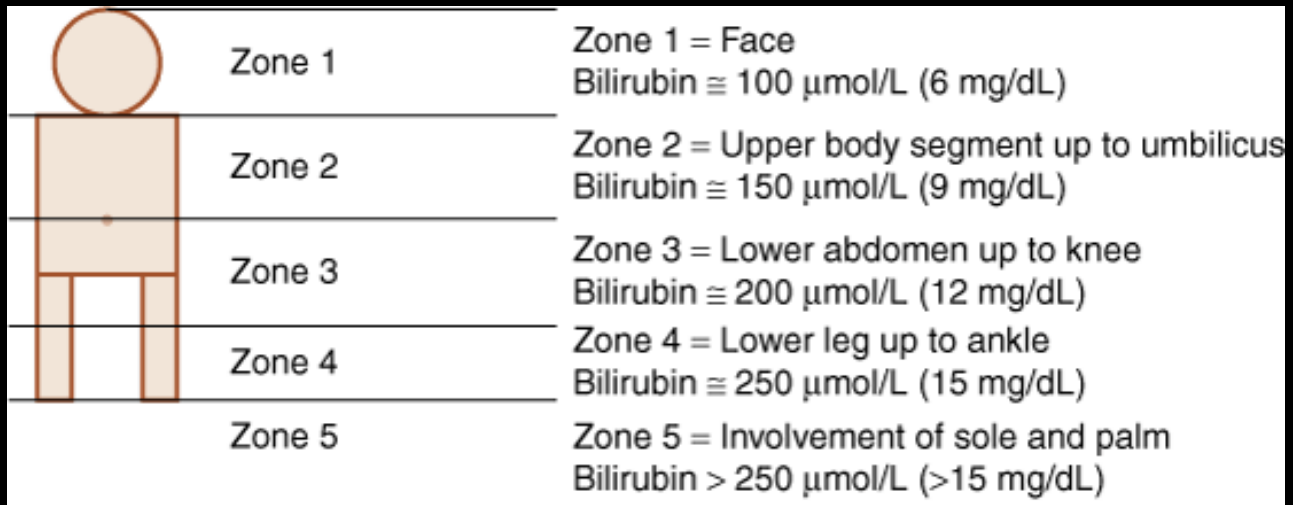
- Appears within 24 hours of age
- Increase of bilirubin > 5 mg / dl / day or at a rate of >0.2mg/dl/hr
- Serum bilirubin >95 percentile for age in hours based on normogram.
- Jaundice persisting after 14 days in fullterm babies.
- Stool clay / white colored and urine staining clothes yellow
- Direct bilirubin> 2 mg / dl or >20% of TSB.

ABO Incompatibility

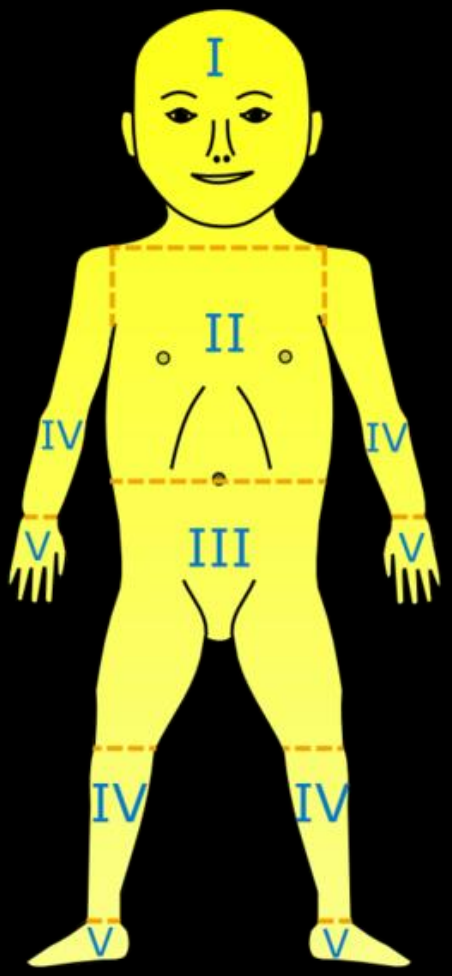
- ABO Incompatibility is the most common cause of hemolytic jaundice (10-20%)
- Most ABO antibodies are IgM (some have IgG)
- Commonly Anti A haemolysin occasionally group B
- Coombs positive ABO is more likely to cause hemolysis but less sever then Rhesus
- Hb. is usually normal or slightly reduced
- No hepato-splenomegaly

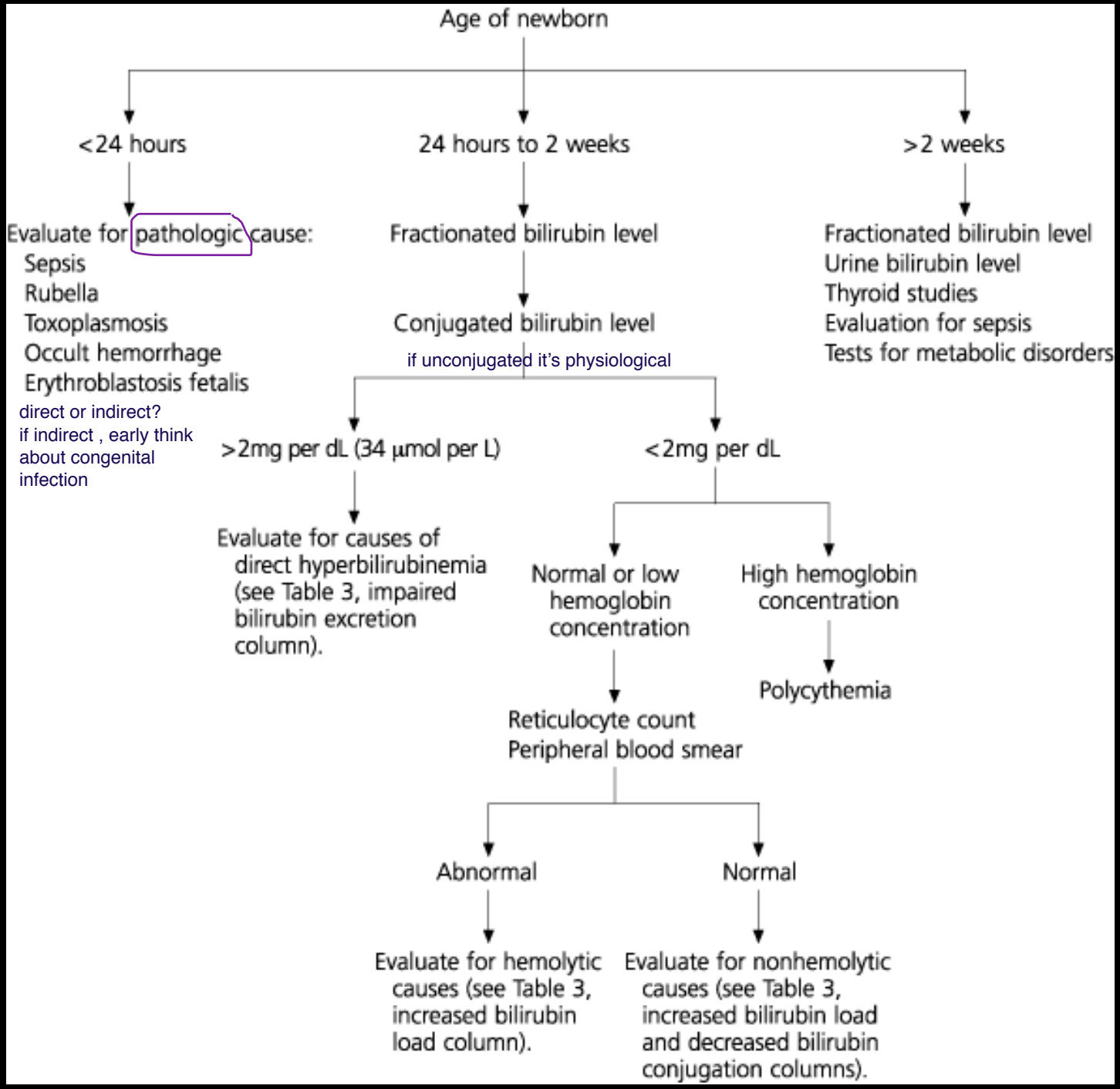
Diagnosis

- History
- Physical Examination
- Investigation



Source: Stevenson DK, Maisels MJ, Watchko JF: *Care of the Jaundiced Neonate*: www.accesspediatrics.com



