# New Modalities in the management of Traumatic Intracranial Injury

#### **Essay**

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### Abbreviation

| aSDH  | acute subdural hematoma           |
|-------|-----------------------------------|
| BCVI  | blunt cerebral vascular injury    |
| BP    | blood pressure                    |
| BTF   | Brain Trauma Foundation           |
| BUN   | blood urea nitrogen               |
| CAI   | carotid arterial injury           |
| CBF   | cerebral blood flow               |
| CCF   | carotid cavernous fistula         |
| CCF   | carotid cavernous fistulas        |
| CMD   | Cerebral microdialysis            |
| CMRO2 | cerebral metabolic rate of oxygen |
| CPF   | cerebral perfusion pressure       |
| CSF   | contains cerebrospinal fluid      |
| CT    | Computed tomography               |
| CTA   | Computed Tomographic Angiography  |
| CVP   | central venous pressure           |
| DAI   | Diffuse axonal injury             |
| DC    | Decompressive craniotomy          |

| DFVA  | digital four-vessel cerebral angiography |
|-------|--|
| DTICH | delayed traumatic intra cerebral         |
|       | hematoma                                 |
| DVT   | deep vein thrombosis                     |
| DVTE  | developing venous thromoembolic          |
|       | events                                   |
| EDH   | Epidural hematomas                       |
| EEG   | electrocardiography                      |
| EEG   | Electroencephalogram                     |
| EMS   | ermedi-cal system                        |
| FLAIR | fluid attenuated inversion recovery      |
| GCS   | Glasgow coma scale The GCs               |
| HBOT  | Hyperbaric oxygen therapy                |
| HIE   | hypoxic-ischemic encephalopathy          |
| НМРАО | hexamethyl propyl eneaminoxime           |
| HTS   | Hypertonic saline                        |
| ICA   | internal carotid artery                  |
| ICH   | intracranial hemorrhage                  |
| ICP   | Intracranial pressure                    |
| ICU   | intensive care unit                      |

| IIT   | intensive insulin therapy            |
|-------|--------------------------------------|
| IJV   | internal jugular vein                |
| IVH   | intraventricular hemorrhage          |
| LMWH  | low molecular weight heparin         |
| LPR   | lactate: pyruvate ratio              |
| MAP   | mean arterial pressure               |
| MRI   | Magnetic resonance imaging           |
| NBCA  | N-butyl cyanoacrylate                |
| PaO   | partial pressure of oxygen           |
| PbtO2 | brain tissue oxygen tension (PbtO2)  |
| PCO2  | pressure CO2                         |
| PT    | parenteral nutrition                 |
| PTS   | posttraumatic seizures               |
| SAH   | subarachnoid hemorrhage              |
| SEP   | Sensory-evoked potentials            |
| SjvO2 | The jugular venous oxygen saturation |
| SOV   | superior orbital vein                |
| SSEPs | Somatosensory evoked potentials      |
| TBI   | traumatic brain injury               |
| TCD   | Transcranial Dopple                  |

### Abbreviation

| VAI | vertebral artery injury |
|-----|-------------------------|
| VBV | cerebral blood volume   |

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#### Introduction

The brain and the CSF have nearly the same specific gravity (only about 4 % different), so that the brain simply floats in the fluid (**Guyton and Hall, 2006**).

The brain neither sinks nor floats in the CSF but remains suspended in it. A human brain removed from the body weighs about 1,500 g, but when suspended in CSF its effective weight is only about 50 g. By analogy. Neutral buoyancy allows the brain to attain considerable size without being impaired by its own weight. If the brain rested heavily on the floor of the cranium, the pressure would kill the nervous tissue. (Saladin, 2003)

The primary function of the CSF is to serve as a shock-absorbing medium. It protects the delicate tissues of the brain and spinal cord from jolts that would otherwise cause them to hit the bony walls of the cranial cavity and vertebral canal (**Tortora and Nielsen, 2012**).

