BRAIN INJURY MONITORING:
IMPLICATIONS AND RECENT MODALITIES

Essay

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The Master Degree (M.Sc.) in
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بسم الله الرحمن الرحيم

"ويسئلونك عن الروح قل الروح من أمر ربي
وما أوتيتم من العلم إلا قليلا"

صدق الله العظيم
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Wael Mahmoud Hossam

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ABBREVIATIONS

3D CT	:	3-dimension computed tomography angiography
AAJ	:	Atlantoaxial joint
ABP	:	Arterial blood pressure
ASVA	:	Atlantoaxial segment vertebral artery
ATPase	:	Adenosine tri-phosphatase
$AVDO_2$	:	Arteriovenous difference of oxygen
BAEP	:	Brainstem auditory evoked potentials
Ca^{2+}	:	Calcium
CBF	:	Cerebral blood flow
CBV	:	Cerebral blood volume
$CMRO_2$	:	Cerebral metabolic rate of oxygen
CO_2	:	Carbon dioxide
CPP	:	Cerebral perfusion
CSF	:	Cerebrospinal fluid
CT	:	Computed tomography
DNA	:	Deoxyribonucleic acid
DSA	:	Digital subtraction angiography
EAC	:	External auditory canal
ECG	:	Electrocardiography
EEG	:	Electroencephalography
EMG	:	Electromyography
EPs	:	Evoked potentials
GCS	:	Glasgow coma scale
GMP	:	Glutamate monophosphate
HbO_2	:	Oxygenated hemoglobin
Hz	:	Hertz
ICP	:	Intracranial pressure
ICU	:	Intensive Care Unit
IJV	:	Internal jugular vein
LDF	:	Laser Doppler flowmetry
LOC	:	Loss of consciousness
LP ratio	:	Lactate pyruvate
MAP	:	Mean arterial pressure
MD	:	Microdialysis
MIP	:	Maximum intensity projection

MTT	:	Mean transit time
NA	:	Not applicable
Na	:	Sodium
NICU	:	Neurosurgery intensive care
NIRS	:	Near infrared spectroscopy
NSU	:	Neuroscience critical care
$pBrO_2$	:	Oxygen extraction fraction
PET	:	Positron emission tomography
SEP	:	Somatosensory evoked potentials
$SjvO_2$	:	Jugular venous oximetry
SPECT	:	Single photon emission computerized tomography
TBI	:	Traumatic brain injury
TCD	:	Transcranial Doppler
TDF	:	Thermal diffusion flowmetry
VA	:	Vertebral artery
VEP	:	Visual evoked potentials

ABSTRACT

Central nervous system (CNS) trauma is a significant cause of morbidity and mortality all over the world. In USA, each year, 500,000 patients with head injury are seen in the emergency department and of these more than 50,000 die from their injuries . Major cause of this menace is cerebral ischemia that ensues minutes to hours after the primary head injury. This is commonly known as secondary injury. Secondary brain lesions resulting from cerebral metabolic and hemodynamic reactions can be prevented by neurocritical care management. It must be initiated as early as possible, ideally in a prehospital setting.

In the last decade, there has been significant progress in the area of neurotrauma management. Elucidation of important pathologic mechanism leading to secondary brain lesions, a better understanding of the consequences of therapeutic agents for brain physiology, and the development of multimodality monitoring have lead to changes in standard practice. First of all, the influence of initial critical care on outcome is now clearly documented. Critical care management of severe head injuries must be regarded as a continuum that starts with initiation of criticare at the scene, uses of multimodality cerebral monitoring for further adaptation of therapeutic agents to brain hemodynamic and metabolism and ends with intense neurologic rehabilitation.

The two most important secondary injury processes that can be monitored, anticipated, and treated in the head injured patient are intracranial hypertension and cerebral ischemia. Recent monitoring practices as well as most of the new technologies available for monitoring patients with a traumatic brain injury address one or both of these processes.

Keywords:

Brain Injury
Implications
Recent Modalities

INTRODUCTION

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Chapter I

NEUROPHYSIOLOGY

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PHYSIOLOGY OF CEREBRAL CIRCULATION:

The adult brain (1200–1400 g) comprises 2–3% of total body weight and receives 15–20% of cardiac output. The central nervous system has a high metabolic rate for oxygen (CMRO2) and uses glucose predominantly as the substrate for its energy needs. Although glial cells make up almost 50% of the brain, they consume less than 10% of total cerebral energy due to their low metabolic rate. Neurons expend most of the available energy. Fifty percent of the total energy generated is used for maintenance and restoration of ion gradients across the cell membrane, and the remaining 25% is used for molecular transport, synaptic transmission and other processes. Normal CBF in humans averages 50 mL/100 g brain tissue per minute. It is usually higher in children and adolescents and drops further with age. Irreversible neuronal damage occurs when CBF drops below 10–15 cc/100 g/min, whereas reversible neuronal injury occurs with CBF between 15 and 20 cc/100 g/min .Because the brain has no significant storage capacity, cerebral metabolism, CBF, and oxygen extraction are tightly coupled. This relationship is expressed by the Fick's equation: $CMRO_2 = CBF \times AVDO_2$, in which $CMRO_2$ represents cerebral metabolic rate for oxygen and $AVDO_2$, arteriovenous difference of oxygen. Under normal conditions, the brain maintains a constant AVDO2 by responding to changes in metabolism, cerebral perfusion pressure (CPP), and

blood viscosity with changes in vessel caliber, a phenomenon referred to as autoregulation.[1,2,3,4]

Cerebral Auto-regulation:

Definition:

Cerebral Auto-regulation defined as the maintenance of a constant level of CBF in the presence of alterations in the perfusion pressure. The normal physiological limits average 50 mm Hg and 150 mm Hg.

Mechanism:

1. The Myogenic theory:

The evidence supporting the *myogenic theory* consists of experiments in which alterations in transmural pressures have been shown to trigger immediate changes in the auto-regulatory response.[5]

2. The Metabolic theory:

It is based on the hypothesis that changes in the microenvironment alter vasomotor responses.

Factors affecting CBF:

(1) Variations in the partial pressure of arterial carbon dioxide ($PaCO_2$) At $PaCO_2$ levels within the normal range.
(2) The partial pressure of arterial oxygen (PaO_2).