

# RESPIRATORY FAILURE

TUCOM

Dep. of Medicine

3<sup>rd</sup> year

Dr. Hasan I. Sultan

# RESPIRATORY FAILURE

## Learning objectives:

1. Define respiratory failure
2. Review normal arterial blood gases levels
3. Classify respiratory failure
4. Explain the mechanisms of respiratory failure
5. Review the clinical approach to respiratory failure
6. Explain the management of respiratory failure
7. Recognized the types of oxygen therapy

# Respiratory failure

The term is used when pulmonary gas exchange fails to maintain normal arterial oxygen and carbon dioxide levels.

**Normal arterial blood gases levels =**

$PaO_2$  = 12 - 15 kPa (90 - 113 mmHg)

$PaCO_2$  = 4.5 - 6 kPa (35 - 45 mmHg)

$HCO_3$  = 22 - 26 mEq/liter

pH = 7.35 - 7.45

Oxygen saturation > 97 %

# classification

Its classified into types I and II relates to the absence or presence of hypercapnia (raised  $PaCO_2$ ).

- **Type I (Hypoxemic) respiratory failure:**  
characterised by Hypoxia  $PaO_2 < 8.0$  kPa (60 mmHg)  
with normal or low  $PaCO_2 < 6.6$  kPa (50 mmHg).
- **Type II (Hypercapnic) respiratory failure:**  
characterised by Hypoxia  $PaO_2 < 8.0$  kPa (60 mmHg)  
and Raised  $PaCO_2 > 6.6$  kPa (50 mmHg).

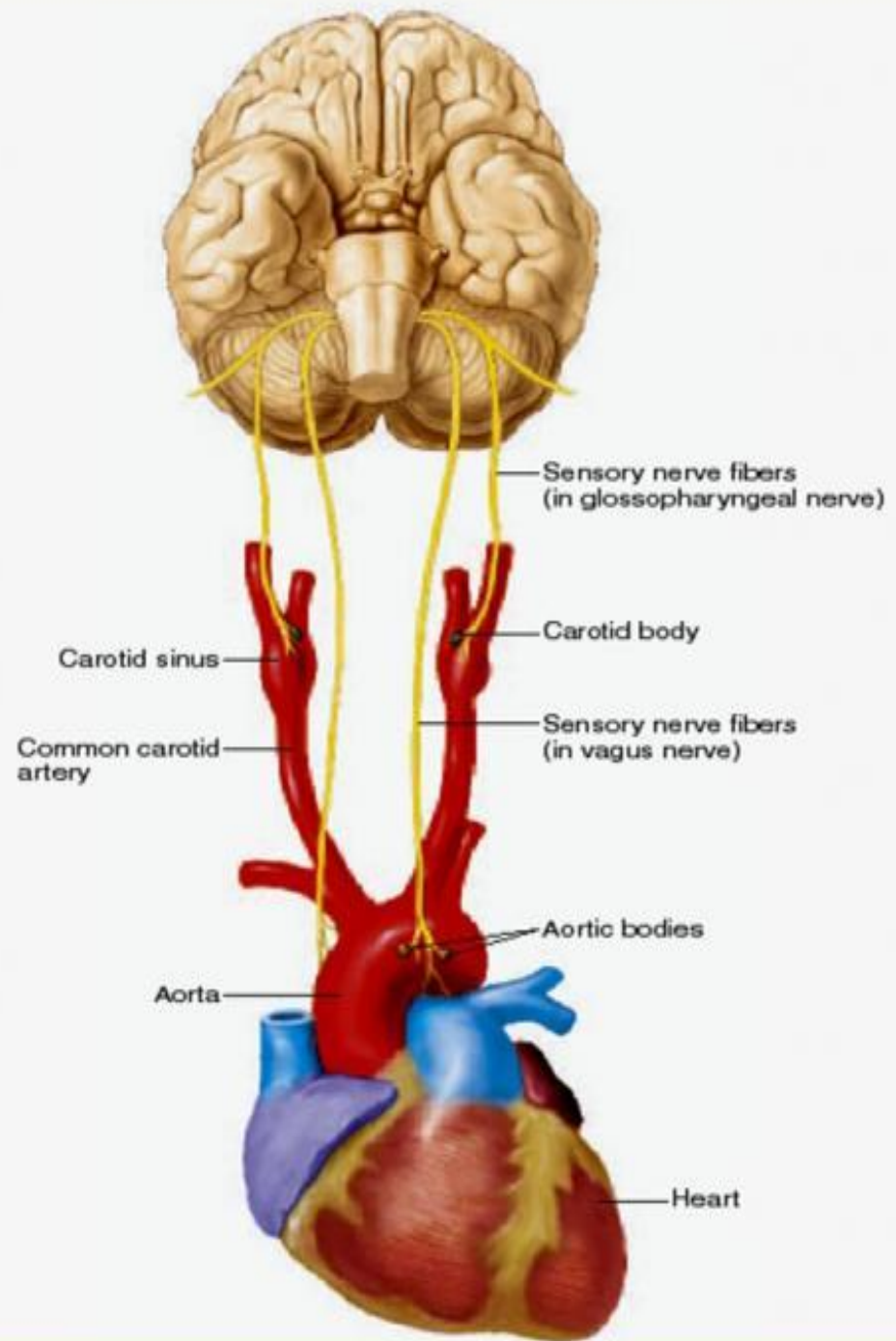
**Acute respiratory failure:** develop over minutes or hours.

