Neonatology

إعداد:

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Aim of this lecture

- By the end of this lecture students will learn:
  1- The normal newborn baby and how to deal with, and the most common diseases and problems of neonate.

2- The preterm baby and the most common problems may have

3- The most common respiratory problems affecting neonate

Neonatal period: starts from birth till 28 days of life.

Full term newborn: a baby born after 37 weeks of gestation.

Preterm(premature) baby: a live born infant before 37 weeks of gestation.

Postmature baby: a baby born after 42 weeks of gestation.

Low birth weight infant(LBW): infant having birth weight less than 2500g.

Very low birth weight infant(VLBW): infant having less than 1500g at birth.

Assessment of newborn baby

- **History taking:** antenatal(primary health care visit, medical illness, infection, drug exposure, radiation, early rupture membrane) and natal history(gestational age, onset of respiration, birth weight), also maternal history of still birth, recurrent abortion.

- **Physical examination:**
 to look for various manifestations of neonatal disease, and to look for any congenital malformations.

Appearance:

- Look for cyanosis, nasal flaring, grunting and intercostal retraction suggest pulmonary disease.
- Meconium stained umbilical cord, nails and skin, may suggest meconium aspiration.

Vital signs and growth measures:

HR: normal 120-160 beats/min

RR: normal 30-60 cycles/min

Temp. :per rectum or axillary route.

Length: normal 50cm

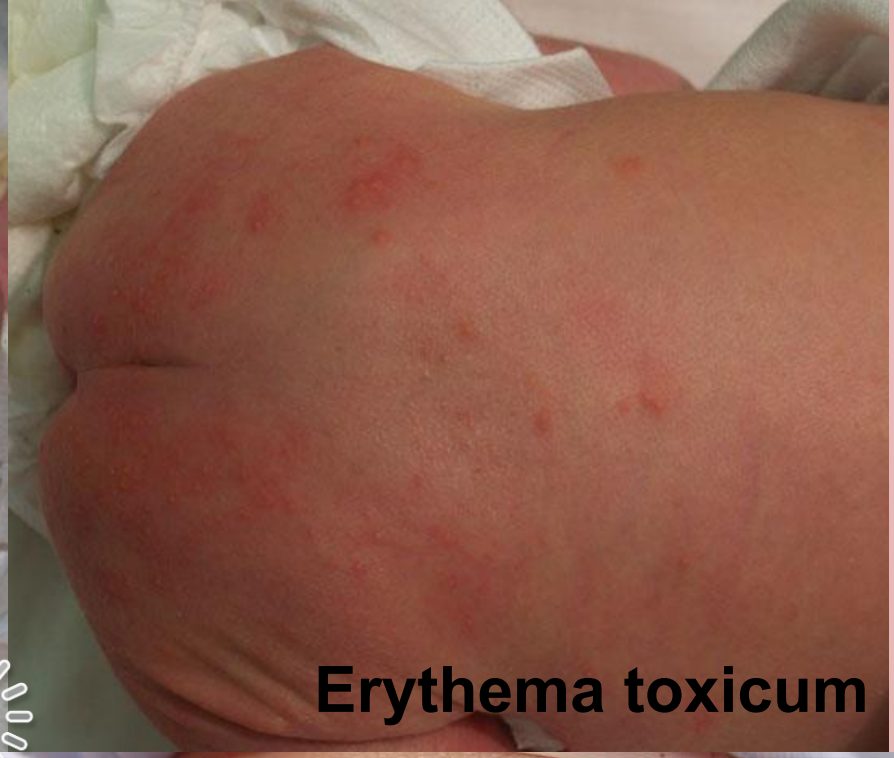
Weight: normal 3-3.5 (4.5) kg

Head circumference: normal 35cm

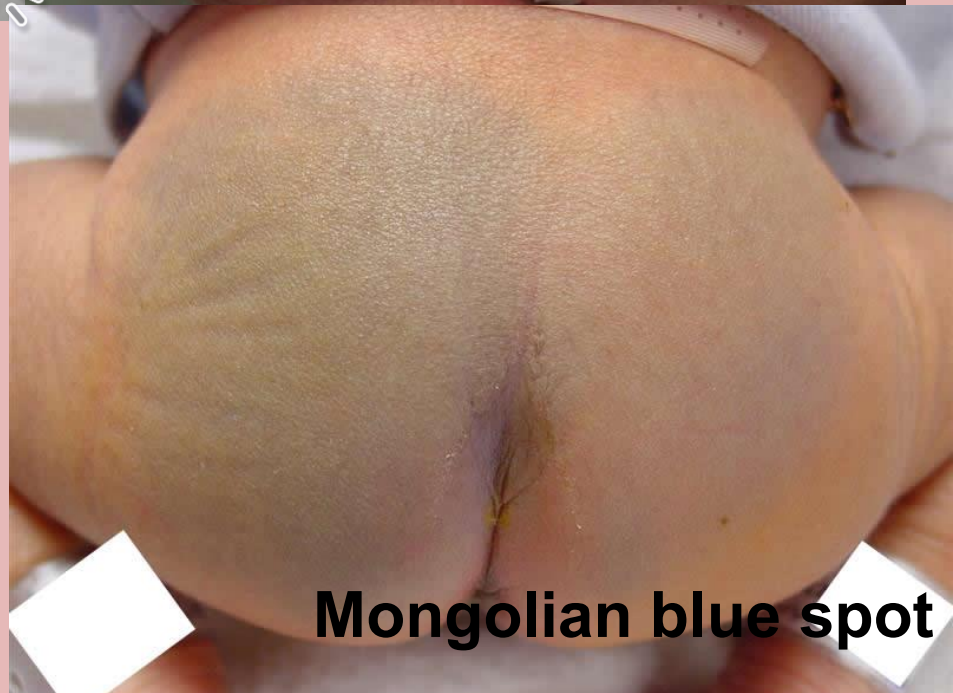
Skin examination:

- **Look for pallor, plethora, jaundice, cyanosis, petechiae, congenital nevi and rash.**
- **Acrocyanosis (of the feet and hands) which is normal in full term and premature infants in the first day after delivery.**
- **Vernix caseosa, a soft white cream layer covering the skin of term infants (not present in preterm).**
- **Mongolian blue spots are transient, dark blue pigmented macules over the lower back and buttocks, usually disappear by one year of age.**
- **Erythema toxicum: is an erythematous papular-vesicular rash are common in the neonate during the first week of life, it contains eosinophil inside, disappear spontaneously.**


Acrocyanosis


Erythema toxicum


Vernix caseosa


Mongolian blue spot

•**Milia**, is a yellow-white epidermal cysts of the pilosebacious follicles that are noted on the nose.

•Capillary and cavernous **hemangiomas** may be seen and usually resolves 1-4 years of age.

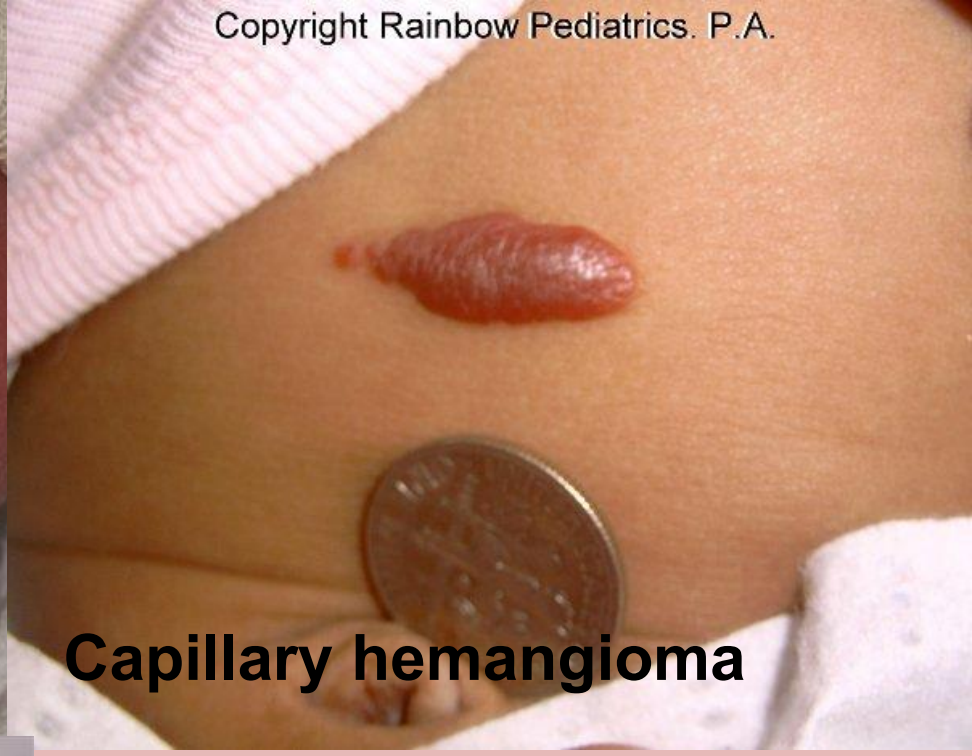
•**Portwine stain** that is seen on the face should consider **Sturge Weber syndrome**: convulsions, mental sub-normality, and ipsilateral intracranial calcification.

•Mild edema may be present in premature infants but may suggest hydrops fetalis, hypoalbuminemia, or Turner syndrome.

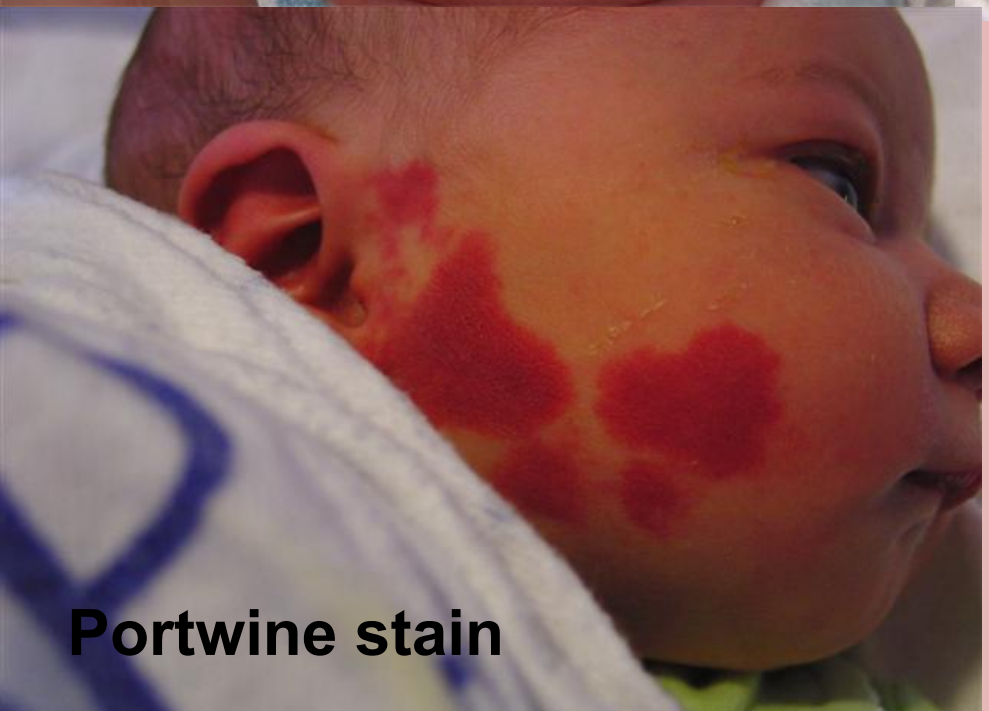
•**Sclerema**: it is a form of hardening of the skin and subcutaneous tissues, it is a sign of septicemia.

•**Hair tufts** over the lumbosacral spines suggest spinal cord defects.

