RESPIRATORY INSUFFICIENCY

Acute respiratory failure (ARF) usually manifests in an older age population with primary obstructive or restrictive disease and a younger, previously healthy population who have suffered an insult resulting in secondary lung injury and dysfunction known as the adult respiratory distress syndrome (ARDS).

THE COMPONENTS OF ARF:
I. The patient is acutely dyspneic;
II. The pO2 is less than 50mmHg;
III. The pCO2 is greater than 50mmHg
IV. The arterial pH shows significant acidemia.
   At least two of the following components are present.

CLASSIFICATION OF ARF HAS BEEN PROPOSED:
A. Type I. – hypoxemia with eucapnia or hypocapnia;
B. Type II – Hypoxemia and hypercapnia.

CAUSES OF ARF:
I. BRAIN:
   1. Bulbar poliomyelitis;
   2. Overdose;
   3. Central alveolar hypoventilation syndrome.
II. SPINAL CORD:
   1. Guillan-Barre syndrome;
   2. Spinal cord trauma;
   3. Polio.
III. NEUROMUSCULAR STRUCTURES:
   1. Myasthenia gravis;
   2. Tetanus;
   3. Drug blockade;
   4. Botulism;
   5. Organic phosphate insecticide.
IV. THORAX:
   1. Kyphoscoliosis;
   2. Massive obesity;
   3. Muscular dystrophy;
   4. Flail chest;
   5. Rheumatoid spondylitis.
V. UPPER AND LOWER AIRWAYS:
   1. Obstructive sleep apnea;
   2. Vocal cord paralysis;
   3. Tracheal obstruction;
   4. Epiglottitis/laryngotracheitis;
   5. Large tonsils and adenoids;
   6. Postintubation laryngeal edema.
VI. CARDIOVASCULAR AND THROMBOEMBOLIC:
1. Cardiogenic pulmonary edema;
2. Pulmonary embolism.

VII. ALVEOLI AND LOWER AIRWAYS:
1. COPD;
2. Asthma;
3. Cystic Fibrosis;
4. Bronchiolitis;
5. ARDS;
6. Interstitial lung disease.

CLINICAL FEATURES
The symptoms of hypoxemia:
   a. disorientation;
   b. confusion;
   c. restlessness;
   d. impaired intellectual function.

EARLY PHYSICAL SIGNS OF HYPOXEMIA:
   a. tachypnoe;
   b. tachycardia;
   c. mild hypertension;
   d. peripheral vasoconstriction.

ARF IN COPD:
1. expiratory wheezing, use of the accessory respiratory muscles;
2. hyperresonance of the chest to percussion;
3. distant breath sounds;
4. bibasilar rales;
5. airflow obstruction during pulmonary function testing;
6. an arterial oxygen tension \( pO2 \) less than 50mmHg and/or an arterial carbon dioxide tension \( pCO2 \) greater than 50mmHg

TREATMENT
OXYGEN THERAPY:
1. nasal canula;
2. air entrainment oxygen masks (Venturi masks).

MECHANICAL VENTILATION.

OTHER TREATMENT
1. Bronchodilators;
2. Theophylline;
3. Postural drainage and chest physiotherapy;
4. Antibiotics;
5. Digitals;
6. Adequate nutritional support.
ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)

ARDS is defined in clinical terms as syndrome of acute respiratory failure characterized by hypoxemia and generalized pulmonary infiltrates in the absence of cardiac failure.

For the diagnosis of ARDS are necessary:
1. Respiratory distress;
2. Severe hypoxemia;

The overall mortality associated with ARDS is approximately 60%. Mortality is linearly related to age, increasing from 40% in the younger age-groups to 90% in patients over 65 years of age.

PHYSIOLOGICAL ABNORMALITIES:

A. Impaired gas exchange;
B. Altered lung mechanics;
C. Pulmonary vascular changes.

Hypoxemia is seen early in the course and is believed to be caused by venoarterial shunting due to alveolar flooding and collapse.

PULMONARY INFLAMMATION - a prominent feature of ARDS.

THERAPY – is directed at the correction of physiological function, especially hypoxemia and tissue hypoxia, while avoiding therapeutic complications.

I. Supplemental oxygen therapy;
II. Mechanical ventilation;
   1. Volume-controlled ventilation in either the assist/control mode (A/C);
   2. Intermittent mandatory ventilation (IMV);
   3. Positive end-expiratory pressure (PEEP).
III. Fluid management;
IV. Pharmacologic therapy (corticosteroids).

COMPLICATIONS AND PROGNOSIS

1/3 of the death in ARDS patients are related to the disease or injury, that is, death is caused by events occurring prior to the onset of ARDS.
2/3 of ARDS deaths are due to complications that have their onset either coincident with or following ARDS onset.

In survivors with previously normal lung function, the long-term prognosis for recovery appears to be remarkably good. Lung volumes and arterial blood gases have been shown to return to normal levels within 4 to 6 months after respiratory failure.
**COR PULMONARE**

*Cor pulmonare* is defined as a right ventricular dysfunction caused by lung disease. Primary diseases of the left side of the heart and congenital heart disease must be excluded.

