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Assessment of Intracranial Pressure in Severe Traumatic Brain Injury Using Optic Nerve Sheath Diameter and Transcranial Doppler

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Abstract

Background:Head trauma is defined as any physical hit or blow towards the head, which may or may not lead to an injury of the underlying brain. We consider a traumatic brain injury (TBI) to be a possible consequence of the traumatic event towards the head.

Severe trauma is responsible of more than 5 million deaths every year worldwide and this incidence is expected to increase in the coming decades.

TBI is the most severe condition observed in trauma patients, given that nearly 33% of patients with TBI die in hospital and another 33% have poor neurological recovery.

Prevention and treatment of intra-cranial hypertension (ICH) are the cornerstones of treatment for patients with TBI in intensive care units (ICUs), as uncontrolled ICH worsens brain damage and remains the most common cause of death after severe TBI.

Key words:Traumatic Brain Injury (TBI), Optic Nerve Sheath DiameterTranscranial Doppler.

1.Introduction:

Head trauma is defined as any physical hit or blow towards the head, which may or may not lead to an injury of the underlying brain. We consider a traumatic brain injury (TBI) to be a possible consequence of the traumatic event towards the head **(1)**. Severe trauma is responsible of more than 5 million deaths every year worldwide and this incidence is expected to increase in the coming decades **(2)**.TBI is the most severe condition observed in trauma patients, given that nearly 33% of patients with TBI die in hospital and another 33% have poor neurological recovery **(3)**.Prevention and treatment of intra-cranial hypertension (ICH) are the cornerstones of treatment for patients with TBI in intensive care units (ICUs), as uncontrolled ICH worsens brain damage and remains the most common cause of death after severe TBI **(4)**.

Intracranial pressure (ICP) monitoring is widely used in neurointensive care, especially for the management of patients with TBI. ICP levels are used to decide interventions, to verify the efficacy of therapeutic maneuvers, and in formulating a prognosis. For all these purposes accurate measurements are essential **(4)**.

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Clinicians rely on numbers provided by different methods, and generally believe that those numbers reflect actual ICP with a high degree of accuracy. For instance, a recent trial on decompressive craniectomy randomized patients to different treatments when ICP exceeded 20 mmHg **(4)**.

TBI remains one of the most complex diseases known in the most complex of all organs in the body. The causes of TBI are many and varied and include penetrating and non-penetrating injuries that, based on their overall level of severity, can evoke different degrees of morbidity, typically framed within the context of the Glasgow Coma Scale (GCS) score **(5)**.

TBI is the foremost cause of death in children and young adults and is a key socioeconomic concern for modernized and developing countries alike. The medical and surgical interventions for TBI can be complex and far reaching, often requiring a multidisciplinary approach involving critical care, trauma, and neurosurgical specialists **(6)**.The management of severe TBI in adults is derived primarily from the guidelines for the management of severe TBI, published by a joint effort of the Brain Trauma Foundation (BTF), the American Association of Neurological Surgeons, and the Congress of Neurological Surgeons **(6)**.

Intracranial hypertension and cerebral hypoperfusion are common occurrences after severe TBI and are associated with worse outcome, whereas a response to ICP lowering treatment is associated with a decreased mortality rate **(7)**.The monitoring of ICP in patients with severe TBI is recommended in the Brain Trauma Foundation Guidelines for the Management of Severe TBI and endorsed by the American Association of Neurological Surgeons(AANS), the Congress of Neurological Surgeons (CNS), and the Joint Neurotrauma and Critical Care section of the AANS/CNS **(8).**

2. Intracranial Pressure (ICP) Monitoring:

Methods for ICP monitoring can be divided into invasive and non- invasive approaches. Invasive methods include fluid-based systems and implantable micro-transducers. Non-invasive methods, most of which offer indirect measurement of ICP. Of the invasive methods, ICP monitoring using an EVD is considered as the gold standard, not only for its accuracybut also because it additionally serves a therapeutic purpose by allowing CSF drainage **(9)**.

2.1. Invasive ICP Monitoring:

EVDs allow for fluid-based monitoring as the pressure in the catheter equilibrates with the intraventricular pressure. This pressure transmits into an external saline-filled tube through a stain-gauge transducer from which the pressure measurement is made. The insertion of an EVD may be difficult in patients with inherently small ventricles size or those with ventricular compression attributable to advanced brain swelling **(9)**.