Milia



Capillary hemangioma



Portwine stain

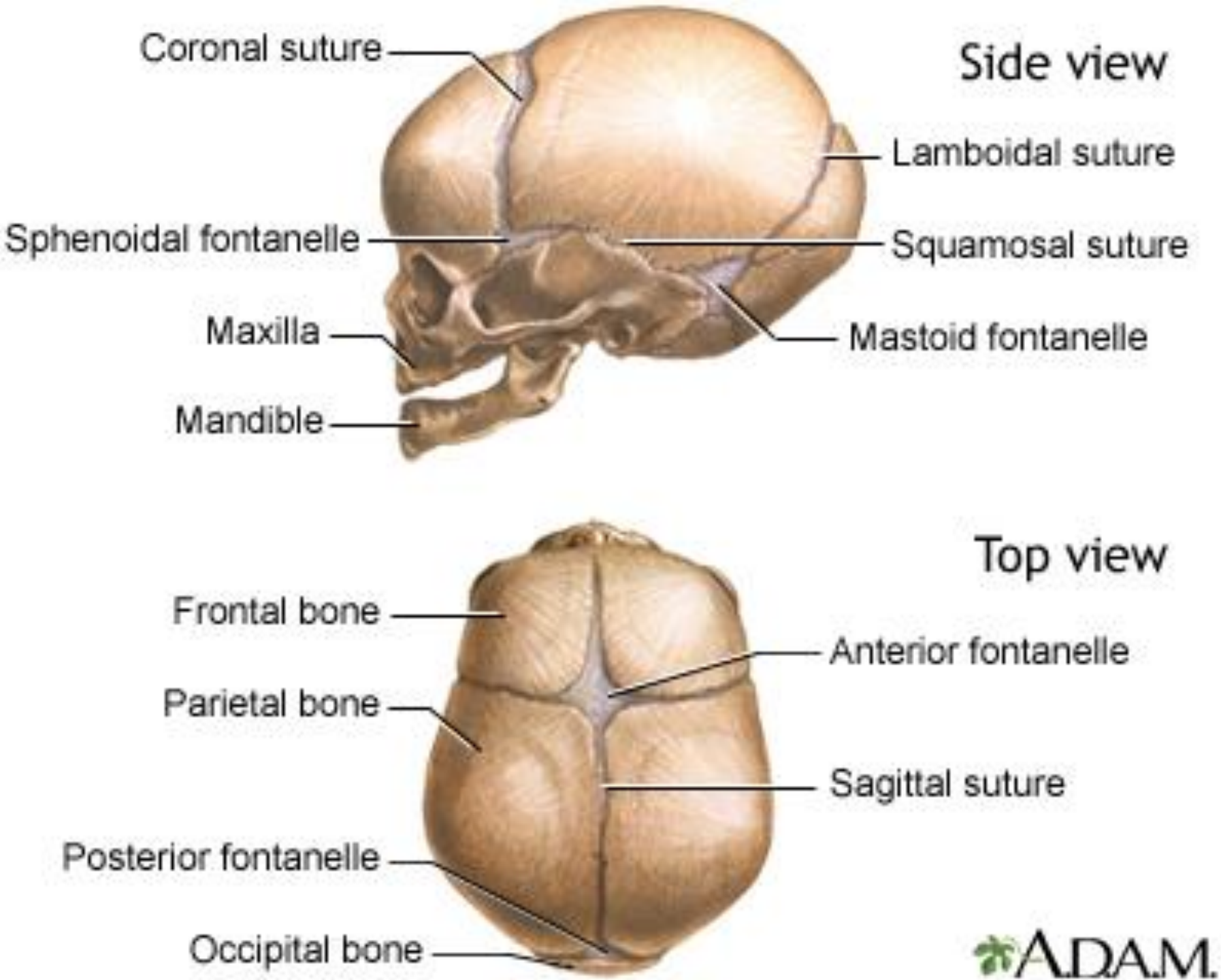
Skull:

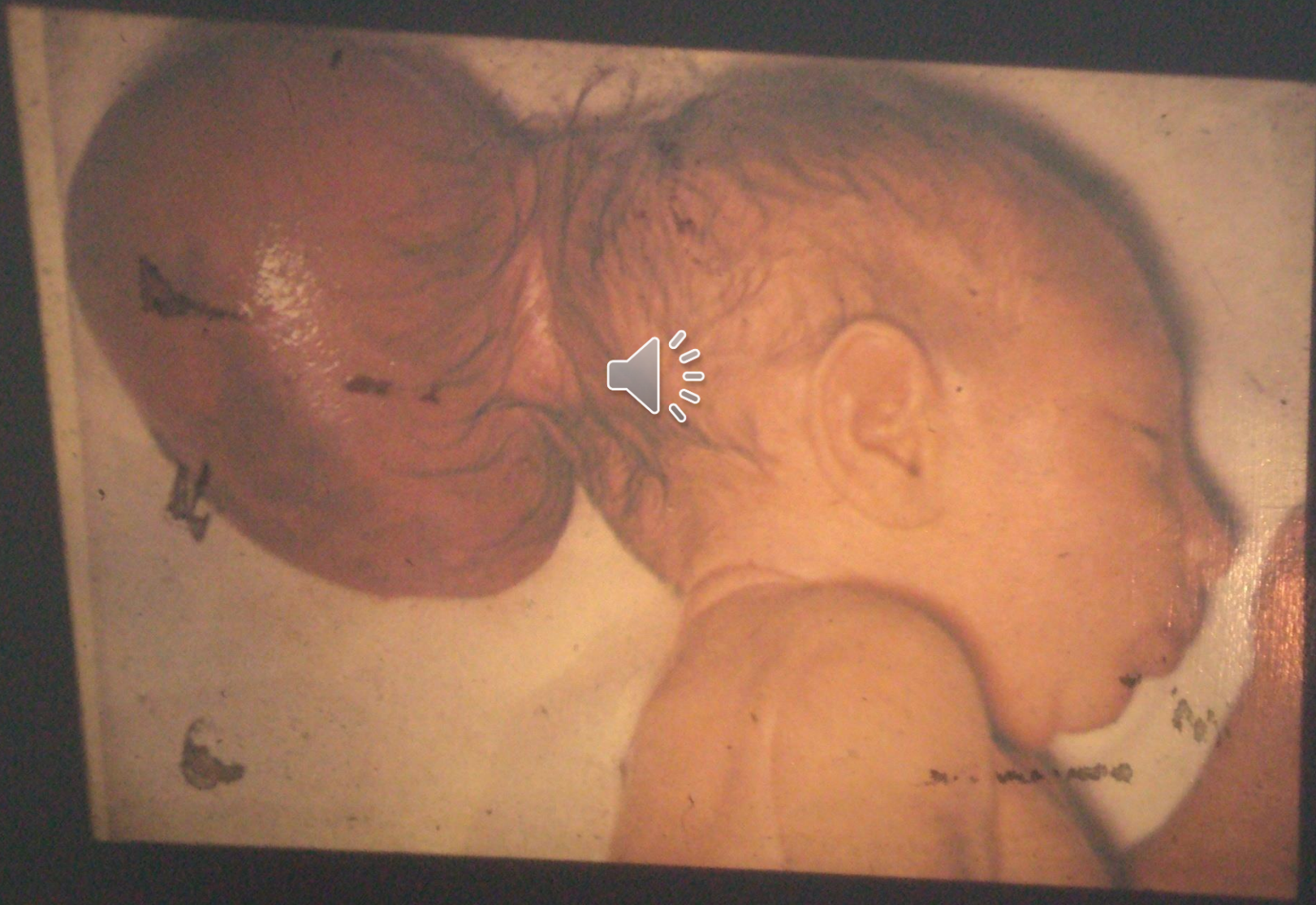
•Look for premature fusion of the cranial sutures (**craniostenosis).**

•**Size** of the fontanels: posterior is small less than 1 cm, anterior is diamond shape measures 1.5x2 cm

•**Causes of large(wide) fontanels**: achondroplasia, congenital hypothyroidism, congenital infection, hydrocephaly, osteogenesis imperfecta.

•Also look for **encephalocele**, **cephalhematoma, caput succedaneum.**


Coronal suture
Side view
Lamboidal suture
Sphenoidal fontanelle
Squamosal suture
Maxilla
Mastoid fontanelle
Mandible
Top view
Frontal bone
Anterior fontanelle
Parietal bone
Sagittal suture
Posterior fontanelle
Occipital bone
A.D.A.M.



Face, eyes and mouth:

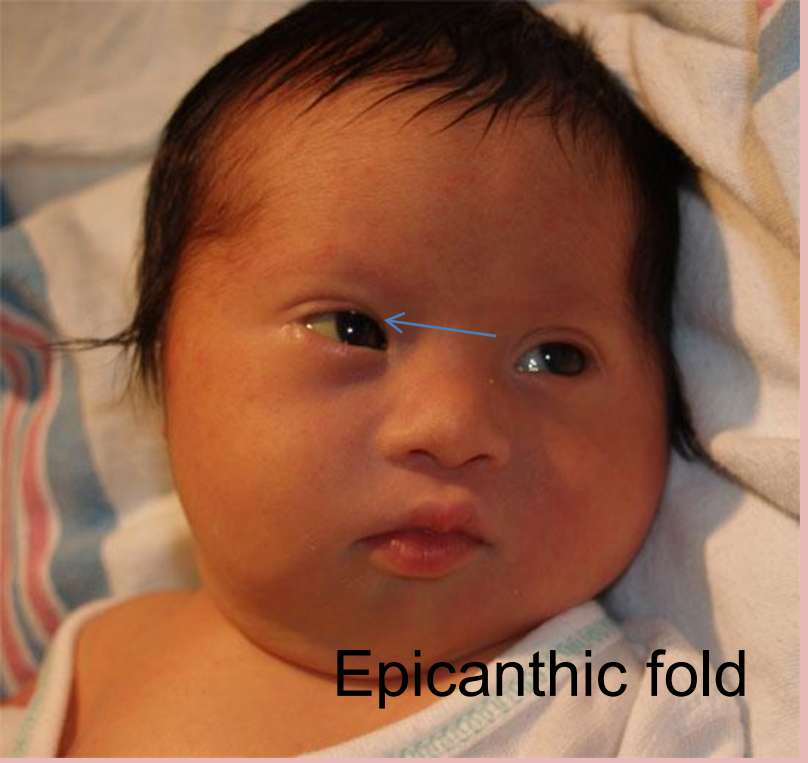
•**Look for dysmorphic feature:**
Epicanthic folds (Down syndrome), low set ears, cleft lip and palate, microphthalmia, cataract (congenital infections or chromosomal disorders)

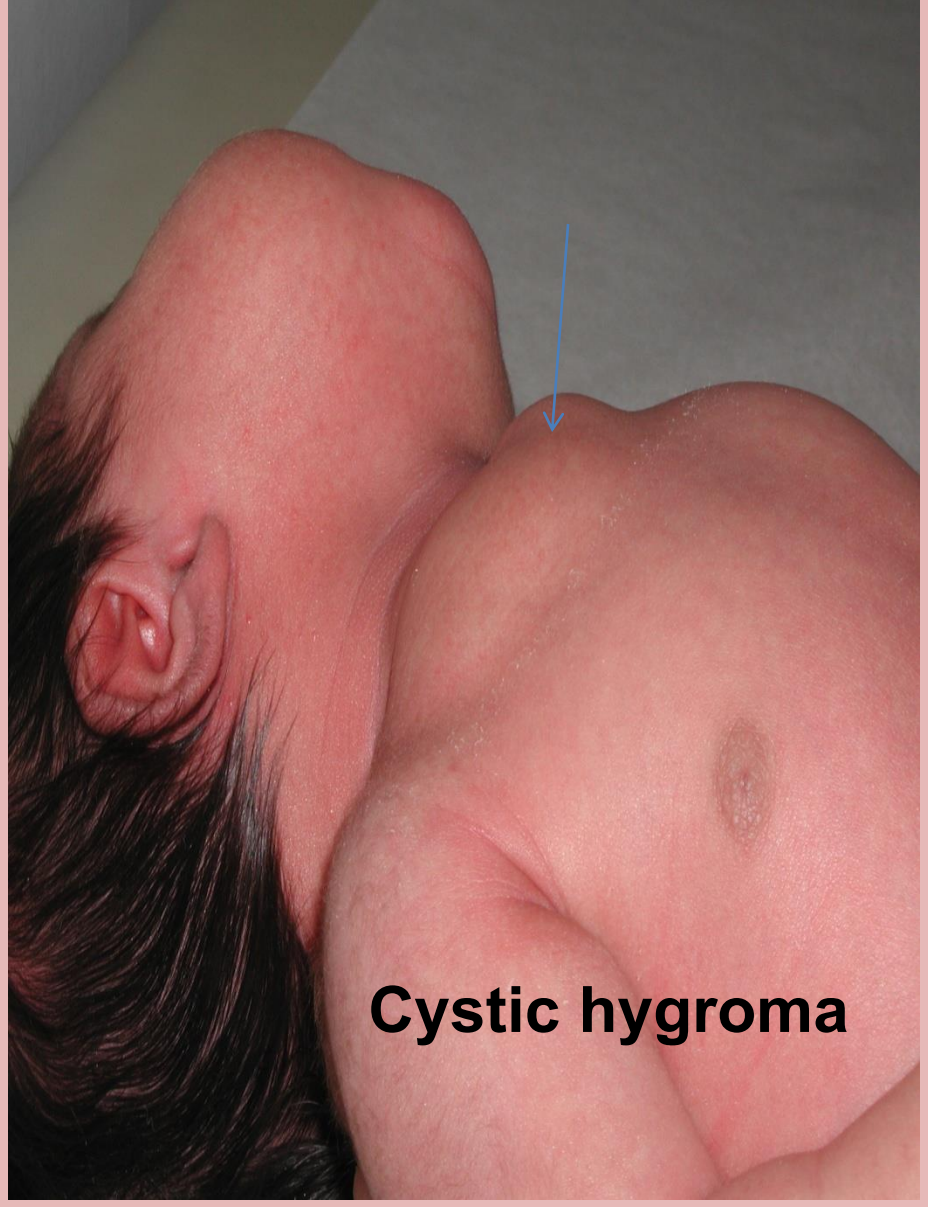
•White pupillary reflex may indicates cataract, ocular tumors, severe chorioretinitis, and retinopathy of prematurity.

