



PCCN-CCRN Pulmonary Review

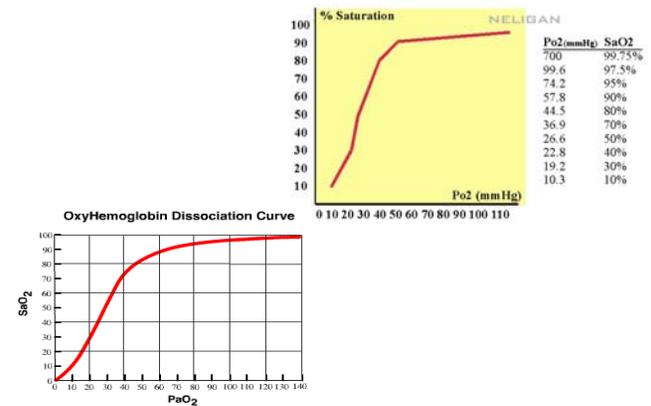
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Alterations in Respiratory Function

Disease States

Normal Ventilation Normal Perfusion	Normal Ventilation Reduced/No Perfusion	Reduced/No Ventilation Normal Perfusion	No Ventilation No Perfusion
	Pulmonary Embolism Pulmonary Vein Stenosis	Pulmonary Edema COPD Sarcoidosis Interstitial Pulmonary Fibrosis ARDS	

What is the oxyhemoglobin dissociation curve and why is it important?



Shunt and Dead Space are Extremes of V/Q mismatching.

- Shunt
 - Perfusion of lung units without ventilation
 - Unoxygenated blood enters the systemic circulation
 - $V/Q = 0$

- Dead space
 - Ventilation of lung units without perfusion
 - Gas enters and leaves lung units without contacting blood
 - Wasted ventilation
 - V/Q is infinite

Acute Pulmonary Embolism

Acute Pulmonary Embolus

- Obstruction of blood flow by a thrombus lodged into one or more of the pulmonary arteries.
 - Annually >600,000 cases in the US
 - 70% are undiagnosed
 - 30% result in death

Sources of Emboli

- Blood clots
 - >90% arise from major deep veins
- Non thrombotic sources
 - Fat
 - Air
 - Amniotic fluid
 - Particulate matter
 - Tissue, parasite, tumor, catheter fragments

Risk Factors for VTE

- Immobility/
Prolonged bed rest
- Sluggish circulatory states
- A. Fib
- Prior DVTs
- Oral contraception and HRT
- Long distance travel
- Long surgical procedures
- Central venous catheters
- Obesity

PE Signs/Symptoms

- **MASSIVE**
 - Tachycardia
 - Dyspnea
 - Hypotension
 - Pleuritic chest pain
 - Feeling of doom
- **SUB MASSIVE**
 - *Fleeting symptoms:*
 - Hypotension
 - Diaphoretic
 - Dyspnea
 - Chest discomfort
 - Pale or cyanotic
 - Pleural friction rub
 - Hemoptysis

Diagnostic Tests

- Pulmonary angiogram
- Spiral CT scan
- Chest MRI
- V/Q scan
- EKG
- Chest x-ray
- ABGs

Chronic pulmonary embolism



Pulmonary angiogram shows mosaic oligemia with localization of pulmonary blood flow to the lower lobes. This may be regarded as a Westermark's sign with oligemia affecting the subtended pulmonary parenchyma of the upper lobes.

Courtesy of Paul Stark, MD.

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Treatments for PE

- Prevention:
 - SCD
 - LMWH
 - SQ heparin
 - Mobility
- Acute:
 - Anti-coagulation
 - Thrombolytic
 - IVC filters
 - Transvenous catheter embolectomy
 - Thrombo-endarterectomy
 - Surgical embolectomy

Pneumonias

1. Community Acquired Pneumonia
2. Nosocomial Pneumonia
3. Aspiration Pneumonia

Community Acquired Pneumonia (CAP)

- Introduction
 - 6th leading cause of death & the number one cause of death from infectious disease
 - 5.6 million cases annually
 - 1.1 million require hospitalization
- Common pathogens
 - *Strep. pneumoniae*, *H. influenzae*, *C. pneumoniae*, and *M. pneumoniae*

