Introduction and Overview of Acute Respiratory Failure
Definition: Acute Respiratory Failure

- Failure to oxygenate
  - Inadequate PaO\textsubscript{2} to saturate hemoglobin
  - PaO\textsubscript{2} of 60 mm Hg ~ SaO\textsubscript{2} of 90%
  - PaO\textsubscript{2} of 50 mm Hg ~ SaO\textsubscript{2} of 75%
- Failure to ventilate
  - Elevation in PaCO\textsubscript{2} associated with decreased pH
  - PaCO\textsubscript{2} of 60 mm Hg ~ pH 7.20
Differentiate ↓O₂ from ↑CO₂

- Hypoxemia (PaO₂<60 mm Hg) and hypercarbia (elevated PaCO₂ with low pH) may occur independently or together
- Hypoxemia:
  - ventilation-perfusion mismatching
  - shunt physiology
- Hypercarbia:
  - alveolar hypoventilation
  - increased dead space fraction
  - increased production (overfeeding, hypermetabolism)
Acute Respiratory Failure

- The goal is to maintain adequate tissue oxygenation to avoid anaerobic metabolism.
- When oxygen supply ≠ demand:
  - Organ dysfunction
  - Lactate generation
- Determinants of supply
  - Cardiac output (5L/minute)
  - Blood oxygen content (20mL O₂ /100mL blood)
    - Hemoglobin
    - PaO₂ / saturation
Normal oxygen transport

- $\text{O}_2$ delivery = cardiac output x blood oxygen content
  - $\text{DO}_2 = \text{C.O.} \times \text{CaO}_2$
  - $\text{DO}_2 = 5\text{L/min} \times 20 \text{mL/100 mL}$
  - $\text{DO}_2 = 1000 \text{mL/minute}$

- Tissue metabolism
  - $\text{VO}_2 = 250 \text{mL/minute or 4 mL/kg}$
- Extraction fraction = 0.25
  - Mixed venous saturation = 75%
  - Mixed venous PaO$_2$ = 40 mm Hg
Inadequate tissue oxygen delivery

- Reduced O$_2$ delivery
  - Cardiac output: 2.5L/min
  - Anemia: Hgb 7.5 gm/dL
  - Hypoxemia: Hgb saturation 75%/PaO$_2$ 40 mm Hg

- If constant O$_2$ consumption (250mL/min):
  - Extraction fraction will be 50%
  - Mixed venous saturation = 50%
  - Mixed venous PaO$_2$ = 27 mm Hg
  - Driving pressure for O$_2$ diffusion from capillaries to cells is reduced: anaerobic metabolism
General management principles

• Optimize oxygen delivery
  – Transfusion of packed RBC
  – Increase cardiac output
  – Relieve arterial hypoxemia

• Reduce oxygen consumption
  – Endotracheal intubation: deliver higher FiO₂ and relieve work of breathing
  – Treat fever: to reduce tissue metabolic rate
Patient-related risk factors

- **Smoking:**
  - Must stop >8 weeks preoperatively to decrease risk

- **General health:**
  - Inability to exercise (supine bicycle) for 2 minutes with HR>99/min

- **COPD:**
  - Must optimize medical management/functional capacity preoperatively

- **Asthma:**
  - If well-controlled preoperatively, not a risk factor

- **Age and obesity are not significant risk factors**
Procedure-related risk factors

- **Surgical site:**
  - Risk increases as incision approaches the diaphragm
  - Open procedures higher risk than laparoscopic
- **Duration:**
  - Procedures >3 hours carry higher risk
- **Anesthesia/pain control:**
  - Consider spinal or regional anesthesia for high risk patients

Diagnosis of high risk patients

• History: Chronic cough, unexplained dyspnea, poor functional status
• Physical: Wheeze, prolonged expiration
• Chest X-ray: Hyperinflation
• Spirometry:
  • Obstruction present if FEV$_1$/FVC <70%
  • Severity based on % predicted FEV$_1$:
    – Mild: 65-80%
    – Moderate 50-65%
    – Moderately Severe 30-50%
    – Severe <30%
Intra-Pulmonary Shunt

- Blood that enters the arterial system without going through ventilated areas of lung

- Same physiology as extrapulmonary cardiac shunt:
  - i.e. Atrial septal defect, patent foramen ovale

