Ministry of Education and Science of Ukraine
V. N. Karazin Kharkiv National University

# RESPIRATORY DISEASES IN NEWBORNS

Methodical recommendations
for students of 5th course of medical faculty

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Methodical recommendations are worked out on the basis of the Program of discipline of "Pediatrics, children infectious diseases" for students of higher medical educational institutions of III–IV levels of accreditation, authorized by Ministry of Health of Ukraine. These recommendations are intended for the 5th years English-speaking medical students.

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# CONTENTS

# LIST OF ACRONYMS

ASD – atrial septal defect
AVL – artificial lung ventilation
BP – Blood pressure
BPD – Bronchopulmonary dysplasia
CAA – Congenital airway abnormalities
CBC – Complete blood count
CCAM – Congenital cystic adenomatoid malformation
CDH – Congenital Diaphragmatic Hernia
CHD – Congenital Heart Disease
CMV – Cytomegalovirus
CPAP – Continuous positive airway pressure
CRP – C-reactive protein
CS – Cesarean Section
ECMO – Extracorporeal Membrane Oxygenation
GBS – group B Streptococcus
HSV – Herpes simplex virus
IDM – Infants of diabetic mothers
IUGR – Intrauterine growth restriction
MAS – Meconium Aspiration Syndrome
MSAF – Meconium-stained amniotic fluid
MV – Mechanical ventilation
PDA – patent ductus arteriosus
PFO – patent foramen ovale
PIE – Pulmonary Interstitial Emphysema
PPHN – Persistent Pulmonary Hypertension of the Newborn
PPV – Positive pressure ventilation
RDS – Respiratory Distress Syndrome
TTN – Transient Tachypnea of the Newborn
VSD – ventricular septal defect

# INTRODUCTION. PECULARITIES OF FETAL AND NEONATE RESPIRATION. COMMON CAUSES OF NEONATAL RESPIRATORY DISTRESS. DOWNE'S SCORE

Respiratory disorders are one of main causes of morbidity and mortality of the term and preterm newborns.

Respiratory disorders account for 30–35 % of early neonatal mortality occupy the leading place in structure of early neonatal mortality.

Airways in newborn baby, as well as in adults, are consist of three sections: the upper (nose, throat), average (larynx, trachea, bronchi), bottom (bronchioles, alveoli).

During intrautering development, the fetus receives a constant supply of oxygen and nutrients via the placenta and umbilical vessels, with carbon dioxide excretion also managed by the maternal circulation. The lungs are filled with fluid secreted by the respiratory epithelium which is important for promoting lung growth.

During the first breathe (gasp) immediately after birth, the neonate fills the airways down to alveolar level with air to commence extra-uterine gas exchange; simultaneously, decreasing pulmonary vascular pressure to allow increased blood flow to the lungs; additionally, reabsorption of the fetal lung fluid occurs.

A preterm neonate born at <37 weeks' gestation has the additional complication of achieving these changes with relatively immature lungs. Extremely preterm (≤28 weeks' gestation) and late preterm neonates (≤32 weeks' gestation) need to survive without adequate alveolar development, which generally commences after 32 weeks' gestational age.

Neonatal respiratory conditions can arise for several reasons: delayed adaptation or maladaptation to extra-uterine life, existing conditions such as surgical or congenital anomalies or from acquired conditions such as pulmonary infections occurring either pre- or post-delivery.

The most common reasons of respiratory disorders in neonates consist of 4 main groups:

1. Preterm pathology:
   – Respiratory distress syndrome;
   – Pneumothorax;
   – Pneumonia;
   – Pulmonary haemorrhage;
   – Aspiration;
   – Pleural effusion (chylothorax);
   – Chronic lung disease.
2. Term pathology:
   – Transient tachypnoea of the newborn;

– Respiratory distress syndrome;
– Meconium aspiration;
– Primary or secondary persistent pulmonary hypertension of the newborn;
– Pneumonia;
– Pneumothorax;
– Aspiration;
– Pleural effusion (chylothorax);
– Pulmonary haemorrhage;
– Surfactant protein deficiency syndromes;
– Alveolar capillary dysplasia.

3. Congenital anomalies/surgical conditions:
– Congenital pulmonary airway malformation;
– Congenital diaphragmatic hernia;
– Tracheo-oesphageal fistula;
– Choanal atresia;
– Pulmonary sequestration;
– Congenital lobar emphysema.

4. Non-respiratory causes of respiratory distress:
– Heart failure (due to congenital heart disease);
– Neuromuscular disorders;
– Hypoxic ischaemic encephalopathy;
– Metabolic acidosis (due to inborn error of metabolism).

**Clinical estimation of severity of respiratory disorders of newborn (Downe's Score)**

| **Test** | **Score** | | |
|---|---|---|---|
| | **0** | **1** | **2** |
| Respiratory rate | <60/minute | 60-80/minute | >80/minute |
| Retractions | No retractions | Mild retractions | Severe retractions |
| Cyanosis | No cyanosis | Cyanosis relieved by O2 | Cyanosis on 40% O2 |
| Air entry | Good bilateral air entry | Mild decrease in air entry | No air entry |
| Grunting | No grunting | Audible by stethoscope | Audible with ear |
| **Evaluation** | | | |
| **Total** | **Diagnosis** | | |
| <4 | No respiratory distress | | |
| 4–7 | Respiratory distress | | |
| >7 | Impending respiratory failure; blood gases are required | | |

**Recommended therapy due to Downes' Score results:**

| Estimation results | Recommended measures |
|---|---|
| 3 or less, minimal distress | Monitoring of oxygen saturation of hemoglobin and gas content of the blood, clinical condition; oxygen donation or continuous positive pressure ventilation in the respiratory tract with spontaneous respiration |
| 4–7, moderate respiratory distress | Continue monitoring of oxygen saturation of hemoglobin and gas content of the blood, clinical condition; chest X-ray; detect glucose in the blood; oxygen donation or PPV, in some causes – AVL. |
| >7, severe respiratory distress | Provide lung ventilation – tracheal intubation, maintain respiratory function of the lungs by apparatus |

## TRANSIENT TACHYPNEA OF THE NEWBORN

Transient tachypnoea of the newborn (TTN) is mild self-limited respiratory disorder of near-term or term infants, which is characterized by an obstructive pattern with normal functional residual and increased total lung capacity. This respiratory disorder occures immediately after birth with pathological retaining of internal pulmonary fluid in the alveolar spaces.

**Risk Factors are:**

– Elective CS delivery;

– Macrosomia and IDM's;

– Prolonged labor;

– Excessive maternal sedation or fluid overload given to the mother, especially with oxytocin infusion;

– Delayed umbilical cord clamping;

– Absence of "catecholamine splash" in response to delivery stress;

– Acute asphyxia during delivery.

**Clinical presentations:**

– Infant is usually near-term or term and presents within 6 hrs after delivery with tachypnea (>80 breaths/min);

– Mild to moderate respiratory distress with grunting, nasal flaring, rib retraction & cyanosis;

– Auscultation reveals good air entry with or without crackles;

– Manifestations usually persist for 12–24 hrs (up to 72 hrs in more severe cases);

– Exclude other causes of respiratory distress in the first 6 hrs of life (e.g., pneumonia, RDS and other). Spontaneous improvement is an important marker of TTN.

**Diagnostics:**

– CBC with differential and CRP to rule out sepsis;

– Blood gas analysis: hypoxemia, PaCO2 is usually low (or may be mildly elevated); if respiratory failure occurs, another diagnosis should be considered;

– Chest x-ray (the typical findings in TTN are the increased prominent pulmonary vasculature, increased transparency of lung field. Sometimes accumulation of fluid in the interlobar fissures can be found);

– Chest x-ray usually shows evidence of clearing by 12–18 hrs with complete resolution by 48–72 hrs.

**Management:**

– Respiratory support: supplemental O2; may be provided by head box or nasal cannula, but nasal continuous positive airway pressure is a more effective modality (this promotes clearance of retained fluid and stents airway, decreasing obstructive component);

– Moderate fluid restriction in first day of life (no more than 60 ml/kg/d);

– Start antibiotics in case of risk for sepsis ;

– Provide maintenance fluids and electrolytes;

– Maintain thermoregulation;

– Feeding through a tube in case of respiratory rate more than 60 breaths/min

None specific therapy and follow-up is needed.

**Complications and prognosis**

– Excellent prognosis;

– Resolution within 24–72 hrs;

– Rarely complicated by pneumothorax;

– No long-term complications;

– No association w/ childhood asthma.