**Chronic respiratory failure:** develop over days or longer.

**Respiratory chemoreceptors:**

**1-Peripheral chemoreceptors:** located in the aorta and carotid arteries, respond to change in  $\text{CO}_2$ ,  $\text{O}_2$  and  $\text{PH}$ , in blood.

**2-Central chemoreceptors:** located in medulla oblongata respond to change in  $\text{CO}_2$ , and  $\text{PH}$  but not  $\text{O}_2$ , in CSF.



## Type I

Hypoxia ( $PaO_2 < 8.0$  kPa (60 mmHg)) Normal or low  $PaCO_2$  ( $< 6.6$  kPa (50 mmHg))

Acute	Chronic
Acute asthma	Emphysema
Pulmonary oedema	Lung fibrosis
Pneumonia	Lymphangitis carcinomatosa
Lobar collapse	Right-to-left shunts
Pneumothorax	Brain-stem lesion
Pulmonary embolism	
ARDS	

## Type II

Hypoxia ( $PaO_2 < 8.0$  kPa (60 mmHg)) Raised  $PaCO_2 (> 6.6$  kPa (50 mmHg))

### Acute

Acute severe asthma

Acute exacerbation COPD

Upper airway obstruction

Acute neuropathies/paralysis

Narcotic drugs

Primary alveolar hypoventilation

Flail chest injury

### Chronic

COPD

Sleep apnoea

Kyphoscoliosis

Myopathies/muscular dystrophy

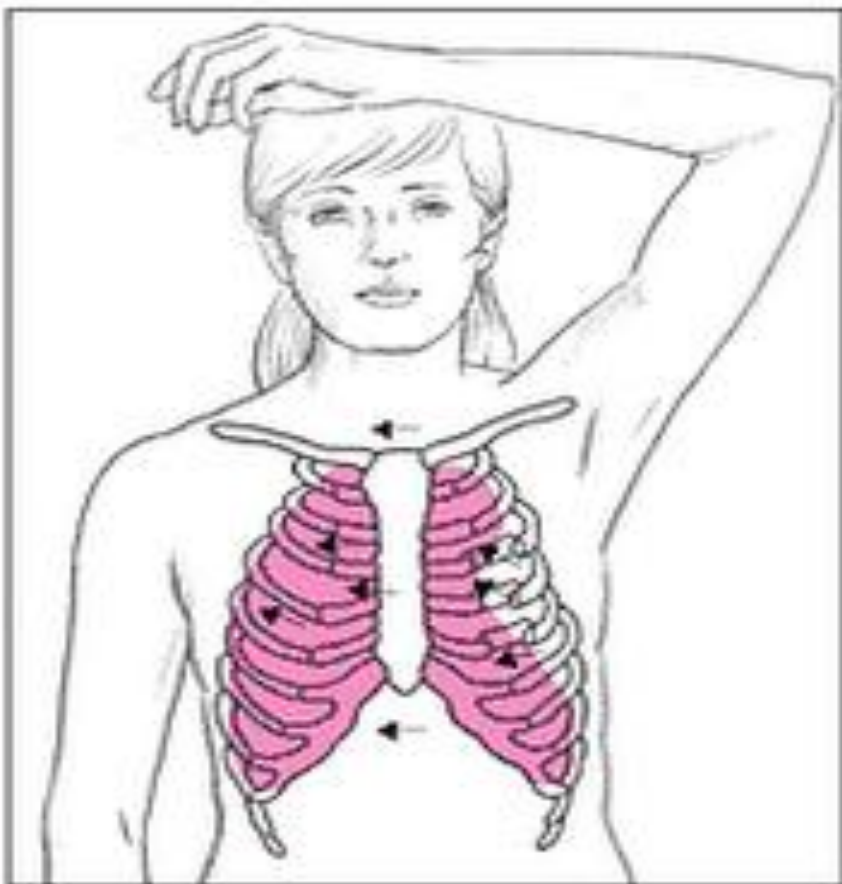
Ankylosing spondylitis

## FLAIL CHEST: PARADOXICAL BREATHING

A patient with a blunt chest injury may develop flail chest, in which a portion of the chest "caves in." This results in paradoxical breathing, described below.