Neck and chest:

•***Periodic breathing*** is a normal pattern of breathing in newborn, characterized by periods of rapid breathing followed by pause of less than 10 seconds then he resume breathing again.

•Look for **cystic hygroma** or **sternomastoid tumor** in the neck, fracture clavicle.


Epicanthic fold


Cystic hygroma

•Breast abscess or engorgement.
•Webbing of the neck, widely spaced nipples in Turner syndrome.


Webbing of neck

Cardiovascular examination:

•Listen for murmurs(congenital heart disease) or patent ductus arteriosus.
•Examination of peripheral pulses and look for femoral-radial delay(coarctation of aorta)

Abdomen:

- Liver is usually palpable 1-2cm below costal margin.
- Feel for any abdominal mass(hydronephrosis, neuroblastoma).

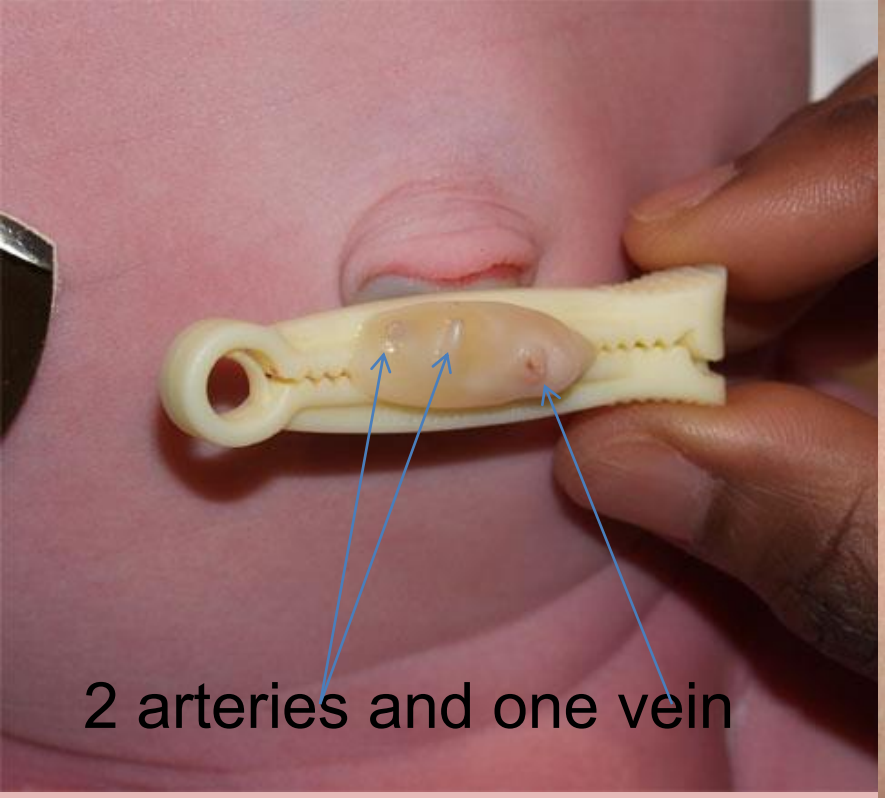
- Abdominal distension may suggest meconium ileus, ileal atresia, imperforate anus.

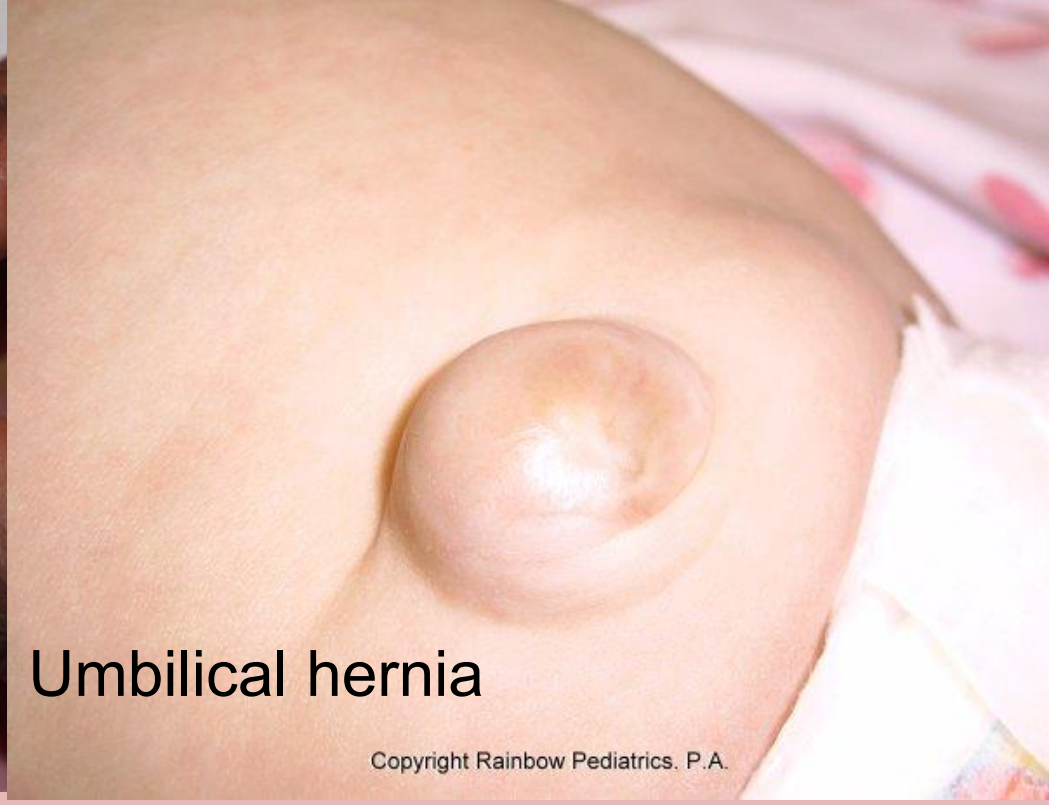
- Meconium stool should pass within the first 48 hr of birth.

- Urine should normally pass within the first 24 hr of birth.

- Umbilical stump should contain 2 arteries and one vein.

- Look for **umbilical hernia**, **omphalocele**(herniation of abdominal content through the umbilical cord)


2 arteries and one vein


Umbilical hernia
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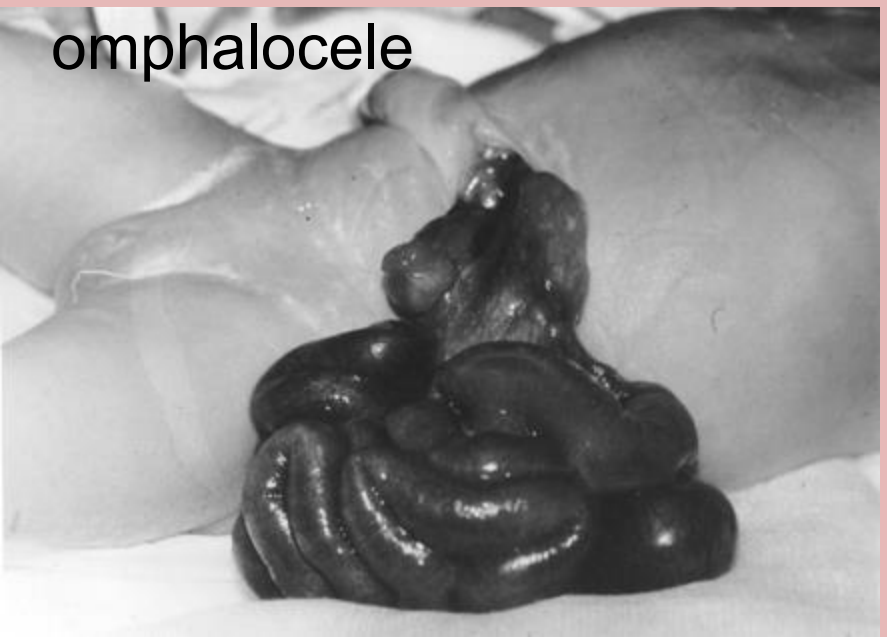
omphalocele

Umbilical granulomas

- when they occur, typically develop after the first week of life. Composed of excess granulation tissue at the base of the umbilical cord.
- The granulomas, which range from 3-10 mm in size, are pink or dark pink in color and have a soft, velvety texture. Because most of these granulomas fail to epithelialize, they are associated with persistent inflammation accompanied by serous or serosanguinous drainage and a tendency for easy bleeding with trauma.
- Treatment options include topical applications of silver nitrate, excision and application of absorbable hemostatic materials, cryosurgery.
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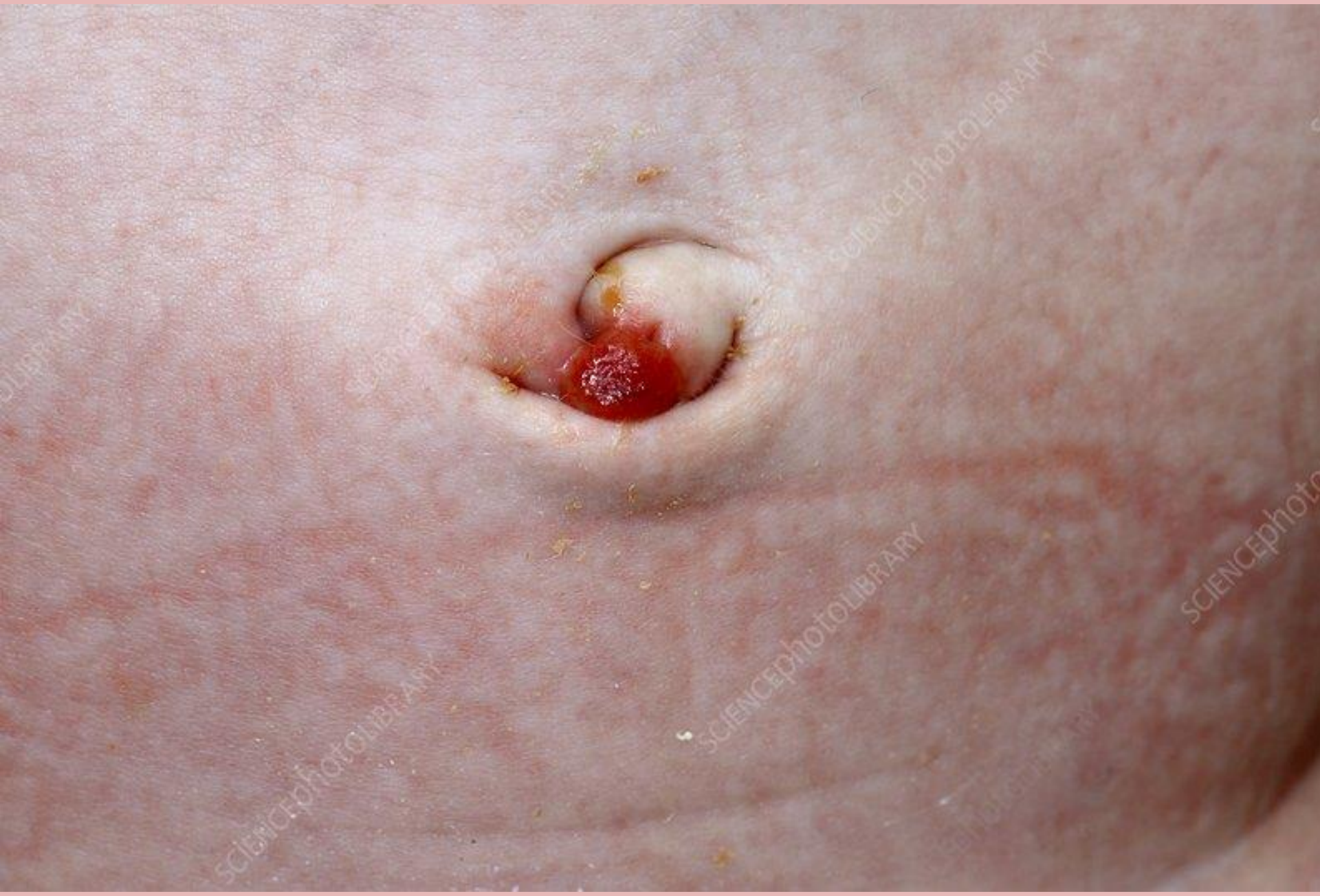
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Umbilical hernias

- are common in the neonatal period and represent a central fascial gap beneath the umbilicus through which abdominal contents may protrude, covered by skin.
- This gap is a consequence of delayed contraction of the encircling fibromuscular umbilical ring.

