بسم الله الرحمن الرحيم

شبكة المعلومـــــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـــســـم ﴾

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد اعدت دون آية تغيرات

يجب أن

تحفظ هذه الأفلام بعيداً عن الغبار

في درجة حرارة من 15 – 20 مئوية ورطوبة نسبية من 20-40 %

To be kept away from dust in dry cool place of
15 – 25c and relative humidity 20-40 %

بعض الوثائق الأصلية تالفة

بالرسالة صفحات

لم ترد بالأصـــل

EFFECT OF LOCAL MODERATE HYPOTHRMIA TO THE HEAD AND NECK ON CEREBRAL METABOLISM IN HEAD INJURED PATIENTS

Thesis

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By

Emad Fathy Fakher
MBBch. (Alex.)

Faculty of Medicine
Alexandria University

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SUPERVISORS

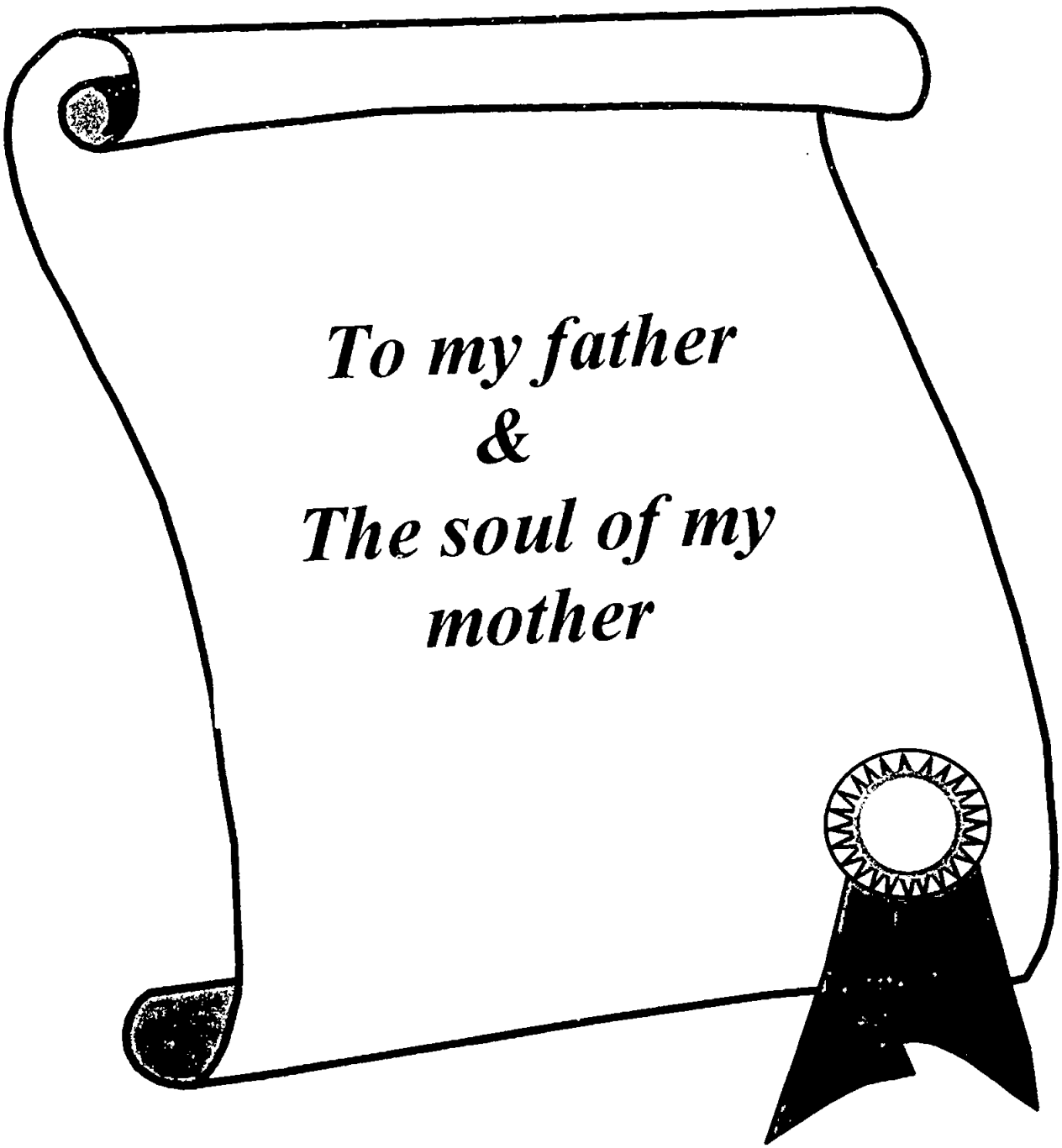
Prof. Dr. Hasan Abdel Aziz Abo Khabar
Professor of Anaesthesia & Surgical intensive care
Faculty of Medicine
Alexandria University

Prof. Dr. Nader Abdel Azem El Gamal
Assistant Professor of Anaesthesia & Surgical intensive care
Faculty of Medicine
Alexandria University

Dr. Yasser Mahmoud El Banna
Assistant Professor of Neurosurgery
Faculty of Medicine
Alexandria University

Co- Worker

Prof. Dr. Maryem Abo Sief Helmy
Professor of Clinical Pathology
Faculty of Medicine
Alexandria University


To my father
&
The soul of my mother

Acknowledgement

Praise to "Allah", the Most Gracious and the Most Merciful Who Guides Us to the Right Way.

