Support to the Patient with Respiratory Failure

Mimoza Canga MD PhD¹, Vito Antonio Malagnino MD DDS²

¹University of Medicine Tirana, Albania 9400

²Sapienza University of Rome, 00185 Italy

Abstract: In Italy respiratory diseases are the third leading cause of death after cancer and cardiovascular disease. In fact, every year in Italy: a person in 100 is hospitalized for a disease that affects the respiratory system, a person in 1,000 people die of respiratory diseases, lung cancer, chronic bronchitis, bronchial asthma and pulmonary emphysema. Not less than five million people are suffering from respiratory diseases. 30,000 people, due to chronic respiratory disease, live in a state of respiratory failure, this means in a so serious situation as to make them dependent on an oxygen supply. The main respiratory diseases in the world are in constant progression predominantly in relation to: raising the age in the population. Increased exposure to risk factors such as active and passive smoking, the type of work activity, air pollution, residence in industrialized countries, genetic, power quality and more. The following epidemiological data ISTAT 2000 demonstrate the importance of the social diseases of the respiratory system: 2,500,000 people suffer from diseases such as chronic bronchitis and emphysema. 1,730,000 people suffer from bronchial asthma with a clear upward trend of new cases, especially for children. Each year there are 600,000 hospital admissions due to respiratory disease. Respiratory diseases, excluding lung cancer, representing 8% of the causes of hospitalization. Respiratory diseases, excluding lung cancer, representing 8% of the causes of hospitalization[2],[15].

Keywords: ventilatoryinsufficience, respiratory failure, diagnostic tests, PSV, oxygen therapy

1. Introduction

The chronic respiratory insufficiency creates a progressively worsening disability state that limits the working abilities of the subjects and in the long term, the performance of a normal social life [46]. The socio-economic implications of this chronic pain are enormous in terms of both social security costs loss of work days, etc. and health expenditure pharmaceutical or hospitalization continuous use of drugs, recurrent hospitalizations with prolonged hospital stay and are accompanied by a progressive deterioration of the quality of life of the patient[31]. The overall mortality rate since about 2005 for chronic respiratory failure is 7%. This is best shown in the graphic.

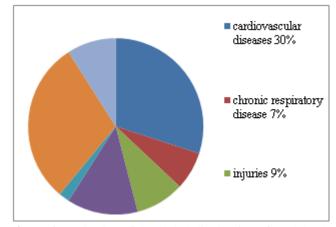


Figure 1: Projection of the global distribution of total deaths (58 million) by leading causes, 2005.

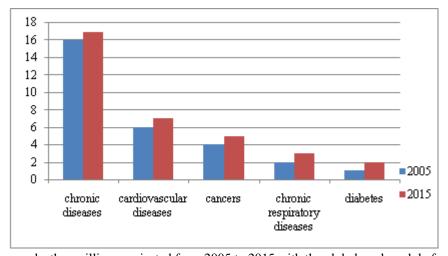


Figure 2: Chronic disease deathes millions projected from 2005 to 2015 with the global goal scedule for people younger than 70 years of age

This update is divided into two chapters, the first one speaks of the respiratory system in general, outline of anatomy, physiology and function of the respiratory system. An important part occupied also the factors that alter respiratory

Paper ID: SUB151366

function, which are smoking, air pollution and allergens. But the second chapter speaks of respiratory failure, its definition, respiratory pathophysiology and classification of respiratory failure in ventilatory failure, respiratory failure

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

by alterations in ventilation-perfusion, respiratory failure from prevalent alterations in alveolar-capillary diffusion, the blood-circulatory insufficiency and intrapulmonary shunt [13],[34]. In the last part we talk about the treatment of respiratory failure, oxygen therapy, invasive and non-invasive mechanical ventilation, the side effects of the noninvasive and the VM, as well as nursing care of the patient with respiratory failure [8].

1.1 The Respiratory System

The lungs are the two main organs of respiration. They are located in the thoracic cavity and have the ability to expand and relax according to the movements of the rib cage and diaphragm [35]. They are the place where the exchange of respiratory gases occurs and can be considered as a collection of small thin-walled saccular compartments that must at least partially be filled with air [44]. This occurs thanks to the rigidity of the chest wall that is able to oppose the natural tendency to collapse part of the lungs. This results in a slight negative pressure between the external lung surface and the internal wall of the chest. A thin layer of fluid covers such surfaces, or pleural membranes, to allow movement of the lungs within the chest cavity.

1.2 Division of the Respiratory Tract

The respiratory route is the route that fulfills the inspired air from the mouth and nose to the pulmonary region where gas exchange takes place. The airways are the conduction system of the lungs, because their function is to distribute the inspired air to the lung regions where gas exchange takes place. The respiratory tract, starting from the nasal nostrils include, in order pharynx, larynx, trachea, bronchi and bronchioles until most of the terminal bronchioles.

1.3 Anatomical Divisions of the Respiratory Tract

An alternative description of the respiratory system is based on anatomical and clinical considerations:

The upper respiratory tract, which includes the external nose,nasal cavity and paranasal sinuses and pharynx. The lower respiratory tract consists of the larynx, trachea, bronchi and intrapulmonary portion of the bronchial tree. The respiratory system has a complex structure designed for that which is the basic function of the lungs: the exchange of carbon dioxide between air of the alveolar cavity and blood of the pulmonary capillaries. Upper respiratory tract, the trachea and bronchi allow the transport of air from the outside to the alveolar region and vice versa, the alveolar capillary structures promote gas exchange, pulmonary circulation allows the transport of dissolved gases in the blood [62].

1.3.1 Bronchi and lungs

Paper ID: SUB151366

The routes start from the nose and oropharynx continuing with the larynx, trachea, and bronchi. The two left and right bronchi are divided into lobar bronchi or second order; these, in turn, in the segmental bronchi or third-order destined for areas or lung segments; they follow in the sub segmental bronchi or bronchioles that the fourth order, and

these are divided into further splitting lobular, terminals and breathing. The last ones already characterized by the presence of alveolar protrusions, continue in the alveolar ducts, in the courts, in the alveolar sacs, alveoli. The structure of the bronchial wall changes as one moves from the trachea to the distal bronchi. In the bronchi of greater caliber, up to the bronchi of the diameter of about 1 mm, are present incomplete rings and then plaques cartilage intended to ensure a certain rigidity to the wall, so as to allow the patency of the lumen during the change of pressure caused by the ventilation or by partial obstructive phenomens. Moreover, together with the discontinuity of the cartilages, the presence of muscle fibers and elastic fibers allows considerable variations of the bronchial lumen. The epithelium that lines the wall up to the bronchioles is pseudo-stratified, cylindrical, and ciliated and goblet cells and contains numerous mucous glands. The cilia of the epithelial cells are of great importance to dismiss out all the organic and inorganic elements which can reach up to that level; mucus, secreted not only by the mucous glands, but probably also from the cells of the more distal terminal bronchioles, also has a protective function. In the respiratory bronchioles with thinner epithelium and low-secreting cells begin toestroflettersisomealveoli and that's why they can already demonstrate the function of gas exchange; from each of these respiratory bronchioles originate three alveolar ducts, in which the wall is almost completely provided with alveoles; each alveolar duct results in some rounded cavities, the athers, in open communication with the alveolar sacs; formations from which protrude numerous alveoli. The structure of the alveolar wall is still under discussion especially with regard to the continuity of the epithelium coating, whose total surface area was evaluated in 60-100 sqm. Therefore, recent electron microscopy images seem to confirm the existence of this thin continuous epithelial layer, which has an average thickness of 0.2 microns, which increases considerably at the level of the nucleus, which, as a rule, is the only structure clearly recognizable by light microscopy. It is known that even in the alveolar walls are present constant solutions of diameter of several microns, so-called pores of Kohn, which allow the passage of air and as it can collect in a cavity in the alveoli alveolar neighbors, even if it originated from different bronchioles, so as to allow a kind of collateral ventilation. Diagram that describes the respiratory system of a human being [62].

1.3.2 Respiratory Physiology

The respiratory system is constituted by a series of organs that operate to allow the exchange of gases between the atmosphere and the blood. Through the processes of ventilation (the transport of gas from the atmosphere to the alveoli of the lungs) and diffusion (gaseous exchange between alveoli and pulmonary capillaries), the respiratory structures work to ensure the maintenance of homeostasis [44]. Forming an interface interposed between the external and internal environment, respiratory system intervenes to meet all the basic needs of cells:

 Oxygen. The oxygen content in the inspired air diffuses from the alveoli in normal conditions the pulmonary capillaries, and then to be transported to the cells throughout the body via the blood.

- 2) Utilization of nutrients. The cellular metabolism is maintained in accordance with the availability of essential oxygen to the aerobic catabolism.
- 3) Elimination. The carbon dioxide produced by cellular metabolism diffuses in normal conditiones from the pulmonary capillaries into the alveoli, from which it is then eliminated by exhalation.
- 4) Acid-base balance. The carbon dioxide reacts chemically with the water contained in the blood, affects the plasma levels of carbonic acid, thus affecting the acid-base balance[10],[47][50].

1.4 Respiratory Patho-Physiology

The body, as a dissipative structure capable of maintaining stable over time its morpho-functional organization, requires a continuous input of free energy that is derived primarily from the oxidation of nutrients directly or indirectly from the external environment. This involves, on the one hand, a continuous supply of oxygen concentration at the sites where respirators must never fall below a certain level, on the other hand, an adequate supply of oxidizable nutrients that must be properly allocated to storage sites in order to ensure a sufficient power generation during periods of fasting or emergency. Oxygen is obtained from the environment through the pulmonary ventilation and is carried by the blood to the cells. Nutrients are taken from the environment with the power supply, degraded molecules used in the processes of digestion, absorbed in the gastrointestinal tract and transported by the blood to the tissues where they are oxidized or stored. Oxygen supply and nutritional status must be kept within compatible limits with a regular supply of energy by the body. In agreement, both the set of processes that keeps the concentration of nutrients into the body fluids at an appropriate level are subject to regulation by complex homeostatic networks that coordinate the activity of a large number of organs and systems [44]. The respiratory essentioal function is to ensure a sufficient oxygenation of the arterial blood and remove a corresponding quantity of carbon dioxide [62]. Respiratory function is implemented through the succession of several stages which consist of:

- a) Pulmonary ventilation
- b) Distribution and mixing of gases (O2 and CO2)
- c) Dissemination of alveolar-capillary gas
- d) Supply of blood in the lung.

Paper ID: SUB151366

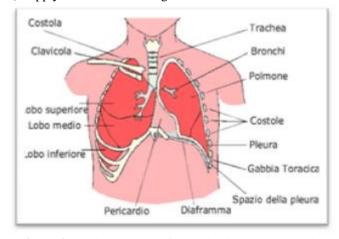


Figure 3: Partial pressure of gases in air environment, in alveola and blood.

