

Respiratory failure – Dr. Kamaran

Respiratory failure refers to a condition in which pulmonary gas exchange fails to maintain normal arterial oxygen and carbon dioxide.

Respiratory failure is a condition in which the respiratory system fails in one or both of its gas-exchanging functions- i.e., oxygenation of, and carbon dioxide elimination from, mixed venous (pulmonary arterial) blood.

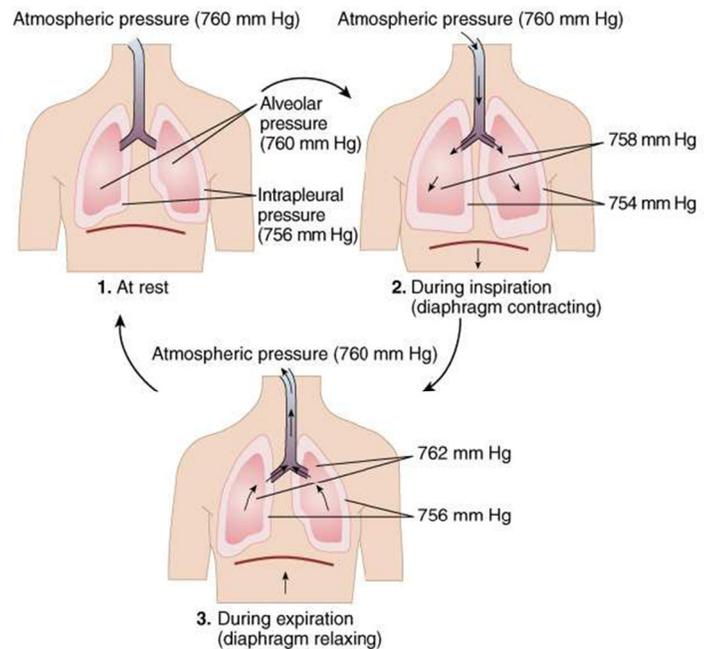
Ventilation: The act of Breathing in (inhaling) which brings in oxygen from the atmosphere into the lungs and the act of Breathing out (exhaling).

Respiration: Is the process of gas exchange between the environment and the tissue level of the human body.

Respiratory failure is a syndrome of inadequate gas exchange due to dysfunction of one or more essential components of the respiratory system: CNS or Brain Stem, Nerves, Chest wall, Airways, Alveolar– capillary units, pulmonary circulation and pleura.

Respiratory muscles:

- Inspiratory Ms expand the rib cage and drive airflow into the lungs.
- Expiratory Ms depress the rib cage and force air out of the lungs.



Inspiratory Muscles:

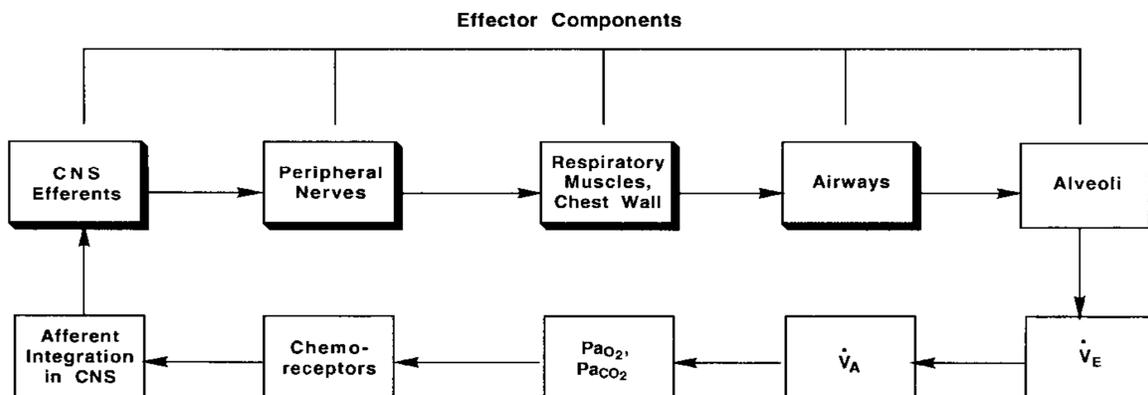
- The sternocleidomastoid elevates the sternum
- The scalenes elevate the top two ribs
- The external intercostals move the ribs upward and outward, expanding the rib cage.
- The diaphragm is the primary inspiratory muscle

Expiratory Muscles

- Internal intercostal muscles pull the ribs downward and inward, reduce the diameter of the rib cage.
- Abdominal muscles depress the lower ribs, and elevate the diaphragm up and into the thorax.