Umbilical hernias are more common in:

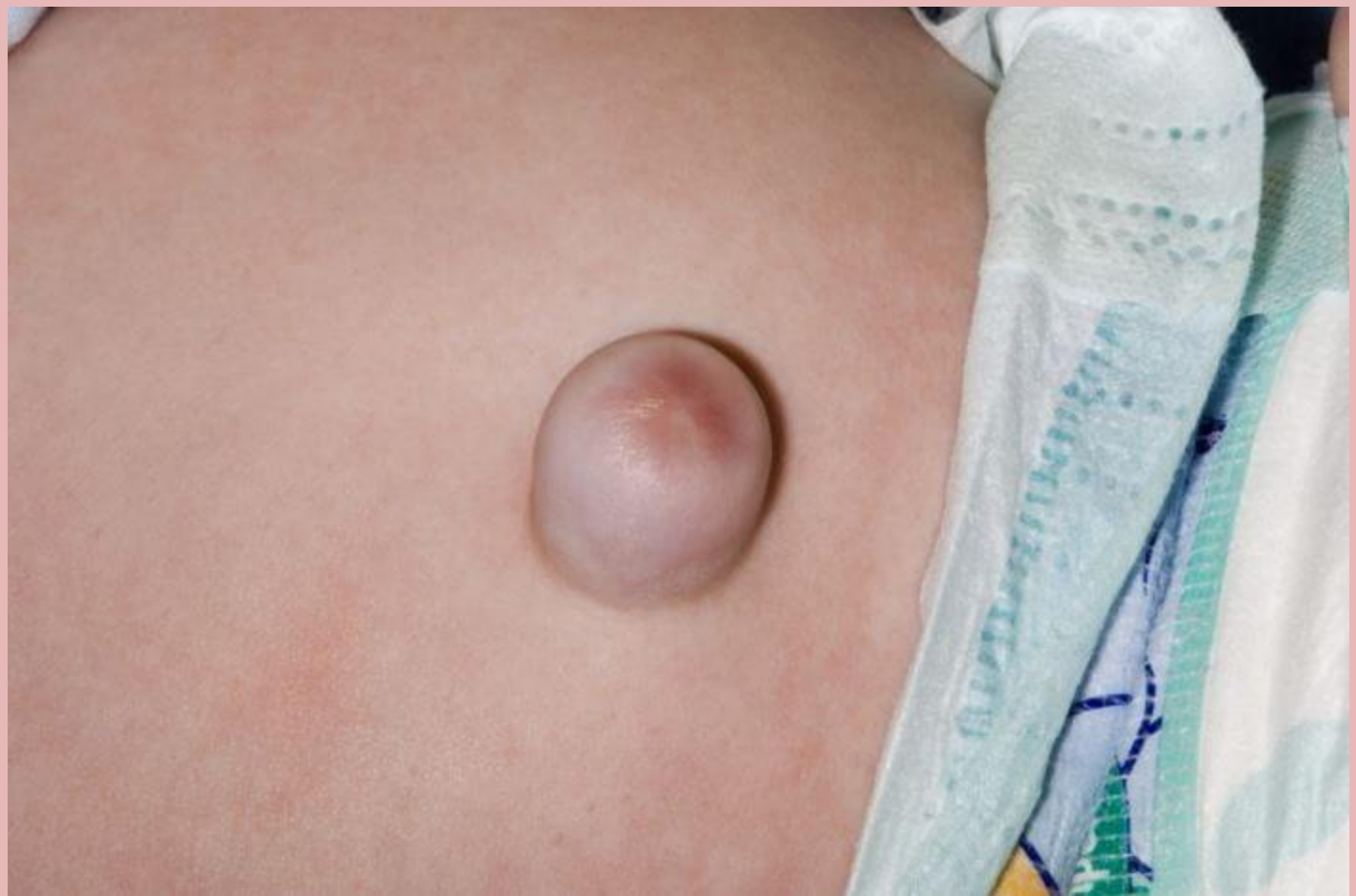
- preterm infants
- Down syndrome
- increased intra-abdominal pressure (such as ascites)
- congenital hypothyroidism.

Although the hernia may be prominent with straining or crying, it should be easily reducible. Incarceration, strangulation and evisceration are rare complications.

Surgical correction is only considered for those who have large defects that are still open at several years of age.

Management

Most children with an umbilical hernia require no intervention. Although some hernias may initially increase in size over the first few months of life, more than 90 per cent will have closed by two years of age.



Genitalia:

•Look for patency of anus.

•The testes should be descendent in a full term infant.

•Look for hernia, hydrocele, hypospadias.

•Female genitalia: look for ambiguous genitalia, in which there is hypertrophy of clitoris and labial fusion, which suggest congenital adrenal hyperplasia.

Extremities:

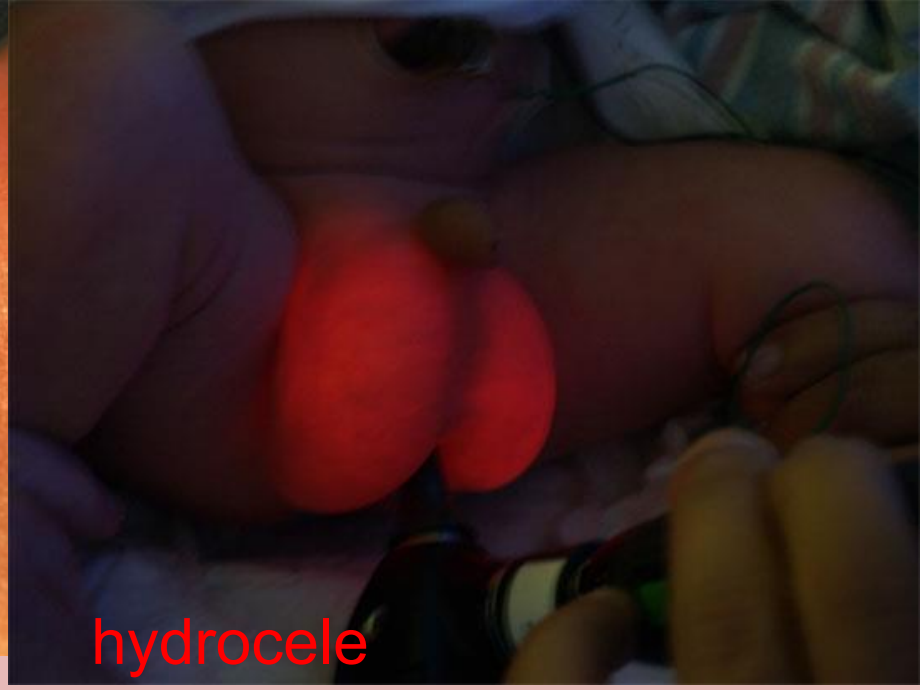
•Look for polydactly(extra finger or digit), syndactly(fusion of the fingers), simian crease(single in Down syndrome) and club feet.

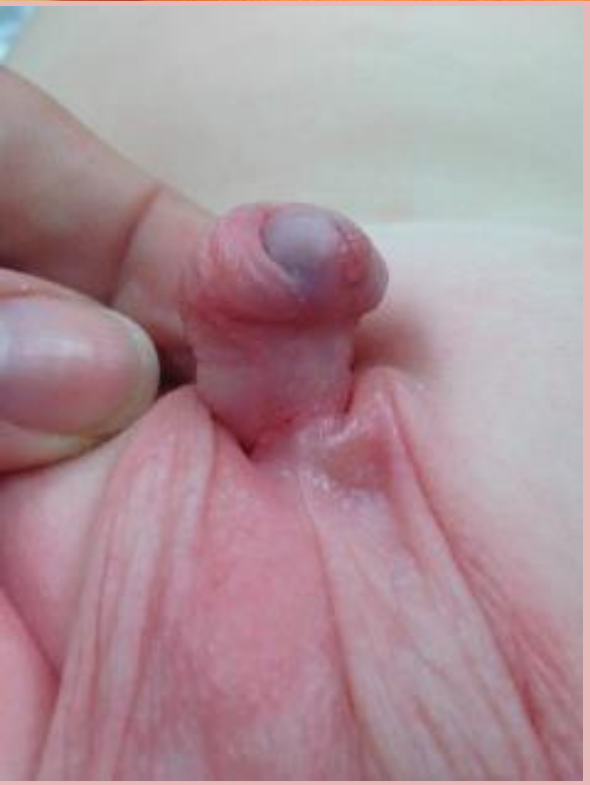
Spines:

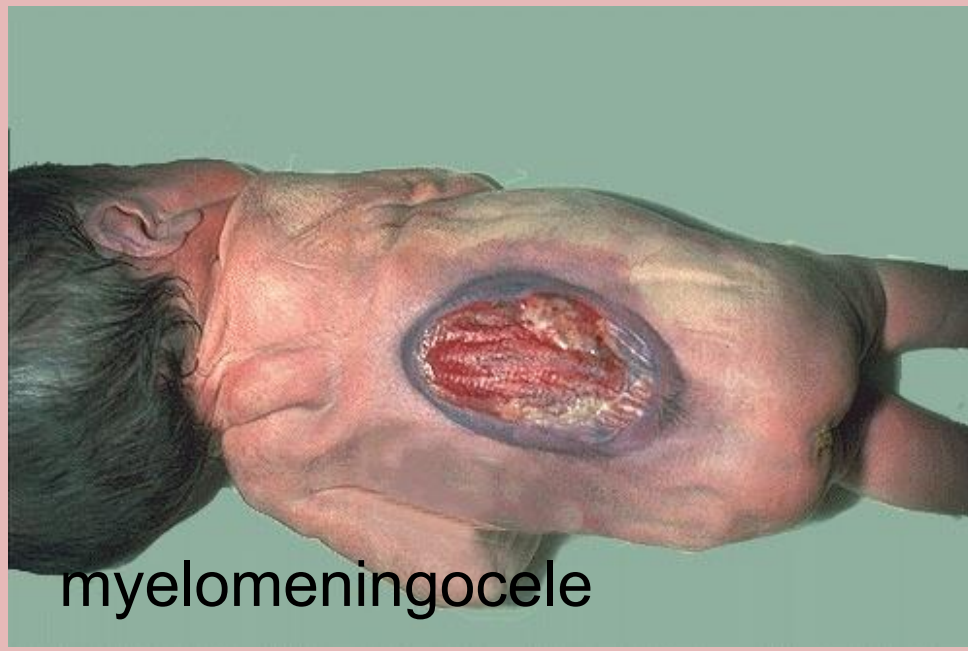
•Look for myelomeningocele, hair tuft.

Hip joints: examine both hip joints for congenital dysplasia by **Barlow's and Ortolani tests.**


Ambiguous genitalia


hydrocele




myelomeningocele

Ortolani test:

Both thighs are held so that the examiner's long finger is placed over the greater trochanters and his thumbs rest on the inner aspects of each thigh. The thighs are then abducted: if a hip is dislocated, a 'clunk' can be felt and heard as the femoral head slips forward into its normal position in the acetabulum.

Barlow's Test:

demonstrates an unstable or dislocatable hip. If the hip is unstable, backward pressure on the lesser trochanter with the thumb on the inner side of the thigh causes the femoral head to slip out of the acetabulum.

Primitive neonatal reflexes:

1. **Moro reflex:**

   elicited by sudden, slight dropping of the supported head from a slightly raised supine position, there will be opening of the hands with extension and abduction followed by adduction and flexion.

- The reflex present at birth and disappear by 3-6 mo of life, its presence beyond 6 mo suggest cerebral palsy.
- Absent moro reflex: in intracranial birth injury, cerebral depression by narcotics, or anesthesia given to the mother just before delivery.
- Asymmetric response(unilateral moro reflex) may be seen in brachial plexuses injury or fracture clavicle.

2. **Palmer grasp reflex:**

- It present by 28 weeks of gestation and gone by 4 mo of age when voluntary palmer grasp begins.

3. **Rooting reflex:**

- Is the turning of the head toward tactile stimulus of the perioral area.

4. **Stepping reflex:**

- When a newborn sole of the foot touches the edge of the table, he will make a step. It disappear by 4 mo of age

5. **Tonic neck reflex:**

- Placing the newborn in a supine position, by lateral rotation of the head to one side, there will be ipsilateral extension of the arm and leg with flexion of limbs on the opposite side (fencing posture).

- The age of the response is 1-6mo of age.
- Absent reflex indicates a spinal cord disease.

❖Routine delivery room care:

•Newborn should be placed head downward immediately after delivery to clear the mouth, nose and pharynx from fluids, mucus, meconium or blood by gentle suction. Delayed cord clamping (30-60 seconds) is recommended to improve transitional circulation and increase neonatal RBC volume.

•Then by gentle tactile stimulation to enhance respiration.

•Maintenance of body heat:

•Relative to body weight, the surface area of newborn is three times that of the adult, so the heat loss of newborn is approximately 4 times that of adult.

•After birth, the newborn infant begins life covered by amniotic fluid and situated in a cold environment(20-25 ˚C).
•An infant skin temperature may fall 0.3 ˚C/min, and the core temperature may decline 0.1 ˚C/min in the delivery room.

