ACUTE RESPIRATORY FAILURE

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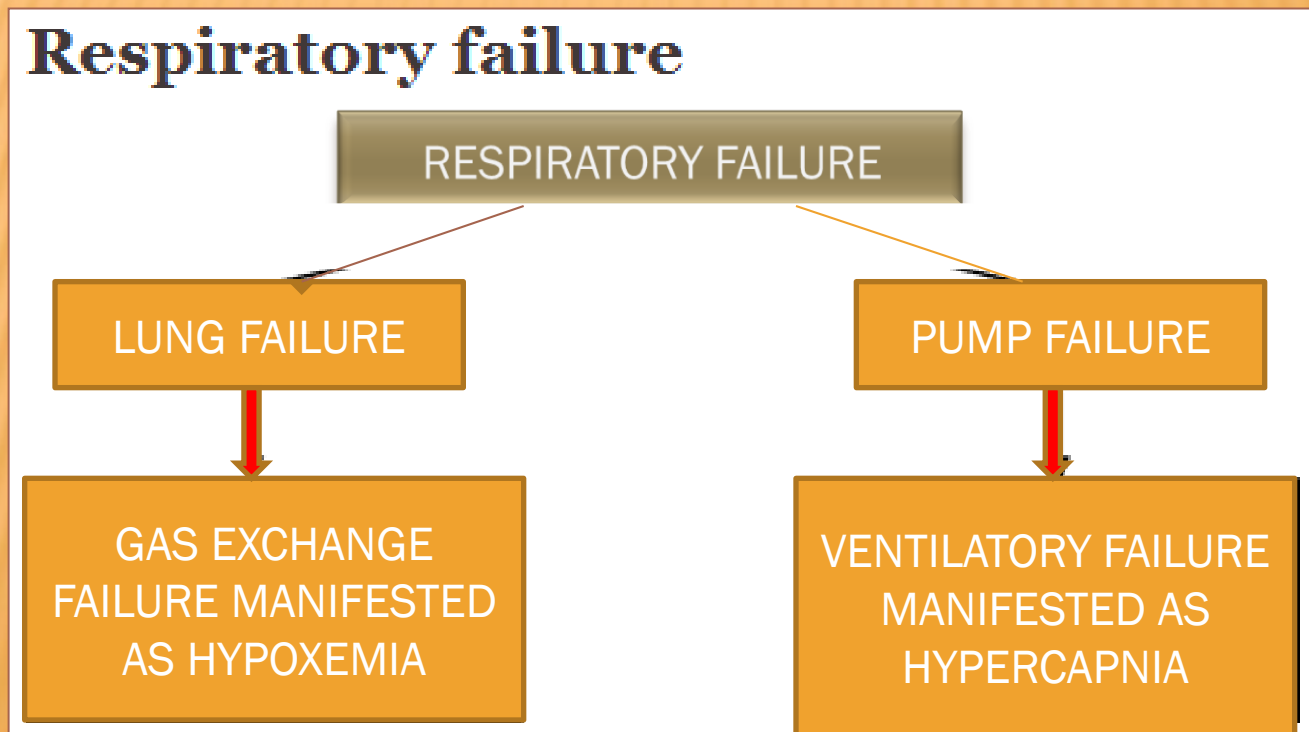
Junior Resident Pulmonary Medicine

- DEFINITION
- TYPES
- ETIOLOGY
- PATHOPHYSIOLOGY
- CATEGORIES OF RESPIRATORY FAILURE
- APPROACH TO THE PATIENT
- PRINCIPLES OF MANAGEMENT
- MONITORING PATIENTS WITH ACUTE
RESPIRATORY FAILURE
- COMPLICATIONS OF ACUTE RESPIRATORY FAILURE
- PROGNOSIS
- CONCLUSION

Respiratory failure occurs mainly either due to lung failure resulting in hypoxaemia or pump failure resulting in alveolar hypoventilation and hypercapnia. Hypercapnic respiratory failure may be the result of mechanical defects, central nervous system depression, imbalance of energy demands and supplies and/or adaptation of central controllers.

Respiratory failure

RESPIRATORY FAILURE

LUNG FAILURE

PUMP FAILURE

GAS EXCHANGE FAILURE MANIFESTED AS HYPOXEMIA

VENTILATORY FAILURE MANIFESTED AS HYPERCAPNIA

CLASSIFICATION
Classified mechanically based on pathophysiologic derangement in respiratory function

TYPE I OR ACUTE HYPOXEMIC RESPIRATORY FAILURE — Often secondary to pulmonary edema and subsequent intrapulmonary shunting

TYPE II RESPIRATORY FAILURE — Secondary to alveolar hypoventilation, resulting in the inability to effectively eliminate carbon dioxide

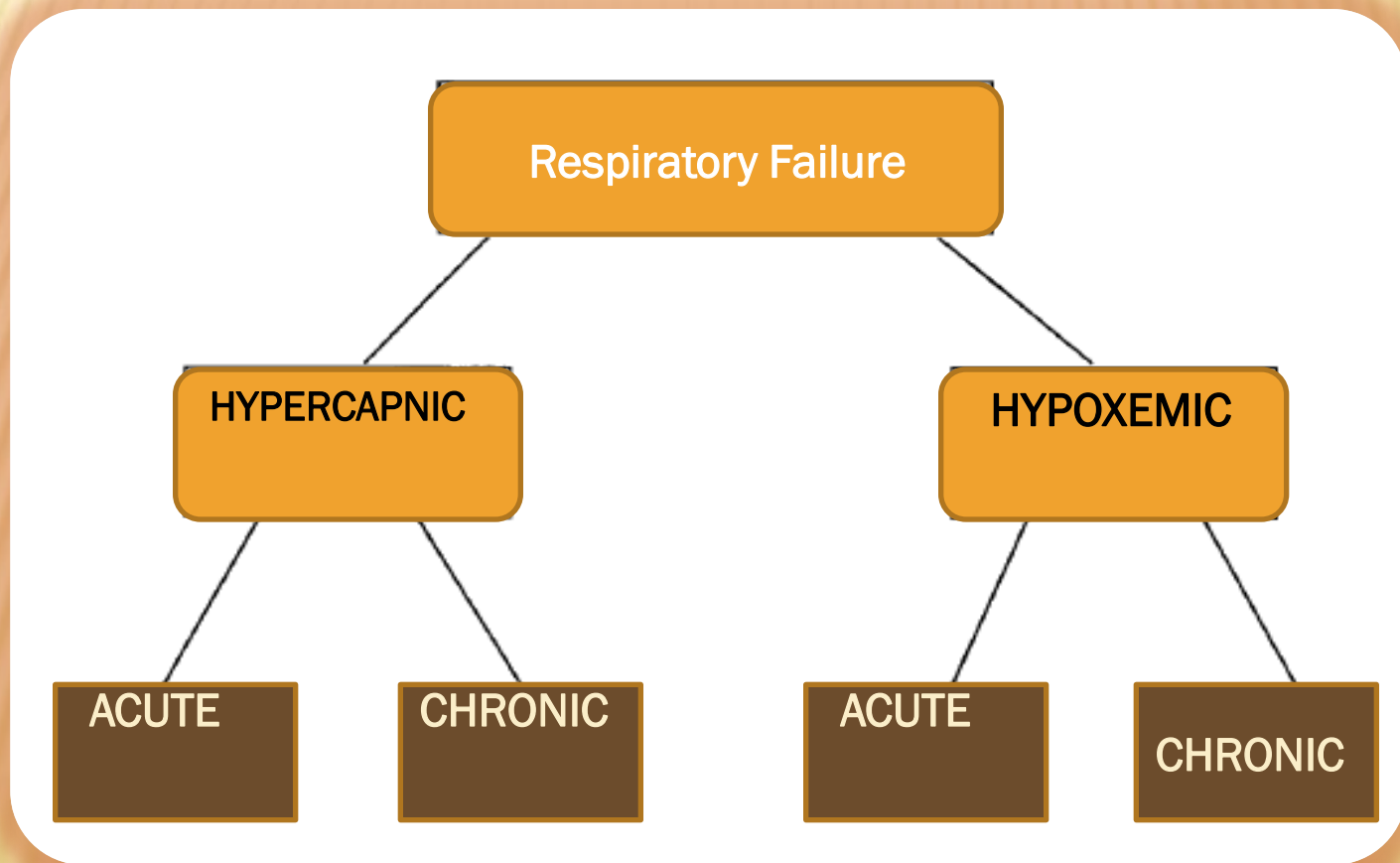**TYPE III PERIOPERATIVE RESPIRATORY FAILURE** — Secondary to lung atelectasis

TYPE IV RESPIRATORY FAILURE — Secondary to hypoperfusion of respiratory muscles in patients in shock

Respiratory Failure may be classified as *hypercapnic or hypoxemic*

Hypercapnic respiratory failure is defined as an arterial Pco2 (Paco2) greater than 45 mmHg.

