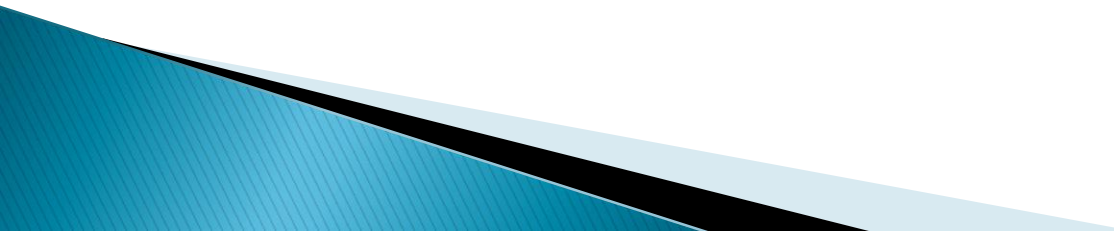



CHRONIC RESPIRATORY FAILURE

BY

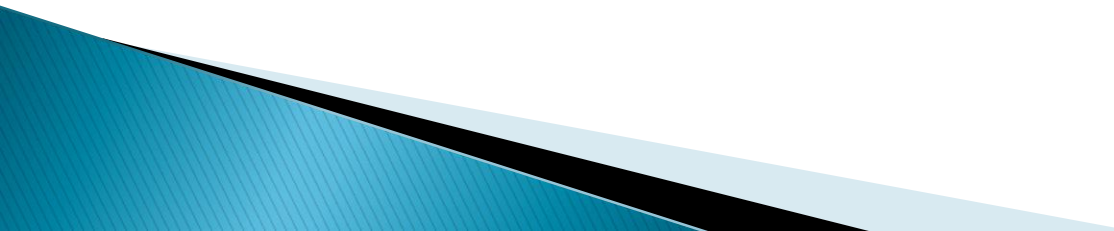
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CHRONIC RESPIRATORY FAILURE

- . DEFINITION
 - . RISK FACTORS
 - . TYPES
 - . CAUSES
 - . PATHOPHYSIOLOGY
 - . CLINICAL FEATURES
 - . DIAGNOSIS
 - . TREATMENT
 - . COMPLICATIONS
- 

RESPIRATORY FAILURE

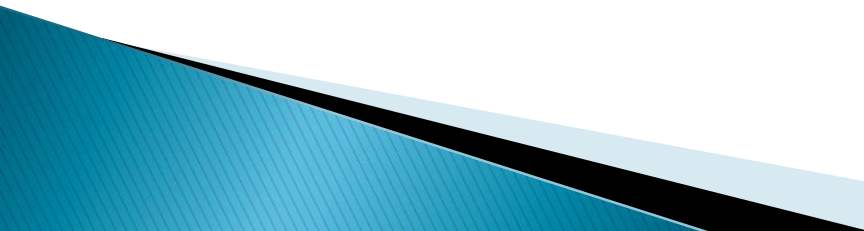
- ▶ The term respiratory failure is used when the lungs are unable to perform their basic task i.e. gas exchange
 - ▶ Respiratory failure is the inability of the respiratory system to supply O₂ or to remove CO₂ from the blood resulting in low blood O₂ or high blood CO₂ respectively.
- 

WHO IS AT RISK FOR RESPIRATORY FAILURE

People who have disease or condition that affect the

- ▶ Tissues (lungs)
- ▶ Muscles
- ▶ Nerves
- ▶ Bones

that support breathing are at risk for respiratory failure..



TYPES

ACUTE RF : It develops over minutes to hours.

It is characterized by

- . an acute lack of O₂ delivery to the blood by the respiratory system.
- . Or acute failure of the respiratory system to remove CO₂ from the blood.

CHRONIC RF: it develops over longer periods of time and is usually defined as long term lack of O₂ delivery to the blood by the RS.

TYPE I AND TYPE II RF

- ▶ **TYPE I : HYPOXEMIA WITHOUT HYPERCAPNIA.**
- ▶ If control of ventilation is intact, ventilation increases in response to a raised paCO_2 so that excess CO_2 is excreted by the normal areas of the lungs.

This requires that the lungs are mechanically normal and are able to respond to the increased drive.