## MECONIUM ASPIRATION SYNDROME

Meconium aspiration syndrome (MAS) is a medical condition affecting newborn infants which can occur before, during, or immediately after birth. It describes the spectrum of disorders and pathophysiology of newborns born in meconium-stained amniotic fluid (MSAF) and have meconium within their lungs.

Meconium is the first intestinal discharge from newborns. It is a viscous, dark-green substance composed of intestinal epithelial cells, lanugo, mucus, and intestinal secretions (bile). Water is the major liquid constituent, comprising 85–95 % of meconium; the remaining 5–15 % of ingredients consists of solid constituents, primarily intestinal secretions, mucosal cells, and solid elements of swallowed amniotic fluid, such as proteins and lipids. Meconium is sterile and does not contain bacteria, which is the primary factor that differentiates it from stool. Intrauterine distress can cause passage of meconium into the amniotic fluid.

MAS more often is found in post term infants (> 40 weeks), but may occur in infants with gestational age >34 weeks. Infants produce meconium due

to varying degrees of asphyxia in utero and it leads to obstruction of large and small airways with aspirated meconium. Aspiration may occur: in utero, intranatal and in postnatal period.

**Risk Factors:**

– post-term pregnancy;
– pre-eclampsia;
– eclampsia;
– maternal hypertension;
– maternal diabetes mellitus;
– IUGR, and evidences of fetal distress (e.g., abnormal biophysical profile).

**Clinical Manifestations:**

– Meconium staining amniotic fluid (ranging from thin, greenstained fluid to thick, pea soup consistency);
– Signs of postmaturity (weight loss, meconium stained nails, skin and umbilical cord);
– Aspiration of large amounts of thick meconium which can lead to acute large;
– Partial distal airway obstruction leads to respiratory distress soon after birth. Infants with severe MAS have "barrel" chest;
– Some infants may have mild initial respiratory distress, which becomes more severe hours after delivery;
– Pneumothorax and/or pneumomediastinum;
– PPHN in severe cases;
– Hypoxia to other organs (e.g., seizures, oliguria);
– Pale or gray color of skin, cyanotic, stained skin.

**Diagnostics:**

– CBC with differential;
– Blood gas analysis;
– Surveillance for end organ hypoxic damage including kidney function tests and cranial ultrasonography.

Chest x-ray:

– Patchy infiltrates;
– Hyperinflation of the chest;
– Atelectasis;
– Pneumothorax;
– Flattening of diaphragm;
– Cardiomegally.

**Management:**

1. Prevention:

– Monitor fetal status;
– Amnioinfusion;
– Avoid harmful techniques;

Suctioning of the oropharynx before delivery of the shoulders is not recommended. Visualization of the vocal cords and tracheal suctioning before ambu-bagging should be done only if the baby is not vigorous.

2. In the Neonatal Intensive Care Unit:

– Optimal thermal environment and minimal handling;

– Respiratory care, Oxygen therapy;

– Keep stable vital sings;

– Empty stomach contents to avoid further aspiration;

– Suction frequently & perform chest physiotherapy;

– Maintain an antibiotic coverage (ampicillin and gentamicin).

– Give supplemental oxygen (maintain PaO2 at least in the range of 80–90 mmHg);

– Mechanical ventilation: in severe cases (paCO2 >60 mmHg or persistent hypoxemia (paO2 <50 mmHg);

– Surfactant therapy (may reduce the severity of respiratory illness and decrease the number of infants with progressive respiratory failure requiring support with extracorporeal membrane oxygenation (ECMO);

– Correct systemic hypotension (hypovolemia, myocardial dysfunction).

**Complications:**

– Air leak;

– PPHN;

– Pneumonia;

– Airway reactivity.

## RESPIRATORY DISTRESS SYNDROME

Respiratory distress syndrome (RDS), also known as hyaline membrane disease, occurs almost exclusively in premature infants. 1/3 infants born between the $28^{th}$ to the $34^{th}$ week, but less than 5 % of those born after the $34^{th}$ week. The disease is mainly caused by a lack of a slippery substance in the lungs called surfactant. This substance helps the lungs fill with air and keeps the air sacs from deflating. Surfactant is present when the lungs are fully developed.

Surfactant is a complex lipoprotein composed of 6 phospholipids and 4 apoproteins, which are produced by the lungs that keeps the alveoli open, making it possible for babies to breathe in air after delivery. It begins to be produced in the fetus at about 26 weeks of pregnancy. When there is not enough surfactant, the tiny alveoli collapse with each breath. As the alveoli collapse, damaged cells collect in the airways and further affect breathing ability. The baby works harder and harder at breathing, trying to reinflate the collapsed airways. As the baby's lung function decreases, less oxygen is taken in and more carbon dioxide builds up in the blood. This can lead to acidosis, which can affect other organs and systems.

**Risk Factors:**

– Prematurity;
– Maternal diabetes;
– Multiple births;
– Elective CS without labor;
– Perinatal asphyxia;
– Cold stress;
– Genetic disorders of surfactant production (e.g., surfactant protein B mutation).

**Secondary surfactant deficiency may occur in infants with the following:**

– Intrapartum asphyxia;
– Pulmonary infections (e.g., group B beta-hemolytic streptococcal pneumonia);
– Pulmonary hemorrhage;
– Meconium aspiration pneumonia;
– Oxygen toxicity along with barotrauma or volutrauma to the lungs;
– Congenital diaphragmatic hernia and pulmonary hypoplasia.

## Clinical Manifestations

Progressive signs of respiratory distress are noted soon after birth and include the following:

– Tachypnea (more than 60 bpm);
– Nasal flaring;
– Expiratory grunting (from partial closure of glottis)
– Subcostal and intercostal retractions;
– Apnea/ irregular respiratory pattern;
– Rales (crackles);
– Diminished breath sounds;
– Cyanosis;
– Extremely immature in neonates may develop apnea and/or hypothermia.

## Investigations

1. Clinical Assessment of Respiratory Distress

### Silverman-Anderson index

| **Feature** | **Score 0** | **Score1** | **Score 2** |
|---|---|---|---|
| Chest Movement | Equal | Respiratory Lag | Seesaw Respiration |
| Intercostal Spaces | None | Minimal | Marked |
| Xiphoid Retractions | None | Minimal | Marked |
| Nasal Flaring | None | Minimal | Marked |
| Expiratory Grunting | None | Audible with stethoscope | Audible without stethoscope |

Results:

- Score 10 = Severe respiratory distress;
- Score ≥ 7 = Impending respiratory failure;
- Score 0 = No respiratory distress;

Chest movement: Synchronized vs minimal lag or sinking of the upper chest as the abdomen rises. In the most extreme instances, a seesaw-like movement of the chest and abdomen is observed and would be given a score of 2.

Intercostal retractions: Retraction between the ribs is rated as none, minimal or marked. This indicates loss of functional residual capacity.

Xiphoid retractions: Similarly, retraction below the xiphoid process are rated as none, minimal or marked.

Nasal flaring: Normally, there should be no nasal flaring. Minimal flaring is scored 1 and marked flaring is scored 2.

Expiratory grunting: Grunting that is audible with a stethoscope is scored 1, and grunting that is audible without using a stethoscope is scored 2.

2. Laboratory and instrumental methods:

– Blood gas analysis. Blood gases show respiratory and metabolic acidosis along with hypoxia. Respiratory acidosis occurs because of alveolar atelectasis and/or overdistension of terminal airways. Metabolic acidosis is primarily lactic acidosis, which results from poor tissue perfusion and anaerobic metabolism.

– Sepsis work-up (CBC with differential, CRP, and blood culture) to rule out early-onset sepsis.

– Serum glucose and electrolyte levels monitoring

– Chest x-ray: findings can be graded according to the severity

Grade 1 (mild cases): fine granulation with marked air bronchograms within the borders of heart shadow are clearly detected

Grade 2: typical diffuse network-granular picture with moderately decreased transparency of the lung fields, air bronchograms beyond the borders of cardiac shadow and thymus are found.

Grade 3: fused shadows formed by numerous network-granular shadows are seen, air bronchograms reach the branches of the 2$^{nd}$ and 3$^{rd}$ degree, general transparency of lung fields decreases.

Grade 4: “white lungs”, complete shadow of all the lung fields, absent air bronchograms, cardiac shadow is not outlined.

**Differential Diagnosis:**

– Acute anemia;
– Aspiration syndromes;
– Gastroesophageal reflux;
– Hypoglycemia;
– Pneumomediastinum;
– Pneumonia;

– Pneumothorax;
– Polycythemia;
– Sudden Infant Death Syndrome;
– Transient Tachypnea of the Newborn.