Hypoxemic respiratory failure is defined as an arterial Po2 (Pao2) less than 55 mmHg when the fraction of oxygen in inspired air (Fio2) is 0.60 or greater.

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                    Respiratory Failure
                    /               \
          HYPERCAPNIC              HYPOXEMIC
           /       \                /       \
       ACUTE    CHRONIC         ACUTE     CHRONIC
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DISTINCTIONS BETWEEN *ACUTE AND CHRONIC RESPIRATORY FAILURE*

CATEGORY	CHARACTERISTICS
Hypercapnic respiratory failure	$Pa_{CO_2} > 45$ mmHg
Acute	Develops in min to h
Chronic	Develops over several days or longer
Hypoxemic respiratory failure	$Pa_{O_2} < 55$ mmHg when $F_{IO_2} \geq 0.60$
Acute	Develops in min to h
Chronic	Develops over several days or longer

Distinction between acute and chronic hypoxemic respiratory failure may not be readily made on the basis of arterial blood gas values.

The presence of markers of chronic hypoxemia (e.g., polycythemia or cor pulmonale) provides clues to CHRONIC disorder, whereas abrupt changes in mental status suggest an ACUTE event.

## Distinctions between *acute and chronic respiratory failure*

Acute hypercapnic respiratory failure is defined as a $Paco_2$ greater than 45 mmHg with accompanying acidemia (pH less than 7.30). The physiological effect of a sudden increment in $Paco_2$ depends on the prevailing level of serum bicarbonate anion.

In patients with chronic hypercapnic respiratory failure—e.g., due to chronic obstructive pulmonary disease (COPD)—a long-standing increase in $Paco_2$ results in renal "compensation" and an increased serum bicarbonate concentration.

Acute-on-chronic respiratory failure

Another entity apart from acute & chronic respiratory failure is Acute-on-chronic respiratory failure
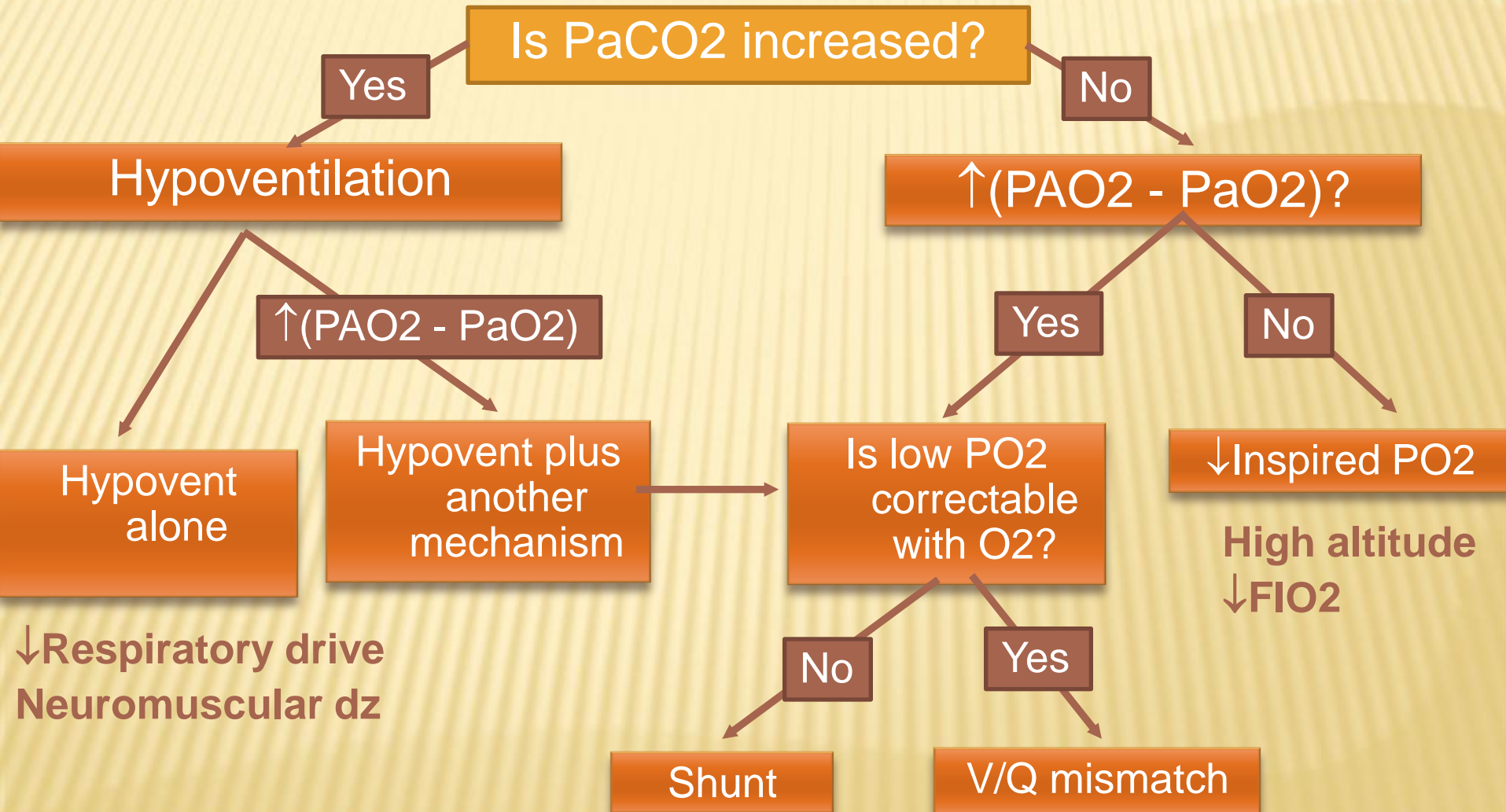
Acute deterioration in a patient with chronic respiratory failure is termed acute-on-chronic respiratory failure. Patients may present with worsening dyspnoea, deteriorating mental status or respiratory arrest after relatively minor, although often multiple, insults. Acute-on-chronic respiratory failure is usually seen in patients known to have **severe COPD**. Patients with severe but stable COPD exist in a very critical balance between increased demands and limited reserves. Any factor that potentially interferes with this balance (either increase in demands or decrease in reserves) leads to respiratory muscle fatigue and acute respiratory failure.
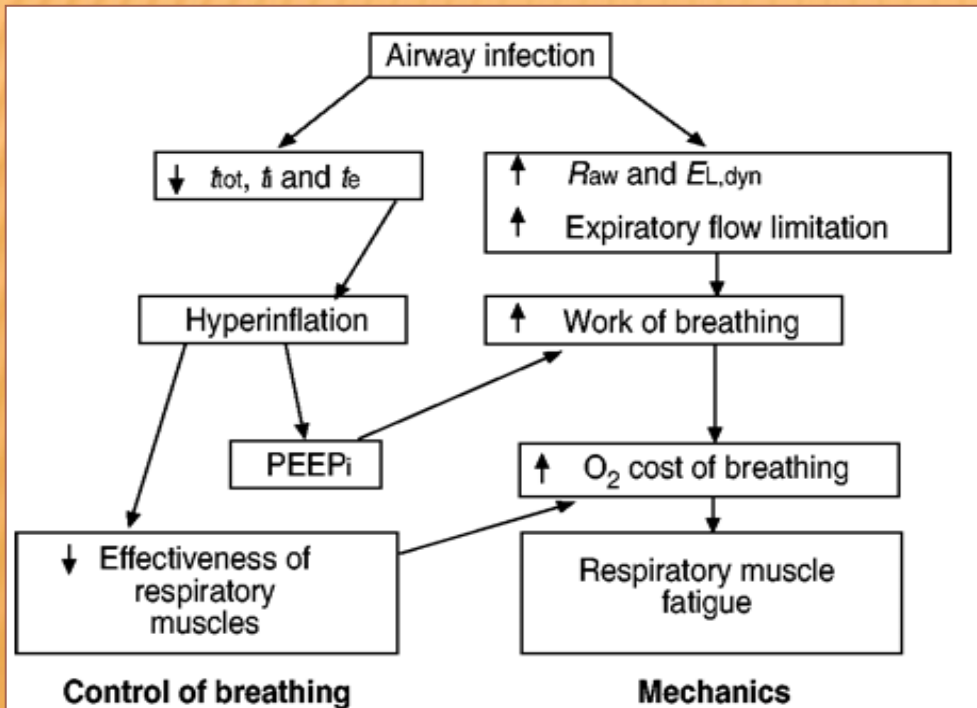
Schematic representation of the sequence of responsible mechanisms that lead to acute-on-chronic respiratory failure in patients with chronic obstructive pulmonary disease.
t_{tot}: *total respiratory cycle;* t_I: *inspiratory time;* t_e: *expiratory time;* R_{aw}: *airway resistance;* $E_{L,dyn}$: *dynamic elastance of the lung;* $PEEP_i$: *intrinsic positive end-expiratory pressure;* ↓

