Neonatology for Obstetricians

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Objectives

By the end of this presentation the student should be able to:

- Know the basics of neonatal resuscitation.
- Know the importance of Apgar's score and its limitations.
- Know the prematurity and its management.
- Know the intrauterine growth restriction (IUGR) and dysmaturity.
- Know the types of congenital malformations

Neonatal

Resuscitation

Who Requires Resuscitation?

Most newly born babies are vigorous.

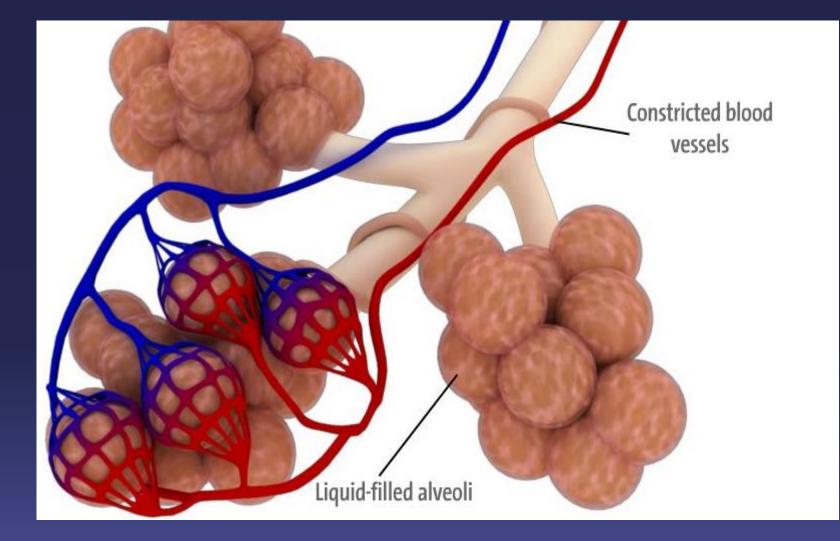
About 10% of newborns require some assistance.

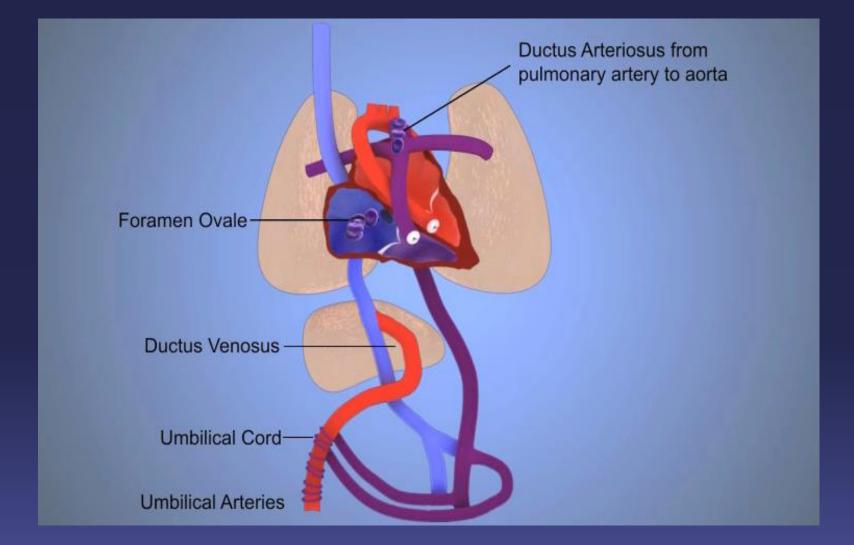
 Only 1% need major resuscitative measures (intubation, chest compressions, and/or medications).

In Utero

Alveoli filled with lung fluid.
Pulmonary arterioles constricted.
Blood flow diverted across ductus arteriosus.

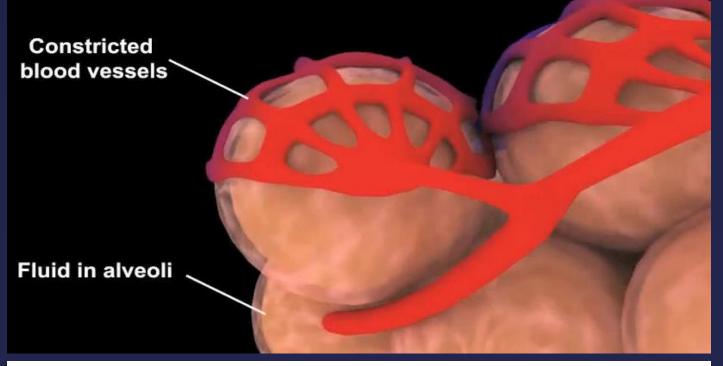
Pulmonary blood flow diminished.