1.4.1. The pulmonary ventilation

Pulmonary ventilation consists in the passage of the gas from the atmosphere to the alveoli and in the opposite direction. The neuro-humoral regulation of breathing takes place in the reticular substance of the lower third part of the bulb and the bridge, where reside the inspiratory and expiratory centers, the pneumotaxic center, the apneustic center and lower expiratory medullary centers and gasping. These centers are integrated and correlated to each other with neuronal connections, which regulate the activity of the respiratory muscles and control the respiratory volume / modifying the frequency and respirazione. They are in turn governed by a set of physical stimulus, chemical and nervous, that act on peripheral receptors or directly at the central level, on the reticular substance (fig. 2). The ventilation is generated by the clinical and rhythmic movement of structures that make up the wall of the chest cavity and the lung parenchyma, structures such as the thoracic vertebrae, twelve pairs of ribs, sternum and muscle structures, such as the diaphragm, the intercostal muscles and the accessories muscles [58], [60-61]. The transit of the air from the atmosphere within the lungs and from these to the outside, is possible thanks to the existence of a gradient pressure, that is, a difference pressure. The breath is composed of two phases: inhalation and exhalation [34]. During the inspiratory phase, occurs the contraction of the muscles dedicated to respiration, namely the diaphragm, the external intercostal, small pectoral and sternocleidomastoid; contraction determines the increase of the size of the chest, consequently case the decrease of the intrapleural and intralveolar pressure, then the air inlet, from the outside, into the lungs [58], [62].

	atmospheric Air	polmonary Alveola	Arterious blood	Venous blood
P O ₂ mmHg	159	100	92	35
P CO ₂ mmHg	0.3	40	40	46
P H ₂ O mmHg	0	47	47	47
P N ₂ mmHg	600	570	573	573
P totale mmHg	760	760	755	706

Figure 4: The quiet breathing at rest, involves an active movement, inspiration, during which the diaphragm lowers and the external muscles, cause the expansion of the chest cavity. At the end of inspiration, the external intercostal muscles and the diaphragm are released, and we have the passive expiration.



Figure 5: Inspiration and expiration

ISSN (Online): 2319-7064

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

During the expiratory phase, is necessary the relaxation of the diaphragm, the contraction of the abdominal and internal intercostal muscles, this leads to a reduction in the volume of the chest cavity, to which follows the increase in intrapleural pressure and intralveolare, which rises from about -3 mmHg, about +3 or +4 mmHg, leading to a positive pressure, then the leakage of air from the lungs [34]. It is necessary to emphasize that the intrapleural pressure is normally lower than atmospheric, this condition is essential to prevent the tendency of the lung to collapse [62]. Only a portion of the air, potentially contained in the airways, participates in gas exchange, becomes essential in order to facilitate breathing in people with respiratory failure, understand the lung volumes and capacities that are described below.

- Total lung capacity (CPT) is the volume of air in the lungs after forced inhalation; is the sum of all volumes of the lung, is 5700ml.
- Total volume (VT, VC) is the volume of air that can be inhaled or exhaled during each breath, corresponds to about 500ml.
- Inspiratory reserve volume (VRI) is the volume of air that can be inhaled, with forced inspiration, at the end of the inhalation of a VC to reach the CPT, corresponds to approximately 3000ml.
- Expiratory reserve volume (VRE) is the volume of air that can be exhaled with forced expiration at the end of exhalation of a VC, is approximately 1100ml.
- Residual volume (VR) is the volume of air remaining in the lungs after exhalation of a VRE, the value is approximately 1200ml.
- Capacity residual pulmonary (CFR) is the sum of VR and VRE, corresponds to the volume of air that remains in the lung following exhalation of a VC, and is 2300ml.

1.4.2. The distribution

The air distribution in ventilated lung is not uniform, so that distribution of a tracer in the <<multicompartment>>. The emptying of the alveoli is also asynchronous, so that we can distinguish slow and rapid alveolar ventilation. The inflow and outflow of gases in the lung are conditioned, in fact, by the phenomenon of mixing between the air coming from the atmosphere and the gas staying in the lungs. Especially in some morbid situations the mixing of gases in the lung occurs in an irregular manner to the early closure of gaps lung zones. It is also important to note that not all the air from the lungs ventilated participates in the respiratory exchange, part of it, in fact, is to fill-in-the normal large airway or - in pathological conditions - some areas not supplying the pulmonary blood flow: this is the socalled << dead space >> of the lung[24].

1.4.3. The alveolar -capillary diffusion

Paper ID: SUB151366

The alveolar - capillary diffusion of a gas involves passing a series of obstacles; a) the alveolar surfactant; b) the alveolar epithelium; c) the interstitium; d) the vascular endothelium; e) the plasma; f) the erythrocyte membrane. The passage of a gas from the lungs to the blood, however, depends-in addition to these obstacles-also; g) by mixing alveolar (ie, the distribution and exchange of air in the alveoli); h) from the distribution of perfusion [11].

1.4.4. The pulmonary circulation

The pulmonary circulation is characterized - in terms of hemodynamic-by: a low-pressureregime, a low level of resistance to blood flow, a high capacity system. The degree of pulmonary perfusion and perfusion distribution are influenced by: 1) the blood pressure in the pulmonary artery; 2) the pressure in the pulmonary veins; 3) from the colloidal osmotic pressure of the blood; 4) from the respectively inspiratory and expiratory phase of the lung; 5) from the patient's decubitus amending the hydrostatic pressure of the pulmonary capillaries. Various factors can modify the pulmonary circulation:

- a) The hypoxia that causes vasoconstriction of the small vessels with increased headache precapillary pulmonary artery.
- b) Hypercapnia and acidosis acting erratically: mostly causing vasoconstriction.
- c) The changes in dynamic lung-thorax district that induce alterations in intrathoracic pressures in different respiratory phases.
- d) The anatomical alterations of the pulmonary vascular network with variations of resistance.
- e) The first three factors interfere especially in acute respiratory failure; all four in chronic respiratory insufficiency [54].

1.5. The factors that may alterate the lungs function

1.5.1. Smoking

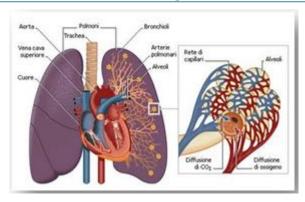
People who have the habit of smoking, have an increased respiratory rate, compared to non-smokers. There habit of cigarette smoking exposes the person to greater risk of damage to the respiratory, than non-smokers. The smoke causes the decrease of the movement of the cilia of the respiratory mucosa and the increased production of mucus; this situation makes it difficult to release from the mucus in the airways, exposing the subject to a greater risk of infection. In addition, smoking increases the risk of lung cancer, cardiovascular disease, Emphysema and bronchitis [2], [62].

1.5.2. Atmosphere

The contaminants present in the atmosphere, originating from the exhaust pipes of cars and industries, are responsible for direct lung damage and increased mucus production and can lead to diseases such as bronchitis or bronchial asthma. Carbon monoxide, present in the fumes, emitted from the exhaust pipes of the cars, has an affinity with the hemoglobin 200 times higher than that hemoglobin has with oxygen, and then binds to the iron atoms of 'hemoglobin of the blood and form a more stable complex oxyhemoglobin, the percentage of oxygen in the atmosphere is about 21%. The partial pressure of oxygen decreases in relation to the increase of the altitude, hence also the subjects not suffering from respiratory diseases may experience dyspnea or intolerance of, at high altitudes [46].

ISSN (Online): 2319-7064

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438



1.5.3. Allergens

These elements, such as pollens, certain types of plants, mites, dust present in the environment, or food substances, which if inhaled or swallowed, trigger an inflammatory response. The events, due to the release of mediators of inflammation bugs are: stinging the eyes, the leakage of runny-nosed, edema of the mucosa, typical signs of hay fever, increased mucus production, edema of the bronchioles and bronchospasm, characteristic signs of allergic asthma.

1.5.4. Deficit lung expansion

There may be present situations or diseases of the respiratory system determinants alterations that occur with the stiffening of the lungs, which have difficulties to expand fully and their alveoli may collapse (atelectasis), with edema and thickening of the lung parenchyma and, finally, with the reduction of the movements of the rib cage. Such situations are defined as restrictive lung disease and cause a considerable increase in the demand for oxygen by the respiratory muscles [62-63].

1.5.5. Occlusion of the respiratory tract

All situations that cause obstruction of the airways, making breathing difficult, because the air has to cross paths with a smaller diameter. The causes of airway obstruction are numerous and can be divided into exogenous factors, such as irritants physical or chemical and inhaled foreign bodies, endogenous factors, such as the excessive production of mucus and edema, which are present in asthma, bronchitis and cystic fibrosis. High endogenous cause is represented by the growth of new tissue, which occurs in the case of lung cancer [62-63].

1.5.6. Body posture

In situations in which the diaphragm is able to move downward and upward, freely, breathing is more effective. Respiratory function is made easier when the person takes standing or sitting positions, which facilitate the expansion of the chest, while the reclining position makes it more difficult to breathe, because the diaphragm is compressed by the abdominal organs.

1.5.7. Nutrition

Paper ID: SUB151366

The efficiency of the respiratory muscles is ensured by a proper intake of calories and protein. An adequate protein intake also promotes the production of hemoglobin and plasma proteins; low hemoglobin levels reduce tissue oxygenation and muscle strength. Proper nutrition is the energy requirements needed to maintain the efficiency of the immune system and prevent respiratory infections, most

frequently affecting the malnourished subjects. Obesity causes a limitation of diaphragmatic movement, making breathing more difficult, causing shortness of breath and tachypnea [3].

2. Respiratory Failure, Definition

The disorders of the ventilation or lung diffusion may give rise to physiological stress of varying severity. If the disease is characterized by a decrease in Pa O2 (partial pressure of O2 in the arterial blood) or by an increase in Pa CO2 (partial pressure of CO2 in arterial blood) in the course of physical effort, being in state of respiratory imbalance. When dysfunction becomes more severe and interferes with the cellular oxygen supply or with the elimination of carbon dioxide even in conditions of rest, it is then present respiratory failure. The respiratory insufficiency is a condition characterized by an altered partial pressure of gas (O2 and CO2) in arterial blood. The criteria for making a diagnosis of IR are an arterial partial pressure of oxygen <60 mmHg while breathing in room air, said hypoxia, whether or not accompanied by hypercapnia which is the arterial partial pressure of carbon dioxide> 45 mmHg.

In clinical practice we speak of respiratory failure when the PaO2 is less than 60 mmHg and Pa CO2 exceeds 45 mmHg (at sea level). When the only detectable data is hypoxemia, the respiratory failure is partial; and when hypercapniaispresent, the respiratory failure and total. We talk of overt respiratory failure when it is evident at rest; if it only occurs after a mild exercise, it is called latent respiratory failure. And this can be caused by an alteration which is realized at the level of one or more organs involved in the complex mechanism of breathing.

2.1 Evolution of Respiratory Failure

In the early stages of respiratory illness, alterations in blood levels of gas are mostly seen during physical activity. In times of normal activity, homeostasis can be maintained by compensatory mechanisms cardiovascular or respiratory diseases. It establishes the existence of an imbalance when respiratory adaptation, represented by an increase in perfusion. and still is effective counterbalancing the effects of illness. The imbalance is often associated with disorders such as emphysema, asthma, bronchitis, and pneumoconiosis [52]. If the dysfunction becomes more acute or develop further complications, often compensatory mechanisms fail to maintain homeostasis. Respiratory failure technically states when PaO2 falls below 50 mm Hg or in PaCO2 reaches or exceeds 50 mm Hg. The failure can be induced by any pathology of the ventilation or diffusion. Although the levels of oxygen and carbon dioxide, respectively, lower or higher, are characteristic of respiratory failure, it is not said that these changes occur simultaneously [62]. The deviations of normality largely dependent on the nature of the disorder from which they originate. It is possible, for example, detect the state of hypoxemia without hypercapnia. In these cases, the decrease in Pa O2 is due to diseases affecting the respiratory membrane or a non-uniform ventilation. Specific disorders include pulmonary fibrosis, pneumonia, and tuberculosis. Hypoxemia and hypercapnia, however, is frequently

ISSN (Online): 2319-7064

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

observed in cases of uniform hypoventilation associated with disorders of nervous origin, malformations of the rib cage and chronic obstructive pulmonary disease. When the respiratory disease concludes with pulmonary high blood pressure, the resistance to the expulsion of the blood from the right ventricle may lead to right heart failure. The resulting heart condition called pulmonary venous congestion onset is characterized by symptoms ranging from neck veins peripheral edema. To further complicate the clinical picture is involved the tendency of pulmonary arterioles in response to force himself to hypoxemia or hypercapnia or both. When the defect is established, respiratory reflex vasoconstriction in the lungs causes a typical increase in pulmonary blood pressure [54]

RESPIRATORY FAILURE

Parzial: hypoxemia (Pa O₂< 60mm Hg)

Total: hypoxemia e hypercapnia (PaCO₂> 49mm Hg)

PATHOGENESIS:

a) Ventilatory failure

b)b) alveolar-capillary Insufficiency

c)c) Circulatory failure

d)d) peripheric metabolic Insufficiency

e)e) Altered composition of air

RESPIRATORY FAILURE ENTITY:

Grade 1: mild hypoxemia after exercise

2nd grade: hypoxemia at rest

3rd grade: hypoxemia and hypercapnia

4th grade: coma

Figure 6: Respiratory failure can be caused by a damage which intervenes at the level of any link in the chain.

