بسم الله الرحمن الرحيم

شبكة المعلومـــــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـــسـم ﴾

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بالرسالة صفحات

لم ترد بالأصـــل

Study Of The Use Of Noninvasive Positive Pressure Ventilation In Patients With Acute Respiratory Failure In Intensive Care Units.

Thesis

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INTRODUCTION

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Respiratory failure, defined as a major abnormality of gas exchange, is the main indication for mechanical ventilation. Reasonable criteria for the gas exchange abnormality include a partial pressure of oxygen (PaO_2) of less than 50 mmHg on room air and/or a partial pressure of carbon dioxide ($PaCO_2$) of greater than 50 mmHg with pH below 7.35.[1,2]

Respiratory failure is commonly defined as "Hypoxemic" characterized by failure of gas exchange, as "Hypercapnic" characterized by failure of the ventilatory pump, or a combination of both.[3]

It is helpful to recall that the cause of hypercapnia is often independent of the cause of hypoxaemia,[4] so treatment of the latter is different from treatment of the former.

Intrapulmonary shunt ($\dot{Q}_S/\dot{Q}_T$) causes hypoxemia refractory to oxygen therapy despite hyperventilation and reduced $PaCO_2$ in type I acute hypoxemic respiratory failure (AHRF).[5,6] Primary failure of alveolar ventilation ($\dot{V}A$) leads to carbon dioxide retention and arterial hypercapnia with reduced PaO_2; this hypoxemia corrects easily with oxygen therapy in type II or acute ventilatory failure (AVF).[5]

Type III respiratory failure occurs in the perioperative period when factors that reduce functional residual capacity (FRC) combine with causes of increased closing volume (CV) to produce progressive atelectasis. The end result can be type I (AHRF), or type II ventilatory failure, or both.[7]

Type IV respiratory failure ensues when the circulation fails and resolves when shock is corrected, as long as one of the other types of respiratory failure has not supervened.[4] The appropriate rationale for ventilator therapy in these patients who are frequently tachypnic with erratic respiratory patterns is to stabilize gas exchange and minimize the steal of a limited cardiac output by the working respiratory muscles until the mechanism for the hypoperfusion state is identified and corrected.[8]

Acute hypoxemic respiratory failure (AHRF)

Hypoxemia defines failure of gas exchange and is usually its primary manifestation, carbon dioxide exchange is also affected but usually can be compensated by increased alveolar ventilation. Thus, in the absence of superimposed ventilatory pump failure, hypercapnia is not a feature of gas exchange failure.[2]

Hypoxemic respiratory failure is characteristic of pneumonia, atelectasis, pulmonary oedema, and pulmonary fibrosis. It also may occur in asthma, chronic bronchitis, and many other respiratory illnesses.[2]

Arterial hypoxaemia arises from collapse or filling of alveoli with the result that a substantial fraction of mixed venous blood traverses non ventilated air spaces, effecting a right – to – left intrapulmonary shunt.[9] In addition to the adverse consequences on gas exchange, diseases characterized by interstitial and alveolar fluid accumulation increase lung stiffness, imposing a mechanical load on the patient with a resulting increase in the work of breathing.[10] Uncorrecting the gas exchange and lung mechanical abnormalities may lead to tissue hypoxia, ventilatory arrest, and death.[11]

The disorders causing acute hypoxaemic respiratory failure may be divided into diffuse lesions such as pulmonary oedema and focal lung lesions such as lobar pneumonia.[12] Since the distribution of airspace involvement may have implications for the response to ventilations such as positive end – expiratory pressure (PEEP),[13,14] this differentiation is of both therapeutic and prognostic value.[12]

Excessive lung liquid accumulation may arise from hydrostatic pressure increase (cardiogenic or hydrostatic edema), or from lung injury that causes an increase in capillary permeability and a failure of the microcirculation to maintain an oncotic pressure gradient between intravascular and interstitial spaces (low pressure edema or acute respiratory distress syndrome [ARDS]).[12]

Diffuse lung lesions (pulmonary oedema)

I) Cardiogenic Pulmonary Edema (CPE).

CPE most commonly arises when left ventricular end-diastolic pressure (LVEDP) increases, as in left ventricular failure or with myocardial ischemia. Mechanical obstruction at the level of mitral valve (e.g., mitral stenosis or ball-valve obstruction) with associated left atrial pressure elevation and mitral valve incompetence with transmission of ventricular pressures to the lung circulation during systole are less common causes of hydrostatic oedema.

In the setting of critical illness, often with multiple factors contributing to impaired ventricular function or impaired renal function, volume overload causes increased hydrostatic pressure. This may be encountered even without exogenous fluid administration, such as when

the Trendlenberg position results in translocation of intravascular volume from the peripheral circulation to the central compartment.[12]

In patients with cardiogenic pulmonary oedema (CPE), the work of breathing is increased from reduced lung compliance and increased airway resistance (interstitial and bronchial oedema).[15] The reduction in lung compliance correlates with derangement in pulmonary gas exchange.[15] In patients with CPE, the inspiratory muscles have to generate large negative swing in pleural pressure (high oesophageal pressure (Pes)), which increase left ventricular transmural pressure and afterload.[16,17] The myocardial depressant effect of hypoxemia together with the increased afterload can result in reduction of the cardiac output, this in turn compromises oxygen delivery to the respiratory muscles and may start a vicious cycle. Increased sympathetic nerve activity and catecholamines levels are important acute compensating mechanisms in maintaining cardiac output and perfusion pressure in patients with CPE.[18] Prolonged increase in sympathetic nerve activity, however, actually may contribute to the progression of CPE by causing cardiac myocyte hypertrophy and necrosis, a reduction in B_1– adrenergic receptors, patchy destruction and depletion of sympathetic nerve terminals, and impairment in systolic and diastolic functions.[18]

In patients with stable chronic heart failure, inspiratory and expiratory muscle strengths are impaired and inspiratory muscle weakness correlates significantly with dyspnea during activity.[19] Respiratory muscle fatigue with retention of CO_2 is seen frequently in patients with severe CPE.[20] Of clinical relevance is the fact that respiratory distress in CPE is not related directly to hypoxemia and cannot be reversed with oxygen administration alone.[21]

II) Low-pressure pulmonary oedema (ARDS).

Low-pressure pulmonary oedema, also termed pulmonary capillary leak or ARDS, results from injury to the lung microcirculation sustained from direct lung insults (e.g., aspiration, inhalation, or infectious agents) or indirectly by systemic processes (e.g., sepsis or traumatic shock with large volume blood product resuscitation). Regardless of the initiating events, the result is leak of fluid and protein into the interstitium and eventually alveolar spaces despite normal microvascular hydrostatic and oncotic pressures.[12]

III) Edema of unclear or mixed etiology.

Some forms of pulmonary oedema, although well described clinically, remain obscure as to their etiology, at least in terms of basic Starling mechanisms. Neurogenic pulmonary oedema, most commonly seen following catastrophic central nervous system injury, has been ascribed to both high-and low-pressure mechanisms.[22]

Reexpansion pulmonary oedema, relatively common unilaterally after inflation of collapsed lung by tube thoracotomy, has been held by many to result from a sudden decrease in lung interstitial pressures and thus from a widened hydrostatic pressure gradient.[12] Some evidence in animal models and from clinical cases, however, has suggested that reperfusion following reexpansion may result in lung injury analogous to reperfusion injury following organ transplantation, with a pulmonary capillary leak.[23]