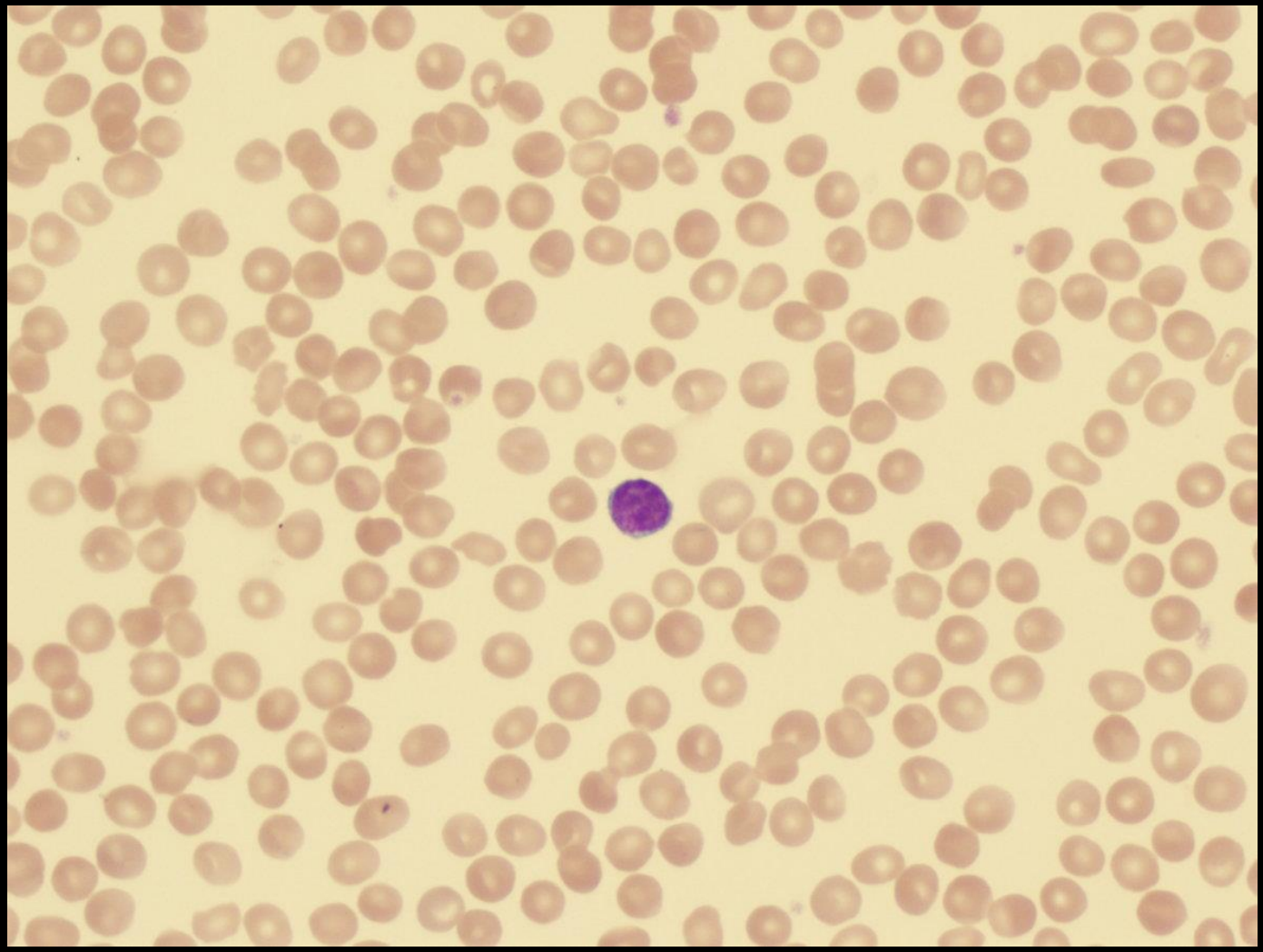

Age of newborn
<24 hours
24 hours to 2 weeks
>2 weeks
Evaluate for pathologic cause:
Sepsis
Rubella
Toxoplasmosis
Occult hemorrhage
Erythroblastosis fetalis
direct or indirect?
if indirect , early think about congenital infection
Fractionated bilirubin level
Conjugated bilirubin level
if unconjugated it's physiological
Fractionated bilirubin level
Urine bilirubin level
Thyroid studies
Evaluation for sepsis
Tests for metabolic disorders
>2mg per dL (34 μmol per L)
<2mg per dL
Evaluate for causes of direct hyperbilirubinemia (see Table 3, impaired bilirubin excretion column).
Normal or low hemoglobin concentration
High hemoglobin concentration
Polycythemia
Reticulocyte count
Peripheral blood smear
Abnormal
Normal
Evaluate for hemolytic causes (see Table 3, increased bilirubin load column).
Evaluate for nonhemolytic causes (see Table 3, increased bilirubin load and decreased bilirubin conjugation columns).

