

Nursing Management of Adults with Respiratory Disorders part 2

ARDS

ARDS is Acute Respiratory Distress Syndrome. It is defined as a sudden, progressive respiratory failure that includes severe dyspnea, hypoxemia despite increasing FiO₂ and diffuse infiltrates (diffuse infiltrates means there is fluid all over the lungs. Fluid shows as white fluffy clouds on the CXR). ARDS can be fatal within 48 hours, so you have to act quickly! Mortality rates are at about 50-70%...not good at all!

The Etiology of ARDS

ARDS is caused by a direct or indirect injury to lungs that causes ischemia or inflammation (there are those “I” words that Dr. B told us to watch out for) that traumatizes the alveolar capillary membrane. Direct injury would be something like pneumonia, *shock*, aspiration, chest trauma...these all involve the lungs directly. Indirect injury would be things like pancreatitis, *sepsis*, *trauma*.

Recall that its the release of chemical mediators that cause the membranes to become “leaky” leading to inflammation.

ARDS Pathophysiology

ARDS involves a massive inflammatory response by the lungs...the permeability of the pulmonary capillary membrane is changed. The alveoli fill with fluid from the interstitial space and there is a loss of surfactant (recall that surfactant keeps those alveoli open)...to add insult to injury the lungs can't even make more surfactant to keep everything nice and happy. You end up with a loss of pulmonary compliance (stiff lungs that can't ventilate well) and impaired oxygenation.

There are 3 phases to ARDS patho

Phase 1: Exudative phase (occurs about 24 hours after insult)

There is damage to the capillary membrane, reduced blood flow to lungs, and fluid starts leaking into the interstitial space...it eventually goes into the alveoli.

Phase 2: Proliferative phase (occurs day 7-10)

The surfactant cells are damaged in this phase. Recall that surfactant increases lung compliance and ease of inflation. As a result of this loss of surfactant, the alveoli collapse which impedes gas exchange and decreases lung compliance. If your pt gets to this stage, they are looking at long-term ventilation.

Assault to pulmonary system
Respiratory distress
Decreased lung compliance
Severe respiratory failure

Phase 3: Fibrotic phase (occurs in 2-3 weeks)

Pulmonary edema worsens, inflammation leads to fibrosis and gas exchange is even worse. Fibrin damage cannot be reversed!

Signs and Symptoms of ARDS

Early signs

- *Restlessness, change in LOC*
- Increasing RR with normal lung sounds
- Dyspnea
- Respiratory alkalosis: low PaCO₂ (< 35 mm Hg)

- Hypoxemia: low PaO₂ (< 60 mm Hg)
- Increased WOB
- *Increasing HR*
- Increased temp
- Normal or only patchy white infiltrates on CXR
- Increased PIP (if on a vent) *PIP = Peak Inspiratory Pressure*

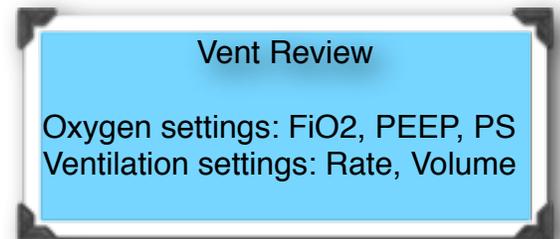
Late Signs

- low PaO₂ despite increasing levels of administered O₂
- Severe dyspnea and WOB (pt may be grunting, retractions)
- Hypercapnia (high PaCO₂) AND hypoxemia
- Metabolic acidosis (recall that with acidosis pt gets lethargic)
- Lungs with crackles and rhonchi (continuous snore-like sound d/t obstruction)
- CXR will show bilateral infiltrates (“white out”)
- Increased PIP, decreased functional residual capacity
- Cyanosis, pallor

So your pt has ARDS...and they are getting worse. What do you DO now?

The pt is going to be intubated, transferred to ICU and sedated. What supplies and equipment need to be at the bedside and what do you need for your pt?

- Working suction
- O₂
- Ambu-Bag
- IV access for sedation
- Pulse ox
- ECG monitoring
- CXR post intubation



What do we do for ARDS?

