고위험신생아의 호흡관리
(Respiratory Management in High Risk Infants)
-Premture Infants with Respiratory Distress Syndrome을 중심으로-

인하대학교 의과대학 간호학과 안 영 미

1. NORMAL LUNG DEVELOPMENT

Stage	Gestation	Characeteristics
Ⅰ. Embryonic period: Formation of proximal airways	4 to 6 wks	Foregut→laryngotracheal groove→a single lung bud→two primary bronchial buds→bronchopulmonary segaments
Ⅱ. Pseudoglandular period: Development of air-conducting system	7 to 16 wks	Differentiation of respiratory epithelium, cilia, cartilage formed.
Ⅲ. Canalicular period: Acinar development	17 to 27 wks	Enlarged bronchi and bronchioles→alveolar ducts & terminal sac formed→ Surfactant rom type Ⅱ alveolar cells (earliest at 19 wks)
Ⅳ. Saccular period: Development of gas exchanging sites	28 to 36 wks	Enough pulmonary alveoli & lymphatic capillaries with vasculature & surfactant by 28 wks for survival at NICU, by 34 wks for survival at nursery.
Ⅴ. Alveolar acquistition period	37 wks to 8 yrs	Pulmonary alveoli continue to develop(at birth, 1/6~1/8 of adult number of alveoli present)

2. PHYSIOLOGIC DIFFERENCES IN RESPIRATORY FUNCTION BETWEEN NEONATES AND ADULTS

(1) **Diaphragmatic respiration** due to horizontal ribs and poorly developed intercostal muscles

(2) **A compliant chest wall but less compliant lungs** with a low functional residual capacity(FRC): heavy breathing
(3) **Lack of established of collateral ventilation** with less developed cilia & pore of Kohn
(4) **A higher metabolic rate for oxygen consumption** in infants than in adults: a more rapid development of hypoxemia.

3. RESPIRATORY DISTRESS SYNDROME(RDS)

1) 정 의

a phenomenon of developmental delay in lung maturity

2) 원 인

(1) a decrease in the total amount of surfactant and a qualitative alteration of the surfactant present.
(2) the anatomic immaturity of the lung parenchyma, capillary endothelium, and the chest wall.
(3) the presence of the PDA with a Lt to Rt shunt, resulting in pulmonary overcirculation.
(4) an increase in the interstitial and alveolar lung water

*Surfactant: a surface-active phospholipid,
(1) to reduces surface tension of fluids that line alveolar and respiratory passages,
(2) to result in uniform expansion and maintenance of lung expansion at low intraalveolar pressure.

3) Pathophysiology of RDS

(1) **Fact. 1**: Deficient surfactant production→unequal expansion of alveoli on inspiration and collapse of alveoli on end expiration→need a great deal of effort to reexpand(e.g. 60 to 75 cmH_2O/breath)→prone to atelectasis.

(2) **Fact. 2**: Absence of alveolar stability and with progressive atelectasis→increase in pulmonary vascular resistance(PVR)→Lt to Rt via PDA(Rt to Lt shunt via foramen ovale, too)→decrease in pulmonary blood flow→pulmonary hypoperfusion.

(3) **Fact. 3**: Inadequate perfusion and ventilation→hypoxemia, hypercapnia & acidosis→vasospasm in pulmonary arterioles→vasoconstriction→a marked increase in PVR.

(4) **Fact. 4**: Prolonged hypoxia→increase in glycolysis→increase in lactic acid→metabolic acidosis(+respiratory acidosis)→further vasoconstriction→decrease in pulmonary blood flow, then further hypoperfusion→no circulation of materials for surfactant production.

(5) **Fact. 5**: PDA, renal insufficiency, Lt ventricle dysfunction, low serum protein concentration, etc.(due to prematurity+hypoxemia)→pulmonary edema.

(6) **Fact. 6**: Immaturity of the nervous system(e.g. a high threshold of the respiratory center to stimuli, weak gag & cough reflex), fetal hemoglobin.