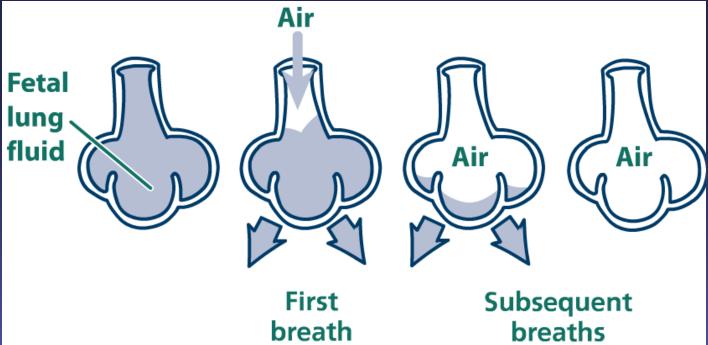


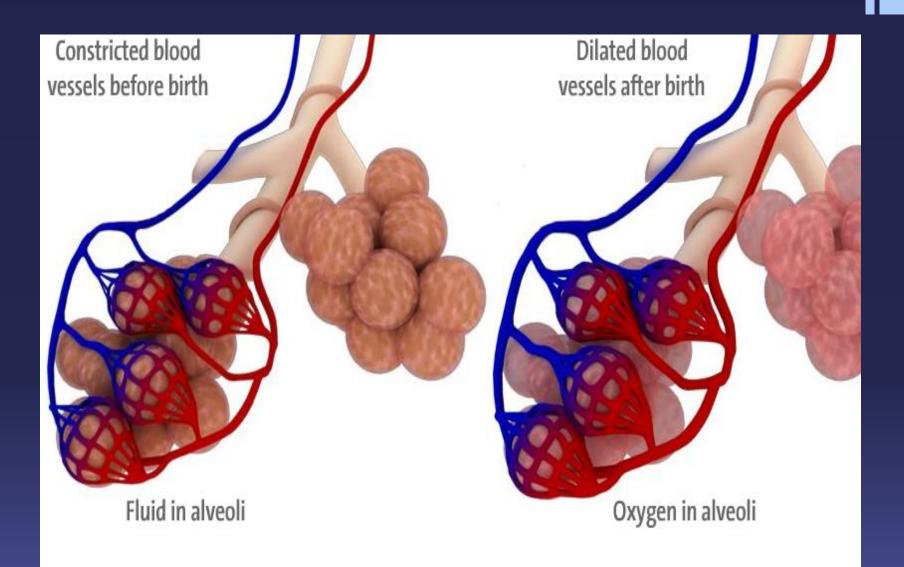


After Delivery

Lungs expand with air
Fetal lung fluid leaves alveoli
Pulmonary arterioles dilate
Pulmonary blood flow increases

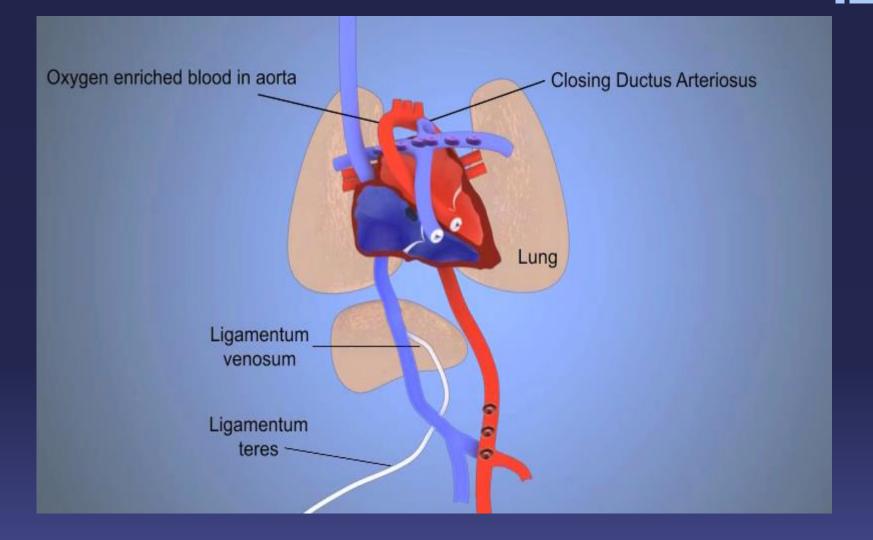






Normal Transition

- The following changes take place soon after birth:
 - Fluid in alveoli absorbed
 - Umbilical arteries and vein constrict thus increasing blood pressure
 - Ductus arteriosus constricts
 - Blood vessels in lung relax
 - Blood flows through lungs to pick up oxygen
 - Blood oxygen levels rise



Transition Abnormalities

Lack of ventilation of the lungs -> sustained constriction of the pulmonary arterioles, preventing systemic arterial blood from being oxygenated
 Prolonged lack of adequate perfusion and

 Prolonged lack of adequate perfusion and oxygenation to the baby's organs can lead to damage to many organs (especially the brain), or death

Signs of the perinatal compromise

Poor muscle tone Depressed respiratory drive Bradycardia Low blood pressure Tachypnea Cyanosis

