Multimodality Monitoring in Neurocritical Care

Essay

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By

Hossam Mamdouh Kotb Saad
M.B.B.Ch.

Under Supervision of

Prof. Dr. Bassel Mohammed Essam Nour El-Dien
Professor of Anesthesiology and Intensive Care
Faculty of Medicine- Ain Shams University

Prof. Dr. Hazem Mohamed Abd-Elrahman Fawzi
Professor of Anesthesiology and Intensive Care
Faculty of Medicine- Ain Shams University

Dr. Yasser Ahmed Abd-Elrhman
Lecturer of Anesthesiology and Intensive Care
Faculty of Medicine- Ain Shams University

Faculty of Medicine
Ain Shams University
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Hossam Mamdouh Kotb Saad

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List of Abbreviations

Abb.	Mean
ABC	ATP-binding cassette
AEP	Auditory evoked potentials
A-VDO$_2$	Cerebral arteriovenous oxygen difference
BAER	Brainstem auditory evoked responses
BBB	Blood–brain barrier
BCRP	Breast cancer resistance protein
CAMAP	Compound muscle action potentials
CBF	Cerebral blood flow
CBV	Cerebral blood volume
cEEG	Continuous electroencephalographic monitoring
CEO$_2$	Cerebral oxygen extraction
CMRO$_2$	Cerebral metabolic rate of oxygen
CPP	Cerebral perfusion pressure
CSA	Compressed Spectral Array
CSF	Cerebrospinal fluid
CVO	Circum ventricular organs
DSA	Density Modulated Spectral Array
DWI	Diffusion-weighted imaging
EEG	Electroencephalogram
FV	Flow velocity
GCS	Glasgow Coma Scale
ICH	Intracerebral hemorrhage
ICP	Intracranial pressure
ICUs	Intensive care units
IEL	Internal elastic lamina
ISF	Interstitial fluid

Abb.	Mean
LDL	Low-density lipoprotein
MAP	Mean arterial pressure
MCA	Middle cerebral artery
MDR	Multidrug resistance
MRP	Multidrug resistance-associated proteins
MTT	Mean transit time
PbtO2	Direct brain tissue oxygen tension
PET	Oxygen-15-positron emission tomography
P-gp	P-glycoprotein
PVI	Pressure Volume Index
PWI	Perfusion-weighted imaging
rSO_2	Near infrared spectroscopy
SAH	Subarachnoid hemorrhage
SjVO2	Jugular venous oxygen saturation
SPECT	Single photon emission CT
SSEP	Somatosensory evoked potentials
SSS	Superior sagittal sinus
TBI	Traumatic brain injury
TCD	Transcranial Doppler Ultrasonography
tcMMEP	Transcranial magnetic motor evoked potentials
VEP	Visual evoked potentials
VPR	Volume Pressure Ratio

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INTRODUCTION

Multimodality monitoring is a recently developed method that aids in understanding brain physiology. Early detection of physiological disturbances is possible with the help of multimodality monitoring, which allows identification of underlying causes of deterioration and minimization of secondary brain injury. Multimodality monitoring is especially helpful in comatosed patients with severe brain injury because neurological examinations are not sensitive enough to detect secondary brain injury **(Kramer et al., 2011).**

Continuous monitoring of the central nervous system in the intensive care unit can serve two functions. Firstly it will help early detection of these secondary cerebral insults so that appropriate interventions can be instituted. Secondly, it can help to monitor therapeutic interventions and provide online feedback **(Dohmen et al., 2007*).***

While the pathophysiological mechanisms which cause brain injury are complex. Continued research in the basic and clinical sciences is making these mechanisms more clearly understood, with current knowledge it is clear

that there is little that we can do to reverse the primary damage caused by an insult **(van Santbrink et al., 2003)**.

The common secondary brain injurys are brain tissue hypoperfusion or ischemia due to intracranial pressure (ICP) surges, brain tissue hypoxia, brain tissue hypoglycemia, or excitotoxic damage due to recurrent seizures. Even though secondary brain injury is frequently encountered in the NeuroICU, neurological examination alone is not sensitive enough for monitoring on secondary brin injury because such patients are usually comatosed. Moreover, when the neurological examination shows worsening, it is usually too late to adequately treat, and permanent damage has already occurred **(Hamphill et al., 2011).**

Although serial cranial imaging such as computerised tomography (CT) or functional magnetic resonance imaging (fMRI)) provides useful information, they are neither continuous nor bedside monitors **(Miller and Suarez, 2005).**

Multimodality monitoring refers to the tracking of several parameters of brain physiology and function that can be affected by direct medical or surgical intervention. These parameters include brain electrical activity

(continuous electroencephalography-cEEG), brain oxygenation (jagular venous and brain tissue oxygen partial pressure-PbO_2), neurochemistry (cerebral microdialisis), intracranial pressure, and cerebral blood flow monitoring (transcranial Doppler ultra sound) **(Metz et al., 2003).**

AIM OF THE WORK

The aim of this essay is to highlight the new era of neurological monitoring regarding the different parameters and significance of each in critical care medicine.

