Hot Topics in Acute Care Surgery and Trauma

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Traumatic Brain Injury

WORLD SOCIETY OF EMERGENCY SURGERY

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Hot Topics in Acute Care Surgery and Trauma

Endorsed by the World Society for Emergency Surgery (WSES) and the American Association for Trauma Surgery, the series covers the most debated topics in acute care and trauma surgery, from perioperative management to organisational and health policy issues, to the complex management of acute and trauma surgical patients. It is a valuable resource for both trainees and acute surgical professionals.

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Foreword to the Series

Research is fundamentally altering the daily practice of acute care surgery (trauma, surgical critical care, and emergency general surgery) for the betterment of patients around the world. Management for many diseases and conditions is radically different than it was just a few years previously. For this reason, concise up-to-date information is required to inform busy clinicians. Therefore, since 2011 the World Society of Emergency Surgery (WSES), in a partnership with the American Association for the Surgery of Trauma (AAST), endorses the development and publication of the "Hot Topics in Acute Care Surgery and Trauma," realizing the need to provide more educational tools for young in-training surgeons and for general physicians and other surgical specialists. These new forthcoming titles have been selected and prepared with this philosophy in mind. The books will cover the basics of pathophysiology and clinical management, framed with the reference that recent advances in the science of resuscitation, surgery, and critical care medicine have the potential to profoundly alter the epidemiology and subsequent outcomes of severe surgical illnesses and trauma.

Pisa, Italy — Federico Coccolini
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Leonardo J. M. De Macedo Filho, Buse Sarigul, and Gregory W. J. Hawryluk

1.1 Introduction

Traumatic brain injury (TBI) is a frequent and important wounding mechanism affecting humans now and throughout history. Thanks to medical and technological advancements, even severe brain injury is now survivable in the majority of cases. Although it is often said that the brain injury field has been slow to advance and that it is behind other areas of medicine, the past century has seen tremendous improvement in our understanding of the condition, the resources for patient care, and in patient outcomes. Here we discuss the evolution of brain injury care and the modern neuromonitoring resources that are the end result of these advances.

Key modern advancements include the development of the Glasgow Coma Scale (GCS) and the advent of computed tomography (CT) scanning as well as the development of supportive intensive care. More recently, clinical practice guidelines and neuromonitoring have improved our care of brain-injured patients. Inspired by the landmark Monro-Kellie doctrine, modern therapeutic interventions have focused on decreasing intracranial pressure (ICP) and optimizing cerebral perfusion. This approach and relevant best practices have been central to the Brain Trauma

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Foundation's (BTF) influential guidelines first published in 1996. Use of these guidelines, now in their fourth edition, has been associated with improved outcomes. This chapter focuses on the evolution of TBI management from ancient times to recent advances in neurocritical care.

1.2 History of TBI

Historically, moderate and severe TBI (sTBI) were rarely survivable. Efforts to treat TBI date back to antiquity. Trepanation, the oldest known neurosurgical procedure, dates back to at least 10,000 BC. Human skulls with bony flaws that had the same shape as primitive surgical instruments from the same time period are well described [1–4]. Trepanation (from the Greek *trypanon*, drilling, opening a hole) is a surgical procedure that consists of removing a portion of the skull. This technique was widely used in antiquity and in the Middle Ages, continuing into the eighteenth and nineteenth centuries for therapeutic purposes, mainly in TBI. Trepanned skulls have also been found in prehistoric human cultures dating to the Neolithic period [1–4]. Evidence of bone remodeling in some archeologic specimens suggests that these efforts occasionally met with some success.

The *Edwin Smith Surgical Papyrus*, dated to 1700 BC, was discovered in 1862 but remained unpublished until 1930, when the Egyptologist James Breasted published an extensive, annotated translation of its contents. This papyrus is composed of 48 clinical cases, systematically described, starting with the head and descending through the thorax and spine, where the document is interrupted. Some of these cases describe head and skull trauma and injuries in a standardized format that includes a clinical description of the case, diagnosis, and a glossary that seeks to clarify technical terms [2, 4].

Hippocrates, known as "the father of medicine," documented procedures for management of skull fractures and contusions [5]. Three hundred years later, Aulus Aurelius Cornelius Celsus of Alexandria described epidural and subdural hematoma evacuation via trepanation. There is a long pause in the historical record in terms of subsequent descriptions of brain injury management, with the exception of Avicenna's discovery of cerebral vessel blockage in stroke and management modalities for acute stroke [6].

During the ancient and medieval eras, civilizations developed intricate amalgamations of logic and mythical/religious thoughts. Thus, concepts about the body, mind (or soul), illness, and health were intertwined with religious and cultural concepts [2–4, 7]. Moreover, in the medieval era, as a result of the decline of the western Roman Empire, the Arab world preserved the medical knowledge of the Greeks and Romans. Neuroanatomy, neurophysiology, neuropathology, and surgical technique studies returned in the eleventh century with the work of Roger of Salermo during the Renaissance [3, 4]. At the end of the thirteenth century, Lanfrancus (−1310) elaborated the concept of concussion. In the fifteenth century, Berengario da Carpi (1465–1527) divided brain injuries into lacerations, contusions, and perforations. In addition, he described postconcussion headache [8].