Several diagnoses may coexist with and complicate the course of respiratory distress syndrome, including the following:

– Pneumonia – usually secondary to group B beta-hemolytic streptococci and often coexists with respiratory distress syndrome;
– Metabolic problems – hypothermia, hypoglycemia;
– Hematologic problems – anemia, polycythemia, jaundice;
– Transient tachypnea of the newborn – usually occurs in term or near-term neonates, often after cesarean delivery; the chest radiograph of an infant with transient tachypnea shows good lung expansion and, often, fluid in the horizontal fissure.
– Aspiration syndromes – may result from aspiration of amniotic fluid, blood, or meconium; aspiration syndrome is observed in more mature infants and is differentiated by obtaining a history and by viewing the chest radiographs.
– Pulmonary air leaks – pneumothorax, interstitial emphysema, pneumomediastinum, pneumopericardium; in premature infants, these complications may be due to excessive positive-pressure ventilation (in rare cases, spontaneous pneumothorax may occur in large infants).
– Congenital anomalies of the lungs – diaphragmatic hernia, chylothorax, congenital cystic adenomatoid malformation of the lung, lobar emphysema, bronchogenic cyst, pulmonary sequestration.
– Congenital anomalies of the heart.

## Management

Prevention:

– Antenatal corticosteroid therapy (betamethasone 12 mg/dose IM for 2 doses, 24 hrs apart, or dexamethasone 6 mg/dose IM for 4 doses, 12 hrs apart) for pregnant women 24–34 wks' gestation at high risk of preterm delivery within the next 7 days.
– Prophylactic surfactant therapy in preterm infants <27 wks' gestation.
– Early CPAP administration in the delivery room.

Treatment:

– Follow general management rules.
– Administer oxygen (depending on the severity of illness).
– Initiate CPAP as early as possible in infants with mild RDS who require an $FiO_2$ below 0.4 to maintain the target $SaO_2$ and have $paCO_2$ <55–60 mmHg.
– Start MV if respiratory acidosis ($PaCO_2$ >60 mmHg, $PaO_2$ <50 mmHg or $SaO_2$ <90%) with an $FiO_2$ >0.5, or severe frequent apnea.

– Administer surfactant therapy: early rescue therapy within 2 hrs after birth is better than late rescue treatment when the full picture of RDS is evident.

Surfactant is given in a dose of 100 mg/kg through the endotracheal tube in small aliquots with intermittent bagging to prevent desaturation during administration and it should be followed by ventilatory support.

**Complications:**

– Septicemia;
– Bronchopulmonary dysplasia (BPD);
– Patent ductus arteriosus (PDA);
– Pulmonary hemorrhage;
– Apnea/bradycardia;
– Necrotizing enterocolitis (NEC);
– Retinopathy of prematurity (ROP);
– Hypertension;
– Failure to thrive;
– Intraventricular hemorrhage (IVH);
– Periventricular leukomalacia (PVL) – with associated neurodevelopmental and audiovisual handicaps.

## PNEUMONIA

Neonatal pneumonia is lung infection in a neonate. Onset may be within hours of birth and part of a generalized sepsis syndrome or after 7 days and confined to the lungs. Signs may be limited to respiratory distress or progress to shock and death.

Pneumonia is the most common invasive bacterial infection after primary sepsis. Early-onset pneumonia is part of generalized sepsis that first manifests at or within hours of birth. Late-onset pneumonia usually occurs after 7 days of age, most commonly in neonatal ICUs among infants who require prolonged endotracheal intubation because of lung disease (called ventilator-associated pneumonia).

3 types of pneumonia are defined:

- Congenital Pneumonia;
- Intranatal Pneumonia;
- Postnatal Pneumonia.

Main causes of pneumonia development in neonates are:

– Prematurity;
– Early rupture of membranes;
– Maternal temperature > 38 °C;
– Foul smelling amniotic fluid;
– Fetal tachycardia;
– Meconium aspiration;
– Common pathogen agents.

## Etiology

Common: GBS, gram negative organisms (e.g. E.Coli, Klebsiella, Pseudomonas), Staph. aureus, Staph. epidermidis and Candida.

– **Transplacental pneumonias**: more often cytomegalic inclusion disease, herpes, rubella, toxoplasmosis, lues, listeriosis.

– **Intranatal pneumonias**: genital micoplasmosis, anaerobic bacteria, baccillus tuberculosis, hemophilic infection.

– **Posttranatal pneumonias** more often it is nosocomial agents: klebsiela, coagulazonegative staphylococcus, blue pus bacillus, colon bacillus, Proteidae

Less common: acquired viral infections (e.g., HSV, CMV).

## Clinical Manifestations

– Manifestations are apparent prior to delivery (e.g., fetal distress, tachycardia), at delivery (e.g., perinatal asphyxia) or after a few hours (e.g., respiratory distress, shock).

– Early manifestations may be nonspecific (e.g., poor feeding, lethargy, irritability, cyanosis, temperature instability and the overall impression that the infant is not well).

– Respiratory distress, cyanosis, apnea and progressive respiratory failure may become evident. In preterm infants, these signs may be superimposed upon RDS or BPD.

– In a ventilated infant, the most prominent change may be the need for an increased ventilatory support.

– Signs of pneumonia (dullness to percussion, change in breath sounds, rales or rhonchi) are difficult to appreciate.

– Pyogenic organisms (e.g., GBS). Onset is usually during the first hours or days of life with rapidly progressive circulatory collapse and respiratory failure.

Respiratory assessment:

- Tachypnea;
- Apnea, irregular breathing pattern;
- Grunting;
- Retractions;
- Nasal flaring;
- Bubbling rales, rhonchi;
- Cyanosis.

Clinical assessment:

– Gray, pale skin color;

– Lethargy;

– Temperature instability;

– Skin rash - pettechia;

– Tachycardia;

– Hypoperfusion;
– Oliguria.

Clinical evidence depends on the gestional age and concomitant antenatal and perinatal pathology.

## Investigation

X-Ray:

– Patchy infiltrates (aspiration);
– Bilateral diffuse granular pattern;
– Loss of volume;
– Densities increase.

If the neonate has an underlying RDS or BPD, it is difficult to determine whether the radiographic changes represent a new process or worsening of the underlying disease.

Laboratory research

– Blood test (CBC) – leukopenia and leukocytosis with the shift to the left leucocytal index rise);
– Microscopy of the stained sputum smear and its coloration by Gram;
– Bacterial culture of the tracheal aspirate and blood culture determination;
– Blood gases determination;
– Glucose level in blood;
– Sphygmooxymetric monitoring;
– Lumbal punction and liquor investigation.

## Differential diagnosis between congenital pneumonia and respiratory disorders syndrome

| Diagnostic criteria | Respiratory disorders | Congenital pneumonia |
|---|---|---|
| Mother anamnesis | Detachment of placenta, diabetes mellitus | Infection diseases, long term waterless period |
| Gestational age | > 34 weeks | any |
| Time of appearance | During first hours of life | During first day of life |
| Intoxication | no | yes |
| Inflammatory changes in the blood analysis | no | yes |
| C-reactive protein | no | yes |
| Lecitinsphingomielinic index | Lass than 2 | More than 2 |
| Auscultation | Diminished breath sounds and diffuse crepitation | Diminished or round breath sounds, fine moist rales and crepitation over the locus |
| X-ray film | Nodoso-reticulary net, air bronchogram, “white” lungs | Local or segmental infiltration |
| Course of diasease | Clinical symptoms reduction up to 3 days | Clinical symptoms induction up to 2–3 days |

**Management**

– Follow general management rules for respiratory disorders.

– Initiate ampicillin and gentamicin IV; modify according to culture results and continue therapy for 14 days.

– If there is a fungal infection, an antifungal agent is used.

## AIR LEAK SYNDROMES

Pneumothorax.
Pulmonary Interstitial Emphysema
Pneumomediastinum

**Risk Factors of air leak syndromes:**

– MV;
– MAS;
– surfactant therapy without decreasing pressure support in ventilated infants;
– vigorous resuscitation;
– prematurity;
– pneumonia.

**I. Pneumothorax.**

Pneumothorax is a collection of air or gas between the lung and the chest wall that causes part or all of a lung to collapse.

Features:

- Spontaneous pneumothorax may be asymptomatic or only mildly symptomatic (i.e., tachypnea and increase of oxygen needs).
- In unilateral cases, chest asymmetry is noted, hyper-resonant chest on percussion and mediastinum shift to the opposite side.
- If the infant is on ventilatory support, he/she will have sudden onset of clinical deterioration (i.e., cyanosis, hypoxemia, hypercarbia and respiratory acidosis associated with decreased breath sounds and shifted heart sounds).
- Tension pneumothorax (a life-threatening condition) leads to decreasing of cardiac output and obstructive shock; urgent drainage prior to a radiograph is mandatory.
- Chest x-ray may show just minimal differences in lucency of lung fields (in case of spontaneous pneumothorax) or jet black lung and shift of mediastinum to the opposite side (in case of tension pneumothorax).