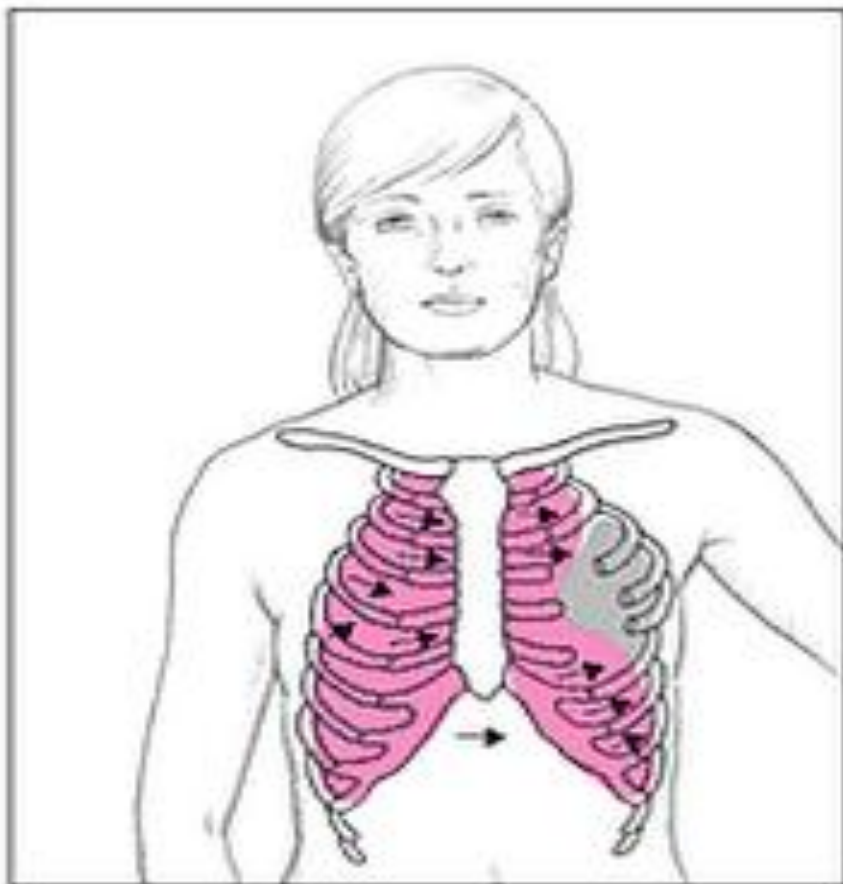
### Inhalation

- Injured chest wall collapses in.
- Uninjured chest wall moves out.



### Exhalation

- Injured chest wall moves out.
- Uninjured chest wall moves in.

