Respiratory Failure
and Acute Respiratory Failure Chapter 66

B. Pulskamp

Function is: gas exchange with transfer of O2 and CO2 between atmosphere and blood.
Respiratory failure results when one or both of O2 and CO2 exchange functions are inadequate.
Respiratory failure can be classified as hypoxemic or hypercapnic.
What is it? It is not a disease.
What makes it Hypoxemic or Hypercapnic??

Hypoxemic or oxygen failure
Alveolar ventilation is inadequate; with inadequate O2 transfer
Lungs can no longer adequately oxygenate the blood.
Usually arterial oxygen pressure (PaO2) is less than 60mm with patient receiving O2 of 60% or greater. (1. inadequate oxygenation 2. In spite of oxygen administration. )
Sa O2 does not fall in direct proportion to PaO2 levels. It falls only slightly until the PaO2 drops below 60 mm Hg. Thus PaO2 is more sensitive measure of gas exchange status than Sa O2. A “small” ↓ in pulse Sa O2 (pulse oximeter ) from 95% to 90% means that PaO2 has decreased more significantly. (CCN secrets by Shell)
Pneumonia, PE, Pulmonary edema, injury from toxic gases, and low-cardiac output states as CHF.

Hypercapnic respiratory failure or ventilatory failure
Insufficient CO2 removal
Arterial carbon dioxide (PaCO2) of more than 45 in combination with acidemia (pH less that 7.35)
PaCO2 is higher than normal
Evidence of body’s inability to compensate for acidemia
The pH is at a level where a further decrease may lead to sever acid-base imbalance.
See Chapter 16 for acid base review
Disorders include what??
What mechanism is involved??
Drug overdose with CNS depressants
Neuromuscular diseases (myasthenia gravis)
Thoracic Trauma or diseases of spinal cord with lung involvement. See table p 1825 for others

Symptoms
Acute dyspnea
Significant respiratory acidemia (pH<7.3)
Can not be defined with this criteria if chronic restrictive or obstructive lung disease
Then Levels of (PaO2) 60 or lower &(PaCO2)levels of 45 or higher
Hypoxemic respiratory failure
- Causes: Respiratory, cardiac, pulmonary
- See table 66-1
- 1. Mismatch between ventilation (V) and perfusion (Q) = (V/Q) mismatch
- 2. Shunt
- 3. Diffusion limitation
- 4. Alveolar Hypoventilation

V/Q mismatch
- Blood perfusing lungs each minute
- 4-5 L = same amount of gas that reaches the alveoli each minute with ratio of 1:1
- 1 ml air/1 ml blood
- Apex may be greater, base may be less = balance.
- See examples page 1827 Fig. 62-4

Diseases of V/Q mismatch
1. With increased secretions or bronchospasms in airways and alveoli limit airflow but no effect on perfusion and blood flow. Pneumonia, asthma, atelectasis (alveoli collapse) chronic and acute bronchitis, severe emphysema.
2. Blood flow limit (i.e. embolus). No effect on airflow but blood flow impaired. Treat with O2 to increase PaO2 to reverse hypoxemia.

Ventilation-perfusion mismatch
- A decrease PaO2 and an elevated PaCO2
- Due to increased work of breathing, secondary to airway resistance (think breathing through a straw)
- Ventilation decreases and PaCO2 increases
- Decrease in pH with respiratory acidosis
- Ventilation does not remove PaCO2
- Kidneys need 48-72 hours to respond

Shunt
- Blood exits the heart without being exposed to O2
- Anatomic-Blood passes through anatomic channel in heart. (ventricular septal defect or ductus arteriosus). With no blood passing through lungs.
- Intrapulmonary shunt—through pulmonary capillaries without gas exchange. Alveoli are filled with fluid. ARDS, and pulmonary edema prevent exchange of O2... O2, may be ineffective and may require mechanical ventilation. High O2 does not effect PaO2 (will effect in V/Q)

Diffusion limitation
- Gas exchange is compromised when alveolar-capillary membrane is compromised that has thickened or destroyed the membrane. P. 1827
- Obstruction from emphysema or recurrent PE. Pulmonary fibrosis, interstitial lung disease and ARDS. Hypoxemia during exercise (Blood is pumped faster with shorter time for diffusion).
Diffusion Abnormalities
- Impairment in equilibration between O2 pressure in alveoli, and in the pulmonary capillaries.
- Causes: decreased contact time for RBC’s at capillary membrane (exercise)
- Pulmonary blood is reduced due to obstruction or destruction
- Blood gas membrane is thickened (pulmonary edema)

Alveolar hypoventilation
- Generalized decrease in ventilation with increase in PaCO2 and decrease in PaO2.
- Result of lung disease, CNS disease, chest wall dysfunction or neuro disease.
- The causes of hypercapnic respiratory failure cause the hypoxemia.
- Alveolar ventilation is volume of gas per breath for gas exchange.
- Adult tidal volume of 500ml with 150 ml of dead space. (+/- 1/3 of persons weight)
- PaCO2 = amount of CO2 produced and inversely related to effective alveolar ventilation.
- Less ventilation = more CO2
- More ventilation = less CO2.