Nosocomial Pneumonia

- Introduction
 - Pneumonia that develops >48 hours after admission
 - Leading cause of death d/t hospital infections
 - Mortality 20-50%
 - Highest at risk population: ventilator patients
- Common pathogens
 - The most common pathogens are gram-negative bacilli and *Staphylococcus aureus*

Aspiration Pneumonia

- Active (vomiting) or passive (regurgitation) with inhalation of stomach contents into the lungs
- Large particles can obscure airway
- Gastric aspirate causes damage to alveolar cells by means of a chemical burn
- Volume of liquid and its content determines extent of damage

Which of the following are signs and symptoms of aspiration?

- Bradycardia
- Tachypnea
- Productive cough
- Decreased oxygen need
- ABG which are acidotic
- Restlessness



Pneumonia: Signs and Symptoms

- | Healthy Individuals | Older/Immunocompromised Patients |
|----------------------------------|-------------------------------------|
| ▪ New Onset Respiratory Symptoms | ▪ May have non-respiratory symptoms |
| ▪ cough | ▪ confusion |
| ▪ sputum production | ▪ failure to thrive |
| ▪ and/or dyspnea | ▪ worsening of chronic illness |
| ▪ Fever | ▪ fever may be absent |
| ▪ Abnormal Breath Sounds | ▪ Tachypnea usually present |
| | ▪ Breath sounds abnormal |

Diagnosis and Treatment of Pneumonia

- Diagnosis
 - Chest X-rays
 - Sputum analysis
 - CBC
- Treatment
 - Treat based on suspected organism with ATB within 1 hour
 - In >50% of cases, a specific organism cannot be identified



REVIEW QUESTION

A morbidly obese patient is admitted to the unit following lap cholecystectomy. You know this patient is at risk for developing nosocomial pneumonia because:

- His body habitus causes him to hypoventilate, leading to atelectasis
- Elevated intra-abdominal pressures related to obesity predispose aspiration
- All patients with cholecystitis are at risk of developing pneumonia
- Morbidly obese individuals have impaired neutrophil activity



Nursing Prevention

- HOB up
- Patients who can get out of bed to eat, need to get out of bed
- Assess for distended abdomens which can lead to vomiting
- Check for residuals on tube fed patients
- Altered LOC at particularly high risk
- Recovery position for patients vomiting

Pulmonary Hypertension

Disease Process

- Narrowing of the pulmonary arteries cause the right side of heart to work harder to pump blood through the lungs
- The heart muscle weakens and loses its ability to pump enough blood for the body
- Right heart failure is the most common cause of death in patients with PAH.

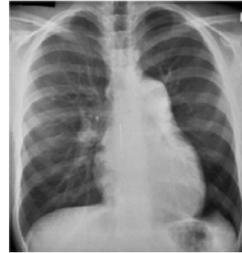
Disease Process...

- Normal pulmonary circulation is a high-flow, low-resistance circuit.
- Increased pulmonary artery pressure and pulmonary vascular resistance characterize pulmonary hypertension.

Pulmonary Hypertension

- Mean PAP is greater >25 mmHg at rest and greater than >30 mmHg during exercise
 - Normal mean PAP is 12-15 mmHg
- Symptoms
 - Progressive exertional dyspnea
 - May or may not have chest discomfort, lightheadedness or presyncope
 - Easily fatigued

Causes of Pulmonary Hypertension



- Cardiac: atrial septal defect, patent ductus arteriosus, aortic stenosis, cardiomyopathy
- Pulmonary: sleep disorders, cystic fibrosis, pneumonia
- Thromboembolic: VTE, parasitic disease, sickle cell anemia, polycythemia
- Hepatic disease: cirrhosis, portal hypertension
- Collagen vascular disease: scleroderma, lupus, rheumatoid arthritis
- Granulomatous disease: sarcoidosis