- First and foremost, we identify pts who are at risk. We need to catch this EARLY!
- Treat the underlying cause
- Prevent further alveoli capillary membrane damage by changing the ventilator mode to “pressure control” to keep the PIP under 25 cm/H₂O
- Support tissue oxygenation (how do we do that?)
- Oxygenation and ventilation support:
 - Small tidal volumes (6mL/kg)
 - Keep inspiratory pressures < 25 by changing to pressure control ventilation
 - Goal is to have the FiO₂ < 70% with a PaO₂ of 60-70
 - Position HOB 30 degrees
 - Exquisite oral care q 2-4 hours
- Maintain tissue perfusion
 - Keep cardiac output up (how do we do that?)
 - Hydration (prevent pt from getting hypovolemic)
- Provide nutritional support to enhance immune function (will need tube food as long as gut works. If gut does not work, you’re looking at TPN)
- Prevent complications...VERY IMPORTANT!
 - Handwashing (most basic and most important)
 - Prevent stress ulcers (Protonix)

- Prevent DVT (SCDs, TEDs, heparin therapy)
- Prevent VAP (exquisite oral care, sterile suctioning)
- Prevent skin breakdown (turn, assess skin around the tube)
- ROM
- Monitor for symptoms of infection (CXR, trend WBCs, temp)
- Provide psychosocial support to patient and family

Weaning your pt off mechanical ventilation...your goals!

- Get the mode to spontaneous with volumes > 500
- FiO₂ to 40%
- PEEP 5
- PS 10 (this will be the last setting to be lowered to goal)
- Minimal secretions
- Clear CXR
- CPAP trials...these may occur over the course of a few days. Start out with 1 hour, then do 2 hours the next day, then maybe 8 hours on day 3 (of course this would vary by pt...this is an example from the case study). Once extubated, pt may be on a mask still but at least they are getting closer and closer to discharge goals.

What do you do for your patient after extubation?

Remain with the pt for 30-60 mins...they are going to need some constant supervision for a while to assess for O₂ sats, WOB, stridor. They will need a swallow eval prior to eating or drinking anything. You also want to remind them to not talk...their airway has been traumatized and you don't want to cause any inflammation at this point.

THORACIC TRAUMA

The most common injury of this type is fractured ribs and atelectasis is your biggest concern because it is extremely painful to breathe with broken ribs and almost impossible to take a deep breath. You are going to be providing excellent pain management to your patient!

There are two mechanisms of thoracic trauma: penetrating and blunt. A penetrating injury damages the lungs, heart and other abdominal structures. A blunt injury is more difficult to detect. The damage is directed toward the same structures. You will want to observe this pt closely and watch for any developments that would require a chest tube.

What data do you always get with a chest trauma pt?

- CBC (determine O₂ carrying capacity)
- CXR (check out the damage)
- ABG (check out the gas situation)

Pulmonary Contusion

In a pulmonary contusion, the lung parenchyma is damaged and there is impaired gas exchange. The injury causes interstitial hemorrhage, alveolar collapse and alveolar flooding. The blood flow continues to truck right on past these damaged/flooded alveoli but they don't pick up any oxygen because the alveoli are full of fluid. This means that blood does not get to load up with oxygen and you end up with shunting and hypoxia. Bad bad bad news.

Assessment findings with pulmonary contusion:

- Dyspnea
- Wheezing hypoxia
- hemoptysis

- Cyanosis (a late sign...someone missed something or he came in too late)
- Bloody sputum

Treatment for pulmonary contusion:

- Careful respiratory assessment...be thorough and accurate!
- Pain relief
- Oxygen therapy

Flail Chest (see video at <http://www.youtube.com/watch?v=e0VNBDbr67U>)

Flail chest is a loss of fixation of the chest in two areas...the same ribs broken in two areas, and there is a decrease in respiratory wall movement. The fractured ribs are not connected to the cartilage anymore and they're just kind of "floating" out there. Freaky!