Hypercapnic Respiratory Failure/
Acute or Chronic Respiratory failure
- Inbalance between ventilatory supply and demand. PaCO2 can not be sustained within normal limits and hypercapnia occurs.
- Ventilatory failure or Pump failure (lungs unable to pump out CO2)
- Low O2 tension, in combination with Hb capacity, cardiac output, and distribution of blood flow

Hypercapnic Diseases:
- 1. Airway and alveoli involvement as asthma, emphysema, chronic bronchitis, cystic fibrosis with air flow obstruction and air trapping.
- 2. CNS with suppressed drive to breath. Drugs, brain stem, head injury. Medulla does not respond to high CO2.
- 3. Chest wall limits lung expansion as trauma, massive obesity.
- 4. Neuro conditions with muscle weakness or paralysis as ASL, cervical cord, MS
- In the last 3 there may be normal lung tissue.

Clinical Manifestations
Table 63-3 p1829
- Related to extent of change and rapidity of change and ability to compensate. If slow onset, kidneys can compensate for the acidosis with compensated respiratory acidosis.
- Related to either increase of PaCO2 or decrease of PaO2 and inability to meet O2 needs.
- Cardiac, anemia and septic shock.
- COPD may have asynchronous respirations.
- Altered ratio of inspiration to expiration
- Asymmetric chest, pursed lips, open mouth arms on bedside table (tripod position).
Symptoms

- Retractions, speak only a few words at a time. Restless, confusion, combative. Are signs of rapid deterioration.
- Watch for fatigue and tiring. Slowing of respirations may indicate possible respiratory arrest. Be alert for possible code! Low respirations may require intubation.
- Hypoxemia is when $O_2$ is less than normal
- Hypoxia is when there are symptoms of inadequate oxygenations.
- Rapid changes of concern: Restlessness, confusion, combative behavior, tachycardia and mild hypertension (brain sensitive to $O_2$ decrease so CNS alterations)
- Morning headache Why?
- Rapid shallow respirations, (lungs have fluid, or are stiff). Worry when???
- When does cyanosis occur? What is the Pa $O_2$ then?
- Diaphoretic, difficulty in speaking, pulsus paradox of 10 to 50. (Systolic pressure minus pressure heard as cuff deflated to continuous respiratory cycle). Evaluate for cardiac tamponade.(fluid in pericardial space with compression of heart)

Other Clinical Manifestations

- Position? Lie down or sit up.
- Change in Inspiratory to expiratory ratio
- Normal I:E is 1 to 2
- Respiratory problems it is 1:3 or 1:4
- Taking longer to empty lungs.
- Late signs are cyanosis (except for anemic or polycythemia patients).
- Diagnostic Studies; ABG (oxygenation with Pa$O_2$ and ventilation with Pa$CO_2$). pulse oximeter, Chest x-ray, CBC, Electrolytes, mixed venous oxygen tension from pulmonary artery port of pulmonary artery catheter. Normal is 35 to 40. Less means tissues using more oxygen, more means tissue not being perfused.

Risk Factors Summary

- Generally the critically ill
- Specifically recent abdominal or thoracic surgery, and compromised due to splinting of incision, abdominal distension, restrictive bandages, tubes, and pain, obese patient, comatose patient or diminished LOC, smokers, those with other infections, immunosuppressed, and geriatric.

Diagnostic Tests

- ABG’s
- CBC
- ECG
- If pulmonary embolus get an V/Q (ventilation perfusion) lung scan.
- Cardiac outputs and hemodynamic monitoring.

Evaluation of Ventilation

- Tidal volume air exhaled during normal breathing
- Less than 300 or more than 700 need to be evaluated
- Forced vital capacity maximal volume of air that can be forcibly exhaled from lungs after maximum inhalation. Usual range is 60 to 80 ml/kg. 15 required for cough
Maximum Inspiratory pressure is maximum inspiration generated against a closed airway. Used to evaluate muscle strength.

Dead space is inspired volume that does not come in contact with pulmonary capillaries for oxygenation. Usually 30% of tidal volume.