Laboratory Evaluation of Term Newborn with Jaundice

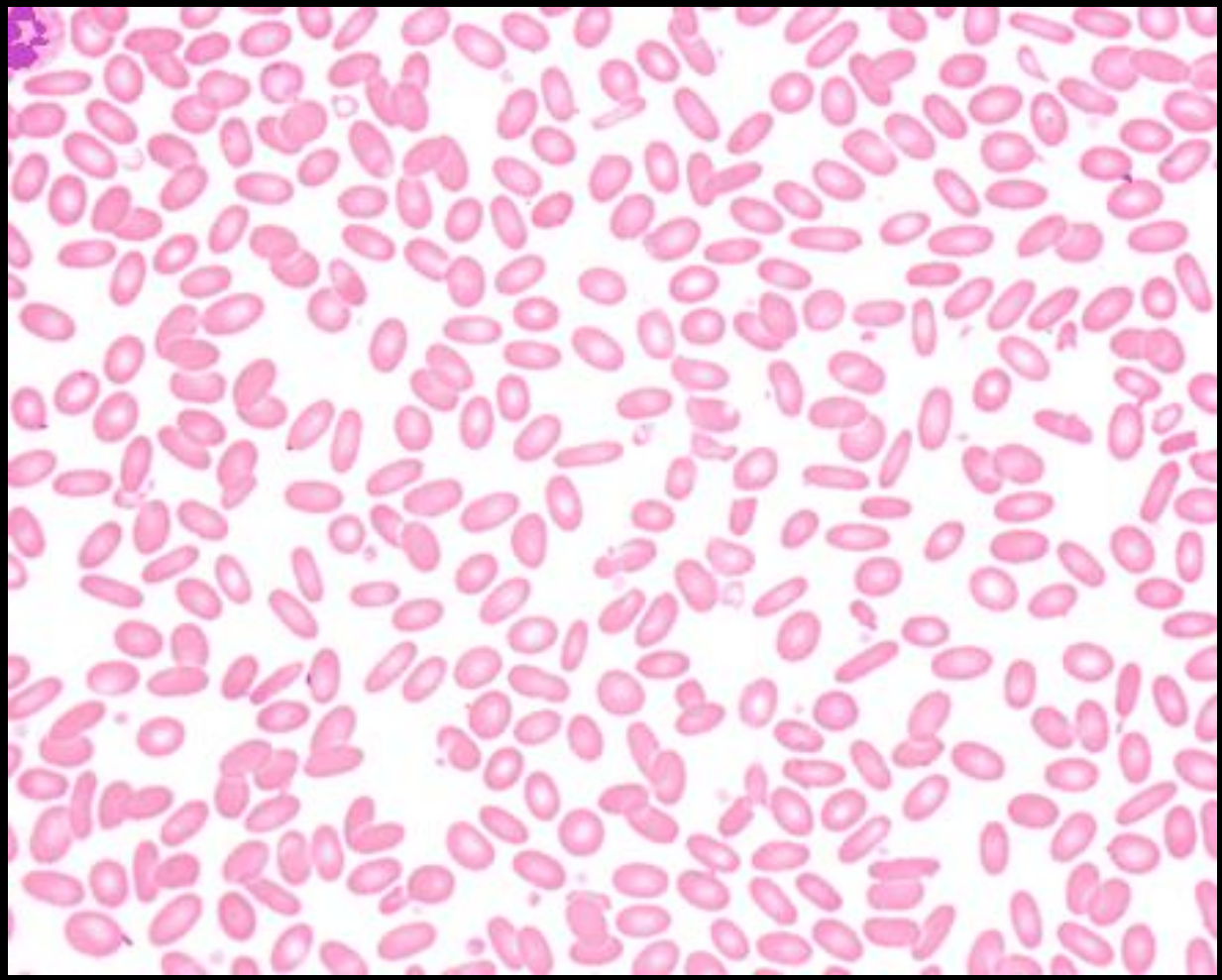
- TOTAL SERUM BILIRUBINE (TSB)
- Bilirubin fraction (conjugated OR non conj.)
- Blood group and comb's test
- CBC. Diff. Retulocytes
- G6PD level
- Peripheral blood smear
- Blood and urine culture **IF** suspected
- Thyroid function & LFT

bilirubin-metry
measure bilirubin level by transcutaneous , very specific
can be used to follow up

spherocytosis



elipticocytosis



In managing 3 days old male infant with indirect hyperbili Jaundice, all of the followings are indicated except:

A. Sun exposure — Myth
B. IV hydration
C. Phenobarbital — old literature , stimulate the liver to get more enzymes
D. Phototherapy — do bilirubin chart if baby is in photo zone ,put him in phototherapy if beyond that in the change zone so we do exchange transfusion
E. Exchange transfusion

Management

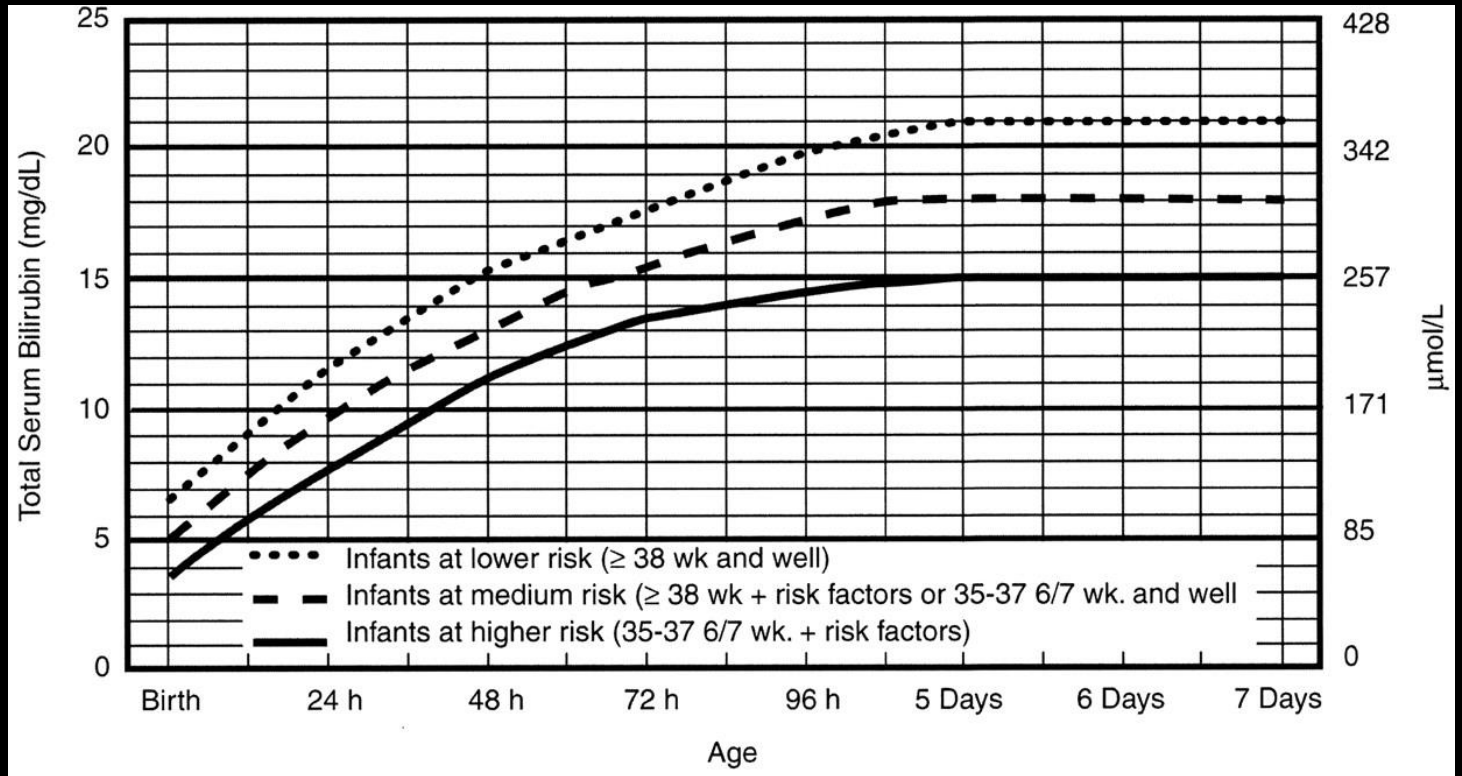
- An increased incidence of kernicterus was found to be associated with total serum bilirubin levels above 20 mg per dL in the presence of hemolysis

✓Hydration And Supportive measures

✓Management guidelines now focus primarily on phototherapy as initial treatment.