Inspiration (expanding the diameter of the thorax and force air into the lungs): pressure in the lungs becomes lower than the pressure in the atmosphere.

Expiration (reduces the diameter of the thorax, and force air out of the lungs), pressure in the lungs is higher than the pressure in the atmosphere



Respiratory failure can arise from an abnormality in any of the “effector” components of the respiratory system.

CNS: Resp. Center drive depression

- Drugs
- Metabolic encephalopathy
- CNS infections
- Increased Intra Cerebral Pressure
- Obstructive sleep Apnea
- Central alveolar hypoventilation

Spinal cord:

- Trauma
- Transverse myelitis

NeuroMuscular Diseases

- Post-Polio syndrome
- Tetanus
- Multiple Sclerosis
- M.Gravis
- Guillain-Barre
- Critical care or steroid myopathy

Chest wall:

- Kyphoscoliosis
- Obesity

Upper airways:

- Obstruction from tissue enlargement
- Infection
- Mass
- Vocal cord paralysis
- Tracheomalacia

Lower airways:

- Bronchospasm
- Infection)

Lung parenchyma, alveoli

- Infection
- Interstitial lung diseases

Cardiovascular system

A variety of pharmacologic, structural, and metabolic disorders of the central nervous system (CNS) are characterized by suppression of the neural drive to breathe in the medulla oblongata. The resultant hypoventilation and hypercapnia may be acute or chronic.

An overdose of narcotic or sedative properties is a common cause of respiratory failure. Acute overdose, long-standing use of some agents (e.g., methadone) may result in chronic hypercapnia.

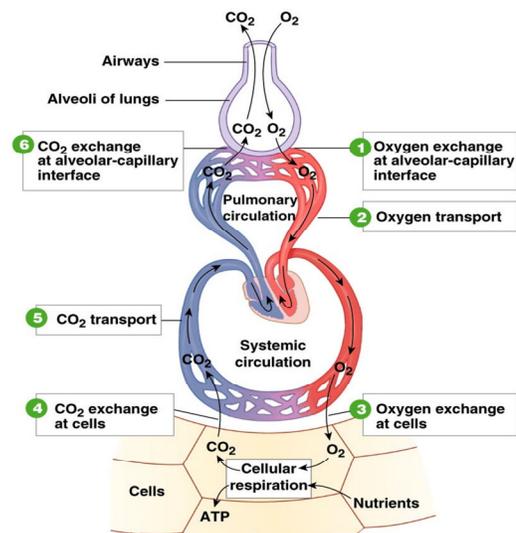
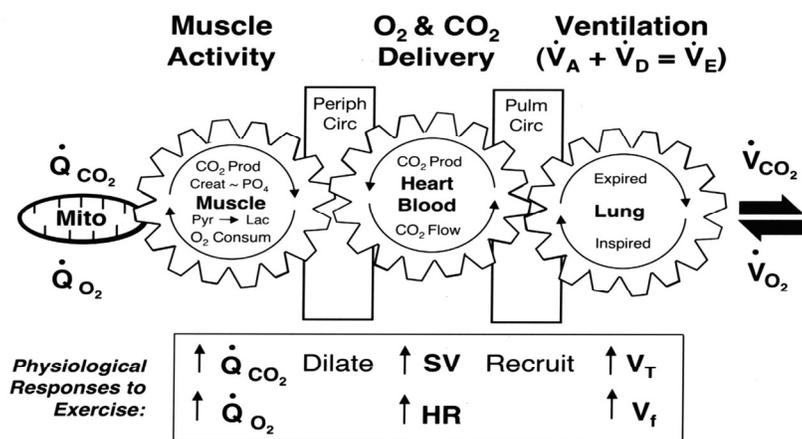
“Structural” CNS abnormalities include meningoencephalitis, localized tumors or vascular abnormalities of the medulla, and strokes affecting medullary control centers.