Investigations by early Egyptian physicians, Hippocrates, Galen, Aulus Cornelius Celsus, and Paul of Aegina led to a better understanding of neurological anatomy, physiology, and therapeutics [9–11]. Their studies also improved our knowledge about cerebrospinal fluid (CSF). Modern concepts of ICP were first introduced by Monro and Kellie in the eighteenth century [10–13].

CSF is an ultrafiltrate produced by the choroid plexus and is present in the cerebral ventricles and subarachnoid space. It is in close relationship to CNS tissue and meninges [12, 13]. CSF was first identified by Nicola Massa in 1538 [13, 14] and was observed by Domenico Felice Cotugno in 1764 beneath the dura mater, within the brain's ventricles, and around the spinal cord [13, 15]. Moro Secundus (1733–1817) described the intraventricular foramen which provides a connection between the lateral ventricles and the third ventricle [11, 14, 16]. The CSF circulation and the correct direction of the flow were confirmed by Francois Magendie (1783–1855) who discovered that the continuation of CSF flow from the ventricular system to subarachnoid space was through the mid-region of the fourth ventricle [13, 17]. Alexander Bochdalek (1801–1883) described the lateral recesses of the fourth ventricle in 1849 and Hubert von Luschka (1820–1875) discovered the connections with the subarachnoid space—known as the foramina of Luschka—and confirmed the presence of the foramen of Magendie [13, 18]. The explanation of how CSF is secreted by the choroid plexus, flows through the ventricular system, and is reabsorbed via subarachnoid villi and Pacchionian granulations was added by Retzius and Key in 1875 [13, 19]. The link between CSF and ICP was defined by Harvey Cushing when he considered CSF to be the third circulatory system [13].

The Monro-Kellie doctrine established that the brain resides in an inelastic and rigid skull. The total intracranial volume has to remain constant. Moreover, along with the consistent volume of blood inside the cranium, the venous blood should be drained perpetually and replaced via arterial oxygenated blood [10, 13, 16]. An increase in the volume of intracranial CSF, brain tissue, or blood should be compensated by a decrease in other components. Otherwise, an increase in ICP is inevitable [10, 13, 16].

During the nineteenth century three major innovations made possible great advances in neurosurgery: anesthesia, antisepsis and aseptic technique, and brain topography [20]. These innovations resulted primarily from a period of consecutive wars and efforts to treat and reduce morbidity and mortality of TBI [8]. The notable brain injury of Phineas Gage in the 1800s brought attention to the localization of function in the brain after he survived an accident in which an iron rod penetrated his head and destroyed a good portion of his left frontal lobe, leading to marked behavioral change [21]. As the twentieth century began, the "neuron theory" was described by Santiago Ramon y Cajal (1852–1934). He postulated that the nervous system constitutes independent cells and defined the nervous system to include neurons that are in contiguity but not continuity [22]. Cajal was the first to use the term "plasticity" in a Congress held in Rome in 1894 in which he described the potential of the brain to adapt to the environment as a force of internal differentiation and plasticity. Until the 1960s, it was considered that the adult nervous system was incapable of generating new neurons. However, Joseph Altman and Gopal Das used

thymidine-H autoradiography to discover newly formed cells, which suggested new neuronal production to the olfactory bulb and the dentate gyrus of the rat hippocampus. These ideas became controversial until two decades later, when Arturo Alvarez-Buylla made his discoveries on neurogenesis and adult neural stem cells via experiments on songbirds and mammals [23]. However, these new discoveries have still not been applied to therapeutic advances in TBI.

The development of neurosurgery accelerated in the first half of the twentieth century. Harvey Cushing (1869–1939) is credited with significant reductions in complications and mortality in cranial surgery. Among his many contributions, he is credited with techniques used to treat head injuries such as subtemporal decompression, which is still frequently used today [8].

Other important developments in the twentieth century were the creation of the GCS and dramatic advances in brain imaging [8, 24]. Also, in the last decades of the twentieth century, the mortality rate for sTBI fell by almost 50% as a result of advancements in supportive care [25].

The BTF, founded in 1986, developed the first evidence-based clinical practice guidelines produced by any surgical specialty. The identification and proliferation of best practices has been repeatedly credited with marked improvement in outcomes from sTBI. The BTF has subsequently produced guidelines on many TBI subtopics including pediatric injuries, prehospital care, prognostication, combat injuries, and concussion. To date, the BTF has published over 15 major guideline projects/editions (Fig. 1.1). Compliance with these guidelines is integral to the American College of Surgeons' trauma center accreditation program and has

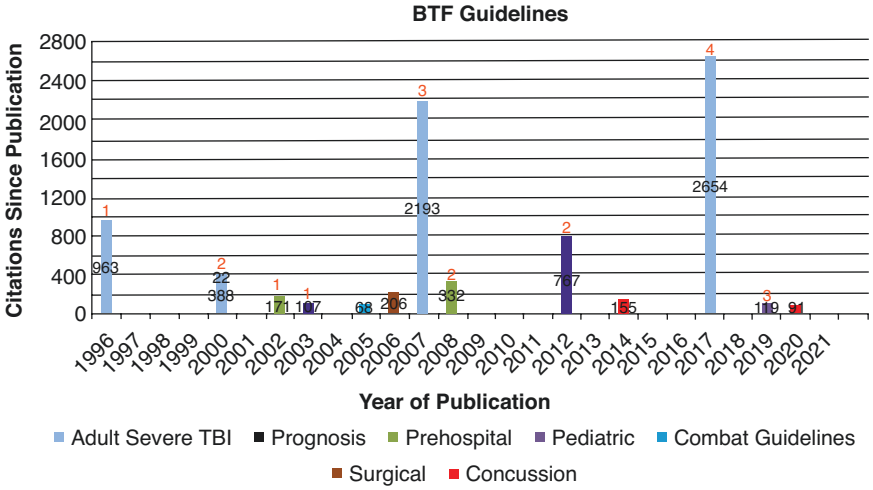

Fig. 1.1 Year of publication and citations of BTF guidelines. The cumulative number of citations calculated by Google Scholar (accessed December 12, 2022). The numbers in red above the bars denote the edition of the guideline. The number within the bars represents the total number of citations

