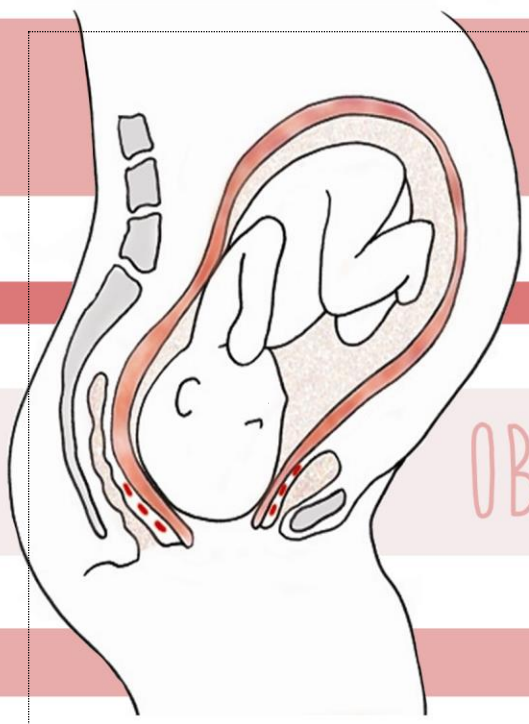

OBSTETRICS AND GYNECOLOGY

26) Neonatology for Obstetricians

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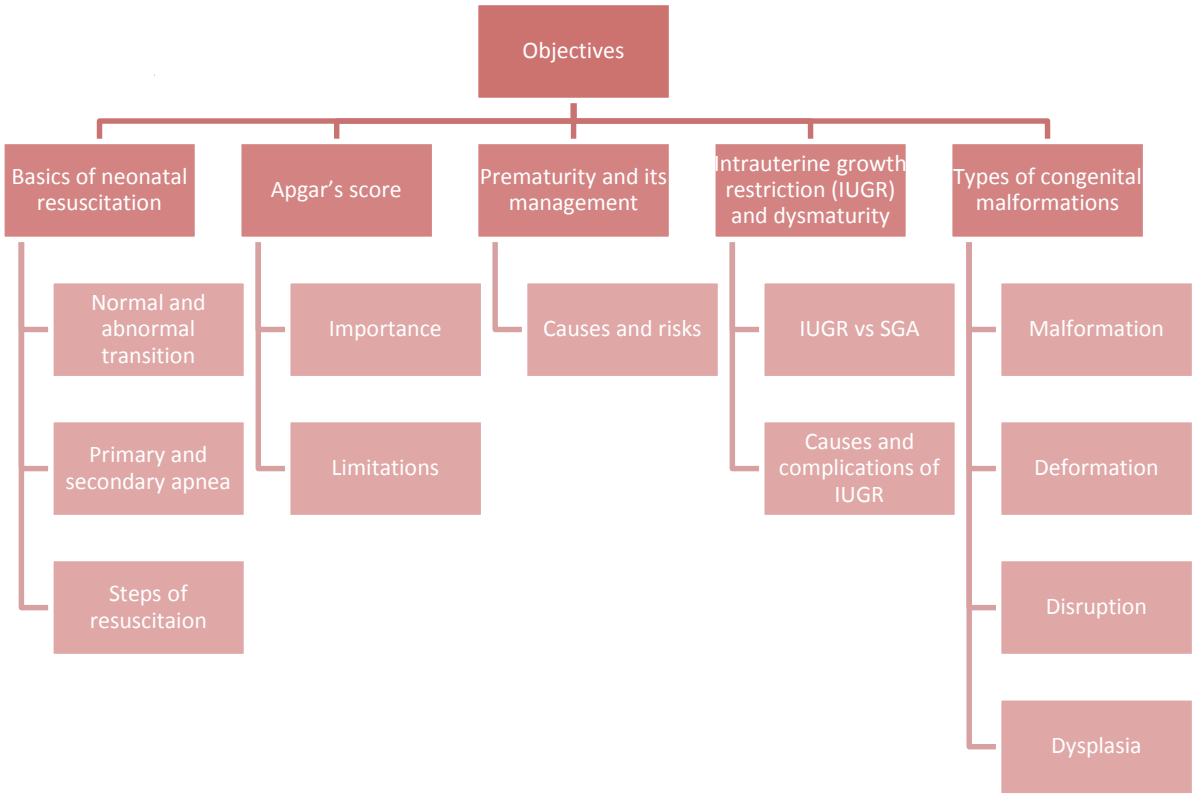
Doctor's note Team's note Not important Important 431 teamwork

Objectives:


Objectives
Basics of neonatal resuscitation
Apgar's score
Prematurity and its management
Intrauterine growth restriction (IUGR) and dysmaturity
Types of congenital malformations
Normal and abnormal transition
Primary and secondary apnea
Steps of resuscitaion
Importance
Limitations
Causes and risks
IUGR vs SGA
Causes and complications of IUGR
Malformation
Deformation
Disruption
Dysplasia

▪ *Neonatal Resuscitation:*

Who Requires Resuscitation?

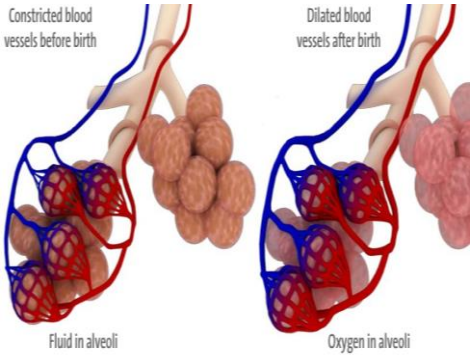
- Most newly born babies are vigorous (strong and healthy)
- About 10% of newborns require some assistance
- Only 1% need major resuscitative measures (intubation, chest compressions, and/or medications (medication is the last resort and very uncommon only 1 in 1000 will need medication which is epinephrine)