Treatments

- Oxygen therapy. If not responsive to O₂ correct hypoxemia add positive pressure ventilation to decrease work of breathing and fatigue. Oxygen delivery must be tolerated by patient and maintain PaO₂ at 55 to 60 mmHg. Hemodynamic monitoring, vasodilators, diuretics, ventilators, and treat underlying cause.

Nursing Management

- Assessment of hemoglobin and CO
- Vitals, oxygen administration,
- Prevent tissue hypoxia, maintain airway with mobilization of secretions and cough, position and suctioning
- Decrease stress and loss of control as in physiologic, cognitive, environmental, & decisional) Interventions to promote comfort unless hypoventilated then need to keep awake to stimulate.

Nursing measures

- Decrease secretions (suction, huff cough etc)
- Assist with increased volume by position (if one lung position with unaffected lung down for better perfusion “Down with the good lung”. Use only if one lung is affected with disease.
- Hydration to thin secretions
- Chest percussions, and postural drainage
- Airway suctioning
- Positive pressure ventilation through invasive (ET tube or naso track) or non invasive (nasal or face mask)

Drug Therapy

- Relief of bronchospasm with Metaproternol (alupent) and albuterol (ventolin) to reverse bronchospasms. IV Aminophylin if sever
- Reduce inflammation: Corticosteroids. Inhaled take 4-5 days for effect so not used in emergency. IV corticoids are immediate.
- Reduction of pulmonary congestion with IV diuretics. (lasix) Digitalis if cardiac problem
- Treat pulmonary infection (pneumonia, bronchitis) with antibiotics after sputum culture.
- Anxiety and restlessness with Lorazepan (ativan)

Therapy

- Medical:
  - 1 Treat underlying cause
  - 2 Maintain adequate CO
3. Maintain adequate Hgb concentration (maintain at 9 to 10)

Nutrition
Remember to evaluate geriatric conditions as the Pa O₂ falls farther and the PaCO₂ rises higher before alteration or rate and depth of breathing.

Acute Respiratory Distress Syndrome
- ARDS is a sudden progressive disorder consisting of high permeability pulmonary edema (not cardiogenic), alveolar-capillary membrane is damaged and fluid and protein leak into the interstitial space and alveoli.
- Patients have decreased lung volumes and decreased lung compliance, alveolar collapse and hypoxemia as lungs are perfused but not ventilated. Respiratory failure and cardiopulmonary arrest.

Causes
- Usually sudden catastrophic situations as direct lung or indirect lung injuries.
- Gram negative septic shock and aspiration most common.
- Stimulation of inflammatory and immune systems
- Direct: gastric aspiration, near drowning, chemical inhalation and oxygen toxicity.
- Indirect: sepsis, multiple trauma, thermal injury, hypoperfusion or hemorrhagic shock, DIC, drug overdose, or massive blood transfusions. Mortality from 50 to 70%

Stage
- Injury stage: Usually 24 to 48 hours after injury (may be 1 to 7 days) Onset may be dyspnea, restlessness and mild hypoxemia. Interstitial and alveolar edema. Hyaline lines lungs.
- Reparative or Proliferative Phase: 1-2 weeks after injury. Lungs have dense fibrous tissue, pulmonary resistance, and reduced lung compliance. Alveolar cells and membranes are damaged.
- Fibrotic Phase: 2-3 weeks after injury. Diffuse scaring and surface areas for lungs are reduced.

Physical Exam findings
- May be insidious.
- Respiratory distress with dyspnea, tachypnea, cough, restlessness, diaphoresis, rate up to 40. Dusky appearance with cyanosis or very pale. Hypoxemia, peripheral pulses rapid and thready, BP normal or elevated initially, then decreased. Auscultation of lungs depends on stage of ARDS. Early has decreased breath sounds, later basilar or coarse crackles later no breath sounds. Wedge pressure does not increase since not cardiogenic cause.

Diagnosis
- One of exclusion. NO specific markers
- Chest x-ray
- ABG's
Clinical progression. If patient survives acute phase and pulmonary edema resolves and may recover in a few days. Late stage has poor prognosis.

Prevention
- Preop screening and evaluation of all high risk patients
- Pulmonary tests and ABG's
- Measures to optimize ventilation. What?
- Which is most critical for nursing?

