26th Annual General Pediatric Review & Self-Assessment

Nicklaus Children's Hospital

# DELIVERY ROOM & WELL-BABY


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# Topics

- Prenatal and Perinatal care
- Maternal conditions affecting newborn
- Delivery Room management
- Routine care of newborn
- Newborn Screening
- Physical exam variants
- Birth trauma
- Hypoglycemia
- Jaundice

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# Antenatal Testing

| | |
|---|---|
| Goal | Identify fetus that will benefit from early intervention and thereby prevent fetal death or neurologic injury |
| Physiologic basis | Premise that fetus responds to hypoxemia with detectable biophysical changes |
| Efficacy | Observational studies lower rate of death when fetal testing performed |
| Timing | As soon as an increased risk of fetal demise is identified and delivery for perinatal benefit would be consider if test results are abnormal. Most pregnancies ~32 wks |
| Interpretation | Abnormal- additional testing<br>Maternal condition identified- prompt Tx |

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## Prenatal Screening

| Routine Maternal Labs |
|---|
| • Blood type, Rh and antibody screen |
| • HBSAg |
| • RPR |
| • Rubella antibodies |
| • HIV testing |
| • GBS screening (35-37 wks) |
| • Rapid glucose screen/GTT |

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## Prenatal Screening

| | |
|---|---|
| Maternal Screening | Birth defects detection uses maternal serum markers<br>**1st Trimester Screening** (Risk for T21 and 18)<br>• Nuchal translucency<br>• β-hCG (Free β-human chorionic gonadotropin)<br>• PAPP-A (pregnancy-associated plasma protein A)<br>**2nd Trimester -Quadruple screen** (T 18, 21, open NTD)<br>• Serum αAFP<br>• Total hCG<br>• Inhibin-A<br>• Unconjugated estriol |
| Genetic diagnostic test | **Cell-free DNA (cfDNA) screening** –fetal cells DNA in maternal blood are analyzed for genetic conditions (Trisomy 13,18, 21) and sex chromosome abnormalities |

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## Prenatal Ultrasound Studies

- Determination of pregnancy viability
- Gestational age calculation
- Evaluation for nuchal translucency
- Detection of malformations (18-22 weeks)
- Diagnosis of multiples
- Determine fetal growth and fetal weight estimate
- Visual guidance- for procedures ( amniocentesis, CVS, PUBS)
- Amniotic Fluid Volume (MVP –Maximum vertical pocket)
  - **Oligohydramnios**: MVP < 2cm-reduced urine production, placental insufficiency, prolonged ROM
  - **Polyhydramnios**: MVP ≥ 8 cm- diabetes, multiple gestation, open NTD, cardiac disease, fetal bowel obstruction,
- Doppler Velocimetry of fetal and funic vessels (uteroplacental flow)

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## Assessment of Fetal Well-Being

| | |
|---|---|
| Fundal Height | Measurement from the top of the uterine fundus to the symphysis pubis |
| Non stress Testing (NST) | FHR is monitored to observe accelerations with fetal movements<br>• **reactive:** ≥2 FHR acceleration/20 min<br>• **nonreactive:** < 2 acceleration/20 min |
| Contraction Stress Test (CST) | Denotes FHR response to uterine contractions (nipple stimulation or oxytocin)<br>Used to evaluate for uteroplacental insufficiency |
| Biophysical profile | Done in 3rd trimester in high risk pregnancies to evaluate fetal well being (U/S and FHR monitoring) |

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## Biophysical Profile (BPP)

| Categories (each scoring 2 or 0) |
|---|
| • NST (non stress test)- FHR |
| • Fetal movements/activity ( 30 min) |
| • Fetal breathing (30 min) |
| • Fetal muscle tone (30 min) |
| • Amniotic fluid volume |
| Score: 8-10 well fetus |
| <6 possible fetal compromise |

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## Electronic Fetal Heart Rate Monitoring (EFM)

| | Definition | Causes |
|---|---|---|
| Fetal tachycardia | FHR >160 bpm, severe >180 bpm | Maternal or fetal infections, hypoxia |
| Fetal bradycardia | FHR <110 bpm | Fetal compromise, CHB |
| Variability | Rapid fluctuations in baseline FHR (moderate, decreased, absent) | Most sensitive indicator Fetal well-being |
| Early decelerations | Occur at same time as contraction, benign | Due to fetal head compression during contraction |
| Variable decelerations | No uniformity in shape, pattern or timing | Due to compression of umbilical cord |
| Late decelerations | Onset, nadir and recovery 10-30 sec after contraction | Due to uteroplacental insufficiency ->fetal hypoxia |

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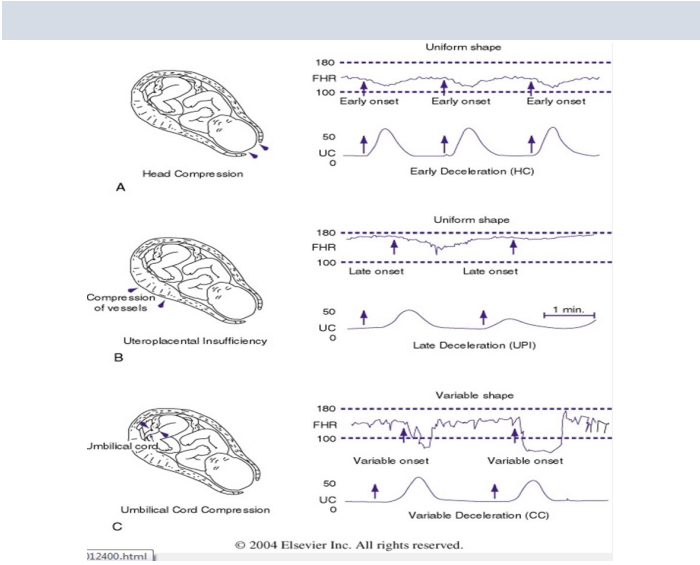
# Fetal Decelerations

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# Antenatal Therapies

| Therapy | Comments |
|---|---|
| RhoGAM | Administered at 28 wks and after delivery to Rh negative women as immunoprophylaxis |
| Antenatal Steroids (ANS) | Standard of care for women at 24 0/7-33 6/7 weeks gestation at risk of preterm delivery within 7 days<br>• Single course recommended<br>• Given to accelerate fetal lung maturation<br>• Associated with improved survival and outcome in premature infants<br>• Decreased incidence of RDS, IVH, NEC, CLD, neonatal mortality |
| GBS prophylaxis | • Active prevention by culturing all mothers 35-37 wks and offering<br>• IAP (intrapartum Antibiotic prophylaxis) per GBS guidelines<br>• Penicillin IV(drug of choice), ampicillin or cefazolin administered at least 4 hrs before delivery |

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## Maternal Conditions Affecting Newborn

| Condition | Effect in Newborn |
|---|---|
| Chorioamnionitis, PROM | Sepsis |
| SLE | Congenital heart block |
| Myasthenia Gravis | Hypotonia, respiratory failure |
| Hyperthyroidism | Hyperthyroidism or hypothyroidism from maternal therapy |
| Withdrawal of maternal hormones | Vaginal discharge (whitish to bloody) |
| Autoimmune thrombocytopenia | Neonatal thrombocytopenia |

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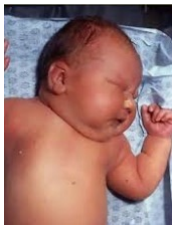
## Infant of Diabetic Mother (IDM)



| Condition | Comment |
|---|---|
| Intrauterine Death | Most common in type I maternal DM |
| Congenital Malformations | CHD (TGV, TA, Tricuspid Atresia, HLHS)<br>Small left colon<br>Caudal regression syndrome<br>NTD |
| Hypoglycemia | Maternal/fetal hyperglycemia-> Fetal hyperinsulinism |
| Macrosomia | Fetal hyperinsulinism promotes growth |
| Birth Trauma | Macrosomia |
| Other Associations | Hyperbilirubinemia, hypocalcemia, hypomagnesemia, polycythemia<br>RDS , hypertrophic cardiomyopathy, renal vein thrombosis |