•Hypothermia may lead to metabolic acidosis, hypoxemia, hypoglycemia and increased renal excretion of water and solutes which occur in term infants to compensate for heat loss.

•Therefore it is important to dry the newborn infant from secretions and fluids immediately after delivery and to put them under radiant heater during resuscitation, while normal term infants should be dried and wrapped in blanket.

•Antiseptic skin and umbilical cord care:

•Careful removal of blood from the skin of newborn

•The entire skin and umbilical cord should be cleansed with warm water or mild soap solution and then rinsed with water.

•Then the newborn is dried and should be wrapped in sterile blanket.

•Umbilical cord may be treated with bactericidal or antimicrobial agents.

•Rigidly enforcing hand to elbow washing of the nursery personnel with iodophor containing antiseptic soap or chlorhexidine before caring each newborn.

•**Other measures:**

•Vitamin k should be given as a prophylaxis to all newborn 1mg (IM) for full term and 0.5mg for preterm, to prevent hemorrhagic disease of newborn

•Then if the newborn in a satisfactory condition, is given to his mother for immediate bonding and feeding.

•Hepatitis B immunization before discharge from the nursery is recommended for newborns with weight >2 kg, irrespective of maternal hepatitis status.

•Neonatal screening is available for various genetic, metabolic, hematologic,and endocrine disorders.

Large for gestational age:

- Newborn baby with birth weight at the 90th centile.
- Causes:

1. Maternal DM
2. Hydrops fetalis as in Rh isoimmunization.
3. Congenital heart disease and heart failure.
4. Some post term infants.

Low birth weight infant:

Newborn baby whose birth weight less than 2.5 kg.

It could be either:

1. Preterm (a common cause in the developed countries)

2. Full term and small for gestational age(intrauterine growth retardation), a common cause in the developing countries.

Large for gestational age infant/ infant of diabetic mother

Small for date infant(intrauterine growth retardation):

- It describes a newborn who is smaller than the usual number of weeks of gestation, his birth weight at 10th centile for babies of same gestational age.
- IUGR occurs when the fetus does not receive the necessary nutrients and oxygen needed for proper growth and development of organs and tissues.
- **Clinical features:**
- The weight is most affected while the head circumference is the least.
- The skin is thick, pale and crackled.
- If meconium present, the skin, umbilical cord and nails are greenish.
- There is little subcutaneous fat, decreased muscle mass.

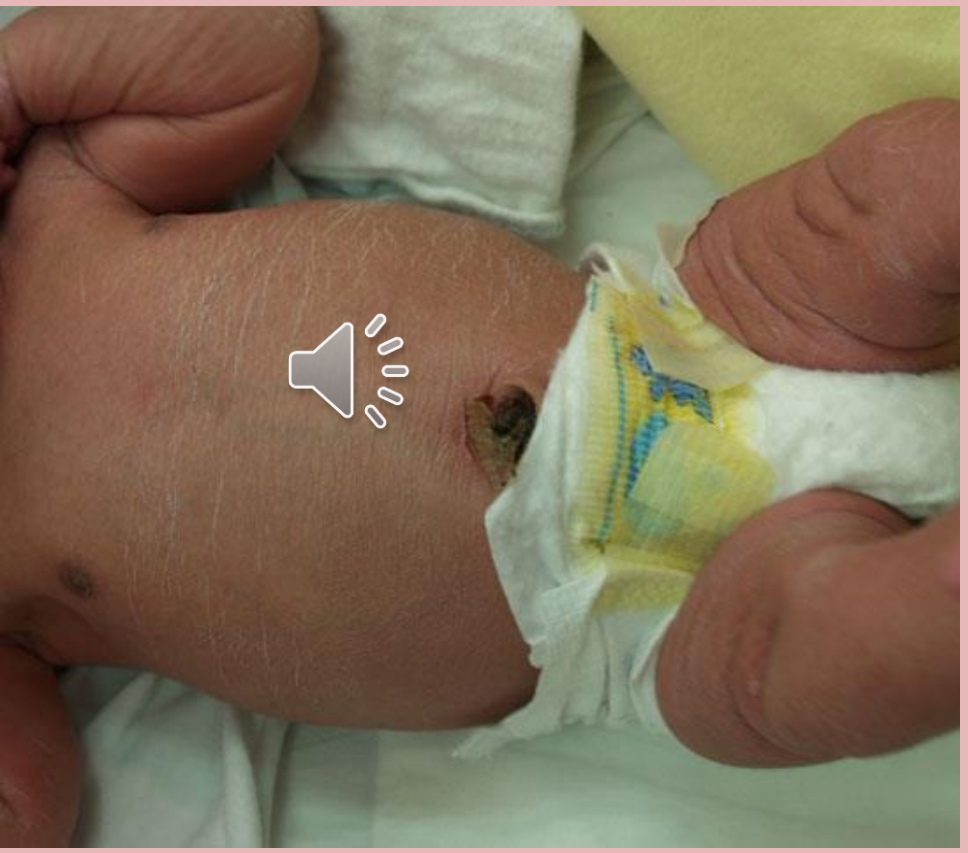
•larger heads for the size of the body (CNS sparing), widened anterior fontanels.

•Physical examination should detail the presence of dysmorphic features might suggest underlying congenital malformations, chromosomal defects, or exposure to teratogens..

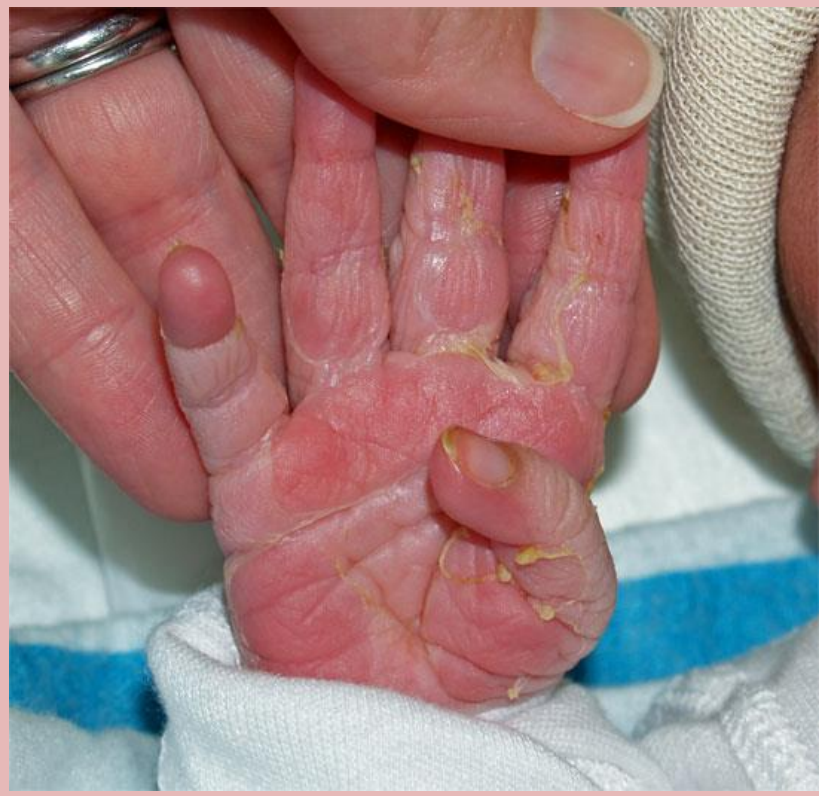
•Hepatosplenomegaly, jaundice, and skin rashes in addition to ocular disorders, such as chorioretinitis, cataracts, glaucoma, and cloudy cornea, suggest the presence of a congenital infection or inborn error of metabolism.

•Mortality rates of infants severely affected are 5 to 20 times the mortality rates of infants who are appropriate for gestational age.

Figure 2: Normal Neonate (R) and Asymmetric IUGR Neonate(L)



Post mature(peeling skin, dry) long nails



Causes of LBW or small for date:

A. Maternal factors	B. Fetal causes	C. Placental causes
1. Previous LBW baby 2. Low socioeconomic state 3. Low level of maternal education 4. No antenatal care 5. Maternal age<16yr or>35yr 6. Short interval between pregnancies. 7. Cigarette smoking 8. Physical & psychological Stresses. 9. Low pre pregnancy weight 10. Hypertension, DM, PET.	1. Congenital infections(rubella) 2. Chromosomal abnormalities. 3. Congenital malformations. 4. Multiple gestations.	1. Placental tumors. 2. Chronic abruptio placentae. 3. Twin-to-twin transfusion syndrome. 4. Placental insufficiency

Problems(complications) of small for date newborn:

1.Temperature instability, mostly hypothermia, due to loss of subcutaneous fat, hypoxia and hypoglycemia.

2.Hypoglycemia, due to decreased glycogen stores, decrease gluconeogenesis, and hypothermia.

3.Polycythemia-hyperviscosity from fetal hypoxia with increased erythropoietin production.

4.Dysmorphology due to syndrome anomalies or chromosomal genetic disorders.

5.Pulmonary hemorrhage due to hypothermia, polycythemia and hypoxia.

6.Meconium aspiration.

7.Perinatal asphyxia due to decreased uteroplacental perfusion during labor or from meconium aspiration.

Preterm infant:

•A newborn baby who is delivered before 37 weeks of gestation.

Characteristic features of prematurity:

•Thin transparent skin with pitting edema over the dorsum of hands and feet.

•Small baby but plump.