Hypoxemic Respiratory Failure

Is PaCO2 increased?

Yes → Hypoventilation

No → ↑(PAO2 - PaO2)?

↑(PAO2 - PaO2)

Hypovent alone

Hypovent plus another mechanism

↓Respiratory drive
Neuromuscular dz

↑(PAO2 - PaO2)?

Yes → Is low PO2 correctable with O2?

No → ↓Inspired PO2

High altitude
↓FIO2

Is low PO2 correctable with O2?

No → Shunt

Yes → V/Q mismatch

HYPOXEMIC RESPIRATORY FAILURE

V/Q mismatch

Atelectasis
Intraalveolar filling
Pneumonia
Pulmonary edema

ARDS
Interstitial lung dz
Pulmonary contusion

SHUNT
V/Q = 0

DEAD SPACE
V/Q = ∞

Intracardiac shunt
Vascular shunt in lungs

Pulmonary embolus
Pulmonary vascular dz
Airway dz
(COPD, asthma)

Hypercapnic Respiratory Failure

PaCO2 >46mmHg

Not compensation for metabolic alkalosis

(PAO2 - PaO2)

normal → Alveolar Hypoventilation

increased → V/Q abnormality

Alveolar Hypoventilation:
- Central Hypoventilation
- ↓ PI max → Neuromuscular Problem

V/Q abnormality:
- NI VCO2 → V/Q Abnormality
- ↑ VCO2 → Hypermetabolism Overfeeding

Hypercapnic Respiratory Failure

Alveolar Hypoventilation

nl PI max

↓ PI max

Central Hypoventilation

Neuromuscular Disorder
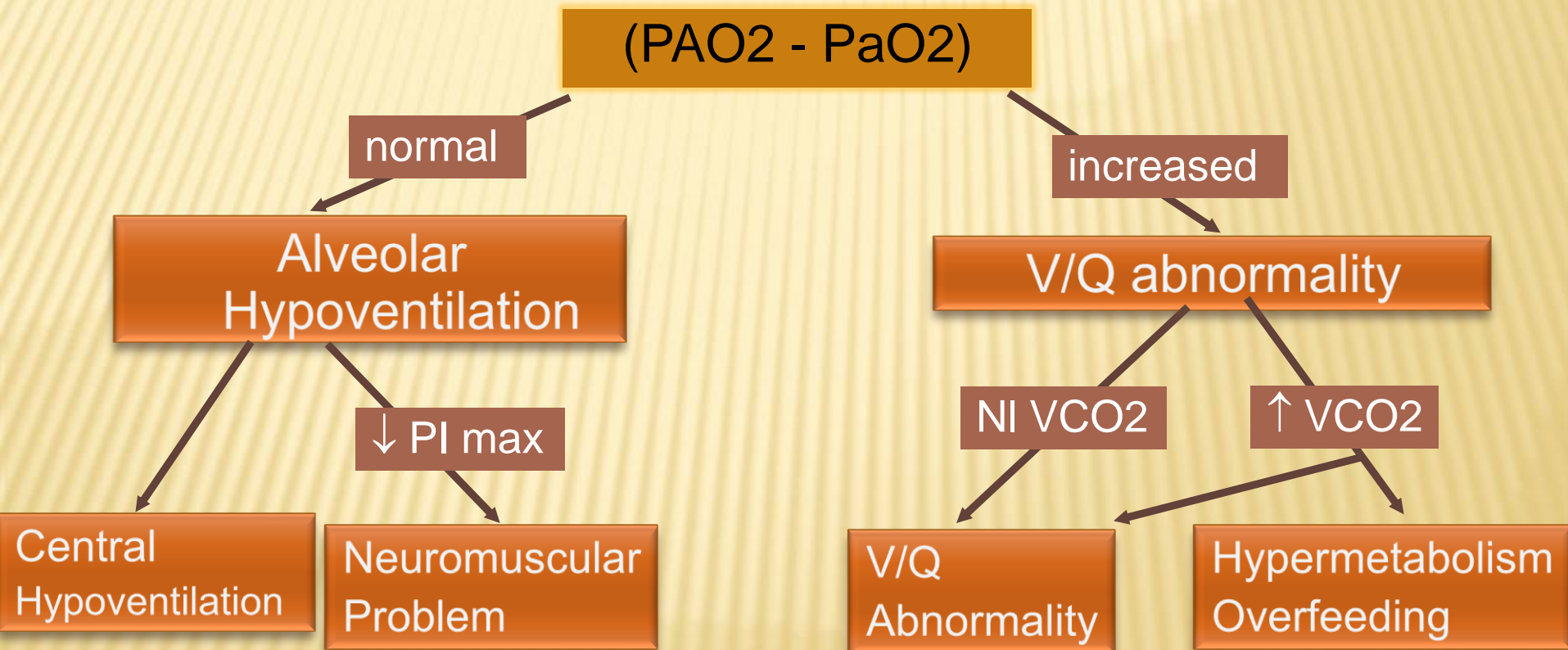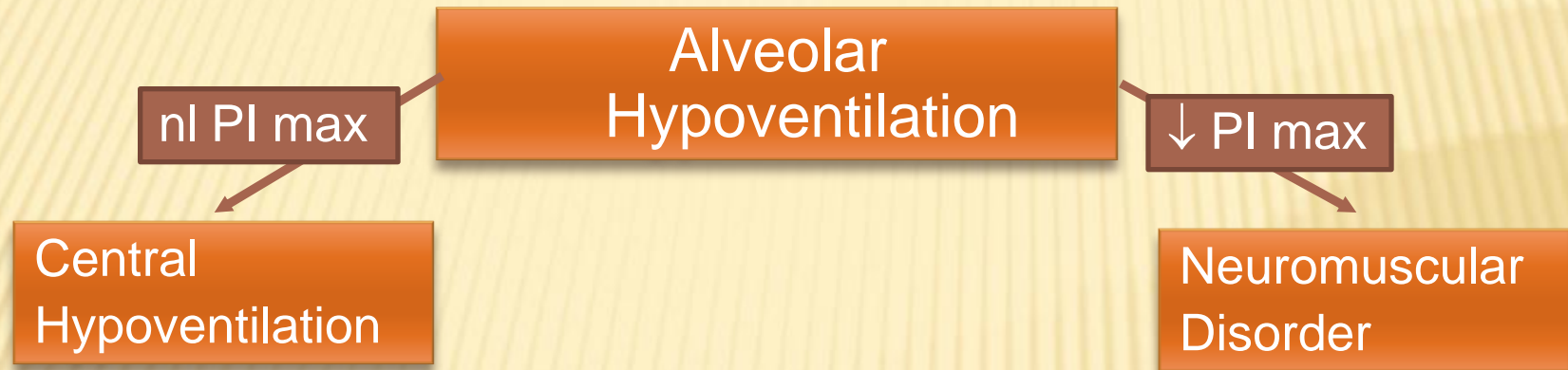
Brainstem respiratory depression
  Drugs (opiates)
  Obesity-hypoventilation syndrome