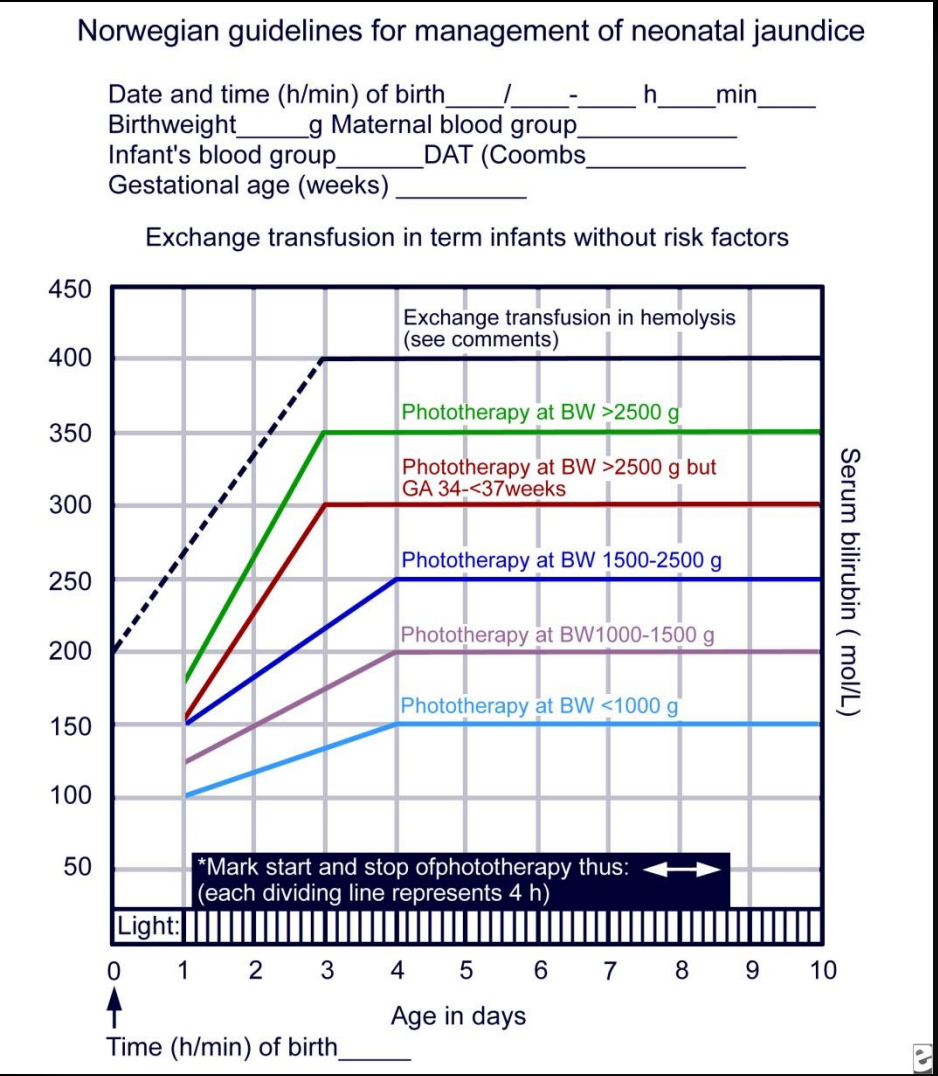
✓Aggressive guidelines recommending the use of exchange transfusion in all infants with significant hyperbilirubinemia

Guidelines for phototherapy in hospitalized infants of 35 or more weeks' gestation

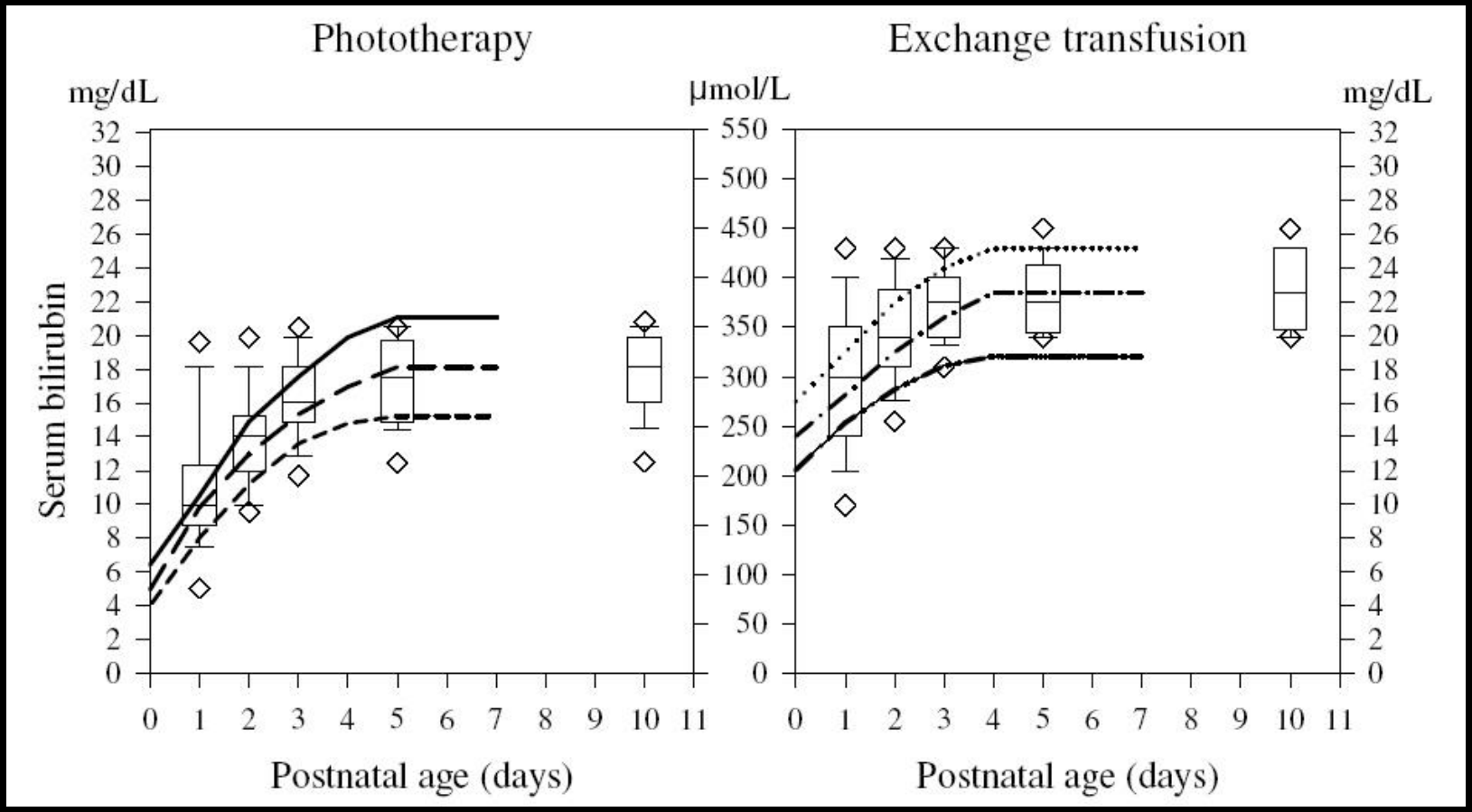


- Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin.
- Risk factors = isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis, or albumin < 3.0g/dL (if measured)
- For well infants 35-37 6/7 wk can adjust TSB levels for intervention around the medium risk line. It is an option to intervene at lower TSB levels for infants closer to 35 wks and at higher TSB levels for those closer to 37 6/7 wk.
- It is an option to provide conventional phototherapy in hospital or at home at TSB levels 2-3 mg/dL (35-50mmol/L) below those shown but home phototherapy should not be used in any infant with risk factors.

Guidelines for management



AAP recommendations



PHOTOTHERAPY

light at blue or blue-green wavelengths converts the bilirubin molecule into a form that is either easier to excrete or is less toxic to the neonate
The effective spectrum for this process has been identified *in vitro* to peak at around 450nm (blue light)

it isomrize bilirubin so it can be excreted easily ,it can also evaporate from skin

side effect : burn from the phototherapy

PHOTOTHERAPY

Conjugated hyperbilirubinemia is never physiologic, and it may indicate the presence of a potentially serious underlying disorder
HOWEVER

ELEVATED CONJUGATED BILIRUBIN LEVELS ARE NOT DIRECTLY TOXIC TO BRAIN CELLS IN THE NEONATE.