Complications
Table 66-9
- Infection
- Respiratory complications of emboli, pneumothorax, fibrosis, and oxygen toxicity.
- GI of ulcers, ileus
- Acute renal failure
- Cardiac with arrhythmias.
- Anemias and ET intubation complications

Treatments /Nursing/Medical
- Treat underlying disorder.
- Oxygenation with Trachs and suctioning, ventilators, Positioning often using prone position.
- Cardiac output and fluid balance.
- Skin and eye care, PROM,
- Ns Dx: Impaired gas, ineffective breathing, risk of infection and injury, impaired communication

Sever Acute Respiratory Syndrome
- Caused by caronavirus and probably spread through droplets.
- Fever, then cough followed by trouble breathing.
- Treat symptoms. Since virus antibiotics not helpful.

Artificial Airways
- ET tubes
- Nasal-More stable, can be placed “blindly”.
- Oral- for most emergencies because more rapid. DO NOT use if head and neck compromised with injury. Oral has larger bore and easier to suction. Problems of increased salivation, difficulty in swallowing and biting down on tube.

Artificial Airways
Nursing considerations
- To verify placement, auscultate for bilateral breath sounds, observe chest wall movement, check oxygen values then have x-ray for verifying placement.
Document ET position (nostril or lip @ x cm.)
Tube placement: Is it at same length as charted? Monitor q 2 hours. Check ties for security and cleanliness.

Maintain proper cuff inflation. Inject air till no leak is heard at peak inspiratory pressure.

While suctioning, if dysrhythmias, stop and ventilate with 100% oxygen or place back on ventilator.

If displaced ET tube what will you do??

Establish air way or ventilate with 100% oxygen??

Why??

With risk for aspiration, what intervention will you perform?

When will you suction??

Alveolar hypoventilation

- Alveolar hypoventilation: What happens??
- Low rate causes hypoventilation.
- Inappropriate vent settings, leakage of air, excessive secretions.
- What would happen to pH with low respiratory rate?
- Retention of CO₂ which produces _______??

Alveolar hypertension

or mechanical overventilation

- Vent rate set too high.
- They then do not retain what? CO₂
- Blowing off CO₂ causes alkalosis because of retained bicarb.
- Causes: pain, fear, anxiety.

Mechanical Ventilation Types

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Negative Pressure

- Negative pressure: Does not require artificial airway. Negative pressure around chest wall causes the chest to be pulled outward reducing intrathoracic pressure with air rushing in via upper airway. Expiration is passive with the machine in the “off” cycle. This mimics how normal respirations occur. Poncho and Pulmowrap are types. It is a “body wrap” that ties at the neck, arms or hands and upper legs. Think of someone hooked up to a vacuum cleaner suction. ☺ These are often used in the home with CNS, spinal cord and COPD problems.

Positive pressure: Volume, time or pressure are the parameters.

- Used with acutely ill patients. Where air is “pushed” into the lungs on inspiration under pressure from the ventilator. Intrathoracic pressure is raised during this process, not lowered as in normal respirations.
1. **Volume cycled** most common used to intubated patients. Volume delivery is preset and constant despite lung resistance.

Ventilators cont
- **Time cycled or time limited** is regulated by time duration of inspiration. Volume and pressure may vary with each breath.
- **Pressure cycled or pressure limited**—terminate inspiration when a preset pressure is reached. Used in acute settings for short time, home or patients who are relatively free of disease. Tidal volume is dependent on airway resistance. Therefore secretions or disease may signal volume limits and little air may in fact be delivered.

**Settings of Ventilator**

Table 64-11 p.1783
- Rate (4-20), volume,
- \( O_2 \) concentration (21% to 100% to maintain \( SaO_2 \) greater than 90%)
- Inspiration /expiration ratio (I:E ratio usually 1:2)
- Flow rate (40 to 100 L/min)
- Sensitivity is amount of effort the patient must generate to initiate ventilator
- Pressure limit is the maximum pressure delivered before the remaining air is shunted off to room air (10-20 cm H2O above peak inspiratory pressure)

**Modes of Volume controlled ventilation**
- Mode is the manner in which the the breath is initiated and volume is controlled by patient or ventilator. Controlled by ventilatory status, respiratory drive and ABG’s.
- Controlled mechanical
- Assist control
- Synchronized intermittent mandatory
- Controlled: breaths are delivered regularly and independently of patient’s effort. Used infrequently except for anesthesia, or paralyzed.
- **Assist control** delivers a preset tidal volume at preset frequency but patient may initiate a breath by attempting to inhale. When the ventilator senses a decrease in intrathoracic pressure the machine delivers a preset volume. The patient can override the rate (breathe faster) but not slower. Used with neuromuscular(Guillain-Barre syndrome), pulmonary edema, ARDS.

**ACV guidelines**
- Hyperventilation if patient over breathes or hypoventilation if machine set with too low limits. Check with ABG’s. Monitor negative pressure: too difficult will require too much effort, too little will end in over ventilation and respiratory alkalosis.