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INTRODUCTION

INTRUDUCTION

Head injuries continue to be a major health problem, not only in terms of their frequency but also because of the fatalities and handicaps that result in the population they most often affect.[1]

Traumatic brain injury (TBI) was found to be responsible for 45-50% of all traumatic fatalities and remains the leading cause of death among trauma victims.[2] Road traffic crashes account for most head injuries. Significant injury to the head and neck is present in over 50% of all fatal road traffic accidents involving vehicle drivers and passengers.[3]

Epidemiology of head injury

The incidence of head injury in the United States is approximately 200 to 400 per 100,000 populations per year. Similar incidence rates have been documented for other countries. Male to female incidence ratios vary between 2:1 and 3:1, and incidence peak is in the second and third decades of life. It was estimated that, over 10 millions people experience head injuries each year, of whom several thousands will die, and several thousands will be permanently disabled.[4] While in Britain, it was estimated that there are 250-300 hospital admissions involving head injury per 100,000 of the population. It is found that about 75% of these injuries are mild to moderate and as few as 7% are severe.[5] In a study done in the ICU, at Alexandria Main University Hospital (Alexandria, Egypt), it was found that trauma ranks the second (19.7%) after circulatory diseases (28.3%) as a cause of ICU admissions. The same study revealed that head

trauma showed the highest percent (38.2%) of all trauma cases followed by trauma to extremities and chest (23.8% and 21.8% respectively), abdominal trauma showed the least percentage (16.2%) of all trauma cases.[6]

Etiology of head injury

The most common cause of closed head injury all over the world is road traffic accidents. These include injuries to vehicle occupants, pedestrians, motorcyclists, and bicyclists. Falls are the next most common cause of injury. While gunshot injuries are the major cause of penetrating head injury.[7]

Etiology of head injuries varies considerably e.g. with patients demographics, age, proximity to major highways ...etc. older patients are more often injured as a result of falls and have a higher incidence of mass lesions.[7]

Intoxication with alcohol or other agents is a significant cofactor in all causes of head injury and across virtually all age groups except the very young and very old.[7]

Pathophysiology of head injury

Brain trauma results in widespread abnormalities in brain anatomy, perfusion, metabolism and function. With closed head trauma, the initial impact results in mechanical disruption of the integrity of cellular and supporting tissue membranes.[8,9] Subsequently, numerous secondary phenomena results, that can cause the patient's condition to deteriorate.[10] Blood and oedema fluid accumulate as normal compartmentation is disrupted, secondary brain ischemia then assumes.[11]

All head injuries can be viewed as being caused by one of two phenomenae: contact forces or inertial forces. Since clinical injuries are rarely the result of only one mechanism, both contact and inertial forces may be involved in many of these situations.[12]

Contact forces

Contact forces are generated when an object strikes the head or when the head strikes an object, irrespective of whether the head is set into motion or is stopped by the blow.[13]

Local deformations of the skull produce local or remote compressive, shear, and tensile strains in the underlying skull and brain. Shock waves emanate from the point of impact and travel rapidly through the skull and brain creating additional tissue strains. These can summate with the strains produced by local deformation, the result of which can be skull fracture, contusion, and some of the intra or extra axial hematomas.[13]

Inertial forces

Inertial forces are generated by head motions that occur during the traumatic event. Most commonly described by acceleration or deceleration of the head as it is set into motion or stopped from moving.[12]

Magnitude of head accelerations or decelerations, rate of onset, and direction are the determinants of the type and severity of the inertial injuries. Impact over the brain is not necessary as long as sufficient head motion occurs to cause injurious levels of shear, tensile and compressive strains within the head.[12,13]

Concussive injuries, diffuse injury without hematoma (diffuse axonal injury) and most acute subdural haematomas are produced solely due to acceleration of the head.[12,13]

Brain metabolism and lactate in head injury

Cerebral oxygen consumption can be divided conceptually into two categories: the metabolic need to maintain structural integrity and the energy expended on neuronal functions[14]. At the whole organ level, the brain depends almost exclusively on the aerobic consumption of glucose for energy production. For many years, it has been assumed that both neurons and glia used glucose as their sole energy substrate, evidence is now emerging that glucose is consumed anaerobically in astrocytes, producing lactate that is released into the extracellular space and subsequently consumed aerobically by neurons. According to this schema, astrocytes and neurons are functionally coupled.[15]

Under physiologic conditions, the coupled astrocytic- neuronal unit leads to several important consequences on measurements of tissue level metabolism. Increased neuronal activity leads to increased astrocytic glycolysis, which is measured as the cerebral metabolic rate (CMR) of glucose. The lactate produced by the astrocytes is then consumed aerobically by neurons; this metabolic activity is reflected by CMR of oxygen. Because this lactate consumed by neurons is not derived directly from the blood, its consumption will not be reflected in arterivenous lactate difference.[16]