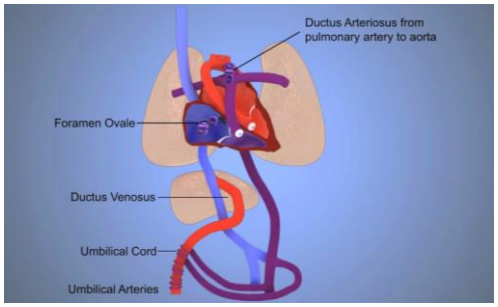
In Utero

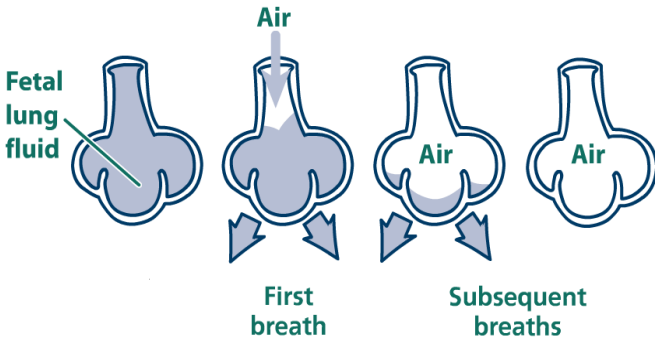
- Alveoli filled with lung fluid
- Pulmonary arterioles constricted (cause only minimal blood needs to go to the lungs)
- Blood flow diverted across ductus arteriosus (passes the blood from the pulmonary artery to descending aorta)
- Pulmonary blood flow diminished (only 10% of blood will go to the lungs)

After Delivery

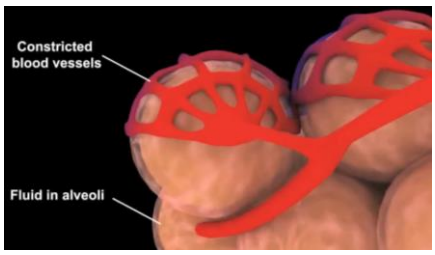
- Lungs expand with air
- Fetal lung fluid leaves alveoli
- Pulmonary arterioles dilate
- Pulmonary blood flow increases
- whereby the lungs take over the lifelong function of respiration

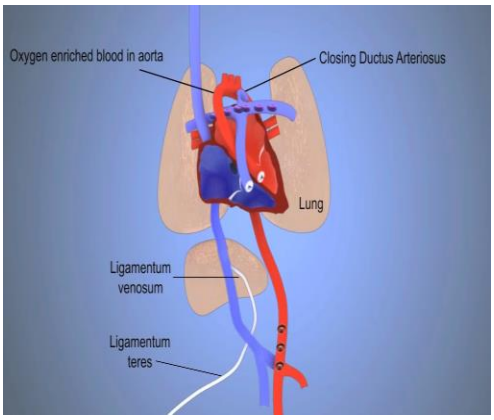






-Transition period: the lungs replace the function of the placenta
-Normal oxygen saturation of fetus is 60% hypoxic environment in utero.





Normal Transition

The following changes take place soon after birth:

- Fluid in alveoli absorbed into lung tissue and replaced by air.
- Umbilical arteries and vein constrict, (removing the low resistance placental circuit), thus increasing blood pressure
- Ductus arteriosus constricts
- Blood vessels in lung relax (increasing pulmonary blood flow)
- Blood flows through lungs to pick up oxygen
- Blood oxygen levels rise (over 5 to 10 minutes to reach normal levels)

Transition Abnormalities

- A baby may encounter difficulty before labor, during labor, or after birth. Some of the problems that may disrupt normal transition
- The baby may not breathe sufficiently to force fluid from the alveoli, or foreign material such as meconium may prevent air from entering the alveoli.
- Excessive blood loss may occur, or there may be inadequate cardiac contractility or bradycardia from hypoxia and ischemia.
- Lack of ventilation of the lungs → sustained constriction of the pulmonary arterioles, preventing systemic arterial blood from being oxygenated (saturation will fall this is called persistent pulmonary hypertension of the newborn)
- Prolonged lack of adequate perfusion and oxygenation to the baby's organs can lead to damage to many organs especially the brain (called hypoxic ischemic encephalopathy or neonatal encephalopathy), or death

Signs of the perinatal compromise

- Poor muscle tone
- Depressed respiratory drive
- Bradycardia(heart rate most important indicator to be observed during resuscitation of the newborn)
- Low blood pressure (usually not measured in the delivery room)
- Tachypnea (as a compensation for the decrease saturation)
- Cyanosis

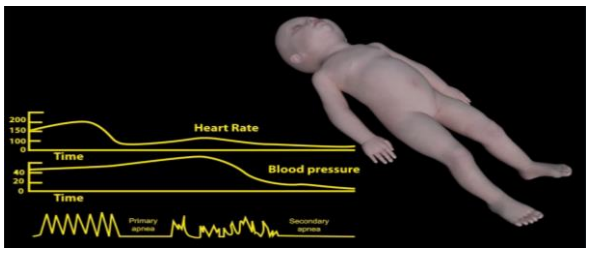
Primary Apnea

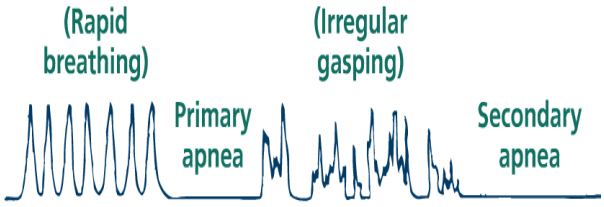
- When a fetus/newborn first becomes deprived of oxygen, an initial period of attempted rapid breathing is followed by primary apnea and dropping heart rate that will improve with tactile stimulation

- When babies are deprived of oxygen (in utero or after delivery), they undergo a well-defined sequence of events that starts with cessation of respiration

Secondary Apnea

- If oxygen deprivation continues,(deep gasping respirations develop), then secondary apnea ensues, accompanied by a continued fall in heart rate and blood pressure
- Secondary apnea cannot be reversed with stimulation (will not restart the baby's breathing); assisted ventilation must be provided (positive-pressure ventilation)