**II. Pulmonary Interstitial Emphysema.**

Pulmonary interstitial emphysema (PIE) is a common comorbidity of neonates that is mainly associated with the etiologic triad of prematurity, respiratory distress syndrome in the first 48 hrs of life, and mechanical ventilation therapy.

Chest x-ray reveals radiolucencies (linear (radiate from lung hilum) or cyst-like (1–4 mm in diameter)).

**III. Pneumomediastinum**

Pneumomediastinum (PM) is defined as a mediastinal air leak.

The infant is often asymptomatic. This condition may follow gas trapping associated with the neonatal respiratory distress syndrome, pneumonia, or the use of mechanical ventilation. Neonatal pneumomediastinum is also associated with the aspiration of meconium or blood and birth-related trauma.

Chest x-ray showes a large quantity of air in the mediastinum which may cause elevation of the thymus and partial collapse of the lungs on both sides.

## Differential diagnosis

- Obstructed/displaced endotracheal tube.
- Pneumopericardium:

– Acute onset;
– Drop in BP, weak/absent pulse;
– Distant/absent heart sounds;
– May transilluminate;
– Chest x-ray: halo around heart;
– Treatment: pericardiocentesis.

- Pneumomediastinum:

– May be asymptomatic, unless accompanied by pneumothorax;
– May present w/ respiratory distress, but not w/ sudden deterioration;
– Chest x-ray: “wind-blown spinnaker sail” (thymus elevated off heart);
– Usually resolves spontaneously, but may progress to pneumothorax or pneumopericardium.

- Congenital lobar emphysema:

– Overdistention of one lobe (usually left upper) secondary to air trapping;
– Onset usually not acute;
– May transilluminate.

- Atelectasis with compensatory hyperinflation:

– Chest x-ray: compensatory emphysema mimics pneumothorax;
– Onset usually not acute;
– May transilluminate.

## Management

- Prevention: judicious use of ventilatory support, close attention to distending pressures and Ti. Appropriate weaning as the clinical condition improves.
- Follow general management rules.
- Specific therapy

• Conservative therapy (in spontaneous pneumothorax and nonventilated cases): close observation of the degree of respiratory distress and SaO2 aiming at spontaneous resolution.

• Decompression of pneumothorax.

## CONGENITAL ABNORMALITIES OF THE LUNG AND THORAX

- Congenital Heart Disease (CHD);
- Congenital Diaphragmatic Hernia (CDH);
- Congenital Cystic Adenomatoid Malformation;
- Tracheal Abnormalities;
- Esophageal Atresia;
- Pulmonary Hypoplasia.

### Congenital Heart Disease

- Defect presents at birth is often diagnosed while the early ultrasound;
- Increased risk factors:
  – Parents have CHD?
  – Siblings have CHD?
  – Maternal diabetes;
  –If mother had German measles, toxoplasmosis while pregnancy, or if mother has HIV+;
  –Alcohol usage during pregnancy;
  –Cocaine usage during pregnancy.

**Types of CHD:**

– Acyanotic (or white). Blood usually returns to the Right side of heart passes through lungs. If CHD presences the defect in heart wall, or obstructed valve or artery may be diagnosed.

- Pink baby;
- Child activity is within norm;
- Blood sate is within norm;

– Cyanotic (or blue). It is noticed mixing of arterial blood with venous blood.

They are: Botallo duct patency, patent foramen ovale (PFO), atrial septal defect (ASD), ventricular septal defect (VSD).

- Blue baby;
- Child activity low;
- Blood sate is low.

**Respiratory Assessment:**

Respirations:

– Normal;

– Tachypnea.

Saturations depend on defect:
– Acyanotic CHD sates are more normal;
– Cyanotic CHD sates are low.

**Clinical Assessment:**

*Heart rate:*
– Slow, fast, variable;
– Different noises in heart.

*Blood Preassure:*
– Check in all 4 extremities.
– Pulses check in all extremities.

*Color of skin:*
– Acyanotic – pink;
– Cyanotic – blue.

**Labs and Tests:**
– Arterial blood gases (ABGs) – depend on defect;
– Lactic Acid increase.

**Chest X-Ray:**
– Heart shape and size;
– Pulmonary blood flow.

**Echocardiogram:**
– The best test to diagnose.

## PERSISTENT PULMONARY HYPERTENSION OF NEONATES (PPHN)

PPHN is a rare lung disorder characterized by increased pressure in the pulmonary artery (pulmonary hypertension). The pulmonary artery carries oxygen-poor blood from the lower chamber on the right side of the heart (right ventricle) to the lungs where it picks up oxygen.

Pulmonary hypertension is a result of severe secondary hypoxemia where right shunt is larger than left through PFO and/or PDA. Usually PPHN affects term or in near-term infants. It may be extremely difficult to manage such patients. If newborn is not responding to available therapy it is necessary to transport patient to an ECMO center.

### History

■ Meconium-stained amniotic fluid
■ Fetal distress: abnormal fetal heart race tracings, abnormal biophysical profile
■ Perinatal asphyxia (hypoxia, hypercapnia, acidosis)
■ Resuscitation req in delivery room
■ IUGR

■ Evidence of infection (e.g., chorioamnionitis)
■ Polycythemia/hyperviscosity
■ Postdates GA
■ Maternal use of nonsteroidal anti-inflammatory drugs (e.g., aspirin, ibuprofen)
■ Hydrops fetalis
■ Congenital malformations
– Congenital diaphragmatic hernia (CDH)
– Congenital cystic adenomatoid malformation (CCAM)
– Pulmonary hypoplasia
– Congenital heart disease

**Signs**

■ Respiratory distress soon after birth (tachypnea, grunting, flaring, retracting)
■ Cyanosis
■ Post-dates appearance: peeling skin, dystrophic umbilical cord, wasting
■ Meconium-stained nails, skin, umbilical cord, hair
■ Meconium-stained amniotic fluid suctioned from trachea
■ Heart murmur (often due to tricuspid regurgitation); S2 often single due to increased pulm artery pressure
■ Scaphoid abdomen w/ CDH
■ Oligohydramnios sequence w/ pulmonary hypoplasia

**Tests**

■ Chest x-rays (findings vary w/ underlying etiology)
– Hypovascularity (esp w/ primary PPHN)
– Hyperinflation, diffuse, patchy infiltrates (meconium aspiration syndrome or congenital pneumonia)
– Granular, ground-glass appearance w/ respiratory distress syndrome
– Stomach/intestinal gas present in chest and chest shifted to opposite side w/ CDH
– Pneumothorax or pneumomediastinum
– Pleural effusions w/ hydrops fetalis
– "Wet-lung" appearance
– Cystic areas in lungs (w/ CCAM)
■ Arterial blood gas
– Hypoxemia
– Hypercapnia
– Metabolic acidosis
– Preductal to postductal d PaO2 >=20–25 mmHg difference w/ right-to-left shunting across patent ductus arteriosus; not seen if shunting only at level of foramen ovale;

– Marked lability in oxygenation (PaO2 values may vary markedly from one moment to next on same or minimally different ventilator settings)

■ O2 saturations (pulse oximetry)

– Frequently low

– Preductal to postductal d PaO2 >=20–25 mmHg difference w/ right-to-left shunting across patent ductus arteriosus; not seen if shunting only at level of foramen ovale;

– Marked lability in oxygenation (O2 sats may vary markedly from one moment to next on same or minimally different ventilator settings)

■ Hyperoxia test

– Obtain arterial blood gas after 10–15 min in FiO2 1.0 (some advise intubation and assisted ventilation for hyperoxia test)

– W/ parenchymal lung disease, PaO2 should be >100 mmHg, except w/ V/Q mismatching

– If cyanotic heart disease, PaO2 changes little

– If PPHN, PaO2 may either rise slightly or remain same; w/hyperventilation w/ 100% O2 for 10–15 min to decrease PaCO2 to 25–30 mmHg & increase pH to >=7.50, a dramatic increase in PaO2 suggests PPHN

*n o t e:* Some believe even brief hyperventilation is contraindicated

■ Echocardiography (mandatory)

– R/o structural congenital heart disease (total anomalous pulmonary venous return is particularly difficult to exclude)

– Assess direction of shunting across the foramen ovale, ductus arteriosus

– Evaluate size of RA & RV

– Estimate pulmonary artery pressure (by evaluating regurgitant tricuspid "jet")

– Assess myocardial function

■ CBC/differential

– Check hemoglobin, hematocrit to r/o polycythemia

– Leukocytosis w/ left shift on WBC differential common in pneumonia, sepsis, meconium aspiration syndrome, w/ pneumothorax

– Increased nucleated RBC suggests in utero hypoxia

– Increased lymphocytes soon after birth w/ recent in utero hypoxia

■ Other

Fatal cases should have autopsy performed to assess for histopathological findings (e.g., capillary-alveolar dysplasia)

– Evaluation for asphyxia, meconium aspiration syndrome, pneumonia, other underlying etiology.