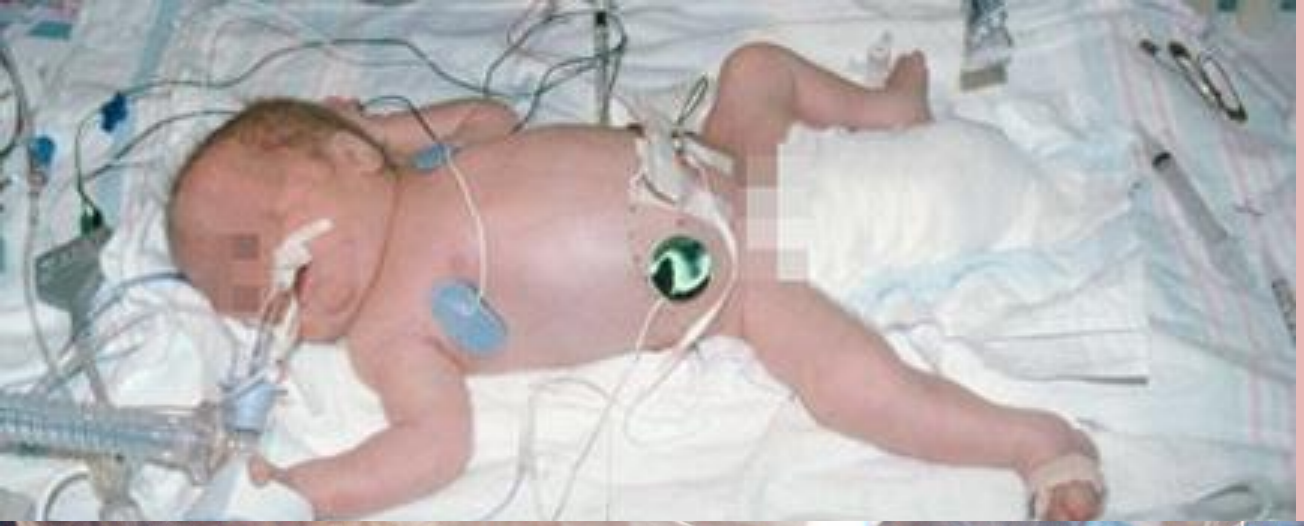
•Hypotonic

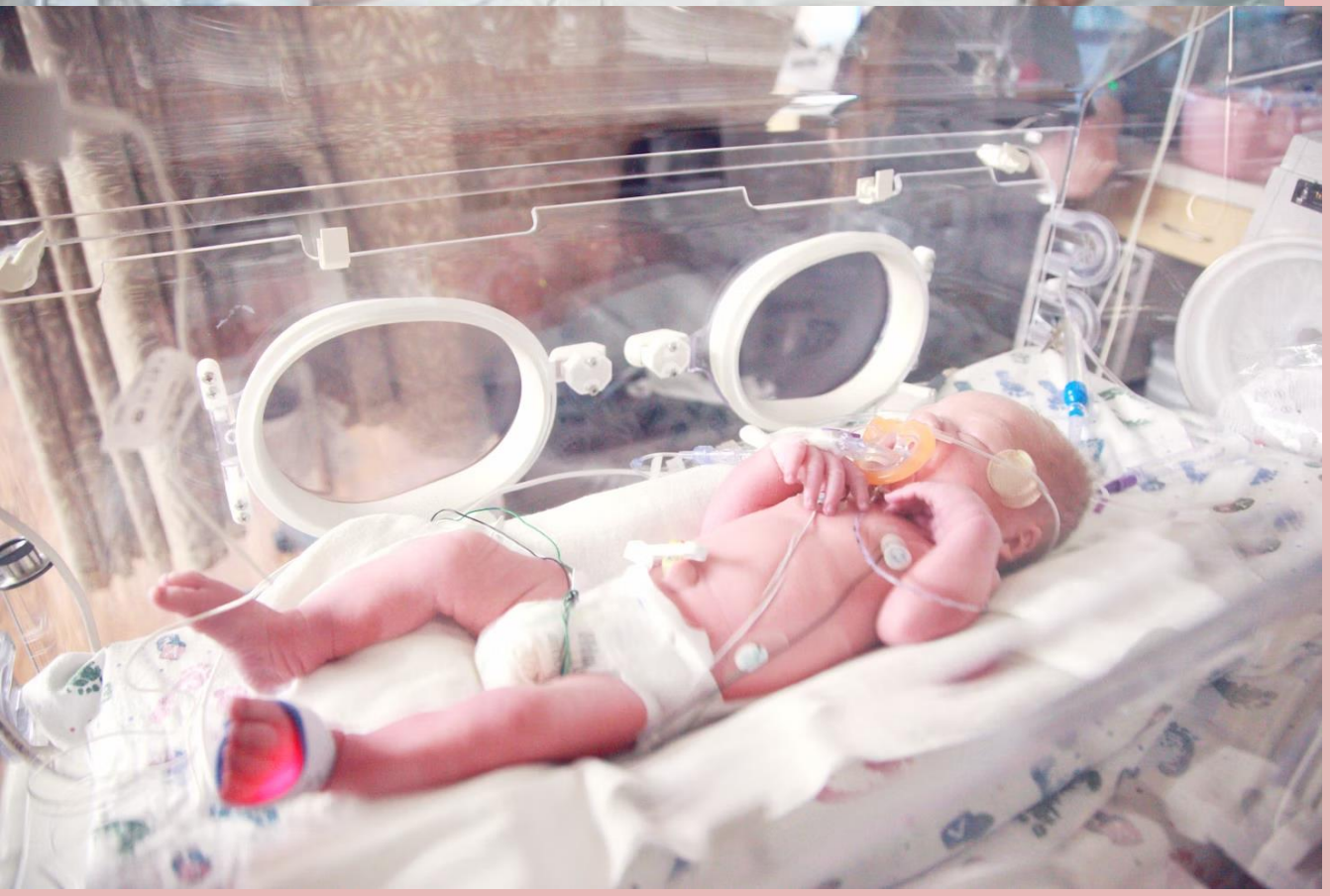
•Decrease firmness of ear lobe, decrease size of breast tissues, and few or absent creases over the sole of foot.

•Underdeveloped genitalia, in male, testes may not be descendent, and wide separation of labia majora not covering the minora in female.

•Excess lanugo hair over the back, trunk and shoulders.

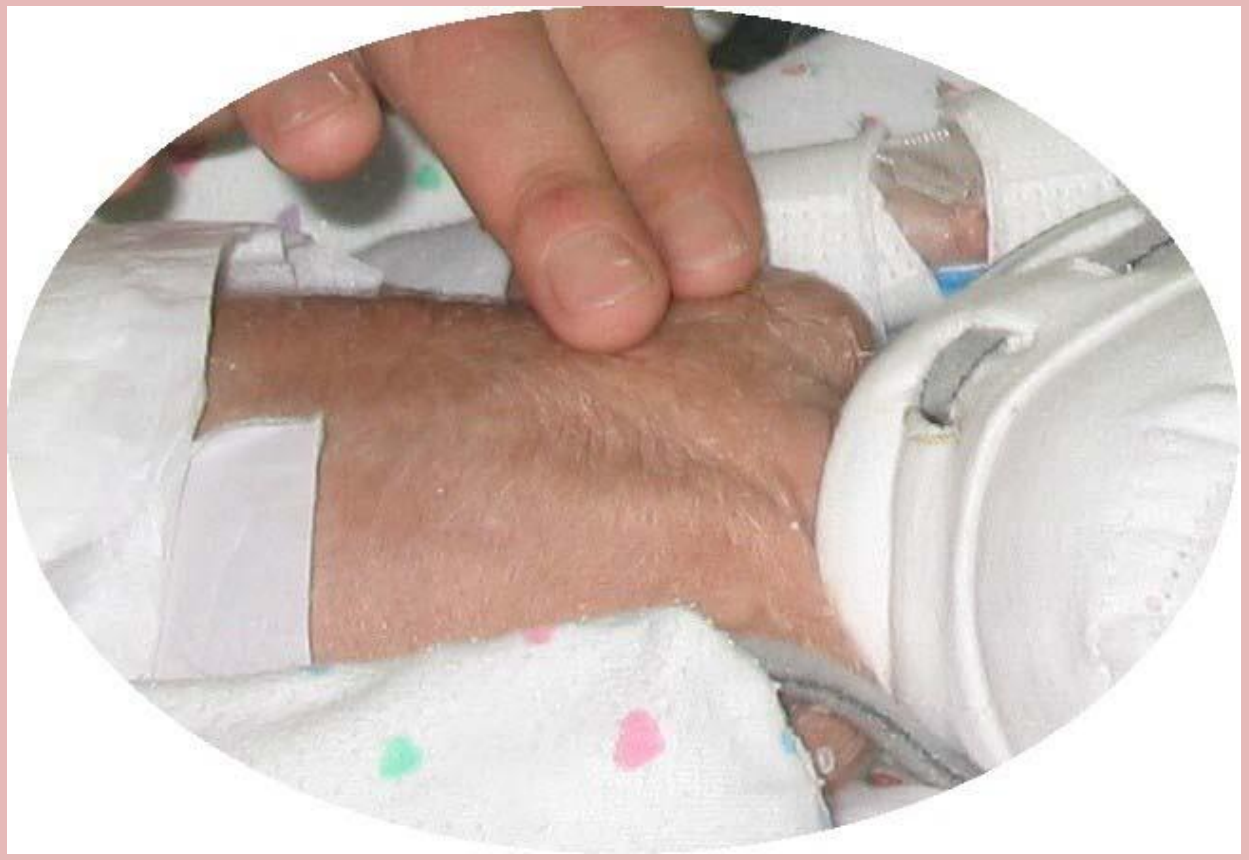
•Posture: the more the premature the baby is, the more he is flaccid and the flexion posture not assumed (frog posture).





Preterm babies

Extended frog like posture



Lanugo hair

Causes of preterm birth:

Fetal	Placental	Uterine	Maternal	Others
1. Fetal distress. 2. Multiple gestations 3. Erythrobl-astosis 4. Congenital anomalies	1. Placenta previa. 2. Abruptio placentae.	1. Bicornuate uterus 2. Incompetent cervix	1. PET 2. DM 3. Hypertension 4. Infection with group B strep. 5. Herpes simplex 6. Syphilis 7. Chorioamnionitis 8. Drug use	1. Premature rupture of membrane 2. Polyhydramnios 3. Iatrogenic (CS) 4. trauma

Problems(complications) of preterm infant:

1. Respiratory distress syndrome and apneic attacks.
2. Metabolic: hypoglycemia, hypocalcaemia and jaundice of prematurity.
3. Hypothermia due to immature thermoregulatory centre, and little subcutaneous fat.
4. Infection and septicemia, due to underdeveloped immune system and excessive instrumentation.
5. Gastrointestinal problems: Poor sucking and swallowing and immature bowel function, and necrotizing enterocolitis.

7. Neurologic: intraventricular hemorrhage, hypotonia.

8. Retinal problems: retinopathy of prematurity secondary to hyper oxygenation.

9. Cardiovascular problems: bradycardia with apnea, patent ductus arteriosus.

10. Blood problems: exaggeration of physiological anemia, hemorrhage.

Management of premature baby:

1. **Thermal control:**
   to keep the infant core temperature 36.5-37C, humidity 40%, inside an incubator care with temperature set at 34 Cº.
2. **Oxygen:**
   humidified oxygen in 2-3L/min.
3. **Fluid requirement:**
   in the first day of life starts with 70ml/kg, then add 10-20ml/kg daily until it reaches 150ml/kg at fifth day of life. Type of fluid 10% glucose water in the first 2 days of life, then changed to 1/5th glucose saline. Avoid giving excessive fluid because of risk of patent ductus arteriosus.

4. Feeding:

- do not give feeding until there is, a good bowel function i.e. passage of meconium, active bowel sounds, no abdominal distension, normal respiration and no apneic attacks and after the baby is clinically stable and can tolerate oral feeding.
- Bottle feeding is easier than breast feeding for 34 wk old neonate.
- Nasogastric tube feeding is appropriate for 32wk premature.

5. Prevention of infection:

- Minimum handling of premature.
- Avoid excessive instrumentations.
- Appropriate hand to elbow washing before nursing a preterm baby.
- Early recognition and treatment of infection.

6. Immaturity of drug metabolism:

- Renal excretion is slower than that of older children, so the drug interval should be at least 12 hourly.

Respiratory distress syndrome(Hyaline membrane disease) RDS:

- **Pathophysiology:**

- The normal mature lung during fetal life secrets a substance from type2 cells called **surfactant,** which is a phospholipids containing lecithin, it is essential for the stability of the lung, it prevent atelactasis by reducing surface tension in the alveoli at end expiration.
- In preterm baby this surfactant is deficient, so the surface tension forces of the alveoli are not reduced, therefore ***atelactasis*** develops during end expiration as the alveolus collapses leading to decrease functional residual capacity, so hypoxemia, pulmonary artery vasoconstriction and respiratory distress will develop.

- Factors that further reduce surfactant synthesis include, hypovolemia, acidosis and hypoxemia.

Infants at a greater risk for RDS :

1. Prematurity
2. Delivery of previous preterm baby with RDS.
3. Maternal DM.
4. Hypothermia
5. Fetal distress, asphyxia.
6. Male sex, white race.
7. Being a second born of twin.

Clinical manifestations:

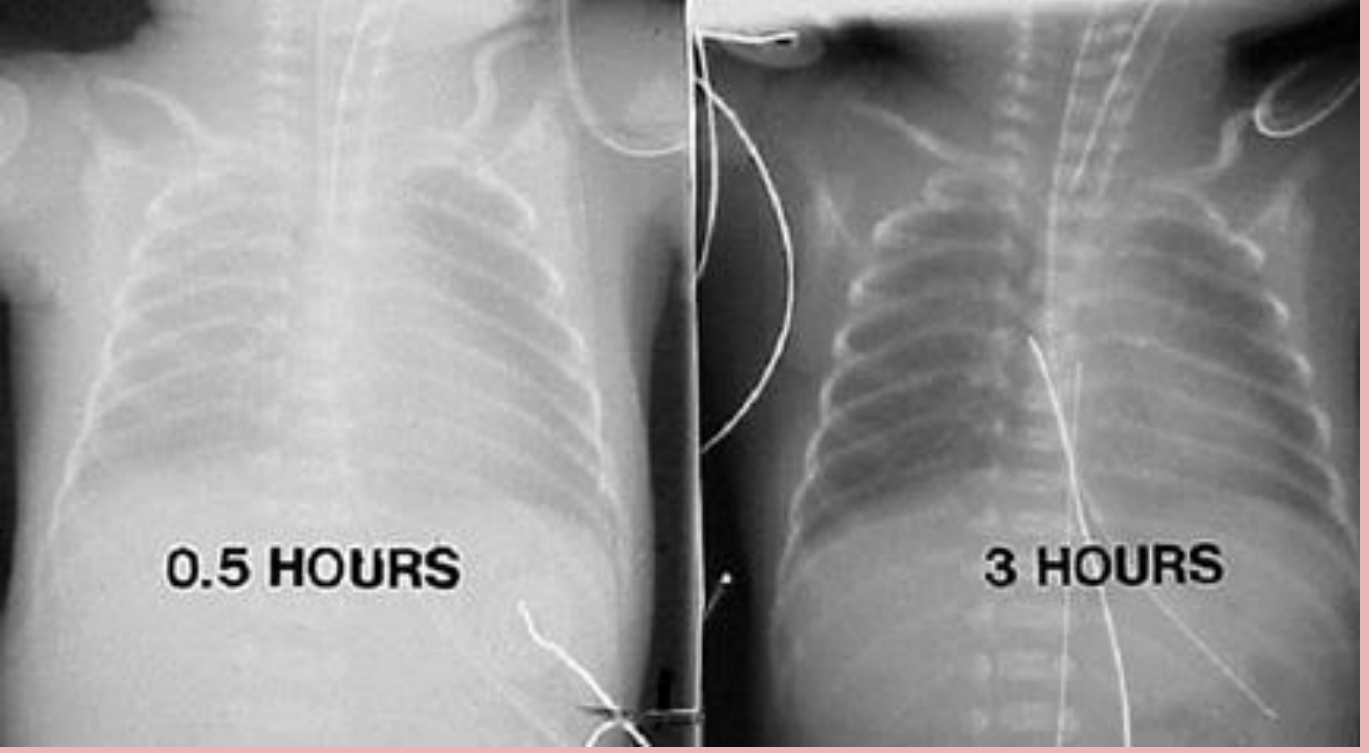
- Signs of RDS usually appear within minutes of birth, although they may not be recognized for several hours in larger premature infants

•Tachypnea, cyanosis, nasal flaring, intercostals and subcostal retractions, grunting(caused by closure of the glottis during expiration to maintain adequate gas exchange during exhalation).