The therapeutic effect of phototherapy depends on

1. the light energy emitted in the effective range of wavelengths
2. the distance between the lights and the infant
3. the surface area of exposed skin,
4. the rate of hemolysis

During phototherapy :

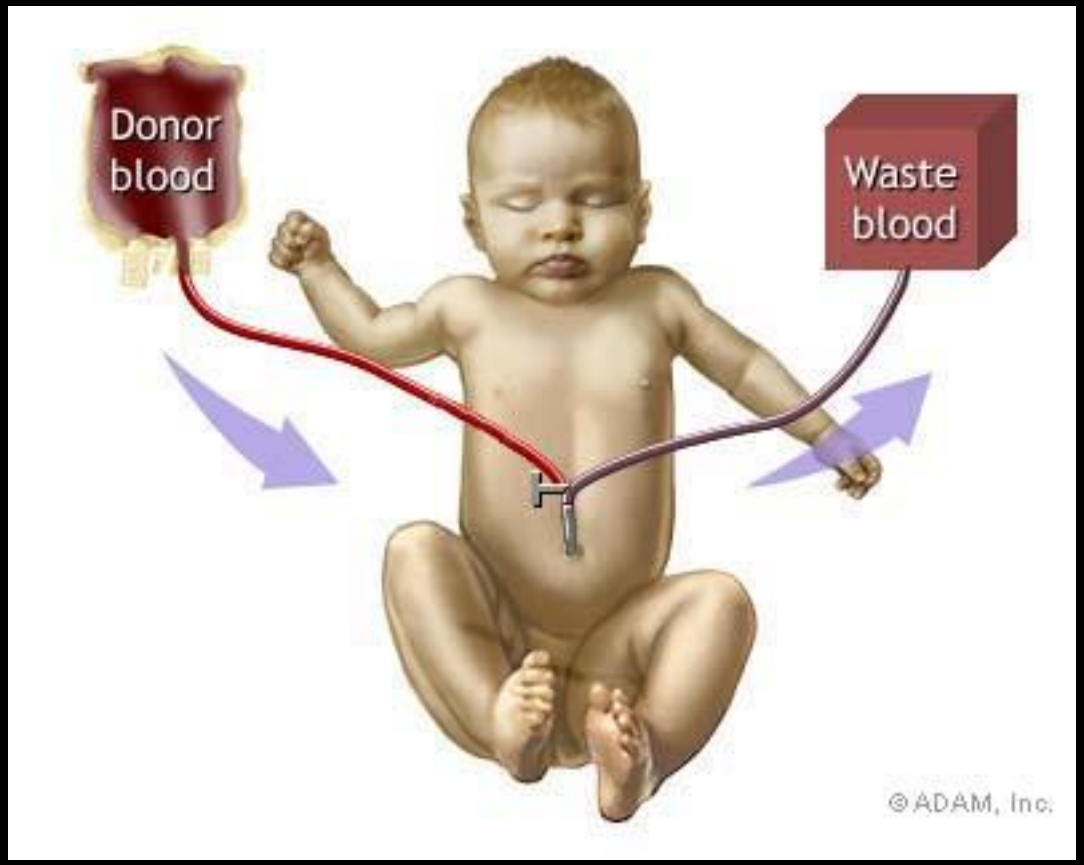
1. Cover the eyes and Genitals
2. Supplemental hydration
3. monitoring for side effects
4. Monitering of bilirubin level

Side effects of phototherapy

- **Increased insensible water loss**: Frequent Breast feeding.
- **Loose green stools**: weigh often and compensate with breast milk.
- **Skin rashes**: Harmless, no need to discontinue phototherapy.
- **Bronze baby syndrome**: occurs if baby has conjugated hyperbilirubinemia. If so, discontinue phototherapy.
- **Hypo or hyperthermia**: monitor temperature frequently.