Pulmonary Hypertension

- Goals of treatment
 - Reduce symptoms & improve quality of life
 - Treat the underlying cause
 - Slow the development of blood clots.
 - Increase the supply of blood and oxygen to the heart, while reducing its workload.
- Treatment options:
 - Medication
 - Oxygen
 - Lung transplantation

Medications

- Prostacyclins
 - Epoprostenol (Flolan) given as a continuous infusion
 - Remodulin – given continuously SubQ or as a continuous infusion
 - Infused for life- Don't ever turn it off!
- Phosphodiesterase inhibitors
 - Revatio® (Sildenafil) - oral
- Anticoagulants
- Diuretics

REVIEW QUESTION

Treatment for pulmonary artery hypertension with a mean pulmonary artery pressure of 30mm Hg and signs of right sided heart failure consists of administration of oxygen and:

- A. Phlebotomy to maintain hematocrit at 48%
- B. Fluid bolus to increase right ventricular output
- C. Epoprostenol (Flolan) to dilate pulmonary arteries
- D. Inotropic agents to increase right ventricular contractility



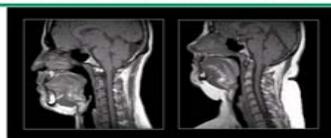
Sleep disordered breathing (SDB)

- SDB is a group of disorders characterized by pauses in breathing or the quantity of ventilation during sleep
- A condition characterized by repeated episodes of hypopnea (under breathing) and apnea (not breathing) during sleep

Sleep Apnea

- Greater than five apneas per one hour of sleep
- Cycle of sleep apnea:
 - Apnea + Hypopnea leads to Hypoxemia + Hypercapnia leads to the patient awakening

Reduced upper airway size in obstructive sleep apnea



Comparison of a mid-sagittal image of a normal subject (left) and a patient with sleep apnea (right). Soft palate and tongue area are larger in the patient with sleep apnea, leading to a reduction in upper airway size.
Courtesy of Richard J Schwab, MD.

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Obstructive Sleep Apnea

- Chest and abdomen move even though breathing has stopped due to upper airway obstruction
- Dilator muscles have continued narrowing

Risk Factors

- Genetic/Familial
- Neck circumference
- Obesity
- Male
- Middle age

Central Sleep Apnea

- Insufficient respiratory drive
- No coordination between diaphragm and upper airway muscles
- Increased CO₂ retention at times
- Decreased minute ventilation
- Movements of chest and abdomen also cease

Results of Sleep Apnea

- Hypersomnolence
- Loss of libido
- Decreased intellect
- Personality changes
- Morning headache
- Not feeling refreshed upon awakening
- Tossing and turning
- Choking and gagging
- Snoring
- Diaphoresis
- Quiet periods ending with loud gasp/grunt

Can lead to both short and long-term adverse problems if left untreated

Diagnostics

- Sleep Study with Respiratory Distress Index
 - $RDI = (\text{Respiratory Effort Related Arousals} + \text{Hypopneas} + \text{apneas}) \times 60 / \text{Total Sleep Time}(\text{in hours})$.
- Lateral cephalometry
 - Small posteriorly placed mandible
 - Narrow posterior airway space
 - Enlarged tongue
 - Inferiorly placed hyoid bone

Treatment Options for Sleep Apnea

- Surgery (UPP, tracheostomy)
- Devices
- CPAP-continuous positive airway pressure
- Electrical stimulation
- Antidepressants
- Treat hypothyroidism
- Acetazolamide
- Weight loss

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

- Least invasive AND most successful treatment modality for OSA
- Delivery of low levels of continuous pressure via a nasal or oronasal interface to “splint” open the airway during sleep

Continuous positive airway pressure (CPAP) for sleep apnea



The CPAP mask gently blows air into your nose while you sleep. It puts just enough pressure on your airway to keep it from closing. The mask in this picture fits over just the nose. Other CPAP devices have masks that fit over the nose and mouth.

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Acute Respiratory Failure

The inability of the cardiac & pulmonary systems to maintain an adequate exchange of oxygen & CO₂ in the lungs.

