MANAGEMENT OF SEVERELY INJURED PATIENT

Essay

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By

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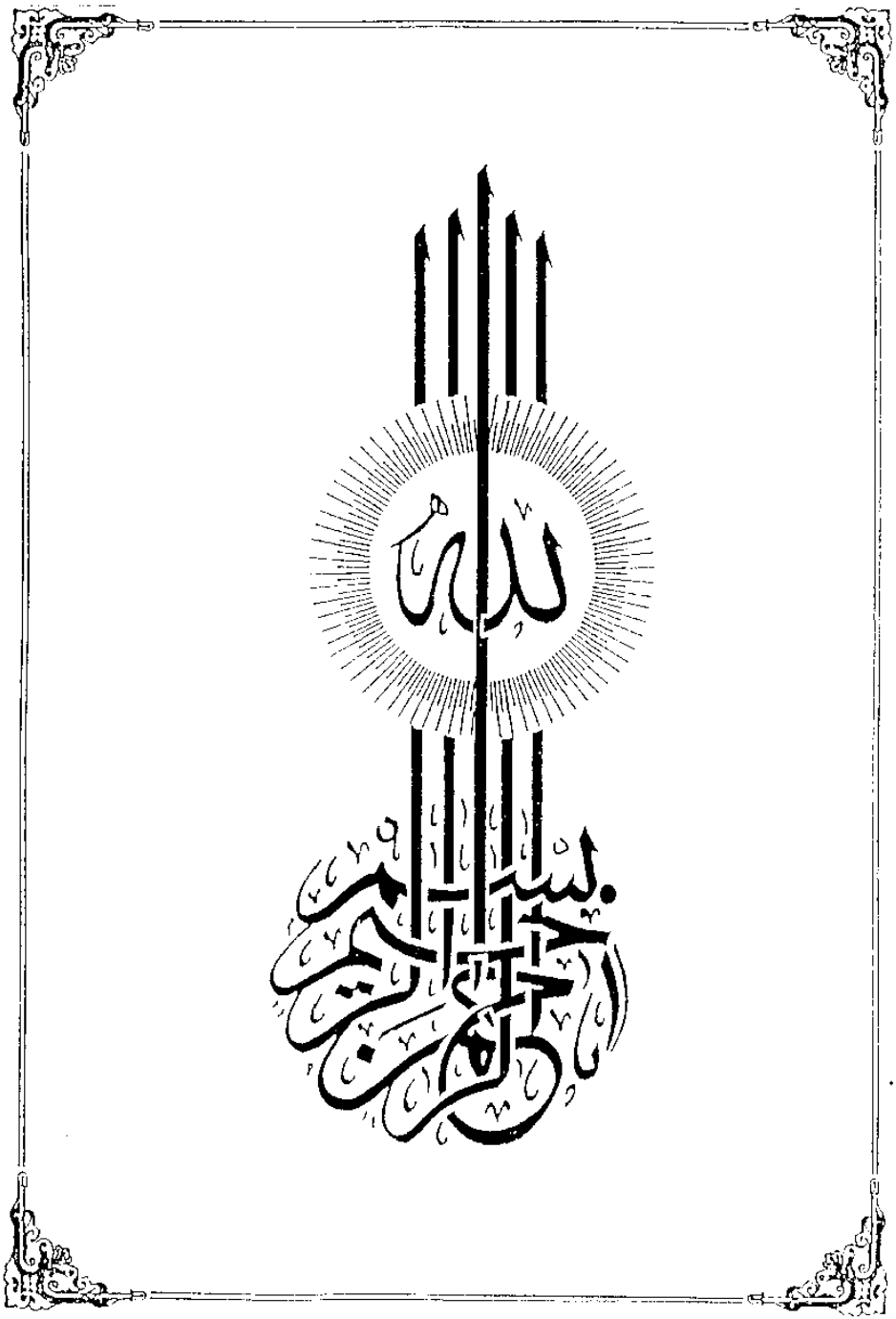

To My Wife

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Introduction

INTRODUCTION

Our modern, fully motorized world, with its modes of high velocity transportation, heavy industry, and increasing population, has seen a rise in the volume of patients with severe injuries.

Statistical reports mentioned that there is an increasing number of severely injured patients resulting from either road trauma or war injuries, however, trauma and accident surgery is falling more and more into providence of specialists who deal primarily with severely injured patients.

The management of the severely injured patients requires a team approach. These critically ill patients are best treated in trauma centers that are equipped and staffed 24 hours a day to manage these complex problems.

The management begins with the surgeon who assumes the responsibility for coordinating the efforts of the personnel in the emergency room. The surgeon who has the prespective to treat the patients as a whole is best qualified to take the charge. However, the

presence of this qualified surgeon does not absolve other physicians or surgeons from the obligation to understand the orderly management of severely injured patients.

Major scientific advances in the management of severely injured patients have been realized in the last two decades.

Hemodynamic support and aggressive pulmonary care have improved the survival rate appreciably.

The development of techniques of intravenous hyperalimentation enhanced awarness of the need for nutritional support of these patients to help to prevent late death from sepsis and protein malnutrition.

The aim of this work is to review literatures concerning the approach to the severely injured patient, the complications encountered during his treatment and the management of multiple musculo-skeletal injuries in the poly traumatized patient.

The severely injured patient is that patient with multiple Trauma or a single trauma which endanger life.

Physiology of the cardio-respiratory system, and effect of hypoxia and poor perfusion on the vital organs.