**Differential diagnosis:**

■ Severe pulmonary parenchymal disease (w/ or w/o associated PPHN)

– Meconium aspiration syndrome
– Congenital pneumonia
– Respiratory distress syndrome
■ Congenital cyanotic heart disease

**Management:**

■ Prenatal/intrapartum

– Identify high-risk infants: Structural anomalies, Evidence of infection, Post-dates, Abnormal fetal heart rate tracings

■ Delivery room

– Optimize resuscitation
– Avoid hypothermia, hypoglycemia, hypovolemia, acidosis

■ Postnatal: general measures

– Establish diagnosis
– Treat underlying disorder
– Correct mechanical problems, such as: pneumothorax, pleural effusions, other
– Maintain adequate systemic BP, cardiac output: volume expansion, inotropes as necessary (dobutamine, dopamine)

■ Oxygen

- Use sufficient to maintain O2 saturations >=95 % or PaO2 levels 50–80 mmHg (some clinicians prefer even higher levels, but no data support this as better approach)
– Follow oxygenation index (OI) to determine effect of mgt, when to escalate to other therapies: OI = (mean airway pressure)(FiO2)(100)/PaO2

■ Mechanical ventilation

– Conventional mechanical ventilation – i.e., intermittent mandatory ventilation – typically the primary mode
– Patient-triggered ventilation (Assist/Control, SIMV) may be considered
– Vol-controlled ventilation an option
– Many employ hyperventilation/alkalosis: take advantage of vasodilatory effect of alkalosis, hypocapnia on pulmonary vasculature; decrease PaCO2 to the "critical" value: below this, sharp rise in PaO2 or O2 saturation

***Hypocapnia may be harmful to brain, lungs;*** avoid PaCO2<25–30 mmHg

– Many advocate "gentle" ventilation (lower PaO2s & higher PaCO2s than above) to minimize ventilator support, prevent "volutrauma"
– No randomized, controlled trials demonstrate superiority of any mechanical ventilation method
- When weaning, make slow, small changes in ventilator settings (because of lability, infants may "flip-flop" w/ large changes)

■ Pharmacologic alkalosis: sodium bicarbonate to achieve alkalosis
– pH usually kept above 7.50
– No efficacy data

■ Consider sedation w/ mechanical ventilation, but avoid suppression of spontaneous ventilatory effort

■ Minimize stimulation
– Avoid loud noises, consider ear plugs
– Consider mask
– Minimize handling (needle punctures, suctioning, chest physiotherapy, etc.)

■ Surfactant
– Efficacious for respiratory distress syndrome
– May be of benefit w/ meconium aspiration syndrome, congenital pneumonia. Give w/ OI >= 15–20; some use at lower levels; use standard (or 1.5× ) dose

■ High-frequency ventilation (HFV)
– No clear-cut advantages of HFV
– Some consider high-freq oscillatory ventilation to "optimize" lung volume
Hyperventilation may be easier to achieve w/ HFV
– Consider HFV w/ air leaks
– Consider HFV w/ OI >=10–15 on conventional mechanical ventilation

■ Inhaled nitric oxide (iNO)
– Selective pulmonary vasodilator
– Dose: 5–20 ppm
– Some use iNO in combo w/ high-freq oscillatory ventilation

■ Non-iNO pulmonary vasodilators
– None specifically approved by FDA for PPHN pts
– Sildenafil, enterally; case reports only
– IV magnesium sulfate – effective in several small trials
– Prostaglandins – little data to support efficacy
– Adenosine infusion – minimal data
– Nitroglycerine – no data to support
– Arginine infusion – experimental

■ Extracorporeal membrane oxygenation (ECMO)
– Therapy of last resort
– Generally, rescue modality when predicted mortality >=80 % (typically at OI levels >=40)
– Overall survival > 80%; best w/ meconium aspiration syndrome, worst w/ CDH
– Systemic anticoagulation necessary; bleeding complications not uncommon
– Venoarterial bypass results in loss of right carotid artery

**Specific therapy** depends on underlying etiology.

**Follow-up**

■ Follow for neurodevelopmental outcomes, pulmonary complications (chronic lung disease, altered pulmonary mechanics, reactive airway disease)

■ Depending on underlying etiology

**Complications and Prognosis**

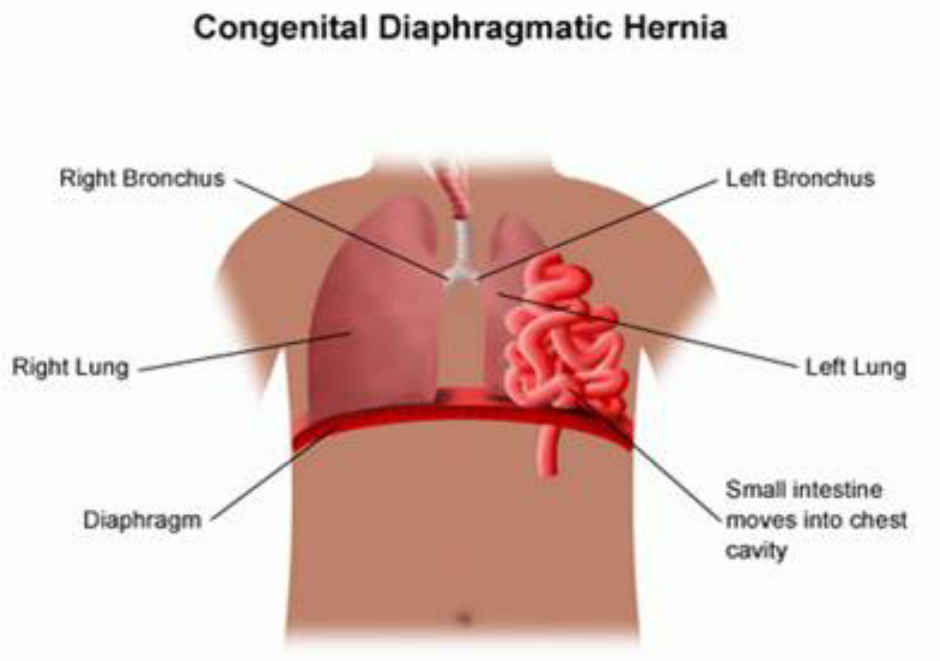
■ 10–20 % mortality

■ Chronic lung disease (need for O2/mechanical ventilation at age 28 days) in 5–10 %

■ Abnormal neurodevelopmental outcome in 20–40 % of survivors: cerebral palsy, seizures, cognitive delays, deafness, autism.

## CONGENITAL DIAPHRAGMATIC HERNIA (CDH)

Congenital Diaphragmatic hernia is a condition characterized by a defect in the diaphragm leading to protrusion of abdominal contents into the thoracic cavity interfering with normal development of the lungs.




The defect may range from a small aperture in the posterior muscle rim to complete absence of diaphragm.

**History and Physical**

■ Usually diagnosed antenatally: heart and stomach in same plane on US; polyhydramnios

■ Diagnosed postnatally in term newborn w/ significant respiratory distress, asymmetric breathing, scaphoid abdomen

■ May be associated w/ congenital heart disease

■ No pattern of inheritance

**Diagnostics**

■ Chest X-ray

■ Echocardiogram

**Differential diagnosis**

■ Cystic adenomatoid malformation, congenital, pulmonary

■ Diaphragm eventration

**Management**

■ Intubation ASAP after birth, do not suppress spontaneous respiration, permissive hypercapnea, minimize baro/volutrauma

■ Base treatment decisions on preductal SaO2

■ Experience w/ nitric oxide, surfactant, oscillating ventilators largely anecdotal

■ ECMO only w/:

– Evidence of sufficient lung development based on preductal SaO2 and Expected mortality >90 % despite best conventional care

■ Elective operation when stable and weaned from most respiratory support

■ Reduce hernia, build diaphragm, close abdomen at operation

■ Continue above respiratory strategy postoperation

■ No specific therapy is available

**Follow-up**

Coordinated multidisciplinary (Surgical, Pulmonary, Cardiological, Developmental, GI) follow-up is optimal.