TYPE I AND TYPE II RF

▶ TYPE II RF : HYPOXEMIA WITH HYPERCAPNIA

Hpercapnia usually occurs in hypoxia only when hypoxia is caused by:–

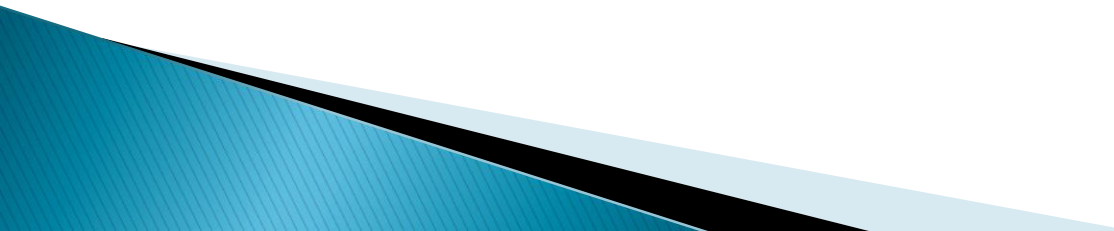
- Hypoventilation : CO₂ transfer b/w the alveoli and the atmosphere is affected as much as O₂ transfer causing hypercapnia.
- Circulatory failure : diminished flow of blood decreases CO₂ removal from the tissues resulting in tissue hypercapnia in addition to tissue hypoxia.

CHRONIC HYPOXEMIA IS BETTER TOLERATED THAN ACUTE HYPOXEMIA

Some compensatory mechanisms are fully developed only in chronic hypoxemia. In such patients :

- ▶ Erythrocytosis (d/t increased production of erythropoetin)
- ▶ Angiogenesis and increased perfused capillary density (reduces diffusion distances for O₂ in the tissues)

Tend to ameliorate the deleterious effects of hypoxemia



CHRONIC HYPOXEMIA IS BETTER TOLERATED THAN ACUTE HYPOXEMIA

- ▶ It is of note that while patients with CRF can remain conscious with paO_2 of 30mmhg and acclimatized mountaineers have remained conscious at high altitude with paO_2 as low as 20mmhg , uncompensated subjects with acute hypoxemia are unlikely to remain conscious with paO_2 less than 27mmhg.

COMPENSATORY MECH IN CHRONIC RESPIRATORY FAILURE

CRF causes progressively worsening respiratory acidosis (chronic acid accumulation resulting from inefficient expulsion of CO₂ from respiratory system).

The failure of RS to remove sufficient CO₂ from the blood over time leads to renal compensation. Baseline O₂ & CO₂ levels in the arterial blood may be normal and compensated for by

- ▶ The renal system : by increasing bicarbonate to compensate for respiratory acidosis
- ▶ The haemodynamic system : by increasing red cell volume to compensate for decreased O₂.

➤ ACUTE RESPIRATORY FAILURE

- ▶ Develops over minutes to hours
- ▶ Decrease pH quickly to less than 7.2
- ▶ Eg. Pneumonia

➤ CHRONIC RESPIRATORY FAILURE

- ▶ Develops over days
Increase in HCO_3
- ▶ Decrease in pH slightly
- ▶ Polycythemia
- ▶ Eg. COPD

CAUSES OF CHRONIC RESPIRATORY FAILURE

- ▶ **COPD** : chronic obstructive pulmonary disease
- ▶ **OSA** : obstructive sleep apnea/ hypopnea
- ▶ **CYSTIC FIBROSIS**
- ▶ **BONY DEFORMITIES**: scoliosis
- ▶ **NEUROMUSCULAR DISORDERS**:
Amyotrophic lat. Sclerosis(severe NMDs that causes ms weakness and disability)
Guillain barre syndrome(AI nerve disorder)
Myasthenia gravis(AI NM disorder that causes ms weakness)

COPD : A HIGH PENALTY TO PAY FOR SMOKING

MECHANISM

- ▶ Entrapment of air n overstretching and destruction
- ▶ Chronic irritation of bronchi and bronchioles by smoke
- ▶ Partial paralysis of the cilia of respiratory epithelium
- ▶ Mucous cannot be moved easily of the passageways
- ▶ Chronic obstruction of smaller airways

EFFECTS ON LUNGS

- ▶ Bronchiolar obstruction causing increased airway resistance
- ▶ Loss of alveolar walls decreasing the diffusion capacity of the lungs
- ▶ Loss of alveolar walls causing decrease in no of pul cappilaries leading to increased pul. Vascular resistance : Pul.HT

OBSTRUCTIVE SLEEP APNOEA

WHO ARE AT RISK

OBESE PERSONS :

Increased fat deposition around the upper airway particularly lateral to pharynx : causing upper airway obs

PTS WITH BONY DEFORMITIES:

Like small maxillae and mandibles with resulting retrognathia causing AP shortening of face and narrowing of upper airway.