ICP can also be measured using implantable micro transducers such as strain gauge devices, pneumatic sensors, and fiber-optic sensors **(10)**.Pneumatic sensors have a balloon in the distal end of the probe, where pressure exerted on the balloon is equal to the pressure of the surrounding

tissue (i.e., ICP). Pneumatic sensors have also been used to measure intracranial compliance. In fiber-optic sensors, changes in ICP move a displaceable mirror at the tip of the sensor, altering the intensity of the light reflected along the fiber optic cable **(11)**.

2.2. Non-Invasive ICP Monitoring:

2.2.1. Transcranial Doppler (TCD):

In the neuro-critical setting, transcranial Doppler (TCD) is most commonly used as a tool to monitor changes in cerebral blood flow (CBF) in the setting of subarachnoid hemorrhage-associated vasospasm. A number of models using TCD-derived data have shown correlation withinvasively measured ICP; these models have used measurements of flow velocity (FV) in the middle cerebral artery, arterial blood pressure and pulsatility index (PI) **(12)**.

2.2.2. Optic Nerve Sheath Diameter (ONSD):

When the optic nerve exits the intracranial space into the orbit, it is still surrounded by the dural sheath. As such, the subarachnoid space surrounding the nerve is contiguous with the intracranial subarachnoid space. Elevation in ICP can transmit through the CSF in the subarachnoid space, leading dilatation of the optic nerve sheath, which can be detected using trans ocular ultrasonography **(13)**.

2.2.3. Imaging-Based Methods:

There are a variety of gross anatomic changes associated with elevated ICP that can be detected using computed tomography (CT) and magnetic resonance imaging (MRI). For instance, the presence of a mass occupying lesion can cause compression of the ventricles and midline shift. Similarly, enlarged ventricles can be indicative of hydrocephalus, and cerebral edema can cause a loss of differentiation of grey and white matter junctions **(13)**.

3. Management During Acute Phase of Head Injury:

Control of Intracranial Pressure and Cerebral Edema:

Edema is a hallmark of traumatic injury to the brain and spinal cord. It also frequently accompanies stroke and often complicates the clinical course of brain tumors. Intracranial pressure can be elevated due to mass effect from intracranial hematomas, contusions, diffuse brain swelling, or hydrocephalus. It can rapidly lead to cell death by either destroying nervecells or by physical compression of surrounding brain tissue. Intracranial hypertension can also lead to brain ischemia by reducing the cerebral perfusion pressure. Intracranial hypertension after TBI is associated with an increased risk of death in most studies **(14)**.

The monitoring of intracranial pressure and the administration of interventions to lower intracranial pressure are routinely used in patients with TBI, despite the lack of level 1 evidence. The full extent of recovery from injury can be improved by controlling edema, but the outcome is not predictable. Current treatments for cerebral edema are very limited and include osmotherapy and glucocorticoids (dexamethasone). The former involves administration of hypertonic mannitol to help reverse the swelling **(14)**.

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Hypertonic solutions:
Hypertonic solutions such as mannitol and hypertonic saline (HTS) are recommended early in the management of ICH after severe TBI **(15)**.They provide therapeutic benefit along with a wide therapeutic margin. The most recent BTF guidelines stated "although hyperosmolar therapy may lower intracranial pressure, there was insufficient evidence about effects on clinical outcomes to support a specific recommendation, or to support use of any specific hyperosmolar agent **(16)**.

The current recommendation for the use of mannitol to treat ICH is carried from the previous edition of the guidelines "to maintain sufficient recognition of the potential need for hyperosmolar therapy to reduceintracranial pressure, while acknowledging that more research is needed to inform more specific recommendations" **(15).** While mannitol has been the traditional agent of choice supported by older studies, the use of HTS is increasing and is supported by several recent studies, albeit small or heterogeneous ones. Reduction in intracranial pressure (ICP) has been consistently demonstrated with both mannitol and HTS, but there is a suggestion that HTS provides a more robust and durable effect in lowering ICP **(17)**.