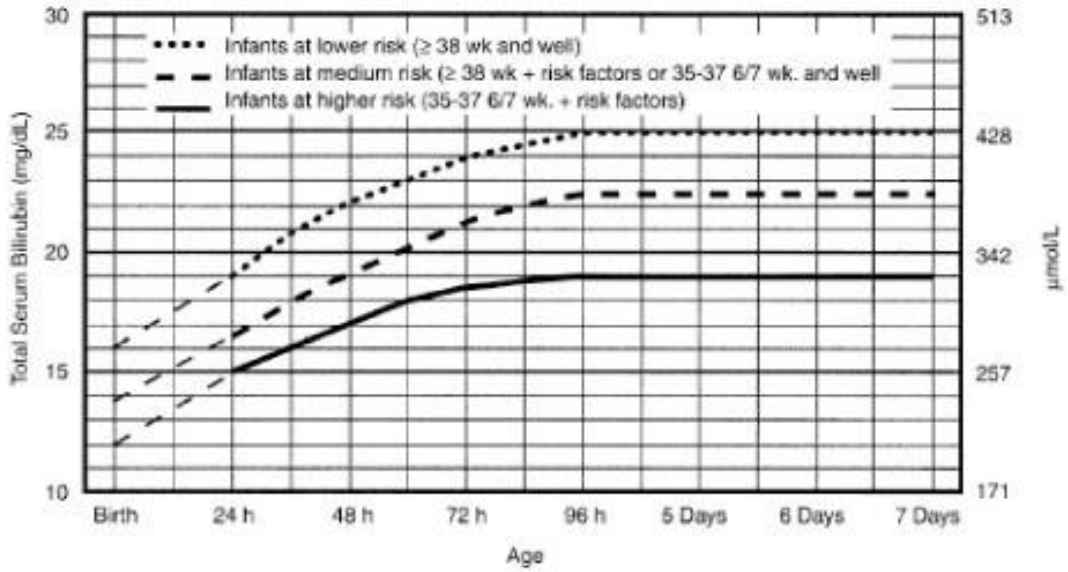
EXCHANGE TRANSFUSION

done if bilirubin goes above or bellow 350



https://youtu.be/ywFFyzjqbJQ
https://youtu.be/0aRHSgBF_Is

EXCHANGE TRANSFUSION

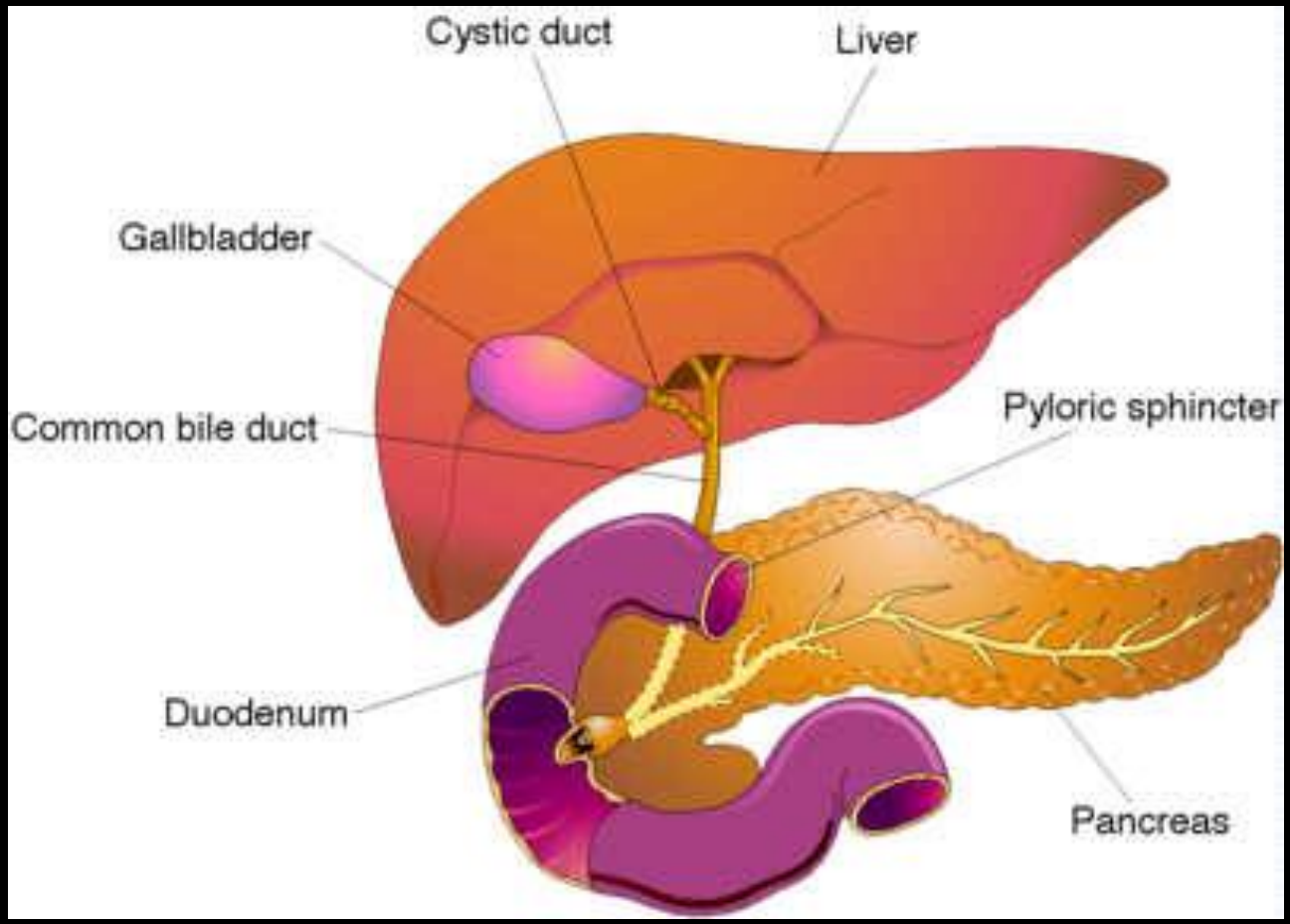


- The dashed lines for the first 24 hours indicate uncertainty due to a wide range of clinical circumstances and a range of responses to phototherapy.
- Immediate exchange transfusion is recommended if infant shows signs of acute bilirubin encephalopathy (hypertonia, arching, retrocollis, opisthotonos, fever, high pitched cry) or if TSB is ≥ 5mg/dL (85 umol/L) above these lines.
- Risk factors = isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis
- Measure serum albumin and calculate B/A ratio.
- Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin.
- If infant is well and 35-37 6/7 wk (median risk) can individualize TSB levels for exchange based on actual gestational age.

OTHER

- Immunoglobulin commonly used in ABO incompatibility and other autoimmune
- Albumin transfusion
- Antibiotics if there is suspicion of sepsis
- Fluid and Electrolytes
- D5% water sun exposure NO
- Phenobarbital ? old litriture 70s :)
- Mesoporphyrin Still under investigation