Respiratory Failure Acute vs Chronic

The inability to maintain adequate gas exchange, ventilation/gas transport, or tissue gas exchange

	Acute Respiratory Failure	Chronic Respiratory Failure
Etiology	Pneumonia, Acute Respiratory Distress Syndrome (ARDS), acute hypoxemia	COPD, lung CA, pulmonary fibrosis
Progression	Most patients are healthy and well prior to respiratory failure. Acute Respiratory Failure develops in hours to days	Underlying disease with progressively severe symptoms.
Prognosis	In-hospital death rate 42% 1-year mortality 11% (ARDS)	In-hospital death rate 11% 1-year mortality 43% (COPD)

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Classification of Respiratory Failure

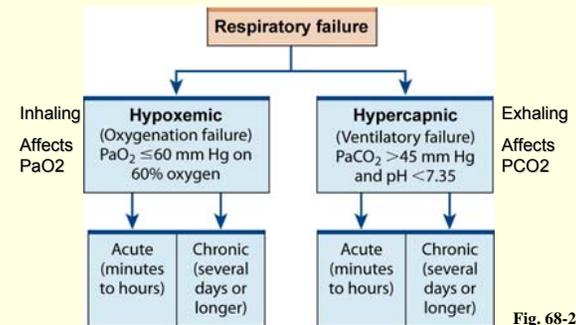


Fig. 68-2

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Hypoxemic Respiratory Failure- (Affects the pO₂)

- V/Q Mismatch
- Shunt
- Diffusion Limitation
- Alveolar Hypoventilation
 - ↑ CO₂ and ↓ PO₂

Etiologies of Hypoxemic ARF

- Pneumonia
- PE
- Plural Effusions
- Pneumothorax
- Early stages of ARDS
- Pulmonary Edema

Presentation

- Compensatory Mechanisms- early
 - Tachycardia- more O₂ to tissues
 - Hypertension- fight or flight
 - Tachypnea –take in more O₂
- Restlessness and apprehension
- Dyspnea
- Cyanosis
- Confusion and impaired judgment
- ABG: pO₂<50-60

Treatment of Hypoxemic Respiratory Failure

- Treat the underlying cause
- Support oxygenation / ventilation
 - Decrease the work of breathing
 - Decrease myocardial workload
 - Prevent or reverse tissue hypoxia

Hypercapnic Respiratory Failure Etiologies

- Abnormalities of the chest wall
 - Flail chest, morbid obesity, kyphoscoliosis
- Neuromuscular Conditions- respiratory muscles are weakened:
 - Guillain-Barre, muscular dystrophy, myasthenia gravis and multiple sclerosis, spinal cord injury
- Abnormalities of the CNS that suppress the drive to breathe.
 - Drug OD, Narcotics, Head injury
- Obstruction
 - Tumor, Mucus plug, Foreign body

Presentation

- Dyspnea to respiratory depression
 - CO₂ narcosis
- Headache
 - Vasodilation
 - Increases ICP
- Papilledema
- Tachycardia and inc. B/P
- Drowsiness and coma
- Respiratory acidosis

Treatment of Hypercapnic ARF

- Treat the underlying cause
- Non-invasive ventilation
- Invasive Mechanical ventilation
- Remove obstruction
- Bronchodilators

Acute Lung Injury (ALI) & Acute Respiratory Distress Syndrome (ARDS)

ALI and ARDS

- Affects ~150,000 -250,000 / year
- 50-70% mortality from multiple organ dysfunction syndrome
- Death is usually from complications of ARDS
- Syndromes characterized by
 - Acute respiratory failure
 - Non cardiac pulmonary edema
 - Refractory hypoxemia caused by intrapulmonary shunting

Acute Lung Injury/ Acute Respiratory Distress Syndrome

- Acute Lung Injury (ALI)
 - Severe diffuse lung injury
 - Pao₂/FiO₂ ratio < 300
 - Bilateral infiltrates
 - Pulmonary occlusive pressure ≤ 18
- Acute Respiratory Distress Syndrome (ARDS)
 - Most severe form of ALI
 - Same characteristics as ALI except Pao₂/FiO₂ ≤ 200
- Syndromes characterized by
 - Acute respiratory failure
 - Non cardiac pulmonary edema
 - Refractory hypoxemia caused by intrapulmonary shunting