Decompressive Craniectomy:
A randomized study in adults with severe diffuse TBI and refractory intracranial hypertension, showed that early bifrontotemporoparietal decompressive craniectomy decreased intracranial pressure and the length of stay in the ICU, but was associated with more unfavorable outcomes **(18).**

4. Transcranial Doppler
TCD technique is based on the Doppler effect, according to which, a sound wave, emitted with a certain frequency, strikes a moving object (e.g., red blood cells moving in an insonated vessel), the wave is then reflected with a different frequency (the Doppler shift), directly proportional by the velocity of the object **(Figure 1)** (**19).**

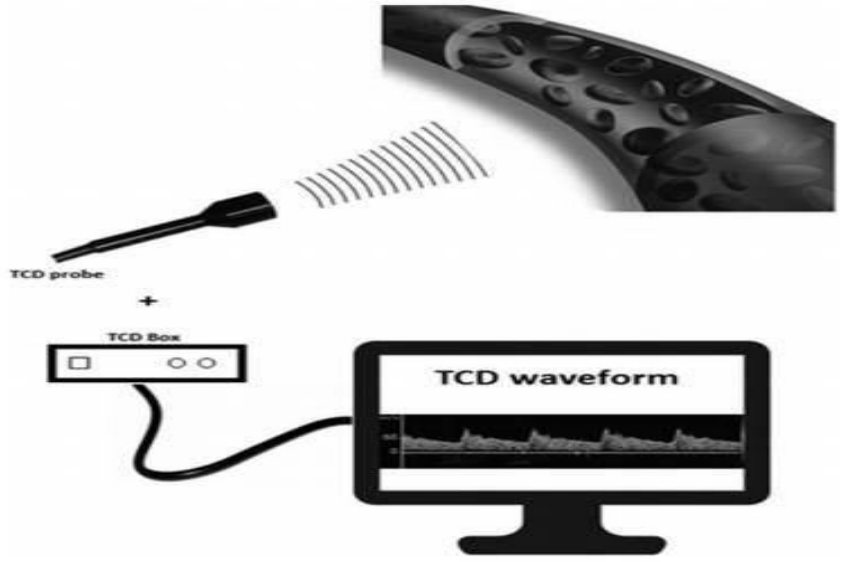

FIGURE 1 Schematic representation of the transcranial Doppler ultrasonography. The equipment basically consists of a ultrasound probe (usually of 2 mhz) and a hardware box. This system can be assembled as a stand-alone device or coupled to a computer for data storage and visualization

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In neurocritical ill patients, multimodality monitoring is of mainstay importance, because clinical examination alone is fairly insensitive to the following disease progression or detecting clinical deterioration **(20).**

Received echoes generate an electrical impulse in the ultrasound probe and are processed to calculate velocity, and therefore to produce a spectral waveform with cerebral blood peak systolic velocity, mean velocity, and end-diastolic velocity values. A constant vessel diameter and cerebral blood flow are the two main assumptions that govern the use of TCD as an indirect measure of CBF **(19)**.

Transcranial Doppler (TCD) has several clinical applications of in critically ill patients (Table 1) as a noninvasive ultrasound, cheap, quick, repeatable tool and its portability allows continuous bedside monitoring of cerebrovascular dynamics, which is particularly useful in a neurointensive care setting **(19)**.

While invasive intracranial pressure (ICP) assessment is considered a standard tool **(21)** and is widely used, this procedure is not exempt from risks, principally bleeding and infections, as well as the possibility of erroneous readings and the consequent inappropriate treatments **(22).**

5. Optic Nerve Sheath Diameter

Ultrasound devices are nowadays widely available in ICUs, but while Transcranial Doppler sonography for the identification of cerebral circulatory arrest requires trained and skilled sonographers, ONSD evaluation, is used to early detect the optic nerve sheath swelling in cases of intracranial hypertension may be performed with minimal training and, above all, noninvasively **(23)**.

High intracranial pressure (ICP) is a serious pathophysiology of TBI leads to poor prognosis. ICP is a fundamental parameter for monitoring the neurocritical patients since, when it overrides mean arterial blood pressure, cerebral perfusion stops and brain damage occurs after cerebral circulatory arrest **(24)**.

Ultrasound is an ICP monitoring method that is safe, available, reliable, and noninvasive. Many studies testified that ultrasound measured optic nerve sheath diameter (ultrasound-ONSD) can predict intracranial hypertension **(25)**.Eye sonography detection of an increased ONSD has been considered a reliable noninvasive indicator of intracranial hypertension **(26)**. Ultrasound or computed tomography (CT) was generally used to measure ONSD, and magnetic resonance imaging (MRI) or CT was used to measure ETD. Results have shown that the values of ONSD or ETD measured by ultrasound are in good agreement with those by MRI or CT **(27)**.