Critical illness polyneuropathy
Critical illness myopathy

Hypophosphatemia
Magnesium depletion

Myasthenia gravis
Guillain-Barre syndrome

ETIOLOGY

Respiratory failure occurs when ≥ 1 essential components of the respiratory system fail.

1.Airway dysfunction

- Asthma
- Emphysema/chronic bronchitis
- Bronchiolitis
- Endobronchial tumor, mass, or stricture
- Chronic obstructive lung disease

2.Alveolar dysfunction

- Pneumonia
- Pulmonary edema
- Pulmonary hemorrhage
- Adult respiratory distress syndrome
- Drug reaction
- Pulmonary contusion
- Interstitial lung disease

3.Pulmonary vascular dysfunction

- Acute pulmonary embolism
- Pulmonary hypertension
- Arteriovenous malformation or intracardiac shunt

4. Nervous system dysfunction

- Sedative medications
- Toxic overdoses
- Postoperative hypothermia
- Brainstem stroke

5. Musculature dysfunction

- Medications/toxins
  - Paralytics
  - Aminoglycosides
  - Steroids
  - Botulism
- Myopathy
- Myositis
- Metabolic abnormalities
  - Hypothyroidism
  - Hypophosphatemia
- Myasthenia gravis
- Guillain-Barré syndrome
- Paraneoplastic syndromes
- Polyradiculopathy of critical illness
- Postoperative or postradiation phrenic nerve dysfunction
- Postoperative pain/splinting

SYMPTOMS & SIGNS

Respiratory failure is not a disease itself, but the end result of many disorders. Some of the symptoms and signs relate to the underlying disorder.

- Shortness of breath/dyspnea
- Flaring of nostrils
- Pursed-lips breathing
- Use of accessory muscles of respiration
- Supine abdominal paradox (diaphragmatic paralysis)
- Peripheral muscle weakness
- Central and/or peripheral cyanosis
- Increased or decreased respiratory rate
- Altered level of consciousness
    Confusion
    Disorientation
    Coma

- Stridor
  Suggests the presence of large airway or laryngeal obstruction
- Wheezing
- Rhonchi
- Evidence of consolidation on auscultation
  Tubular breath sounds
  Dullness to percussion
  Egophony
- Evidence of right-heart dysfunction
  Elevated jugular venous pressures
  Pronounced or delayed pulmonic component of the second heart sound
  Right-sided heave
  Right-sided third heart sound
  Murmur

MECHANISM

Hypoxemia results in central cyanosis that is best assessed by examining the oral mucous membranes, since blood flow at these sites is well maintained when the periphery may be vasoconstricted.

Cyanosis is more easily observed in polycythaemic patients, whereas in anemic patients there may be insufficient reduced hemoglobin to produce a blue color to the mucus membranes.

Hypoxemia affects the central nervous system (CNS), causing irritability, impaired intellectual function and clouding of consciousness, which may progress to convulsions, coma and death.

A level of acute hypoxemia that might be dangerous to a previously healthy individual may be well tolerated by patients with chronic hypoxia. Hypoxemia stimulates ventilation via the carotid chemoreceptor, increases heart rate and cardiac output and dilates peripheral vessels. Cardiac dysrhythmias may occur, which may be exaggerated by concomitant digitalis or hypokalemia.

The pulmonary arteries respond to hypoxia by vasoconstricting, producing increased vascular resistance and pulmonary hypertension, with the later development of right ventricular enlargement or cor pulmonale. Persistent hypoxia results in secondary polycythaemia due to increased production of erythropoietin.