***Mothers with DM should be offered 2nd trimester U/S**

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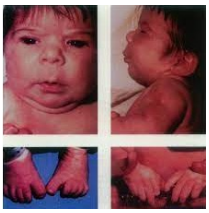
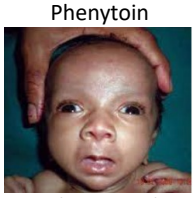
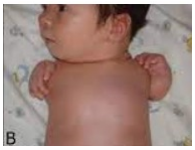
# Maternal Drug Use Affecting Newborn



Phenytoin

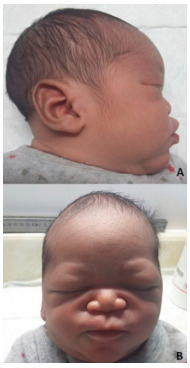


Valproic acid



Thalidomide

| Drug | Effect on Fetus |
|---|---|
| Lithium | Ebstein's anomaly (1-5% risk)<br>Excreted in milk- Not compatible with breast milk feedings |
| Phenytoin | Hypertelorism, epicanthal folds, cleft lip/palate<br>Broad low nasal bridge, short upturned nose<br>Nail hypoplasia, CHD |
| Valproic acid | Midface hypoplasia, NTD, cardiac, limb, renal anomalies |
| Thalidomide | Severe limb reduction |
| Warfarin (Coumadin) | Nail hypoplasia, depressed nasal bridge, CHD, stippled epiphyses |
| Isotretinoin | CHD, limb reductions, microtia or anotia |



Warfarin

Isotretinoin

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# Fetal Alcohol Syndrome (FAS)

(Courtesy of M Rimsza)

- Symmetric growth failure
- Microcephaly
- Short palpebrae
- Flat nasal bridge
- Long smooth philtrum
- Thin upper lip
- Maxillary hypoplasia
- Radioulnar synostosis
- Nail hypoplasia
- CHD (VSD)
- Behavioral problems
- Developmental delay, MR

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## Maternal Substance Abuse

| Substance | Findings |
|---|---|
| Nicotine | Increased incidence of miscarriage, abruption, stillbirth, Prematurity, LBW, IUGR, risk for facial clefting<br>Increased risk SIDS, childhood wheezing |
| Cocaine | Placental infarction/abruption<br>Prematurity, IUGR<br>Cerebral infarction (rare)<br>Gastroschisis<br>poss. GU/limb anomalies |
| Marijuana | Not associated with birth defects |
| Opiates | Low birth weight, FTT, NAS |
| Amphetamines | Fetal growth restriction, agitation, irritability, hypersensitivity to stimuli, potential developmental/cognitive delays |

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## Neonatal Abstinence Syndrome (NAS)

| | |
|---|---|
| Onset | Heroin < 2days<br>Methadone 2-7 days, up to 2 wks<br>Cocaine- does not cause withdrawal |
| Diagnosis | urine or meconium toxicology |
| NAS scores | tremors, irritability, poor feeding, vomiting, loose stools, sweating, fever, sneezing, tachypnea |
| Treatment (opiate withdrawal) | **NAS <7 comfort measures (swaddling)**<br>**NAS >8 morphine sulfate, methadone** |

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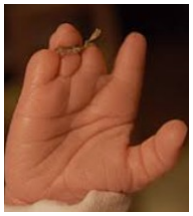
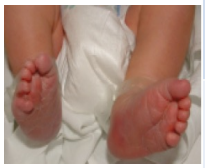
## Defect of Morphogenesis

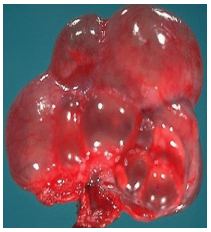


Disruption

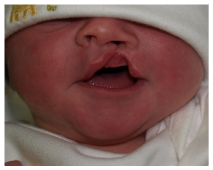


Deformation

| Type | Cause | Example |
|---|---|---|
| Disruption | Destruction of a tissue that initially developed normally | Amniotic bands, ring-like constriction of limbs, amputation of digits |
| Dysplasia | Abnormal cellular organization or function in a specific tissue or organ | Multicystic renal dysplasia, hemangiomas |
| Deformation | Extrinsic intrauterine constraint or deformity due to neuromuscular or skeletal abnormality | Positional talipes equinovarus, torticollis |
| Malformation | Incomplete or abnormal progression of one or more developmental processes in early gestation | Cleft lip, palate, myelocele, CHD |



Dysplasia



Malformation

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## Delivery Room

| Prior to delivery |
|---|
| Review history |
| Communication with Ob |
| Communication with RN/RT |
| Equipment check |

| Questions to ask? |
|---|
| Gestational age? |
| Amniotic fluid clear? |
| Additional risk factors? |
| Umbilical cord management plans? |

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# Delayed Cord Clamping

| |
|---|
| Endorsed by ACOG and AAP |
| • Cord clamping is delayed for at least 30-60 seconds after birth FT/PT<br>• Immediate clamping- maternal hemorrhage or hemodynamic instability, immediate need for infant resuscitation |
| FT: Increases Hb<br>Increases iron stores for several months<br>May favorably affect infant development<br>PT: Decreases need for blood transfusions<br>Lower incidence of NEC and IVH |
| May lead to increased jaundice |

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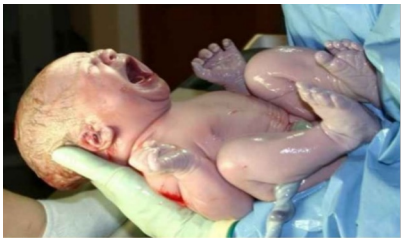
# Delivery Room

Assessment at birth

- Term gestation?
- Good muscle tone?
- Breathing or crying?

Yes
Routine care
Place infant directly on mother's chest if desired
Cover with dry linen
Observation

No
↕
Resuscitate



- ~10% of newborns require some assistance to begin breathing
- Less than 1% require extensive resuscitation measures

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## Delivery Room Management

| Resuscitation Steps |
| --- |
| • Warm (move to radiant warmer/maintain normal temp) |
| • Dry |
| • Stimulate |
| • Position head and neck |
| • Suction (only if copious or obstructing airway) |
| • Establish patent airway |
| • Ventilate and Oxygenate (establish adequate ventilation) |
| • Initiate chest compression (establish adequate circulation) |
| • Administer epinephrine and/or volume |

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## Delivery Room Resuscitation

"Golden Minute": approximately 60 seconds are allotted for completing the initial steps, reevaluation and beginning ventilation if required

Decision to progress beyond initial steps are based on:

| | |
| --- | --- |
| Respirations | apnea, gasping or labored breathing |
| **HR** | less than 100/min- **most sensitive indicator** |
| Oxygenation | oximetry reading- once PPV is instituted |

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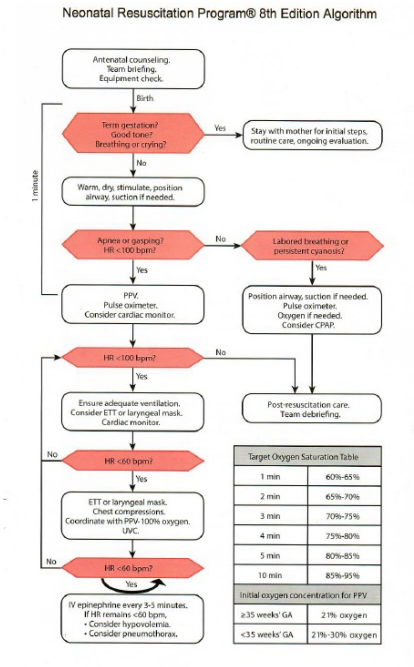
# NRP: 8th edition Update




Neonatal Resuscitation. American Academy of Pediatrics.