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

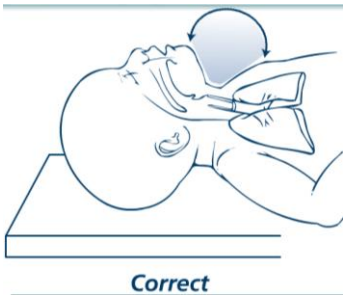
- A – Airways
- B – Breathing
- C – Circulation
- D – Drugs (epinephrine)

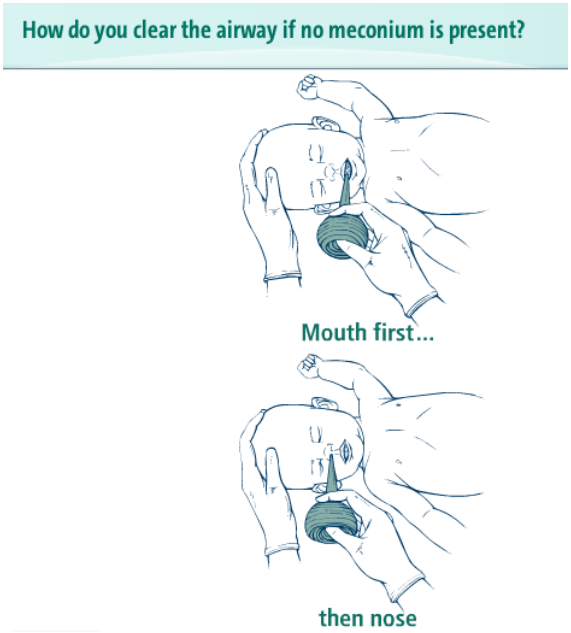
Initial Steps

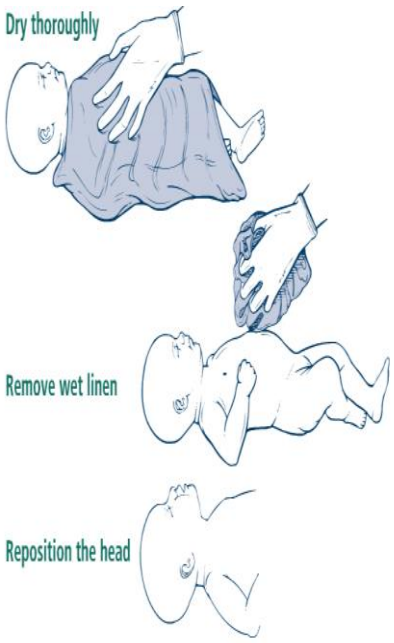
- Provide warmth
- Position; clear airway (as necessary)
- Dry, stimulate, reposition

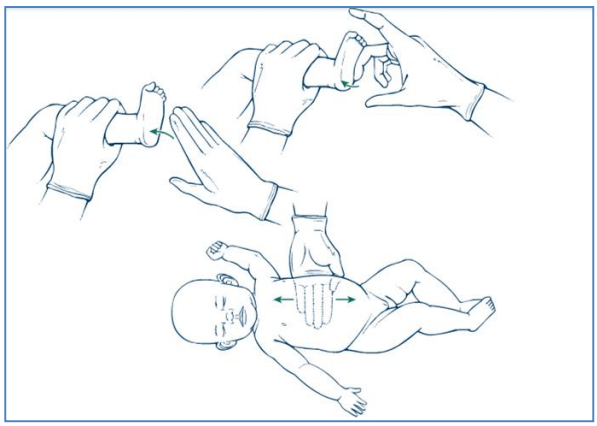
-Position the baby is by putting him in sniffing position (extended neck).

-Clear the airways by suction (suction the mouth before the nose) if necessary. Severe suction can cause bradycardia or respiratory suppression.

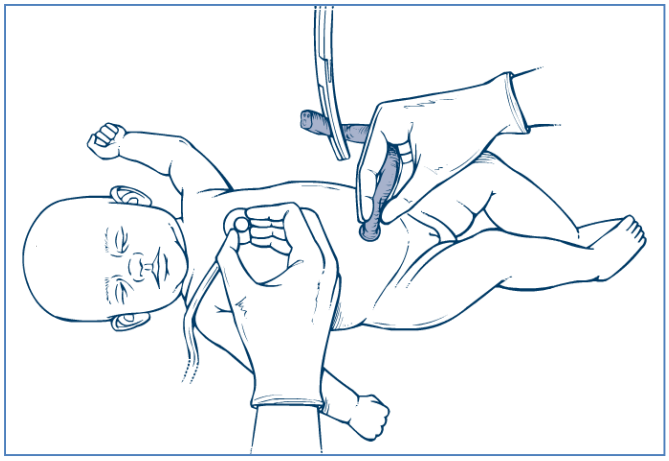








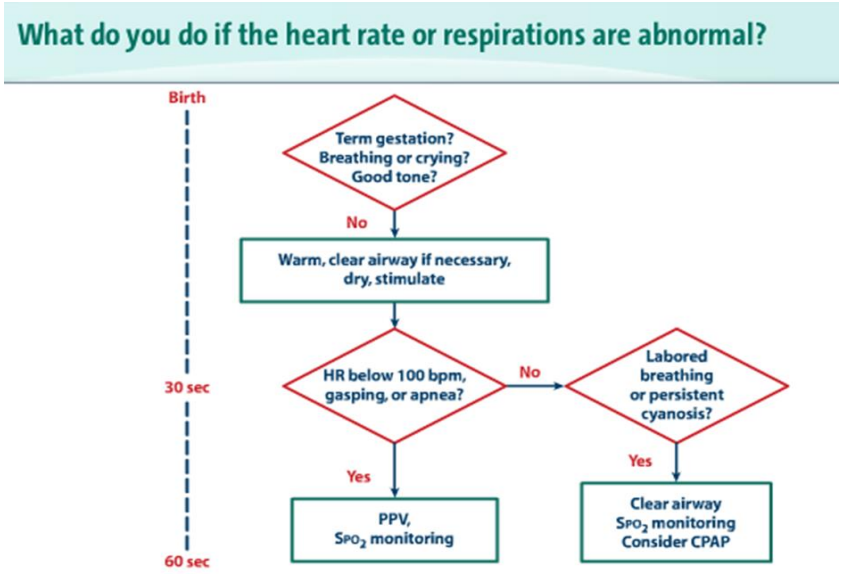
Two accepted ways to stimulate the baby:
1- Flicking the sole of the foot
2-rubing the back



Checking the heart always during resuscitation.
Starting from 2010, calculating the heart rate with statoscope. For 6 seconds then multiply by 10, or by holding the cord and counting the pulses for 6 seconds and multiplying by 10. If the pulse is weak it won't be felt. Better to use stethoscope.