MECHANISM

- ▶ Airway narrowing causing increased respiratory effort leading to arousal at night and day time sleepiness.
- ▶ Chronic CO₂ retention may occur in a pt with COPD whose RC is insensitive to CO₂ and has increased no of apneic episodes during sleep. Another pt with equally severe copd who has high sensivity to CO₂ and normal sleep may not retain CO₂

CYSTIC FIBROSIS

- The abnormality in CFTR i.e. CF Transmembrane conductance Regulator func result in relative dehydration in the pericellular layer above the epithelial cells.
- ▶ This facilitates bacterial adherence and inhibit mucociliary clearance.
 - ▶ Chronic bacterial colonisation with intermittent acute inflammatory responses
 - ▶ Bronchitis and bronchiolitis causing dilatation of bronchial wall with resultant bronchiectasis
 - ▶ Progressive lung damage causing deterioration of lung function
 - ▶ Pul HT and RESPIRATORY FAILURE.

SCOLIOSIS

Defined as lateral curvature of the spine and is associated with rotation of spine and viscera adjacent to it. In scoliosis:–

- ▶ Total lung volume is reduced
- ▶ Lungs differ in size
- ▶ Distortion of lobar shapes d/t deformity

The higher the curve, the more severe its effect on function.

In patients with severe scoliosis:–

- ▶ Severe desaturation may occur during sleep
- ▶ Episodes of central / obstructive apnea/hypopnea
- ▶ Occurrence of CO₂ retention
- ▶ Defective chemical control over ventilation.
- ▶ Rapid deterioration of lung function
- ▶ RESPIRATORY FAILURE

CLINICAL FEATURES

- **CLINICAL EVIDENCE OF
HYPOXEMIA**
- **CLINICAL EVIDENCE OF
HYPERCAPNIA**

CLINICAL EVIDENCE OF HYPOXEMIA

- ▶ **CENTRAL CYANOSIS** = Assessed by examining the oral mucous membrane.
- ▶ **On CNS** = Irritability, impaired intellectual function & clouding of consciousness. May progress to convulsions, coma & death
- ▶ **STIMULATES VENTILATION** = Via carotid chemoreceptors causing increased HR & CO & dilates peripheral BV.
- ▶ **ON HEART** = Tachycardia and dysrhythmias
- ▶ **PULMONARY ARTERIES** = vasoconstriction=increased PVR =pul. Hypertension.
- ▶ **PERSISTENT HYPOXEMIA** = secondary polycythemia d\ t increased production of erythropoetin..

CLINICAL EVIDENCE OF HYPERCAPNIA

- * Vary from patient to patient
- * Changes in paCO_2 are more important than the actual level..
- 1. Irritability, confusion, somnolence and coma.
- 2. Tremors , myoclonic jerks, astrexis and even seizures.
- 3. Dilate blood vessels=
 - . CRANIAL= increased BF = headache n papilloedema
 - . PERIPHERAL= warm flushed skin
 - . GENERALIZED = hypotension
- 4. Sympathetic stimulation= tachycardia & sweating

DIAGNOSIS

1. HISTORY

2. CLINICAL EXAMINATION

3. LAB INVESTIGATIONS



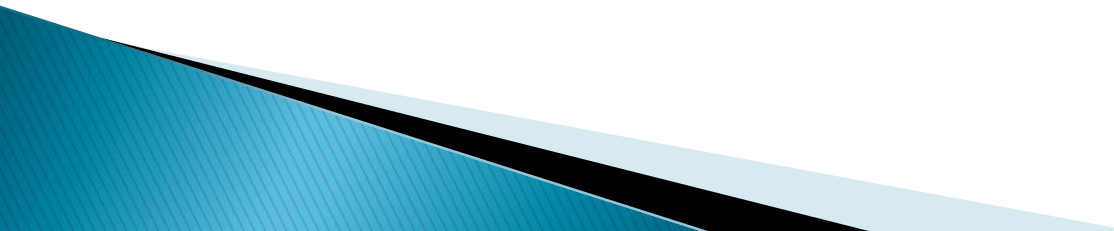
1. HISTORY

A. RESPIRATORY SYMPTOMS : dyspnoea

B. NON RESPIRATORY SYMPTOMS: In patients with CRF 2*to NMDs ; central ventilatory defects have few or no respiratory complaints..