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# NRP

| | |
|---|---|
| High risk delivery | Source of blended air/oxygen, pulse oximeter and cardiac monitor should be immediately available in event of an unexpected resuscitation |
| FiO2 (initial) | • ≥ 35 wks RA<br>• <35 wks FiO2 between 21-30% |
| O2 Saturations | Should be measured continuously using preductal saturation probe and titrated to meet target guidelines |

| Target Pre-ductal SpO2 after birth | |
|---|---|
| 1 min | 60-65% |
| 2 min | 65-70% |
| 3 min | 70-75% |
| 4 min | 75-80% |
| 5 min | 80-85% |
| 10 min | 85-95% |

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## Intermittent Positive Pressure Ventilation (IPPV)

| | |
|---|---|
| Indications | HR <100 bpm or ineffective respirations after initial steps |
| Settings | Initial PIP 20-25 cm H20, rate 40-60 bpm<br>PT-PEEP is recommended (starting PEEP 5 cm H20) using T-Piece resuscitator (Neopuff) |
| After 15 sec r**eassess**-<br>- no response | Use MR SOPA corrective measures |
| -no response to MR SOPA | an alternate airway (ETT or laryngeal mask) should be inserted |

*When alternate airway becomes necessary a cardiac monitor is recommended

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## Neonatal Resuscitation Program (NRP)

| MR SOPA |
|---|
| **M** ask adjustment |
| **R** eposition head |
| **S** uction mouth/nose |
| **O** pen mouth |
| **P** ressure (↑to no more than 40 cm H2O) |
| **A** lternative airway (ETT, Laryngeal mask) |

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## Chest Compressions

| | |
|---|---|
| Indication | HR < 60 bpm, despite effective PPV at a 3:1 ratio<br>FiO2 should be increased to 100% |
| Methods | Thumb Technique (preferred technique)<br>2-Finger Technique |
| Placement | Lower third of the sternum |
| Depth of compression | 1/3 the A-P diameter of the chest |

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## Medications

| | |
|---|---|
| Epinephrine | Concentration: 1:10,000<br>Dose:<br>• IV/OI: 0.02 mg/kg (equals 0.2mL/kg)<br>• ETT: 0.1 mg/kg (equals 1 ml/kg)<br>Route: UVC is the preferred route<br>Enhances cardiac contractility, increases rate and effectiveness of cardiac contraction, constricts peripheral circulation |
| Volume Expander | N/S, RL or O Rh neg PRBC<br>Dose: 10 ml/kg via UVC |

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## Golden Hour for Term Infant

| | |
|---|---|
| Counseling/team briefing | If risk factors |
| Delayed cord clamping | 30-60 sec |
| Preventing hypothermia | Radiant warmer, warm blankets, |
| Respiratory support | Pulse oximeter, start FiO2 at 21%, follow target Sats Support as needed |
| Initiation of breast feeding | Well newborns as soon as possible, skin to skin |
| Preventing hypoglycemia | Monitor BS at risk infant (IDM, LGA, SGA) |
| Therapeutic hypothermia for asphyxia | Turn warmer off, monitor Temp, start hypothermia within 6 hrs |

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## Golden Hour for Preterm Infant

| | |
|---|---|
| Counseling/team briefing | Plan management of expected complications |
| Delayed cord clamping | 30-60 sec |
| Preventing hypothermia | Radiant warmer, warm blankets, cap, polyethylene wrap, thermal mattress, heated incubator for transport |
| Respiratory support | Pulse oximeter, targeted O2 Sat, CPAP, Invasive ventilation, surfactant (support as needed) |
| Cardiac support | Monitor VS, B/P, maintain normal perfusion and B/P |
| Prevention of neurologic injury | Gentle handling, head midline, avoid high PIP, PEEP, |
| Early initiation of nutrition | TPN and enteral nutrition priority |
| Preventing Hypoglycemia | Measure glucose within 1 hr, glucose infusion as soon as possible |
| Infection prevention | If suspicion of sepsis, B/C and antibiotics within the 1st hour |
| Laboratory test and X-rays | To be done during golden hour |
| Communication with parents | Parents should be informed of condition and POC |

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## Apgar Score

| | 0 | 1 | 2 |
|---|---|---|---|
| Heart Rate | Absent | <100 bpm | >100 bpm |
| Respirations | Absent | Slow, irregular | Good, crying |
| Muscle tone | Limp | Some flexion of extremities | Active motion |
| Reflex irritability | No response | Grimace | Cough, sneeze, cry |
| Color | Blue, pale | Body pink Limbs blue | Completely pink |

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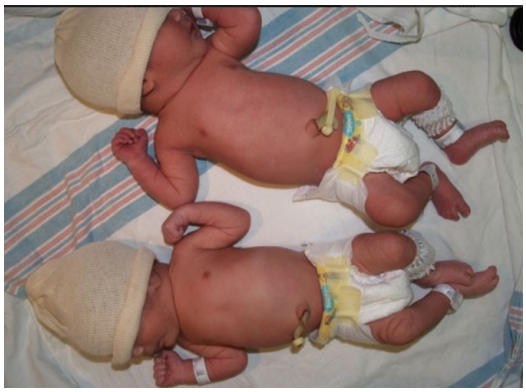
## Multiple Gestations

| | |
|---|---|
| Incidence | Spontaneous<br>Twins 1/80 pregnancies<br>Triplets 1/8000 pregnancies<br>IVF- increased risk of multiple gestations |
| Types | • Identical twin: diamniotic, monochorionic<br>• Fraternal twin: dichorionic, diamniotic |
| Fetal Risks | congenital anomalies, growth restriction or discordant growth, twin to twin transfusion, fetal demise,<br>PT delivery twins ~60% |



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## Definitions

| | |
|---|---|
| Preterm | <37 completed wks gestation |
| Late Preterm | 34 wks to 36 wks and 6 days |
| Full Term | 37-41 completed wks gestation |
| Post Term | ≥ 42 completed wks gestation |
| Low birth weight (LBW) | Weight <2500gms |
| Very low birth weight (VLBW) | Weight <1500gms |
| Extremely low birth weight (ELBW) | Weight <1000gms |

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## Late Preterm

| | |
|---|---|
| Definition | 34 0/7-36 6/7 completed weeks gestation |
| Characteristics | Metabolically and physiologically immature |
| Increased risk | • Hypoglycemia<br>• Feeding problems<br>• Temperature instability<br>• Respiratory distress, apnea<br>• Hyperbilirubinemia<br>• Feeding difficulties<br>• Increased hospital readmission rates<br>• Higher mortality then full term infants |

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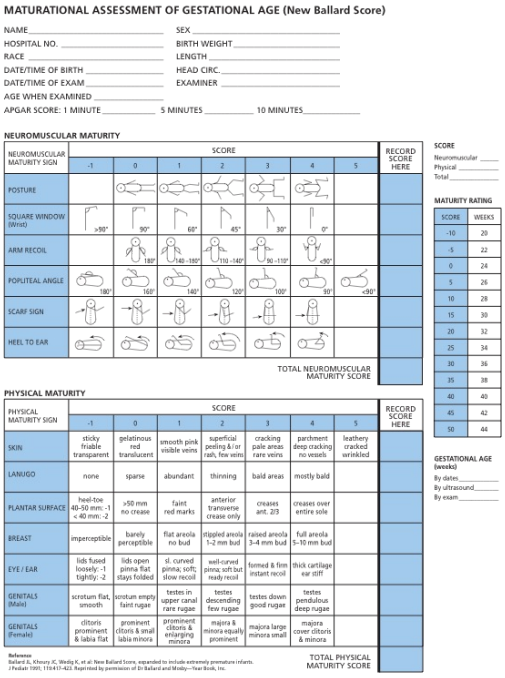

MATURATIONAL ASSESSMENT OF GESTATIONAL AGE (New Ballard Score)