Evaluation **after 30 seconds**

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%

Circulation

- If heart rate <60 bpm despite adequate ventilation for 30 seconds:
    - Provide chest compressions as you continue assisted ventilation(PPV)
    - Consider intubation of the trachea at this point
    - Evaluate again if heart rate <60 bpm proceed to the next step(go to drugs epinephrine)

HR >100 bpm no need PPV
HR <100bpm indication for PPV
HR <60 indication to add chest compression to PPV

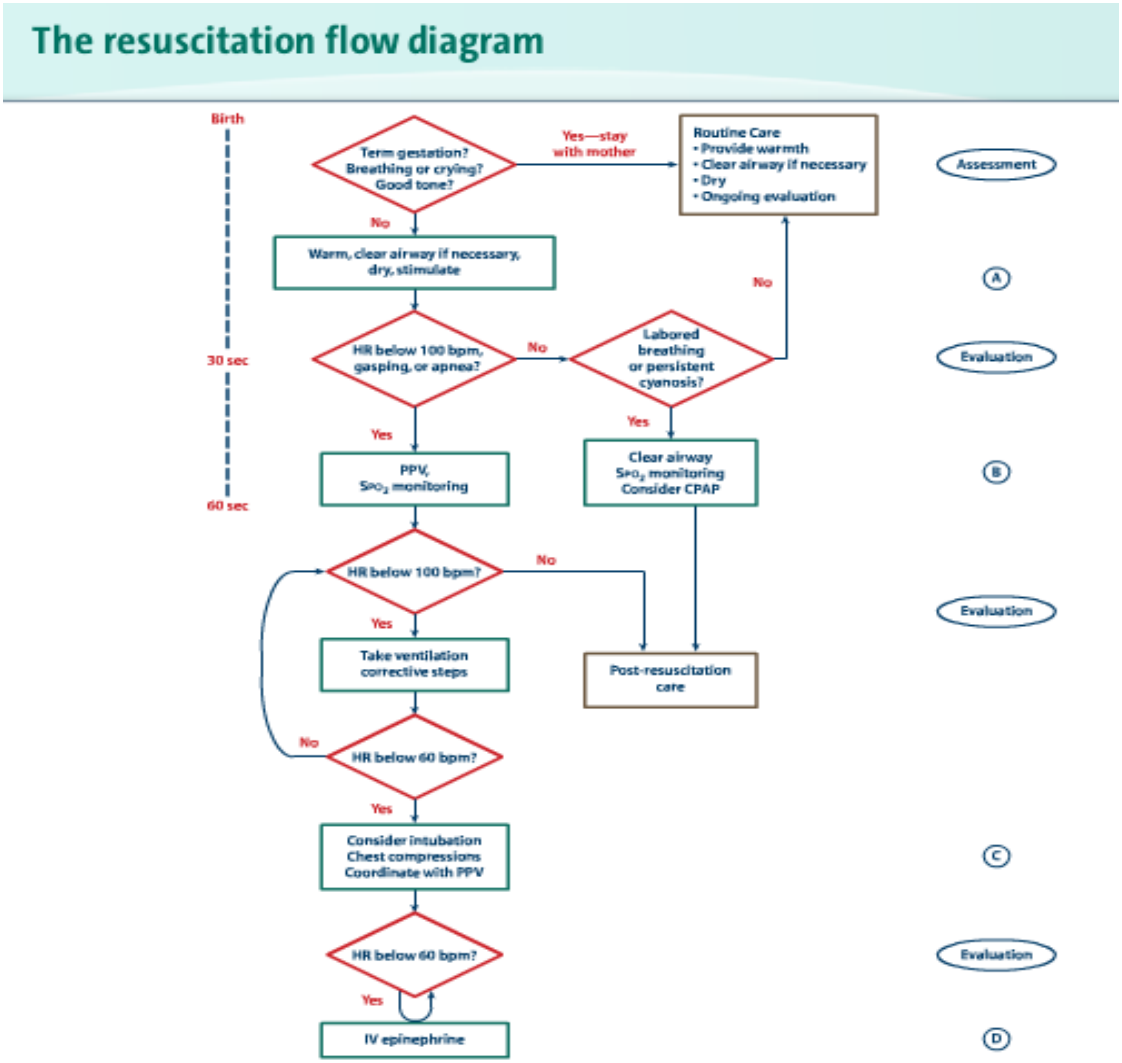
Drugs

- If heart rate <60 bpm despite adequate ventilation and chest compressions:
    - 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 compress-ions and epinephrine may be necessary
- HR <60 bpm → Additional steps needed (chest compression and/or drugs)

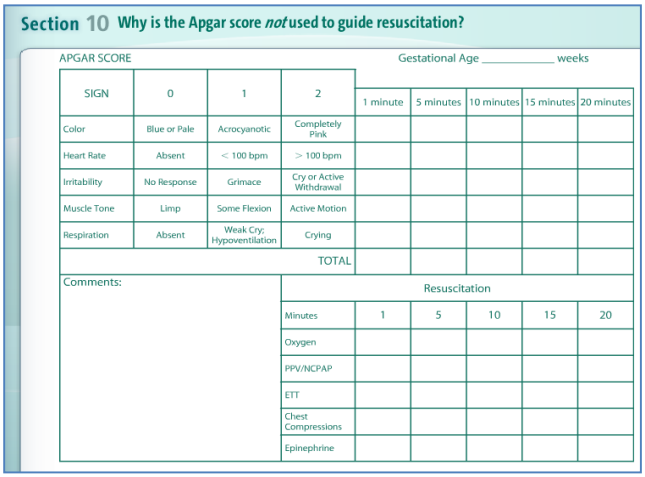
- HR >60 bpm → Chest compressions can be stopped
- HR >100 bpm and breathing → PPV can be stopped
- Always proceed to the next step if no improvement after 30 seconds



If after 10 minutes of resuscitation the baby does not respond, resuscitation is stopped and announce death.