PaO₂/FiO₂ ratio
 350-450 = Normal
 <300 = Acute Lung Injury
 <200 = ARDS

For Example...
 95/.21=450 80/.50= 160

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Potential etiologies

Direct Injury

- Pulmonary contusion
- Gastric aspiration
- **Pneumonia**
- Pulmonary embolism
- Hypervolemia
- Near drowning

Indirect Injury

- **Sepsis**
- Shock/ prolonged hypotension
- Cardiopulmonary bypass
- Multiple blood transfusions
- Burns
- DIC
- Drug overdose
- Pancreatitis

ALI/ARDS: Pathophysiology

Acute Phase (1-7 days)

Damage to alveolar epithelial cells → increased endothelial permeability → interstitial edema & leakage of protein-containing fluid into alveoli, loss of surfactant

- Hypoxemia resistant to high FiO₂
- Increased dead space (tissue that does not contribute to ventilation)
- Decreased lung compliance



Reparative or Prolific (1-2 weeks after injury)

Inflammatory response, ↑ pulmonary vascular resistance → Pulmonary HTN, destruction of the alveoli → worsening hypoxia

Recovery Phase (2-3 weeks after injury)

Gradual resolution of hypoxemia
 Improved lung compliance
 Resolution of radiographic abnormalities

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ALI & ARDS: clinical presentation

- S/s severe hypoxemia then refractory hypoxemia
- Diffuse bilateral infiltrates on CXR
- Marked reduced lung compliance
- PAP \uparrow , PAOP normal or low, CVP \uparrow
- ABGs show acute hypoxemia



ALI & ARDS: Treatment goals

- Improve delivery and reduce consumption of oxygen
 - Treat underlying cause
 - Maintain airway and ventilation
 - MV: low tidal volume and high PEEP, pressure control and I/E inverse ratio
 - \downarrow alveolar fluid: diuresis and fluid restriction

ALI & ARDS THERAPIES

- Hemodynamic Monitoring
- Inotropes as needed
- Antibiotics / Steroids / Sedation / Paralytics
- Proning/CLRT
- Extracorporeal Membranous Oxygenation (ECMO)
- Inhaled vasodilators
- Inverse I:E ratio
- PEEP
- Enteral Nutrition

Review Question

In a patient with ARDS, which of the following contribute to the development of atelectasis?

- A. Increased pulmonary vascular resistance / hypoxemia
- B. Increased pulmonary compliance/ hypoxemia
- C. Loss of surfactant / interstitial fluid accumulation
- D. Mucosal edema / mucous plugging



Mechanical Ventilation

Mechanical Ventilation: Indications

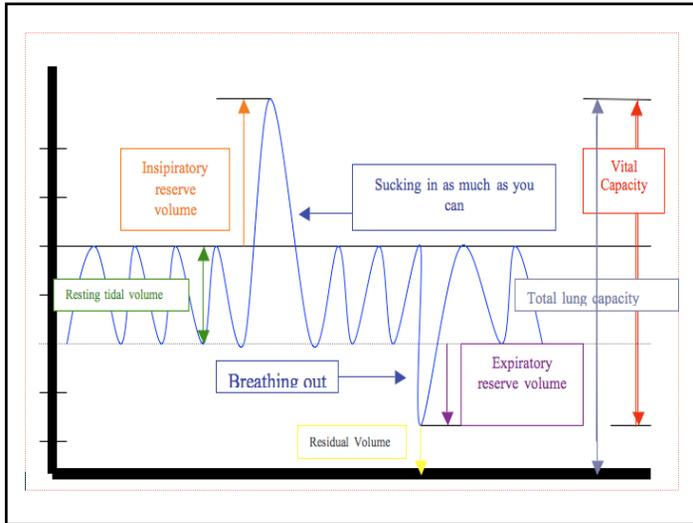
- Acute ventilatory failure with acidosis
- Hypoxemia despite adequate O₂
- CO₂ retention
- Apnea
- Marked increase in work of breathing and fatigue

