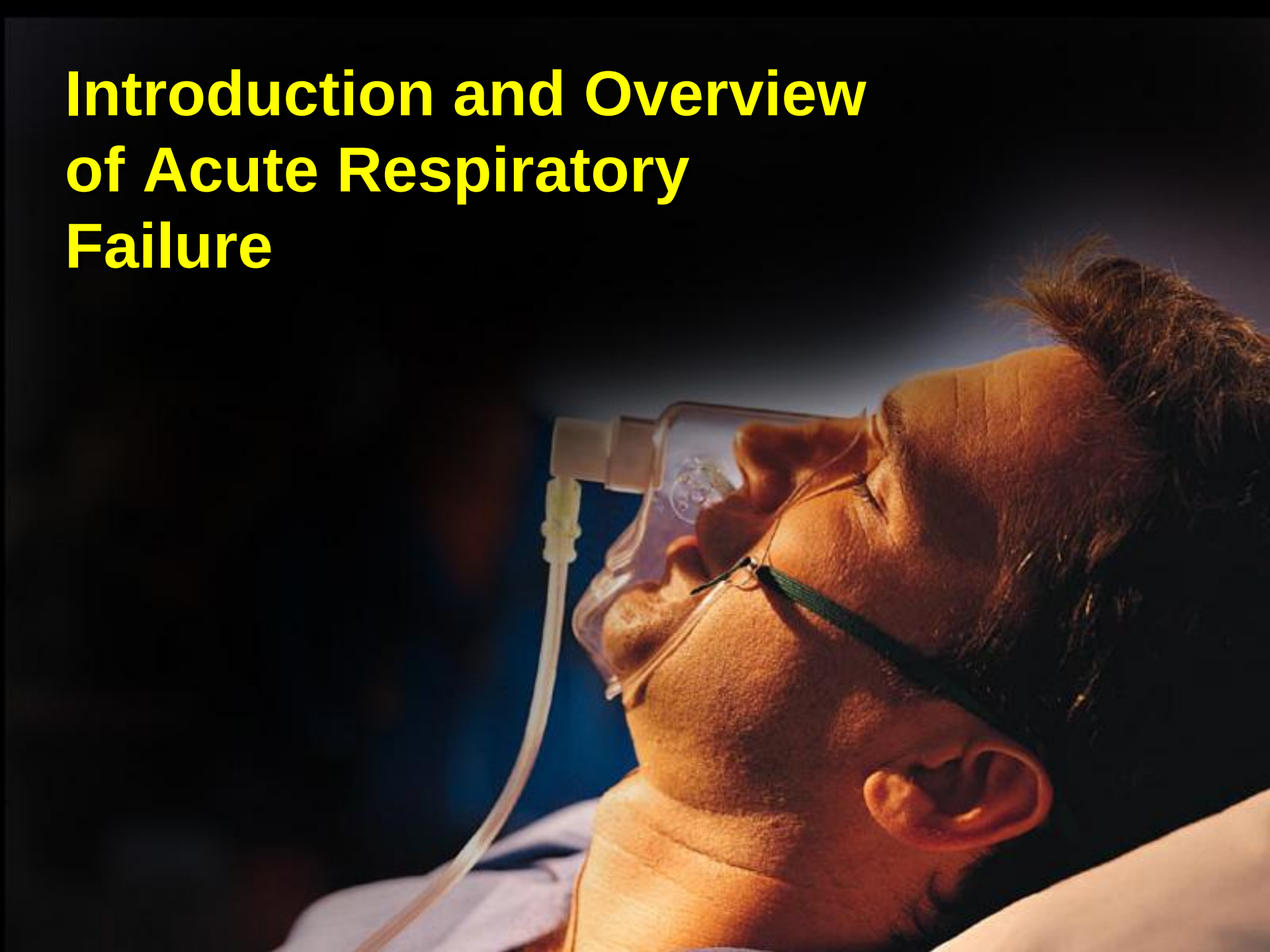
Introduction and Overview of Acute Respiratory Failure



Definition: Acute Respiratory Failure

- Failure to oxygenate
  - Inadequate PaO_2 to saturate hemoglobin
  - PaO_2 of 60 mm Hg ~ SaO_2 of 90%
  - PaO_2 of 50 mm Hg ~ SaO_2 of 75%
- Failure to ventilate
  - Elevation in $PaCO_2$ associated with decreased pH
  - $PaCO_2$ of 60 mm Hg ~ pH 7.20

Differentiate $\downarrow O_2$ from $\uparrow CO_2$

- Hypoxemia (PaO_2<60 mm Hg) and hypercarbia (elevated $PaCO_2$ 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 O_2 /100mL blood)
    - Hemoglobin
    - PaO_2 / saturation

Normal oxygen transport

- O_2 delivery=cardiac output x blood oxygen content
  - DO_2 = C.O. x CaO_2
  - DO_2 = 5L/min x 20 mL/100 mL
  - DO_2 = 1000 mL/minute
- Tissue metabolism
  - VO_2 = 250 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 FiO_2 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