Depending on the mode of onset respiratory failure is divided into:

• Acute Respiratory Failure

Usually the most severe form, where it assumes a lot of importance the rapidity with which manifests respiratory failure, such as a high rate of abnormal values can manifest failure, even if the values themselves remain in norm. It is characterized by hypoxemia and without increase of carbon dioxide in arterial blood and is caused by alterations in the ventilation / perfusion ratio, shunt and impaired circulation. It is typical for diseases involving the lung parenchyma [62].

• Chronic Respiratory Failure

It occurs more slowly, and there is a more severe form called "riacutization of chronic respiratory insufficiency," at a fast rapid increase in Pa CO2 during the chronic form. It characterized by hypoxemia and hypercapnia, there is a severe alveolar hypoxentilation, as it defers respiratory insufficiency due to COPD in an advanced state of evolution, progressive damage to the respiratory function, or by superimposing an acute bronchopulmonary [62]

2.2 Pathophysiology of respiratory failure

The pathogenic mechanisms of respiratory insufficiency can affect the different phases of respiration; Therefore, respiratory failure may be produced by: a) ventilatory failure; b) impaired alveolar-capillary; c) respiratory failure by alterations in ventilation-perfusion d) respiratory failure by prevalent alterations in alveolar-capillary diffusion and blood-circulatory insufficiency; f) oxygen) composition of the air with peripherals metabolic abnormalities g) Intrapulmonary veno-arterial shunt. These different mechanisms may be responsible of respiratory failure alone, or may be present simultaneously in different size and each of them, in turn, can recognize different causes. In relation to the size of the induced alterations in blood chemistry from respiratory failure, we can distinguish different degrees,

that not raraely, happen in time [62]. A first degree of latent or compensated respiratory failure, is characterized by the appearance of hypoxemia only after mild exercise. A second degree of respiratory failure occurs, is also characterized by the persistence of hypoxemia during sleep. The third stage is characterized by the addition of hypoxemia hypercapnia. In the fourth grade, there is also respiratory acidosis. The fifth grade, sometimes terminal, is characterized by hypercapniccoma [20].

2.2.1 Ventilatory Insufficience

So that the ventilation process can take place as a rule certain conditions are necessary: that the airway to be patency, that the motility of the chest to be normal, the normal alveolar cavities to be extended and freely pervious to the ebb and flow of air. Any alteration of any of these conditions may result in an impairment of alveolar ventilation and this can be severe enough to lead to respiratory failure. Schematically, we can distinguish the types of respiratory failure who are also present at the same time in the same subject: restrictivventilatory failure, obstructive airway disease, depression of the respiratory centers or anatomical and functional damage of the respiratory centers, abnormal nerve conduction or neuromuscular transmission and also for limiting the movement of the chest.Restrictiv respiratory failure means the decrease in ventilation caused by a <<restriction >> or anatomical - functional decrease of thoraco - lung complex that is manifested by a decrease in running respiratory surface [1]. The causes that can determine an insufficiency of this type are different: a) anatomical or functional injury of the nerve centers or air breathing capable of preventing a normal motility of the chest and what can be observed as a result of head trauma, such encephalopathies of various nature, such poisonings to barbiturates or oppiacci, or polio, to radicoloneuriti, etc. b) lesions of the bones or muscles of the chest trauma, bone disease, arthropathy, myopathy, etc. c) marked obesity, such as to inhibit the motility of the rib cage; d) pleural and mediastinal disorders such as abundantversaments, pachipleurite, pneumomediastinum.; such as to cause a compression of the lung parenchyma. e) reduction of the lung parenchyma, caused by bronchopneumonia[34] ,[56].By obstructive respiratory failure is intended the alteration of the ventilation caused by an obstruction at the level of

ISSN (Online): 2319-7064

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

pharyngo-bronchiolar routes that, hindering primarily exhalation, due to the small forces involved in this phase of respiration, favor the onset of chronic obstructive pulmonary emphysema [57]. Among the causes of this obstruction we can remember, next to the paralysis of the vocal cords, and the obstructive laringopathy and tracheopath, y) bronchitis, bronchial asthma, bronchial tumors, endobronchial foreign bodies; but are especially chronic bronchitis the most frequent cause of obstructive ventilatoryfailure [21-22].

	Restrictive	Obstructive
Vital Capacity	Decreased	Decreased
Residual Volume	Decreased	Increased
Total lung Capacity	Decreased	Normal or increased
Maximal Ventilation	Decreased	Decreased
VEMS/ CV	Normal	Decreased
VR/CPT	Normal	Increased

The diagnosis of the type of ventilatory failure in individual cases may be sufficient some simple spirometric investigations: in the restrictive form, in fact, there is a parallel decrease in total lung capacity, vital capacity, residual volume and maximum volume of ventilation, with normal relations of FEV / CV and VR / CPT. Instead, in the obstructive there is an increase in residual volume to total lung capacity and possibly, with a marked decrease in forced expiratory volume in accordance with the ratio FEV / CV decreased and the VR / CPT is increased. The ratio of FEV / CV under obstructive ventilatory insufficiency is gradually decreasing. From values 60-70 in chronic bronchitis to 60-55 in initial respiratory insufficiency, to significantly lower values in severe emphysema. The ventilatory failure, whatever the cause that determines it, can arise and occur acutely or chronically, therefore clinically can be distinguished an acute and a chronic form. Ventilatory failure due to depression of the respiratory centers, or anatomical and functional damage of the respiratory centers: is due to overdose of sedatives or anesthetics, head trauma, intracranial hypertension, bulbar paralysis, myelitis and encephalitis, cerebral ischemia, shock, prolonged hypoxia, chronic hypercapnia , metabolic alkalosis, Pickwick syndrome (obstructive sleep apnea), or primary alveolar hypoventilation or idiopathic depression of the respiratory centers [15],[58]. Ventilatory failure due to the alteration of nerve conduction or neuromuscular transmission, with inability of the respiratory muscles, particularly the diaphragm, to support a normal dynamics of the respiratory tract by:

- Traumatic injuries of the spinal cord;
- Infectious diseases: polio, peripheral neuritis;
- Neuromuscular block: curare, succinilcolina, neurotrophic gas, botulism, nicotinic poisoning;
- Diseases of the respiratory muscles: poliomiosite. Ventilatory failure to restriction of movement of the chest
- thoracic trauma, thoracic deformities

Paper ID: SUB151366

• Ankylosing spondylitis, severe obesity.

${\bf 2.2.2}$ Respiratory failure from changes in the ventilation-perfusion

The failure of gas exchange at the alveolar - capillary, which may result in the establishment of respiratory failure, recognizes two fundamental pathogenetic causes: 1) the

alteration of the ventilation / perfusion ratio, 2) the alteration of the gaseous diffusion. The various changes in the ventilation / perfusion ratio are related to the alteration of both the ventilation and the circulation at the alveolar level [15]. They can be summarized thus: a) Non-uniform distribution of air in the alveolar region with homogeneous distribution of spraying, well being quantitatively normal global alveolar ventilation and perfusion: the relationship between alveolar ventilation and perfusion (VA / Q) is less than 0.8 is when occurs in chronic bronchitis, bronchial asthma, chronic obstructive emphysema; b) Normal spraying and no ventilation in a group almost extended of alveoli: the ratio VA / O is reduced until it is reduced to zero inatelectasisor hepatized lobe in the course of pneumonia; c) Homogeneous distribution of the air with reduction of spraying, being quantitatively normal global alveolar ventilation and perfusion: the ratio VA / Q is greater than 0.8; it is observed in chronic pulmonary thrombosis, pulmonary arteritis etc..; d) Normal alveolar ventilation and absence of spraying: the ratio VA / Q increases and reaches its highest expression in acute occlusion of a major branch of the pulmonary artery. Normal alveolar ventilation and right to left shunt with placing of venous blood into the arterial blood: the ratio VA / Q is less than 0.8 and occurs in certain congenital diseases, etc. into the lung arteriovenous aneurysm. Alterations in the gaseous diffusion are established in those diseases that alter the structure of the alveolar-capillary membrane by increasing or reducing the thickness, however, the permeability or reducing the extent as occurs in chronic obstructive emphysema to the rupture of alveolar septa, and these occur in collagen, sarcoidosis, interstitial fibrosis, tuberculosis and so on[59].

2.2.3 Respiratory failure from the prevailing changes in alveolar-capillary diffusion of oxygen

- a) For thickening alveolar-capillary <<membrane >> that can occur in:
 - Diffuse interstitial lungdesease: interstitial fibrosis, sarcoidosis, interstitial pneumonia, professional interstitial pneumopathy, collagenopathy, laryngitis carcinomatosis;
 - Diffuse alveolar lungdesease: alterations in alveolar surfactant, pulmonary edema.
- b) For reduction of the exchange of the alveolar-capillary surface

For reduction of functioning lung tissue: atelectasis, fibrosis, bronchopulmonary tumors, pleural effusion, pneumothorax, raising of the lung. The limitation of the spread is a relatively rare cause of clinically significant hypoxemia. In normal circumstances the blood flowing through the pulmonary capillary bed reaches an O2 saturation of hemoglobin almost complete in about 1/3 of the available time of exposure to the alveolar surface. Even in the presence of a limitation to the diffusion of O2, there is still a sufficient time for the O2 saturation of hemoglobin. Exceptions may occur during the exercise, with a much increased cardiac output associated with a marked reduction of the transit time of the capillary and of other units. The limitations of the spread should not be confused with pulmonary function tests of diffusing capacity. The diffusion capacity is measured by means of labeled amount of carbon monoxide and is mainly influenced by the relationships between the surfaces of blood and alveolar gas.

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

Therefore even a diffusion capacity of carbon monoxide very abnormal may not significantly affect the transport of O2. Associated hypoxemia is most often due to an imbalance between ventilation and perfusion V / Q For all practical purposes, the restriction of diffusion does not cause acute respiratory failure. When present, a limitation of the diffusion can be easily corrected by administering a mixture enriched with O2.

2.2.4 Hemato-circulation insufficience

The transport of oxygen is bound its fixation to hemoglobin and to the normality of pulmonary and systemic circulation. As is known, a small amount of oxygen is dissolved in the plasma, but most is fixed to hemoglobin. 1 gram of hemoglobin binds O2, and 1.4 ml of oxygen saturation of arterial blood is approximately 97%. This oxygen is easily transferred to the tissues whose Pa O2 is less than 40 mm Hg so that in the venous blood oxygen content decreases in the pulmonary artery and the O2 saturation in the blood is 75%. For these different operations may be fulfilled, normally, it is necessary that from the hemodynamic point of view, circulation to be normal and is included as a reduction of the circulatory flow. It is obvious that an alteration in the erythrocyte may result in a state of hypoxemia. Even the CO2 transport from peripheral tissues to the lung capillaries is in close dependence on the pressure gradient between the blood and CO2 conditions of circulation in all its districts [59].