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 muscle 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					

-Score ranging from 0 to 10. Each parameter will take a point of 0, 1, or 2.
-Normal score is more than 7,
-If less than 7 we do extensive APGAR score (will need resuscitation.)
-It is done at 1 minute then repeated at 5 minutes. If abnormal score we repeat it every 5 minutes.
-Should mention what has been done to the baby if resuscitated.

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:
    - Birthweight > 90th percentile for gestational age ***or***
    - Greater than 4,000 g
  - More in IDMs (15% - 45%) vs. normal infants (8% to 14%)

- Growth chart will measure the head circumference, the weight and the length of baby. Most of newborns are within the 50th percentile, the normal is from 10 to 90.
- Less than 10 centile is small for gestational age (weight of baby more than 2 standard deviation below the average gestational age.)
- More than 90 centile is large for gestational age (more than 2 standard deviation above the standard gestational age).
  - 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
  - Macrosomia is a risk factor for intrapartum injury (shoulder dystocia, Erb's palsy asphyxia) and for cesarean delivery

The preterm infant:

Prematurity (before 37 weeks):

- **By gestational age**
  - Early preterm – 23 to 33 6/7.
  - Late preterm – 34 to 36 6/7.

- **By weight**

  - ELBW (extreme low birth weight) → < 1000g.
  - VLBW (very low birth weight) → < 1500g.
  - LBW (low birth weight) → < 2500g.

Prematurity Causes:

- **Maternal**

  - Chronic illnesses.
  - Uterine anomalies (small or malformation or mass).
  - Others.

- **Placental**

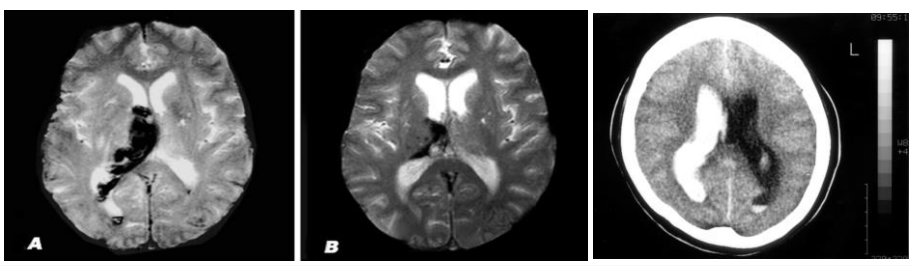
  - Insufficiency.
  - Hemorrhage.

- **Fetal**

  - Infections (TORCH infection).
  - Genetic and chromosomal (most serious and kill the fetus).

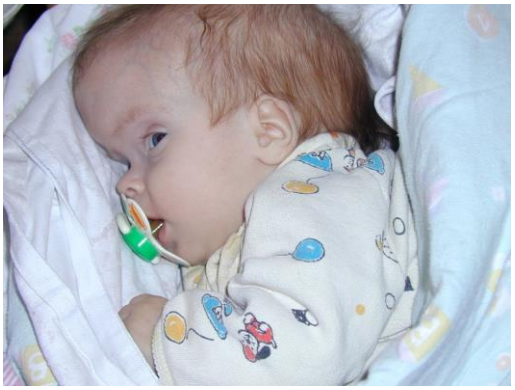
Why Are The Preterm At Risk?

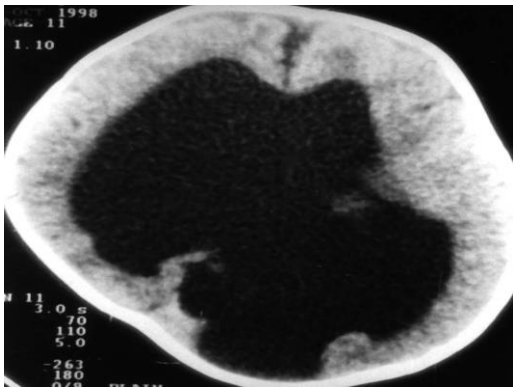
- Surfactant deficiency (lungs are not functioning).
- Poor temperature control (can lead to hypoglycemia, acidosis).
- Decreased respiratory drive.
- Weak muscles make spontaneous breathing difficult.
- Oxygen toxicity (sensitive to oxygen, during resuscitation we should not push the O_2 more than 90%, if more than 95% may cause damage to the lungs, to the brain, and the most serious is retinopathy of prematurity (ROP)).
- Possible infection (due to decrease immunity).
- Susceptibility to IVH (spontaneous intraventricular hemorrhage is increased with birth weight of less than 1.5 kg, the lower the weight the higher the risk).
- Susceptibility to hypovolemia due to blood loss (cause preterm neonates have low blood volume).