There occurs nocturnal exaggeration of CO_2 that disrupts sleep patterns causing nightmares, enuresis, and morning headaches..

During the day = fatigue, hypersomnolence and mood disorders..



2. CLINICAL EXAMINATION

- A. Morbid obesity, retrognathia, tonsillar hypertrophy & macroglossia == point to obstructive sleep apnoea.
- B. Examination of thoracic cage for scoliosis.
- C. Neurologic examination seeking muscle weakness suggestive of a neuromuscular syndrome.
- D. Bilat. Diaphragmatic paralysis = seen by observing paradoxical motion of abdomen during inspiration

3. LAB INVESTIGATIONS

A.

NON-INVASIVE :

1. PULSE OXIMETRY
2. PFTs
3. POLYSOMNOGRAPHY

B.

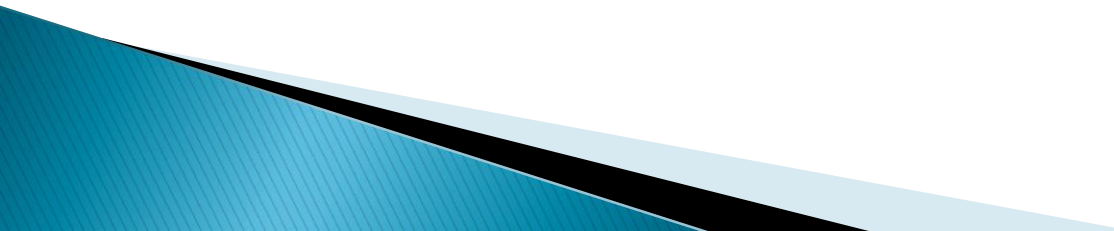
INVASIVE :

1. ROUTINE
2. ABG ANALYSIS

- ▶ 1. **PULSE OXIMETRY** : have been used to measure the degree of oxygenation non-invasively , but provide no information on paCO_2 .Moreover the 95% confidence limits of pulse oximeters is 4%.
- ▶ 2. **PFTs including SPIROMETERY** : measurement of lung vol., maximal inspiratory and expiratory , VC will detect pts. with severe obstructive and restrictive ds. If FEV_1 is $> 1\text{l}$ in a pt with CRF lung ds is probably not the sole cause, & other factor should be sought..

3. POLYSOMNOGRAPHY : consist of an overnight study with monitoring of the EEG, EMG, EOG, airflow at the mouth & nose, chestwall motion & oximetry. It is indicated for any pt with CRF and syptoms to suggest OSA such as snoring or excessive daytime sleepiness.

4. MEASUREMENT OF TRANSDIAPHRAGMATIC PRESSURES : using gastric and esophageal balloon is useful to confirm the diagnosis of bilateral diaphragmatic paralysis.



INVASIVE INVESTIGATIONS

- ▶ **COMPLETE BLOOD COUNT AND OTHER BLOOD TESTS:** to exclude polycythemia , thyroid abnormalities, serum chemistry studies.
- ▶ **ABG ANALYSIS :** the diagnosis of RF rests on the measurement of arterial blood gases. The chronicity of the respiratory failure should be assessed by evaluating the pH & the degree to which this is compensated by an increase in serum bicarbonate.
- ▶ An acute increase in paCO_2 of 10mmHg is roughly associated with a decrease in pH of 0.08 U and an increase in S. HCO_3 of 1mEq/l. whereas a chronic increase in paco_2 of 10mmHg is associated with a decrease in pH of 0.03 U and increase in S. HCO_3 of 3.5 mEq/l

PITFALLS IN ABG ANALYSIS

Delivery of O₂ to the tissues demands 4 steps:-

1. Ventilation (transfer of O₂ from the environment to the lungs).
2. Pulmonary O₂ exchange.
3. O₂ transport to the tissues.
4. Tissue gas exchange (utilization of O₂ and release of CO₂ by the peripheral tissues).