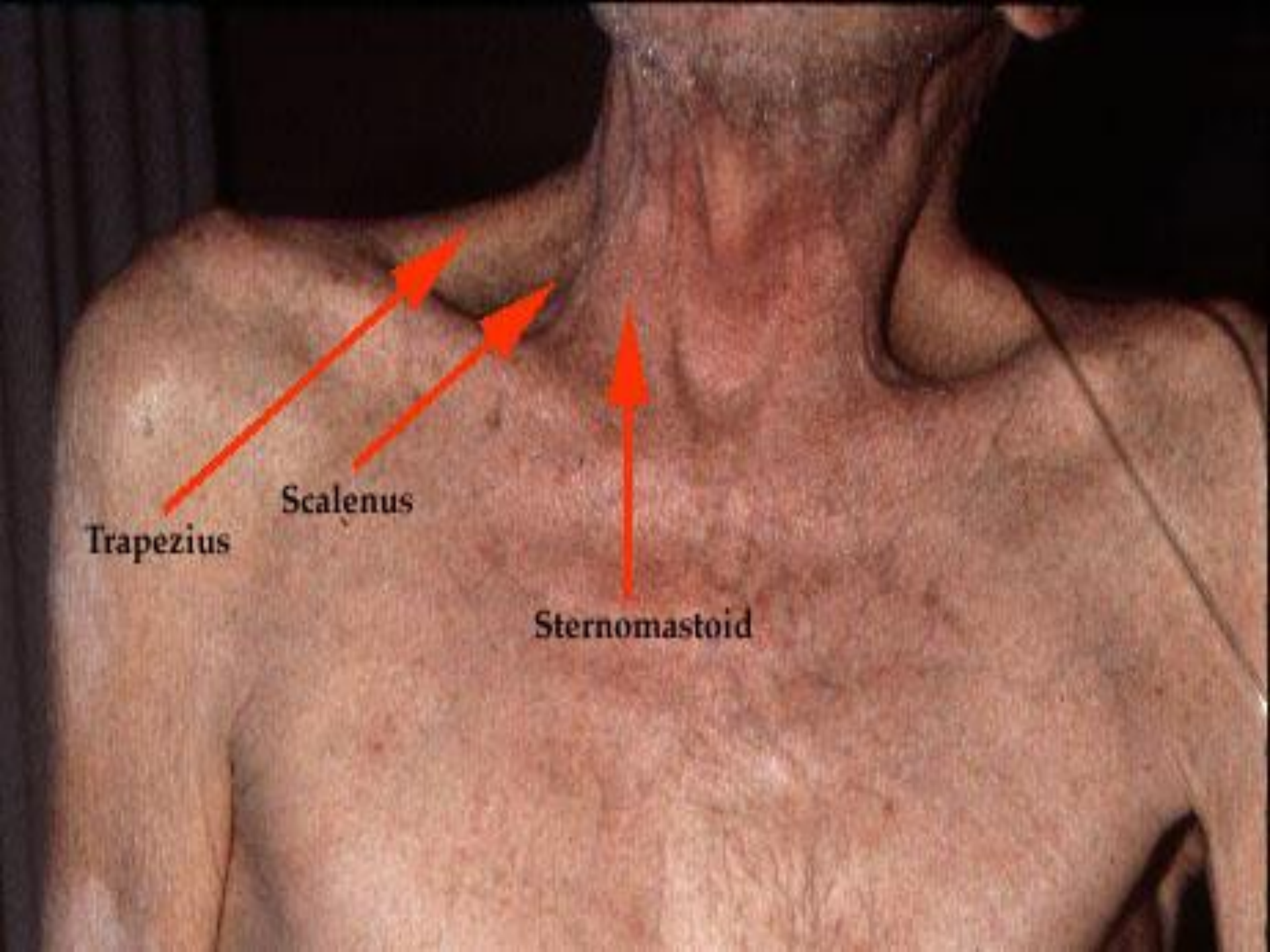




# Clinical evaluation

## 1- Initial assessment:

- History of chest trauma (flail chest, ARDS, or pneumothorax). Asthma. COPD. DVT. Smoking.
- Assessment of conscious level (response to commands, ability to cough)
- Measurement of respiratory rate, depth and pattern of breathing.
- Signs of respiratory distress: flaring of nostrils, pursed lips breathing, use accessory muscles of respiration (sternocleidomastoid, scalene and intercostal muscles), tripod position and tracheal tug.