PHYSIOLOGY OF THE CARDIO-RESPIRATORY SYSTEM, AND EFFECT OF HYPOXIA AND POOR PERFUSION ON THE VITAL ORGANS

The mechanism of respiration:

The mechanics of the respiratory system are dependant on the interaction of its two fundamental constituents with elastic properties; the lungs and the chest wall. The pleural cavity is the potential space which separates these two constituents, with a subatmospheric pressure of about -2.5 cm H_2O. During inspiration, the intrapleural pressure becomes increasingly sub-atmospheric, inspiration is limited by the mechanical properties of the lung. Expiration occurs passively to the resting expiratory level. (Mc A.Ledingham and T. Woodcock, 1988).

Breathing is a dynamic process and work must be done to overcome resistance to airflow within the lungs. Resistance is expressed as the pressure gradient necessary to move a unit volume of gas inunit time. Resistance depends on the radius of the airway, so bronchospasm or intraluminal obstruction increases the resistance. (Nunn J., 1977).

The static elastic properties of the lung and the chest wall are described by their compliance. In the intact respiratory system, the lung volume at the resting expiratory level is known as the functional residual capacity (F.R.C.). At this point, the elastic tendency of the chest wall to expand from the (F.R.C.) is balanced by the tendency of the lung to retract. Changes in the mechanical properties of the lung and chest wall due to age, disease, or trauma, will result in changes in the F.R.C. and work of breathing. (Nunn, 1977).

During inspiration, the expansion of the chest cavity is brought about by contraction of the diaphragm and rotation of the ribs upwards. Quiet expiration is brought about by relaxation of the inspiratory muscles, aided by elastic recoil of the lungs. (Mc. A. Ledingham, 1988).

In the critically-injured patient, with low pulmonary compliance, poor gas exchange and low cardiac output, the high work of breathing demands a high proportion of the total available oxygen and lead to metabolic acidosis and rapid deterioration of this patient. This patient can only be helped by muscular

paralysis and intermittent positive pressure ventilation (I.P.P.V.) which will improve the arterial oxygenation. (Cohen et al., 1982).

Regulation of respiration:

The muscles of respiration are innervated by lower motor neurons within the phrenic (C 3, 4 and 5), cervical and intercostal nerves; breathing stops if the cord is transected above the level of C 3. Automatic rhythmic breathing is controlled by a respiratory centre in the brain stem; complete absence of respiratory effort is a feature of the clinical syndrome of brain death.

(Conference of the Medical Royal Colleges and their Faculties in the United Kingdom, 1979).

Breathing can also be controlled voluntarily by cortical activity via the corticospinal tracts. Reciprocal innervation of the respiratory muscles brings about relaxation of the expiratory muscles during inspiration and vice-versa.

The respiratory centre:

The respiratory centre is in the medulla oblongata and consists of two groups of neurones, the ventral and

dorsal groups. Within both groups, inspiratory and expiratory neurons can be identified by their pattern of discharge during the respiratory cycle. The dorsal group drives the intercostal muscles and the accessory muscles of respiration. (Longs and Duffin, 1984).

Two higher brain stem centres exert a modifying effect on the rhythmicity of the medullary respiratory centre, the apneustic and pneumotaxic centres (Mitchell and Berger, 1975).

Respiratory reflexes from the lung:

Inflation and deflation reflexes called the Herring-Breuer reflexes, are initiated by stimulation of the stretch receptors and J-receptors respectively and cause a vagally-mediated inhibition of inspiration or expiration. The J-receptors (deflation) are particularly sensitive to pulmonary congestion and embolisation and may contribute to the rapid shallow respiration seen in these conditions.

The cough reflex involves irregular deep inspiration followed by forced expiration through suddenly opening glottis and this reflex is carried by the vagi. Sneezing is another expulsive reflex. (Mitchell and Berger, 1975).

Chemical Control of Respiration:

The single most important factor in the control of respiration is the carbon dioxide tension (PCO_2) of blood perfusing the medullary chemoreceptors which are present in the respiratory centre. Respiratory adjustment also occur to combat increases in blood PH and dangerous falls in the oxygen tension (PO_2) of arterial blood. Carbon dioxide is a very soluble gas and readily crosses the blood brain barrier. Changes in the PCO_2 of brain interstitial fluid and C.S.F., and consecuently a change in brain and C.S.F-PH as CO_2 combines with water and forms carbonic acid. The fall in brain and CSF-PH stimulates the medullary chemoreceptors increasing the rate and depth of respiration. A fall in arterial PCO_2 has an opposite effect. The peripheral chemoreceptors in the aortic and carotid bodies are also sensitive to PCO_2 but play a minor role in maintenance of normocapnia (West J.B., 1979).

A fall in the PO_2 of arterial blood perfusing the aortic and carotid bodies to 8 Kpa begins to cause stimulation of the chemoreceptors and to increase the rate and depth of respiration. Stagnant hypoxia of the chemoreceptors is in part responsible for the

hyperventilation seen in circulatory shock. A rise in PO_2 does not depress respiration. The direct central effect of hypoxia is to depress the respiratory centre. (West J.B., 1979). Facilities for blood gas analysis are widely available in modern hospitals. Determination of arterial blood gases in the patient with serious post- operative and traumatic respiratory complications is essential for the proper management of these disorders. While a mild degree of hypoxaemia can be treated with added inspired oxygen, refractory hypercapnia and severe hypoxaemia demand institution of I.P.P.V. before cardiorespiratory arrest. (Nunn J., (1977).

Haemodynamic changes in health and disease:

In normal circulatory system, the blood is moving parallel to the axis of the vessels, i.e: undisturbed, and this results in laminar flow, where the central laminae have higher velocity than the peripheral laminae. When the blood flow becomes disturbed, due to stenosis, aneurysm or stasis, the blood elements move randomly, some at right angles to the axis of flow and this may result in thrombus and occlusion of the blood vessel. (Nicolaides, 1975).