NAME _____ SEX _____
HOSPITAL NO. _____ BIRTH WEIGHT _____
RACE _____ LENGTH _____
DATE/TIME OF BIRTH _____ HEAD CIRC. _____
DATE/TIME OF EXAM _____ EXAMINER _____
AGE WHEN EXAMINED _____
APGAR SCORE: 1 MINUTE _____ 5 MINUTES _____ 10 MINUTES _____

NEUROMUSCULAR MATURITY

| NEUROMUSCULAR MATURITY SIGN | SCORE -1 | 0 | 1 | 2 | 3 | 4 | 5 | RECORD SCORE HERE |
|---|---|---|---|---|---|---|---|---|
| POSTURE | | | | | | | | |
| SQUARE WINDOW (Wrist) | >90° | 90° | 60° | 45° | 30° | 0° | | |
| ARM RECOIL | | 180° | 140 - 180° | 110 - 140° | 90 - 110° | <90° | | |
| POPLITEAL ANGLE | 180° | 160° | 140° | 120° | 100° | 80° | <80° | |
| SCARF SIGN | | | | | | | | |
| HEEL TO EAR | | | | | | | | |

TOTAL NEUROMUSCULAR MATURITY SCORE

SCORE
Neuromuscular _____
Physical _____
Total _____

MATURITY RATING

| SCORE | WEEKS |
|---|---|
| -10 | 20 |
| -5 | 22 |
| 0 | 24 |
| 5 | 26 |
| 10 | 28 |
| 15 | 30 |
| 20 | 32 |
| 25 | 34 |
| 30 | 36 |
| 35 | 38 |
| 40 | 40 |
| 45 | 42 |
| 50 | 44 |

GESTATIONAL AGE (weeks)
By dates _____
By ultrasound _____
By exam _____

PHYSICAL MATURITY

| PHYSICAL MATURITY SIGN | SCORE -1 | 0 | 1 | 2 | 3 | 4 | 5 | RECORD SCORE HERE |
|---|---|---|---|---|---|---|---|---|
| SKIN | sticky friable transparent | gelatinous red translucent | smooth pink visible veins | superficial peeling & / or rash, few veins | cracking pale areas rare veins | parchment deep cracking no vessels | leathery cracked wrinkled | |
| LANUGO | none | sparse | abundant | thinning | bald areas | mostly bald | | |
| PLANTAR SURFACE | heel-toe 40-50 mm: -1 < 40 mm: -2 | >50 mm no crease | faint red marks | anterior transverse crease only | creases ant. 2/3 | creases over entire sole | | |
| BREAST | imperceptible | barely perceptible | flat areola no bud | stippled areola 1-2 mm bud | raised areola 3-4 mm bud | full areola 5-10 mm bud | | |
| EYE / EAR | lids fused loosely: -1 tightly: -2 | lids open pinna flat stays folded | sl. curved pinna; soft; slow recoil | well-curved pinna; soft but ready recoil | formed & firm instant recoil | thick cartilage ear stiff | | |
| GENITALS (Male) | scrotum flat, smooth | scrotum empty faint rugae | testes in upper canal rare rugae | testes descending few rugae | testes down good rugae | testes pendulous deep rugae | | |
| GENITALS (Female) | clitoris prominent & labia flat | prominent clitoris & small labia minora | prominent clitoris & enlarging minora | majora & minora equally prominent | majora large minora small | majora cover clitoris & minora | | |

TOTAL PHYSICAL MATURITY SCORE

Reference
Ballard JL, Khoury JC, Wedig K, et al: New Ballard Score, expanded to include extremely premature infants. J Pediatr 1991; 119:417–423. Reprinted by permission of Dr Ballard and Mosby—Year Book, Inc.

# GA Assessment

Preterm

- Visible veins
- Ears slow recoil
- Few creases in soles
- Immature areola
- Few rugae, labia majora and minor equally prominent

Post term

- Dry, cracked skin
- Ear stiff
- Creases in entire soles
- Full areola
- Testes pendulous/labia majora covers minora

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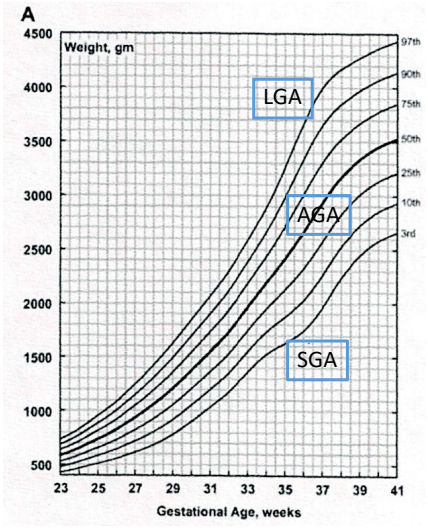
# Classifications

| | | |
|---|---|---|
| LGA | BW > 90% | At risk for perinatal asphyxia, birth trauma, hypoglycemia<br>IDM, Beckwith-Wiederman |
| AGA | BW 10-90% | |
| SGA | BW < 10% | At risk for hypoglycemia, polycythemia, temperature instability<br>• Chromosomal anomalies<br>• Congenital infections<br>• Congenital malformations<br>• Maternal smoking or drugs<br>• Genetic factors<br>• Metabolic disorders |




Modified from Olsen I E et al. Pediatrics 2010;125:e214-e224

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# Intrauterine Growth Restriction

Reduction of expected fetal growth

| IUGR | Ponderal index <10% | PI: $\frac{BW \times 100}{(\text{crown-heel})^3}$ |
|---|---|---|
| Symmetrical (HC proportional to body) | Early onset | • Congenital infection Congenital malformations<br>• Chromosomal anomalies<br>• Maternal chronic HTN |
| Asymmetrical (HC %> wt) | Late onset | • Uteroplacental insufficiency (chronic fetal hypoxia) |

- Higher mortality than AGA infants
- Increased incidence of **perinatal asphyxia** and fetal demise
- Prone to hypoglycemia, hypothermia, hyperbilirubinemia, polycythemia
- At increased risk for hypertension and metabolic syndrome as adults

39

# Routine Newborn Care

| | |
|---|---|
| Diet | Encourage early breast feeding |
| Cord care | Dry cord care, wash soap and water and dry thoroughly |
| Vitamin K | Prophylaxis for hemorrhagic disease of the NB (IM) |
| Eye prophylaxis | Erythromycin OU |
| Glucose | IDM, LGA, IUGR/SGA |
| NBS | PKU, hypothyroidism, HbS, thalassemia, CF, others |
| • CCHD screen | Saturation test - to detect cyanotic CHD (LE) |
| • Hearing screen | Otoacoustic emission (OAE) |
| Transcutaneous bilirubin | At discharge/infants at risk |
| Hepatitis B vaccine (prophylaxis) | $\geq$ 2000 gms within 24 hr, $\leq$ 2000gms at 1 mo or D/C whichever comes first |
| Preterm screens | ROP screen, IVH screen, Car seat test |

40

## Newborn Screening

| | |
|---|---|
| Healthy Newborns | Requirement for collection is at least 24 hours of age. |
| Newborns admitted to the NICU | • Collected upon admission, prior to any treatments/transfusions<br>• Second screening specimen should be collected between 48-72 hours of life<br>• Third specimen should be collected at 28 days of life or before discharge; whichever comes first, on all infants less than 2000 grams at birth or with prior abnormal screen |