PATIENTS JOURNEY
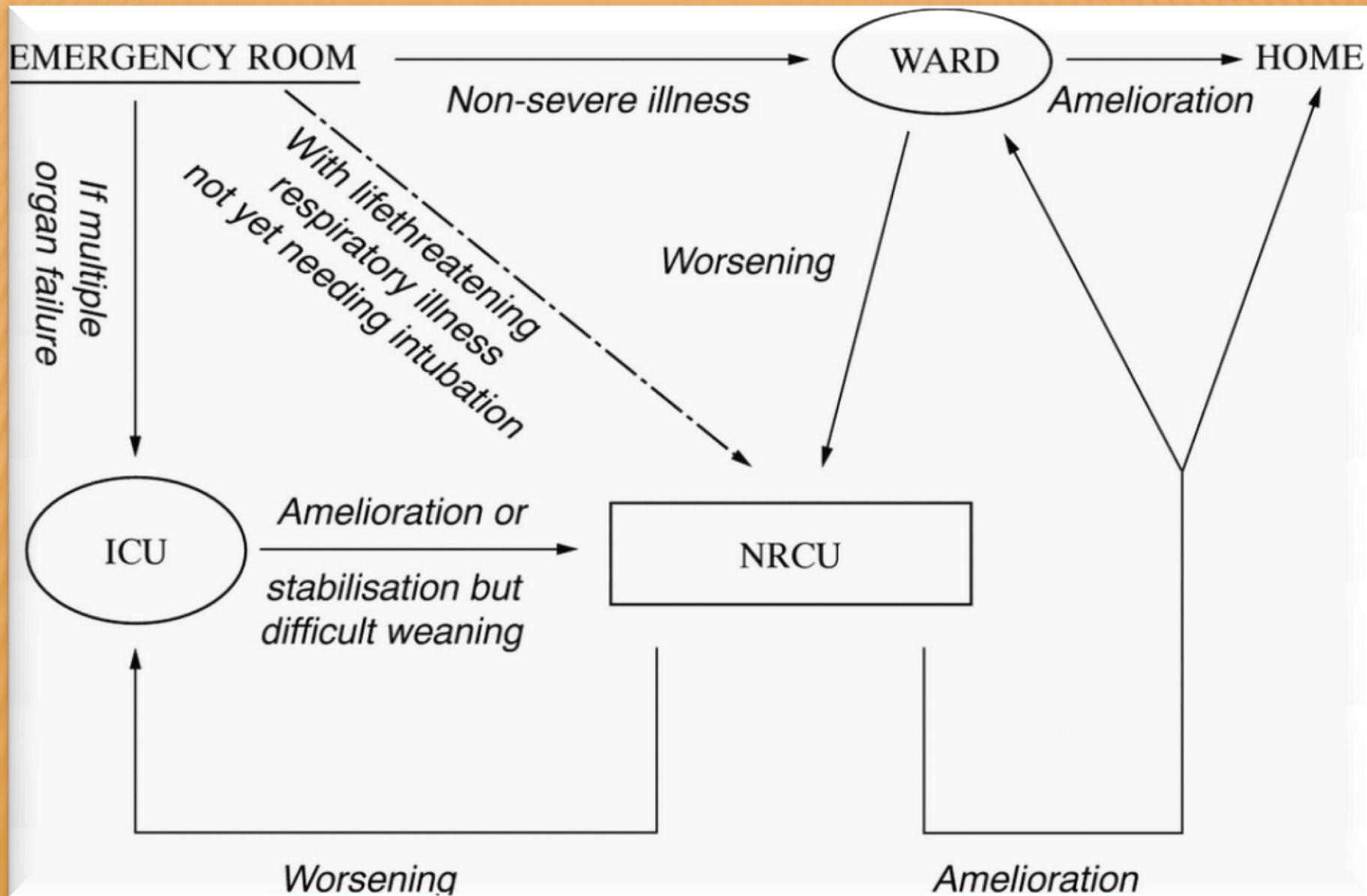
General

❖*Initial evaluation of the respiratory system*
➤Immediate determination of upper-airway patency
➤Examination for central and peripheral cyanosis
➤Measurement of respiratory rate,depth and pattern of respiration
➤Presence or absence of signs of respiratory distress, including:
- •Flaring of nostrils
- •Pursed-lips breathing
- •Use of accessory muscles of respiration
➤Assessment of the configuration of the chest wall and its movement during the respiratory cycle
➤Palpation and auscultation over each hemithorax

❖*Pulse Oximetery & Arterial blood gas measurements for oxygen and carbon dioxide tensions*

❖*Initial stabilization should be implemented before the specific etiology of respiratory failure is diagnosed and treated*.
➤Airway protection, oxygenation, and ventilation, including mechanical ventilation (when indicated)
➤Cardiovascular stability must be rapidly assessed and achieved.
➤After stabilization, a thorough evaluation of the cause of respiratory failure can be safely undertaken.

PHYSIOLOGIC APPROACH

Approaching respiratory failure from a physiologic perspective can provide important clues about specific etiology, leading to a more effective diagnostic approach.

MUSCULAR DYSFUNCTION
- Common cause of respiratory failure in ICU setting
- Usually, multifactorial causes
    Various medications, prolonged periods of mechanical ventilator support, and polyradiculopathy associated with critical illness can all adversely affect the respiratory muscles.
- Suggested by supine abdominal paradox (diaphragmatic paralysis), peripheral muscle weakness, reduced maximal inspiratory pressure generation (inspiratory force)
- Tests: vital capacity, inspiratory force
- Method of determination: bedside measurement in awake patient
- Findings consistent with muscular dysfunction
    Presence of paradoxical respiratory motion
    Vital capacity < 10 mL/kg
    Inspiratory force < –20 cmH$_2$O
    Rapid shallow breathing index: ratio of respiratory rate to tidal volume in liters > 105

Airway dysfunction

- Upper-airway dysfunction is suggested by stridor.
- Lower-airway dysfunction is suggested by wheezing.
- Tests

     Wheezing or rhonchi on auscultation

     Airway resistance measurement in patients on ventilator

          Values exceeding 3–8 cm H_2O/L per second indicate airway obstruction.

     Evidence of intrinsic positive end-expiratory pressure (auto-PEEP)

Alveolar compartment dysfunction

- Signs of pulmonary consolidation on auscultation, with tubular breath sounds and dullnes
- Respiratory system compliance < 30 mL/H_2O measured with patient on ventilator
- Alveolar infiltrates on chest radiography

Pulmonary vascular dysfunction

- Signs of right-heart failure on examination
     - Elevated jugular venous pressure and central venous pressure
     - Right ventricular hypertrophy or right bundle-branch block

Nervous system dysfunction

The most frequent cause is use of medications that impair respiratory drive, many of which also impair the level of consciousness.

Finding consistent with dysfunction: respiratory rate < 12 breaths/min in spontaneously breathing patient in presence of hypoxia or hypercarbia and acidemia

LABORATORY TESTS

Arterial blood gases :

- Arterial blood gas analysis provides information on the following:
  1] Oxygenation of blood through gas exchange in the lungs.
  2] Carbon dioxide (CO_2) elimination through respiration.
  3] Acid-base balance or imbalance in extra-cellular fluid (ECF)

Complete blood count:.

- A complete blood count may indicate anemia, which can contribute to tissue hypoxia, whereas polycythemia may indicate chronic hypoxemic respiratory failure

RFT & LFT :.

- helpful in the evaluation & management of a patient in respiratory failure. Abnormalities in renal and hepatic function may either provide clues to the etiology of respiratory failure or alert the clinician to complications associated with it.Abnormalities in electrolytes such as potassium, magnesium, and phosphate may aggravate respiratory failure

CK: Measuring serum creatine kinase with fractionation and troponin I helps exclude recent myocardial infarction in a patient with respiratory failure. An elevated creatine kinase with a normal troponin I may indicate myositis, which occasionally can cause respiratory failure.

OTHERS :

In chronic hypercapnic respiratory failure, serum thyroid-stimulating hormone should be measured to evaluate the possibility of hypothyroidism, a potentially reversible cause of respiratory failure.

CHEST RADIOGRAPHY

Chest radiography is essential because it frequently reveals the cause of respiratory failure. However, distinguishing between cardiogenic and noncardiogenic pulmonary edema often is difficult.

Increased heart size, vascular redistribution, peribronchial cuffing, pleural effusions, septal lines, and perihilar bat-wing distribution of infiltrates suggest hydrostatic edema; the lack of these findings suggests acute respiratory distress syndrome (ARDS).

ECG

ECG should be performed to evaluate the possibility of a cardiovascular cause of respiratory failure; it also may detect dysrhythmias resulting from severe hypoxemia and/or acidosis.

ECHOCARDIOGRAPHY

Echocardiography need not be performed routinely in all patients with respiratory failure. However, it is a useful test when a cardiac cause of acute respiratory failure is suspected.

•The findings of left ventricular dilatation, regional or global wall motion abnormalities, or severe mitral regurgitation support the diagnosis of cardiogenic pulmonary edema.

•A normal heart size and normal systolic and diastolic function in a patient with pulmonary edema would suggest acute respiratory distress syndrome (ARDS).

•Echocardiography provides an estimate of right ventricular function and pulmonary artery pressure in patients with chronic hypercapnic respiratory failure.

PULMONARY FUNCTION TESTS

Patients with acute respiratory failure generally are unable to perform pulmonary function tests (PFTs).

However, PFTs are useful in the evaluation of chronic respiratory failure.

Normal values of forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) suggest a disturbance in respiratory control.

A decrease in FEV_1 -to-FVC ratio indicates airflow obstruction, whereas a reduction in both the FEV_1 and FVC and maintenance of the FEV_1 -to-FVC ratio suggest restrictive lung disease. Respiratory failure is uncommon in obstructive diseases when the FEV_1 is greater than 1 L and in restrictive diseases when the FVC is more than 1 L.

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DIAGNOSTIC PROCEDURES

If respiratory failure is suspected to be secondary to muscular dysfunction, consider:	• Transdiaphragmatic pressure measurements by using esophageal balloons • Electromyography • Nerve conduction velocity studies • Tensilon test for myasthenia gravis
If respiratory failure is suspected to be secondary to airway dysfunction, consider:	• Pulmonary function testing • Functional response to bronchodilators
If respiratory failure is suspected to be secondary to alveolar dysfunction, consider:	• Bronchoscopy • Lung biopsy
If respiratory failure is suspected to be secondary to pulmonary vascular disease, consider:	• Electrocardiography • Echocardiography • Right-heart catheterization: Measurement of pulmonary capillary wedge pressure may be helpful in distinguishing cardiogenic from noncardiogenic edema.

ESTABLISH PATENCY, OXYGENATION, AND VENTILATION

AIRWAY PATENCY

Obstruction of the upper airway is a medical emergency. In an unconscious patient, occlusion often caused by the tongue or soft tissues of the pharynx.