Metabolic disorders may produce hypercapnia, examples myxedema, hepatic failure, and advanced uremia. A common clinical setting in which elevation of $Paco_2$ is observed is chronic metabolic alkalosis (e.g., due to diuretic use),

Obesity - HypoVentilation Syndrome (OHVS), hypercapnia due to hypoventilation on a central basis.

Type 1 RF:

- It is typically caused by a ventilation/perfusion (V/Q) mismatch; the volume of air flowing in and out of the lungs is not matched with the flow of blood to the lungs (Failure of oxygenation).
- The (V/Q) is a ratio of the amount of air reaching the alveoli (V) to the amount of blood reaching the alveoli. “Q” and it is a measurement used to assess the efficiency and adequacy of the matching of two variables.
- 1 liter of blood can hold about 200 mL of oxygen.
- 1 liter of dry air has about 210 mL of oxygen.
- The ideal (V/Q) would be about 0.9 to 1.05
- The humidified air (with less oxygen), then the ideal (V/Q) would be about 1.0, thus leading to concept of (V/Q) equality or V/Q matching.
- Type I respiratory failure, hypoxia depends upon balance between O_2 Delivery and O_2 Consumption



- (V/Q) mismatch: V and Q of a gas exchanging unit are not matched.
- The V/Q ratio: Apex of lung is higher than Base of lung.

The main reason for lower V/Q ratios at the base is that both ventilation and perfusion increase when going from the apex to the base, but Q increases more than ventilation.

The principal factor of V/Q dishomogeneity between the apex and the base of the lung is gravity.

Ventilation

Gravity and lung's weight act on ventilation by increasing pleural pressure at the base (making it less negative) and thus reducing the alveolar volume.

At the base smaller volumes mean the alveoli are more compliant (more distensible) and so capable of wider oxygen exchanges with the external environment.

The apex, though showing a higher oxygen partial pressure, ventilates less efficiently since its compliance is lower and so smaller volumes are exchanged.

The diagnosis of acute or chronic respiratory failure begins with clinical suspicion of its presence. Confirmation of the diagnosis is based on arterial blood gas analysis

The first step in management is to determine the appropriate setting for care - admission to a standard inpatient facility or to an intensive or intermediate care unit and it depends upon :

- The acuity of the respiratory failure;
- The degree of hypoxemia, hypercapnia, and acidemia;
- The presence of co-morbidities (e.g., cardiac or renal insufficiency);
- The clinical direction that the patient takes over the first few minutes or hours of observation

RF is characterized by inadequate blood oxygenation or carbon dioxide removal. "Adequacy" is defined by tissue requirements for oxygen uptake and carbon dioxide elimination.

In the absence of bedside techniques for direct measurement of these metabolic parameters, clinicians must rely on arterial blood gas values.

Hypoxemic RF, Type I: PaO₂ <55 mmHg (< 8 kpa)

- Acute, develops in minutes to hours
- Chronic, develops over several days or longer

Hypercapnic RF Type II: PaCO₂ >45 mmHg (> 6 kpa)

- Acute Develops in minutes to hours
- Chronic Develops over several days or longer

Type I: Hypoxia, PaO₂ < 8kpa (60mmHg), normal or low PaCO₂

Acute:

- Acute Asthma
- Pulmonary oedema
- Pneumonia
- Lobar collapse
- Pneumothorax
- Pulmonary embolism
- ARDS

Chronic:

- COPD
 - Lung fibrosis
 - Lymphangitis
 - Carcinomatosa
 - R- to- L shunts
 - Brain-Stem lesion
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Type II, hypoxia PaO₂ < 8kpa and Hypercapnia PaCO₂ > 6kpa

Acute:

- Acute severe asthma
- Acute Severe COPD
- Upper airway obstruction
- Acute neuropathy
- Narcotic drugs
- Primary alveolar HypoV
- Flail chest

Chronic:

- COPD
 - Sleep apnea
 - Kyphoscoliosis
 - Myopathies
 - Dystrophy
 - Ankylosing spondylitis
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Morbidity and Mortality in Acute Hypercapnic Respiratory Failure

In general, several parameters presage a higher mortality in patients admitted with hypercapnic respiratory failure:

- 1) The patient's "physiological reserve," as determined by concurrent cardiopulmonary, renal, hepatic, or neurological disease and the age;
- 2) The underlying cause of the acute deterioration;
- 3) The severity of the RF, as defined by arterial pH and Pco₂
- 4) Development of complications after onset of acute respiratory failure

Older patients who are significantly more acidemic, hypotensive, or uremic appear to have a higher mortality. The magnitude of the hypoxemia or hypercapnia at the time of presentation may not reliably foretell mortality.

Complications of Acute Respiratory Failure

<p>Pulmonary</p> <ul style="list-style-type: none"> • Pulmonary emboli • Pulmonary barotrauma (interstitial emphysema, pneumothorax, subcutaneous emphysema, pneumoperitoneum, tension lung cyst, subpleural air cyst) • Pulmonary fibrosis <p>Related to Use of Mechanical Devices</p> <ul style="list-style-type: none"> • Complications of mechanical ventilation (infection, arterial desaturation, hypotension, barotrauma, others) • Complications of insertion and maintenance of pulmonary artery catheter (pneumothorax, air