THE NORMAL PULMONARY CIRCULATION AND PULMONARY HYPERTENSION

The pulmonary circulation is normally a high-flow, small resistance and low pressure circuit. The pulmonary vascular bed has a remarkable capacity to both regulate its vascular tone and recruit unused vessels in order to adopt to physiological changes. However, when pulmonary vascular reserve has been exhausted by progressive reduction in the extent and distensibility of the pulmonary vascular tree, even, the modest increments in pulmonary blood flow associated with daily living may suffice to elicit marked pulmonary hypertension.

There are two main mechanisms leading to the pulmonary hypertension:

A. Alveolar hypoxia
B. Limitation of the pulmonary vascular bed.

Alveolar and arterial hypoxia and/or hypercapnic acidosis produce functional vasoconstriction that is apparently sufficient to elicit chronic pulmonary hypertension and cor pulmonale. This phenomenon is known as hypoxic pulmonary vasoconstriction – mechanism Von Eulera and Liljestranda. The mechanism responsible for hypoxic pulmonary vasoconstriction remains unclarified, although it has been suggested, that vasoactive substances (histamine, serotonin, angiotensin II) may be responsible for the hypoxic pressor response.

Classification has been proposed:

I. Acute cor pulmonale- reserve for the dilatation of the right side of the heart which follows acute embolization of the lung.

II. Chronic cor pulmonale- is judged by the type and duration of the respiratory disorder that led to the cardiac enlargement.

Stages of chronic cor pulmonare:

A. COR PULMONALE OCCULTUM:
   1. Normal pressure in pulmonary artery in rest.
   2. Pressure increase during exercise > 4kPa (30mmHg).
   3. No symptoms in clinical examination.

B. COR PULMONALE COMPENSATUM:
   1. Middle pressure higher than 2,5kPa (20mmHg) in rest.
   2. Clinical symptoms in examination.
   3. Pulmonary hypertension and enlargement of the right ventricle.

C. COR PULMONARE DECOMPENSATUM:
   1. Pulmonary hypertension.
   2. Respiratory and circulatory failure
   3. Left and right ventricle failure.
CLASSIFICATION OF THE DISEASES LEADING TO CHRONIC COR PULMONALE

I. Disorders leading to alveolar hypoxia:

A. Diseases with the diffuse bronchus constriction:
   1. Chronic bronchitis.
   2. Emphysema.
   3. Bronchial asthma.
   4. Cystic fibrosis.

B. Interstitial lung diseases:
   1. Inorganic dusts (asbestosis, silicosis, coal worker’s pneumoconiosis, berylliosis).
      Organic dusts (cotton dust, grain dust, farmer’s lung).
   2. Fibrosis of unknown reason.
   4. Tuberculosis.
   5. Sarcoidosis.
   6. Collagenosis.
   7. Lung neoplasms.

C. Disorders of chest bellows.
   8. Neuromuscular respiration disorders.
   10. Kyphoscoliosis.

II. Disorders leading to restriction and stiffening of pulmonary vascular tree.
   1. Primary pulmonary hypertension.
   2. Recurrent pulmonary emboli.
   3. Pulmonary vasculitis.
   4. Schistosomiasis.

CLINICAL FEATURES

The signs and symptoms are usually nonspecific, due to primary pulmonary diseases. Central cyanosis is often present. Exertional dyspnea, tachypnea, chest pain and lightheadedness are common and are related to the increased right ventricular work load and the pulmonary vascular limitation. The other symptoms – syncope, pedal edema, ascites, hemoptysis, hoarseness, Raynoud’s phenomenon.

PHYSICAL EXAMINATION.

   2. Parasternal lift.
   3. Prominent subxiphoid cardiac impulse.
   4. Tender hepatomegaly.
On auscultation:
   1. Pulmonic component of the second heart sound.
   2. Pulmonic ejection click.
   3. Third and fourth heart sounds heard at the left parasternal region.
   4. The murmur of tricuspid insufficiency.
   5. Graham Steell murmur of pulmonic insufficiency.
II. Disorders of the central nervous system.
   Hypoxemia- tremor-hypersomnia-coma.

III. “P pulmonale” in ECG.

IV. Catheterization of the pulmonary artery.
   This is the only examination for sure diagnosis of cor pulmonale.

V. X-ray examination.
   Assessment of enlargement of the right ventricle.

VI. Pulmonary function testing.
   Obstruction, restriction or mixed disorders.

VII. Arterial blood gas.

VIII. Biochemical investigation.

IX. Haematological investigation (polycythaemia, DIC).

TREATMENT OF THE COR PULMONALE DECOMPENSATUM.

Therapy should be directed towards addressing the factors responsible, if possible.

A. Casual treatment – avoid bacterial or viruses infection (antibiotics).

B. Symptomatic treatment – supplemental oxygen therapy.
   Long – term oxygen therapy reduces the frequency of hospitalization in respiratory failure.

   1. Bronchopulmonary drainage.
   2. Orally administered expectorants or aerosol delivery of mucolytic agents.
   5. Glucocorticosteroids.
   6. Diuretics.
   7. Cardiac glycosides.
   8. Phlebotomy.
   10. Vasodilators – the use of vasodilators is controversial. The good solution may be a combination of oxygen supplementation and vasodilator drugs.

PROGNOSIS.

The presence of coexistent pulmonary hypertension in the setting of chronic respiratory diseases is a poor prognostic sign, with death resulting from either respiratory failure or combined cardiopulmonary decompensation commonly occurring within three to five years.