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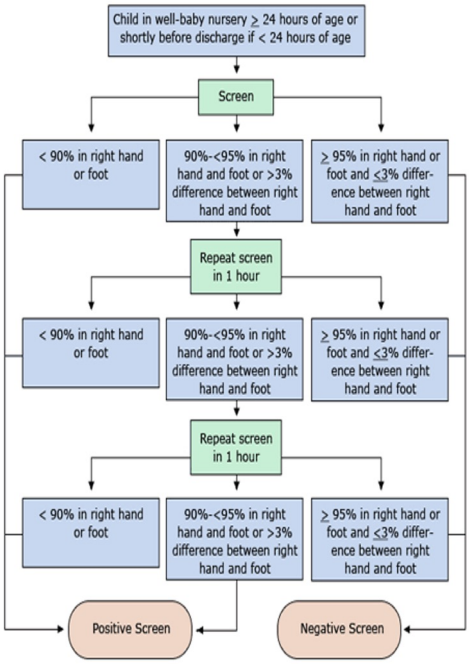
## Critical Congenital Heart Disease (CCHD ) Screening

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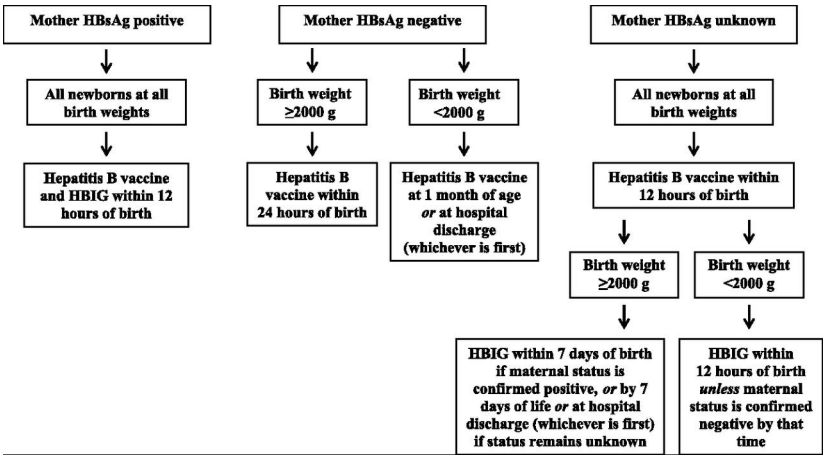
**Administration of the birth dose of hepatitis B vaccine by maternal HBsAg status.**




COMMITTEE ON INFECTIOUS DISEASES, and COMMITTEE ON FETUS AND NEWBORN Pediatrics 2017;140:e20171870



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# Hepatitis B Exposure (Mat +HBSAg)

| | |
|---|---|
| Transmission | + Mat HBsAg / - HBeAg 5-20%:<br>+ Mat HBsAg / + HBeAg 70-90%: |
| Risks | At risk of developing chronic hepatitis B infection |
| Management | • HBIG and HBV within 12 hrs of birth<br>• HB vaccine series at 1-2mo, 6mo<br>• Testing ->Anti-HBs and HBsAg at 9-18 months to identify chronic carriers and possible need for repeat vaccination) |

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## Hearing Screen

| | |
|---|---|
| Hearing loss | Affects 1-6/1000 infants |
| Screening | Universal screening at birth and no later than 1 month |
| Highest Risk | • Family history<br>• Syndromes known to have hearing loss<br>• Infections (CMV, TORCH, meningitis)<br>• Hyperbilirubinemia<br>• Preterm<br>• NICU admission |
| **SCREENING TESTS** | |
| **OAE** (Evoked otoacoustic emission) | Assesses function of peripheral nervous system |
| **ABR** (Auditory brainstem response) | Measures neural activity in cochlea, auditory nerve and brainstem |
| Treatment | Should be started before 6 months<br>Delays in language, speech and cognitive development if hearing impairment not identified<br>If patient fails hearing screen --> CMV testing,<br>if unable to do hearing screen –> CMV testing |

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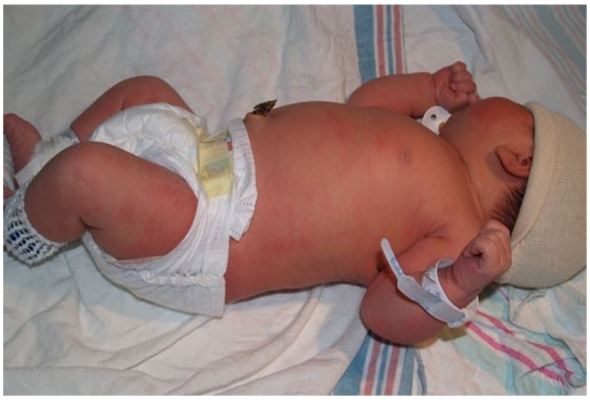
## Newborn Exam



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## Neonatal Reflexes

| Reflex | Age at Disappearance | Comments |
|---|---|---|
| Rooting reflex | 2-3 months | Aids attaching to nipple |
| Moro | 5-6 months | Some vestige may persist |
| Parachute reflex | Persists throughout life | Protect in event of a fall |
| Asymmetric Tonic neck reflex | 2-3 months | Most prominent at 1 month |
| Palmar Grasp | 5-6 months | Appears at 28 wks |
| Plantar Grasp | 9-10 months | Appears at 38-40 weeks |

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## Umbilical Cord Assessment

| | |
|---|---|
| Description | 3 vessels ->2 arteries and 1 vein, pearly white in color |
| Length | • Averages 55 cm in length<br>• **Short cords** - associated with fetal hypotonia, intrauterine compression, oligohydramnios<br>• **Long cords** -may twist or know an cause fetal distress |
| Course | Cords usually sloughs by day 10-14. |
| Delayed separation | May be seen in neutrophil chemotactic defects (**leukocyte adhesion deficiency type I)** |
| Single UA | 0.2-0.6 % of live births<br>30% can have other abnormalities (GU, CV, GI, Musculoskeletal system, T 18 |

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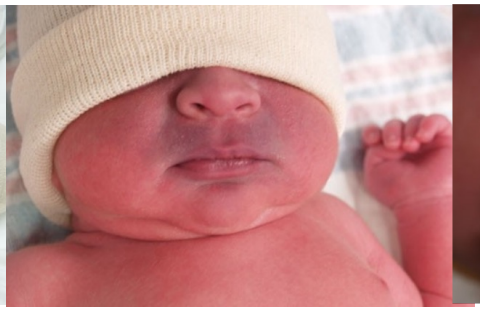
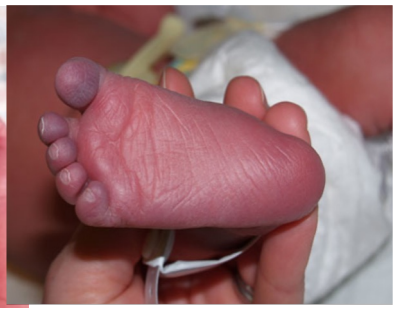
## Physical Exam Variants

Facial Bruising



Circumoral Cyanosis



Acrocyanosis

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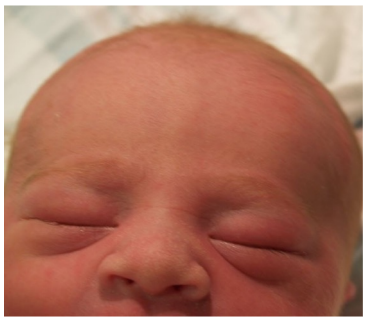
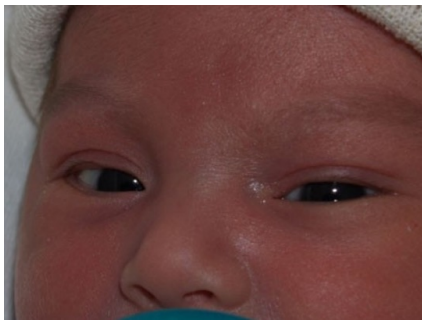
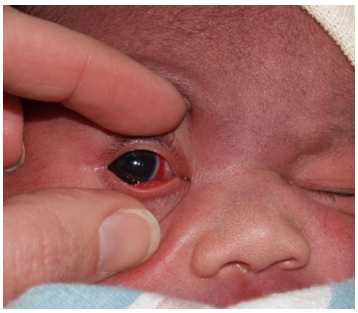
## Physical Exam Variants