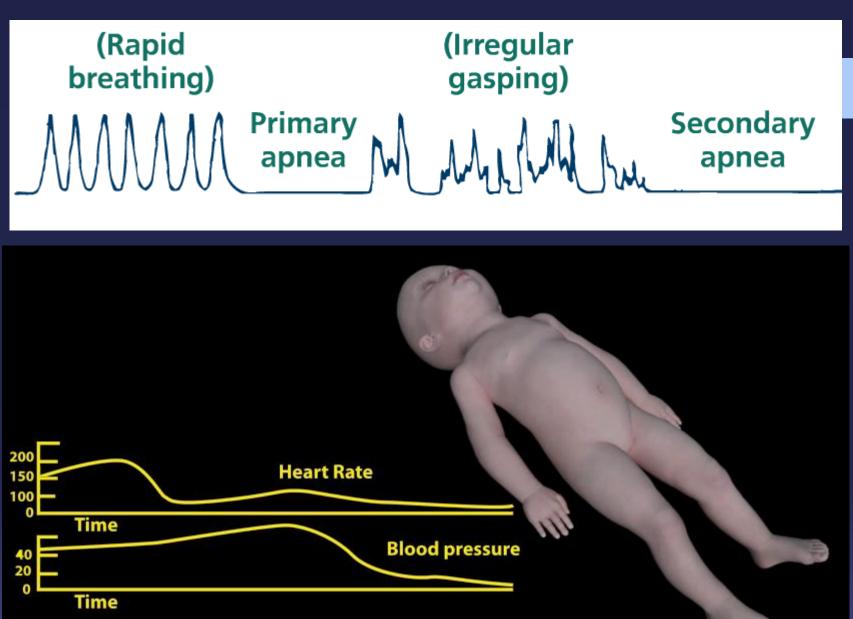
Primary Apnea

When a fetus/newborn first becomes deprived of oxygen, an initial period of attempted rapid breathing is followed by primary apnea and dropping heart rate that will improve with tactile stimulation

Secondary Apnea

 If oxygen deprivation continues, secondary apnea ensues, accompanied by a continued fall in heart rate and blood pressure.

Secondary apnea cannot be reversed with stimulation; assisted ventilation must be provided.



Primary Aprea Secondary aprea

Preparation for Resuscitation

EVERY delivery should be attended by at least one person whose only responsibility is the baby and who is capable of initiating resuscitation

Prepare necessary equipment

Turn on radiant warmer

Check resuscitation equipment

Remember your ABCs

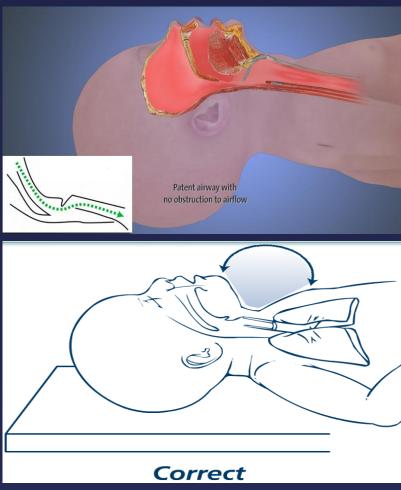
- A Airways
- B Breathing
- C Circulation
- D Drugs



Initial Steps

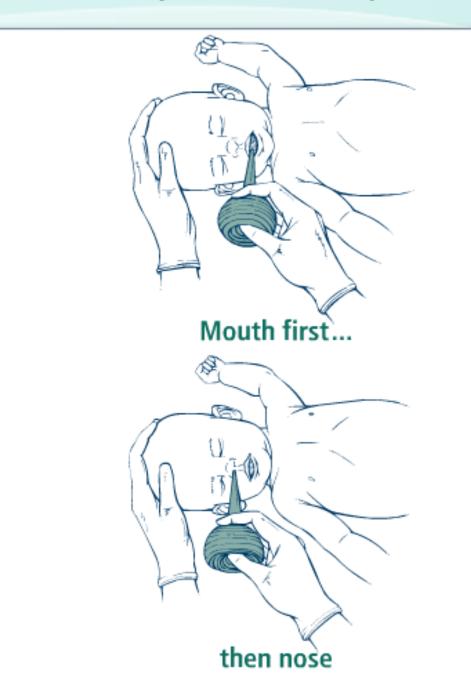
- Provide warmth
- Position; clear airway (as necessary)
- Dry, stimulate, reposition