Trapezius

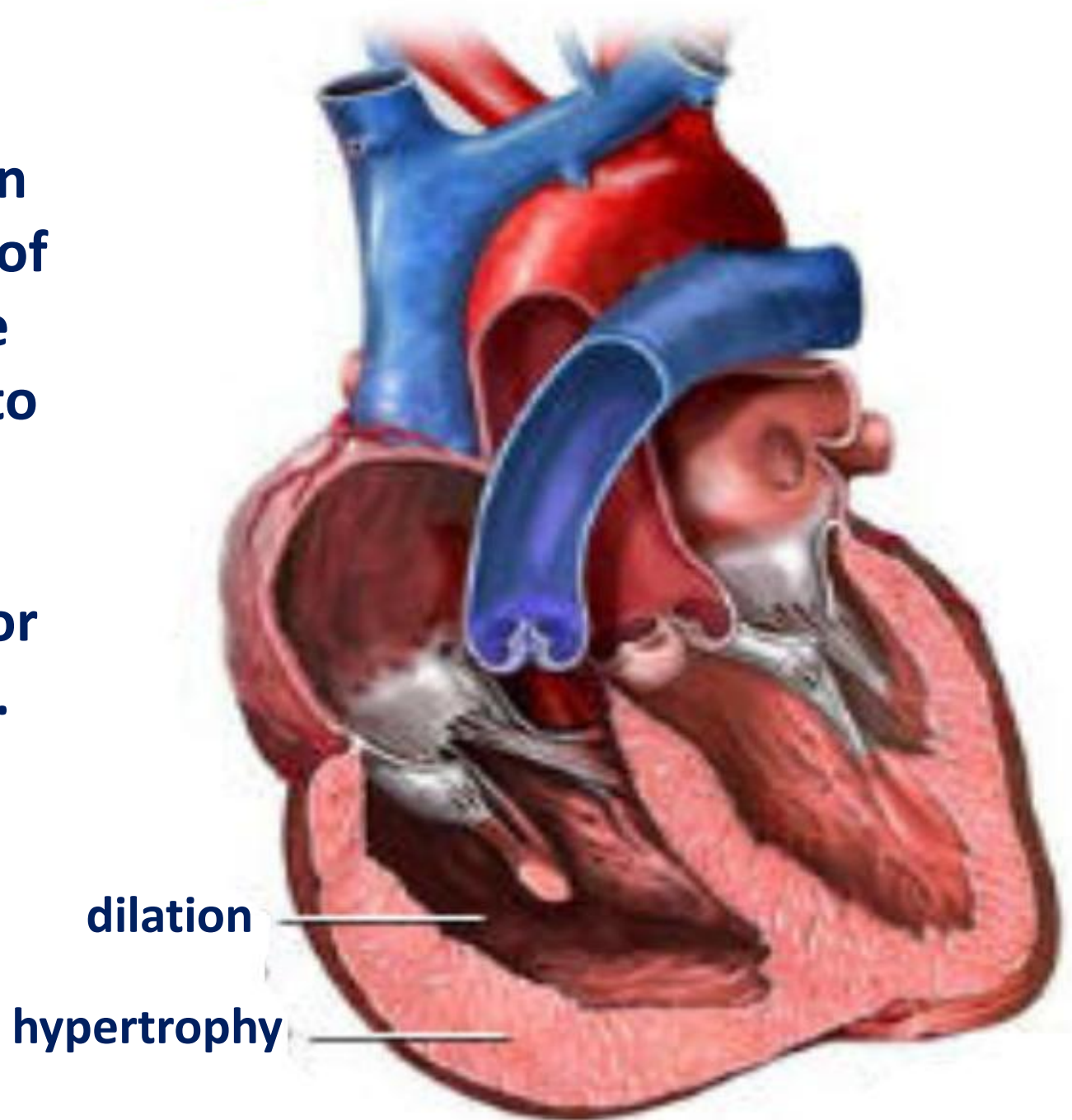
Scalenus

Sternomastoid

- **Signs of sever hypoxemia: cyanosis, systemic hypotension, pulmonary hypertension, polycythemia, tachycardia, and cerebral dysfunction.**
- **Signs of CO<sub>2</sub> retention: warm periphery, bounding pulses and flapping tremor.**
- **Signs of cor pulmonale: peripheral oedema, raised JVP, hepatomegaly and ascites.**



**Note:** Cor pulmonale: is defined as dilation and hypertrophy of the right ventricle (RV) in response to diseases of the pulmonary vasculature and/or lung parenchyma.



## **2- Auscultation over chest:**

- **No air entry: pneumothorax**
- **Expiratory ronchi with prolong expiratory phase: airway obstruction.**
- **Bronchial breathing: pneumonia, lung collapse or lung fibrosis.**
- **Crepitation: pneumonia, pulmonary odema, or brochiactaesis.**

### **3- Investigations:**

- 1. Arterial blood gases (severity of hypoxaemia, hypercapnia, acidaemia, bicarbonate)**
- 2. Pulse oximetry: it is a rapid and simple way for detection of blood oxygen content but not CO<sub>2</sub> .**
- 3. Chest X-ray**
- 4. Others: depend on the causes of respiratory failure, e,g. complete blood count, sputum Gram stain and culture, pulmonary function test or chest CT scan.**