Benefits

- Airway Protection
- Secretion Management
- Muscle Rest
- Restore and maintain gas exchange
- Reduce both systemic and myocardial O₂ requirements
- Permit sedation

It's breathtaking 😊

- TIDAL VOLUME (TV)
volume of air inhaled/exhaled with each breath
- INSPIRED RESERVED VOLUME (IRV)
volume of air inhaled during *maximal* inspiration
- EXPIRATORY RESERVE VOLUME(ERV)
volume of air maximally exhaled beyond the normal expiratory normal tidal volume
- RESIDUAL VOLUME (RV)
volume of air in lungs at end of maximal expiration
- FUNCTIONAL RESIDUAL CAPACITY (FRC)
volume of air remaining in the lungs at the end of expiration
- TOTAL LUNG CAPACITY
volume of air lungs can hold with a maximum inspiration



Methods of Mechanical Ventilation

- Non-invasive positive pressure ventilation via mask
 - CPAP: positive pressure throughout the ventilatory cycle + PEEP
 - IPAP = EPAP
 - Bi-PAP: provides two distinct levels of support during inspiration and expiration + PEEP
 - IPAP > EPAP
- Invasive positive pressure ventilation
 - Endotracheal tubes
 - Oral
 - Nasal
 - Tracheostomy tubes

MECHANICAL VENTILATION

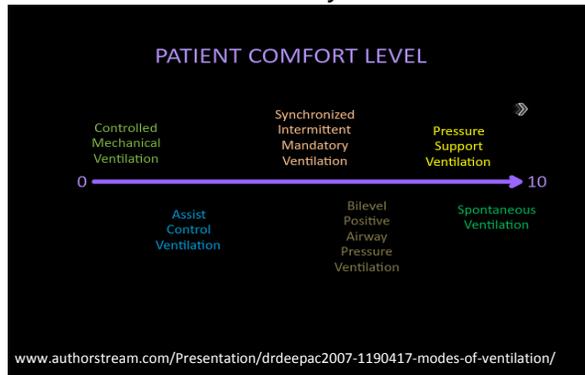
- VENTILATOR SETTINGS
 - FiO₂
 - Respiratory rate
 - Tidal Volume ~6-8 ml/kg of ideal body weight
 - Reduced volume protects against barotrauma
 - I:E Ratio
 - Normal breathing pattern is 1:2
 - Sigh
 - Programmed to occur several times per hour 1.5x the tidal volume
 - Pressure support
 - PEEP

Intubation

- Preparation
- Preoxygenation
- Pretreatment
- Paralysis with induction
- Protection and positioning
- Placement with proof (ETCO₂ detector)
- Postintubation management

Modes of Ventilation

Mode describes the *style* of the breath



High Frequency Ventilation

- Indications
 - Acute lung injury (ALI)/ Acute Respiratory Distress Syndrome (ARDS)
 - Bronchopleural fistula
 - May promote fistula closure by limiting alveolar distention
- Potential Complications
 - Pulmonary barotrauma
 - Hemodynamic instability
 - Necrotizing tracheobronchitis

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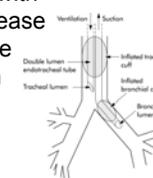
High Frequency Ventilation

- Mode that combines very high respiratory rates with small tidal volumes
- High frequency jet ventilation
 - A smaller tube is inserted into the ETT
 - 100-150 breaths per minute are delivered at a pressure of 35 psi
- High frequency oscillatory ventilation
 - Oscillatory pump delivers up to 900 breaths per minute through the ETT
 - Constant airway pressure maintains alveolar recruitment and impacts oxygenation

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Independent Lung Ventilation

- Ventilates each lung separately
- Requires 2 ventilators and a double lumen ETT
- Used for patients with unilaterial lung disease or different disease processes in each lung
- Indications
 - Massive unilateral hemoptysis
 - Lung abscess
 - Aspiration
 - Pulmonary contusion
 - Unilateral pneumonia or pulmonary edema
 - Bronchopleural fistula
 - Single lung transplant