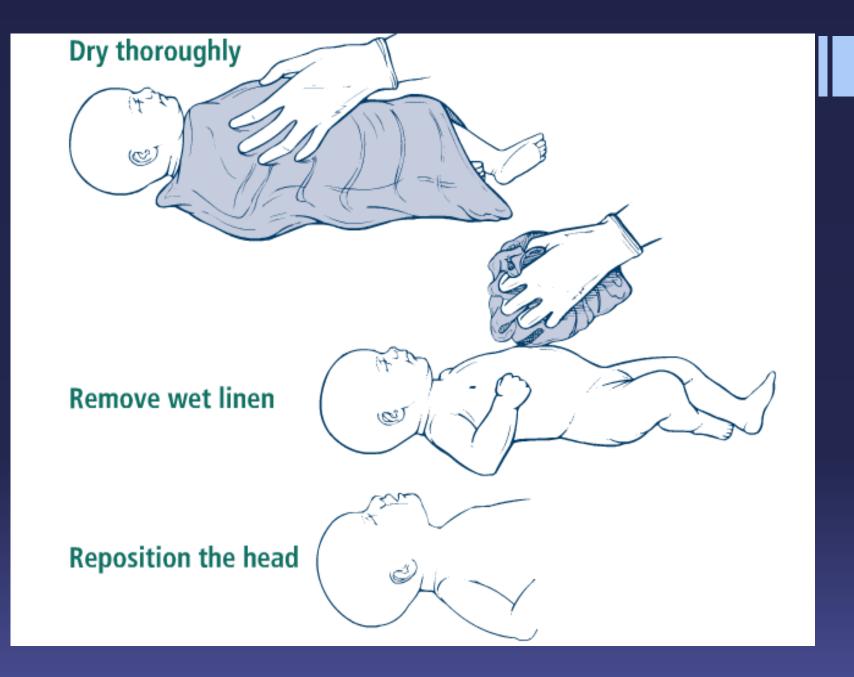


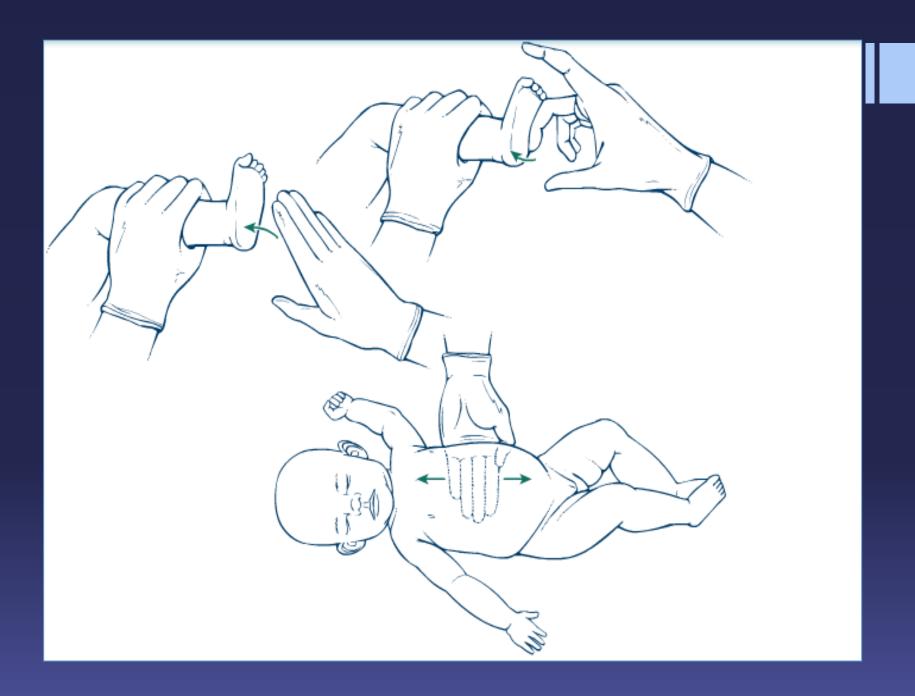


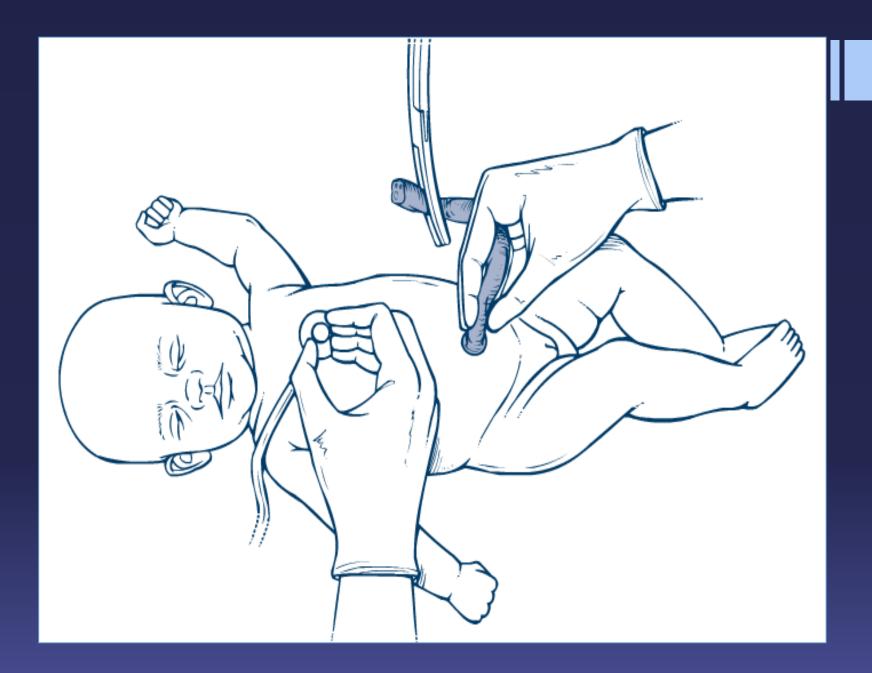


How do you clear the airway if no meconium is present?





