شبكة المعلومات الجامعية

بسم اللّه الرحمن الرحيم

شبكة المعلومات الجامعية

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

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

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

شبكة المعلومات الجامعية

بعض الوثائق الأصلية تالفة

شبكة المعلومات الجامعية

بالرسالة صفحات
لم ترد بالأصل

Traumatic Brain Injury

An Essay
Submitted for partial fulfillment of
Master Degree in Intensive care
By

AHMED KHALAF ABD EL - GHAFOUR
M.B.,B,CH

Under Supervision of :

PROF. DR. MOHAMED ABD EL- KHALEK MOHAMED ALI

Professor of Anaesthesiology and Intensive Care,
Ain Shams University

DR. HESHAM MOHAMED MAHMOUD EL-AZAZI

Assistant Professor of Anaesthesiology and Intensive Care,
Ain Shams University

DR. DALIA ABD EL-HAMID MOHAMED NASR EL-DIN

Lecturer of Anaesthesiology and Intensive Care,
Ain Shams University

Faculty of Medicine
Ain Shams University
2009

CONTENTES

DEDICATION

I dedicate this work to

My Parents and all members of

my family . For their help

and assistance.

INTRODUCTION

Traumatic brain injury (TBI ,also called **intracranial injury**) occurs when an outside force injures the brain . Traumatic Brain Injury can be classified based on severity (mild, moderate,or severe), mechanism (closed or penetrating head injury), or other features. Head injury usually refers to TBI, but is a broader category because it can involve damage to structures other than the brain, such as the scalp and skull . Traumatic brain injury is a major cause of death and disability worldwide, especially in young people. Causes include falls, vehicle accidents, and violence . Prevention measures include use of technology to protect those who are in accidents , such as seat belts and sports or motorcycle helmets, as well as efforts to reduce the number of accidents, such as safety education programs and enforcement of traffic laws. *(Rehman T, et al,2008)* .

. Severe traumatic brain injury is a problem approaching epidemic proportions in different parts of the world. Traumatic brain injury accounts for approximately 40% of all deaths from acute injuries in the United States . Approximately 52,000 US deaths per year result from TBI. The mortality rate for deaths outside the hospital is approximately 17 per 100,000 people; it is approximately 6 per 100,000 people for patients who are hospitalized. That incidence of mild TBI is about 131 cases per 100,000 people, the incidence of moderate TBI is about 15

cases per 100,000 people,and the incidence of severe TBI is approximately 14 per 100,000 people. *(Tieves KS,et al,2005)*

Brain trauma can be caused by a direct impact or by acceleration alone . In addition to the damage caused at the moment of injury, brain trauma causes secondary injury, a variety of events that take place in the minutes and days following the injury.These processes, which include alterations in cerebral blood flow and the pressure within the skull , contribute to the damage from the initial injury. This presents the opportunity to discover new treatments that limit damage by interfering with these cascades. *(Rehman T, et al.,2008)*

Diagnosis is suspected based on lesion circumstances and clinical evidence , most prominently a neurological examination . Neuroimaging helps in diagnosis and prognosis and in deciding what treatments to give . *(Barr RM, et al.,2007)*

The primary concerns are ensuring proper oxygen supply, maintaining adequate cerebral blood flow, and controlling raised intracranial pressure (ICP). Other methods to prevent damage include management of other injuries through surgery and prevention of seizures. *(Parikh S, et al.,2007)*

Prognosis worsens with the severity of injury. Most TBIs are mild and do not cause permanent or long-term

disability ; however, all severity levels of TBI have the potential to cause significant , long-lasting disability. Permanent disability is thought to occur in 10% of mild injuries, 66% of moderate injuries, and 100% of severe injuries. *(Brown AW, et al.,2008)*

Complications are distinct medical problems that may arise after TBI; they include physical, cognitive , emotional , and behavioral complications . Traumatic brain injury can cause prolonged effects on consciousness, such as coma , brain death , persistent vegetative state. Lying still for long periods can cause complications including pressure sores , pneumonia or other infections , progressive multiple organ failure , and deep venous thrombosis . *(Hall RC and Chapman MJ ,2005)*

PATHOPHYSIOLOGY

The knowledge of the pathophysiology after traumatic brain injury is necessary for adequate and patient-oriented treatment . As the primary insult , which represents the direct mechanical damage , cannot be therapeutically influenced , target of the treatment is the limitation of the secondary damage (delayed non-mechanical damage). It is influenced by changes in cerebral blood flow (hypo- and hyperperfusion), impairment of cerebrovascular autoregulation , cerebral metabolic dysfunction and inadequate cerebral oxygenation . Furthermore , excitotoxic cell damage and inflammation may lead to apoptotic and necrotic cell death . Understanding the multidimensional cascade of secondary brain injury offers differentiated therapeutic options .