***N Engl J Med* 1999 340: 937**

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

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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%

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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 4th 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

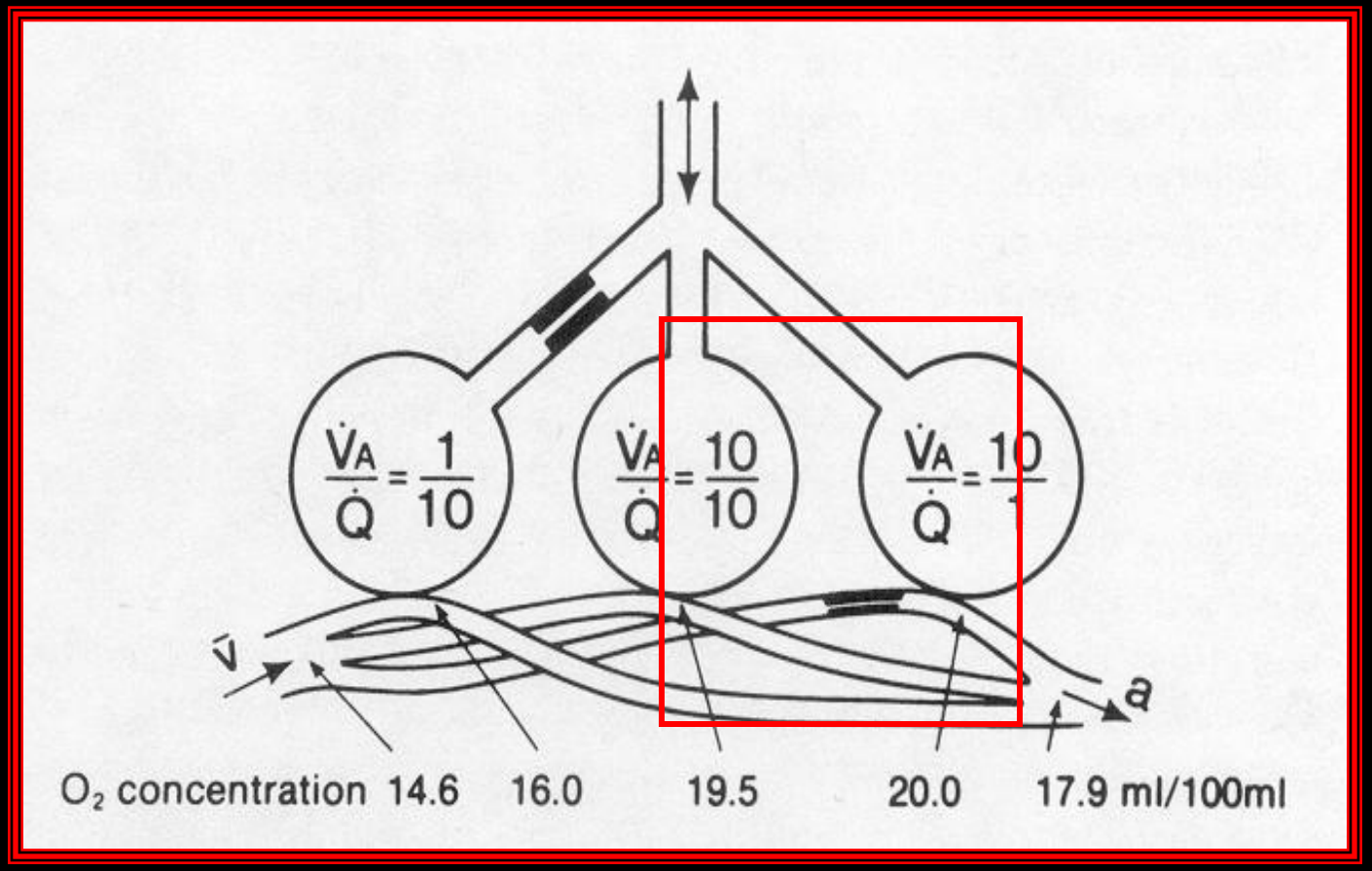
Key elements of respiratory system competence

- 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 CO_2

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 SaO_2 ≥90%

- Bronchodilators for reversible component of COPD:
  - Salbutamol 2-4 puffs q1-4h: β-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, SaO_2, 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

Contraindications to non-invasive ventilation

Table 1 Contraindications to use of noninvasive ventilation

Impending cardiovascular collapse or respiratory arrest
Excessive secretions or massive upper gastrointestinal bleeding
Upper airway obstruction
Recent facial, upper airway, or upper gastrointestinal surgery
Patient unable to protect airway, including altered mental status
No monitored beds available (relative)

***Am J Med* 2005 118: 584**

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 3 Suggested criteria for consideration of extubation readiness

Underlying disease process that necessitated intubation is improving
Hemodynamically stable
Appropriate mental status
Capable of upper airway protection
Ventilator settings: fraction of inspired oxygen ≤0.40; positive end-expiratory pressure ≤8 cm H_2O

***Am J Med* 118: 584**

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

Summary: Acute Respiratory Failure

- 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.