(7) **Fact. 7**: Increased transudation of fluid into lung+fibrin in transudated fluid from necrotic cells→Hyaline membrane formation; impaired gas exchange→diminished lung distensibility(poor compliance)→need more pressure to expand.

4) Management of RDS

Focused on maintaining respiration with adequate oxygenation

- −Employment of mechanical ventilation and surfactant replacement therapy.
- −Employment of chest physiotherapy(CPT) and endotracheal suctioning(ETS) as an essential part of nursing care

(1) Mechanical ventilation

a. **PIP**(positive inspiratory pressure) to inflate surfactant deficient alveoli

b. **PEEP**(positive end-expiratory pressure) to prevent alveolar collapse

during expiration

c. TV(tidal volume) delivered into the airways

d. MAP(mean airway pressure)

* High Frequency Ventilation(HFV)

- HFPPV(high frequency positive pressure ventilation): 60 to 150 bpm
- HFO(high frequency oscillation): 480 to 1200 bpm
- HFJV(high frequency jet ventilation): 250 to 900 bpm

* Surfactant replacement therapy

- Survanta: a natural bovine surfactant
- Exosurf neonatal: an artificial surfactant

(2) Chest Physiotherapy(CPT)

a. **Postural drainage**: use gravity to assist removal of secretions from specific lung areas
   - inefficient due to diaphragmatic breathing, weak intercostal muscles & an unstable chest wall
   - an increased risk of ICH & ICH

b. **Percussion**(clapping): a series of gentle blows to the outer thorax over the distribution of the bronchopulmonary tree
   - produce an alteration of airway pressure & intrathoracic pressure to help dislodge of plugs
   - an increased risk of ICH & IVH, pulmonary hemorrhage, rib fractures
   - no need with less thick secretions

c. **Chest vibration**
   - an application of fine, oscillatory movements from lower to upper part of lungs for 30 seconds using a mechanical vibrator
   - to thin lung secretions and to propel secretions from small to larger airways
   - to improve intrapulmonary gas mixing & oxygenation
   - S/E: hypoxia

d. **Issues on the effects of CPT**

- the combined use of multiple CPT techniques
- the lack of control of the population variables
- the lack of standardization in the performance of various CPT techniques
- the lack of standardized time points in the protocol at which to measure the effects of CPT

(3) **Endotracheal Suctioning**(ETS)

a. **Respiratory tract:**

mucociliary damage→necrosis, inflammation, the loss of cilia→need more frequent ETS

b. **Cardiopulmonary response:**

· Disconnection from ventilator→hypoxia, bradycardia

· Hitting the carina(deep ETS)→stimulation of parasympathetic fiber→vagal stimulation→decrease in HR, tracheobronchial contraction, increase in mucous secretions→more prone to hypoxia

c. **Cerebrohemodynamic response:**

· Deep ETS→the contraction of the expiratory and abdominal muscles→increase in intrathoracic pressure and intraabdominal pressure→decrease in venous return to the heart(cerebral venous congestion)→increase in ICP→prone to IVH(⇒valsalva's response).

· Presence of Geminal matrix (cerebral cortex):

a high vascular, gelatinous area without blood vessels for 24 to 36 GA, receives large portion of CBF before 32 GA, reversal of venous blood flow in this area (U-turn)→cerebral venous stasis, ICH, IVH.

d. **Issues on ETS in high risk infants**

- **Depth**: length of suction catheter inserted
- **Duration**: duration of ETS(open vs closed circuit)
- **Frequency**: number of catheter passage
- **ID / OD ratio**: ratio of internal diameter of endotracheal tube to external diameter of suction catheter
- HV(Hyperventilation: rate of Ambu bagging), HI(hyperinflation: PIP or volume of Ambu bagging) & HO(hyperoxygenation: level of FiO_2) before and after ETS