•During next 72 hr baby either show signs of improvement or he may deteriorate into severe respiratory distress, apneic attacks and respiratory failure.

•Auscultation of the chest show diminished air entry bilaterally.

•CXR: show atelactasis seen as a ground glass haze in the lung surrounding air-filled bronchi(air bronchogram= aerated bronchioles superimposed on a background of collapsed alveoli)Blood gas analysis show decreased Po2, increase Co2 and low PH.



Chest radiographs in a premature infant with respiratory distress syndrome before and after surfactant treatment. Left: Initial radiograph shows poor lung expansion, air bronchogram, and reticular granular appearance. Right: Repeat chest radiograph obtained when the neonate is aged 3 hours and after surfactant therapy demonstrates marked improvement.

❖After birth, RDS may be prevented or its severity reduced by early intratracheal administration of surfactant.

Differential diagnosis:

•Early onset sepsis, pneumonia, congenital diaphragmatic hernia, pneumothorax, cyanotic congenital heart disease and lobar emphysema.

Treatment of RDS:

•Adequate humidified oxygen to keep O2 saturation 90%.
•O2 can be administered by nasal cannula or oxygen hood.
•The goal of therapy for patients with respiratory distress syndrome is to maintain a pH of 7.25-7.4, a partial pressure of oxygen (PaO2) of 50-70mm Hg, and a carbon dioxide pressure (PCO2) of 40-65 mm Hg.

•If hypoxemia(PaO2<50mmHg) present then nasal continuous positive airway pressure should be added.

•If respiratory failure ensures(Pco2>60mmHg, PH<7.2, PaO2 <50mmHg) with 100% oxygen then assisted ventilation using a respirator is indicated.

•10% glucose water is given in the first 2 days then changed to 1/5th glucose saline.

•Because it is difficult to distinguish between sepsis and pneumonia from RDS, broad spectrum antibiotics using ampicillin and gentamicin is added.

•Surfactant administration through endotracheal tube will improve alveolar oxygen, increase pulmonary compliance and improve CXR findings. Repeated doses is given every 6-12hr.

Complications of RDS:

1. Pulmonary air leaks: pneumothorax, subcutaneous emphysema and pneumomediastinum.
2. Patent ductus arteriosus(PDA):

   in term infants the ductus closes within 24-48 hr of birth, while in preterm infant specially with RDS may remain patent.

   blood will be shunted from left to right direction, causing heart failure, pulmonary edema and machinery murmur

   (systolic and diastolic).

   it can be prevented by initial fluid restriction and diuretic use, if not closed, so give indomethacin IV injection and finally if not closed surgical correction needed later on.

3. Bronchopulmonary dysplasia:

it is a form of chronic lung disease in which there is oxygen dependency.

it is caused by oxygen mediated lung damage, manifested by hypercapnea, hypertension, poor growth and right sided HF.

4. Retinopathy of prematurity(Retrolental fibroplasia):

it is caused by acute and chronic effects of oxygen toxicity on the developing blood vessels of premature infants retina.

it is the leading cause of blindness in very LBW infants.

the excessive arterial oxygen tension will cause vasoconstriction of retinal vasculature, this will lead to vaso-obliteration and fibro vascular proliferation with retinal detachment.

Perinatal asphyxia(Hypoxic Ischemic Encephalopathy):

•It is the state of reduced gaseous exchange through the placenta or through the lungs, leading to lack of oxygen and perfusion to various organs.

•***Pathophysiology:***

•It is a process of hypoxia, hypercapnea, poor cardiac output and metabolic acidosis.

•Asphyxia associated with severe bradycardia or cardiac insufficiency will lead to ischemia.

•Fetal and neonatal circulatory system responds to hypoxia by shunting blood preferentially to the brain, heart, adrenals and away from the intestine, kidney, lungs and skin.

Causes of birth asphyxia:

Intrauterine	Intrapartum	Postpartum
1. Hypoxia-ischemia: uteroplacental insufficiency, abruptio placentae, prolapsed cord, maternal hypotension 2. anemia-shock: placenta previa, fetomaternal hemorrhage, erythroblastosis.	1. Birth trauma: cephalopelvic disproportion, shoulder dystocia, breech presentation 2. Hypoxia-ischemia: umbilical cord compression, tetanic contraction of uterus(oxytocine overuse).	1. CNS may be affected by maternal medications, anesthesia, trauma 2. Congenital neuromuscular disease like cong. Myasthenia gravis, myopathy, myotonic dystrophy. 3. Infection: pneumonia, shock. 4. Airway disorder: choanal atresia, laryngeal webs. 5. Pulmonary disorder: severe immaturity, pneumothorax, pleural effusion, diaphragmatic hernia.

Effects(complications) of birth asphyxia:

CNS: hypoxic-ischemic encephalopathy(H.I.E), intraventricular hemorrhage, cerebral edema, seizures, hypotonia, hypertonia, later on infant may have delayed development, cerebral palsy and mental retardation.

CVS: myocardial ischemia, poor contractility, hypotension.

Pulmonary: persistent pulmonary hypertension, RDS.

Renal: acute tubular or cortical necrosis.

Adrenal: adrenal hemorrhage.

GIT: perforation, ulceration, and necrosis.

Metabolic: inappropriate ADH secretion, hyponatremia, hypoglycemia, hypocalcaemia.

Hematology: DIC.

Apgar score examination:

•It is a rapid scoring system based on physiological responses to the birth process and it is used to assess the need to resuscitate a newborn.

•This scoring system is based on 5 physiological criteria, which should be observed by qualified examiner at interval of 1 and 5 min after birth.

•Full term infant with normal cardiopulmonary adaptation should score 8 to 9 at 1 and 5 min.

•Apgar score of 4 to 7 warrant close attention

•Apgar score of 0 to 3 represents either cardiopulmonary arrest, hypoventilation, severe bradycardia or CNS depression.

Apgar scoring parameters:

signs	0	1	2
Heart rate	0	<100/min	>100/min
Respiration	None	Weak cry	Vigorous cry
Muscle tone	None	Some extremity flexion	Arms, legs well flexed
Reflex irritability	None	Some motion	Cry, withdrawal
Body color	Blue	Pink body, blue extremities	Pink all over

Clinically:

•Early signs of fetal distress, fetal heart rate show deceleration pattern, scalp PH<7.20 and meconium stained liquor.

•After birth the newborn fail to breath spontaneously and may remain hypotonic, pale, cyanosed, apneic, and unresponsive to stimulation.

•Other presentations may occur like RDS, HF, pulmonary hypertension, hematuria, convulsion……

Prognosis:

•Mortality rate 10-20-%

•Those who recover usually had mild presentations, while those with moderate and severe presentations many of them will have cerebral palsy, MR, epilepsy, hearing defect, and microcephaly.

Investigations:

•Blood gas analysis, PH.
Brain and abdominal ultrasound(has limited utility in evaluation of hypoxic injury in term infant, but it can be useful for excluding hemorrhagic lesions).

- MRI is the most sensitive imaging modality for detecting hypoxic brain injury in the neonate

•CT scan of the brain, to look for infarction, bleeding or intraventricular hemorrhage.

- Amplitude-integrated electroencephalography (aEEG) may help to determine which infants are at highest risk for developmental sequelae of neonatal brain injury, can provide valuable prognostic information, for infants who will have adverse neurodevelopmental outcome.

APGAR

- 7 - 10
  - Normal Infant
  - Suction oropharnyx
  - Keep warm

APGAR

- 4 - 6
  - Moderate asphyxia
  - Suction oropharnyx
  - Keep warm
  - Oxygenate
  - If 5 minute score < 7, repeat every 5 minutes for 20 minutes

APGAR

- 0 - 3
  - Asphyxia neonatorum
  - Resuscitate aggressively

Treatment(resuscitation):

- **Therapeutic hypothermia** , whether head cooling or systemic cooling (by servo-control to a core rectal or esophageal temperature of 33.5°C within the 1st 6 hr after birth and maintained for 72 hr), has been shown in various trials to reduce mortality and major neurodevelopmental impairment at 18 mo of age.

- High-dose **erythropoietin** given as an adjunct to therapeutic hypothermia shows some promise in decreasing MRI indicators of brain injury and short-term motor outcomes.

•Complications of induced hypothermia include thrombocytopenia (usually without bleeding), reduced heart rate, and subcutaneous fat necrosis as well as the potential for overcooling and the cold injury syndrome.

•The resuscitation usually follows A,B,C,D.

•**A**: secure patent airway by gentle oropharyngeal suction of amniotic fluid, meconium or blood.

•**B**: maintain breathing through gentle tactile stimulation, oxygen by face mask or ambu bag with positive pressure ventilation.

•If still there is no spontaneous respiration, HR <100/min, then ventilation is done through endotracheal intubation, with external cardiac massage over the lower third of the sternum at rate of 120/min.

•The ratio of cardiac compression to ventilation is **3:1**.

•**C:**if HR remain below 60/min despite effective cardiac compression and ventilation, epinephrine is given in a dose of 0.1-0.3 ml/kg of 1:10000 solution IV or intratracheal, the dose may be repeated every 3-5min.
•Sodium bicarbonate 2meq/kg may be given in prolonged acidosis

•Dopamine or doputamine infusion 5-20 µg/kg/min to improve cardiac output in neonate with poor peripheral perfusion, weak pulse and hypotension.

•10% glucose water or normal saline infusion in case of shock.

•Naloxone is given as antidote if the mother received narcotics, in a dose of 0.1 mg/kg

•Anticonvulsants is given when indicated, phenobarbitone given IV as 20mg/kg loading dose followed by 5mg/kg maintenance dose, or phenytoin as alternative.
•Lorazepam 0.1mg/kg/ day or **levetiracetam** may be needed in refractory cases.

•<u>Good response is determined by</u>

• Adequate chest rise
•Symmetric breath sounds
•Improved pink color
•HR>100/min and
•Spontaneous respiration.

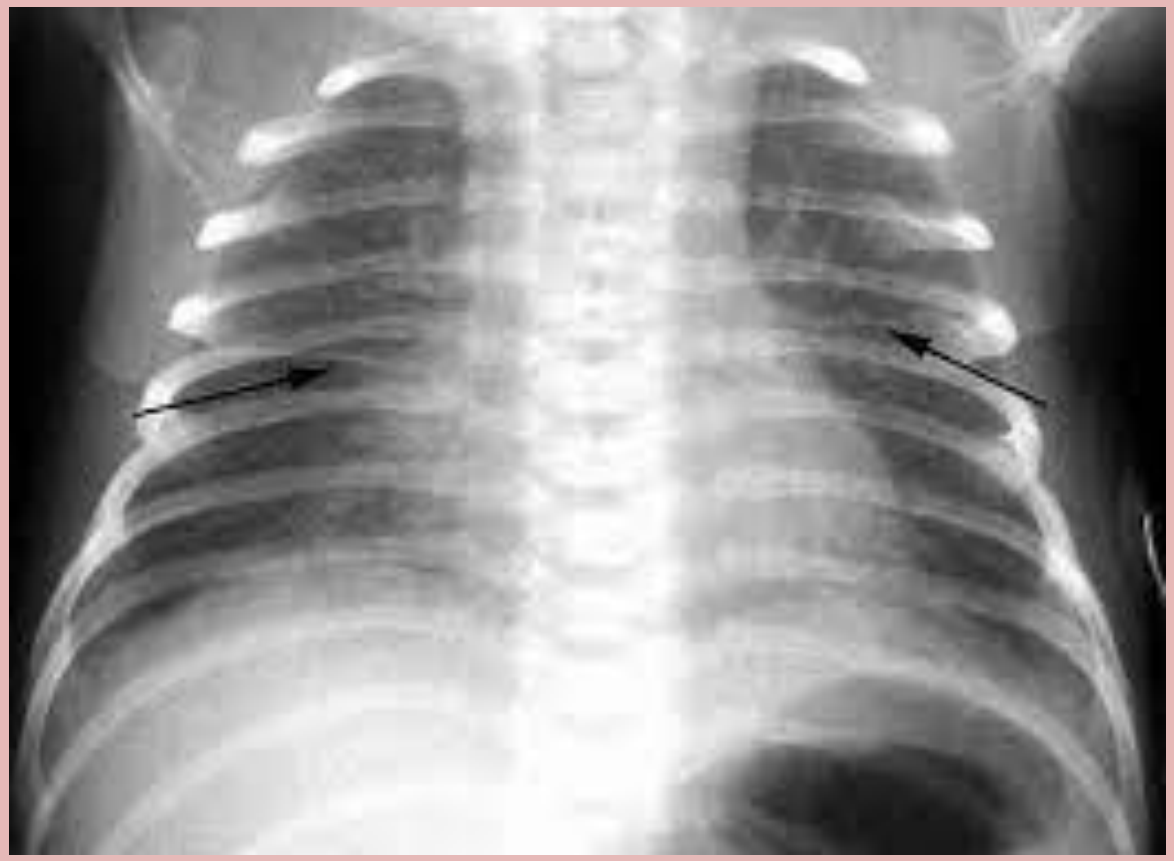
Transient Tachypnea of the Newborn

- is a clinical syndrome of self-limited tachypnea associated with delayed clearance of fetal lung fluid.
- TTN the most common etiology of tachypnea in the Newborn
- <u>common associated risk factors</u>

- Twin gestation
- Maternal asthma
- Late prematurity
- Precipitous delivery
- Gestational diabetes
- And cesarean delivery without labor

- TTN is believed to result from ineffective activity of epithelial sodium channels and Na+ ,K+ -ATPase, which slows absorption of fetal lung fluid and results in decreased pulmonary compliance and impeded gas exchange.