2.2.5 Altered composition of the air and metabolic alterations devices

Alteration in the composition of the air we breathe can cause a state of hypoxemia and hypercapnia may be when there is a deficiency of oxygen, as can occur for example at high altitudes or when there is an excess of carbon dioxide as may occur in indoors or in the workplace. Are to take account also those conditions in which substances are present in the inspired air that can hinder the fixing of oxygen to hemoglobin, such as carbon monoxide which binding to hemoglobin to form carboxyhemoglobin prevents the fixation of oxygen: remember that carbon monoxide is related to hemoglobin 200 times more than oxygen[55].

2.2.6 Respiratory failure by intrapulmonary shunt

The term shunt refers to the possibility that blood from the arterial system steps in the venous circulation without crossing any region of the ventilated lung.

- 1) The areas of intrapulmonary shunt represent an episode of altered V / Q ratio in which a part of the lung is not ventilated, but continues to be perfused.
- 2) The intrapulmonary shunt can result from anatomic abnormalities such as pulmonary arteriovenous fistulas. Most commonly derived from the shunt perfusion of non-ventilated areas of the lung tissue. Pulmonary ventilation can be prevented when the alveoli are collapsed (for example atelectasis) or filled with liquid (for example, pulmonary edema or hemorrhage intralveolare).
- 3) Besides the intrapulmonary shunt, right-to-left intracardiac shunts can cause severe hypoxemia.
- D. Unlike hypoxemia caused by the altered V / Q ratio, hypoxemia due to shunt does not improve with the administration of oxygen.

2.3 Clinical view of respiratory failure

Respiratory failure, whatever its pathogenesis, is manifested by a clinical - functional picture well characterized by specific signs and symptoms. The type of breathing provides important information on the functioning of the respiratory system and on the general state of health of the patient. We define a breathing eupnea in which the frequency is normal, rhythm and regular breaks are absent. In children over 10 years and in adults, the normal frequency, in rest conditions, is 12-20 / minute; the normal frequency in the elderly is slightly higher, up to 25 breaths per minute. In newborns and infants, the respiratory rate is considered normal if 30-60 breaths per minute [24], [43].

- *Dyspnea*. It is a subjective symptom, which consists in the feeling of difficulty ventilatory and then determines the need for increased respiratory effort. Dyspnea can be classified with different severity levels: level 1, level 2, level 3, level 4, orthopnea.
- Bradypnea. Breathing frequency shorter than normal, with greater depth and with regular rhythm. It occurs physiologically during sleep, or it can be present in patients with impaired alertness and / or intake of breath depressant drugs, such as opiates.
- *Tachypnea*. Breathing frequency higher than normal and superficial. The pace may appear regular or irregular. It is often present in patients with restrictive lung disease, hypovolemic shock, and pleuritis.
- *Apnea*. Cessation of breathing. It can occur during extreme bradipnee and arrest of breathing.
- Hyperpnea. Is an increase in depth of breathing, which may occur with increased frequency or normal? It is common during exercise, in the attack of anxiety or in some cases of hypoxia.
- Cheyne Stokes respiration. Periodic breathing alternated with moments of apnea. The respiratory cycle increases, gradually, both frequency and depth, then progressively decreases, until the apnea. The alternation between the number of apnea and respiratory cycles is regular. One of the main factors for the production of these oscillations in ventilation is the delay of the feedback information to the nerve centers of the brain, relative to the effects of ventilation on the gas content in the pulmonary capillary blood. It can occur physiologically in older people during sleep, may be present in diseases that cause intracranial hypertension.
- Ataxic breath -Biot. Alternates a cycle of breaths surface, of equal depth, with a period of apnea. The alternation is irregular. May be present in meningitis and in lesions of the fossa posterior.
- Kussmaul Breathing. Typical Breath of diabetes, and nomorecompensated and induced by the decrease of blood pH (acidosis). It consists of a 'deep inspiration, followed by a short break and short exhalation, followed by another pause, with an increased frequency than normal.
- Gasping Breathing or (gasping). It is characteristic of severe cerebral hypoxia, present in patients with shock, severe reduction of cardiac output, pre-agonal stage. It consists of rapid, irregular breaths, followed by a long expiratory pause.

ISSN (Online): 2319-7064

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

- The cough. The most common symptom of lung disease, is an act of reflection, but controllable by the higher nerve centers. The irritation that triggers can be localized anywhere in the airways, from the pharynx to the terminal bronchioles. The irritation activates the bulbar centers of cough and ventilation, generates vigorous inspiratory contraction, followed by a rapid exhalation expulsion, with closure of the rima glottis; the high pressure (130-160mmHg) is free with the opening of the glottis, with speed of 121 / sec. Based on the presence or less expectoration, the cough may be productive of "sputum", or dry.
- The sputum. The characteristics of sputum may communicate important information about the disease process, the elements observed are: volume, color, texture and smell. The viscous and difficulty to expel sputum is typical of those dehydrated. The presence of yellow or green sputum, putrid odor is indicative of respiratory infection. Pulmonary edema occurs with a frothy and rosy sputum.
- The hemoptysis. It is the expectoration of frankly material blood, blood-streaked, pink or containing small clots; chronic bronchitis and cancer of the airways and lungs are common cause of hemoptysis.
- The chest pain. It is linked to inflammatory processes of the airways, such as pleuritis, or to alterations in skeletal muscle of the chest wall. Chest pain, in addition to being the result of a pulmonary disease, deteriorates the mechanical ventilatory.
- Hypoxemia. Is the reduction of the partial pressure of oxygen in the blood, is detected through a blood arterial, it is often accompanied by tachypnea, dyspnea, cyanosis, confusion.
- *Hypoxia*. It is the decrease of oxygen ratio in the tissues.
- The Hypercapia. It is the increased partial pressure of carbon dioxide in the blood, is detected through anarterialblood, it is often accompanied by tachypnea, tachycardia, lethargy, mental confusion.
- The alterations in the level of consciousness. Brain function can be impaired by altered partial pressures of respiratory gases in the blood (hypoxemia or hypercapnia), with following a lung disease.
- The alterations in respiratory rate. The respiratory rate increases with fever, sepsis, hypoxemia, hypercapnia, acidosis, anxiety, shock, pain, and with every cause which results in the increased work of breathing. A respiratory rate> 28 breaths / min. is an index of significant respiratory distress. A minor bradypnea to 8 breaths / min, or a respiratory rate less than that expected, because of pathologies such as sepsis or pulmonary edema, is a sign of urgent cardiac arrest.
- The alterations of the depth of ventilation. Are difficult
 to evaluate because the movements of the diaphragm and
 rib cage are not measurable. Ventilatory activity
 particularly blatant, possibly accompanied dall'alitamento
 of the pinna and the use of accessory muscles, is an
 indication of the effort to increase the respiratory minute
 volume and should be investigated.
- The alteration in the symmetry of ventilation. Normally, the expansion of the two hemythorax is symmetric, the asymmetry can be observed in ventilatory pleural diseases, obstruction of a main bronchus, or

Paper ID: SUB151366

- neuromuscular alterations in the rib cage, in patients with single or multiple rib fractures, or (surgical or traumatic) unilateral thoracic injury.
- The alteration of abdomen / chest synchronization. In eupnoica ventilation, chest and abdomen rise and fall synchronously; in paradoxical ventilation, the expansion of the chest during inspiration, is accompanied with the sinking of abdomen; during expiration the chest is lowered while the abdomen is projected outside.
- The difficult breathing. Consists of an intense inspiratory effort, required to ensure entry into the lungs of an air flow. The excessive negative intrathoracic pressure causes the retraction of the supra-sternal, above-clavicle, intercostal and sub-sternal regions. The difficult exhalation. It is manifested by an intense expiratory effort, required to ensure the output from the lungs of an air flow, which otherwise takes place passively. To exhale, the patient needs to raise the pressure in the lungs; expiratory accessories muscles are recruited, those of the neck, back and abdominal muscles.
- The cyanosis. It is the blueness of the skin layers and mucous membranes caused by increased concentration of reduced hemoglobin in capillary blood; cyanosis is found in the nail bed, in the oral mucous membranes, conjunctiva, nasal apex and in the earlobe; its manifestation requires at least 5mg/100ml of reduced hemoglobin in the capillaries.
- The subcutaneous emphysema. It manifests as swelling and distension of the integument, with a characteristic crackling on palpation, the presence of air in the subcutaneous planes may be due to several factors, such as the alveolar rupture in patients ventilated with positive compression and spontaneous rupture of an emphysematous bubble.
- *The posture*. Patients with respiratory distress prefer the sitting position while gripping the back of the chair. This position allows the subject to stabilize the shoulders and recruit several muscle groups at the ventilatory effort.
- The breath sounds. Can be frankly audible without the aid of tools, or audible through a stethoscope. The most characteristic are 1. Wheezing (continuous noise at high frequency and with music features, due to the reduction of the lumen), 2. Ronchi (continuous noise, high intensity and low frequency, similar to snoring, due to the production of mucus, or obstruction of the airways), 3. Rales (noise is not continuous, high-intensity explosive and bubbling, due to the production of mucus in the airway), 4. Crackles (non-continuous noise, like the rustling of hair, audible in the final phase of exhalation).
- Fingers to "drumstick" or digital clubbing (clubing): this alteration is manifested by rounding and enlargement of fingers and toes, the cause is not certain, but it seems to be due to chronic tissue hypoxia; and often present in respiratory diseases of the heart [3].

2.4 Treatment of respiratory failure

The treatment of I.R.C. provides for the use of therapeutic resources such as the long term oxygen therapy and mechanical ventilation, in order to stabilize and slow the progression of chronic disease [14],[16-19], [26-27], [48-50], [53].

ISSN (Online): 2319-7064

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

2.4.1. Noninvasive mechanical ventilation

A ventilator is a machine designed to transmit and relatively simple to apply, following a pattern set, an energy that is used to perform useful work. The energy is supplied to the fan in the form of electricity or compressed gas, and from which transmitted to increase or replace the work that the respiratory muscles of the patient must fulfill in order to support the work of breathing.

When and why to start the ventilatory treatment.

There are two reasons, the first is that the non-invasive ventilation has been and still is used mainly in acute respiratory failure in the absence of the pump. It is almost always a result of an exacerbation of a chronic lung disease such as chronic obstructive pulmonary disease, neuromuscular diseases or fibrothorax and therefore the pathogenic mechanisms that lead to its onset is slower and more gradual than the often dramatic paintings that we face in the classical renal parenchyma.

The second reason that may standardize the use in acute of this type of ventilation is related to the fact that it, being proposed only recently as an alternative to the classical intubation, due to follow patterns methodological rather rigid. The table shows the parameters used for NIMV.

When to start the non-invasive ventilation

- Respiratory rate> 35 breaths / min
- SaO2 <88% oxygen
- Sudden Increase PaCO2> 15,20 mmHg
- pH <7,35

clinical signs of respiratory "distess"

- (Dyspnea-cyanosis, paradoxical abdominal motion)
- alteration of sensorium (excluding coma)

Figure 7: Scheme on the main indications for the start of non-invasive ventilationthe noninvasive mechanical ventilation has been developed in order to prevent complications related to the use of invasive mechanical ventilation (MV), while ensuring, a similar degree of effectiveness. The efficacy of NIV in both positive pressure mode and negative pressure, depends on close clinical monitoring of the patient by well-trained nursing staff in the use of these ventilatory techniques and with extensive experience in this kind of patients. The success rate reported with NIV with positive pressure range from 51 to 91%. The short-and long-term survival is higher than with medical therapy alone and those reported with the VM-invasive. Physiological studies suggest that mechanical ventilation negative pressure is able to improve the pattern of breathing, arterial blood gases and decrease the workload of the respiratory muscles, thereby fulfilling the objectives of mechanical ventilation.