Eyelid Edema



Dysconjugate Eye Movements



Subconjunctival Hemorrhage

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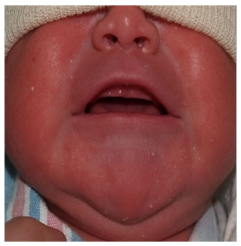
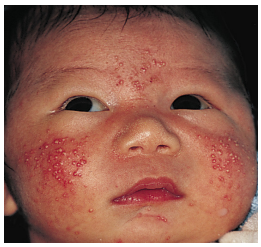
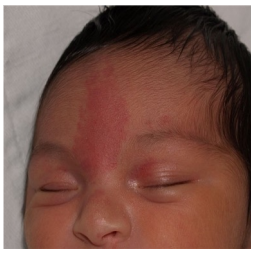
## Physical Exam Variants



Milia



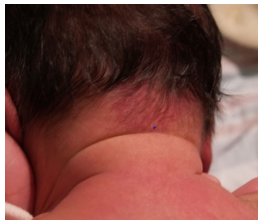
Neonatal Acne



Salmon Patch

Sebaceous Hyperplasia

Hemangioma



Stork Bite

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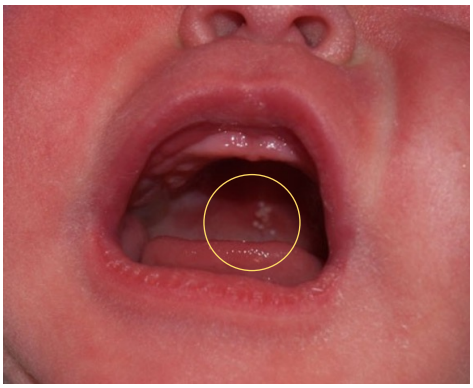
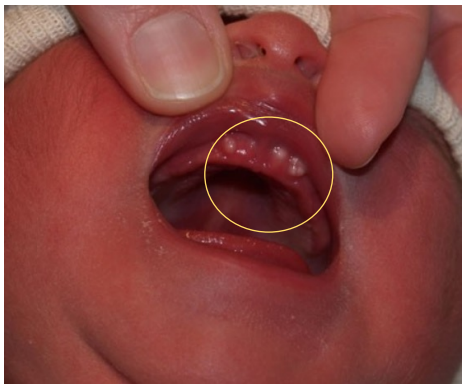
## Physical Exam Variants



Ebstein's Pearls



Bohn's Nodules

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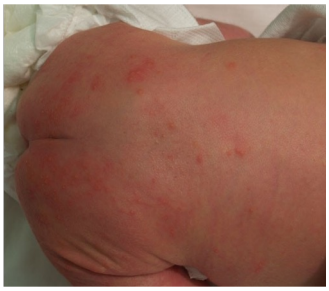
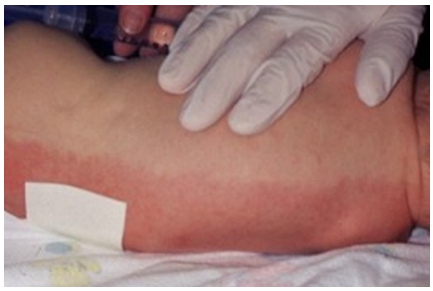
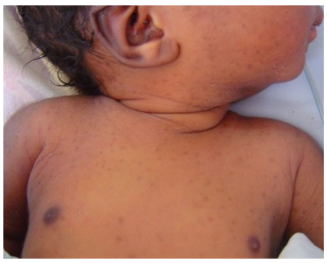
# Physical Exam Variants



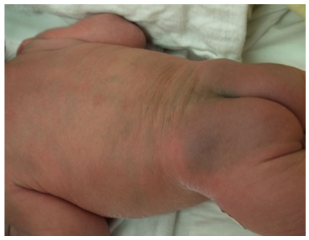
Erythema Toxicum



Harlequin Color Change



Transient Benign Pustular Melanosis



Mongolian Spots

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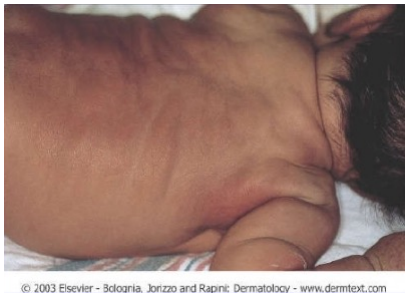
# Subcutaneous Fat Necrosis

| | |
|---|---|
| Etiology | Unknown |
| Presentation | Noninfectious panniculitis characterized by well circumscribed, indurated and nodular area of fatty necrosis in back, buttocks, proximal extremities or cheeks |
| Associations | IUGR and perinatal distress<br>Hypothermia therapy |
| Treatment | Benign, does not require treatment<br>only when significant metabolic of hematologic complications are present<br>50% develop hypercalcemia |

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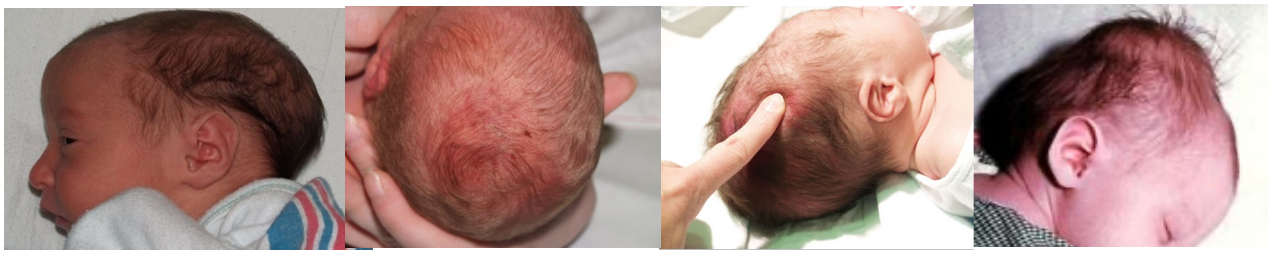
# Physical Exam Variants



Molding | Bruising | Caput Succedaneum | **C**ephalohematoma

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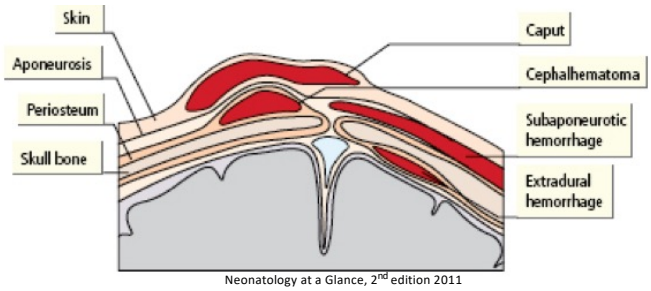


Neonatology at a Glance, 2nd edition 2011

| Lesion | Features of swelling | Crosses suture lines | Marked blood loss |
|---|---|---|---|
| **Caput Succedaneum** | Soft, pitting | Yes | No |
| **Cephalohematoma** | Firm, tense | No | No |
| **Subgaleal Hemorrhage (subaponeurotic)** | Firm, fluctuant, anterior displacement of ear | Yes | Yes |

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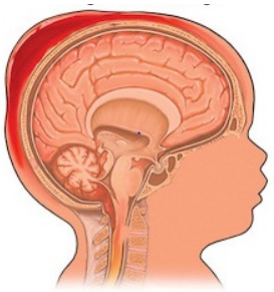
## Subgaleal Hemorrhage (SGH)

| | |
|---|---|
| Etiology | • Secondary to rupture of the emissary veins that course between the periosteum of the skull and the galea aponeurotica<br>• Associated with vacuum-assisted deliveries and may also have ICH or skull fracture |
| Clinical findings | • Firm, fluctuant, anterior displacement of ear, not confined by suture lines<br>• May present with hypotensive shock secondary to acute blood loss and hypovolemia. |
| Management | • Early recognition is crucial<br>• Follow serial Hct<br>• Supportive care |