Flail chest clinical findings:

- Paradoxical chest movement (easy to see in the above video)
- Unequal breath sounds
- Respiratory distress

Flail chest treatment

- Position the good lung down
- Provide adequate oxygenation and ventilation (may require intubation!)
- Closed chest drainage (I imagine there's going to be blood in there)
- Frequent respiratory assessments
- Pain control

Sub-Q Emphysema (crepitus)

With SQE, air has gotten out of the normal airway and found its way into the interstitial space. Subcutaneous clinical findings include edema and crackles on palpation (like bubble wrap!). Mediastinal clinical findings are crepitus in suprasternal notch and mediastinal shift (a bad thing).

Closed Chest Drainage (chest tube)

The purpose of a chest tube is to remove air or fluid from the pleural space and re-expand the lung! The system is a CLOSED system...which means you have to work to keep it closed! The drainage units can be a "wet suction" unit or a "dry suction" unit...which type to use will be prescribed by the MD.

VAP (ventilator acquired pneumonia)

VAP is a nosocomial respiratory infection that should be totally preventable! It accounts for 15% of all hospital-associated infections and 27% of all MICU acquired infections (according to CDC). The primary risk factor for getting VAP is mechanical ventilation! The upper respiratory tract is colonized within 48 hours and 10-65% of people with colonization will go on to get VAP. Early-onset VAP develops within 48-72 hours of intubation. (Late VAP develops > 72 hours post) If pneumonia occurred earlier than that, the pt came in with it and it's not necessarily your fault. Whew!

Nosocomial pneumonias are the leading cause of mortality

Common VAP Microorganisms

- S. pneumoniae
- S. aureus
- H. influenzae
- or normal oropharyngeal flora

Late VAP Microorganisms

- pseudomonas
- acinetobacter
- S. aureus

from nosocomial infections, with a mortality rate of 20-70% (that's terrible!). VAP increases ventilatory support requirements and lengthens the ICU stay by 4.3 days (not cheap and not pleasant at all for the pt). The overall length of stay is increased by 4-9 days with a cost increase of \$20K to \$40K per episode. Ouch!

VAP Prevention

- Use orotracheal intubation rather than nasotracheal
- HOB 30 degrees
- Sxn above the endotracheal tube cuff frequently (q 12 hours at a minimum)
- Clear secretions prior to deflating ET cuff
- Frequent oral care (q 2-4 hrs)
- Brush teeth 2 x per day (q shift)
- Change in-line sxn catheter when it is soiled or malfunctioning...not often!
- Use aseptic technique when changing trach tube and replace with a sterile tube
- Use only sterile fluid to clear secretions from the sxn catheter
- Verify placement of feeding tube (aspiration pneumonia is bad news!)
- Assess pt intestinal motility and adjust enteral feeding rate/volume to avoid regurgitation
- Remove ET and tracheostomy tubes as soon as medically indicated...get 'em out!
- Avoid repeated endotracheal intubations (don't extubate too aggressively!)

Set wall suction to
80-120 cm H₂O.

ATI REVIEW FOCUS

- *What is a thoracentesis?* A thoracentesis is a surgical perforation of the chest wall and pleural space with a needle to obtain specimens for dx, to instill meds into the pleural space, and for the removal of air and fluid from the pleural space.
- *What clinical condition does a thoracentesis treat?* Pleural effusion. This most commonly occurs with cancer, where oncotic pressures change and fluid collects in the pleural space, or when the lymph system does not drain this area properly. A thoracentesis can also drain infection (empyema) from the infected area.
- *Why would a pt have a thoracentesis instead of a chest tube?* Pt would get thoracentesis if effusion is small (< 25% of lung volume) and is experiencing SOB, increased RR...but is not clinically unstable. Also used when dx tests are needed or if there is less than 1500 mL of fluid. The procedure can be done as an outpatient procedure...more of a chronic disease type patient and not so much for trauma patients.
- *What are the nursing responsibilities?* Position the patient upright to enable pooling of fluid and leaning over to facilitate access. The patient MUST hold still, so you will pre-medicate them for pain and also provide a calming presence during the procedure.

Black, Joyce M., and Jane Hokanson Hawks. *Medical-Surgical Nursing: Clinical Management for Positive Outcomes - Single Volume (Medical Surgical Nursing- 1 Vol (Black/Luckmann))*. St. Louis: Saunders, 2009. Print.

Brady, D. (2009) *Respiratory Disorders. Advanced Med/Surg. Lecture conducted from CSU Sacramento, Sacramento.*