CEREBRAL CIRCULATION

Introduction:

As an organ, the brain comprises only about 2% of body weight yet it receives 15–20% of total cardiac output making the brain one of the most highly perfused organs in the body. The brain is also unique in that it is enclosed by the skull which is a bony rigid structure that does not allow for expansion of either tissue or extracellular fluid without significant deleterious effects. Swelling of the brain due to vasogenic edema can increase intracranial pressure (ICP) and cause severe neurologic complications and even death **(Dunn et al., 2004).**

Because of the importance to maintain ICP within normal ranges and also to provide an appropriate ionic milieu for neuronal function, water and solute transport from the blood into the brain parenchyma is controlled in very special ways. The large arteries account for a- greater proportion of vascular resistance in the brain than in many other vascular beds. This prominent role of large arteries in vascular resistance likely helps to provide constant blood flow to neuronal tissue and protect the cerebral microcirculation during fluctuations in arterial pressure **(Hamel, 2006).**

Anatomy and Ultrastructure

The Arteries

The brain is one of the most highly perfused organs in the body. The arterial blood supply to the human brain consists of two pairs of large arteries, the right and left internal carotid and the right and left vertebral arteries. The internal carotid arteries principally supply the cerebrum, whereas the two vertebral arteries join distally to form the *basilar artery* **(Nishimura et al., 2007).**

Branches of the vertebral and basilar arteries supply blood for the cerebellum and brain stem. Proximally, the basilar artery joins the two internal carotid arteries and other communicating arteries to form a complete anastomotic ring at the base of the brain known as the circle of Willis **(Zlokovic, 2008)**.

The circle of Willis gives rise to three pairs of main arteries, the anterior, middle, and posterior cerebral arteries, which divide into progressively smaller arteries and arterioles that run along the surface until they penetrate the brain tissue to supply blood to the corresponding regions of the cerebral cortex **(Fig. 1) (Cipolla et al., 2004).**

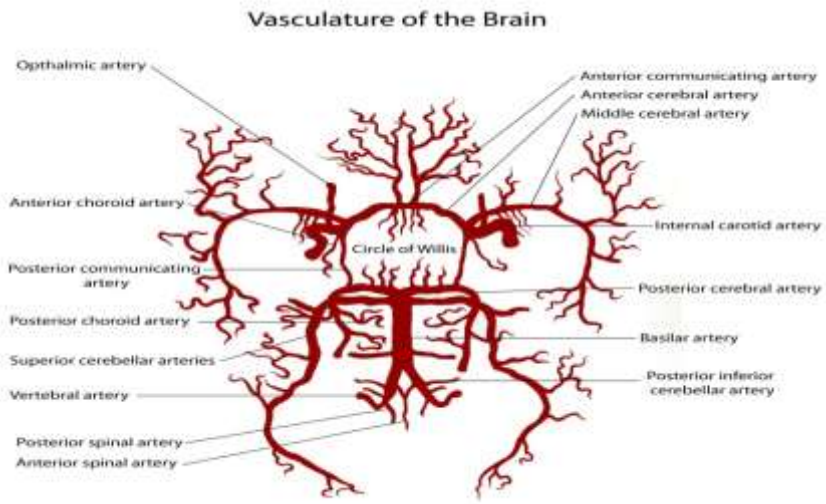

Fig. (1): The internal carotid and vertebral arteries: **(Cipolla et al., 2004).**

Cerebral Vascular Architecture

The pial vessels are intracranial vessels on the surface of the brain within the pia-arachnoid (also known as the leptomeninges) or glia limitans (the outmost layer of the cortex comprised of astrocytic end-feet). Pial vessels are surrounded by cerebrospinal fluid (CSF) and give rise to smaller arteries that eventually penetrate into the brain tissue **(Goadsby and Edvinsson, 2002)**.

Penetrating arterioles lie within the Virchow–Robin space and are structurally between pial and parenchymal arterioles. The penetrating arteries become parenchymal arterioles once they penetrate into the brain tissue and