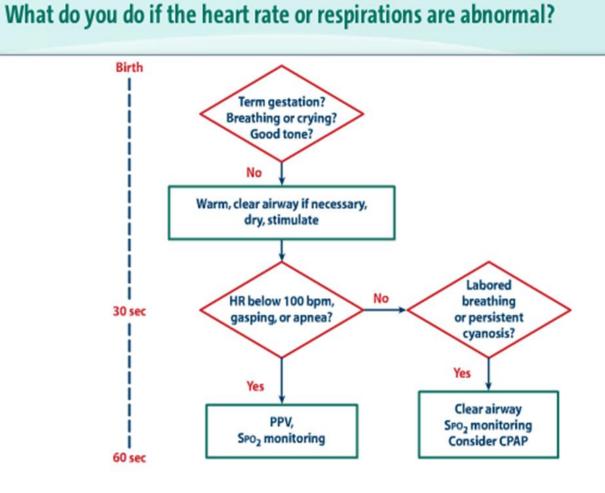




Evaluation

B

Breathing



Targeted Pre-ductal SPO ₂ 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%				





If heart rate <60 bpm despite adequate ventilation for 30 seconds:</p>

- Provide chest compressions as you continue assisted ventilation
- Consider intubation of the trachea at this point
- Evaluate again if heart rate <60 bpm proceed to the next step





If heart rate <60 bpm despite adequate ventilation and chest compressions:</p>

- Administer epinephrine as you continue assisted ventilation and chest compressions
- Consider intubation of the trachea at this point if not intubated yet

Points to remember

- The most important and effective action in neonatal resuscitation is to ventilate the lungs.
- Effective PPV in secondary apnea usually results in rapid improvement of heart rate.

If heart rate does not increase, ventilation may be inadequate and/or chest compressions and epinephrine may be necessary.

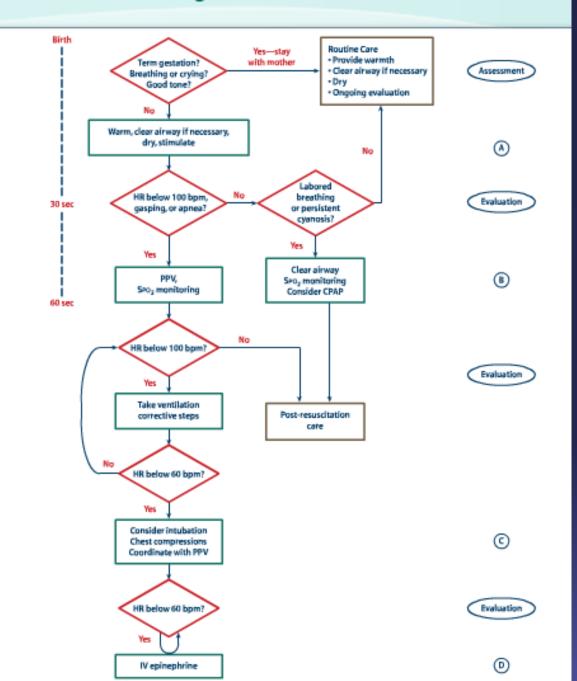
Points to remember

■HR <60 bpm → Additional steps needed</p>

- ■HR >60 bpm → Chest compressions can be stopped
- ■HR >100 bpm and breathing → PPV can be stopped

Always proceed to the next step if no improvement after 30 seconds

The resuscitation flow diagram





APGAR's Score

Apgar's score

In 1952, Dr Virginia Apgar devised a scoring system that was a rapid method of assessing the clinical status of the newborn infant and the need for prompt intervention to establish breathing.

Parameters assessed:

Color, HR, RR, reflexes and muscular tone.



Section 10 Why is the Apgar score *not* used to guide resuscitation?

APGAR SCORE			Gestational Age			weeks		
SIGN	0	1	2	1 minute	5 minutes	10 minutes	15 minutes	20 minutes
Color	Blue or Pale	Acrocyanotic	Completely Pink					
Heart Rate	Absent	< 100 bpm	> 100 bpm					
Irritability	No Response	Grimace	Cry or Active Withdrawal					
Muscle Tone	Limp	Some Flexion	Active Motion					
Respiration	Absent	Weak Cry; Hypoventilation	Crying					
			TOTAL					
Comments:				Resuscitation				
			Minutes	1	5	10	15	20
			Oxygen					
			PPV/NCPAP					
			ETT					
			Chest Compressions					
			Epinephrine					

Limitations of Apgar's score

Might be depressed due to

- Maternal causes (anesthesia, drugs).
- Neurological conditions.
- It is not always a good indicator for later outcomes.
- It does not correlate well with perinatal or intrapartum insults.



Infant of diabetic mother (IDM)

Why IDM infant is "big"

Macrosomia:

- Defined as:
 - Birth weight > 90th percentile for gestational age <u>or</u>
 - Greater than 4,000 g
- More in IDMs (15% 45%) vs. normal infants (8% to 14%).