ABGs reflect changes in the first 2 steps and they offer no information at all for the crucial factor which is the adequacy of tissue gas exchange.

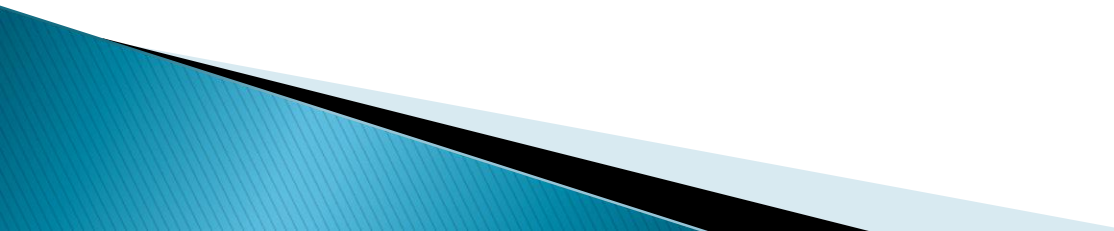
TREATMENT

▶ GENERAL PRINCIPLES OF MANAGEMENT

1. To correct life threatening hypoxia
2. To correct life threatening acidemia
3. To treat the underlying cause
4. To prevent complications

O₂ THERAPY

Since the immediate threat to the patients of CRF is d/t inadequate level of O₂ delivered to the tissues, oxygenation is the basic therapy for RF d/t lung disease. O₂ enriched air is given to the pt. by nasal prongs, venturi mask or by placing an airtube into the trachea . Since high O₂ levels can be toxic, the conc. Of O₂ must be carefully controlled for both long term and short term..



O₂ THERAPY

- ▶ Venturi mask produces the most predictable inspired O₂ concentration.
- ▶ Conc. from nasal prongs are less predictable but the device is better tolerated by the patients. On an average , PaO₂ increase by 10mmhg as the FiO₂ is increased from 21 to 24%. O₂ flow of 2l/min by nasal prongs produces an FiO₂ of 25 – 35%.

ASSISTED VENTILATION WITH MECHANICAL DEVICES : may be the first priority for neuromuscular ds. Pts. going into RF

- ▶ **ENDOTRACHEAL TUBE INSERTION** : it permits delivery of precisely determined amounts of O₂ to the lungs and removal of secretions & ensures adequate ventilation. Combined with the mech. ventilation endotracheal intubation is the cornerstone of the therapy of respiratory failure.
- ▶ **MECHANICAL VENTILATION** : if the pt is tiring despite the ongoing therapy, a mechanical ventilator , also called a respirator is used. The ventilator assists or controls the pts. breathing.

- ▶ **PEEP** : is used with mechanical ventilation to keep the air pressure in the trachea @ the level that increase the vol of gas remaining in the lung after breathing out. This keeps the alveoli open, reduce the shunting of blood th' lungs & improve gas exchange. Most ventilators have PEEP adjustment.
- ▶ **MANAGEMENT OF FLUIDS AND ELECTROLYTES** : Pulmonary edema, the build up of abnormal amounts of fluid in the lung tissues, often occurs in respi failure. Therefore fluids are carefully managed and monitored to maintain fluid balance & avoid fluid overload which may further worsen gas exchange.

PHARMACOLOGIC THERAPY

- ▶ Reversal of airway obstruction using bronchodilators or steroids.
- ▶ Treatment of CHF using diuretics.
- ▶ Correction of metabolic alkalosis.
- ▶ AMINOPHYLLINE is an acute respiratory stimulant and when given i/v to COPD pts with chronic CO₂ retention , may actually reduce hypercarbia. However long term studies on effects of theophylline in pts with COPD & ch. Hypercarbia have not shown consistent reversal of CRF.

PHARMACOLOGIC THERAPY

- ▶ **ALMITRINE BISMESYLATE:** a peripheral chemoreceptor agonist, has been use to enhance oxygenation in pts with COPD during both sleep and wakefulness. However , the drugs effects appear to be related more to an improvement in v/q relationships rather than increased respiratory drive and role of almitrine in treatment of CRF remains questionable.