- TTN is characterized by the early onset of tachypnea (>60 breaths/min), sometimes with retractions or expiratory grunting and occasionally with cyanosis that is relieved by minimal O2 supplementation (<40%).
- The chest generally sounds clear without crackles or wheeze
- The chest radiograph shows prominent perihilar pulmonary vascular markings, fluid in the intralobar fissures, and rarely small pleural effusions.
- Hypercapnia and acidosis are uncommon.
- Respiratory failure requiring positive pressure support also is uncommon.



- **Treatment**
- Is supportive.

- Inhaled β2 - agonists such as albuterol (salbutamol) increase expression and activation of epithelial Na channels and Na+ ,K+ m ATPase and facilitate fluid clearance.

- Emerging evidence suggests that when given early in the course of TTN, albuterol may improve oxygenation, shorten the duration of supplemental O2 therapy, and expedite recovery.

Meconium Aspiration

- Meconium-stained amniotic fluid is found in 10–15% of births and usually occurs in term or post-term infants.

- **Meconium aspiration syndrome (MAS)** develops in 5% of such infants; 30% require mechanical ventilation, and 3–5% die.

- Infants with MAS are at increased risk of **persistent pulmonary hypertension**

- Either in utero or with the first breath, thick, particulate meconium is aspirated into the lungs.

- The resulting small airway obstruction may produce respiratory distress within the 1st hours, with tachypnea, retractions, grunting, and cyanosis observed in severely affected infants.

- Partial obstruction of some airways may lead to pneumomediastinum, pneumothorax, or both.
- Overdistention of the chest may be prominent.

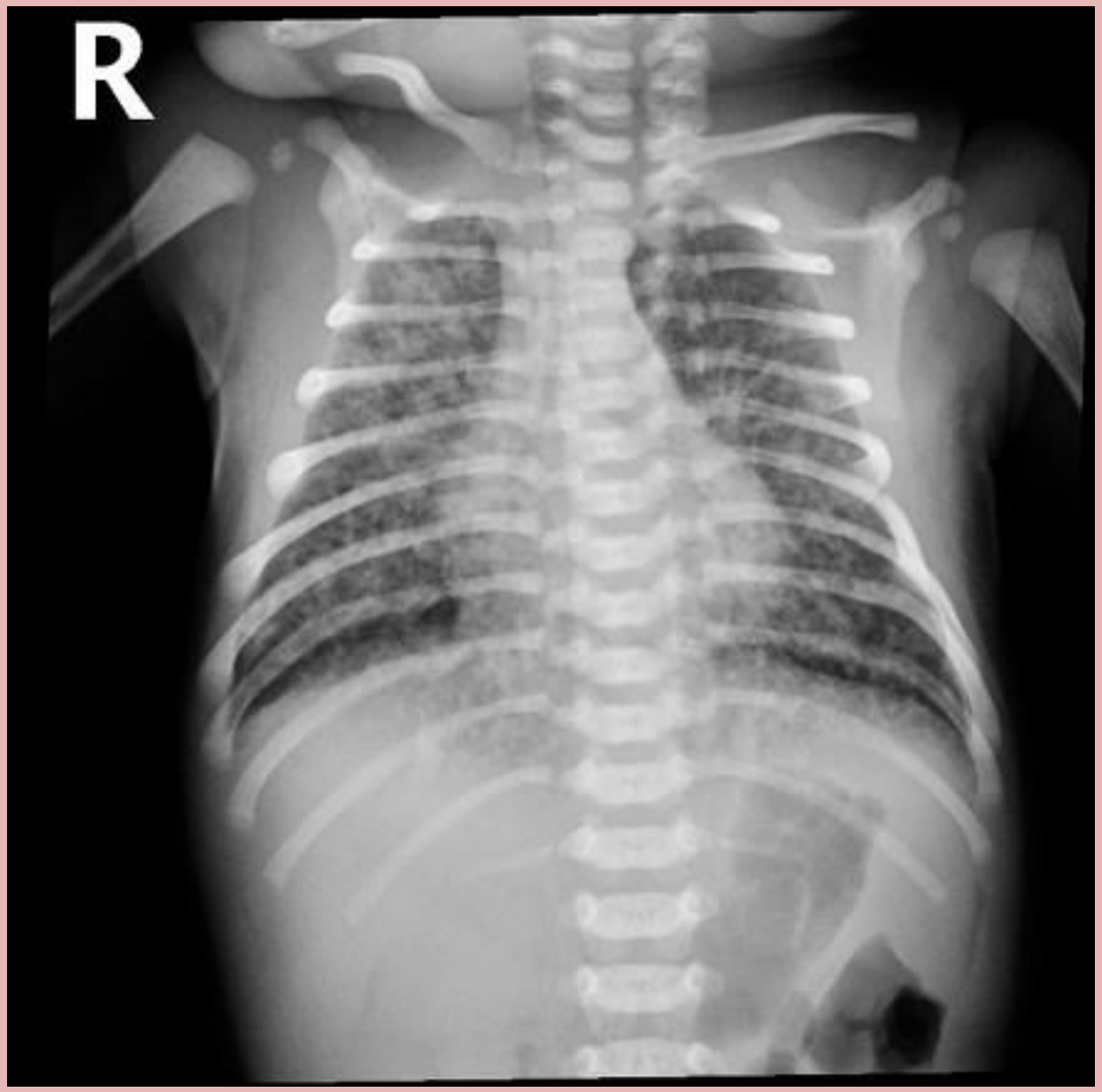
- The condition usually improves within 72 hr, but when its course requires assisted ventilation, it may be severe with a high risk for mortality.

- The typical chest radiograph is characterized by patchy infiltrates, coarse streaking of both lung fields, increased anteroposterior diameter, and flattening of the diaphragm.

Treatment

- Includes supportive care and standard management for respiratory distress.
- Administration of exogenous surfactant and/or nitrous oxide to infants with MAS and hypoxemic respiratory failure, or pulmonary hypertension the need for extracorporeal membrane oxygenation (ECMO), which is required by the most severely affected infants

- In infants with MAS who demonstrate no other signs of sepsis, there is no role for routine antibiotic therapy.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Prognosis

- The mortality rate of meconium-stained infants is considerably higher than that of nonstained infants.

- Residual lung problems are rare but include symptomatic cough, wheezing, and persistent hyperinflation for up to 5-10 yr.

Diaphragmatic Hernia

- A *diaphragmatic hernia* is defined as a communication between the abdominal and thoracic cavities with or without abdominal contents in the thorax
- In **congenital diaphragmatic hernia (CDH)** the Bochdalek hernia accounts for up to 90% of the hernias seen, with 80–90% occurring on the left side.
- a major limiting factor for survival is the associated **pulmonary hypoplasia**
- The incidence of CDH is between 1 in 2,000 and 1 in 5,000 live births, with females affected twice as often as males.
- Defects are more common on the left (85%) and are occasionally bilateral (<5%).

Diagnosis and Clinical Presentation

- In >50% of cases, CDH can be diagnosed on prenatal ultrasonography (US) between 16 and 24 wk of gestation.
- High-speed fetal MRI can further define the lesion.
- US findings may include polyhydramnios, chest mass, mediastinal shift, gastric bubble, and fetal hydrops.

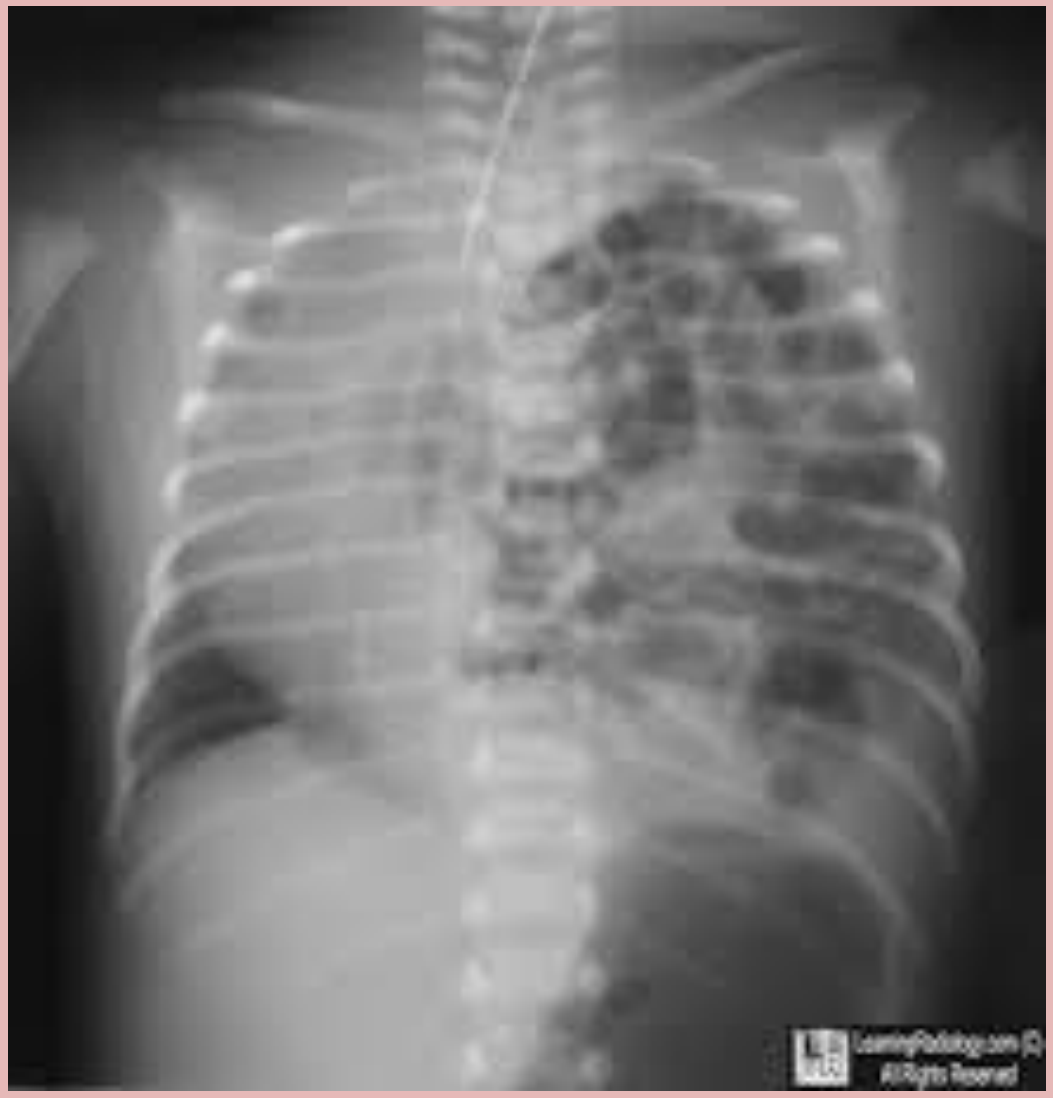
•Respiratory distress is a cardinal sign . It may occur immediately after birth, or there may be a “honeymoon” period of up to 48 hr during which the baby is relatively stable.

•There will be tachypnea, grunting, use of accessory muscles, and cyanosis.

•may also have a scaphoid abdomen and increased chest wall diameter.

- Bowel sounds may also be heard in the chest with decreased breath sounds bilaterally.
- The point of maximal cardiac impulse may be displaced away from the side of the hernia if mediastinal shift has occurred.

- A chest radiograph and passage of a nasal gastric tube are usually sufficient to confirm the diagnosis.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Treatment

- In the delivery room, infants with respiratory distress should be rapidly stabilized with endotracheal intubation.
- *Prolonged mask ventilation in the delivery room, which enlarges the stomach and small bowel and thus makes oxygenation more difficult, must be avoided* and a naso- or orogastric tube placed immediately for decompression.
- Conventional mechanical ventilation, high-frequency oscillatory ventilation (HFOV), and ECMO are the 3 main strategies to support respiratory failure in the newborn with CDH.

- **Surgical Repair:** Most experts wait at least 48 hr after stabilization and resolution of the pulmonary hypertension.