embolism, arrhythmias, infection, thrombosis, pulmonary artery rupture) • Complications of tracheal intubation <ul style="list-style-type: none"> ○ Related to prolonged intubation attempt (hypoxemic brain injury, cardiac arrest, seizures, others) ○ Related to right main bronchus intubation (hypoventilation, pneumothorax, atelectasis) ○ Self- or inadvertent extubation ○ Endotracheal tube dislodgment ○ Endotracheal tube cuff leak ○ Injury to pharynx, larynx, trachea • Complications of tracheotomy (pneumothorax, bleeding, tube dislodgment, tracheoinnominate fistula, tracheoesophageal fistula, tracheal stenosis) 	<p>Gastrointestinal</p> <ul style="list-style-type: none"> • Hemorrhage (including “stress” ulcerations) • Ileus • Diarrhea <p>Cardiovascular</p> <ul style="list-style-type: none"> • Hypotension • Arrhythmias • Decreased cardiac output • Myocardial infarction • Pulmonary hypertension <p>Renal</p> <ul style="list-style-type: none"> • Acute renal failure • Fluid retention <p>Infectious</p> <ul style="list-style-type: none"> • Nosocomial pneumonia • Bacteremia • Sepsis • Paranasal sinusitis <p>Nutritional</p> <ul style="list-style-type: none"> • Complications of underlying malnutrition • Complications of enteral feeding • Complications of parenteral feeding • Complications of refeeding (hypercapnia) <p>Other</p> <ul style="list-style-type: none"> • Psychiatric (anxiety, depression, confusion) • Hematological (anemia, thrombocytopenia)
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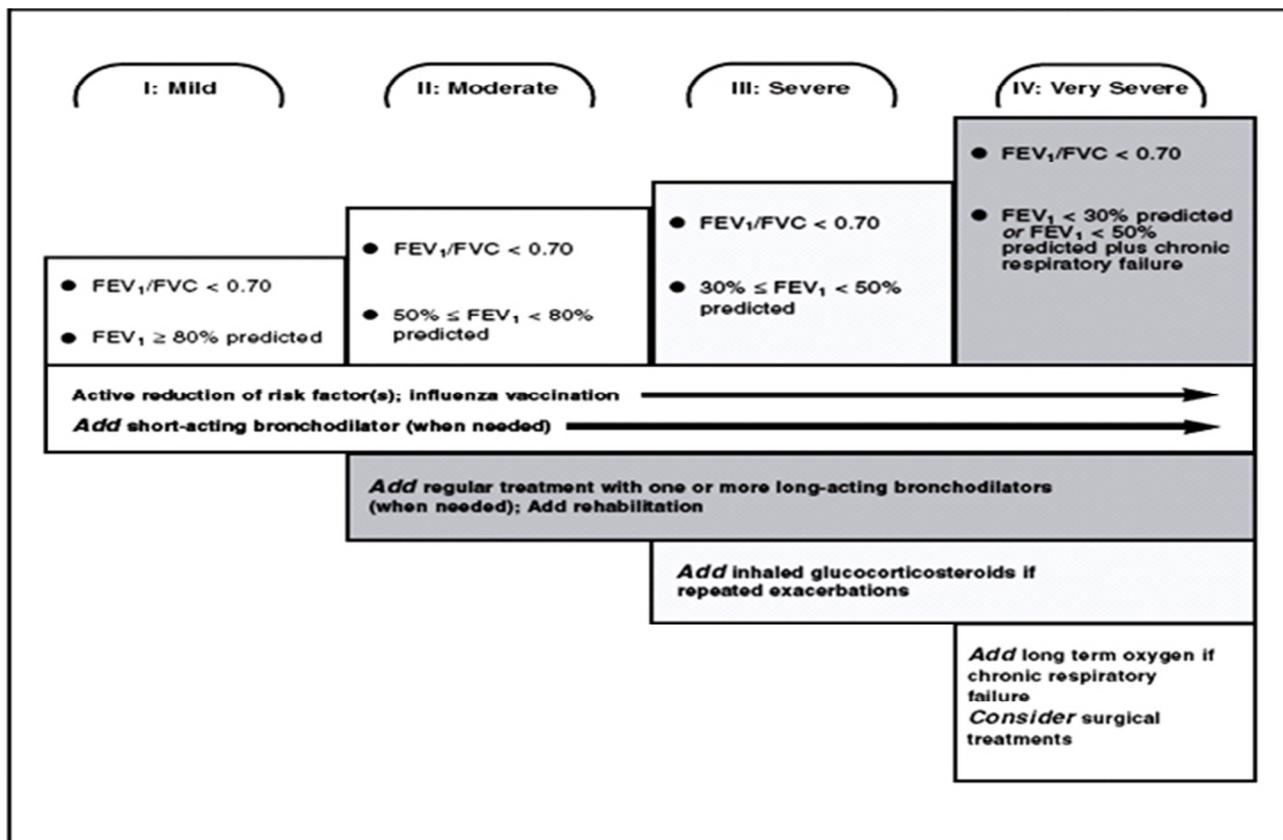
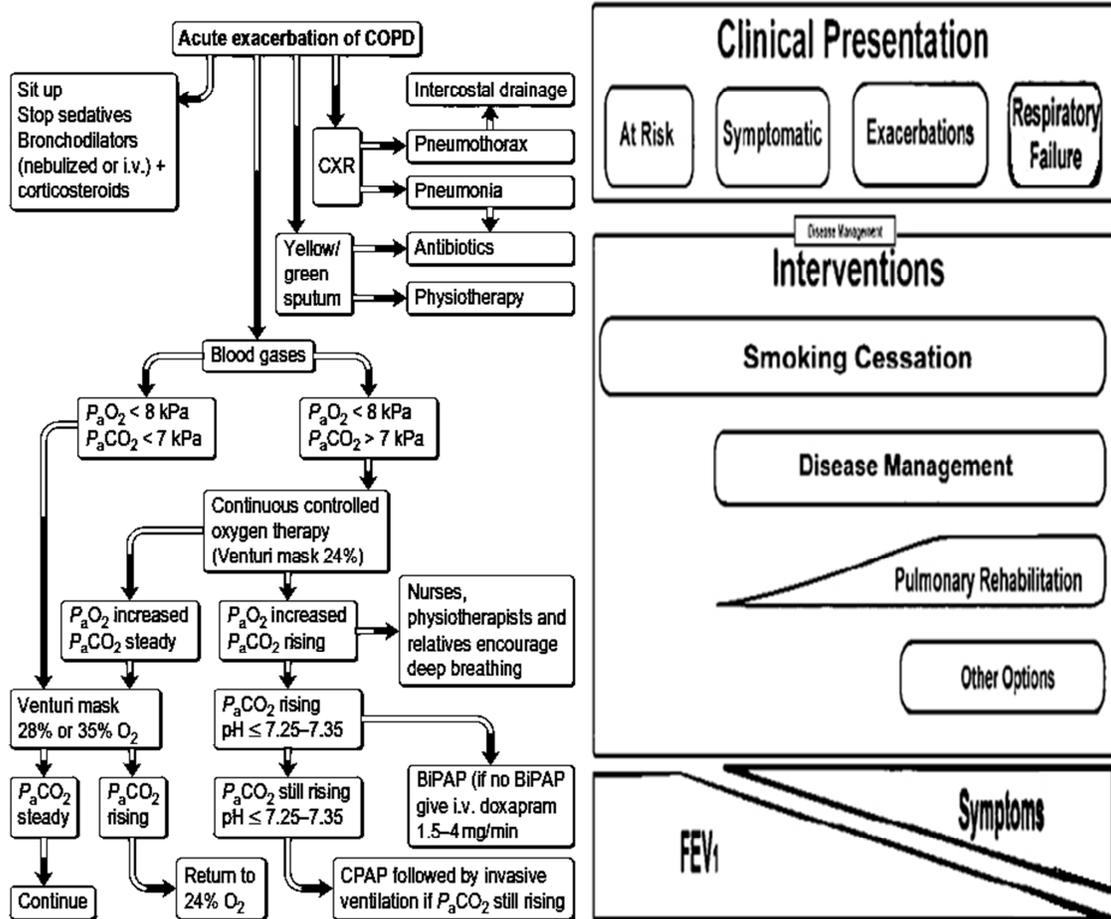
Causes and management of exacerbations of COPD