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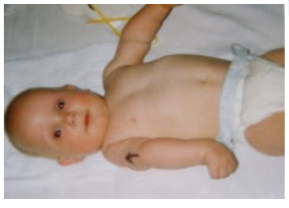
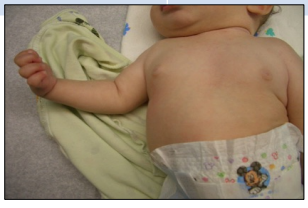
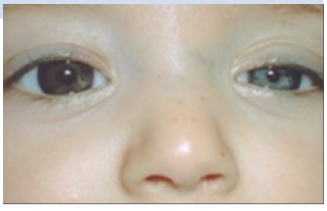
## Birth Injuries

| Injury | Nerve involvement | Findings |
|---|---|---|
| Facial Palsy | Facial Nerve injury | Usually unilateral, facial weakness with crying |
| Erb's Palsy | Nerves involved C5, C6, | Paralysis of shoulder/arm<br>Waiter's tip position,+grasp |
| Klumpke's Palsy | Nerve involved C8, T1 | Paralysis of arm and hand<br>Hand in claw-like posturing |
| Horner's Syndrome | Nerve involved C8,T1 | Ipsilateral miosis, ptosis, heterochromia, anhidrosis |
| Phrenic nerve paralysis | C3, C4, C5 | Diaphragmatic paralysis |


Facial Palsy


Erb's Palsy


Klumpke's Palsy


Horner's Syndrome

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## Neonatal Hypoglycemia

| Category | Example |
|---|---|
| **Physiologic (low glycogen stores** (depletion of hepatic glycogen stores) | • Prematurity<br>• IUGR<br>• PNA<br>• Sepsis |
| **Hyperinsulinism** (high intrauterine production (IDM), dysregulation) | • IDM (Maternal/fetal hyperglycemia -> Fetal hyperinsulinism<br>• Monogenic mutations in ABCC8,KCNJ11, GCK) |
| **Endocrine Deficiency** (GH and Cortisol deficiency inhibit production of hepatic glycogen stores) | • Adrenal Insufficiency<br>• GH deficiency |
| **IEM** (inhibit glucose production) | • Glycogen storage disease<br>• Fatty acid oxidation disorders<br>• Ketogenesis disorders |

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## Neonatal Hypoglycemia

| | |
|---|---|
| Infants at risk | PT, SGA/LGA infants, IDM, IUGR, asphyxia, sepsis, IEM |
| Presentation | Signs may be suttle and nonspecific<br>Jitteriness, lethargy, seizures, weak suck, floppiness, weak or high pitched cry<br>exaggerated Moro, cyanosis, tachypnea, apnea, tachycardia or bradycardia<br>temperature instability, poor feeding<br>If prolonged-> seizures, coma |
| Diagnosis | 1. Low blood glucose concentration<br>2. S/S consistent with NH<br>3. Resolution of S/S after restoring glucose levels to normal values |
| Persistent >48 hrs | Hyperinsulinism (most likely Dx) |
| Long term sequelae | MR, recurrent seizures, developmental delay |

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# Hypoglycemia Management

| Condition | Comment |
|---|---|
| All | *Treat any underlying condition |
| Asymptomatic | • IDM<br>• LGA<br>• Late Preterm<br>• FT SGA<br>With no symptoms should be fed within the first hour after birth (early feeding)<br>Screening glucose at 30 min after 1st feeding (follow per AAP guidelines) |
| Symptomatic | Serum glucose and IV glucose initiated if <40mg/dl |

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## Screening and Management of Postnatal Glucose Homeostasis in Late Preterm and Term SGA, IDM/LGA Infants

[(LPT) Infants 34 – $36^{6/7}$ weeks and SGA (screen 0-24 hrs); IDM and LGA ≥34 weeks (screen 0-12 hrs)]

**Symptomatic and <40 mg/dL ⟶ IV glucose**

**ASYMPTOMATIC**

| Birth to 4 hours of age | 4 to 24 hours of age |
|---|---|
| INITIAL FEED WITHIN 1 hour<br>Screen glucose 30 minutes after 1st feed | Continue feeds q 2-3 hours<br>Screen glucose prior to each feed |
| Initial screen <25 mg/dL | Screen <35 mg/dL |
| Feed and check in 1 hour | Feed and check in 1 hour |
| <25 mg/dL ↓ IV glucose* | 25–40 mg/dL ↓ Refeed/IV glucose* as needed |
| <35 mg/dL ↓ IV glucose* | 35 – 45 mg/dL ↓ Refeed/IV glucose* as needed |

Target glucose screen ≥45 mg/dL prior to routine feeds

* Glucose dose = 200 mg/kg (dextrose 10% at 2 mL/kg) and/or IV infusion at 5–8 mg/kg per min (80–100 mL/kg per d). Achieve plasma glucose level of 40-50 mg/dL.

Committee on Fetus and Newborn Pediatrics 2011;127:575579



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# Neonatal Jaundice

| | |
|---|---|
| Physiologic | Normal transitional process |
| Non-physiologic | Bilirubin production is exaggerated or excretion is reduced beyond normal |
| Concerns | High Bilirubin levels may lead acute bilirubin encephalopathy (ABE) and Kecnicterus |

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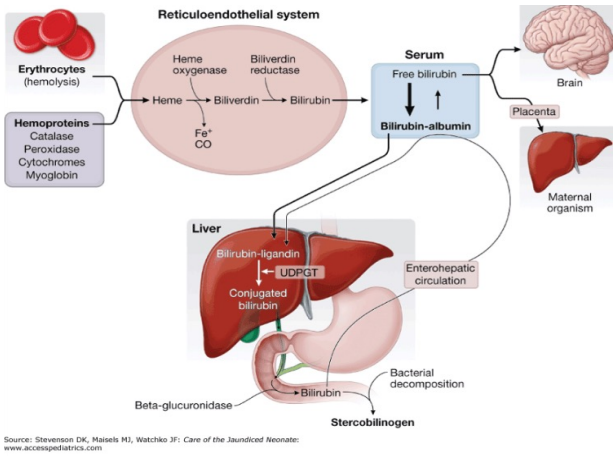
# Bilirubin Metabolism in the Newborn

- Synthesis- RBC breakdown
- Transport- Albumin
- Metabolism

RBCs (80%)
*and*
Ineffective Erythropoiesis, non-hemoglobin hemoprotein (myoglobin,catalases, cytochrome, NO synthase) (20%)




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

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## Indirect Hyperbilirubinemia

| | |
|---|---|
| Causes | • Physiologic Jaundice (most common cause)<br>• Hemolysis (Rh, ABO incompatibility, G6PD deficiency)<br>• Breast Feeding related jaundice<br>• Breast Milk Jaundice<br>• Polycythemia<br>• Blood extravasation<br>• Decreased hepatic uptake or conjugation |
| Management | Phototherapy<br>Exchange transfusion<br>IVIG (Blood group incompatibility) |
| Complications | Bilirubin Encephalopathy<br>Kernicterus |

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## Physiologic Jaundice

| Increased bilirubin load | Deficient Conjugation | Increased Enterohepatic Recirculation |
|---|---|---|
| High RBC mass (Hct often 45-60) | Decrease hepatocyte uptake of bilirubin | High concentration of intestinal β- glucuronidase |
| Shortened RBC lifespan (70-90 days) | UGT1A1 levels at 1% of adult levels at birth | High concentration of bilirubin in meconium |

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## Blood Group Incompatibility

| Mother | Infant | Coombs | Diagnosis |
|---|---|---|---|
| Rh - | Rh+ | + | Rh |
| O | A or B | +/- | ABO |
| O, A, B | O,A,B | + | Minor group |

