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شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

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شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

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سامية محمد مصطفى

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

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بعض الوثائق الأصلية تالفة

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بالرسالة صفحات لم ترد بالأصل

A COMPARATIVE STUDY OF CONVENTIONAL VERSUS LOW TIDAL VOLUME MECHANICAL VENTILATION IN ACUTE SEVERE ASTHMA.

Thesis

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In

Anesthesiology

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To

My

Mother

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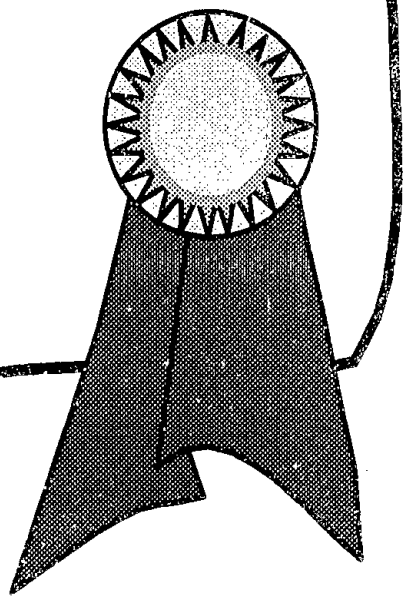
Introduction



INTRODUCTION

Most asthma exacerbations are managed in the community or emergency department while the more severe cases that fail to respond to bronchodilator and anti-inflammatory therapy require admission to intensive care units (ICU).[1-2]

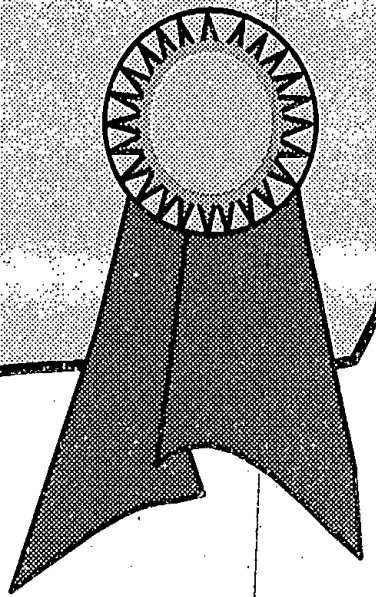
Worldwide asthma prevalence is increasing, and with that the total number of admissions to hospital and intensive care. Although the time between the onset of symptoms and the requirement for ventilation is becoming shorter, the outcome is improving with fewer deaths and lower complication rates. [3]

Acute respiratory failure necessitating intubation and mechanical ventilation in patients with acute severe asthma is relatively uncommon, and there are few data available regarding positive pressure ventilation in critically ill patients with asthma. [4]

Aggressive bronchodilator (continuous nebulised β_2-agonist) and anti-inflammatory therapy must continue throughout the period of mechanical ventilation. [5-6]

Over the last seven to eight years there has been a marked change in the philosophy of how best to mechanically ventilate the patient in severe respiratory failure. Prior to this time most authors suggested large tidal volumes (10-15 ml/kg) for all patients with essentially no concern for the peak alveolar pressure developed. [7-8]

Review of literature



REVIEW OF LITERATURE

ASTHMA

Definition and epidemiology:

Asthma is a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular, mast cells, eosinophils, T lymphocytes, neutrophils, and epithelial cells. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and cough, particularly at night and in early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment. The inflammation also causes an associated increase in the existing bronchial hyper-responsiveness to a variety of stimuli. [9]

Acute severe asthma (Status asthmaticus) is an acute asthmatic attack in which the degree of bronchial obstruction is either severe from the beginning or increases in severity and not relieved by the usual treatment placing the patient at risk of developing acute respiratory failure. [1, 10]

Asthma is the most common chronic lung disease in both the developed and developing countries. There is evidence that over the last 20 years its prevalence has increased worldwide. [11]

A number of circumstances may mimic the diagnosis of acute asthma (COPD, congestive heart failure, upper airway obstruction, hyperventilation syndrome, or vocal cord dysfunction). Usually, they can be identified by history and physical examination. Morbidity and mortality are most often associated with failure to appreciate the severity of the exacerbation, resulting in inadequate emergency treatment and delay in referring to hospital. [12]

Pathophysiology:

Asthma is an inflammatory disease of the airways that appears to involve a broad range of cellular- and cytokine- mediated mechanisms of tissue injury. [13] In asthmatic subjects who die suddenly of an asthmatic attack, the peripheral airways frequently exhibit occlusion of the bronchial lumen by inspissated secretions, thickened smooth muscles, and bronchial wall inflammatory infiltration and edema. [14] These changes observed in the asthmatic airways support the hypothesis that peripheral airways occlusion forms the pathologic basis of the gas exchange abnormalities observed in acute, severe asthma. In such patients, widespread occlusion of the airways leads to the development of extensive areas of alveolar units in which ventilation (V) is severely reduced but perfusion (Q) is maintained (i.e. areas with very low V/Q ratios, frequently lower than 0.1). [15]

Hypoxemia, hypercapnia and lactic acidosis

Hypoxemia is therefore common in every asthmatic crisis of some severity; mild hypoxia is easily corrected with the administration of relatively low concentrations of supplemental oxygen. [16] More severe hypoxemia and the need for higher concentrations of supplemental oxygen may relate to some contribution of shunt physiology.

Analysis of arterial blood gases is important in the management of patients with acute, severe asthma, but it is not predictive of outcome. In the early stages of acute, severe asthma, analysis of arterial blood gases usually reveals mild hypoxemia, hypocapnia and respiratory alkalosis. If the deterioration in the patient's clinical status lasts for a few days there may be some compensatory renal bicarbonate secretion, which manifests as a non-