**Complications and prognosis**

■ Chronic lung disease

■ Pulmonary hypertension

■ Most patients survive w/ normal heart/lung function, but should be followed as needed

■ Foregut dysmotility most persistent problem; usually managed by prokinetic agents/H2 blockers; surgery seldom needed except when stomach will not empty

## CONGENITAL CYSTIC ADENOMATOID MALFORMATION (CCAM)

Congenital cystic adenomatoid malformation of the lung is a relatively common structural lung abnormality. It frequently presents on antenatal scanning as a cystic lesion. It may be associated with fetal hydrops if it is large enough to obstruct venous return to the heart.

**History and Physical**

■ Abnormality often identified on prenatal US, but exact nature of malformation may not be clear until birth

■ Suspected postpartum because of:

– Respiratory distress

– Asymmetric breathing, but normal contour to abdomen

– High-output cardiac failure

– Pulmonary infection (rare in newborn)

**Diagnostic**

■ Chest X-ray

■ CT scan w/ IV contrast

■ MRI

■ Angiography (rarely required)

**Differential diagnosis**

■ Congenital cystic adenomatoid malformation

– Hamartoma of lung w/ cystic, solid components

– Can involve single lobe or whole lung

– May have same physiology as congenital diaphragmatic hernia

■ Bronchogenic cyst

– Cystic dilatation of non-communicating airspace

– Usually upper lobes or behind carina

■ Lobar emphysema

– Paucity of bronchial cartilage w/ air trapping

– Usually in upper lobes

■ Pulm sequestration

– Persistence of systemic arterial vessel from aorta to "sequestered" lobe

– Always lower lobe

**Management**

■ Usually resection or enucleation (bronchogenic cyst only)

■ Sometimes lobectomy

■ Limited observation only if asymptomatic, very small & resolving

■ No specific therapy is available

With proper treatment no complications are observed.

## CONGENITAL AIRWAY ABNORMALITIES (CAA)

The etiologies are varied and include, choanal atresia, pyriform aperture stenosis, and rarely tumors such as glioma, encephalocele, teratoma, or dermoid. More common upper airway congenital anomalies include laryngomalacia, vocal cord paralysis, and subglottic stenosis. It occurs rare than pulmonary parenchymal diseases. Clinical presentations are often with significant respiratory distress.

Stridor is a term used to describe noisy breathing in general, and to refer specifically to a high-pitched intermittent sound associated with croup, respiratory infection, and airway obstruction. It may be an important key to diagnose the abnormality.

**Types of CAA**

1. Supraglottic:

Nose - Choanal Atresia; Craniofacial (Pierre Robin Complex); Macroglossia; Tumors (Hemangioma).

2. Glottic:

– Vocal Cord Paralysis.
– Tumors and Cysts:
– Hemangioma, Cystic Hygroma, Teratoma;

- Tracheal Esophageal Fistula/ Atresia;
- Webs;
- Trauma.

3. Subglottic:

Stenosis – congenital or acquired; Webs; Atresia; Tumors.

4. Trachea:

Tracheomalacia; Stenosis; Cyst; Atresia.

5. Extrinsic:

Vascular Ring; Mediastinal Mass.

**Causes and physical**

- Fast of augmentation of symptoms;
- Presence of feeding abnormalities;
- Previous infection;
- Previous intubation or trauma;
- Presence of associated cardiopulmonary abnormalities.

Respiratory Assessment:

– Tachypnoe, retractions.

- Breath Sounds:

Stridor is the MOST important physical sign created by airway turbulence and indicates obstruction:

– Inspiratory dyspnoe – implies supraglottic or glottic abnormalities;
– Expiratory dyspnoe - implies intrathoracic abnormalities;
– Mixed dyspnoe - implies subglottic abnormalities.

**Clinical Assessment:**

– Heart Rate: tachycardia; bradycardia when obstruction is present.
– Cyanotic color of skin:
– Lethargy;
– Irritability;
– Feeding Difficulty.

Bronchoscopy is used for evaluating abnormality.

Possible Surgical Interventions:

– Cricoid Split;

– Tracheostomy;

– Excise Hygroma;

– Place stents.

As a conclusion should be noted that almost all respiratory disorders of newborns have the same clinical sings. That is why all infants with such disorders require the careful attention, high level of medical stuff and should be treated wholly due to guidelines.

## QUESTIONS FOR SELF-CONTROL

1. Definition of the surfactant system of lungs. Factors of respiratory disorders syndrome development in the neonate.

2. Mechanism of hyoline membranes development. Methods of antenatal prophylaxis.

3. Evaluation of respiratory failure severity in respiratory disorders (SRD) syndrome using Silverman`s and Downes` scales.

4. Methods of antenatal prediction and postnatal diagnostics of SRD. Radiological signs of SRD.

5. Principles of SRD treatment in newborns. Respiratory support. Surfactant therapy.

6. Classification of the pneumonia in the neonate depending on the way and term of infection. Etiology.

7. Risk factors of the pneumonia development in the neonate. Pathogenesis.

8. Features of pneumonia course in newborns depending on the sources of infection and aetiology.

9. Principles of diagnostics and treatment of the pneumonia in the neonate. Features of aethiological therapy.

## TEST

**1.** A term, 4200-g female infant is delivered via cesarean section because of cephalopelvic disproportion. The amniotic fluid was clear, and the infant cried almost immediately after birth. Within the first 15 min of life, however, the infant’s respiratory rate increased to 80 breaths per min, and she began to have intermittent grunting respirations. The infant was transferred to the level 2 nursery and was noted to have an oxygen saturation of 94%. The chest radiograph showed fluid in the fissure, overaeration, and prominent pulmonary vascular markings.

The most likely diagnosis in this infant is:

A. Diaphragmatic hernia.

B. Meconium aspiration.

C. Pneumonia.

D. Idiopathic respiratory distress syndrome.

E. Transient tachypnea of the newborn.

**2.** What are definitive X-ray sings in newborn with severe respiratory distress syndrome:

A. Bowel, stomach, liver in chest.

B. "White lungs", complete shadow of all the lung fields, absent air bronchograms, heart shadow is not outlined.

C. Hyperinflation of the chest, atelectasis, pneumothorax, flattening of diaphragm, cardiomegally.

D. The increased prominent pulmonary vasculature, increased transparency of lung field.

E. Patchy infiltrates (aspiration), bilateral diffuse granular pattern, loss of volume, densities increase.

**3.** A mother delivers a neonate with meconium staining and Apgar scores of 3 at 1 and 5 min of life. She had no prenatal care and the delivery was by emergency cesarean section for severe fetal bradycardia. Which of the following sequelae could be expected to develop in this intubated neonate with respiratory distress?

A. Sustained rise in pulmonary arterial pressure.

B. Hyperactive bowel sounds.

C. Microcephaly with micrognathia.

D. Cataracts.

E. Thrombocytosis.

**4.** A previously healthy full-term infant has several episodes of duskiness and apnea during the second day of life. Diagnostic considerations should include which of the following?

A. Hemolytic anemia.

B. Congenital heart disease.

C. Idiopathic apnea.

D. Harlequin syndrome.

E. Hyperglycemia.

**5.** The infant is now 7 weeks old. She has been extubated for 2 weeks and still requires oxygen to maintain her saturation above 93 %. Her chest radiograph now reveals patchy, fluffy infiltrates with areas of lucency. She requires daily diuretic treatment. What is the diagnosis?

A. Bronchopulmonary dysplasia.

B. Respiratory distress syndrome (hyaline membrane disease).

C. Pulmonary interstitial emphysema.
D. Bronchiolitis.
E. Primary pulmonary hypertension.

**6.** A newborn infant develops respiratory distress immediately after birth. His abdomen is scaphoid. No breath sounds are heard on the left side of his chest, but they are audible on the right. Immediate intubation is successful with little or no improvement in clinical status. Emergency chest x-ray is shown bowel, stomach, liver in chest. The most likely explanation for this infant's condition is

A. Pneumonia.
B. Cyanotic heart disease.
C. Diaphragmatic hernia.
D. Choanal atresia.
E. Pneumothorax.

**7.** What is most common causes of respiratory distress syndrome in newborn:

A. Mechanical obstruction.
B. Absence of "catecholamine splash" in response to delivery stress.
C. Prematurity, asphyxia.
D. Prolonged discoordinated labor activity.
E. Pathogen agents.

**8.** A full-term newborn infant is having episodes of cyanosis and apnea, which are worse when he is attempting to feed, but he seems better when he is crying. The most important next step to quickly establish the diagnosis is

A. Echocardiogram.
B. Ventilation perfusion scan.
C. Passage of catheter into nose.
D. Hemoglobin electrophoresis.
E. Bronchoscopic evaluation of palate and larynx.