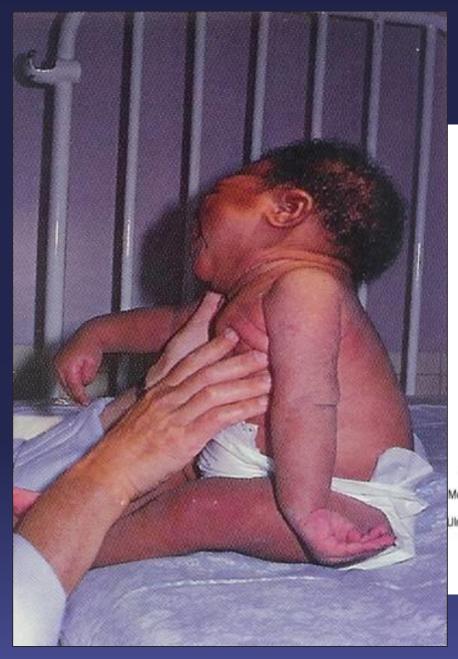
Why IDM infant is "big"

- Fetal hyperglycemia and hyperinsulinemia affect primarily insulin sensitive tissues such as fat.
- The risk of macrosomia is similar for all classes of diabetes (type 1, type 2, and gestational).
- Glycemic control in the 2nd and 3rd trimesters may reduce the macrosomia rate to near baseline.

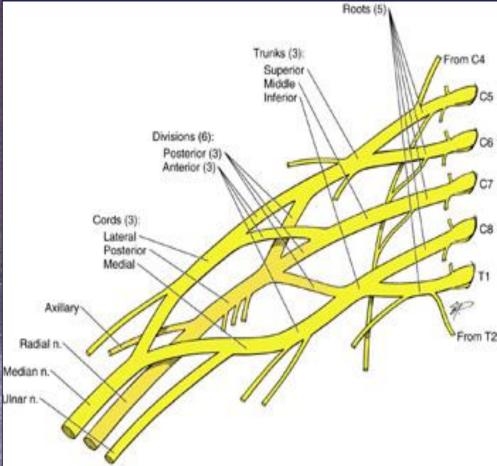
Why IDM infant is "big"

 Macrosomia is a risk factor for intrapartum injury (shoulder dystocia and asphyxia) and for cesarean delivery.





Erb's Palsy





Preterm Infant

Prematurity

By gestational age Early preterm – 23 to 33 6/7 Late preterm – 34 to 36 6/7 By weight ELBW - less than 1000g VLBW - less than 1500g LBW -less than 2500g

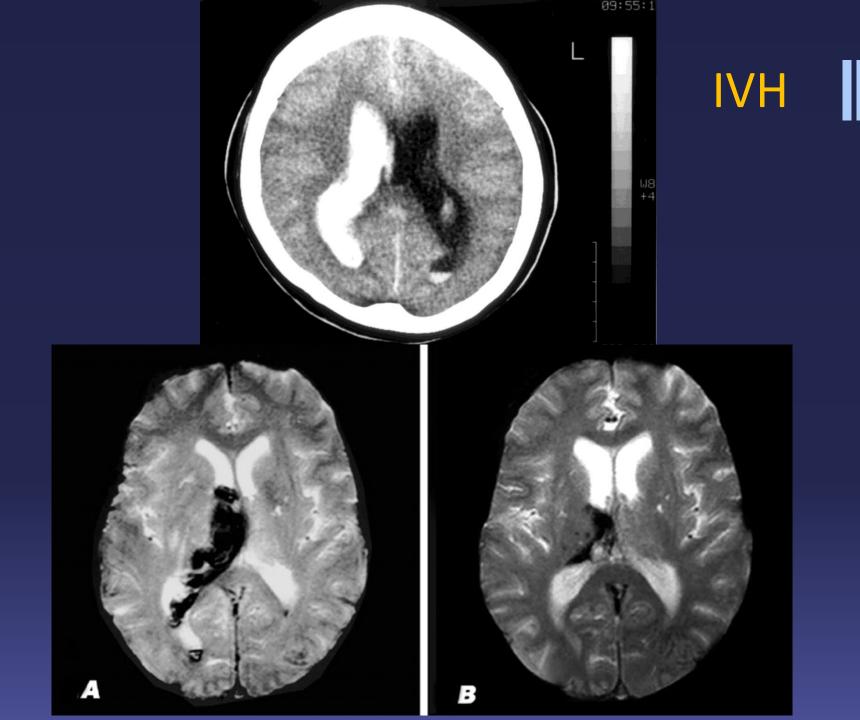
Prematurity Causes

Maternal

- Chronic illnesses
- Uterine anomalies
- Others
- Placental
 - Insufficiency
 - Hemmorhage
- Fetal
 - Infections
 - Genetic and chromosomal

Why Are The Preterm At Risk?

- Surfactant deficiency.
- Poor temperature control.
- Decreased respiratory drive.
- Weak muscles make spontaneous breathing difficult.
- Oxygen toxicity.
- Possible infection.
- Susceptibility to IVH.
- Susceptibility to hypovolemia due to blood loss.



Hydrocephalus



Neonatal Mortality Associated with Prematurity, USA (2003-2005)

Gestational Age (completed weeks)	% Survival if admitted to NICU
23	38-66
24	43-81
25	85-92
26	86-93
27-32	86-98

The Morbidity of Extreme Prematurity

Risk of all significant morbidities relate to degree of prematurity, At 23 weeks:
BPD 50-70%
White Matter Injury 32%
NEC 9%



Dysmaturity

A complex of signs occurring in an infant, such as a relative absence of subcutaneous fat, skin wrinkling, prominent fingernails and toenails, and a meconium staining of the skin and the placental membranes, that is associated with postmaturity or placental insufficiency.

The American Heritage[®] Medical Dictionary Copyright © 2007, 2004 by Houghton Mifflin Company.