The main goals of mechanical ventilation are:

- Increase alveolar ventilation in order to remove the excess CO2 and correct respiratory acidosis;
- Reduce the work of the respiratory muscles.

Paper ID: SUB151366

 To assess the effects of mechanical ventilation is necessary to monitoriseconstantlythe levels of PaCO2. In the course of mechanical ventilation can be administered through the flow of the respirator, even oxygen in the desired concentration and aerosolized medication. It is evident that during the artificial ventilation must be associated an appropriate disostruzione routes, otherwise the pressure of the fan will be largely dispersed to overcome the resistance of the airway.In mechanical ventilation must be distinguished:

- The assisted mechanical ventilation, which facilitates and enhances the patient's spontaneous ventilation, insufficient but still present;
- The controlled mechanical ventilation, which completely replaces the spontaneous ventilation in cases where this is practically suppressed.

There are different types of ventilation:

• Assisted –controlled ventilation (A/C)

It is a volumetric mode, it can be completely controlled when the sensitivity of the trigger is canceled and the patient can not interact with the ventilator, or assisted, and then the patient can influence the activity of the machine. During controlled ventilation the ventilator provides a set number of breaths to a predetermined volume by the operator and remains totally insensitive to any effort of the patient to change the frequency. The controlled mode requires constant monitoring, especially of blood gases, to precede the appropriate adjustments in response to changes in respiratory needs of the patient. During assisted ventilation, the trigger sensitivity is determined by the operator and the patient is able, once topped with a striving inspiratory threshold value, to enable the respirator. A minimum frequency in each case is set to precede a ventilation support if the patient does not begin the respiratory act in a span of time that depends on the respiratory rate set.

• Synchronized Intermittent Mandatory Ventilation (SIMV)

The IMV is a method originally proposed for the treatment of infants and used later for weaning post-surgical patients. Practically the IMV is a ventilatory support in a series of acts which are supplied with mandatory volumetric mode or pressometrica by the fan, but between an act and the other the patient can breathe spontaneously. The IMV arises between the pure controlled ventilation, in which the breath is governed entirely by the machine, and a CPAP of 0 cmH2O. Therefore, the patient can vary independently his breathing, when the respiratory rate set by the operator is high, the spontaneous activity of the patient is virtually suppressed, mind if it is set to zero in a / m, the ventilation is totally supported by the patient.

• Pressure Support Ventilation (PSV) or pressure support (PS)

The pressure support (PS) is a method of ventilation in which each spontaneous breath of the patient receives a pressure support. The inspiratory airway pressure is kept constant at the level established by the operator, and since the PS is cycled to fall flow, the patient should theoretically have complete control of respiratory timing and tidal volume.

• Pressure controlled ventilation (PVC)

The PVC is a mode of ventilation in the metric that approaches the method assisted / controlled volume. The principle on which it is based, is to provide a pressure wave to the airway. The PVC is similar in the basic principle of the PSV, however, characterized by the fact

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

that the respiratory patern is no longer dependent on the patient but by the operator that determines respiratory frequency and inspiratory time. In true PVC the patient would have the opportunity to interact with the machine, starting a spontaneous breath, in the manner of inspiratory time set by the user. The advantage of PVC is that it can be applied without any problems in the case of a patient that does not produce inspiratory efforts, while reducing the baro traumatic risk than a controlled volumetric, in the case when mechanical conditions vary suddenly.

• Ventilation in continuous positive airway pressure (CPAP)

It is considered the ventilatory mode that is closest to spontaneous breathing and in fact the ventilation is entrusted entirely to the patient, while the breath is responsible for maintaining a predetermined positive pressure, so higher than atmospheric pressure, constant throughout the breath. The CPAP should not be confused with PEEP. The first is a specific mode of ventilation, while the second is a superelevation of the base line to apply other methods during positive pressure ventilation with volumetric in A / C or PSV. Since this is a totally spontaneous mode it is clear that the application of CPAP should only occur in patients with respiratory drive intact and good functional state of the respiratory muscles.

2.4.2. Collateral effects of non-invasive ventilation

The mortality of patients with ventilator-associated pneumonia or the incidence of complications from tracheal intubation were not significantly reduced in the last ten years.

- Ventilator-associated Pneumonia
 - Despite efforts to improve the hygienic conditions of the patient-ventilator circuit, nosocomial pneumonia continues to complicate the prognosis in approximately 30% of patients admitted and ventilated.
- Barotrauma
 - Barotrauma refers to the accumulation of extra-alveolar air associated with mechanical ventilation. The possible causes of overt barotrauma, such as pneumomediastinum, not necessarily derived from alveolar rupture, may in fact be due to other phenomenal as facial fractures or nasal, pharyngeal abscess, chest trauma, foreign body perforation and surgical procedures.
- Complications of the intubation: The essential condition for the setting of invasive ventilation is oral or nasotracheal intubation. The frequency of complications related to intubation is probably higher than what is commonly thought. Indeed retrospective studies have quantified the incidence in approximately 60.70% of the cases. These can be divided into complications due to the act of intubation, presence of the tube in the wing and extubation. Regarding the first case, you experience bleeding of the mouth or nose, dental trauma, bruises and bleeding sub mucous membranes of the pharynx, edema, vocal cord injury, dislocation, tracheal perforation and theintubation of the right bronchus. Complications related to the presence of the tube are skin reactions due to the adhesive tape that holds it in place, sinusitis, runny nose, ear infections and pressure ulcers in the pharynx and larynx with the possibility of ulcers themselves.

Other complications:

• Pulmonary embolism

Paper ID: SUB151366

- Pulmonary Fibrosis
- Alterations in intestinal motility
- Gastrointestinal haemorrhage
- Arrhythmias
- Acute renal failure
- Malnutrition
- Complications due to nutritional support
- Disorders of swallowing.

2.4.3. Collateral effects of NIMV

The side effects of the noninvasive are less important than those found during invasive ventilation.

- The haemodynamic effects, is reported a small increase of the wedge pressure compared to the ventilation during spontaneous breathing and a decrease in cardiac output, but only with the addition of external PEEP.
- Intolerance and poor compliance, are problems that can be remedied by careful choice of the form and mode of ventilation.
- Nose Injuries, is the more serious and more frequent side effect. Such injuries are caused by excessive pressure developed by the masks in an attempt to prevent air leaks and therefore, lacking the necessary spraying, develops redness with the same mechanism of pressure sores. Prevention is done by applying the protections, such as those used around the abdominal stoma, on the part in contact with the nasal mask, or trying to minimize the pressure applied by the efforts on the masks that should keep raising the height of the presidium of the skin. The success of these techniques depends on the physical and nutritional status of the patient, and, when possible, from the periodic disconnection of the patient from the ventilator, even if for short periods.
- Rhinorrhea or dry nose
- Gastric distension, it can be annoying when it prevents proper expansion of the abdomen during inspiration or when the patient is not breathing in tune with the fan. Can be solved, in some cases reducing the insufflation pressure or using a very sensitive trigger system so as to shorten the latency time between inspiratory effort and opening of the valves.
- The risk of *hyperventilation* may exist especially during the night hours in patients where the impedance of the neuromuscular system is particularly low and PaCO2 may decrease abruptly causing acute closure of the glottis, to prevent hypocapnia.
- The patient-ventilator asynchrony may occur during PSV.

Side effects of NIMV:

- Bed sore nose
- Nasal Pain
- Gastric distension
- Insomnia
- Losses of the circuit
- Claustrophobia
- Rhinorrhea
- Dryness in the nose and mouth
- Patient-ventilator asynchronism
- Closure of the glottis

Figure 8: Principal side effects of NIMV

ISSN (Online): 2319-7064

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

2.5 Oxygenotherapy

Definition

Oxygen therapy means the administration through the respiratory route of pure oxygen, or in a mixture, in a concentration higher than that of the atmospheric oxygen in order to remove or alter the hypoxic state.

2.5.1 The goal of therapy with oxygen

The body needs oxygen to stay alive. Oxygen is administered to relieve hypoxemia. The normal amount of O2 in arterial blood should range between 80 to 100 mmHg. If the pO2 falls below 55-60 mmHg, effects may be irreversible. Hypoxemia can cause irreversible tissue damage, for this reason it is important to correct it promptly. Once hypoxemia relieved with supplemental oxygen, it is necessary to determine and treat the condition that caused it [16-19], [51]. The administration of oxygen treats the effects (hypoxemia) of the disease, but not the disease itself.

Levels of hypoxemia and associated symptoms

Moderate hypoxemia

- Tachycardia;
- Confusion;
- Agitation;
- Respiratory rate increased;
- need of air;
- Visual disturbances;
- Sweating.

Grave Hypoxemia

- Lethargy;
- Drowsiness;
- Bradycardia;
- Increased respiratory rate;
- Shortness of breath;
- Increased Cyanosis.
- · Serious hypoxemia
- Shock;
- Coma;
- Death.

2.5.2. The goal of oxygen therapy

The objective is to keep the pO2 above 55-60mmHg. The administration of oxygen should not cause elevations of pCO2 above 45 mmHg. The stimulus to breathing for these individuals is given by a low level of oxygen, and consequently they do not tolerate high doses of oxygen such as those administered to people who do not suffer from chronic lung diseases with carbon dioxide retention. Often the oxygen therapy is not effective to maintain an acceptable level of pO2; must be used then to intensive therapy, such as mechanical ventilation [27].

The dosage of oxygen

The amount of O2 that is to be administered is determined based on the desired clinical effect, the medical history of the subject and the result of gas analysis. The devices operating in the oxygen indicate the dose in liters / minute in both (%). The dose of O2 is prescribed based on the device that must be used and the method of measurement.

2.5.3. Procedure

1. If you need to deliver certain percentages of oxygen use the Venturi mask. It can be used if the patient has an irregular breathing that its ventilation / minute does not exceed the number of liters delivered per minute by mask itself. Venturi masks uses the Venturi principle. The oxygen passes through a tube connected to a channel, in the mask, and flows through this passage. At the momentinwhich it outflows, occurs a pressure drop which creates a suction effect that has the express purpose of administering a specific percentage of oxygen. This allows that a large volume of oxygen and the environmental air mix to ensure a satisfactory ventilation. Commercially are available also some types of masks that permit the administration of oxygen from 24%, 28%, 35%, 40%, and other by 50-55%. If you are using a standard template, fill the humidifier with sterile water as the latter adds water vapor to dry gas (O2) to decrease the drying and irritating effects. The standard mask is a clear plastic mask that includes both the oral cavity and the nasal and creates a further area for the collection of oxygen. The exhaled air passes through small holes on the sides of the mask, which also allow the entry of air from the environment in addition to the flow of inspired oxygen, to satisfy the need of the patient's oxygen. This mask is used to administer low percentages of oxygen (40-65%). Fill the humidifier with a solution consisting of 50% distilled water and a 50% saline solution. The lungs respond to a state of irritation with the constriction of the smooth muscle lining the airways; this is called bronchospasm and is also associated with mucosal edema. During the administration of any inhalated substance, the lungs can react with bronchospasm. When a patient is receiving a topical administration and manifests sudden shortness of breath, coughing or audible wheezing, therapy should be interrupted and the incident reported to the doctor. The most common agent used for moisturizing the lung is sterile distilled water, but are also used and saline 0.9% or 0.45%. If the nasal cannula is placed, consider that the nasal passages are pervious. Inform about the importance of a constant use of the oxygen mask, listening with participation to the difficulties of the patient to hold in situ. It is possible that the patient, especially in the early days of the use of the mask, express a feeling of oppression and inability to breathe. Ensure communication despite the positioning of the mask. Often a higher resistance to the use of the mask is due to the fact that his position causes a feeling of inability to communicate, to speak, to convey to staff any difficulties or fears. Ensuring the possibility of communication is a timely intervention in case of need, these are factors of tranquility for the patient. Perform a thorough cleaning of the container of the humidifier at least once a day. All moisturizing topical methods carry a risk of microbial contamination, since all devices have in a tank of water and then there is always the possibility that bacteria or other mechanisms may grow in these tanks. If this occurs, the bacteria can be deposited in the respiratory tract of the patient with the water particles. This danger can be eliminated or reduced with the use of sterile solutions and by means of sterilization of the container and the tubing system that reaches the patient. Recollect patients and visitors not to smoke in the hospital. The rule of prohibited smoking must be observed for all, regardless of the manner in which oxygen is administered. Instruct the patient to take fluids compatible with its

ISSN (Online): 2319-7064

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

pathology and recommend the use of a cleaning solution for your skin every time the mask is removed. The mask is uncomfortable and causes a facial feeling, skin very hot and humid. Ensure that there are no leaks at the connection points. Make sure that the hose connection has not kinked or bent. You should avoid excessive padding or the same belt too tight deform the mask. Evaluate and treat the possible occurrence of side effects following administration of oxygen. The side effects of oxygen therapy are: hypoventilation, atelectasis and toxicity. interventions to reduce any possible hypoventilation from oxygen therapy. Those with chronically elevated CO2 levels, when oxygen is being administered may experience hypoventilation. This is because the administration of oxygen removes the hypoxic stimulus of respiration: the elimination of the stimulus gives rise to apnea.