- Calculated amount of venous blood that must mix with arterial blood to produce the observed PaO\(_2\) is sometimes called “venous admixture”

- Shunt is refractory to supplemental oxygen
Hypercarbic (Type II) respiratory failure

- Also termed acute on chronic respiratory failure
- Usually seen in patients with severe COPD
- Occasionally occurs in a patient that has not been diagnosed with obstructive lung disease

- COPD is the 4\textsuperscript{th} leading cause of death (2000)
- Patients have expiratory airflow limitation
- FEV1/FVC < 70%
- Measured as FEV1 (L); lower value = more severe
Prognosis: Type II respiratory failure

- High short-term risk of death but up to half of discharged patients will survive for one year
- Half of survivors describe quality of life as good or better
- A proportion of these patients will return to work
- Older patient, more co-morbidities, lower baseline level of function predict poor survival but are not refined enough for adequate prognostication
- Extensive critical care resource utilization
- Ideally, management discussion is undertaken before acute deterioration
Type II respiratory failure

- Multiple, minor insults cause acute deterioration of chronic (precariously compensated) respiratory status
- Respiratory failure often is slowly progressive
- Patients are distressed, using accessory muscles, have prolonged expiratory time, wheezing
- In late phases: obtundation, apnea
- Low PaO$_2$ and high PaCO$_2$
- PACO$_2$ is directly related to alveolar ventilation
- Relatively easy to oxygenate in comparison to ARDS
Precipitating factors for type II respiratory failure

• In the postoperative setting, consider effects of:
  – Sedation
  – Site of Incision/Pain
  – Fluid overload, myocardial ischemia
  – Bronchospasm
  – Infection
Respiratory load vs. strength

- Ventilation is accomplished by the diaphragm and other respiratory muscles working as a piston or pump.

- When stimulated by the central nervous system, diaphragmatic and intercostal contraction reduce pleural pressure, causing lung inflation.

- Muscles will fatigue when the load is excessive.
  - Fatigue = failure to develop the normal degree of tension despite maximal stimulation.

- Airways contribute a resistive load that is increased by bronchospasm, secretions, inflammation.

- Lungs contribute an elastic load due to dynamic hyperinflation - a failure to fully empty, resulting in intrinsic PEEP.
Key elements of respiratory system competence

• Adequate central respiratory drive
  – anesthetics, narcotics, benzodiazepines, hypothyroidism

• Neuromuscular competence
  – corticosteroids, malnutrition, phrenic nerve injury, paralytics, Guillain-Barré syndrome

• Diaphragmatic strength/pressure generation
  – hyperinflation, malnutrition, electrolyte disturbance, hypoxemia, myopathy

• Low airways resistance
  – secretions, edema, bronchospasm, sleep apnea

• Adequate lung and chest wall compliance
  – obesity, ascites, ileus, interstitial edema, pleural effusion, infection, atelectasis, rib fracture, pneumothorax, iPEEP
Dead space and hypercarbia

- The portion of ventilation that doesn’t participate in gas exchange: Exhaled unchanged
- The effective portion of minute ventilation is alveolar ventilation
- Apparatus dead space (if using a breathing apparatus)
- Anatomic dead space (volume of conducting passages) ~2mL/kg in adults
- Alveolar dead space (under/unperfused alveoli)
- Physiological dead space = sum of anatomical and alveolar dead space
- Increase in dead space ventilation will raise PaCO$_2$
- Classically seen in pulmonary embolism
Alveolar Dead Space

Ventilation without perfusion - can’t eliminate arterial CO₂
Physiologic effects of CO$_2$

- Major regulator of cerebral blood flow: CBF increases via vasodilation due to CSF acidosis; this may increase intracranial pressure.
- Increased circulating catecholamines (epi > norepi)
- Pulmonary arterial vasoconstriction (less than seen with hypoxia)
- Shifts oxyhemoglobin dissociation curve to the right
- Displaces oxygen in alveolar gas
- Slight net increase in blood pressure
- Bicarbonate retention by kidneys
- Increase in plasma potassium
Approach to type II respiratory failure