Post term infant



IUGR vs. SGA

-IUGR

Failure of normal fetal growth caused by multiple adverse effects on the fetus.

SGA

- When infant birth-weight is < population norms (lower than a predetermined cutoff weight.) or
- Having a birth-weight <10th percentile for gestational age or >2 standard deviations below the mean for gestational age.

IUGR

What is the cause of the IUGR?When was it detected?Are there signs of fetal compromise?

IUGR complications

Increased risk of perinatal complications

- Perinatal asphyxia.
- Cold stress.
- Hyperviscosity (polycythemia).
- Hypoglycemia.

Outcomes of IUGR infants

- The most important determinant of IUGR outcome is its cause
 - Infants with chromosomal disorders or congenital infections (eg, CMV) experience early IUGR, and commonly have a disability.
 - Preterm IUGR infants have a risk of major disability (eg, CP or MR) that is similar to AGA preterm of the same size.



Congenital Malformations

Congenital Anomalies

Congenital

- The presence of the defect at birth
- Major (2% to 3% of live born infants)
 - Medical and social consequences (cleft palate and neural tube defects).
- Minor (Up to 15%)
 - No significant health or social burden (epicanthal folds and a single palmar crease).
- Normal phenotypic variants
 - Physical differences occurring in 4% or more individuals of a general population.

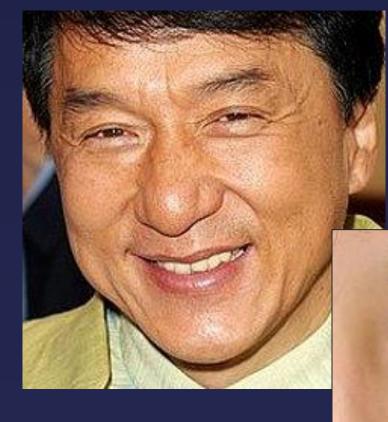
Major





Minor





Normal variant



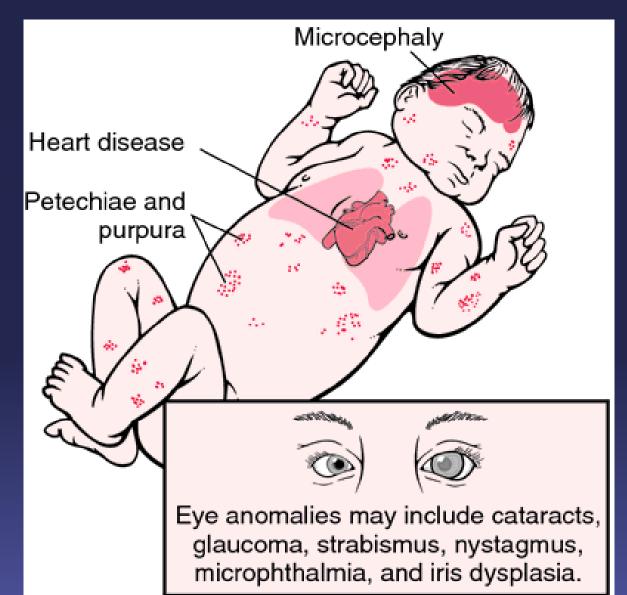
Simian crease



Malformations

- Abnormal processes <u>during</u> the initial formation of a structure
- May result in:
 - Faulty configuration (transposition of the great vessels).
 - Incomplete formation (cleft palate).
 - Agenesis (absence of radius).
- May be the result of:
 - Genetic chromosomal (10%) and single gene defects (4%).
 - Environmental insults (teratogens).
 - Drugs thalidomide .
 - Congenitally acquired viruses Rubella .
- Multifactorial in 25%, unknown in 40%-45%

Congenital Rubella



Malformations - Cleft lip and palate



Deformations

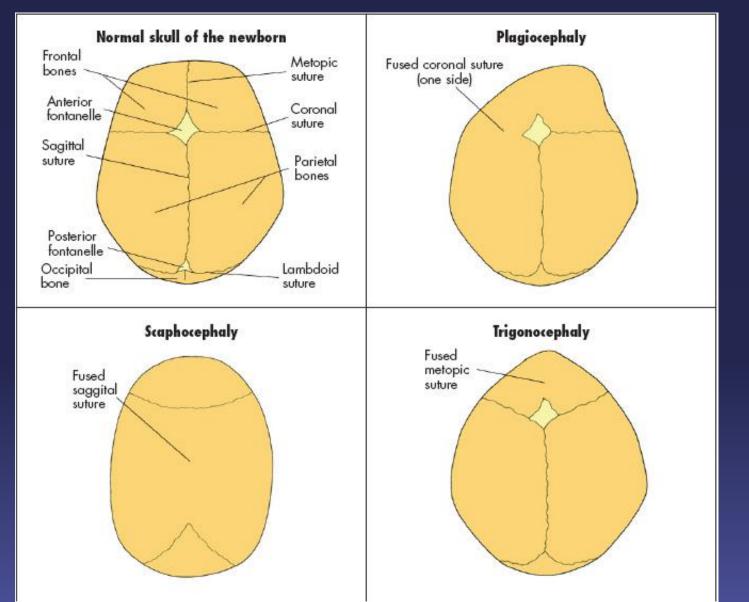
- Unusual and prolonged mechanical forces acting on normal tissue.
- External (uterine constraint) vs. intrinsic (edema).
- Mostly Musculoskeletal tissues
 - Tibial bowing and hip dislocation associated with breech presentation.
 - Webbing of the neck associated with the involution of a giant cystic hygroma).
 - Craniostenosis resulting from in utero constraint.
- Typically improve postnatally.
- Resolution depends on the duration of the abnormal forces and the extent of subsequent growth.



