ACUTE RESPIRATORY FAILURE

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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.
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.
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.
Acute hypercapnic respiratory failure is defined as a Paco2 greater than 45 mmHg with accompanying acidemia (pH less than 7.30). The physiological effect of a sudden increment in Paco2 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 Paco2 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.](image)

t$_{tot}$: total respiratory cycle; $t_i$: inspiratory time; $t_e$: expiratory time; $R_{aw}$: airway resistance; $E_{L,dyr}$: dynamic elastance of the lung; $PEEP_i$: intrinsic positive end-expiratory pressure; ↓
Is PaCO2 increased?

Yes

Hypoventilation

↑(PAO2 - PaO2)

Hypovent alone

Hypovent plus another mechanism

No

↑(PAO2 - PaO2)?

Yes

Is low PO2 correctable with O2?

Yes

↓Inspired PO2

High altitude

↓FIO2

No

Shunt

V/Q mismatch

↓Respiratory drive

Neuromuscular dz
V/Q mismatch

**SHUNT**

V/Q = 0

- Atelectasis
- Intraalveolar filling
- Pneumonia
- Pulmonary edema

- Intracardiac shunt
- Vascular shunt in lungs

**DEAD SPACE**

V/Q = \(\infty\)

- ARDS
- Interstitial lung dz
- Pulmonary contusion

- Pulmonary embolus
- Pulmonary vascular dz
- Airway dz
  (COPD, asthma)
Hypercapnic Respiratory Failure

\[ \text{PaCO}_2 > 46 \text{mmHg} \]

Not compensation for metabolic alkalosis

\( (\text{PAO}_2 - \text{PaO}_2) \)

Alveolar Hypoventilation

- Normal
- \( \downarrow \) PI max

Central Hypoventilation

Neuromuscular Problem

V/Q Abnormality

- NI VCO2
- \( \uparrow \) VCO2

V/Q Abnormality

Hypermetabolism Overfeeding
Alveolar Hypoventilation

Central Hypoventilation

Brainstem respiratory depression
Drugs (opiates)
Obesity-hypoventilation syndrome

↓ PI max

Neuromuscular Disorder

Critical illness polyneuropathy
Critical illness myopathy

Hypophosphatemia
Magnesium depletion

Myasthenia gravis
Guillain-Barre syndrome
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
<table>
<thead>
<tr>
<th>4. Nervous system dysfunction</th>
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<tbody>
<tr>
<td>• Sedative medications</td>
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<tr>
<td>• Toxic overdoses</td>
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<tr>
<td>• Postoperative hypothermia</td>
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<tr>
<td>• Brainstem stroke</td>
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<tr>
<th>5. Musculature dysfunction</th>
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<tr>
<td>• Medications/toxins</td>
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<tr>
<td>• Paralytics</td>
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<tr>
<td>• Aminoglycosides</td>
</tr>
<tr>
<td>• Steroids</td>
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<tr>
<td>• Botulism</td>
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<tr>
<td>• Myopathy</td>
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<tr>
<td>• Myositis</td>
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<tr>
<td>• Metabolic abnormalities</td>
</tr>
<tr>
<td>• Hypothyroidism</td>
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<tr>
<td>• Hypophosphatemia</td>
</tr>
<tr>
<td>• Myasthenia gravis</td>
</tr>
<tr>
<td>• Guillain-Barré syndrome</td>
</tr>
<tr>
<td>• Paraneoplastic syndromes</td>
</tr>
<tr>
<td>• Polyradiculopathy of critical illness</td>
</tr>
<tr>
<td>• Postoperative or postradiation phrenic nerve dysfunction</td>
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<td>• Postoperative pain/splinting</td>
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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.
**Diagnostic Approach**

**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 Oximetry & 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 cmH₂O
  **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 H₂O/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 dullness
• Respiratory system compliance < 30 mL/H₂O 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 (CO2) 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.
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.
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.

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<td>Assess patient for spontaneous respiration.</td>
<td>When a spontaneously breathing patient is unable to dislodge a foreign object, a forceful subdiaphragmatic thrust (Heimlich maneuver) can facilitate removal.</td>
<td>Liquids, such as vomitus or blood, are removed by suctioning under direct vision.</td>
<td>In many cases, an endotracheal tube is required to establish a stable airway.</td>
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<td>Use head tilt–chin lift maneuver to open airway.</td>
<td>Removal may require laryngoscopy and removal with forceps.</td>
<td>When the airway cannot be secured otherwise, a tracheostomy or cricothyroto my should be performed.</td>
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**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 comfort. After 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.
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 acute respiratory failure. The main indications are 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. 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.
ABG
PaO₂ < 60 mm Hg

PaCO₂ ≤ 45 mm Hg

Type 1 RF

O₂ inhalation via nasal prongs or venturi mask

Not able to maintain adequate PaO₂ despite high FiO₂ or development of hypercapnia

Trial of NIPPV

No response

Yes

Is the patient conscious?
Is the respiratory drive intact?
Are the secretions minimal?
Is the patient hemodynamically stable?

NIPPV

No response

Endotracheal intubation & Mechanical Ventilation

No

PaCO₂ > 45 mm Hg

Type 2 RF

Low pH (N) HCO₃

Acute Type 2 RF

Supplemental O₂ via nasal prongs or venturi mask

No response

(N) pH high HCO₃

Acute on chronic Type 2 RF

Chronic Type 2 RF
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 ($\leq$18 mm Hg) and a PaO2/FIO2 ratio $\leq$ 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 $\leq$ 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 intubation. The 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..
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<thead>
<tr>
<th>Pulmonary</th>
<th>Cardiovascular</th>
<th>Gastrointestinal</th>
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<tbody>
<tr>
<td>• Pulmonary embolism</td>
<td>• Hypotension</td>
<td>• Hemorrhage</td>
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<td>• Barotrauma,</td>
<td>• Reduced cardiac output</td>
<td>• Gastric distention, paralytic ileus,</td>
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<tr>
<td>• Pulmonary fibrosis</td>
<td>• Arrhythmia</td>
<td>• Diarrhea</td>
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<td>• Complications secondary to the use of</td>
<td>• Pericarditis</td>
<td>• pneumoperitoneum</td>
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<tr>
<td>mechanical devices.</td>
<td>• Acute myocardial infarction</td>
<td>• Stress ulceration is common in patients with acute respiratory</td>
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<tr>
<td>• Prone to develop nosocomial pneumonia.</td>
<td>These complications may be related to the underlying disease process,</td>
<td>failure; the incidence can be reduced by routine use of antisecretory</td>
</tr>
<tr>
<td>• Pulmonary fibrosis may follow acute lung</td>
<td>mechanical ventilation, or the use of pulmonary artery catheters</td>
<td>agents or mucosal protectants</td>
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<td>injury associated with acute respiratory</td>
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<td>distress syndrome (ARDS). High oxygen</td>
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<td>concentrations and the use of large tidal</td>
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<td>volumes may worsen acute lung injury</td>
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<td>Infectious</td>
<td>Renal</td>
<td>Nutritional</td>
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<tr>
<td>• 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.</td>
<td>• 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).</td>
<td>• 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).</td>
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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%.
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