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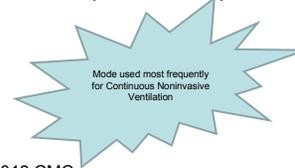
Adjuncts to Vent Modes

- PEEP: Positive End Expiratory Pressure
 - Delivered at the end of expiration
 - Increases functional residual capacity
 - Increases area for gas exchange
 - May decrease CO especially if hypovolemic
- Pressure Support
 - Delivered at the beginning of inspiration
 - Used alone or added to SIMV
 - Preset pressure to augment the tidal volume of each spontaneous breath
 - A "boost" from the ventilator
 - Patient must generate a negative flow or pressure to trigger the support
 - If pressure support is used as a stand-alone mode *all* breaths are spontaneous

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Positive Airway Pressure

- BiPAP
 - Bi-level Positive Airway Pressure
 - The inspiratory pressure is greater than the expiratory pressure
 - IPAP > EPAP
 - Continuous high flow positive airway pressure that cycles between inspiration and expiration
- CPAP
 - Continuous Positive Airway Pressure
 - Inspiratory pressure and expiratory pressure are the same
 - IPAP=EPAP
 - Pressures are generally lower than BiPAP



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MECHANICAL VENTILATION ABG Regulation

- PaCO₂ > 45 mmHg
 - ↑ Ventilation :
 - ↑ Rate
 - ↑ Tidal Volume
- PaO₂ < 60 mmHg
 - ↑ FiO₂, ↑ PEEP
- PaCO₂ < 35 mmHg
 - ↓ Ventilation :
 - ↓ Rate
 - ↓ TV
 - Change mode if AC to IMV
 - Consider Sedation
- PaO₂ > 100 mmHg
 - ↓ FiO₂, ↓ PEEP

Can We Wean??

Clinical criteria used to determine readiness for trials of spontaneous breathing

Required criteria
1. The cause of the respiratory failure has improved
2. PaO ₂ /FiO ₂ ≥ 150* or SpO ₂ ≥ 90 percent on FiO ₂ ≤ 40 percent and positive end-expiratory pressure (PEEP) ≤ 5 cmH ₂ O
3. pH > 7.25
4. Hemodynamic stability (no or low dose vasopressor medications)
5. Able to initiate an inspiratory effort
Additional criteria (optional criteria)
1. Hemoglobin ≥ 8 to 10 mg/dL
2. Core temperature ≤ 38 to 38.5 degrees Centigrade
3. Mental status awake and alert or easily arousable

* A threshold of PaO₂/FiO₂ ≥ 120 can be used for patients with chronic hypoxemia. Some patients require higher levels of PEEP to avoid atelectasis during mechanical ventilation.
Adapted from: MacIntyre, NR, Cook, DJ, Ely, EW, Jr., et al. Evidence-based guidelines for weaning and discontinuing ventilatory support: a collective task force facilitated by the American College of Chest Physicians, the American Association for Respiratory Care, and the American College of Critical Care Medicine. *Chest* 2001; 120:375S.

UptoDate

Potential Ventilator Associated Complications...

- Barotrauma/volutrauma
- Hemodynamic Changes
- Upper Airway Damage
- Oxygen Toxicity
- Ventilator Associated Pneumonia (VAP)

Plus...

- Muscle atrophy
- Anxiety
- Inability to wean from MV
- Skin breakdown

Barotrauma/Volutrauma

- Barotrauma
- Volutrauma
 - High volumes cause damage to the alveoli
 - Increased risk of pneumothorax

Hemodynamic Compromise

- High pressures and PEEP cause high pressure to be transmitted to the mediastinal structures
- Decreased venous return
- Drop in CO

Upper Airway Damage

- Tracheal stenosis
- Laryngeal stenosis
- Vocal cord paralysis
- Tracheal-esophageal fistula
- Innominate artery damage

Ventilator Associated Pneumonia (VAP)

- VAP is the leading cause of death amongst hospital-acquired infections
 - Hospital mortality of ventilated patients who develop VAP is 46% compared to 32% for ventilated patients who do not develop VAP
 - Nosocomial pneumonia
 - Pseudomonas Aeruginosa (gram -)
 - Staphylococcus Aureus (gram +)

VAP

- CONTRIBUTING FACTORS
 - Supine position
 - Restrained
 - Inadequate mouth care
- VAP Prevention
 - Elevation of the Head of the Bed
 - Daily "Sedation Vacations" and Assessment of Readiness to Extubate
 - Peptic Ulcer Disease Prophylaxis
 - Deep Venous Thrombosis Prophylaxis
 - Oral care

REVIEW QUESTION

Which one of the following can be an effect of positive - pressure ventilator therapy?