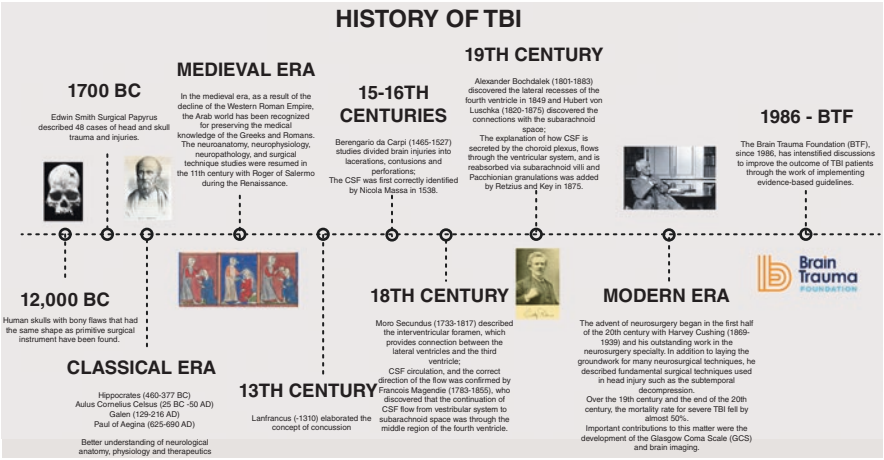

Fig. 1.2 History of TBI—timeline. The x-axis shows important contributions to the understanding of TBI over the centuries and eras, from the Neolithic to the present day, with emphasis on the BTF guidelines and studies on the subject. (Credits: (1) Anterior aspect of Squiers, Inca Skull, showing trephining. Wellcome Collection. Attribution 4.0 International (CC BY 4.0); (2) Les merveilles de l'industrie ou, Description des principales industries modernes/par Louis Figuier. - Paris: Furne, Jouvet, [1873–1877]. - Tome III. PublicDomain; (3) Cranial operation from BL Sloane 1977, Image taken from f. 2 of Chirurgia. Written in French. British Library. Public Domain; (4) Portrait of Gustaf Retzius, extracted from the article Gustaf Retzius som etnograf in Fataburen Kulturhistorisk tidskrift (1919). Nordiska Museet. Public Domain; (5) HarveyWilliams Cushing. Photograph, 1938. Created 1938. Harvey Cushing (1869–1939). Wellcome Collection. Attribution 4.0 International (CC BY 4.0))

intensified discussions on improving the outcome of TBI patients [26–30]. Development and widespread adoption of the BTF guidelines is only a recent advance in the long history of TBI treatment (Fig. 1.2).

1.3 Evolution of Neuromonitoring

1.3.1 Historical Evolution of Intracranial Pressure Monitoring

In 1891, the German physician Heinrich Quincke published the first description of the lumbar puncture technique as well as subsequent investigations of CSF and CSF pressure in relation to various neurological diseases (Fig. 1.3). He determined that a pipette of glass should be affixed to the puncture needle, and through the water column it was possible to measure the CSF pressure [11, 31]. This technique of repetitive CSF opening pressure measurement for assessment of ICP became widely used, becoming the first method for clinical assessment of ICP [11, 31]. However, this method led to the death of some patients with high ICP, presumably by inciting transtentorial herniation [11].

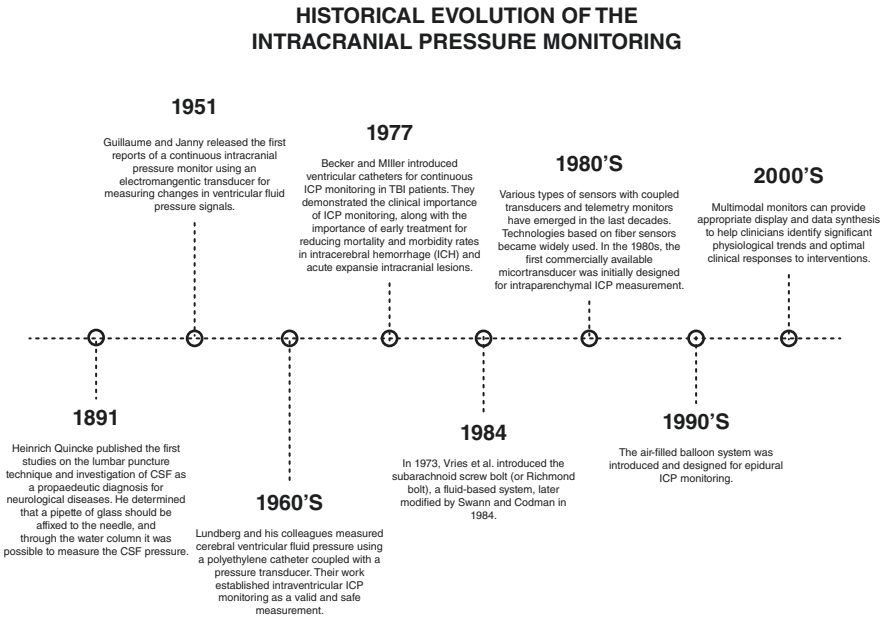

Fig. 1.3 Historical evolution of intracranial pressure monitoring—timeline. The *x*-axis shows contributions from the first studies by Quincke in the nineteenth century to the current multimodal monitors used in TBI