**9.** A postterm infant is born at home after a prolonged and difficult labor. The maternal grandmother brings the infant to the hospital at 1 h of life because of fast breathing. Grandmother notes that the child spit up some dark brown particulate fluid shortly after birth. Physical examination reveals an infant in marked respiratory distress. Other findings include both an umbilical cord and flaking skin with a yellow-green hue. Chest radiograph reveals patchy infiltrates bilaterally. What is the diagnosis?

A. Pneumothorax.
B. Asthma.
C. Meconium aspiration.
D. Transient tachypnea of the newborn.
E. Bacterial pneumonia.

**10.** After an uneventful labor and delivery, an infant is born at 32 weeks' gestation weighing 1500 g. Respiratory difficulty develops immediately after birth and increases in intensity thereafter. The child's mother previously lost an infant because of hyaline membrane disease. At 6 h of age, the child's respiratory rate is 60 breaths per min. Examination reveals grunting, intercostal retraction, nasal flaring, and marked cyanosis in room air. Physiologic abnormalities compatible with these data include

A. Decreased lung compliance, reduced lung volume, left-to-right shunt of blood.

B. Decreased lung compliance, reduced lung volume, right-to-left shunt of blood.

C. Decreased lung compliance, increased lung volume, left-to-right shunt of blood.

D. Normal lung compliance, reduced lung volume, left-to-right shunt of blood.

E. Normal lung compliance, increased lung volume, right-to-left shunt of blood.

**Standards of answers to tasks: 1E, 2B, 3A, 4B, 5A, 6C, 7C, 8C, 9D, 10B.**

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https://emedicine.medscape.com/article/974110-overview