Intraventricular hemorrhage (IVH):

- ***The blood vessels are very fragile (germinal matrix) → increased risk of bleeding.***
- ***The germinal matrix is formed early during embryogenesis and is the site of glial and neuronal differentiation. From here cells migrate peripherally to form the brain. It is densely cellular and vascular. The blood vessels of the germinal matrix are weak walled and predisposed to haemorrhage. A significant stress experienced by a premature infant after birth may cause these vessels to rupture. The bleeding occurs initially in the periventricular areas causing a periventricular haemorrhage (PVH). If this bleeding persists, the expanding volume of blood dissects into the adjacent lateral ventricles leading to an intraventricular haemorrhage (IVH).***





Hydrocephalus: ***as a complication of IVH.***

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

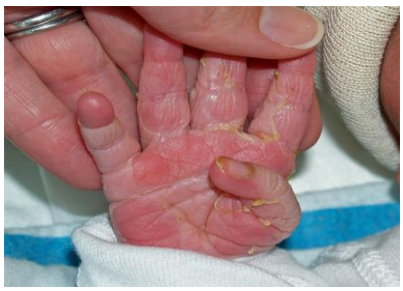
The Morbidity of Extreme Prematurity:

- **Risk of all significant morbidities relate to degree of prematurity At 23 weeks**
  - BPD (bronchopulmonary dysplasia, chronic disease of the lungs and can persist for years) 50-70%.
  - White Matter Injury (periventricular leukomalacia) 32%.
  - NEC (necrotizing enterocolitis) 9%.

Dysmaturity vs. Prematurity:

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 (IUGR).
- The failure of an organism to develop, ripen, or otherwise achieve maturity in structure or function.
- The condition of a fetus or newborn that is abnormally small or large for its age of gestation. Kinds of dysmaturity are small for gestational age and large for gestational age.



Postterm 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.

  - **If the newborn is below 10th percentile can be IUGR or SGA. The difference:**

IUGR	SGA
Can be more than 10th percentile.	Must be below 10th percentile.
Most likely, there is a disease affecting the baby either: maternal, fetal or placental cause. At first, the fetus is growing then with serial US there'll be a gradual decrease in the percentile. This decrease can be minimal (above the 10th percentile) or drastic (below 10th percentile).	Postnatal terminology. There is no disease in the mother, fetus, or placenta. It is normal.
Baby appears as an old man.	---

IUGR complications:

- **Increased risk of perinatal complications**
  - Perinatal asphyxia.
  - Cold stress.
  - Hyperviscosity (polycythemia).
  - Hypoglycemia.

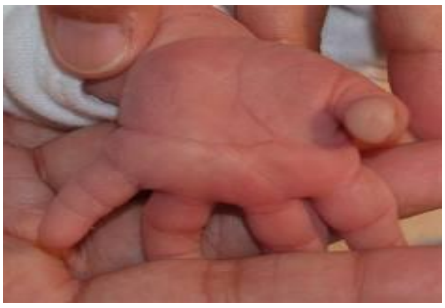
Outcomes of IUGR infants:

- 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, Cerebral Palsy or Mental Retardation) that is similar to AGA (appropriate for gestational age) preterm of the same size.

Congenital Malformations: check the birth defects part in the summary

Congenital: The presence of the defect at birth. Inborn errors of morphogenesis can be classified based on the developmental stage during which the alteration occurs, the process causing the change, and the end result. The four categories of morpho-genic errors are malformation, deformation, disruption and dysplasia.

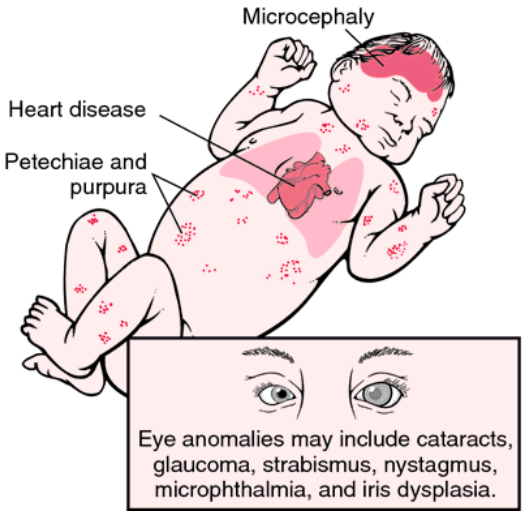
- **Major (2% to 3% of live born infants):**
  - Medical and social consequences (cleft palate and neural tube defects).
- **Minor (Up to 15%):**
  - No significant health or social burden (epicanthal folds and a single palmar crease).
- **Normal phenotypic variants:**
  - Physical differences occurring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



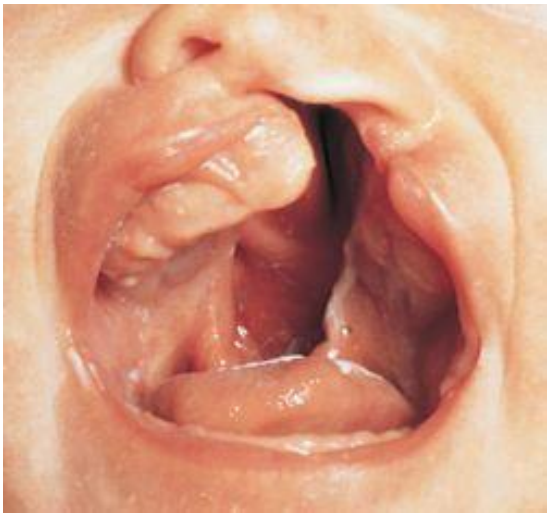
Simian crease: ***Can occur in normal population, but occur more frequently with trisomies such as 13, 18 and 21 (Down syndrome).***

Malformations:

- Abnormal processes during the initial formation of a structure (first 3 months of pregnancy organogenesis).
- **May result in:**
  - Faulty configuration (transposition of the great vessels).
  - Incomplete formation (cleft palate).
  - Agenesis (absence of radius) thrombocytopenia absent radius syndrome.
- **May be the result of:**
  - Genetic - chromosomal (10%) and single gene defects (4%).
  - Environmental insults (teratogens):
    - Drugs – thalidomide (cause short or absence of the limbs).
    - Congenitally acquired viruses – Rubella.
- **Multifactorial in 25%, unknown in 40%-45%.**