(Werner C.and Engelhard K.,2007).

Physiological consideration:

The adult human brain weighs about 1350 gm ; and it receives 12 to 15 % of cardiac output and so high cerebral metabolic rate .Whole brain oxygen consumption is about 20% of total body oxygen utilization . Normal values of cerebral blood flow and cerebral metabolic rate are listed in table (1) .

(Ritter A.M and Robertson C.S.,1994)

NOMAL VALUES**:** Table 1: cerebral normal values

Cerebral blood flow:	.
Global.	**44-55ml / 100g / min**
Cortical (mostly grey matter) .	**75-80 ml / 100g/min**
Subcortical (mostly white matter) .	**20 ml /100g /min**
Cerebral metabolic rate for O2	.
.	**3 – 3.5 ml /100g /min**
Cerebral vascular resistance	.
	1.5 – 2.1 mmHg.100g.min / ml
Cerebral perfusion pressure	**about 80 mmHg**
Threshold of ischemia	**CPP < 50mm Hg**
Cerebral venous O2 tension	**32 – 44 mmHg**
Cerebral venous O2 saturation	**55 – 70 %.**
Intracranial pressure (supine)	**8 – 12 mmHg**

(Ritter A.M and Robertson C.S.,1994)

BASIC PRINCIPLES OF CEREBRAL BLOOD FLOW & BRAIN METABOLISM :

1- Cerebral hemodynamics **:**

(a) Anatomical and physiological considerations: .

. Cerebral blood flow is regulated by a number of factors , as arterial blood pressure , ICP , venous outflow , blood viscosity and *P*aCO2 and *P*aO2 . Each carotid artery contributes 40 % of the total cerebral perfusion , remaining 20 % coming from two vertebral arteries , which fuse to form basilar artery . The major fraction of venous blood draining from brain is collected in transverse sinuses which , together with the inferior petrosal sinuses , form the internal jugular veins . The distribution of capillaries is functionally organized throughout

the central nervous system . The capillary density is an anatomical indicator of oxidative metabolism . Brain areas with high basal levels of glucose metabolism contain a high density of capillaries. *(Zauner A and Paul J.M.1997)*

(b) Cerebral perfusion pressure and cerebrovascular resistance:

. The net driving force for the cerebral circulation is defined as the cerebral perfusion pressure (CPP) , which is the mean arterial blood pressure (MABP) minus the cerebral venous pressure . If the pressure within the thin-walled cortical veins is equal to or less than the external (CSF) pressure, the veins may collapse . Under most circumstances , however , the pressure in these veins is slightly above extravascular , (that is intracranial pressure) , in order to permit continuous flow . Because of the close relationship between cerebral venous pressure and ICP , CPP is generally defined as the difference between mean arterial pressure and ICP . In a normal brain , changes in CPP between 50 and 130 mmHg produce only minimal changes in CBF .A constant flow in this range of CPP is maintained by an increase or decrease in vascular resistance. Normal value of CPP (80 mmHg) is maintained by an increase or decrease in vascular resistance .Outside this range , cerebral vasodilatation or vasoconstriction cannot maintain a normal cerebral blood flow (CBF) . Since CPP = MABP–ICP and the flow in general equals pressure / resistance ; so CBF = CPP / CVR where CVR is cerebrovascular resistance .

Since vascular resistance is inversely proportional to the fourth power of vessel diameter , change in cerebral vessel diameter will produce marked change in resistance. *(Zauner A and Paul J.M.1997)*

(c) Effects of hematocrit on CBF: Blood viscosity is determined by factors , as erythrocyte size and concentration , shear rate , temperature , pH , plasma protein level and plasma lipid concentration . Clinical studies show an inverse relationship between CBF and hematocrit . *(Giller,et al,1993).*

2-Regulation of Cerebral blood flow :

(a) Arterial gas tension and cerebral vasoreactivity:

Hypercapnia relaxes cerebrovascular smooth muscles , whereas hypocapnia produces vasoconstriction . An arterial PaCO2 of 20–25 mmHg may reduce the CBF by 40–50% , and conversely an increase in PCO2 over 50 mmHg increases CBF by more than 50% . Also , an increase in PaO2 may lead to cerebral vasoconstriction and free radical formation . Changes in CBF are due to alterations in CO2 occur almost immediately in healthy brain , but the response may be altered after head injury . Carbon dioxide induces changes in the hydrogen ion concentration (modulating extracellular pH) . Experimental studies have shown that HCO3 solutions decrease CBF whereas low HCO3 solutions increase CBF. *(Giller,et al,1993)*