After a pulse is found,  
a blood sample is  
taken from the artery



ADAM



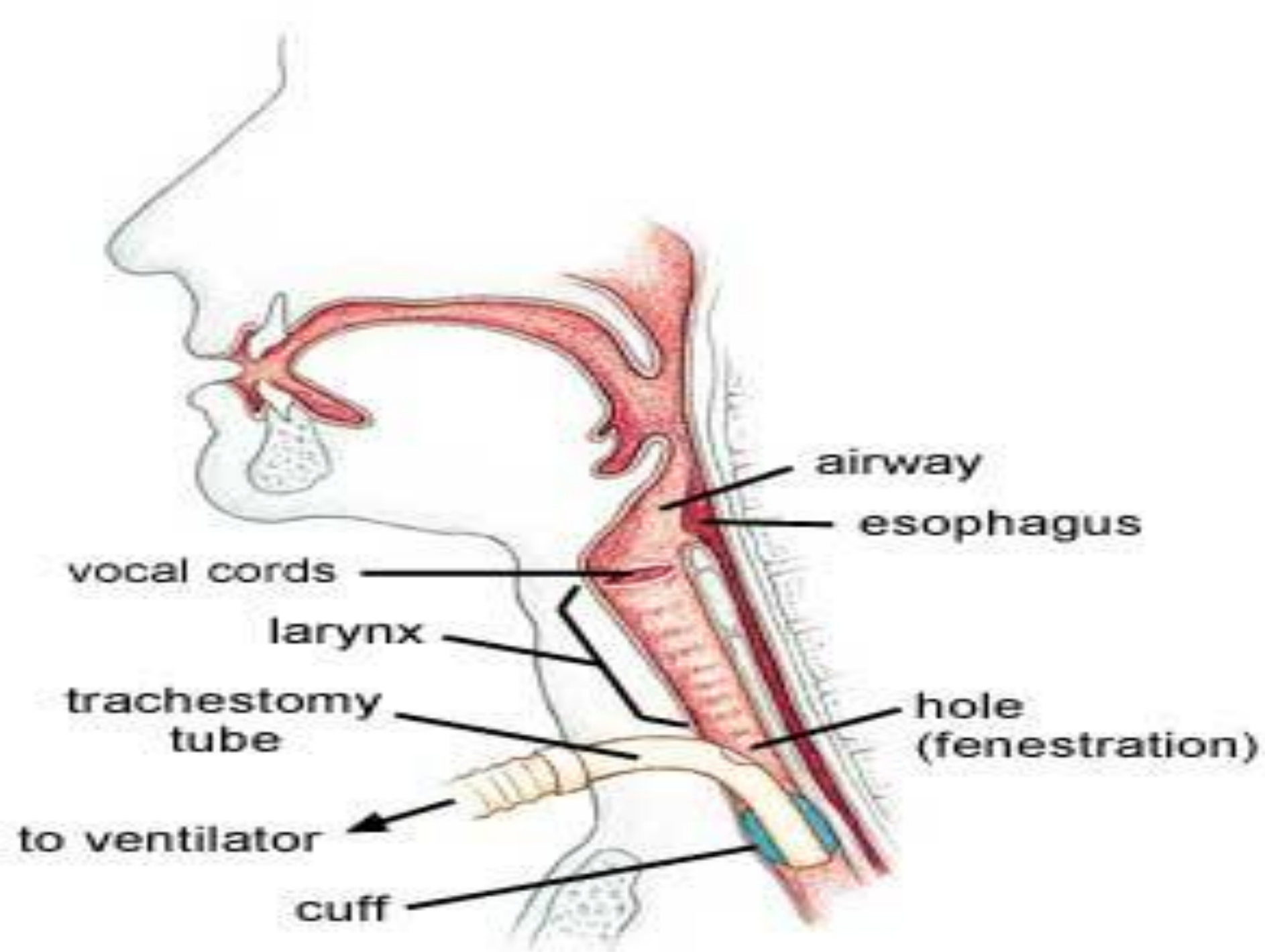


# Management

## 1- Maintenance of airway

## 2- Treatment of specific precipitating cause:

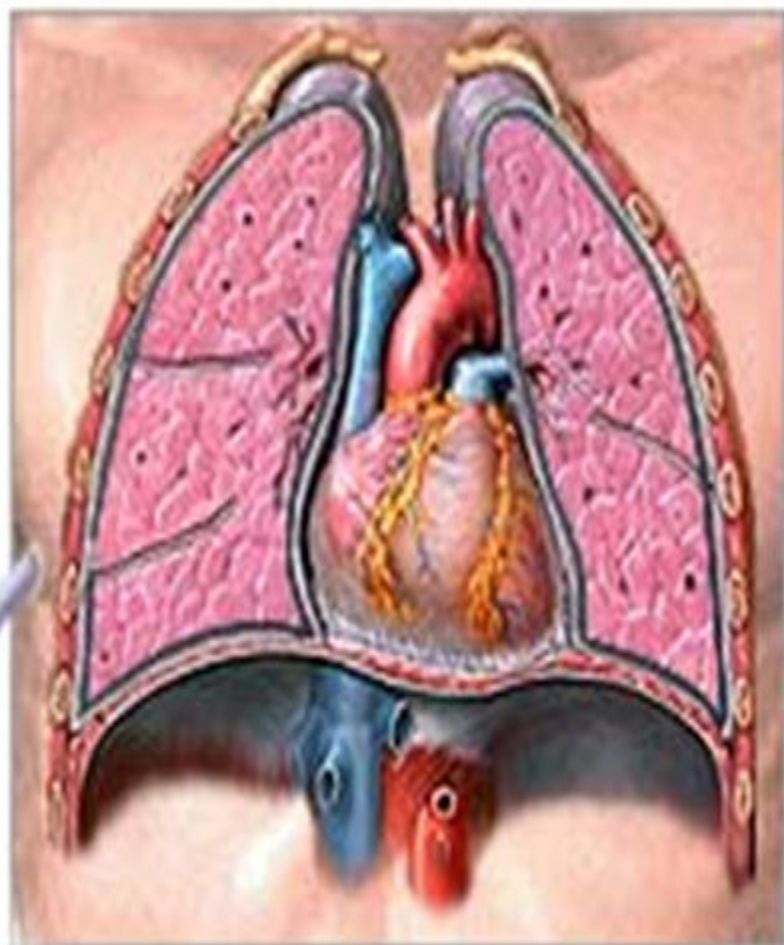
- Tracheostomy for laryngeal obstruction.
- Chest tube for tension pneumothorax.
- Fixation of ribs for flail chest injury.
- Reversal of narcotic drugs poisons.
- Nebulised bronchodilators for bronchospasm.
- Opiates for sever chest pain.
- Frequent physiotherapy  $\pm$  pharyngeal suction.
- Antibiotics if evidence of infection.
- Diuretics if evidence of fluid overload.



# Chest tube



Pneumothorax



Re-expanded lung

### 3- Oxygen therapy

For all types of respiratory failure to restore adequate oxygen level.

**Administration of oxygen:** Oxygen should always be prescribed in writing with clearly specified flow rates or concentrations.

**1-High concentrations:** (40-60% oxygen by a high-flow mask) are particularly useful in acute type I respiratory failure such as commonly occurs in pneumonia, asthma or pulmonary oedema. When high-flow masks are used for prolonged periods, the oxygen should be humidified by passing it over warm water.

**Note:** 100% oxygen is both irritant and toxic if inhaled for more than a few hours. In premature infants cause retrolental fibroplasia and blindness. In adults, it can cause lung damage and fibrosis.