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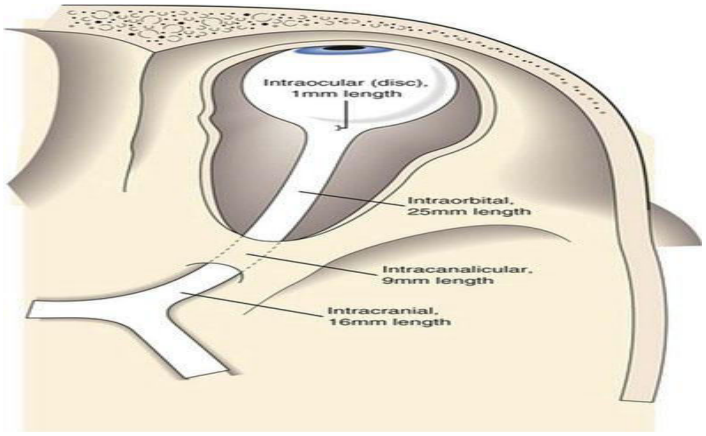

Figure (2): Optic Nerve Anatomy There are four anatomical divisions of the optic nerve: intraocular (1 mm), intraorbital (24– 28 mm), intracanalicular (9 mm), and intracranial (16 mm) **(28).**

The space surrounding the optic nerve is a continuation of the intracranial subarachnoid space. With the compensatory redistribution of cerebrospinal fluid (CSF) seen in cases of intracranial hypertension, the raised ICP instantaneously distends the ONSD **(29)**.
TBI and post-cardiac arrest patients, the ONSD calculated based on ultrasound or CT image is correlated with the invasive ICP **(30)**.

Comparing with the CT and MRI, the ultrasound is much more convenient in particular coma patients and in emergency conditions and also cheaper **(31)**. Optic Nerve Anatomy There are four anatomical divisions of the optic nerve: intraocular (1 mm), intraorbital (24– 28 mm), intracanalicular (9 mm), and intracranial (16 mm) **(28)**.

Acute and chronic diseases with an increase in ICP (e.g., idiopathic intracranial hypertension, craniocerebral trauma, malignant middle cerebral artery stroke, intracranial hemorrhage, decompensated hydrocephalus) lead to an increase of ONSD **(32)**.
In cases where the increase of ICP persists, a congested papilla develops with optic disc elevation (ODE), which can be detected with ultrasonography. Furthermore, some papers indicate that a diminution of ICP can provoke a decrease in ONSD **(32)**.

5. Technique and safety considerations

Ultrasonography of ONSD and the optic nerve can be easily performed using most color ultrasound systems equipped with high- frequency linear probes (7.5MHz or higher) with a lateral spatial resolution of less than 0.4 mm **(33)**.
As a first step, the system settings should be adjusted (mechanical index = 0.23 and the thermal index (TI) = 0.0) in order to prevent the damage of sensitive structures such as the lens, retina and vitreous body **(34)**.

Secondly, all parameters, such as time gain compensation or gray scale, depth and gain are individually adapted in order to achieve the best image quality. Vigilant training in the

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examination technique is advised. Standardization of technique is of great importance to reduce the inter- and intra-observer variation and establish the true axial plane and the exact boundaries of the sheath **(33)**.

For ONSD measurement, the examiner normally sits at the head of the examination table with the patient positioned supine with the head and upper body raised 20–30° to avoid any pressure on the eye. The patient remains in this position for at least 1 min before data are recorded. A thick layer of gel is applied to the closed upper eyelid. The transducer should be positioned on the temporal side of the eye. To help suppress eye movement and to achieve a better delineation of the major anatomical landmarks (optic nerve and lens), the patient is asked to look forward with closed eyes **(35)**.