- Always obtain blood gases prior to administration of oxygen.
- Start a flow of two per minute if the oxygen therapy should be started before you have met the gas analysis results.
- Monitor the patient carefully when administering oxygen to evaluate the efficacy of the therapy. Please note: respiratory rate, breathing pattern, level of consciousness and the presence of tremors.
- Report immediately any side effect.Perform the necessary actions to reduce the potential toxicity from oxygen. The toxicity occurs when oxygen is administered in high doses for a prolonged period of time. The development of toxicity is often difficult to recognize, however, that a patient receives a high dose of oxygen for a long time should be kept under careful observation with regard to the symptoms:
- Pain in the back of the sternum;
- Increased difficulty in breathing;
- Nausea and vomiting;
- Agitation;
- Dry cough.

Evaluate the effectiveness of oxygen therapy.

The effectiveness of oxygen therapy can be assessed objectively and subjectively.

Objectively by arterial blood gas analysis to determine that, the pO2 is between 60 and 80 mmHg, and hypoxemia was relieved.

Subjectively with the observations:

- The frequency of the pulse.
- Blood pressure.
- The level of consciousness.

Paper ID: SUB151366

- The temperature and condition of the skin.
- The frequency and the type of breathing.

A study of Databank titled Gas Medicines estimate the number of patients cared for in 2001 with long-term oxygen at 62,500. From this data we can draw the conclusion that the total number of patients in long-term oxygen therapy (OLT) is equal to 62,500, of which 42% were located in northern Italy, 23% in the center and 35% in the south and the islands.

2.6 Education and respiratory exercises Goals

- Correct bad habits in breathing.
- Encourage the fluid and the expulsion of the secretion.
- Teach proper methods of breathing.

Procedure

- 1. Ventilate the room in which the patient stays, especially before breathing exercises.
- 2. Choose for exercises a bright place with proper humidity.
- 3. Exercises can be performed in the supine position, sitting or standing. Patients 'forced' to bed must be arranged in a supine position without the pillow, because it prevents good ventilation of the lungs.
- 4. Instruct the patient to remove any clothing, if forces.
- 5. Instruct the patient to relax and breathe quietly.
- 6. Teach the therapeutic technique of breathing "a narrow nose" (slightly pressed together is to keep the pine nose with two fingers).
- 7. Explaining the technique of exhalation "tight-lipped" (let the air out through your lips almost completely shut, while the cheeks swell slightly) [13], [29], [36-39].

Standard exercises in the presence of respiratory problems

- 1) Teaching the patient abdominal breathing (promotes blood perfusion).
 - Assume the supine position.
 - Inhale through your nose, pushing the air in the lower abdomen. The wall of the abdomen swells outward.
 - Exhale through your mouth; the abdominal wall is pulled inside and the air can escape from the lungs through the nose.
- Teaching the patient chest breathing (eliminates the pressure on the heart and lungs and activates the blood circulation).
 - Inhale slowly, deliberately pushing the air in the chest area. The ribs are stretched laterally outward.
 - Exhale, tighten the coast and let the air escape through the nose during the exercise, the patient must maintain property shoulders and belly.
- 3) Teaching the patient the complete breathing (improves the supply of oxygen).
 - The air is inhaled slowly, the abdomen is inflated, and the blades are lifted.
 - The lungs fill with air a bit 'at a time, giving the upper body a rocking motion.
 - During exhalation, the abdominal wall swells and shoulders go down.
 - Among the inhalation and exhalation are inserted pauses of favorite lasting.
- 4) Teaching the patient breathing voice:
 - This breathing technique is performed as above, but the inspiration is carried out in three stages, to completely fill the lungs with air.
 - The exhalation occurs from the mouth, uttering together with the air coming out.
- 5) Each exercise is repeated seven times with intervals to avoid fatigue of the patient.
- 6) Patient can perform some "interval exercises":

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

- Inflate a balloon (suitable especially in children).
- Tie to bed smears of gauze that the patient should move by blowing.
- Blowing into a bottle full of water with a straw, by bubbling liquid.
- 7) The use of auxiliary equipment is used by the physical therapist; the nurse can encourage the patient in the exercises [20], [25], [29-30], [36-39].

2.7 The role of the nurse in assessing the functionality of the respiratory system

There are many factors that affect the functioning of the respiratory system: biological, socio-cultural, spiritual factors and environmental factors. The nurse in the development of the treatment plan of the patient must consider how these factors have altered respiratory function of the client. To make this assessment, the nurse uses the interview and physical examination. The physical examination is done using inspection, palpation, percussion and auscultation, which is of medical expertise [63].

2.7.1 The interview

Through the interview, the nurse has the ability to collect tons of data about the patient, his work, the environment in which they live and so on. But it is particularly important to detect the perception that the patient has on his own respiratory activity and the presence of signs of impaired function such as fatigue, dyspnea, cough, pain, previous lung disease.

Therefore, we collect here a data track with this investigation.

- Age: breathing frequency is changed regardless of age.
- Gender: The type of breathing is different in men and women.
- Profession: the type, but especially the working environment may be responsible for some respiratory diseases for precisely the presence of pollutants.
- Socio-cultural environment: may be particularly important data related to education, hobbies, marital status and family relations of the patient. In this situation we can consider lifestyle habits such as smoking, use of alcohol or addictive substances.
- Earlier diseases of the respiratory system: they may have caused disturbance to the normal respiratory function.
- Allergies: we need to check if the patient is aware of any allergies; Allergic rhinitis and asthma are two classic examples and fairly frequent.

2.7.2 The physical examination

To proceed in the collection of data that is used to assess the functionality of the respiratory system is necessary to make the physical examination, which is carried out through observation, and also by inspection, palpation, percussion and auscultation.

2.7.3 The observation

Paper ID: SUB151366

With the observation we may detect the breath and its characters, the color of the skin, the structure of the rib cage, posture.

1. The breath and its characters

When it comes to breathing, it is meant the breath formed from inhalation and exhalation. We assess the frequency of breathing, rhythm, depth, shape. Breathing frequency describes the number of breaths that occur in a minute. In the adult subject and and at rest the normal frequency varies between 12 to 20 breaths per minute; in the newborn varies from 30 to 80 breaths per minute; in the young child from 20 to 40

We talk about:

- Eupnea when breathing is normal.
- Tachypnea, when the frequency of breathing is encreased.
- Polypnea, when it increases the rate and depth of breathing.
- Bradypnea, when it decreases the frequency of breathing.
- Apnea, in the absence of breaths.

2. A method for detecting the frequency of breathing

After observing the patient's pulse, we continue to hold his hand as if it had not yet completed the survey and begin to observe the movements of the rib cage. This is not to tell the patient that you are making the counting of breaths; the breath, in fact, is under voluntaryandinvoluntarycontrol; may be sufficient to know to be observed to cause alterations that may be confused with anomalies. Proceed with the count for a minute [43]. When the movements of the rib cage are not clearly visible, it is necessary to put a hand on the trace or on the back and proceeds to count in this way. After detection, record on the folder breathing frequency and time of detection.

3. Rhythm of the breath

Describes the intervals of time and space that exist between aventilatoryact and another. Under normal conditions, this interval is constant. There are pathological conditions in which the breath assumes specific characteristics such as to give rise to pathological types of breath; like the breath of Biot, Cheyne-Stokes breathing, and the breath of Kussmaul.

4. The depth of breathing

Describes the amount of air that reaches the alveoli and which is subsequently deleted. Normally exhalation takes one and a half the inhalation, but may be longer in the case of respiratory diseases. A breath is defined as superficial or short when the amount of air introduced and low and can not reach the alveoli and engage in trade. You can create a situation of hypercapnia and hypoxia. The shortness of breath or shallow also appears in situations of alkalosis and is considered a buffer mechanism.

5. The shape of the breath

We talk about thoracic or costal breathing when it is mainly used the ribcage. This type of breathing is typical in women and in children. The abdominal or diaphragmatic breathing is typical of man and is characterized by a predominant use of the diaphragm and the abdominal muscles [41-42].

The skin color

Often people with respiratory problems have a bluish color, sometimes localized in specific areas of the body (nail bed, lips and ear lobes). This particular condition is defined with the termine cyanosis and is a consequence of an increase in reduced hemoglobin in blood levels higher than 5 g / 100 ml.

The structure of the rib cage

Normally the diameter of the latero-lateral chest is two times higher than in the anterior-posterior. In the presence of respiratory distress, the person tends to use the accessory muscles to increase ventilatory capacity [13]. The constant use of this muscle determines an increased tropism that the nurse must observe and detect as significant information. Another anomaly is represented by the retraction which is a visible depression of the intercostal, soprasternalis, and interclavicolaris spaces in the inspiratory phase [41-42]. It is caused by the need to increase the forced inspiration and can be seen in patients with airway obstruction, asthma and tracheobronchitis [58].

The posture

The position that the patient assumes may also provide useful information on its respiratory function. The subject with difficulty breathing has a tendency to take positions that facilitate maximum ventilation with minimum effort. Fowler's position can be very helpful in people with cardiopulmonary problems; it allows to increase lung expansion and reduce the abdominal pressure on the diaphragm. The dyspneic subject spontaneously assumes the sitting position that allows maximum chest expansion. The nurse, in these cases, must provide the maintenance of the position, recalling that an adequate level of ventilation and oxygenation is maintained by frequent changes of position.

2.9 The diagnostic tests

To complete the assessment of lung function, the nurse should assess the results of some tests that are used to determine the adequacy of ventilation and oxygenation. These tests are: radiological examinations (with or without contrast), nonradiological investigations (which are ECG, ultrasound, MRI, scintigraphy), functional tests (spirometry, stress tests, and blood gas analysis), laboratory tests of pleural fluid, the bronchoalveolar lavage and sputum. Also important are the skin tests of immediate and delayed hypersensitivity, and serological tests (research antibodies). These are followed by the endoscopies tracheobronchoscopia with or without biopsy, thoracentesis pleural biopsy, mediastinoscopy thoracoscopy. Among these tests of particular significance are: blood gases, spirometry, oximetry, the sputum culture

The hemogasanalisis consists in measuring the partial pressure of oxygen (PaO2) and carbon dioxide (PaCO2), bicarbonate (HCO3) and pH in a sample of arterial blood. The blood sample can be taken from the radial, brachial and femoral artery, but it is preferable to the radial artery for several reasons, such as: easily palpable, easy access, stable and is easily compressible.