**2-Low concentrations:** (24–28% by Venturi mask) are the most accurate method of delivering controlled oxygen therapy in **type II respiratory failure**. **Why?**

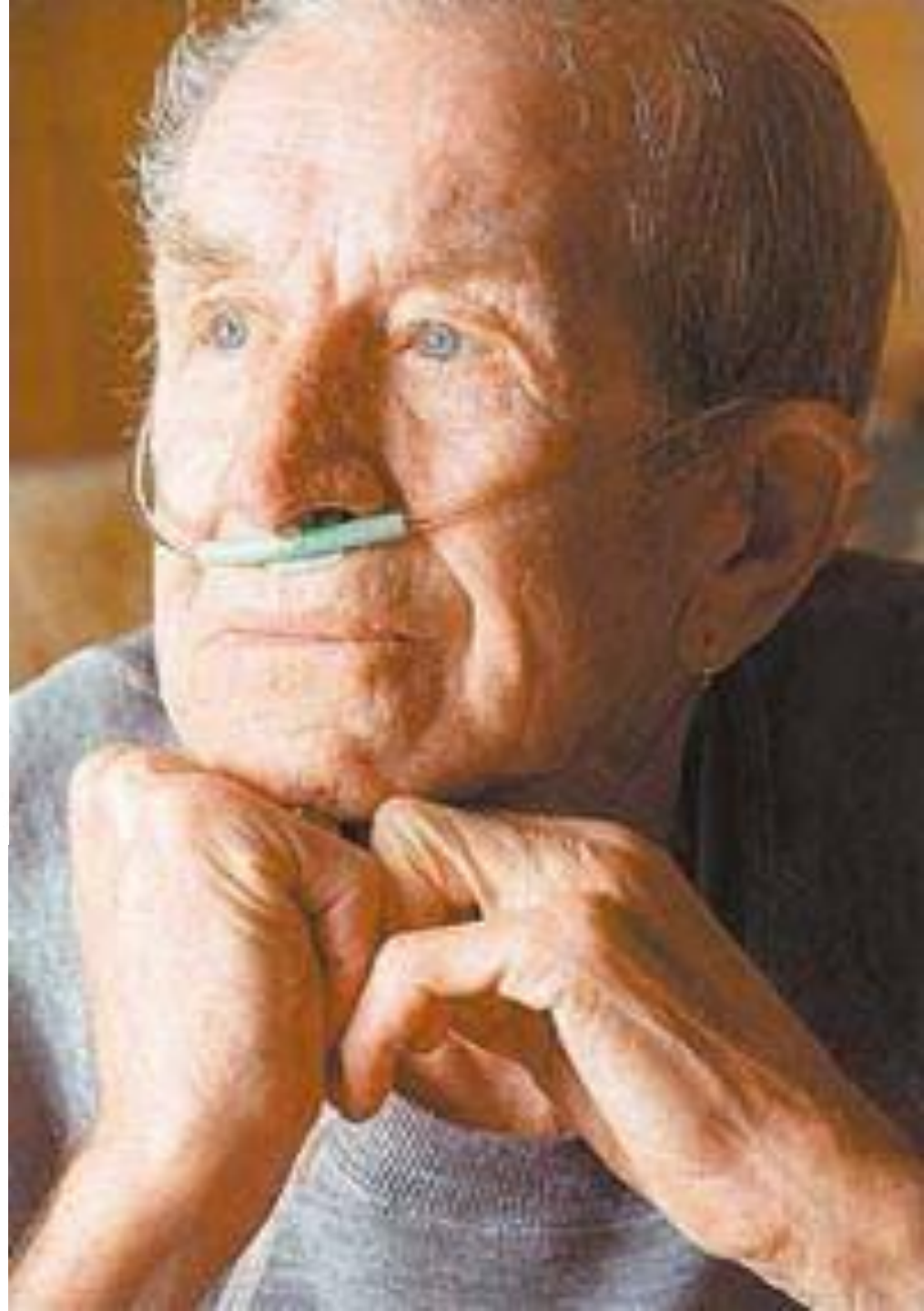
Because of acidaemia corrected by kidneys retention of bicarbonate and there is a loss of chemo-sensitivity to elevated PaCO<sub>2</sub>, so chemoreceptors depend on hypoxia for respiratory drive, and are at risk of respiratory depression if given high concentrations of oxygen.

However, once patients are stable, if a low concentration of oxygen is required continuously for more than a few hours, 1-2 liters per minute delivered via nasal cannula allows patients to eat and to undergo physiotherapy etc. while continuing to receive oxygen.





**O<sub>2</sub> can be administered by nasal prongs or nasal cannula, which are generally well tolerated and allow the patient to cough, speak, eat, and drink while receiving O<sub>2</sub>**



***3-Chronic oxygen delivery:*** from cylinders delivered to the home, from an oxygen concentrator, is often given via a low-concentration mask or nasal cannula. Portable oxygen may increase exercise tolerance in some patients with chronic hypoxic lung disease, and lightweight portable cylinders with oxygen-sparing devices may allow previously housebound patients to resume outdoor activities.

**Note:** If  $\text{PaCO}_2$  continues to rise or a safe  $\text{PaO}_2$  cannot be achieved without severe hypercapnia and acidaemia, mechanical ventilatory support may be required.