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# APPENDIX

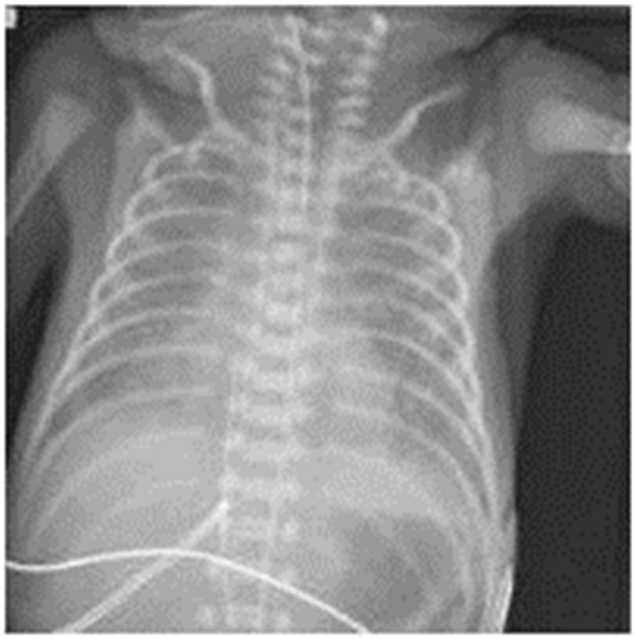


*Fig. 1. Meconium aspiration syndrome*

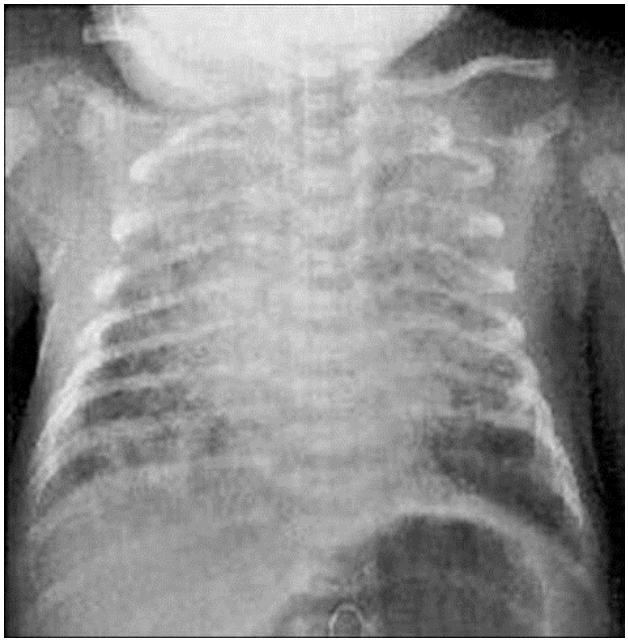


*Fig. 2. Meconium aspiration syndrome*

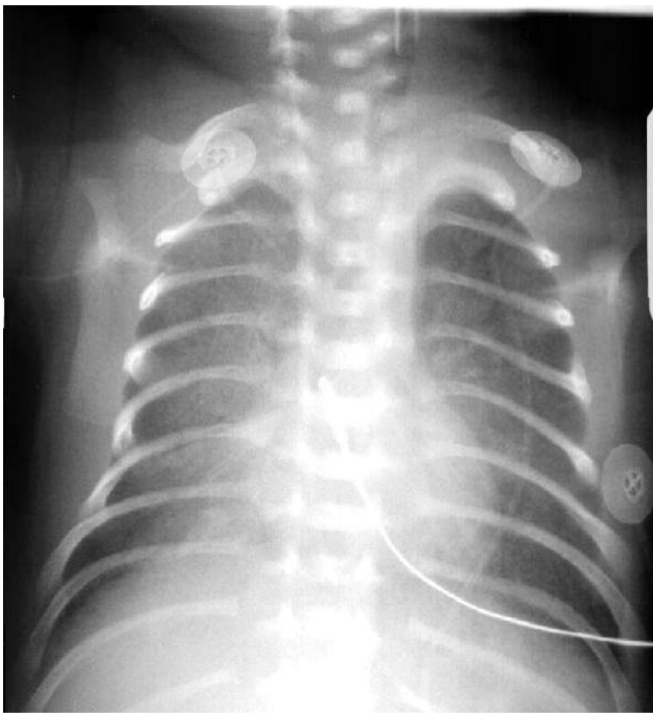


*Fig. 3. Pespiratory distress syndrome. Early stage*

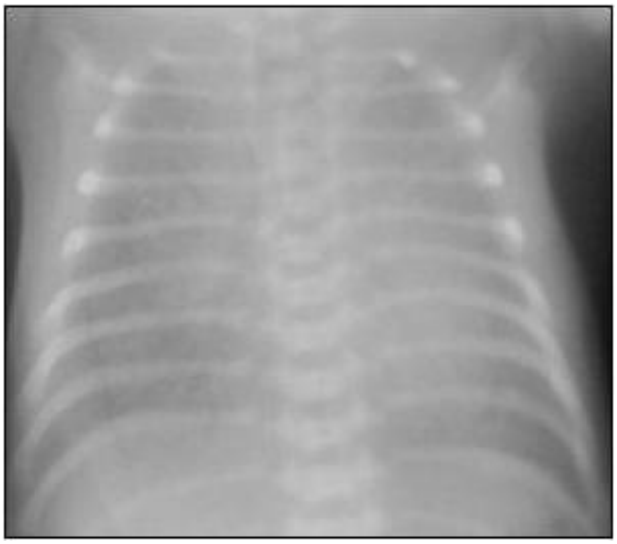


*Fig. 4. Pespiratory distress syndrome. Late stage*

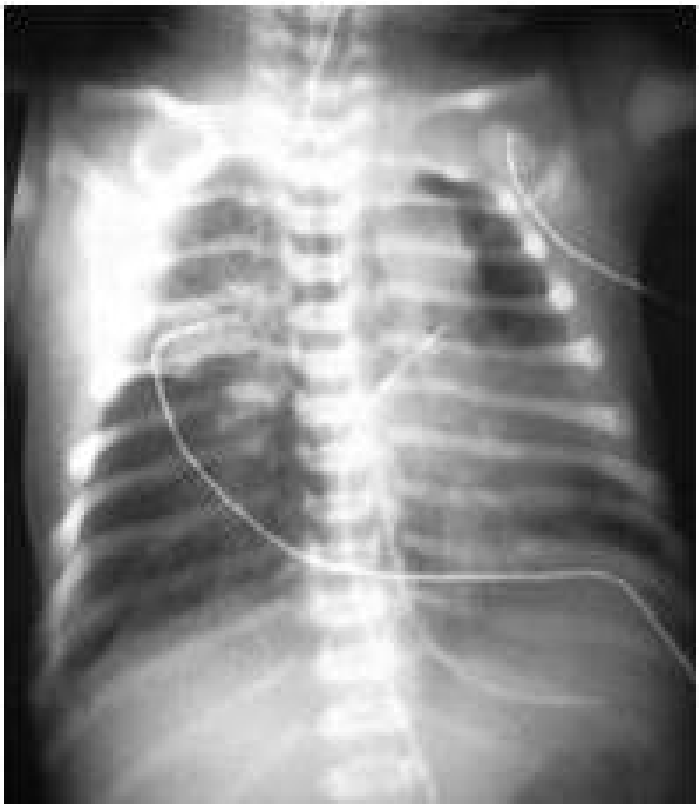
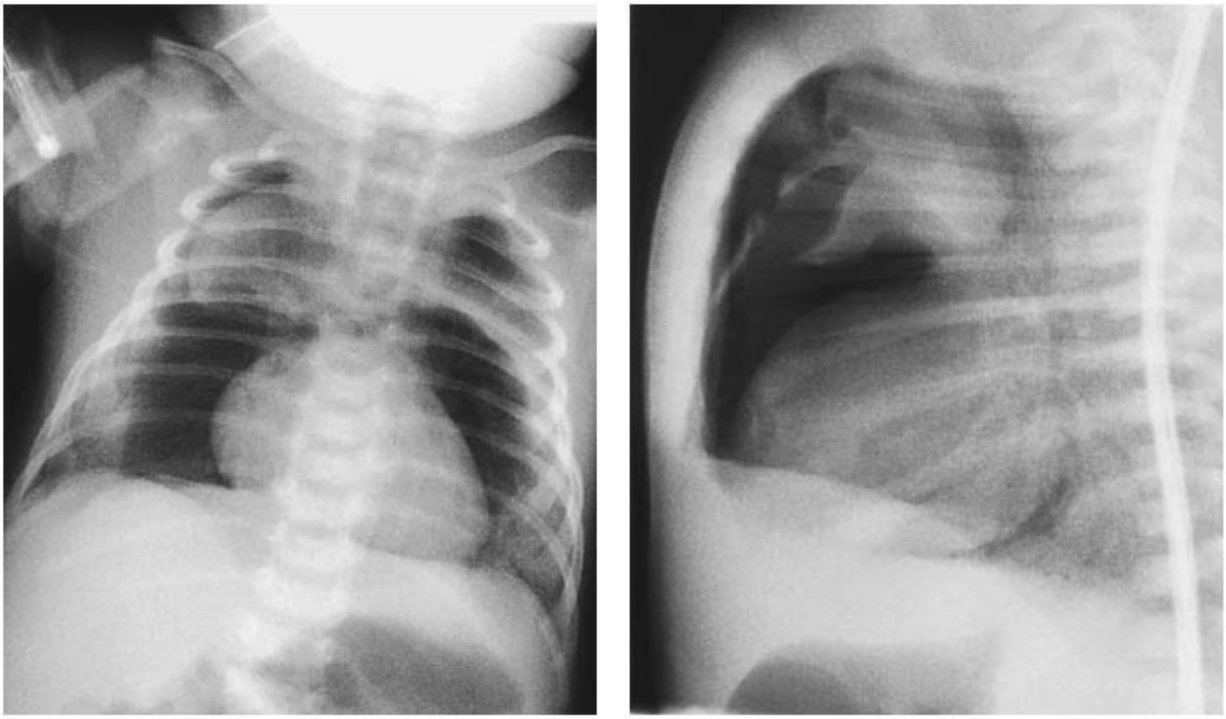


*Fig. 5. Pneumonia*



A B

*Fig. 6. Pneumomediastinum*

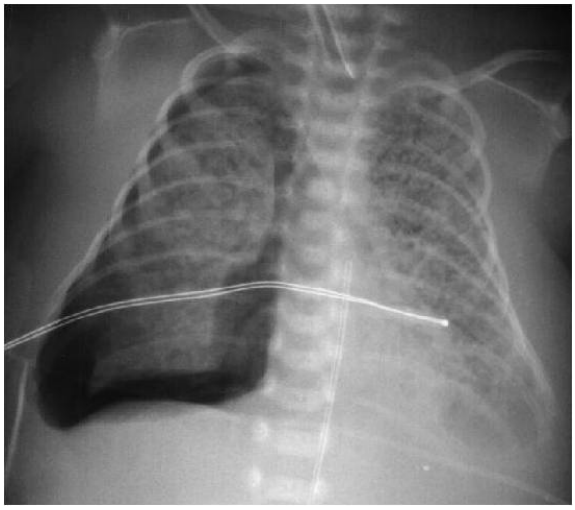


*Fig. 7. Pneumothorax*

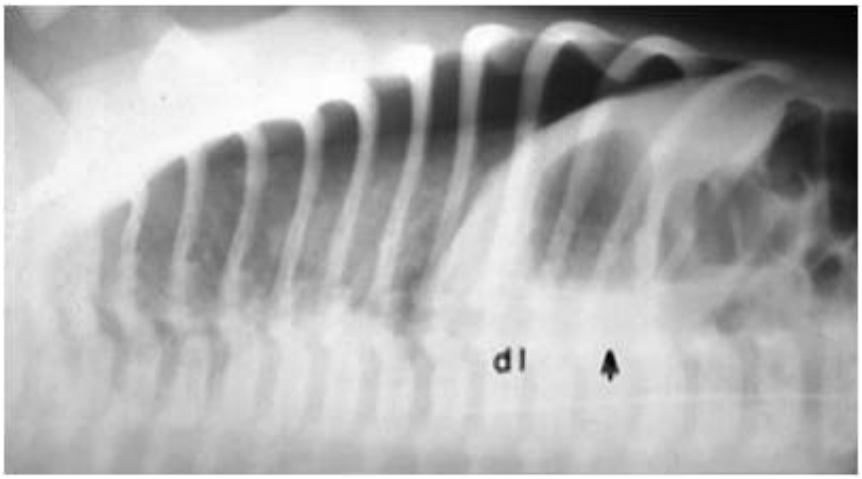



*Fig. 8. Pneumothorax*

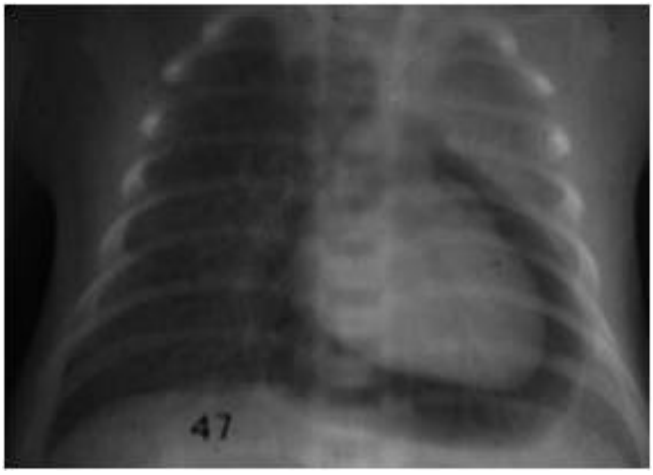



*Fig. 9. Pneumopericardium*

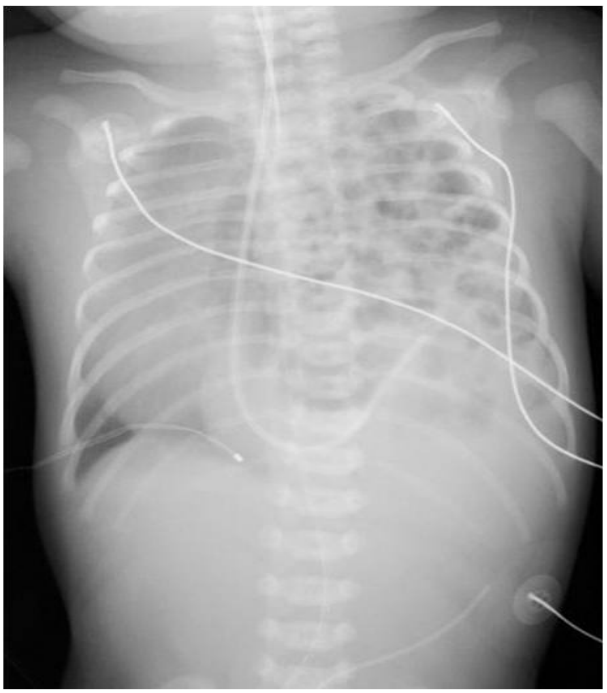


*Fig. 10. Congenital diaphragmatic hernia*

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# РЕСПІРАТОРНІ ЗАХВОРЮВАННЯ НОВОНАРОДЖЕНИХ

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для студентів 5-го курсу медичного факультету

(Англ. мовою)

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