It provides important information on the ventilatory or perfusion defect: PaO2 expresses as much oxygen as the lungs into the bloodstream; PaCO2 expresses the efficiency of pulmonary elimination of carbon dioxide; pH expresses the acid-base balance (increased nell'alcalosi respiratory and metabolic acidosis, decreased respiratory and metabolic nell'acidosi); the value of bicarbonate essentially expresses the metabolic aspect: if the relationship between carbonic

Paper ID: SUB151366

acid and bicarbonate, the first prevales, acidosis occurs, if it prevails the last, alkalosis occurs.

PaO ₂ <55 mmHg	HYPOXEMIA
PaCO ₂ > 45 mmHg	HYPERCAPNIA
p H< 7.35	RESPIRATORY ACIDOSIS

Figure 9: Hemogasanalysis criteria of I.R

Parametra	Unit	Arterios	Venous	Capillar
pН		7.38-7.42	7.36-7.40	7.38-7.42
PO_2	mmHg	80-100	35-45	>80
PCO ₂	mmHg	37-43	45-50	40
Sat O ₂	%	95-97	55-70	95-97
HCO ₃	Mmol/l	21-29	24-30	21-29

Figure 10: Haemogasanalysis

	normal	Respiratory acidosis	Respiratory alcalosis
Usual		alveolar	alveolar
causes		hypoventilation	hyperventilation
		Disorientation,	dizziness,cramps,
Principal		lethargy, sweating	paresthesias,
symptoms		Irregular	sweating,
		breathing	anxiety,rapid and
			deep breathing
pН	7.35-7.45	decreased	increased
PaO ₂	80-100mm	Normal	Normal orincreased
	00 10011111		
2 2.0 2	Hg	ordecreased	
PaCO ₂		ordecreased aumentata	decreased
	Hg		decreased
	Hg 35-45 mm	aumentata	decreased
PaCO ₂	Hg 35-45 mm Hg	aumentata	
PaCO ₂ HCO ₃ -	Hg 35-45 mm Hg 22-26 mEq/l	aumentata	decreased

Figure 11: The alterations dependent on gas analysis respiratory causes

Spirometry allows evaluating the respiratory function by determining the ability to exchange oxygen and carbon dioxide in the lungs. The patient breathes into a tube connected to a device called spirometer which is able to record the quantity of air that enters and exits in each breath. This tool allows you to determine the volumes and capacities of the lungs. Percutaneusoximetry consists in to assess the oxygen saturation at the capillary level. For this exam is used an instrument called oximeter; the most common is the fingertip oximeter. It is a non-invasive detection; to the patient's finger is applied a sensor that is connected to the device that continuously monitors the oxygen saturation and observe the changes according to the time. The culture of sputum consists in verifying the presence of micro-organisms responsible for an infection of the airways and provides opportunities, through sensitivity testing to identify the most active drug on the isolated bacterial strain.

2.10 The Nursing Diagnoses

The nursing diagnoses related to respiratory failure are:

A. ineffective liberation airway related to ineffective cough, increased secretions, pain, immobility;

B.Ineffective breathing pattern related to damage of the respiratory muscles, pain;

C. alteration of gas exchange related to decreased lung expansion, the presence of secretions, altered oxygen supply [63].

ISSN (Online): 2319-7064

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

A. Ineffective liberation of airways.

It is the condition in which the patient is not able to free the respiratory tract secretions or obstructions and maintain patency. The following clinical signs and symptoms may be indicative of inefficient liberation of the airways:

- Abnormal breath sounds such as rales, rhonchi, wheezing.
- Alteration of the characters of breath.
- Cough with or without sputum.
- Dyspnea.
- Cyanosis.
- Fever.

Nursing care plan for patients with nursing diagnosis: ineffective liberation of the airways.

For the realization of this plan, the nurse should ask at least two objectives:

- a. The patient will develop and maintain a state of airway patency;
- b. Secretions will be kept flowing.

The first step to tackle is to investigate and document the respiratory status of the patient, through history and physical examination. Subsequently can be realized interventions described below.

- Explain to the patient that every two hourshe must move, must cough and breathe deeply.
- Instruct the patient to take deep breaths through the nose and exhale through the mouth.
- Teach the patient to cough effectively. If there are no contraindications advise him to take just before hot drinks
- Have the patient assume a position that allows maximum chest expansion (or ortopnoica Fowler).
- If the patient's mobility is reduced, in collaboration with the physical therapist perform postural drainage and clapping (percussion of the chest with hand copa).
- Assist the patient during coughing and provide the necessary to collect the sputum.
- Keep a careful oral hygiene.
- If necessary, provide the aspiration of secretions.
- Assessing the hydric balance and, if there are no contraindications, increase your intake of fluids.
- Humidify hospital rooms. Use humidifiers.
- If the patient is on O2 therapy periodically check the fluid levels of humidification [63].

Evaluation of the intervents

The patient will present breath sounds bilaterally within 48 hours, the cough will be effective with clear sputum and fluid, ventilation will be adequate (analyze blood gas analysis, the absence of cyanosis).

B. Ineffective Breathing Pattern

Indicates a condition in which the pattern of inspiration and expiration of an individual does not allow adequate ventilation. The following clinical signs and symptoms may be indicative of ineffective respiratory activity:

- Dyspnea.
- Shortness of breath.

Paper ID: SUB151366

Tachypnea.

- Thrills.
- Alteration of the values of arterial blood gases.
- Cyanosis
- Cough.
- Use of accessory muscles of respiration.
- Increase in the anterior-posterior diameter of the chest.
- Alteration of the excursion of the chest.

Nursing care plan for patients with nursing diagnosis: ineffective breathing.

What you want to achieve with the realization of this treatment plan is to optimize the ventilation, facilitating the flow of air with the lowest muscle work and the lowest possible energy expenditure [63]. The actions that need to be planned and implemented to achieve these objectives are:

- Determine and document the patient's respiratory status.
- Document the patient's breathing pattern (frequency, rhythm, depth of breathing, use of accessory muscles, presence of cyanosis, arterial blood gas analysis).
- Instruct the patient to take the position that allows maximum thoracic excursion.
- Instruct the patient to control his breathing. Teach him the breathing exercises making him learn to prolong exhalation.
- Accompany the patient during exercises by creating an atmosphere of calm and tranquility.
- Inform the patient for the existence of irritants to the respiratory system and the need to avoid them (smoke, dust, pollutants).
- Administer medications prescribed by your doctor in order to improve the flow of air (bronchodilators, anticholinergics, anti-inflammatories).
- Monitor respiratory status in response to prescribed therapy.
- Reduce the fatigue of the respiratory act by teaching the patient relaxation techniques and diaphragmatic breathing.
- Maintain an adequate nutritional intake in order to support the work of the respiratory muscles and provide necessary energy intake.
- Use techniques to facilitate the removal of secretions from the airways examined in the diagnosis liberation of ineffective airway.
- Avoid, if possible, the performance of invasive techniques.
- Maintain adequate cleaning of the environments and tools that are used for the patient [63].

Evaluation

The patient will present an improvement in symptoms. The noises are bilateral normal, the pattern of breathing is less difficult. Improve the values of vital capacity and forced expiratory volume. The blood gases are normal.

C. Alteration of Gas Exchange

Indicates a condition of imbalance between the intake of oxygen and elimination of carbon dioxide level in the air / blood barrier of the alveolar-capillary membrane. The following clinical signs and symptoms may be indicative of altered gas exchange:

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

- Presence of secretions in the airways and difficulty in eliminating them.
- Hypercapnia.
- Hypoxia.
- Drowsiness.
- Restlessness, irritability.
- Confusion.

Plan nursing care for patients with nursing diagnosis: impaired gas exchange.

Maintaining adequate ventilation and oxygenation to the body's needs is the goal you want to achieve with the schedule for this plan.

- Perform a thorough assessment on lung function and on prescription prepare for running gas analysis.
- In the presence of secretions in the airways instruct the patient in accordance with the provided plan of care for the diagnosis liberation of ineffective airway.
- The patient must take a position that facilitates a proper ventilation / perfusion ratio.
- It is important to avoid sudden temperature changes and maintain a calm and peaceful environment, providing the patient the information about what you are doing and the reasons for it.
- Administration of medication (bronchodilators, expectorants, corticosteroids, antibiotics, antihistamines) and O2 therapy is an action that stands out the complementarity of the figures of the doctor and the nurse. The doctor, in fact, is responsible for the prescription, while the nurse is the proper administration and awareness of the effect of the drugs [63].

Evaluation

The improvement of the patient's condition will be highlighted by a normalization of blood gas values and the disappearance of the signs of hypoxia.

GENERAL ASSISTENCE TO THE PATIENT WITH RESPIRATORY FAILURE AT DISCHARGE			
Bisogno/Problem	Ojective	Intervention	
Security / protection	Reintegration of the patient in his environment.	Encourage the patient and his family specific information about the services to which they refer for periodic inspections or for emergency purposes.	
	Reduce Legal discounts to the request for documents relating to the hospital.	Instruct the patient and his family the practice to perform to get the health records they need.	

3. Conclusions

Paper ID: SUB151366

The nurse assisting a patient with respiratory failure, must daily confront him, in search for better solutions to his physically and mentally problems. The nurse must take care of the patient in its entirety, from all points of view, so as to ensure a good quality of life. Psychological support, that the nurse should give all these patients, is very important, helping and ensuringacorrect information to them, about everything that is done, obtain the collaboration of the patient for the undergoing examinations, offer them psychological support and reassurance. An important task is also the education prior to discharge. We must never forget that the patient with a mechanical garrison poses practical problems completely different than that discharged with medical therapy alone. Therefore, even when the patient is admitted, it is very important to educate both the patient and his family on some knowledge and practical operations. The nurse should teach the invasively ventilated patient proper management of the orifice cleaning, replacement of elastic bands support the bronchial aspiration that can also be performed by the patient himself, and periodic review of any stagnant secretions at the inner cannula. Instead, for the noninvasive ventilation NIMV should be shown the correct positioning of the mask and the support, the inflation of the "cuscino" around the mask itself, and teach prevention regulations in order to prevent the onset of skin nasal lesions.Invece per la ventilazione non invasiva NIMV si deve mostrare il corretto posizionamento della maschera e dei supporti, il gonfiaggio del "cuscino" intorno alla maschera stessa, nonché insegnare le norme di prevenzione al fine di scongiurare l'insorgenza di lesioni cutanee nasali.