In 1951, Guillaume and Janny released the first reports of a continuous ICP monitor that used an electromagnetic transducer for measuring changes in ventricular fluid pressure signals. They used a U-tube manometer in which the CSF continues to flow until it is equalized by a reverse pressure [10, 11, 13, 32]. ICP monitoring was further advanced in the 1960s by Lundberg and his colleagues, who measured cerebral ventricular fluid pressure using a polyethylene catheter coupled to a pressure transducer. Their work established monitoring of intraventricular pressure as a valid and safe alternative [33–35]. The aim of Lundberg's thesis was to provide a method for ventricular cannulation that was minimally traumatic, feasible, had a low risk of infection and leakage, and facilitated recording with continuous flow of the ICP. In addition, he described three ICP wave patterns associated with intracranial pathologies [11, 33–35]. "A" waves represented increase in ICP to levels of 50 to 100 mmHg that maintained a plateau for 5–20 min, followed by an abrupt drop. "B" waves were abrupt rises in ICP up to 50 mmHg, with a frequency of 0.5–2 waves per minute. These waves could be directly related to cerebral blood flow (CBF) and vessel diameter but were of uncertain origin and relevance. "C" waves, also known as Mayer's wave, represented arterial wave reflexes and were associated with cardiac and respiratory cycles [11, 33–35].

In 1977, Becker and Miller introduced ventricular catheters for continuous ICP monitoring in TBI patients. They demonstrated the clinical importance of ICP monitoring, along with the importance of early treatment for reducing mortality and

morbidity rates in intracerebral hemorrhage (ICH) and acute expansile intracranial lesions. The clear evidence of good results among patients in whom ICP elevation could be quickly recognized and treated contributed to the popularization of the method [11, 36, 37]. Between the 1980s and 2000s, ICP monitoring became widespread. However, even today, it is not being used routinely in all ICUs. Also, cost and access limit use in low- and middle-income countries [27–30, 38, 39].

Recommendations that focus on the reduction of ICP and maintenance of adequate cerebral perfusion are central to the BTF guidelines, which review the varying levels of evidence for three types of monitoring in sTBI patients: ICP, cerebral perfusion pressure (CPP), and brain oxygenation [27–30]. Additional monitoring modalities mentioned in the recent guidelines regarding their use for diagnostic, therapeutic, and prognostic purposes include electroencephalography (EEG), partial pressure of brain tissue oxygen ($PbtO_2$), CBF, transcranial Doppler ultrasonography (TCD) for cerebral autoregulatory status, and cerebral microdialysis [11].

1.3.2 ICP Monitoring in Modern Era

1.3.2.1 Invasive ICP Monitoring

ICP can be measured via either invasive or noninvasive methods. Invasive methods include fluid-based systems and implantable microtransducers. Invasive ICP monitoring techniques consist of the insertion of a catheter, which varies in intracranial location and in the pressure transduction method. The devices are typically inserted in the intraventricular or intraparenchymal spaces. Regarding pressure transduction methods, catheters can be connected to an external ventricular drain (EVD) or a microtransducer [11, 13, 40].

The ventricular catheter is traditionally considered as the "gold standard" for reliability in ICP monitoring. The superiority of this technique when compared to others is that it allows CSF drainage for control of the ICP as well as biochemical, cytological, and microbiological CSF sample analysis [11, 13, 40, 41]. In 1973, Vries et al. introduced the subarachnoid screw bolt (or Richmond bolt) [42], a fluid-based system, later modified by Swann and Codman in 1984 [11, 43] in an attempt to reduce the infection rates of ventricular catheters at that time. However, the screw still presented a high risk of infection without allowing CSF drainage. It also had a tendency to underestimate ICP, which inspired the development of newer technologies [11, 13, 41].

Various types of sensors with coupled transducers and telemetry monitors have emerged in the last decades. Technologies based on fiberoptics, strain gauges, and pneumatic sensors are now widely used [11]. In the 1980s, the first commercially available microtransducer was introduced. This was the Honeywell MTC-P5F®, initially designed for intraparenchymal ICP measurement [44]. The first equipment to be used more widely were the Camino [45] and Codman [46] devices. Available technologies for ICP monitoring are made by a relatively small number of manufacturers. Each product or technology has its own benefits and weaknesses related to the technology itself or to the manufacturing process [11, 13, 30, 41, 47–50].

ICP monitoring also affords the opportunity to determine CPP, which represents the vascular pressure gradient that drives oxygen delivery to cerebral tissue. It is calculated as the difference between mean arterial pressure (MAP) and ICP. Decreases in CPP may contribute to secondary brain injury through cerebral hypoperfusion and/or ischemia. The BTF recommends (Level IIB) targeting a CPP between 60 and 70 mmHg—depending upon autoregulatory status—to optimize survival and favorable outcome [11, 27–30].