PHARMACOLOGIC THERAPY

- ▶ **DOXAPRAM HYDROCHLORIDES** : is another agent stimulating ventilation in exacerbation of COPD. However, adverse effects including muscle spasm, agitation and seizures occur frequently, the drug is available only in i/v form & use is not recommended for more than two consecutive hours. Thus, it has no role in therapy of CRF.

OTHER MEASURES

- ▶ **BRONCHOSCOPY** : pts with RF who have excessive lung secretions are sometimes helped by fiberoptic bronchoscopy. It is useful for placing or removing endotracheal tubes, removing foreign body from lung and collecting tissue samples for diagnosis.
- ▶ **IV NUTRITIONAL SUPPORT**: is essential to maintain or restore strength when weakness and loss of muscle mass prevents pts from breathing adequately without ventilatory support.

OTHER MEASURES

- ▶ **PHYSIOTHERAPY:** includes chest percussion, suction of airways and regular changes of body position . It helps drains secretions, maintains alveolar inflation and prevents atelectasis, incomplete expansion of the lungs.
- ▶ **X-RAY MONITORING :** x-ray images of the chest helps the doctor monitor the progress of lung and heart ds in respi failure. The portable chest radiograph taken with an x-ray machine brought to the bedside is often used for this purpose in ICU.

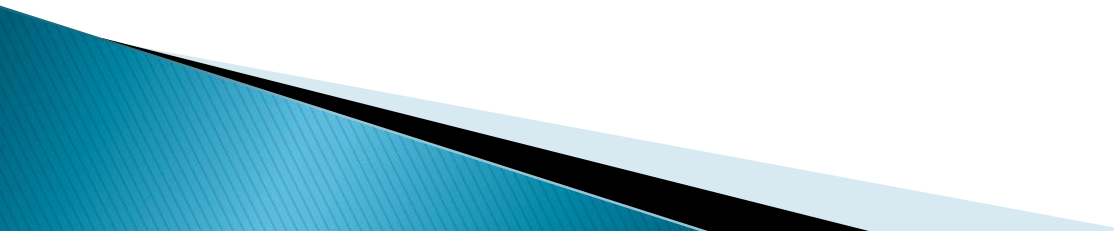
NEWER ADVANCES

- ▶ **EXTRACORPOREAL MEMBRANE OXYGENATOR :**
ECMO is essentially an artificial lung. It is an appropriately cased artificial memb which is attached to the patient externally(extracorporeally) th' an artery or vein. Although the best substitute for a diseased lung that cannot handle gas exchange adequately is a healthy human lung. Bt such substitution is often not possible. Circulating the pts blood th' the ECMO offers another approach. Gas exchange using the ECMO keeps the pt alive while the damaged lungs have chance to heal.

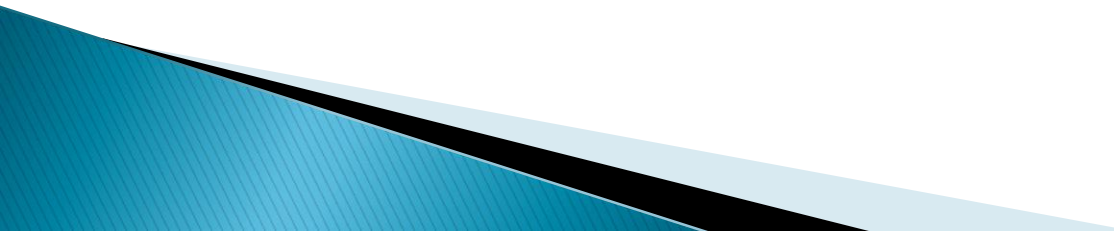
COMPLICATIONS OF TREATMENT

- ▶ Oxygen toxicity
- ▶ Pulmonary embolism
- ▶ Cardiovascular problems
- ▶ Barotrauma
- ▶ Pneumothorax
- ▶ Gastrointestinal bleeding

They result from fluid overload, mechanical ventilation, PEEP, and other procedures used in management of RF.



POTENTIAL COMPLICATIONS OF CRF

- ▶ HEART FAILURE
 - ▶ MYOCARDIAL INFARCTION
 - ▶ ORGAN FAILURE OR DYSFUNCTION
 - ▶ PNEUMONIA
 - ▶ RESPIRATORY ARREST
 - ▶ SHOCK
- 

PREVENTION

- ▶ Because RF is not a disease itself; but the end result of many disorders; the best prevention is to prevent or treat the underlying disease