References

- [1] Celli BR, Cote CG, Marin JM et al."The body –mass index, air flow obstruction, dyspnea and exercise capacity index in chronic obstructive disease".NEngl J Med 2004; 350:1005-1012.CrossRefMedline
- [2] Oga T, Nishimura K, Tsukino M et al. "Analysis of the factors related to mortality in chronic obstructive pulmonary disease: role of exercise capacity and health status". Am J RespirCrit Care Med 2003; 167: 544-549. Abstract / Free Full Text
- [3] Hamilton AL, Killian KJ, Summers E,et al. "Symptom intensity and subjective limitation to exercise in patients with cardiorespiratory disorders". Chest 1996; 110:1255-1263. Abstract / Free Full Text
- [4] Gosselink R, Trooster T et al." Peripheral muscle weakness contributes to exercise limitation in COPD". Am J RespirCrit Care Med 1996; 153:976-980? Medline
- [5] Mahler DA, Harver A. "A factor analysis of dyspnea ratings, respiratory muscle strength and lung function in patients with chronic obstructive pulmonary disease". Am Rev Respir Dis 1992; 145: 467-470.
- [6] Vincken W, Van Noord JA, et al. "Improved health outcomes in patients with COPD during 1 years treatment with tiotropium". EurRespir J 2002; 19: 209-216. Abstract / Free Full Text
- [7] Ikeda A, Nishimura K, Koyama H, et al. "Dose response study of ipratropium bromide aerosol on maximum exercise performance in stable patients with chronic obstructive pulmonary disease". Thorax 1996; 51: 48-53. Abstract / Free Full Text
- [8] Weiner P, Magadle R, Berar –Yanay N et al."The cumulative effect of long acting bronchodilatators, exercise and inspiratory muscles training on the perception of dyspnea in patients with advanced

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

- COPD".Chest 2000; 118: 672-678.Abstract / Free Full Text
- [9] AmericanThoracis Society. "Pulmonary rehabilitation": 1999. Am J RespirCrit Care Med 1999; 159: 1666-1682.Free Full Text
- [10] McGavin CR, Gupta SP, Lloyd EL, et al." Physical rehabilitation for the chronic bronchitic: results of a controlled trial of exercises in the home". Thorax 1977; 32: 307-311. Abstract / Free Full Text
- [11] Degre S, Srgysels R, Messin R, et al." Hemodynamic responses to physical training in patients with chronic lung disease". Am Rev Respir Dis 1974; 110: 395-402? Medline
- [12] Casaburi R, Petty TL." Principles and practice of pulmonary rehabilitation". Philalelphia, PA; W.B.Saunders; 1993.Medline
- [13] Belman MJ, KendreganBA."Exercise training fails to increase skeletal muscle enzymes in patients with chronic obstructive pulmonary disease". Am Rev Respir Dis 1981; 123: 256-261? Abstract / Free Full Text
- [14] Lacasse Y, Brosseau L, Milne S, te al. "Pulmonary rehabilitation for chronic obstructive pulmonary disease". Cochrabase Syst Rev 2002; 3: CD003793.
- [15] Fabbri LM, Hurd SS." Global strategy for the diagnosis, management and prevention of COPD: 2003 update". Eur. Respir J 2003; 22: 1-2. Free Full Text
- [16] "Pulmonary Rehabilitation" Guidelines Panel, American College of Chest Physicianand American Association of Cardiovascular and Pulmonary Rehabilitation. Chest 1997; 112: 1363-1396.Free Full Text
- [17] Donner CF, Decramer M. Pulmonary rehabilitation. EurRespirMonogr; 13:1 -200.
- [18] British Thoracic Society, "Standards of Care Subcommitee on Pulmonary Rehabilitation. Pulmonary rehabilitation". Thorax 2001; 56: 827-834.Free Full Text
- [19] Donner CF, Muir JF." Selection criteria and programmes for pulmonary rehabilitation in COPD patients: rehabilitation and chronic care scientific group of the Europian Respiratory Society". EurRespir J 1997; 10: 744-757.Medline
- [20] National Emphysema Treatment Trial Research Group. "Cost effectiveness of lung-volume-reduction surgery for patients with severe emphysema". N Engl J Med 2003; 348: 2092-2102. CrossRefMedline
- [21] National Emphysema Treatment Trial Research Group." A randomized trial comparing lung volumereduction surgery for patients with severe emphysema". N Engl J Med 2003; 348: 2059-2073.Abstract / Free Full Text
- [22] Gelb AF, McKenna RJ, Brenner M, et al." Lung function 5 years after lung volume reduction surgery for patients with severe emphysema". Am J RespirCrit Care Med 2001; 163: 1562-1566.Abstract / Free Full Text
- [23] Fahy BF." Pulmonary rehabilitation for chronic obstructive pulmonary disease: a scientific and political agenda". Respir care. 2004; 49: 28-38.Medline

Paper ID: SUB151366

- [24] Thompson SG. "Why sources of heterogeneity in meta-analysis should be investigated". BMJ 1994; 309: 1351-1355.Free Full Text
- [25] O'Donnell DE, McGuire M, et al."The impact of exercise reconditioning on breathleness in severe chronic air flow limitation". Am J RespirCrit Care Med 1995; 152:2005-2013. Abstract / Free Full Text
- [26] Reardon J, Awad E, Normandin E, et al."The effect of comprensiveoutpatient pulmonary rehabilitation on dyspnea". Chest 1994; 105: 1046-1052.Abstract / Free Full Text
- [27] Ries AL, Kaplan RM, Limberg TM. "Effects of pulmonary rehabilitation on physiologic and psychosocial outcomes in patients with chronic obstructive pulmonary disease". Ann Intern Med 1995; 122:823-832. Abstract / Free Full Text
- [28] Weiner P, Azgad Y, Ganam R." Inspiratory muscles training combined with general exercise reconditioning in patients with COPD". Chest 1992; 102: 1351-1356.Abstract / Free Full Text
- [29] Simpson K, Killian K, McCartney N, Stubbing DG,et al."Randomised controlled trial of weightlifting exercise in patients with chronic airflow limitation". Thorax 1992; 47: 70-75. Abstract / Free Full Text
- [30] Hernandez MT, Rubio TM, Ruiz FO,etal."Results of a home-based training program for patients with COPD".Chest 2000; 118:106-114.Abstract / Free Full Text
- [31] Cambach W, Chadwick-Straver RV, WagenaarRC,etal."The effects of a community –based pulmonary rehabilitation programme on exercise tolerance and quality of life: a randomized controller trial". EurRespir J 1997; 10: 104-113.Abstract / Free Full Text
- [32] Puete-Maestu L, SantaCruz A, Vargas T. "Effects of training on the tolerance to high-intensity exercise in patients with severe COPD".Respiration(Herrlisheim) 2003; 70: 367-370.
- [33] Troosters T, Gosselink R, Decramer M." Short and long –term effects of outpatient Rehabilitation in patients with chronic obstructive pulmonary disease a randomized trial". Am J Med 2000; 109: 207-212.
- [34] Casaburi R, Patessio A, Loli F, et al."Reduction in exercise lactic acidosis and ventilation as a result of exercise training in patients with obstructive lung disease". Am Rev Respir Dis 1991; 143: 9-18?
- [35] Maltais F, LeBlanc P, Simard C., ETal."Skeletal muscle adaptation to endurance training in patients with chronic obstructive pulmonary disease". Am J RespirCrit Care Med 1996; 154: 442-447?
- [36] Gigliotti F, Coli C, Bianchi R., etal."Exercisetrainig improve exertional dyspnea in patients with COPD: evidence of the role of mechanical factors". Chest 2003; 123: 1794-1802.
- [37] Casaburi R, Porszasz J, Burns MR." Physiologic benefits of exercise training in rehabilitation of patients with severe chronic obstructive pulmonary disease". Am J RespirCrit Care Med 1997; 155: 1541-1551.
- [38] Porszasz J, Emtner M, WhippBJ., et al." Endurance training decreases exercise-induced dynamic hyperinflation in patients with COPD". EurRespir .J 2003; 22: 205s.

Index Copernicus Value (2013): 6.14 | Impact Factor (2013): 4.438

- [39] O'Donnell DE, McGuire M, Samis L., et al." General exercise training improves ventilatory and peripheral muscle strength and endurance in chronic airflow limitation". Am J RespirCrit Care Med 1998; 157: 1489-1497.
- [40] Harver A, Mahler DA, Daubenspeck JA." Targeted inspiratory muscle training improves respiratory muscle function and reduces dyspnea in patients with chronic obstructive pulmonary disease". Ann Intern Med 1989; 111: 117-124.
- [41] Lisboa C, Munoz V, Beroiza T., etal."Inspiratory muscle training in chronic airflow limitation: comparison of two differenttraining loads with a threshold device". EurRespirJ 1994; 7: 1266-1274.
- [42] Lisboa C, Villafranca C, Leiva A., et al."Inspiratory muscle training in chronic airflow limitation: effect on exercise performance". EurRespirJ 1997; 10: 537-542.
- [43] Redelmeier DA, Bayoumi AM, Goldstein RS." Interpreting small differences in functional status: the six minute walk test in chronic lung disease patients". Am J RespirCrit Care Med 1997; 155: 1278- 1282.
- [44] Booker HA." Exercise training and breathing control in patients with chronic airflow limitation". Physiotherapy 1984; 70: 258-260.
- [45] Ringbaek TJ, Broendum E., et al. "Rehabilitation of patients with chronic obstructive pulmonary disease: exercise twice a week is not sufficient". Respir Med 2000; 94: 150-154.
- [46] Wijkstra PJ, Van Altena R, KraanJ."Quality of life in patients with chronic obstructive pulmonary disease improves after rehabilitation at home". EurRespirJ 1994: 7: 269-273.
- [47] Cockcroft AE, Saunders MJ, Berry G. "Randomized controlled trial of rehabilitation in chronic respiratory disability". Thorax 1981; 36: 200-203.
- [48] Engstrom CP, Persson LO, Larsson S., et al." Long term effects of a pulmonary rehabilitation programme in outpatients with chronic obstructive pulmonary disease: a randomized controlled study". Scand J Rehabil Med 1999; 31: 207-213.
- [49] Goldstein RS, Gort EH." Randomized controlled trial of respiratory rehabilitation". Lancet 1994; 344: 1394-1397.
- [50] Finnerty JP, Keeping Me, Jones J." The effectiveness of outpatient'spulmonary rehabilitation in chronic lung disease: a randomized controlled trial". Chest 2001; 119: 1705-1710.
- [51] Sin DD, McAlister FA, Man FS, et al."Contemporary management of chronic obstructive pulmonary disease: scientific review".JAMA 2003; 290: 2301-2312.
- [52] Hajiro T, Nishimura K, Tsukino M. "Analysis of clinical methods used to evaluate dyspnea in patients with chronic obstructive pulmonary disease". Am J RespirCrit Care Med 1998; 158: 1185-1189.
- [53] Dekhuijzen PN, Beek MML, FolgeringHT."Psychological during pulmonary rehabilitation and target –flow inspiratory muscle training in COPD patients with a ventilatory limitation during exercise". Int J Rehabil Res 1990; 13: 109-117.
- [54] O'Donnell DE, D'Arsigny C." Exercise hypercapnia in advanced chronic obstructive pulmonary disease: the role of lung hyperinflation". Am J RespirCrit Care Med 2002; 166: 663-668?

- [55] Barbera JA, Peinado VI, Santos S. "Pulmonary hypertension in chronic obstructive pulmonary disease". EurRespirJ 2003; 21: 892-905.
- [56] Casaburi R. Limitation to exercise tolerance in chronic obstructive pulmonary disease: look to the muscle of ambulation. Am J RespirCrit Care Med 2003; 168: 409-410?
- [57] Laghi F, Tobin MJ. "Disorders of the respiratory muscles". Am J RespirCrit Care Med 2003; 168: 10-48?
- [58] Decramer M, Gosselink R, Troosters T." Muscle weakness is related to utilization of health care resources in COPD patients". EurRespirJ 1997; 10: 417-423.
- [59] Whittom F, Jobin J, Simard PM."Histochemical and morphological characteristics of the vastuslateralis muscle in patients with chronic obstructive pulmonary disease". Med Sci Sports Exerc 1998; 30: 1467-1474.
- [60] Saey D, Michaud A, Couillard A., et al."Contractile fatigue, muscle morphometryand bloodlactate in chronic obstructive pulmonary disease". Am J RespirCrit Care Med 2005; 171: 1109-1115.
- [61] Gosselink R, Troosters T, Decramer M. Distribution of muscle weakness in patients with stable chronic obstructive pulmonary disease. J Cardiopulm Rehabil 2000; 20: 353-360.
- [62] Alan Stevens, James Lowe.Patologia generale e fiziopatologia. Edizione italiana 2002, 2ed,XV:552-570, ISBN 88-408-1179-6.
- [63] Loredana Sasso.Scienze infermieristiche generali e cliniche2007;421-439,ISBN: 9788838616921