An ideal monitor for tracking ICP must be easy to use, accurate, reliable, reproducible, inexpensive, and must be associated with minimal infections and bleeding complications. Invasive transducers are reliable and accurate; however, cost and access to the technology are issues that limit its widespread use [11, 13, 50]. EVD catheters are the gold standard for monitoring ICP, despite having a higher risk of hemorrhage and infection than microtransducers [11, 13, 50].

1.3.2.2 Noninvasive ICP Monitoring

A noninvasive ICP monitor can be defined as a technique that provides information on ICP or the neurological consequences of increased ICP, such as reduced CBF and metabolic changes, without penetrating the skin or skull, thus minimizing the risks to the monitored individuals [51, 52]. Noninvasive modalities may represent the future of ICP monitoring because of their lower risk and greater cost efficiency [11, 13, 50–52]. Noninvasive monitoring methods are divided into two groups, those that use physiological parameters related to intracranial compartments, and those based on extracranial compartments that are anatomically connected to intracranial compartments [52].

Since the 1970s, there has been a strong effort to develop noninvasive monitoring to avoid complications associated with invasive ICP monitoring techniques. Consequently, many different noninvasive modalities have been developed in recent decades and are being studied [11, 13, 50]. The most popular noninvasive ICP monitoring techniques in TBI are brain imaging analysis; optic nerve sheath diameter (ONSD); TCD; tympanic membrane displacement; EEG; near-infrared spectroscopy (NIRS); pupillometry; microdialysis; pressure on the anterior fontanelle via fontanometry; venous ophthalmodynamometry; tonometry; acoustoelasticity; and otoacoustic emissions [11, 13, 50, 53, 54].

Fontanometry

Over the 1970s and 1980s, many studies were conducted to investigate the correlation between anterior fontanelle pressure and ICP in children with open fontanelles [50, 54–56]. Fontanometry is a method developed to measure the pressure beneath the fontanelle and thus provide information about ICP. It is based on placing sensors over the patent anterior fontanelle of children younger than 2 1/2 years. Device attachment has been an important and persistent concern with this technique. The best-known of these devices is the Rotterdam® transducer, which has been used in clinical practice [50, 54–56].

Optic Nerve Sheath Diameter

In 1964, Hayreh et al. [57] showed that, due to the communication of the subarachnoid space with the intracranial cavity, changes in CSF pressure can be transmitted along the optic nerve sheath. Therefore, when there is an increase in CSF pressure, the optic nerve sheath diameter (ONSD) can expand [54, 57]. The optic nerve sheath is continuous with the brain dura mater and is surrounded by the subarachnoid space, which contains the CSF [41]. ONSD expansion may be accompanied by papilledema, but unlike papilledema, ONSD expansion occurs almost immediately after an acute increase in ICP [54, 58]. ONSD sonographic measurement is a rapid modality for monitoring ICP increase. However, measuring ONSD is an operator-dependent technique, and conditions including tumors, inflammation, sarcoidosis, and Graves' disease can affect ONSD measurements. It is also difficult to measure ONSD in patients with orbital or optic nerve injuries [50, 54, 59].

Ophthalmodynamometry

Ophthalmodynamometry was originally described by Baurmann [60] in 1925 and consists of measuring the pressure in the ophthalmic artery and vein through an application of known pressure to the eyeball. In 2000, Firsching et al. observed that the venous outlet pressure has a close linear relationship with ICP [61]. The central retinal vein passes through the optic nerve and is surrounded by CSF, and changes in ICP can affect the optic nerve and central retinal vein. Like other ophthalmic ICP monitoring techniques, venous ophthalmodynamometry can be used to screen patients with a suspected increase in ICP before performing an invasive technique. It cannot replace invasive techniques. However, it can be used as a follow-up screening tool in some patients [50, 54, 61].

Tympanic Membrane Displacement

Reid et al. published the first study to compare tympanic membrane displacement (TMD) values with ICP measured via invasive methods in 1990 [62]. Three essential criteria are necessary to perform a tympanometry test: patent cochlear aqueduct, normal middle ear pressure, and intact stapedius reflex. In normal circumstances, the pressure in the intracranial compartment is transmitted to the perilymphatic fluid of the cochlea and thus displaces the stapedius, changing the acoustic reflex. Changes in ICP are thus transmitted through the cochlea, allowing indirect measurement of ICP. When a baseline ICP is established, TMD is useful to calculate normal or raised ICP, and repeated TMD measurements could be used to find changes in ICP [50, 54, 62, 63].

Brain Imaging

A variety of brain CT scan findings, such as loss of gray and white matter differentiation, midline shift, and basal cistern and ventricular effacement, have been associated with elevated ICP, and CT still remains the most-used diagnostic modality in the evaluation of patients with TBI. However, present evidence suggests that CT is not a very sensitive tool in the sense that CT may remain normal even with a raised

ICP [50, 63, 64]. Conversely, Rotterdam and Marshall criteria including midline shift, presence of space-occupying lesions, and status of basal cisterns have been suggested to be predictive of ICP increase [65, 66].

The current role of brain MRI as a diagnostic and monitoring tool in neurosurgery far outweighs its role as a purely noninvasive technique for assessing ICP. MRI techniques for the assessment of ICP are based on the relationship between intracranial compliance and pressure. MRI has also been used to assess optic nerve sheath diameter as a marker of elevated ICP and appears to be more accurate than ultrasound in assessing the CSF-filled subarachnoid space surrounding the optic nerve [63, 67, 68].