Synchronized intermittent mandatory ventilation
- **SIMV**: preset tidal volume also at preset frequency. In synchrony with the patients spontaneous breathing. Between ventilator breaths patient is able to breath spontaneously therefore receiving preset inspired oxygen concentration spontaneous breaths but **self-regulates rate** and depth of those breaths. (ACV all breaths are of same preset volume.)
SIMV
- Most common and used for continuous and during weaning.
- It minimizes “fighting” ventilator, avoids respiratory alkalosis, lower mean pressure, more uniform gas distribution and prevention of muscle atrophy. SIMV enhances venous return due to intrathoracic pressures, and CO is better maintained.
- Weaning patients allows a smooth transition to spontaneous ventilation by gradually decreasing the ventilator rate while the patient assumes more control over the breathing.
- Disadvantages: If rate set low and spontaneous breathing decreases advent of poor ventilation. Watch for fatigue.
- PEEP: positive end expiratory pressure-positive pressure is applied during exhalation. Increased distension of alveoli, with prevention of alveolar collapse and aeration of previously collapsed alveoli.
- Used in pulmonary edema, diffuse lung disease (ARDS), continued hypoxemia when oxygen is 50%. Use in extreme caution with COPD, hypovolemia, and low CO. PEEP may cause increased intracranial pressure by impeding blood flow from head due to increased thoracic pressure.

Continuous Positive Airway Pressure
- CPAP is PEEP in spontaneous breathing patients with a constant flow of gas at a rate greater than the Patient’s spontaneous inspiratory rate.

Medications for Respiratory system
- Classification:
  - A. Mucolytics
  - B. Bronchodilators:
    - 1. Beta agonists albuterol, epinephrine, isoproterenol, alupent, brethine,
    - 2. Anticholinergics
    - 3. Xanthine derivatives.
  - C. Anti-inflammatory agents
    - 1. Inhaled glucocorticoid steroids
    - A. desamethasone, triamcinolone
    - 2. Mast cell stabilizers
    - 3. Leukotriene receptor antagonists

Case studies
- Mrs. C is a 75 year old married woman with severe oxygen and corticosteroid dependent COPD. She is admitted to the MICU with Acute respiratory failure and pneumonia.
- Subjective: shortness of breath and difficulty breathing
- Objective Data: ABG’s on 2 L. pH 7.3, Pa CO2 55, PaO2 60 SaO2 84%
- O2 @2 L, Albuterol (Ventolin, Proventil) q 1 hr
- IV aminophylin, antibiotics and corticosteroids.
1. What type of failure? How does this illustrate concept of acute on chronic respiratory failure?
2. What contributed to her respiratory failure?
3. What is patho effects and clinical manifestations of her respiratory failure?
4. How do the tripod and pursed lip breathing contribute to respiratory function?

- What is NIPPV? When is it contraindicated?
- Which of the treatments instituted is the most important in returning her to her usual level of respiratory function?
- What are some examples of nursing diagnoses?

Review
- Criteria for acute respiratory failure?
- Who is most at risk for hypoxemia respiratory failure? Think of cause of low supply of oxygen.
- Who is most at risk for hypercapnic respiratory failure? Think of unable to blow off CO₂
- Explain the ventilation perfusion mechanics and what are examples.
- What do ABG’s evaluate?
- What are some early and late symptoms of respiratory failure? When is the patient failing to maintain the respiratory function?
- When would a patient be a candidate for intubation?
- Know normals for ABG’s. What are they used to evaluate?
- What is usual medical treatment for:
  - PE, and COPD
  - Nursing treatment for ineffective cough in systemic disease.
- Early onset of ARDS shows?
- ARDS pathology

Evaluate each ventilator condition for the defining characteristics
A. Alveolar Hyperventilation Mechanical overventilation
B. Alveolar hypoventilation:
1. pH ↑  2. pH ↓ Normal what value? _____
3. pCO₂ ↑  4. pCO₂ ↓ Normal what value?
5. HCO₃ ↑  6. HCO₃ ↓ Normal what value?
Cause??? ↓

. Evaluate each ventilator condition for the defining characteristics
A. Alveolar Hyperventilation Mechanical overventilation) pH ↑  pCO₂↓  HCO₃ n or ↓ (K ↓)
B. Alveolar hypoventilation: pH ↓ pCO₂↑  HCO₃ ↑
1. pH ↑  2. pH ↓ Normal what number? _____ 7.35-45
3. pCO₂ ↑  4. pCO₂↓ Normal what number? 35-45
5. HCO₃ ↑  6. HCO₃ ↓ Normal what number? 20-30
Cause??? ↓