## **4- Mechanically assisted ventilation**

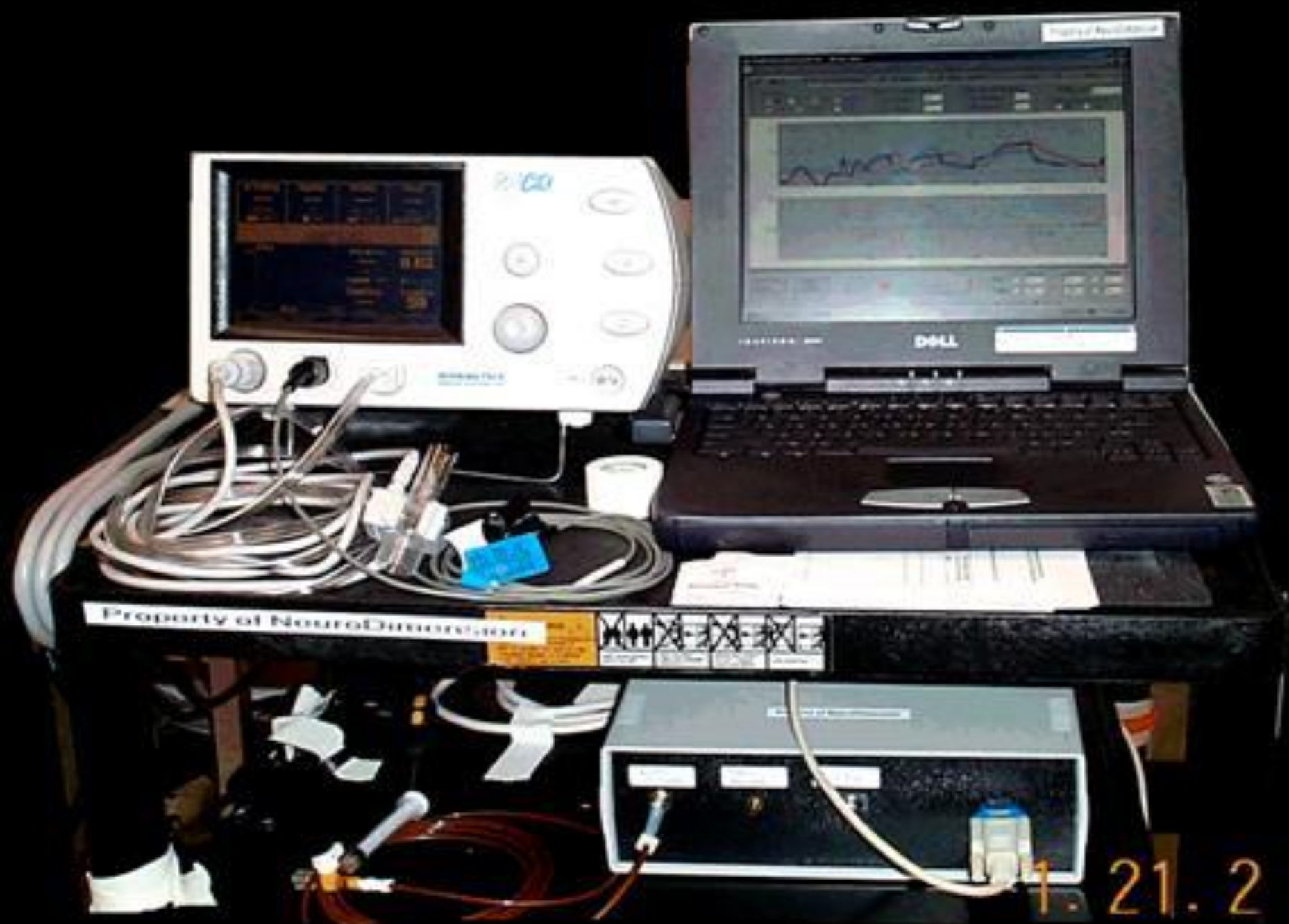
**Patients with initially severe respiratory failure (type I or type II) or those who fail to improve despite optimal medical therapy may require mechanical ventilation.**

### **The various types of**

- **Non-invasive (via a face or nasal mask) or**
- **Invasive (via an endotracheal tube) ventilation.**



**Non-invasive ventilation for the respiratory distressed patient**



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Patient Data		Respiratory	
SpO2	99.8	Total Rate	17
HR	12	PIP	18.4
RR	17	EPAP	15
PS Level	7.4	EPAP	5
Total Vol	583	FiO2	70
Total VE	10.5		

**5- Doxapram:** (1.5- 4 mg/min) by slow intravenous infusion should only be used as a respiratory stimulant where non-invasive ventilation is not available or is poorly tolerated, or in those with reduced respiratory drive. Even in these circumstances this agent provides only minor and transient improvements in arterial blood gas parameters.



**6- Lung transplantation:** is an established treatment for carefully selected patients with advanced lung disease unresponsive to medical treatment.

- Single-lung transplantation: may be used for selected patients with advanced emphysema or lung fibrosis.
- Bilateral lung transplantation: indicated in patients with chronic bilateral pulmonary infection, such as cystic fibrosis and bronchiectasis
- Combined heart–lung transplantation: for patients with advanced congenital heart disease, such as Eisenmenger’s syndrome, and primary pulmonary hypertension unresponsive to medical therapy.

**Thanks**