1 Assess patient for spontaneous respiration.

2 Use head tilt–chin lift maneuver to open airway.

3 When a spontaneously breathing patient is unable to dislodge a foreign object, a forceful subdiaphragmatic thrust (Heimlich maneuver) can facilitate removal.

4 Removal may require laryngoscopy and removal with forceps.

5 Liquids, such as vomitus or blood, are removed by suctioning under direct vision.

6 In many cases, an endotracheal tube is required to establish a stable airway.

7 When the airway cannot be secured otherwise, a tracheostomy or cricothyrotomy should be performed.
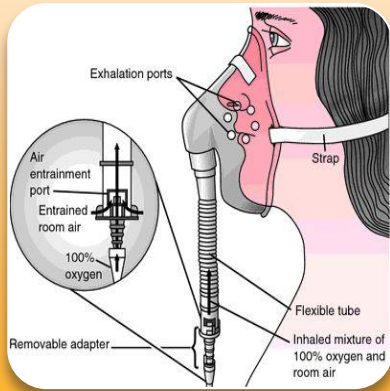
Oxygen

- Aids in correcting hypoxemia
- Can sometimes be provided through mask ventilation, but mechanical ventilation is more often required

Mechanical ventilation
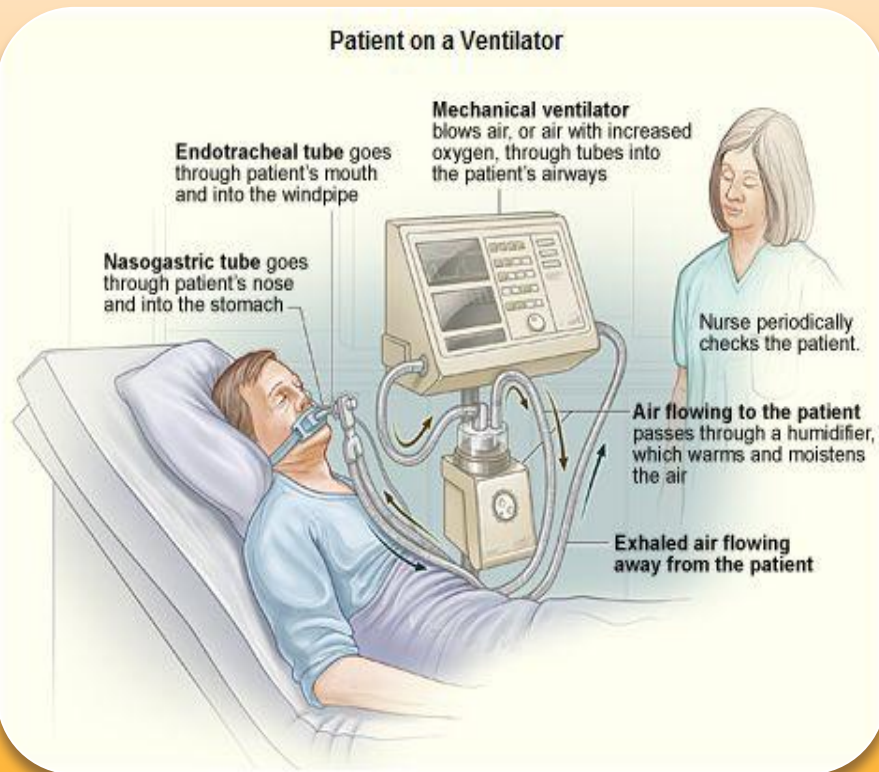
✓Mainstay of supportive care for respiratory failure .

✓Aids in correcting hypercarbia, acidosis, and hypoxemia.

✓Usually requires placement of an endotracheal tube.Either the orotracheal or nasotracheal route, usually using a combination of parenteral and local anesthesia to ensure patient comfortAfter placement, the tube must be secured and its position verified.

✓After oxygenation and elimination of carbon dioxide have been documented, oxygen content and other ventilator settings can be adjusted.Oxygen delivery to the alveoli can be improved by applying PEEP in patients receiving mechanical ventilatory support.

NIPPV

In selected patients with hypercapnic respiratory failure due to an acute exacerbation of chronic obstructive pulmonary disease (COPD), noninvasive positive pressure ventilation, added to usual medical therapy, reduces the need for endotracheal intubation, the length of hospital stay, and the risk of death.

The patient wears a tight-fitting nasal or full facial mask, which is connected to a continuous positive airway pressure unit, a bi-level airway pressure unit, or a standard ventilator.
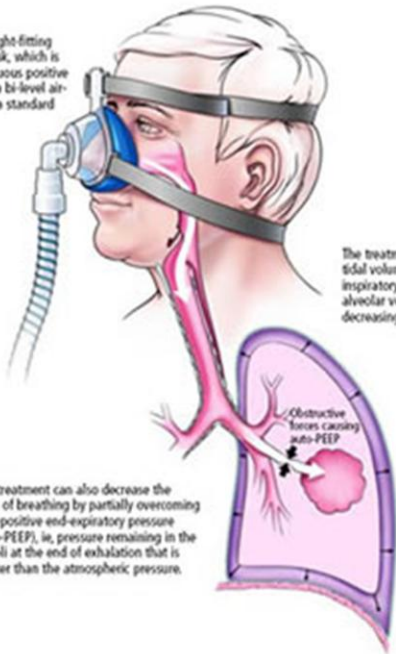
The treatment provides a larger tidal volume with the same inspiratory effort, thus improving alveolar ventilation and decreasing the work of breathing.

This treatment can also decrease the work of breathing by partially overcoming auto-positive end-expiratory pressure (auto-PEEP), ie, pressure remaining in the alveoli at the end of exhalation that is greater than the atmospheric pressure.

Obstructive forces causing auto-PEEP

Medical Illustrator: Joseph Pangrace

Non-invasive mechanical ventilation has been increasingly used to avoid or serve as an alternative to intubation. Compared with medical therapy, and in some instances with invasive mechanical ventilation, it improves survival and reduces complications in selected patients with <u>acute respiratory failure</u>.
The main indications are *<u>exacerbation of chronic obstructive pulmonary disease, cardiogenic pulmonary oedema, pulmonary infiltrates in immunocompromised patients, and weaning of previously intubated stable patients with chronic obstructive pulmonary disease</u>*

This technique can be used in postoperative patients or those with neurological diseases, to palliate symptoms in terminally ill patients, or to help with bronchoscopy; however further studies are needed in these situations before it can be regarded as first-line treatment.
 Non-invasive ventilation implemented as an alternative to intubation should be provided in an intensive care or high-dependency unit. When used to prevent intubation in otherwise stable patients it can be safely administered in an adequately staffed and monitored ward

AFTER STABILIZATION

When patient has been stabilized, treatment of underlying disorder can begin.

Examples
- Remove excess secretion by suctioning
- Treat infections with effective antimicrobials.
- Suppress inflammation with anti-inflammatory drugs.
- Treat obstruction with bronchodilators.
- Avoid harmful effects of excess oxygen or mechanical forces from mechanical ventilator.
- Prevent recurrent pulmonary emboli with anticoagulants.
- Remove transudated fluid with diuretics.
- Some forms of chronic respiratory system failure, such as sleep apnea syndrome and post-polio syndrome, are ultimately responsive to nocturnal mechanical ventilation or continuous positive airway pressure (CPAP).
- Selected patients with isolated severe chronic respiratory failure may have improved quality of life with lung transplantation.

The pharmacotherapy of cardiogenic pulmonary edema and acute exacerbations of COPD is discussed here. The goals of therapy in cardiogenic pulmonary edema are to achieve a pulmonary capillary wedge pressure of 15-18 mm Hg and a cardiac index greater than 2.2 L/min/m^2, while maintaining adequate blood pressure and organ perfusion. These goals may need to be modified for some patients. Diuretics, nitrates, analgesics, and inotropics are used in the treatment of acute pulmonary edema.