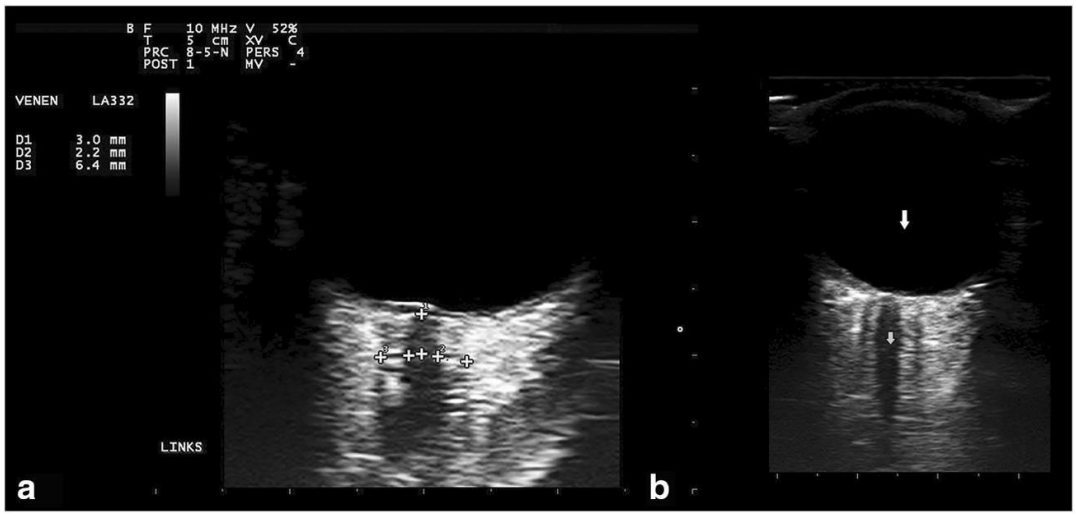

Figure (3): Measurement of the optic nerve diameter (OND) and optic nerve sheath diameter (ONSD) 3 mm behind the papilla (1) (a), using an electronic caliper and an axis perpendicular to the ON. Optic nerve diameter (OND) was measured as the distance inside the pia mater (2) and ONSD as the distance inside the dura mater on the hyperechogenic area surrounding the optic nerve (3). b Overview of the eyeball and the retroocular space using B mode. Ultrasonography makes an axial cut through the eye including a longitudinal section of optic nerve: hypoechogenic ocular globe (big arrow) and hypoechogenic optic nerve (small arrow) **(36).**

The correct way to measure the ONSD is between the outer hyperechogenic borders of the subarachnoid space. The sonographic aspect of the optic nerve is from the center to the periphery: hypoechogenic nerve fibers are closely surrounded by the hyperechogenic pia mater. The subarachnoid space appears hyperechogenic due to the trabecular structure and is surrounded by dura mater and periorbital fat **(36)**.

Measurement of the optic nerve diameter (OND) and optic nerve sheath diameter (ONSD) 3 mm behind the papilla (1) (a), using an electronic caliper and an axis perpendicular to the ON. Optic nerve diameter (OND) was measured as the distance inside the pia mater (2) and ONSD as the

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The risk benefit of invasive ICP monitoring has to be evaluated, considering that it requires specific personnel for the catheter insertion, maintenance, and troubleshooting, and it has been associated with cerebral damage and infective risks. Thus, since all these factors must be kept in mind when deciding whether to use invasive ICP monitoring, to date thereare no sufficient evidence to suggest the routine use of ICP invasive monitoring **(37)**.
On the other hand, in neurocritical, sedated, and mechanically ventilated patients, brain death (BD) must be recognized as soon as possible, in order to identify the potential candidates to be selected for organ donation, thus minimizing the time that could lead to organs deterioration **(25)**.

Clinically, BD diagnosis is suspected in case of unresponsive coma, persistent apnea and absence of brainstem reflexes. All signs that are under diagnosed during sedation. Moreover, these patients are usually already admitted to ICUs since days before BD occurrence and their clinical neurological state is continuously clinically monitored to early detect these signs of deterioration **(25)**.

When BD is suspected, a clinical and instrumental diagnostic protocol is then soon started. The diagnostic protocol, slightly different in each country, is usually composed of a multidisciplinary approach; beyond clinical and respiratory parameters evaluation, it usually includes an EEG, showing the absence of cortical electrical activity, as well as several “ancillary tests” (i.e., somatosensory evoked potentials and, in specific cases, blood flow evaluation with Transcranial Doppler, cerebral angiography and other conventional radiological imaging) **(25).**

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6. Conflict of Interest: No conflict of interest.

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