Conjugated Hyperbili



basal ganglia

hippocampus

Kernicterus

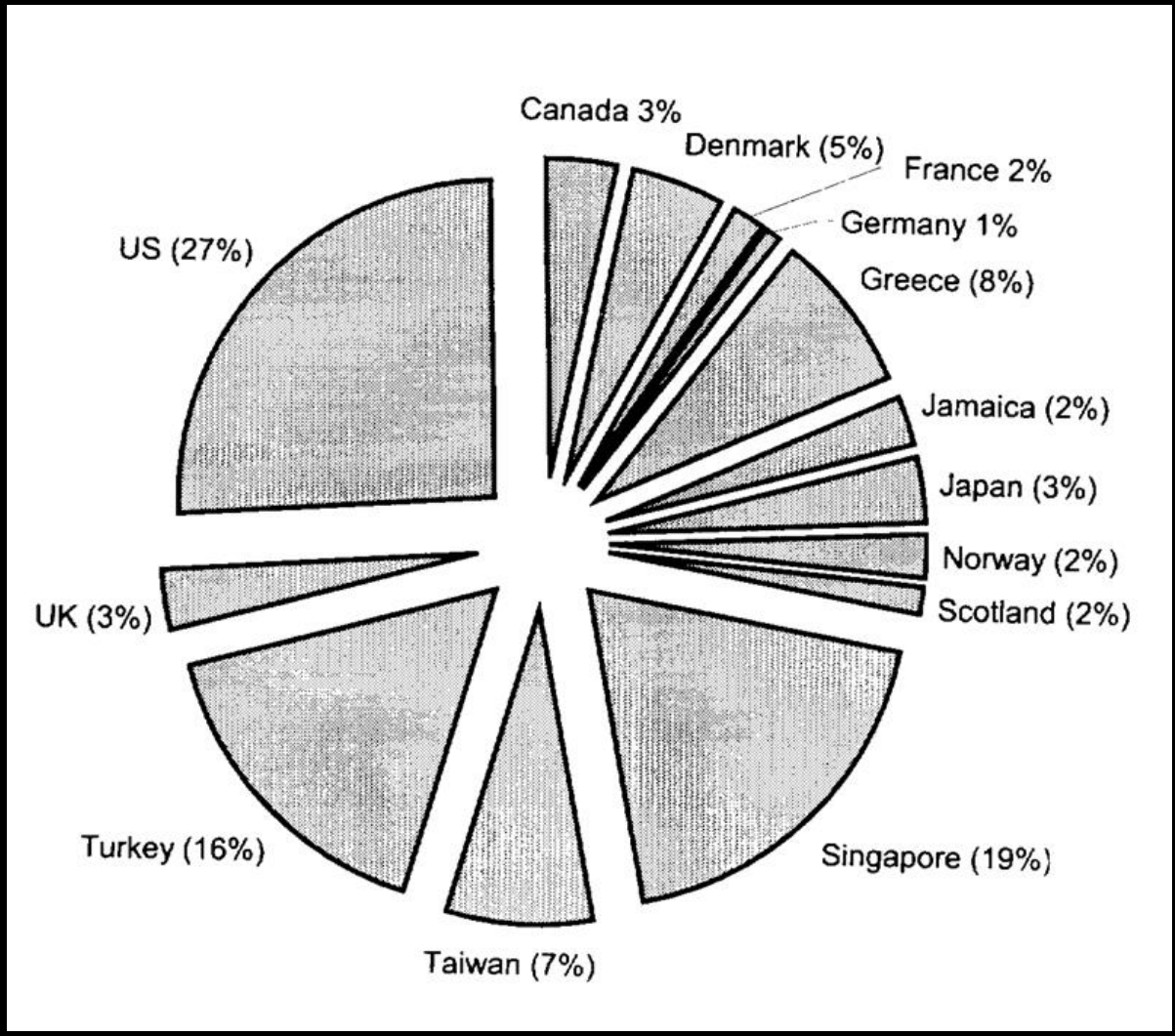
geniculate bodies

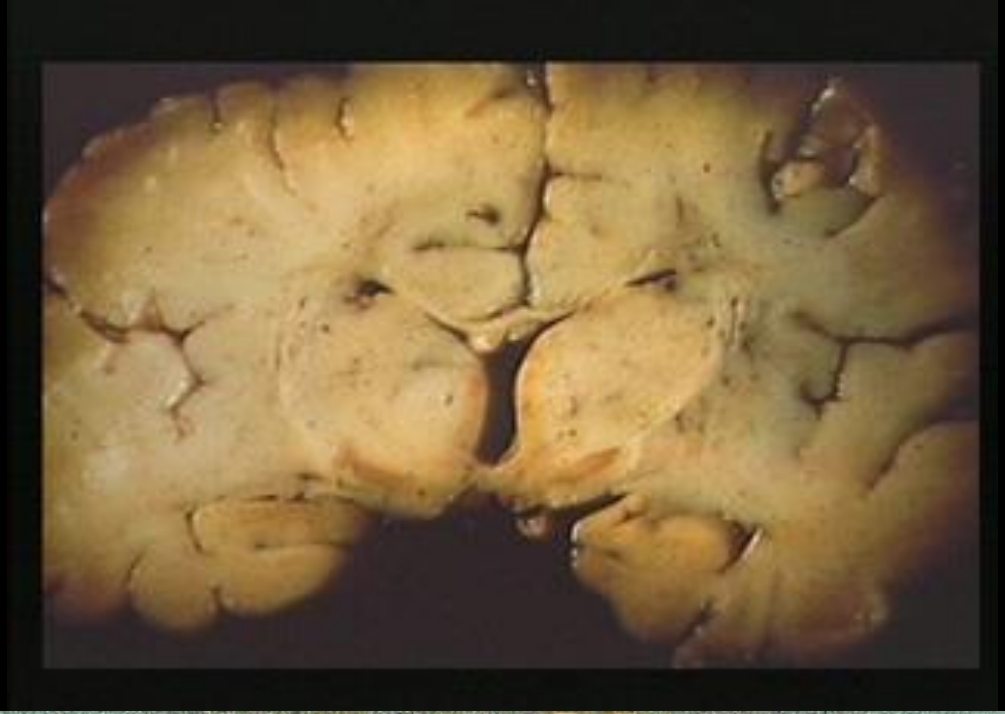
cranial nerve nuclei

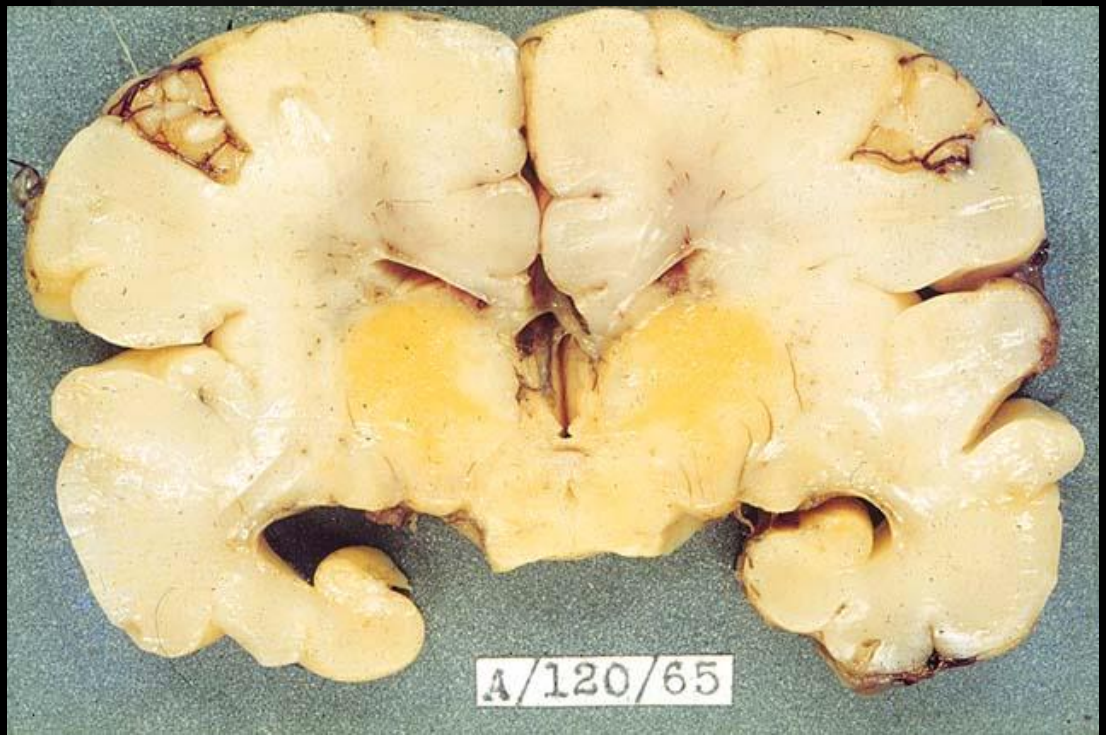
Pathophysiology

- Bilirubin staining in the regions of the basal ganglia, hippocampus, substantia nigra, and brainstem nuclei
- Staining can occur in the absence of severe hyperbilirubinemia
- Characteristic patterns of neuronal necrosis

KERNICTERUS






A/120/65

Causes

- Severe hemolytic processes were identified 25% most of the cases in saudi arabia is criggler najjar
- Glucose-6-phosphate dehydrogenase (G6PD) deficiency was diagnosed in 22%
- galactosemia occurred in 2.5%
- Crigler-Najjar syndrome type I occurred in
- NO etiology for the severe hyperbilirubinemia was discovered in 73% of cases

Incidence

- Incidence of bilirubin levels >30mg/dl (1/10,000)
- Do we have any registry in Saudi Arabia??
- All reported cases from Saudi literatures were secondary to Crigler Najjarr syndrome

Am J MedGenet. 1998 Aug 27;79(1):12-5

Term Infant with Jaundice

- High pitched cry
- Arching of the baby's body into a bow
- Weakness, limpness, floppiness
- Difficulty nursing and/or sucking

- WHAT IS THE TREATMENT ?

KERNICTERUS

- Early symptoms-acute bilirubin encephalopathy
  - ✓ poor feeding
  - ✓ abnormal cry
  - ✓ hypotonia,
- Intermediate phase
  - ✓ -stupor, irritability, hypertonia
- Late
  - ✓ shrill cry, no feeding, opisthotonus, apnea, seizures, coma, death cerebral palsy

Clinical Spectrum: Adverse Effects of Newborn Jaundice

Acute Bilirubin Encephalopathy

affect cranial nerves most commonly hearing

Death: respiratory failure

from hypertonia

Chronic Post-icteric Sequelae (Kernicterus)

Auditory Neuropathy (isolated)

Subtle manifestations (extra-pyramidal and central posturing disorders) *suspected but not yet proven*

Bilirubin Induced Neurologic Dysfunction (BIND)

KERNICTERUS

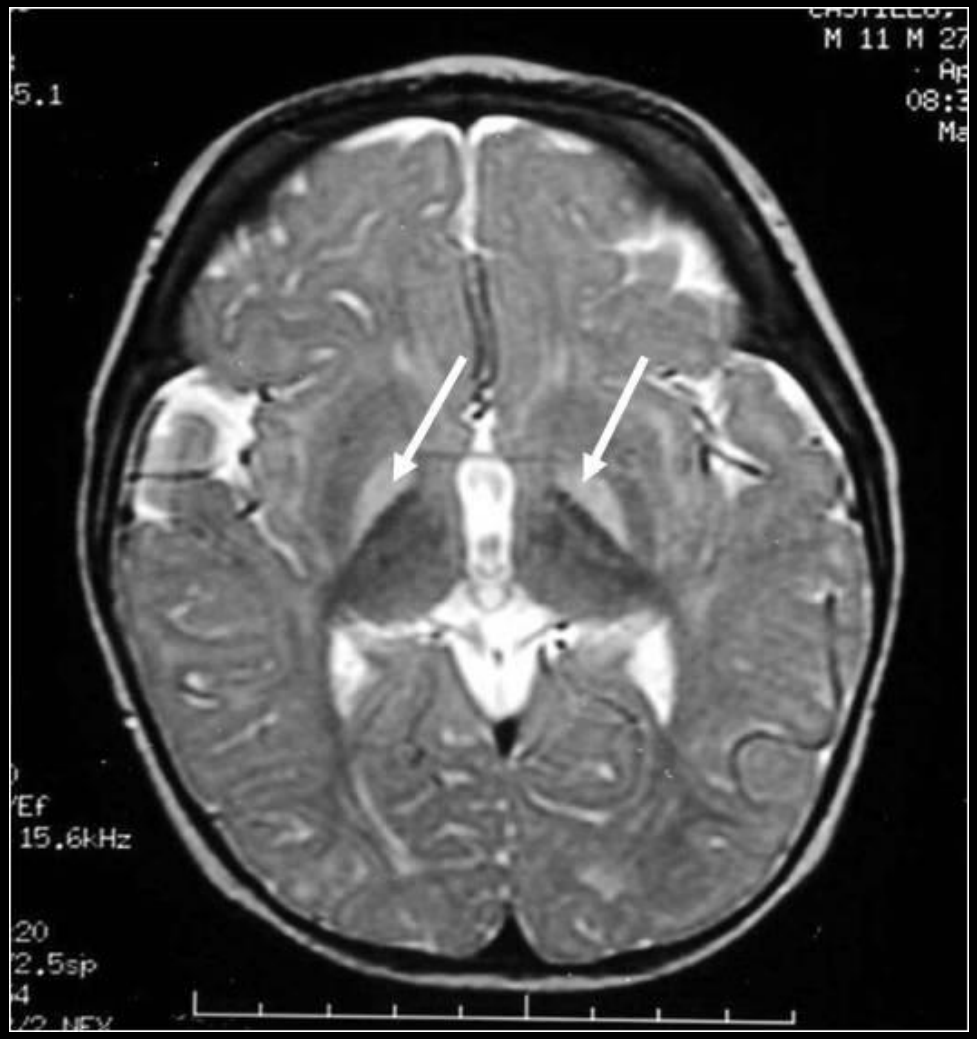
- Late sequelae can include
  - ✓ gaze abnormalities
  - ✓ feeding difficulties
  - ✓ dystonia
  - ✓ incoordination
  - ✓ choreoathetosis
  - ✓ sensorineural hearing loss
  - ✓ painful muscle spasms