If the jolt is severe, however, the brain still may strike the inside of the cranium or suffer shearing injury from contact with the angular surfaces of the cranial floor. This is one of the common findings in child abuse (shaken child syndrome) and in head injuries and PC (Post Concussions) from auto accidents, boxing, and the like. (Saladin, 2003).

If lesion occurred on the opposite side, it's called contrecoup. It occurs mainly when a severe blow applied to the head, so, no damage occurs to the brain on the side of the blow but on the opposite side. (Guyton and Hall, 2006)

Skull fracture, intracranial hemorrhage, or cerebral injury can be caused in humans due to a strong impact to the head. The following 2 types of cerebral injuries are often observed: one type is cerebral contusion which is a local brain damage to the brain, and the other is diffuse axonal injury (DAI) which is a diffuse brain damage to the brain. In various head injuries caused by external impact, cerebral contusion and DAI mainly result in direct failure of the cerebral parenchyma. (Zhang et al., 2012)

Traumatic brain injury (TBI) continues to be an enormous public health problem, even with modern medicine in the 21st century. Most patients with TBI (75-80%) have mild head injuries; the remaining injuries are divided equally between the moderate and severe categories. The cost to society of TBI is staggering, from both an economic and an emotional standpoint. Almost

100% of persons with severe head injury and as many as two thirds of those with moderate head injury will be permanently disabled and will not return to their premorbid level of function. (Greenes, 2006)

Every year, millions of people sustain a head injury. Most of these injuries are minor because the skull provides the brain with considerable protection. The symptoms of minor head injuries usually go away on their own. More than half a million head injuries a year, however, are severe enough to require hospitalization. Learning to recognize a serious head injury, and implementing basic first aid, can make the difference in saving someone's life. In patients who have suffered a severe head injury, there is often one or more other organ systems injured. For example, a head injury is sometimes accompanied by a spinal injury. (Marx et al, 2002).

The management of head injury has been based on the concept of primary and secondary brain injury. The primary brain injury was defined as the irreversible pathology sustained at the time of trauma where the secondary brain injury has been considered the subsequent or progressive brain damage that occurs due to an evolving pathology following the primary insult. It has been the general contention that the primary injury is irreversible and management should be directed at preventing or treating secondary pathology. However it is now clear that some of the biochemical events are potentially preventable or even reversible if treatment is instituted early enough (Kaye, 2005).

Several functional assessment measures are commonly used in clinical practice to describe functional status, monitor response to therapies, and assess long-term outcome. The Glasgow outcome scale has been used for almost 30 years and has been the most commonly used tool in traumatic brain injury outcome research (**Diamond and Stewart, 2004**).

Head injuries include both injuries to the brain and those to other parts of the head, such as the scalp and skull. Head injuries may be closed or open. A closed (non-missile) head injury is one in which the skull is not broken. A penetrating head injury occurs when an object pierces the skull and breaches the dura mater. Brain injuries may be diffuse, occurring over a wide area, or focal, located in a small, specific area. Brain injury can be at the site of impact, but can also be at the opposite side of the skull due to a countercoup effect (Small, 2002).

It is common for head trauma patients to have drowsiness; but to be easily aroused, headaches, and vomiting after injury. If these symptoms persist > 1 or 2 days, a CT of the head is needed (Small, 2002).

CT advantages for evaluation of the head-injured patient include its sensitivity for demonstrating mass effect, ventricular size and configuration, bone injuries, and acute hemorrhage regardless of location. Other advantages include its widespread availability, rapidity of scanning, and compatibility with other medical and life support devices. Its limitations include insensitivity in detecting small and predominantly nonhemorrhagic lesions associated with trauma such as contusion, particularly when adjacent to bony surfaces. Early and repeated CT scanning may be required for clinical or neurologic deterioration, especially in the first 72 hours after head injury, to detect delayed hematoma, hypoxic-ischemic lesions, or cerebral edema (Davis et al., 2006).

Recovery in patients with neurologic deficits will vary. Patients with neurologic deficits who improve daily are more likely to recover. Patients who are vegetative for months are less likely to improve. Most patients without deficits have full recovery. However, persons who sustain