Tissue Resonance Analysis

The tissue resonance technique was developed by Michaeli [69] in 2002. It is based on the premise that different tissues vibrate at different frequencies when exposed to a particular sound wave in order to digitally obtain an echopulsogram, which shows a good correlation with invasive ICP. This method is a promising technique for noninvasive ICP monitoring, but it requires further validation [50, 54, 69].

EEG

The EEG represents the spontaneous electrical activity of the cerebral cortex recorded through electrodes placed on the scalp. These electrical signals are then amplified, filtered, and displayed in an 8- or 16-channel system [50, 54]. Aside from the importance of detecting the seizures and subclinical seizures that are common after TBI, many studies show that neurophysiological changes precede ICP changes [70, 71]. Moreover, certain components of EEG spectrum analysis are useful in correlating with ICP. EEG power spectrum analysis was reported in 2012 by Chen. Power spectral analysis allows a graphical representation of EEG readings over time and produces an ICP index (IPI) which correlates to ICP. However, more studies are needed to establish the correlation of EEG spectrum analysis with changes in ICP [50, 72].

Pupillometry

Examination of the pupils has long been a part of neurological assessment. Advances in technology have resulted in development of infrared pupillometry to quantitatively measure subtle changes in pupil size in response to light stimuli, establishing that the velocity of pupillary constriction is sensitive to increases in ICP and that a 10–20% reduction in pupil size is associated with intracranial hypertension [73–75]. In 2003 Taylor et al. suggested a new point-and-shoot hand-held pupillometer for quantitative evaluation of pupillary function. Their study enrolled 404 subjects. It was concluded that pupillary changes may suggest subtle changes in ICP, and the velocity of pupillary constriction was sensitive to increased ICP. A reduction of pupillary size by 10% was associated with ICP levels higher than 22 mmHg [74].

In 2011, Chen [76] introduced the concept of the pupillary neurological index using an algorithmic approach to predict changes in ICP with pupillary reactivity. This algorithm is produced by combining such parameters as minimum and

maximum pupillary diameter, the latency of the light reflex, and constriction and dilation velocity. The index includes a scale ranging from 0 to 5 points, and <3 is considered as abnormal. Quantitative pupillometry is shown to be more precise and more consistent than standard flashlight pupil assessment, especially in neurological intensive care units. Conversely, pupillometry has limitations. Evaluation of agitated or confused patients and patients with scleral edema, periorbital edema, intraocular lens replacement, and prior ocular surgery may be challenging. Moreover, the measurements may be affected by the light of the environment [77]. Although promising, the clinical applicability of this technique requires further investigation [50, 54].

TCD

In 1982, Aaslid [78] described TCD as a technique for evaluating cerebral hemodynamics, and since then, it has been used to measure the blood flow velocities and the cerebral vasoreactivity in the basal brain arteries and in the Circle of Willis, albeit mainly in the context of aneurysmal subarachnoid hemorrhage and vasospasm. The most commonly evaluated parameters using the arterial waveform are peak systolic and diastolic velocity, mean velocity, resistance index, and pulsatility index [50, 54, 69, 79, 80].

The measurement is made over regions of the skull with the thinnest bone windows (temporal, transorbital, or back of head). TCD is best suited to provide a qualitative estimate (low, normal, or high) of ICP. It appears to be a promising modality for noninvasive ICP monitoring, but it cannot replace invasive monitoring. Important disadvantages are the requirement for a trained and qualified operator to perform and interpret the measurements and the limited accuracy in estimating absolute ICP values [50, 54, 79, 80].

Near-Infrared Spectroscopy (NIRS)

Near infrared is the name given to the region of the electromagnetic spectrum immediately above the visible region in terms of wavelength. NIRS is an emerging technology that works on the principle of differential absorption of infrared light to detect changes in oxygen and deoxyhemoglobin concentration of blood. NIRS works with wavelengths of 700–1000 nm, where the low absorption allows it to easily pass through skin and bone, resulting in deep tissue penetration that enables it to measure regional changes in cerebral blood oxygen saturation (rSO_2) and cerebral blood volume. Moreover, it can be used to detect changes in CBF and ICP [50, 54, 81–84].

In 1997, Kampfl demonstrated a significant difference in rSO_2 values between normal and elevated ICP in sTBI patients [81], and changes in cerebral oxygenation correlated well with ICP vascular slow waves during CSF infusions and TBI studies [82]. NIRS allows the calculation of certain indices that have been correlated with cerebrovascular pressure reactivity in TBI patients [67]. However, it does not provide an absolute estimate of ICP or facilitate the detection of changes in ICP [50, 54, 84]. This method shows promise, but it cannot currently be used to estimate ICP values [50, 54, 84].