What is bilirubin level ? that cause kernictures

- Over 120 cases kernicterus documented since 1990
- Majority term, breastfed
- Majority of those had levels in high 30s to 40s.
- Lowest level recorded in case series of 111 from 1991-2002 was 20.7 (The mean was 38.)
- Many cases had no planned follow up and had been discharged early (<48 hours).

Risk Factors

- Asphyxia
- Acidosis
- Sepsis
- Hypoalbuminemia
- Young gestational age
- Low birth wt
- Hyperthermia
- Respiratory distress



Magnetic resonance imaging of the head. Hyperintense basal ganglia lesions on T2-weighted images

Prevention

- Recommend:
  - Promote and support successful breastfeeding.
  - Universal systematic pre-discharge assessment.
  - Provide targeted follow-up based on the risk.
  - Track outcome for timely treatment to prevent excessive hyperbilirubinemia and possibly, kernicterus.

AAP 2004: Recommendations

I. Primary Prevention: lactation support
II. Risk assessment for severe hyperbilirubinemia:
III. Interpretation of TSB values
IV. Cause of jaundice/hyperbilirubinemia.
V. Pre-discharge risk assessment
VI. Hospital policies and procedures
VII. Treatment

Summary

- Bilirubin physiology
- Prevent neurotoxicity no sun exposure no water with sugar :)
- Identify and treat illness associated with
- Excess production
- Impaired conjugation or inadequate elimination
- Combination of therapy

MCQs

A 3-day old full term infant with hemolytic disease of the newborn due to Rh incompatibility has a serum indirect bilirubin concentration of 33 mg/dL. You perform an exchange transfusion with no further elevations of bilirubin above 19 mg/dL. Among the following, the **MOST** appropriate study to use to follow up on this infant is:

A. Another Coomb's test
B. Brainstem auditory evoked response b.c acutely it affect the cranial nerves
C. Computed tomography of the head
D. Hemoglobin electrophoresis
E. Indirect retinoscopy

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7-day old breastfeed infant born at term has had decreased appetite, irritability and vomiting for 24 hours. On Physical examination, the infant appears listless. Respiratory Rate: 40/min, Heart Rate : 160/min, and blood pressure: 68/38 mm Hg. The skin and sclera are icteric but no other abnormalities noted. Laboratory studies reveal: Hemoglobin: 12 gm/dL. Urinalysis is negative for reducing substances. Of the following, the **MOST** likely diagnosis is:

A. Bacterial sepsis
B. Blood group incompatibility
C. Breast milk jaundice
D. Hypothyroidism
E. Intrauterine infection

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A 3-day old , breast fed infant develops jaundice. The serum bilirubin level is 12 mg/dL with a direct bilirubin component of 0.5 mg/dL. The infant's mother asks whether the jaundice might be associated with breastfeeding. Which of the following statements regarding hyperbilirubinaemia associated with breast feeding is **TRUE:**

A. Indirect hyperbilirubinaemia associated with breast feeding may occur as early as the first day of life.
B. Water supplementation in breast-fed infants will significantly reduce serum concentrations of indirect bilirubin
C. Hyperbilirubinemia associated with breast feeding may persist for 8 to 12 weeks
D. Decreased clearance of bilirubin may play a role in breast feeding jaundice, breast milk jaundice.

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Of the following conditions, which is the **MOST** consistent with findings of mild cholestasis without evidence of biliary atresia?

A. Lead intoxication
B. Chronic hemolytic disease
C. Alpha – antitrypsin deficiency
D. Breast milk jaundice
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A 4-week old, breast-fed boy has had mild jaundice since birth. Weight gain has been poor. The urine is light yellow-brown, and the stools are pale yellow-green in color. At this point, the **MOST** appropriate next step in management is to: pale stool & dark urine meaans direct bilirubiin , biliary obstruction maybe

A. Observe the child clinically for 2 to 4 weeks
B. Stop breastfeeding and re-examine the child in 7 to 10 days
C. Obtain a cholecystogram
D. Obtain a total and direct serum bilirubin levels and studies of liver function

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You are presenting to 5th year medical student on Neonatal jaundice . Which statement is True ?

A. Is normally excreted in the urine following its conjugation to glucuronic acid

B. Achieve high blood levels due to haemolysis associated with glucose-6-phosphate dehydrogenase deficiency

C. Must be prevented from reaching 340 umol/L in well term babies by use of exchange transfusion if necessary

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-Neonatal jaundice is associated with all of the following except :

A. prematurity

B. cystic fibrosis

C. Gilbert's syndrome

D. breast milk feeding

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A term baby is found to have serum bilirubin of 250 umol/l at 18 hours of age. Which of the following is true?

A. Physiological jaundice is the most likely cause
B. An urgent conjugated bilirubin level is indicated
C. It is unlikely to be due to haemolysis
D. The infants blood group and Coombs test are the most important investigations
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In an infant who appeared healthy at birth, vomiting and diarrhea developed at 1 week of age. She gained weight poorly despite a change from breast milk to infant formula feeding at 2 weeks of age. At 3 weeks of age, she is brought to the emergency department where she is found to be lethargic and to have hepatomegaly. *Of the following, the most likely diagnosis is*

A) Inspissated bile syndrome
B) Crigler-Najjar Syndrome
C) Galactosemia
D) Gilbert Syndrome
E) Dubin-Johnson Syndrome

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6 week old infant presented with early signs of kernicterus. His blood work showed high indirect non hemolytic hyperbilirubinemia. The MOST likely diagnosis :

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An apparently term infant who was born at home was noted to be very yellow on the fifth postnatal day. he has no symptoms or clinical signs of bilirubin encephalopathy. His bilirubin concentration is 36.5 mg/dL (624.2 mcmol/L), with a direct bilirubin measurement of 1.5 mg/dL (26.7 mcmol/L). You draw blood to investigate the cause of the hyperbilirubinemia and place the infant under intense phototherapy. Of the following, the MOST appropriate treatment plan is:

A. administration of a bolus of 20 mL/kg normal saline,
B. administration of intravenous fluids with 10% glucose at rate of 150 mL/kg per day
C. administration of salt-poor albumin (1g/kg) over the next hour,
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