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Up to 10% of acute hospital admissions in the UK are the result of exacerbations of COPD

BTSociety reported that 20% of annual deaths are due to respiratory diseases and almost 25% due to COPD.

- **Mild:** those requiring an increase in usual therapy.
- **Moderate:** those requiring the introduction of antibiotics and/or steroids.
- **Severe:** those needing hospital admissions.



**Postbronchodilator FEV₁ is recommended for the diagnosis and assessment of severity of COPD.*

Management of RF:

- Diagnose and manage the underline causes.
- ABG is mandatory in the assessment of the initial treatment.

- History of the present illness from the patient or from a company person.
- Opiate antagonist is occasionally successful.
- Don't delay intubation.
- Sedative poisoning or CO₂ narcosis, eventually coma, is a primary failure of neurological drive and don't forget (Intra Cerebral Hemorrhage or head injury).

- Type I RF, O₂ therapy high concentration 40-60 % or more FIO₂ which increases the alveolar PaO₂ in poorly ventilated lung unit.
- Patients need high concentration more than 60% should receive humidified oxygen.
- For COPD 24-28% oxygen by venturi mask to avoid worsening the condition.

At the Emergency Room (ER)

- Initial treatment, the degree of severity and further planning takes place at the ER.
- On call physician (pumlmonologist, internist, or anesthesiologist) should be contacted in case of NIV or intubation.
- When the patient has been transferred from ER, the next post must be registered and well planned.

❖ *Is it Type I or type II respiratory failure?*

Group A: ICU and most probably intubation:

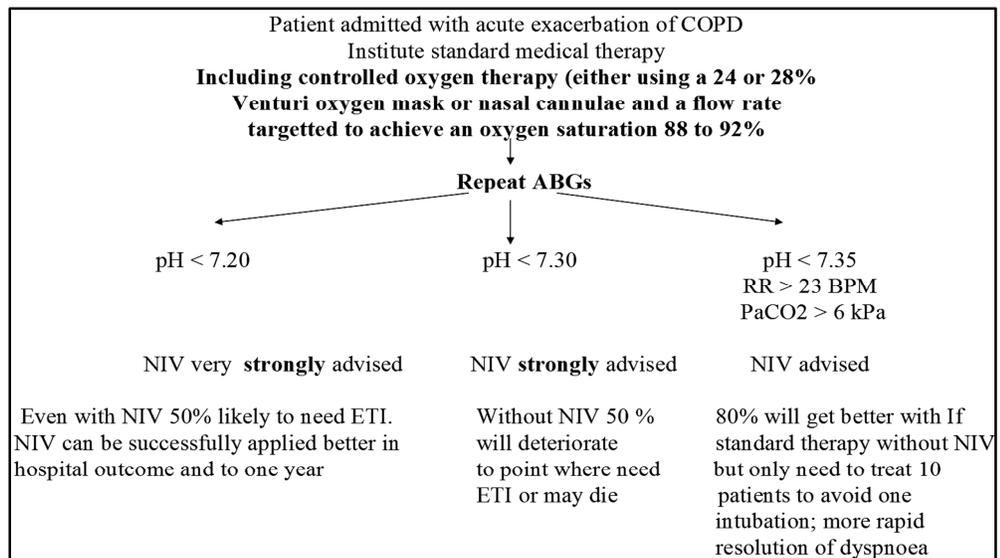
- PaCO₂ narcosis or pH <7.20
- Unstable hemodynamic status. (shock
- Life-threatening arrhythmias (VF) or recurrent (VT)
- Mental unstable, irritable, panic status, facial trauma and unfitness for mask therapy)
- Worsening of the clinical status after a few hours of NIV therapy (unsuccessful NIV).
- PaO₂ <7 kPa despite maximal FiO
- General tiredness, respiratory muscle weakness and intolerable clinical status. Breath frequency > 30. BPM

Group B: Internal M.ICU, CCU, intermediate post

- Acute RF type 1: hypoxia- First choice - CPAP.
- PaO₂ <8 kPa. Normal pH and PaCO₂.
- Pneumonia. Sepsis (Multiorgan dysfunction syndrome- MODS).
- Pulmonary embolism
- Acute Heart Failure (pulmonary edema)
- Acute Asthma Attacks
- FiO₂ %: Start with the high level 100%, then step down gradually.
- CPAP (2.5 cmH₂O to 10 cmH₂O), will increase gradually to the pressure that gives the desired effect.

Acute RF: B- pH under 7.35

- Chronic RF :B- PaCO₂ over 6 kpa
- Assessment and management of Acute RF
 - Acute RF type 1
 - Acute RF type II
- Assessment and management of Acute on Chronic RF
- Assessment and management of Chronic RF



AR Type II – hypercapnia- First choice NIV

- PH <7.35 and PaCO₂ high, but PaO₂ low or normal.
- Acute on chronic respiratory failure (pH <7.35 and PaCO₂ high and low or normal PaO₂).
- Acute exacerbation of COPD.

Group C: Medical ward / lung clinic. For conventional therapy.

- Acute RF that does not meet the criteria in Group A and B.
- Patients must be monitored closely with ABG and should be observed the first few hours in case of reevaluate the treatment concerning (Rescue CPAP or NIV).

Contraindications of NIV:

- Comatose patient.(not hypercapnic comatose syndrome).CO₂ narcosis.
- Facial injury, surgery, deformity.
- Recent upper abdominal operation.
- Poor compliance and panic
- Patient communication- failure.
- Hemodynamic Unstable, severe arrhythmias
- Massive upper airway secret stagnation.
- Serious bulbar dysfunction
- Acute severe upper gastrointestinal bleeding
- Pneumothorax (with chest tube is not contraindicated).
- Acute sinusitis.

NIV Setting:

- Mode = ST (Spontaneous Timed).
- IPAP(Inspiratory positive Airway Pressure) = 12 cmH₂O
- EPAP(Expiratory positive airway Pressure) = 4 cmH₂O
- RF = 12 - 15 or 5BPM under the patient's own
- IPAP min: 0.1 msc
- IPAP max: See table on the machine accordingly.
- Rise Time: 150 msc for COPD and more than 300 msc for NMD
- Trigger: mild, moderate and high.
- Cycle: mild, moderate and high.