Congenital Rubella



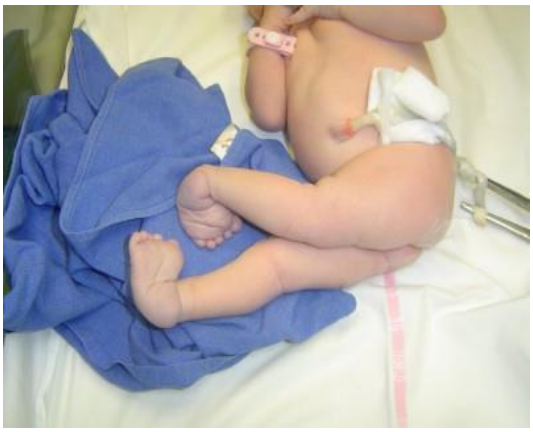
Malformations - Cleft lip and palate

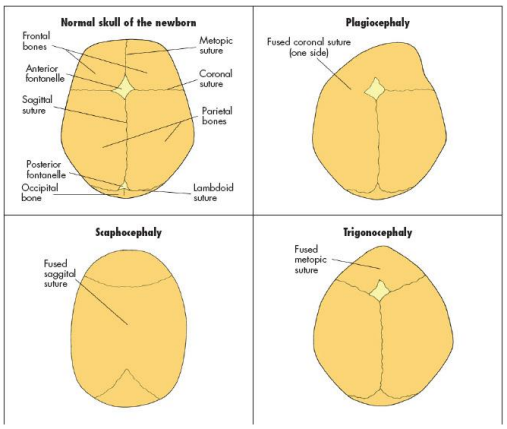
Deformations:

- 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 (deformity of the skull due to premature closure of the cranial sutures) resulting from in utero constraint.

- Typically improve postnatally.
- Resolution depends on the duration of the abnormal forces and the extent of subsequent growth.

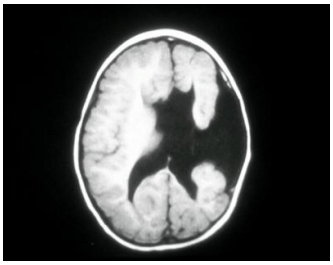
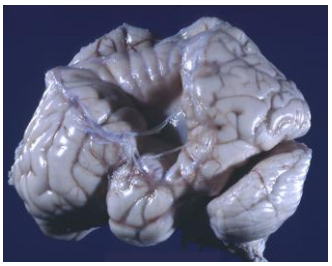
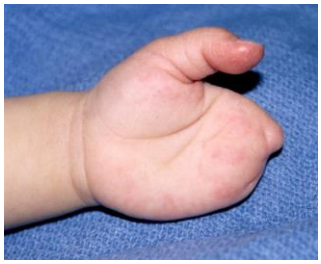




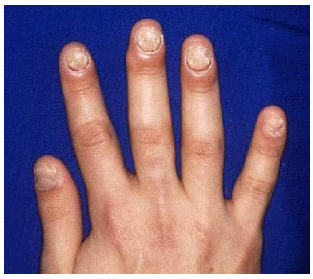
Disruptions:

- 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.


Dysplasia:




Ectodermal dysplasia.

- Abnormal cellular organization or function.
- Typically affects a single tissue type.
- Examples:
  - Hamartomas, ectodermal dysplasia, and skeletal dysplasias.

Multiple malformations:

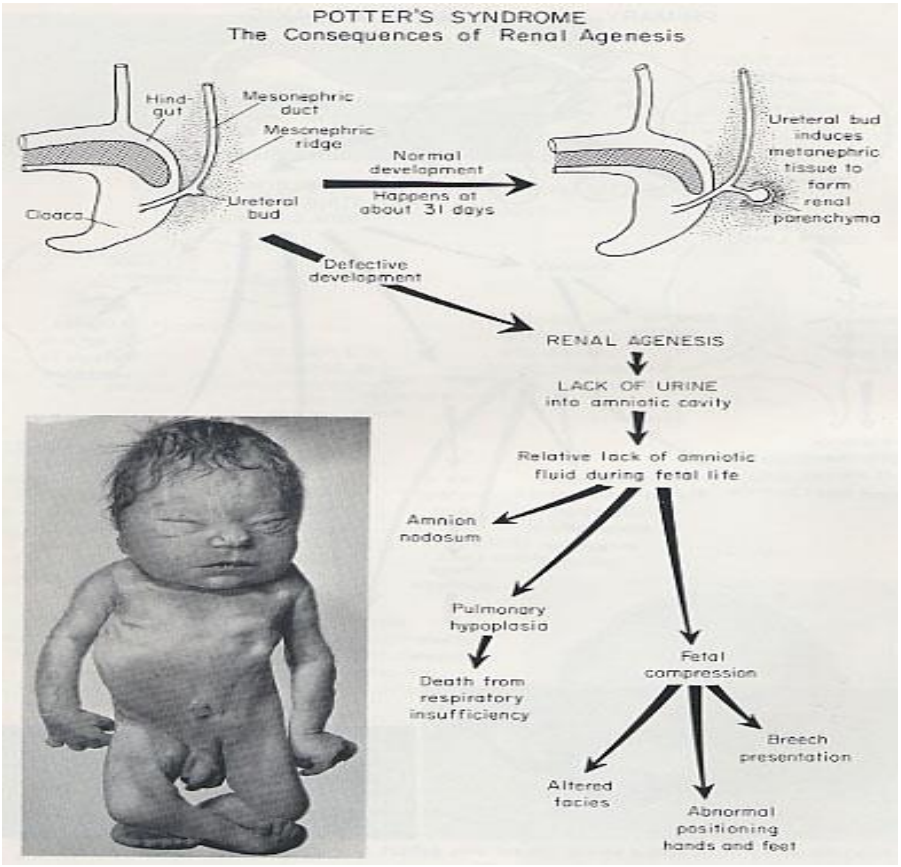
- 0.7% of live births.
- Sequence vs. Syndrome.