- Recognize the factors that create an imbalance between ventilatory load and reserve
- Position patient to facilitate ventilation (upright or semi-erect)
- Administer oxygen: to obtain $\text{SaO}_2 \geq 90$
- Bronchodilators for reversible component of COPD:
  - Salbutamol 2-4 puffs q1-4h: $\beta$-agonist (rapid onset, short-acting) or nebulizer if unable to control breathing pattern
  - Ipratropium 2-4 puffs qid: Anticholinergic, slower onset, longer duration
  - Consider corticosteroids (more for asthmatics than COPD)
- Antibiotics
- Diuretics
- Anti-ischemic therapy (nitrates); blood transfusion if indicated
Early acute on chronic respiratory failure

- Recognize impending respiratory failure:
  - Deteriorating mental status
  - Subjective sense of exhaustion
  - Tachypnea
  - Accessory muscle use
  - Thoraco-abdominal paradox
  - Cardiovascular instability (arrythmia, labile BP)
Non-invasive ventilation (NIPPV)

- Face mask with higher inspiratory than expiratory pressure support (BiPAP)
- Reduces need for intubation and mortality
- Strongest evidence is in exacerbation of COPD
- Other indications:
  - cardiogenic pulmonary edema
  - immunocompromised host with respiratory insufficiency
  - weaning from ventilator or post-extubation
NIPPV

- Compliant patient; able to synchronize with ventilator
- Appropriately fitting mask/seal
- Monitoring: HR, SaO₂, BP
- Bedside presence for clinical response (usually starts in 10 minutes or less)
  - Improved mental status
  - Decreased respiratory rate/increased tidal volume
  - Improved oxygen saturation/ABG

- If not tolerated or not improving: consider mechanical ventilation
### Table 1  Contraindications to use of noninvasive ventilation

<table>
<thead>
<tr>
<th>Condition</th>
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<tbody>
<tr>
<td>Impending cardiovascular collapse or respiratory arrest</td>
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<td>Excessive secretions or massive upper gastrointestinal bleeding</td>
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<td>Upper airway obstruction</td>
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<td>Recent facial, upper airway, or upper gastrointestinal surgery</td>
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<td>Patient unable to protect airway, including altered mental status</td>
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<td>No monitored beds available (relative)</td>
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Goals of ventilation: Type II respiratory failure

- Immediate stabilization of airway, oxygenation, hemodynamics
- Rest fatigued respiratory muscles
  - 48-72 hours; usually volume-controlled mode of ventilation
- Reverse dynamic hyperinflation/iPEEP
  - Ensure adequate inspiratory flow rate
  - Ensure adequate expiratory time to allow full lung emptying
- Treat causes of increased airways resistance
  - Bronchodilators, antibiotics, diuretics, steroids
- Provide time for reversal of other specific precipitating factors
  - Nutrition, electrolyte correction, etc.
Resolution of acute respiratory failure

<table>
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<th>Table 3</th>
<th>Suggested criteria for consideration of extubation readiness</th>
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<tr>
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<td>Underlying disease process that necessitated intubation is improving</td>
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<td></td>
<td>Hemodynamically stable</td>
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<td></td>
<td>Appropriate mental status</td>
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<td>Capable of upper airway protection</td>
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<td>Ventilator settings: fraction of inspired oxygen ≤0.40; positive end-expiratory pressure ≤8 cm H₂O</td>
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Summary: Acute Respiratory Failure

• The main function of the respiratory system is to oxygenate tissue and remove CO$_2$ by ventilation

• ARDS is a primarily a failure to oxygenate caused by acute lung inflammation and alveolar flooding with shunt physiology

• Acute on chronic (type II) respiratory failure is primarily due to a failure of adequate ventilation caused by depressed respiratory drive, altered neuromuscular coupling or increased airways resistance/collapse
Summary: Acute Respiratory Failure

- **Disease-related factors**
  - Aspiration, pneumonia, trauma, shock: ARDS

- **Patient-related factors**
  - Obstructive lung disease: Type II respiratory failure

- **Procedure-related factors**
  - Proximity to diaphragm, duration of procedure
Management:
- Anticipate ARF in high risk patients and initiate postoperative interventions (adequate pain control, spirometry, etc.)
- Recognize need for ventilatory assistance
- Lung protective strategy: ARDS
- Non-invasive ventilation: Type II failure
- Supportive care and reversal of provocative factors
  - Antibiotics, bronchodilators, reduction of steroids, etc.