1.3.3 Ancillary Monitoring

1.3.3.1 Cerebral Autoregulation and CBF

Cerebral autoregulation (CA) is defined as the mechanism by which the brain maintains a constant nutrient supply across a breadth of physiologic conditions. CBF is directly proportional to CPP and the fourth power of vascular diameter, and it is indirectly proportional to blood viscosity and cerebral vascular length. CPP is determined by the difference between MAP and ICP [85–87]. The CA curve was first described by Lassen in 1959 [88] as a triphasic curve, and it was suggested that the brain is capable of maintaining a constant perfusion pressure throughout a wide range of mean arterial pressures [85–87]. A systematic review and meta-analysis by de-Lima-Oliveira [86] in 2018 selected 35 studies about the relationship between CA and ICP since the 1980s and observed that there was a clear tendency toward CA impairment with increased ICP [86]. At least four mechanisms are proposed for autoregulation: myogenic (vascular changes), neurogenic (vascular autonomic nerve supply), metabolic (changes in the microenvironment such as pCO_2 and H^+), and endothelial factors (such as nitric oxide) [85]. The assessment of cerebral autoregulation could be static (relationships between CBF and MAP are considered constant) or dynamic (assessment is based on determination of dynamic changes of CBF in response to dynamic changes in MAP) [85].

Cerebral autoregulatory status may also be determined via measuring the cerebrovascular pressure reactivity index (PRx) or the CBF velocity via TCD and near-infrared spectroscopy [13, 54, 85–87]. Increasing CPP, in some cases, may be the only way to increase oxygen delivery to the brain, but this has some costs. Vascular regulation in the traumatized brain is often impaired, causing dissociation between the CBF and the cerebral metabolic demand. Therefore, measuring the CBF may be more important in severely traumatized patients. Thermal diffusion flowmetry and laser Doppler flowmetry are some methods for measuring CBF [85–87].

The concept of the PRx was introduced by Czosnyka [89] in 1997 based on the principle that in MAP elevations there would be cerebral vasoconstriction with a reduction in cerebral blood volume and, consequently, in ICP. PRx reflects the smooth muscle tone of arteries and cerebral arterioles in response to changes in transmural pressure, forming part of the more elaborate physiological phenomenon of CA. The PRx indirectly reflects the CA status and may be utilized to delineate the optimal CPP for a patient [85–87]. In 2000, Luzius Steiner suggested the U-shape relationship between PRx and CPP. He and his colleagues demonstrated that the lowest level in this curve correlated with the CPP level that was associated with the best autoregulation, and this level was termed as the optimal CPP or CPPopt [90]. This was interpreted as the middle point of the upper and lower levels in Lassen's curve. The COGiTATE trial is currently investigating whether therapy based on the targeted value for CPPopt improves outcome [91].

Usually, CA maintains normal CBF when MAP is between 60 and 140 mmHg. CBF of 50–60 mL/100 g/min at a MAP of 80–100 mmHg is normally maintained by vasodilation (when MAP drops to the limit of 60 mmHg) or cerebral

vasoconstriction (when MAP rises up to the limit of 150 mmHg), which protects the brain from ischemia or hyperemia despite the physiological fluctuations of CPP. Patients with TBI may have a decrease or loss of CA. In this case, the CBF becomes dependent on the MAP. So if MAP rises, CBF rises too and can cause an increase in brain volume. If MAP drops, CBF also decreases, reducing ICP but possibly causing ischemia and necrosis [84–87].

1.3.3.2 Cerebral Oxygenation

Jugular Venous Oxygen Saturation ($SjvO_2$) and Arterio-Jugular Differences of Oxygen ($AVDO_2$)

Brain oxygenation may be monitored via two invasive modalities: jugular bulb oxygen saturation and $PbtO_2$ by the insertion of a catheter in the brain parenchyma. NIRS is a noninvasive bedside monitoring technique which detects changes in oxygen and deoxyhemoglobin concentration similarly to pulse oximetry [50, 54, 92]. Jugular bulb oximetry ($SjvO_2$) reflects the difference between brain oxygen and brain metabolic rate of oxygen, assuming that arterial oxyhemoglobin saturation, hemoglobin concentration, and the oxygen/hemoglobin dissociation curve remain stable [93, 94]. Myerson [95] first described the percutaneous sampling and analysis of human cerebral venous blood from the jugular bulb in 1927. Gibs [96] observed the arteriovenous difference between oxygen, glucose, and lactate. Moreover, he proposed that cerebral venous blood oxygen saturation measurement allows an estimate of global metabolic demand in relation to oxygen consumption [94]. Catheterizing the dominant internal jugular vein to correctly assess global cerebral oxygenation is recommended for this type of monitoring. The catheter tip should be positioned in the jugular bulb and placement confirmed by lateral skull radiography. $SjvO_2$ provides an indirect measure of CBF. If it is low (<50% for more than 10 min duration), it may reflect hypoperfusion (decreased supply) or an increase in cerebral metabolism (increased demand) [30, 93, 94].

The arteriovenous difference in oxygen supply ($AVDO_2$ = $CMRO_2$/CBF; $CMRO_2$ = cerebral metabolic rate of oxygen) is the best estimate of the balance between brain metabolism and CBF [97]. When $AVDO_2$ increases, the cerebral metabolic demand is low, and when $AVDO_2$ decreases, this may be suggestive of hyperperfusion or tissue death [89]. $SjvO_2$ levels are correlated with $AVDO_2$ and may be useful in detecting ischemia or hyperemia [93, 94].

However, this method is limited by potential changes in arterial oxygen content, hemodilution, and position of the jugular bulb catheter, as well as by the need for frequent calibration and infrequent complications related to catheter insertion, such as infection, increased ICP, thrombosis, and pneumothorax [93, 94, 98].