A malformation sequence:

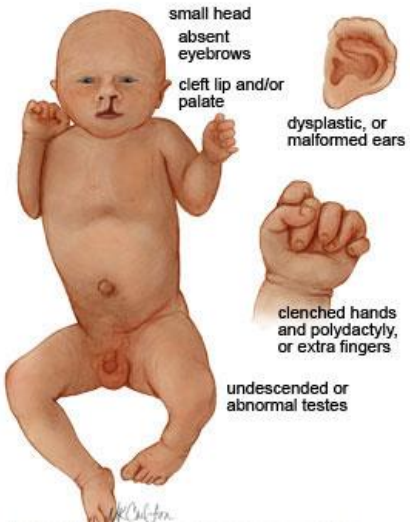
- All of the anomalies can be explained on the basis of a single problem.
- Examples:
  - Oligohydramnios sequence (Potter syndrome): The fetus has a renal problem causing oligohydramnios, which will lead to deformities in upper and lower limbs, a small head and chest with hypoplastic lungs.
  - Pierre Robin sequence: Pierre Robin syndrome (or sequence) is a condition present at birth, in which the infant has a smaller-than-normal lower jaw, a tongue that falls back in the throat, and difficulty breathing.

A malformation syndrome:

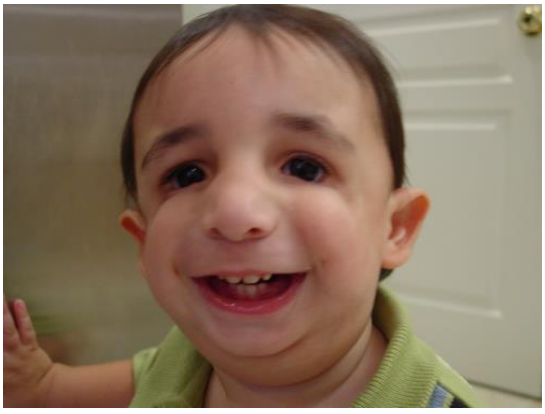
- Multiple structural defects that are NOT explained on the basis of a single initiating defect but share a cause (chromosomal abnormalities, mutant gene disorders, or environmental teratogens).



Trisomy 18 (Edward's Syndrome)



Trisomy 13 (Patau Syndrome)



Treacher Collins Syndrome

Or mandibulofacial dysostosis: is a rare autosomal dominant congenital disorder characterized by craniofacial deformities. The typical physical features include: absent cheekbones, downward-slanting eyes, micrognathia (a small lower jaw), conductive hearing loss, underdeveloped zygoma, drooping part of the lateral lower eyelids, and malformed or absent ears.

Summary

- **Primary Apnea will improve with tactile stimulation**
- **If oxygen deprivation continues then Secondary apnea will develop and assisted ventilation must be provided (positive-pressure ventilation)**

HR >100 bpm no need PPV

HR <100bpm indication for PPV

HR <60 indication to add chest compression to PPV if continued then administer epinephrine

- **Parameters of Apgar score:**

Color, HR, RR, reflexes and muscle tone

- **Macrosomia is a risk factor for intrapartum injury (shoulder dystocia, Erb's palsy asphyxia) and for cesarean delivery**

- **If the newborn is below 10th percentile it can be IUGR or SGA. The difference, IUGR can be more than 10th percentile and most probably there is a pathological cause for it. While SGR must be below 10th percentile but the baby is totally normal with no pathological cause, not in the mother nor the placenta or even in the fetus .**

  Birth Defects (further explanation)
  - **Major are often defined as anomalies or malformations that create significant medical problems for the patient or that require specific surgical or medical management. Major anomalies or malformations generally are not considered a variation of the normal spectrum, minor and normal phenotypical variants .**
  - **Minor anomalies are often described as features that vary from those that are most commonly seen in the normal population but that, in and of themselves, do not cause increased morbidity.**
  - **both major and minor anomalies may be associated with particular syndromes or, in many cases, may be an isolated finding in an otherwise healthy individual.**

When considering dysmorphic features it is also important to keep in mind the various ways in which structures and tissues may become abnormal:

Deformation:

- **For instance a structure may be visibly abnormal due to a deformation. A deformation is caused by an abnormal external force on the fetus during in utero development that resulted in abnormal growth or formation of the fetal structure. For example, in fetuses that grow in a uterine environment where not enough amniotic fluid is present (oligohydramnios) as described above when discussing Potter sequence, the fetus may have a flattened face due to compression of the face against the uterine wall with no room for significant movement and full development of the face or facial features.**

Disruption:

- **Another type of abnormality is known as a disruption where a fetal structure is growing normally and then growth is arrested due to something which disrupts the process. This is seen in the condition of amniotic bands where a digit or extremity may be growing normally but then growth is disrupted or discontinued due to the development of amniotic band at the end of that extremity. This may result in missing fingers, toes, or hands and feet. Oftentimes disruptions and deformations are relatively isolated and not associated with multiple congenital anomalies.**

Malformation:

- **A malformation signifies that fetal growth and development did not proceed normally due to underlying genetic, epigenetic, or environmental factors that altered the development a particular structure.**

Dysplasia:

- **Another type of generalized anomaly is related to an underlying tissue dysplasia where the intrinsic cellular architecture of a tissue is not normally maintained throughout growth and development. Many of the skeletal syndromes of short stature are due to dysplasia in the developing bone and cartilage.**

A syndrome is generally recognized and defined as a well-characterized constellation of major and minor anomalies that occur together in a predictable fashion presumably due to a single underlying etiology which may be monogenic, chromosomal, mitochondrial, or teratogenic in origin. For instance Down Syndrome trisomy 21, (a numeric chromosome disorder - 47 XX or XY, +21) is a syndrome associated with a predictable constellation of major and minor anomalies that create a recognizable phenotype that allowing people who have seen other individuals with trisomy 21 to immediately suspect the diagnosis when they see another individual with the same condition, even though all of the characteristic anomalies (or features) are generally not present in any one affected individual.

A sequence is a group of related anomalies that generally stem from a single initial major anomaly that alters the development of other surrounding or related tissues or structures. Potter's sequence is recognized by a constellation of physical findings where the outward appearance of the newborn is often characterized by flattened abnormal facial features and deformations of the hands and feet. These features along with poor lung development are secondary to decreased amounts of amniotic fluid (oligohydramnios) which is most often due to major renal (kidney) abnormalities (eg a single major anomaly) associated with decreased fetal urine output.

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