DIURETICS

First-line therapy generally includes a loop diuretic such as furosemide, which inhibits sodium chloride reabsorption in the ascending loop of Henle.

FUROSEMIDE :Administer loop diuretics IV because this allows for both superior potency and a higher peak concentration

METOLAZONE

Has been used as adjunctive therapy in patients initially refractory to furosemide. Has been demonstrated to be synergistic with loop diuretics in treating refractory patients and causes a greater loss of potassium. Potent loop diuretic that sometimes is used in combination with Lasix for more aggressive diuresis. Also used in patients with a degree of renal dysfunction for initiating diuresis.

NITRATES

These agents reduce myocardial oxygen demand by lowering preload and afterload. In severely hypertensive patients, nitroprusside causes more arterial dilatation than nitroglycerin. Nevertheless, due to the possibility of thiocyanate toxicity and the coronary steal phenomenon associated with nitroprusside, IV nitroglycerin may be the initial therapy of choice for afterload reduction.

ANALGESICS

Morphine IV is an excellent adjunct in the management of acute pulmonary edema. In addition to being both an anxiolytic and an analgesic, its most important effect is venodilation, which reduces preload. Also causes arterial dilatation, which reduces systemic vascular resistance and may increase cardiac output.

CORTICOSTEROIDS

Have been shown to be effective in accelerating recovery from acute COPD exacerbations and are an important anti-inflammatory therapy in asthma. Although they may not make a clinical difference in the ED, they have some effect 6-8 h into therapy; therefore, early dosing is critical.

Methylprednisolone

Usually given IV in ED for initiation of corticosteroid therapy, although PO should theoretically be equally efficacious

INOTROPIC DRUGS

Principal inotropic agents include dopamine, dobutamine, inamrinone (formerly amrinone), milrinone, dopexamine, and digoxin. In patients with hypotension presenting with CHF, dopamine and dobutamine usually are employed. Inamrinone and milrinone inhibit phosphodiesterase, resulting in an increase of intracellular cyclic AMP and alteration in calcium transport. As a result, they increase cardiac contractility and reduce vascular tone by vasodilatation.

Dopamine

Stimulates both adrenergic and dopaminergic receptors. Hemodynamic effects depend on the dose. Lower doses stimulate mainly dopaminergic receptors that produce renal and mesenteric vasodilation. Cardiac stimulation and renal vasodilation are produced by higher doses. Positive inotropic agent at 2-10 mcg/kg/min that can lead to tachycardia, ischemia, and dysrhythmias. Doses >10 mcg/kg/min cause vasoconstriction, which increases afterload.

Norepinephrine

Used in protracted hypotension following adequate fluid replacement. Stimulates beta1- and alpha-adrenergic receptors, which in turn increases cardiac muscle contractility and heart rate, as well as vasoconstriction. As a result, increases systemic blood pressure and cardiac output. Adjust and maintain infusion to stabilize blood pressure (eg, 80-100 mm Hg systolic) sufficiently to perfuse vital organs.

Dobutamine

Produces vasodilation and increases inotropic state. At higher dosages, may cause increased heart rate, thus exacerbating myocardial ischemia. _Strong inotropic agent with minimal chronotropic effect and no vasoconstriction._

BRONCHODILATORS

These agents are an important component of treatment in respiratory failure caused by obstructive lung disease. These agents act to decrease muscle tone in both small and large airways in the lungs. This category includes beta-adrenergics, methylxanthines, and anticholinergics.

Terbutaline

Acts directly on beta2-receptors to relax bronchial smooth muscle, relieving bronchospasm and reducing airway resistance.

Albuterol

Beta-agonist useful in the treatment of bronchospasm. Selectively stimulate beta2-adrenergic receptors of the lungs. Bronchodilation results from relaxation of bronchial smooth muscle, which relieves bronchospasm and reduces airway resistance.

Theophylline

Has a number of physiological effects, including increases in collateral ventilation, respiratory muscle function, mucociliary clearance, and central respiratory drive. Partially acts by inhibiting phosphodiesterase, elevating cellular cyclic AMP levels, or antagonizing adenosine receptors in the bronchi, resulting in relaxation of smooth muscle. However, clinical efficacy is controversial, especially in the acute settING

Ipratropium bromide

Anticholinergic medication that appears to inhibit vagally mediated reflexes by antagonizing action of acetylcholine, specifically with the muscarinic receptor on bronchial smooth muscle. Vagal tone can be significantly increased in COPD; therefore, this can have a profound effect. Dose can be combined with a beta-agonist because ipratropium may require 20 min to begin having an effect.
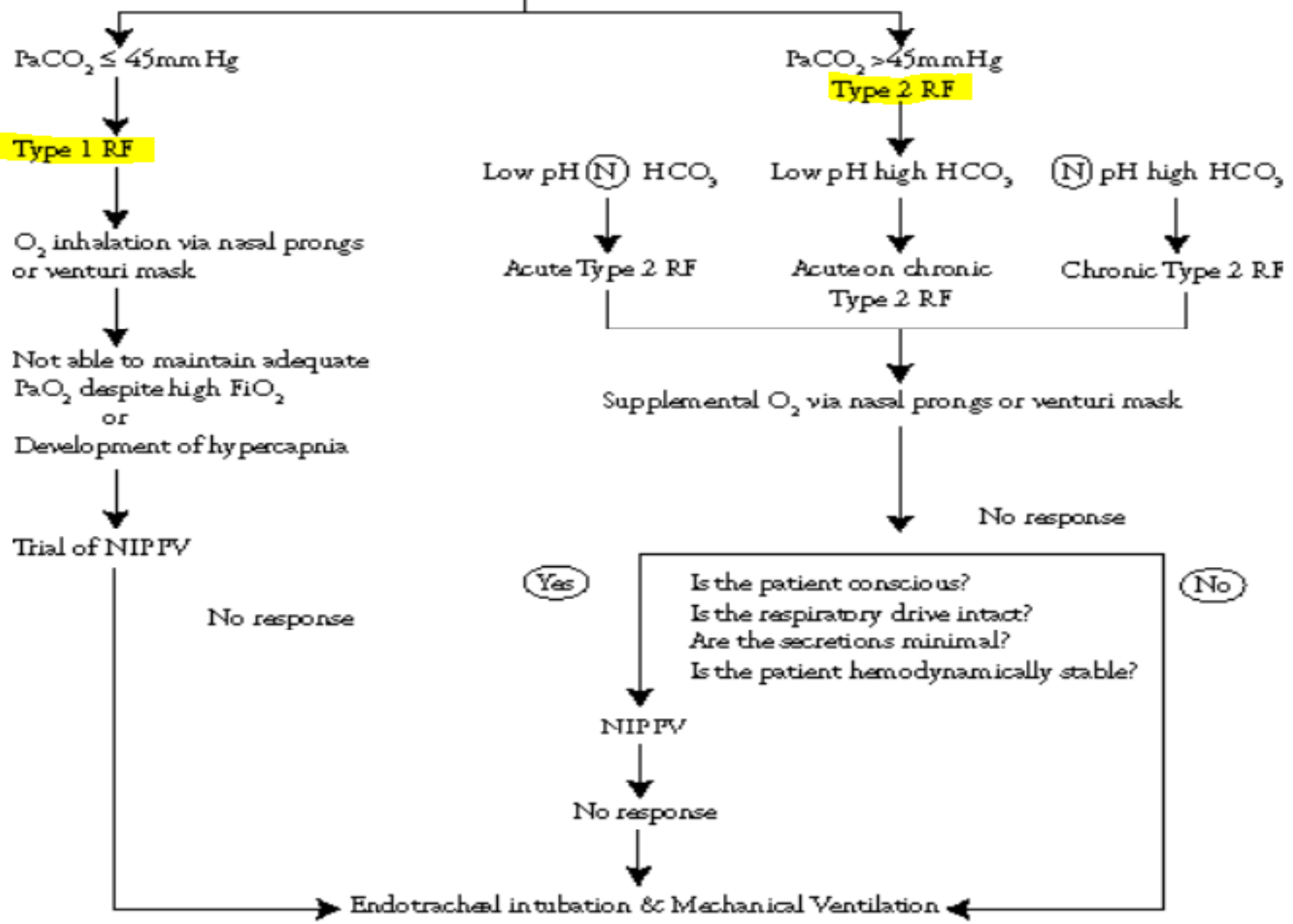
TYPE 1 RESPIRATORY FAILURE MANAGEMENT

This condition usually presents little difficulty and apart from the use of oxygen, treatment of the primary cause (e.g. antibiotics for lobar pneumonia), may be all that it required.
Arterial hypoxemia when extremely severe can be life threatening and therefore should have the highest priority when managing acute respiratory failure.