Deformations



Deformations



Disruptions

Breakdown of normal tissue <u>after</u> formation

Causes

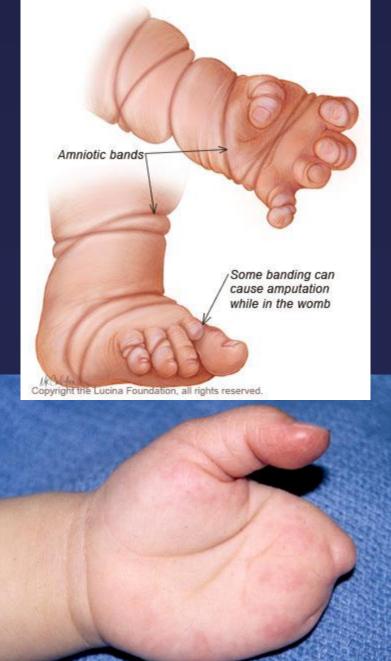
 Mechanical compressive forces, hemorrhage, thrombosis, and other vascular impairments.

Manifestations

 Alterations of configuration, division of parts not usually divided, fusion of parts not usually fused, and the loss of previously present parts.

Examples

- Porencephalic cyst secondary to a vascular accident.
- Limb amputations caused by amniotic bands.



Disruptions





Dysplasia

 Abnormal cellular organization or function.

- Typically affects a single tissue type.
- Examples

Hamartomas, ectodermal dysplasia, and skeletal dysplasias.

Ectodermal dysplasia



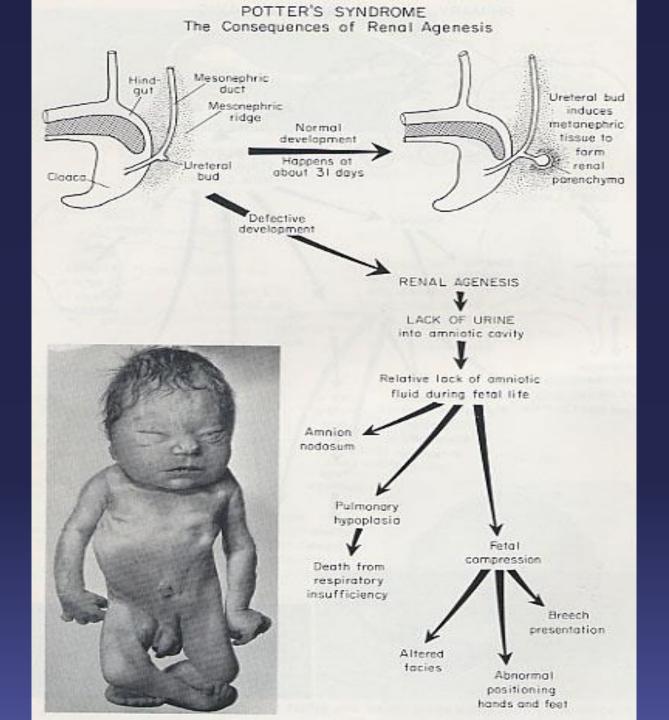
Multiple malformations

0.7% of live birthsSequence vs. Syndrome

A malformation sequence

- All of the anomalies can be explained on the basis of a single problem
- Examples
 - Oligohydramnios sequence
 - Pierre Robin sequence





A malformation syndrome

Multiple structural defects that are NOT explained on the basis of a single initiating defect but share a cause (chromosomal abnormalities, mutant gene disorders, or environmental teratogens)



Trisomy 13 and Trisomy 18

small mouth, small jaw, short neck

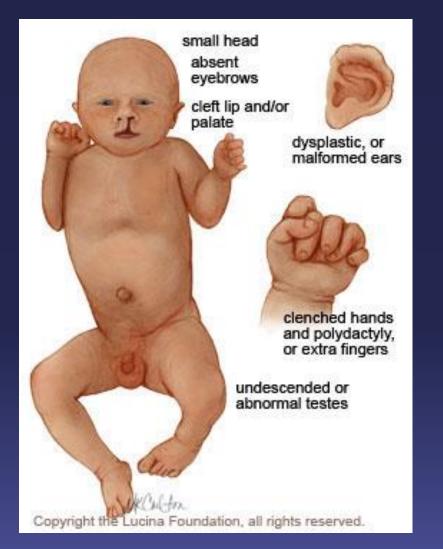
shield chest, or short and prominent sternum; and wideset nipples occiput, or back part of the skull, is prominent

> dysplastic, or malformed ears



clenched hands with overlapping fingers

flexed big toe; prominent heels Copyright the Lucine Foundation, all rights reserved.



Tracher collins syndrome



Thank You