- A. Decrease in CO
- B. Decrease in bronchial secretions
- C. Increase in lung compliance
- D. Increase in CO



REVIEW QUESTION

A nurse preceptor is working with an orientee who just admitted a patient believed to have ventilator-associated pneumonia. In reviewing the various interventions that may be helpful in preventing this disorder, which should the preceptor emphasize as the most important?

- A. Locating the tip of the NG in the post-pyloric area
- B. Maintaining HOB elevation at 30-45 degrees
- C. Suctioning the oropharynx and ETT hourly
- D. Using hyperalimentation instead of enteral feedings



Modes of Mechanical Ventilation

- The following slides describe commonly used modes of mechanical ventilation
- As you prepare for your test remember to think about the nursing actions and assessment findings associated with mechanical ventilation
 - ABG interpretation
 - Complications: barotrauma, VAP
 - Monitoring parameters
 - Readiness to wean

Continuous Mandatory Ventilation (CMV)

- Set tidal volume and number of breaths per minute
 - The patient does not initiate additional ventilation
 - Requires sedation or paralysis
- Advantage: Provides maximum respiratory muscle rest
- Disadvantages: requires sedation, causes significant respiratory muscle atrophy
- Nursing Care
 - Maintain sedation or paralysis
 - Monitor for hypoxia or hypercapnea
 - Passive and active range of motion
 - Maintain oral hygiene
 - VAP prevention measures

Assist Control A/C

- Set tidal volume and number of breaths per minute
 - The respiratory rate is typically set four breaths per minute below the patient's native rate
 - For each breath the patient takes above the set rate, they get the full tidal volume
- Advantage: provides respiratory muscle rest
- Disadvantages
 - Air hunger may occur if flow or tidal volume are set too low
 - high pressures can cause barotrauma to the lungs
 - Respiratory muscle atrophy
- Nursing Care
 - Monitor for hypoxia or hypercapnea
 - Assess for dyspnea, feelings of suffocation or other distress (signs of air hunger)
 - Passive and active range of motion
 - Maintain oral hygiene
 - VAP prevention measures
 - Observe the patient's respiratory rate
 - Spontaneous breaths vs breaths delivered by the vent

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Synchronized Intermittent Mandatory Ventilation (SIMV)

- Set tidal volume and number of breaths per minute
- Not all breaths are assisted
 - For each breath the patient takes above the set rate, they only get the size of breath they can generate on their own
 - The patient can breathe on their own between the assisted breaths delivered by the ventilator
- Advantages
 - allows patients to adjust the size of their extra breaths to account for metabolic or respiratory changes
 - Better preservation of respiratory muscle function
- Nursing Care
 - Monitor for hypoxia or hypercapnea
 - Passive and active range of motion
 - Maintain oral hygiene
 - VAP prevention measures
 - Observe the patient's respiratory rate
 - Spontaneous breaths vs breaths delivered by the vent

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Pressure Support Ventilation PSV

- Used alone or added to SIMV
 - Preset pressure to augment the tidal volume of each spontaneous breath
 - A "boost" from the ventilator
- Patient must generate a negative flow or pressure to trigger the support
- As inspiratory efforts decline, the flow decelerates and gradually terminates
- If pressure support is used as a stand-alone mode *all* breaths are spontaneous
- Nursing Care
 - Monitor for hypoxia or hypercapnea
 - Passive and active range of motion
 - Maintain oral hygiene
 - VAP prevention measures
 - Observe the patient's respiratory rate
 - Assess for fatigue or other signs the patient may not be able to continue spontaneous respiration

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