Brain Tissue Oxygenation ($PbtO_2$)

Brain hypoxia has been shown to be harmful after a TBI, and it is recognized as a key secondary insult after injury [92, 99, 100]. In recent years, there has been growing evidence that patient outcome is improved after the application of therapy

targeted at cerebral tissue oxygen pressure. In this targeted therapy, MAP and percentage of inspired oxygen fraction are often used to maintain this parameter at adequate levels [92, 100].

In 1956, Clark et al. [101] reported the possibility of monitoring oxygen tension in blood and tissue, and in 1993 Meixensberger [102] first demonstrated the concept of $PbtO_2$ monitoring and its potential to assist in management of TBI patients [100, 101]. The use of direct $PbtO_2$ monitors was approved by the US Food and Drug Administration in 2001 [100]. $PbtO_2$ may be measured focally in the brain via either Licox (Integra, USA) or Neurovent (Raumedic, Germany) catheters, both of which have been shown to be safe and to provide accurate data. These devices provide information about the balance between oxygen demand and delivery in an injured brain. They may be affected by changes in capillary perfusion, distance from the capillaries in an edematous brain, and barriers to oxygen diffusion [103].

Recent data suggests that $PbtO_2$ values are directly correlated with patient outcomes. Cerebral hypoxia is an independent predictor of poor prognosis, disconnected from ICP, CPP, and brain imaging changes [100, 104]. $PbtO_2$ monitoring devices appear to discriminate reliably between normal oxygenation, threatened ischemia, and critical ischemia [100]. After elevation of the fraction of inspired O_2, PaO_2 increases to supraphysiological levels, or hyperoxemia. However, the relationship between hypoxemia and outcome in patients with TBI is controversial [85, 100, 104]. The randomized, controlled, multicenter phase III BOOST-3 trial is investigating the outcomes of maintaining a management protocol based on $PbtO_2$ combined with ICP [105]. For now, $PbtO_2$ values should be interpreted in the context of other monitored parameters to establish optimal management in clinical practice [92]. Cerebral hypoxia is a known cause of worse neurological outcome in patients with TBI. It has been observed that a higher frequency of daily episodes of cerebral hypoxia and a longer duration are common in nonsurvivors. Hypoxia is defined as alveolar oxygen pressure (PaO_2) $\leq$ 60 mmHg or O_2 saturation $\leq$ 90% or $PbtO_2 < 20$ mmHg [103–105].

1.3.3.3 Cerebral Microdialysis

Microdialysis consists of inserting an intraparenchymal catheter which allows diffusion of water and soluble substances at the distal end of the catheter across a semipermeable membrane. This permits constant assessment of the biochemical state of the brain tissue and interstitial fluid [106]. This information can help to guide therapy such as MAP parameters, ventilatory rate, and pCO_2 levels, and hyperosmolar therapy as well as the potential need for surgical interventions [106–108]. It could predict secondary damage before detection by clinical manifestations and conventional monitoring [106, 108, 109].

This method was first described in animal studies measuring neurotransmitters by Gaddum (1961) [110] and Myers (1972) [111]. In 1966, Bitto [108, 112] reported a dialysis technique using small volumes of interstitial tissue (e.g., brain), and Delgado (1972) [113] improved it using an electrode in a solution

continuously perfused through a dialysis bag, later called a dialytrode. Ungerstedt [114, 115], in the late 1970s and early 1980s, improved the efficiency of microdialysis by enlarging the surface area of the dialysis membrane. The successful use of microdialysis to quantify monoamine levels in neural tissue contributed significantly to the worldwide use of cerebral microdialysis [109].

Microdialysis can reveal the chemical composition of the interstitial fluid. Water and solutes diffuse between the interstitial fluid and perfused solution, which is called the perfusate, and the concentration gradient between these two chambers allows the diffusion of solutes at a constant speed, enabling their measurement in the dialysate. A number of metabolites have been studied and are believed to serve as biomarkers following TBI. The most commonly measured metabolites include glucose, lactate, pyruvate, glycerol, and glutamate [106–109].

Microdialysis has provided important information about TBI pathophysiology and continues to be an important tool as new biochemical markers are being investigated and utilized. However, further studies are necessary to clarify whether interventions based on microdialysis data may improve patient outcomes [106–109].

1.3.4 Multimodal Monitoring

Several parameters can be evaluated at the same time and can be used to establish a patient's prognosis after TBI, detect secondary injuries before irreversible damage occurs, allowing more thorough assessment of patient condition [116–118].

Among the commonly evaluated parameters and techniques are ICP, MAP, central venous oxygen saturation, TCD, ONSD, microdialysis, NIRS, continuous EEG, and other invasive and noninvasive physiologic trends at the bedside [116–118].

The use of a data acquisition and integration device, such as the Moberg CNS monitor, can provide the appropriate display and data synthesis to help clinicians identify significant physiological trends and optimal clinical responses to interventions. By condensing individual monitors and numeric feedback onto a single screen and formatting data into a graphical display, this system can help clinicians increase their understanding and recall of significant patient physiology, thereby improving the quality of patient care [116–118].

1.4 Conclusion

Traditionally, TBI management has focused on treating increased ICP and low CPP. Technological advancement has led to new tools which may provide this information with greater safety. However, research has expanded our knowledge of pathophysiological mechanisms underlying secondary damage to the brain after TBI far beyond these two parameters. Multimodal monitoring holds promise for analyzing a broader set of physiologic parameters to enable more extensive optimization of brain physiology following injury.

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