 The goal should be to _increase hemoglobin O2 saturation to at least 85-90%_ without risking significant oxygen toxicity. Very high FiO2 levels can be safely used for brief periods of time.

The use of positive end-expiratory pressure (PEEP), changes in position, sedation and paralysis may be helpful in lowering FiO2. Fever, agitation, overfeeding, vigorous respiratory activity and sepsis can all markedly increase VO2.

Prolonged exposure to high concentration of oxygen (FiO2>50%) should be avoided because pulmonary toxicity depends on both the duration of treatment and FiO2.

Failure of high FiO2, to improve PaO2 implies a significant intrapulmonary shunt, as occurs in ARDS.
ARDS is the most severe form of acute lung injury and is characterized by bilateral, widespread radiographic pulmonary infiltrate, normal pulmonary capillary wedge pressure (≤18 mm Hg) and a PaO2/FIO2 ratio ≤ 200 regardless of level of positive end-expiratory pressure (PEEP). Acute lung injury (ALI) is a mild form of ARDS, and differs from ARDS based on less severe hypoxemia (PaO2/FIO2 ratio ≤ 300).The mainstay of supportive care of ALI/ARDS is mechanical ventilation.

TYPE 2 RESPIRATORY FAILURE

By far the commonest cause of type 2 or hypercapnic respiratory failure is an exacerbation of COPD.

The development of arterial hypoxaemia occurs insidiously in most patients with COPD, although in some the fall in PaO2 can be rapid. Hypoxaemia that develops slowly may produce little effects and chronic hypercapnia can be tolerated for many years with few symptoms, although early morning headache is relatively common.Once acute respiratory failure is suspected the diagnosis must be confirmed by arterial blood gas analysis. The pH (hydrogen ion concentration) is helpful in assessing the degree of acute vs. chronic respiratory failure.

The general principles of management are :

(i) to correct life threatening hypoxaemia;

(ii) to correct life threatening acidosis;

(iii) to treat the underlying cause; and

(iv) to prevent complications.

✓Non-invasive positive pressure ventilation (NIPPV) is increasingly being used in the care of patients suffering from acute respiratory failure. NIPPV is a novel method of giving positive pressure ventilation without endotracheal intubationThe distinct advantage of NIPPV is avoiding endotracheal intubation and its associated complications

✓Mechanical ventilation

✓Approximately half of patients suffering from hypercapnic Respiratory failure (COPD exacerbations) respond favourably to medical therapy, half of those within first 24 hrs and 92% within 72 hrs.

TYPE III OR PERIOPERATIVE RESPIRATORY FAILURE

❖Atelectasis can be treated by:
- •Frequent changes in position
- •Chest physiotherapy
- •Upright positioning
- •Aggressive control of incisional and/or abdominal pain
- •Noninvasive positive-pressure ventilation may also be used to reverse regional atelectasis.

TYPE IV RESPIRATORY FAILURE

❖Intubation and mechanical ventilation can allow redistribution of the cardiac output away from the respiratory muscles and back to vital organs while the shock is treated.
So its mainly depends on treatment of Shock..

COMPLICATIONS

Pulmonary	Cardiovascular	Gastrointestinal
• Pulmonary embolism • Barotrauma, • Pulmonary fibrosis • Complications secondary to the use of mechanical devices. • Prone to develop nosocomial pneumonia. • Pulmonary fibrosis may follow acute lung injury associated with acute respiratory distress syndrome (ARDS). High oxygen concentrations and the use of large tidal volumes may worsen acute lung injury	• Hypotension • Reduced cardiac output • Arrhythmia • Pericarditis • Acute myocardial infarction. These complications may be related to the underlying disease process, mechanical ventilation, or the use of pulmonary artery catheters	• Hemorrhage • Gastric distention, paralytic ileus, • Diarrhea • pneumoperitoneum • Stress ulceration is common in patients with acute respiratory failure; the incidence can be reduced by routine use of antisecretory agents or mucosal protectants

Infectious	Renal	Nutritional
• Nosocomial infections, such as pneumonia, urinary tract infections, and catheter-related sepsis, are frequent complications of acute respiratory failure. These usually occur with the use of mechanical devices. The incidence of nosocomial pneumonia is high and associated with significant mortality.	• Acute renal failure and abnormalities of electrolytes and acid-base homeostasis are common in critically ill patients with respiratory failure The development of acute renal failure in a patient with acute respiratory failure carries a poor prognosis and high mortality. The most common mechanisms of renal failure in this setting are renal hypoperfusion and the use of nephrotoxic drugs (including radiographic contrast material).	• These include malnutrition and its effects on respiratory performance and complications related to administration of enteral or parenteral nutrition Complications associated with nasogastric tubes, such as abdominal distention and diarrhea, also may occur. Complications of parenteral nutrition may be mechanical due to catheter insertion, infectious, or metabolic (eg, hypoglycemia, electrolyte imbalance).

PROGNOSIS

Respiratory failure is associated with poor prognosis.

> In the U.S., about one-third of patients with respiratory failure requiring ICU care and mechanical ventilation die during hospitalization.

> Mortality from respiratory failure increases with age and in the presence of other comorbid conditions.

Advances in mechanical ventilation and airway management have improved the prognosis for patients with respiratory failure.

> Even patients with irreversible chronic respiratory failure can now be provided with ventilator support systems that allow acceptable quality of life and management at home.

The mortality rate for acute respiratory distress syndrome (ARDS) is approximately 40%. Younger patients (< 60 y) have better survival rates than older patients. Approximately two thirds of patients who survive an episode of acute respiratory distress syndrome (ARDS) manifest some impairment of pulmonary function 1 or more years after recovery.

Significant mortality also occurs in patients admitted with hypercapnic respiratory failure. This is because these patients have a chronic respiratory disorder and other comorbidities such as cardiopulmonary, renal, hepatic, or neurologic disease. These patients also may have poor nutritional status.
 For patients with COPD and acute respiratory failure, the overall mortality rate has declined from approximately 26% to 10%.

CONCLUSIONS

First principle in managing patients at risk for respiratory failure is to prevent progression of the underlying disease.

Pneumonia and asthma, for example, have specific therapies that include antibiotics and bronchodilators that should be instituted promptly.Simultaneously, the respiratory failure must be addressed.

If it is acute and severe, it is a **medical emergency**. Oxygen levels must be normalized as quickly as possible by providing supplemental oxygen. Patients with high carbon dioxide levels need ventilatory support, so they receive pressurized gas from devices (ventilators) that increase pressure when triggered by the patient's inspiratory effort or by a timer.

The pressurized gas can be delivered via a plastic tube inserted into the trachea (**invasive ventilation**) or via a mask strapped over the nose and mouth or just the nose (**noninvasive ventilation**).

Not all respiratory failure has a dire outlook. Patients with acute respiratory failure can be ventilated until the acute disease is successfully treated. They may then return to a normal life.

Nevertheless, respiratory failure is serious and has potential life-threatening consequences but with proper management & effort there is hope for better prognosis................

"Inhale. Exhale. Repeat. The simple act of breathing is surely the most natural, primal, and universal human experience..........."

Thank You