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## Breast Feeding Related Jaundice

| | |
|---|---|
| Breast Feeding Jaundice | • Common<br>• Presents in the first 3 days of life<br>• Breastfeeding- Important risk factor for hyperbilirubinemia in healthy term and near term infant<br>• **Lack of volume => inadequate production or poor intake**<br>• Effective breast feeding can reduce risk of severe hyperbilirubinemia |
| Breast Milk Jaundice | • Rare<br>• Late onset, may persist 1-3 months<br>• Related to unidentified component in breast milk that causes increased enterohepatic circulation |

-

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## G6PD Deficiency

| | |
|---|---|
| Deficiency | G6PD (Most common enzyme defect )<br>Decreases protection against oxidative distress |
| Inheritance | **X-linked disorder**, affects mostly males<br>Affect 13 % Males, 4% females of African American descend<br>Most no family Hx |
| Clinical findings | Sudden increase in TSB, no lab evidence of hemolysis<br>Can cause **severe hyperbilirubinemima** and kernicterus |
| Ancestry | Sub-Sahara, Africa, Middle East, Mediterranean, Arabian Peninsula or South East Asia |
| Diagnosis | measuring G6PD activity in RBC (best 3 month after event) |
| Crisis Prevention | Should avoid medications (antimalarials, antibiotics-nitrofurantoin and sulfonamides), contact with moth balls and eating fava beans |

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## Conjugation Defects

| | Crigler-Najar Type I | Crigler-Najar Type II (Arias Syndrome) | Gilbert Syndrome |
|---|---|---|---|
| Inheritance | AR | AR or AD | AR or AD |
| UDPGT activity | Absent | <10% | 50% |
| TSB | >20 mg/dl | 5-15 mg/dl | 3-5 mg/dl |
| Kernicterus Risk | High | Low risk | No apparent risk |

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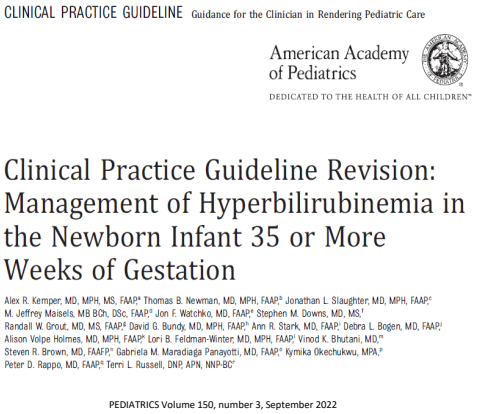
# Management of Hyperbilirubinemia



CLINICAL PRACTICE GUIDELINE Guidance for the Clinician in Rendering Pediatric Care

American Academy of Pediatrics

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Clinical Practice Guideline Revision: Management of Hyperbilirubinemia in the Newborn Infant 35 or More Weeks of Gestation

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- Update and replace the 2004 Guidelines
- Central to this guideline is having systems in place including policies in hospitals and other types of birthing locations to provide the care necessary to minimize the risk of kernicterus
- Universal predischarge bilirubin screening with measures of total serum bilirubin (TSB) or transcutaneous bilirubin (TcB) linked to specific recommendations for follow-up
- Addresses:
  - Prevention
  - Risk Assessment
  - Monitoring
  - Treatment

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# Risk Factors for Developing Significant Hyperbilirubinemia

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|---|
| • Lower GA (risk increases with every week less than 40 wks) |
| • Jaundice in first 24 hrs |
| • Predischarge TcB or TSB close to phototherapy threshold |
| • Hemolysis from any cause |
| • Phototherapy before discharge |
| • Parent or sibling requiring phototherapy or exchange transfusion |
| • Family Hx or genetic ancestry suggestive of inherited red cell disorder (G6PD) |
| • Exclusive breast feeding with suboptimal intake |
| • Cephalohematoma or significant bruising |
| • Trisomy 21 |
| • Macrosomic IDM |

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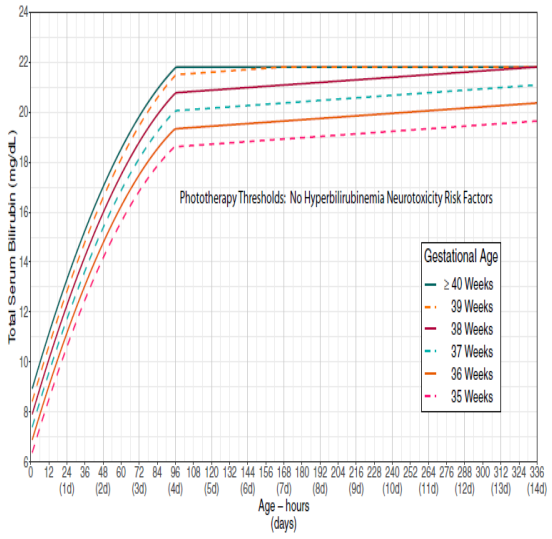
# Treatment of Hyperbilirubinemia




## Phototherapy
## No Risk Factors

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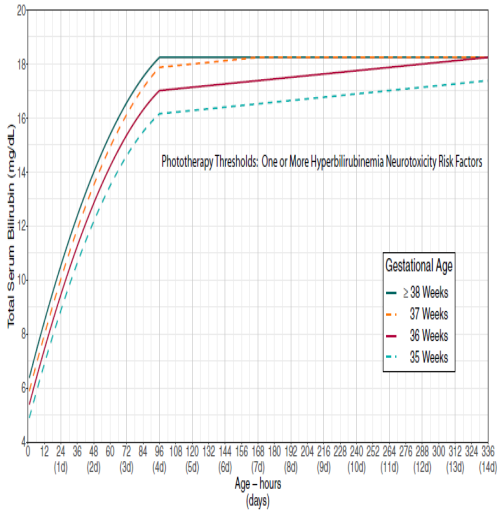
# Phototherapy




## NEUROTOXICITY RISK FACTORS:

- GA <38 wks ,risk increases with degree of prematurity
- Albumin < 3
- Isoimmune hemolytic disease (+Direct Coombs, G6PD deficiency or other hemolytic condition)
- Sepsis
- Significant clinical instability in the previous 24 hrs.

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## Treatment of Hyperbilirubinemia Escalation of Care/Exchange Transfusion

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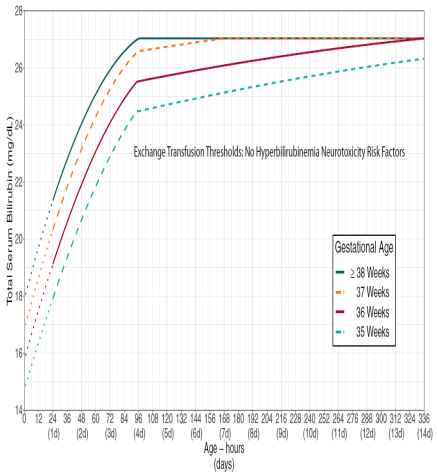


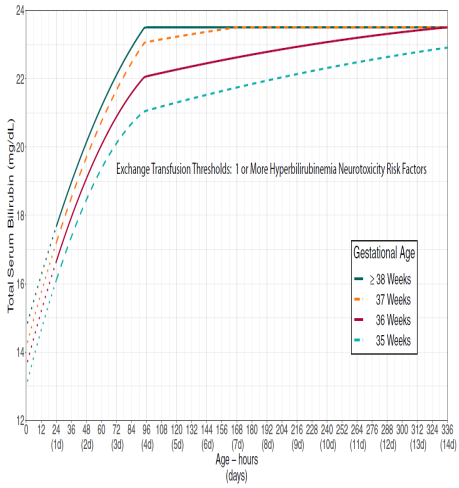


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## Prevention of Severe Hyperbilirubinemia

- Evaluate all infants for risk factors
- Increase the frequency of breast feedings
- Phototherapy if indicated
- Screening prior to hospital discharge
- Follow up with Pediatrician as indicated by guidelines

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Good luck
and
Thank you!!

Nicklaus Children's Hospital

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