Chapter 55
Decompressive Craniectomy in the ICU: Usefulness of Transcranial Doppler (TCD/TCCS) in the Monitoring of Hemodynamic Changes

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Key Points

1. TCD/TCCS is a noninvasive monitoring technique in patients with acute neurological injury and/or neurosurgical pathology. TCD/TCCS is safe, which facilitates the study of cerebral blood flow velocities (CBFVs) and allows detection of possible complications.
2. TCCS allows visualization of possible anatomical changes of the brain parenchyma and blood vessels, and achieves a greater accuracy measures of CBFVs compared with TCD method (blind technique), pre- and post-craniectomy.
3. TCD/TCCS allows assessment of cerebral hemodynamic changes in real time during the intracranial hypertension management, as well as the hemodynamic changes due to decompressive craniectomy (DC).

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4. Decompressive craniectomy is considered a life-saving surgical technique to improve refractory intracranial hypertension, where ICP elevation is not controlled with medical management. TCD/TCCS provides useful information during clinical evolution and could contribute to determining the neurological prognosis of the critically ill patient.

55.1 Introduction

Transcranial Doppler/transcranial color-coded duplex sonography (TCD/TCCS) are noninvasive and reproducible monitoring tools that facilitate the study of the cerebral hemodynamics, allowing for the assessment in real time the CBFV variations and its association with clinical changes at bedside [1]. These noninvasive monitoring techniques are part of the diagnostic arsenal to provide timely and directed-therapy. Therefore, TCD/TCCS measurements can provide information to individualize interventions adjusted to the clinical needs of each patient [1].

TCCS, compared to other monitoring techniques, provides images of the brain parenchyma anatomical (B-mode) and cerebral blood vessels in real time, enabling a more accurate acoustic access [2, 3].

In patients with traumatic brain injury (TBI), TCD/TCCS provides information on the cerebral hemodynamic status. Hyperemia and vasospasm are pathological conditions present during TBI. These clinical situations can be differentiated from each by calculating the Lindegaard ratio [6]. If the latter is elevated, then vasospasm is the most likely reason for the elevated CBFVs. The vasospasm and CBFV changes (spectral Doppler waveform analysis) may precede the clinical manifestations of neurological worsening (e.g., delayed cerebral ischemia). Therefore, TCD/TCCS is a useful technique to anticipate neurological worsening at bedside [4, 5]. It has been described that vasospasm findings evidenced by TCD/TCCS predict and are associated with documented vasospasm through invasive angiography (CTA, DSA) [6].

A three-phase CBF pattern has been described after TBI [6]. Initially, overall CBF is reduced leading to a hypoperfusion status followed by a hyperperfusion phenomenon in the following 24–72 hours [5, 6].

There are patterns for rapid and simple recognition of CBF disturbances (e.g., hyperflow, cerebral circulatory arrest), which can be identified in the interpretation of spectral Doppler waveform analysis and hemodynamic indexes (pulsatility indexes (PI), resistance index (RI)) derived from it [2], thus facilitating rapid real-time therapeutic decision (Fig. 55.1).

55.2 TCD/TCCS Technique

TCD technique uses a low-frequency transducer (2 MHz), and TCCS uses phased array transducer whose frequency oscillates between 1 and 5 MHz, being automatically configured at frequencies 1 and 2 MHz when the B-mode is selected [1, 2, 38].

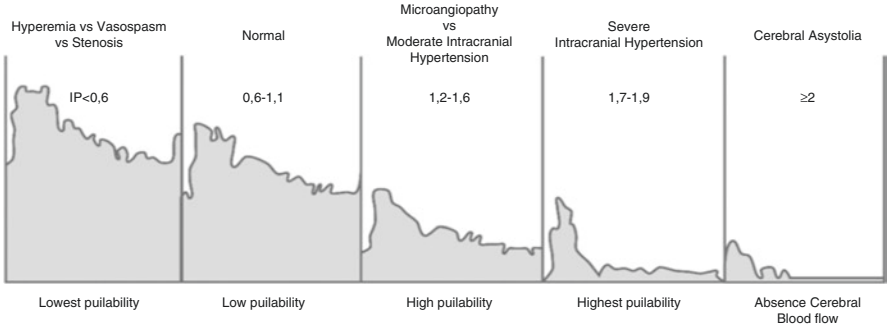


Fig. 55.1 Morphological spectrum of waves reflecting cerebral blood flow velocities and PI documented by transcranial Doppler and examples of relevant conditions. HIC Intracranial hypertension, PI Pulsatility index ([Maximum systolic flow velocity – minimal diastolic flow velocity/ Mean flow velocity])

CBFVs should be measured with an insonation angle less than 60 degrees. The following precaution is recommended [1]:

1. Importance of keeping the correct insonation angle-correction. It can increase 20–30% in CBFVs measurement (more details see Chap. 72).

One of the impediments for an adequate transcranial insonation of CBFVs is the absence of transtemporal and suboccipital acoustic windows (prevalent in elderly women). Therefore, in craniectomized patients [1, 38, 39], the evaluation of cerebral basal arteries is easier.

55.3 Descompressive Craniectomy (DC)

The utility of DC to manage intracranial hypertension syndrome has been well described. A space-occupying lesion (intracerebral hemorrhage, subdural hematoma, etc.) or diffuse lesions (brain edema) may generate midline shift and/or different classic herniation syndromes. According to the classical Monro-Kellie doctrine, intracranial components shift to compensate the ICP changes to maintain adequate brain oxygen delivery and cerebral perfusion pressure (CPP). The following have been described as compensatory mechanisms: cerebrospinal fluid (CSF) redistributes from skull into the spinal canal (less production and increase reabsorption), venous systems compression (increase venous outflow), and arterial system compression (decrease blood entering the skull). These mechanisms are only temporarily effective. Hence, when these compensatory mechanisms are exhausted, neurological worsening ensues [4, 5, 17].

DC is a surgical procedure whereby a bone fragment with a diameter of at least 12–15 cm (supratentorially) is temporarily removed. This provides additional space for brain tissue expansion, thus restoring some balance of intracranial pressures [14, 15], to decrease ICP and CPP [3, 8]. After this procedure, the pressure gradient

generated by the displacement of brain structures decreases according to the Monro-Kellie doctrine, therefore avoiding brain edema, cerebral hypoperfusion, and brain herniation syndromes [4, 16, 19, 20, 22, 23].

ICP control through DC leads to a theoretical improvement of cerebral hemodynamics and maintain adequate brain oxygen delivery due to optimization of CBF [10–12]. TCD/TCCS has a potential as a diagnostic and monitoring tool to indirectly record these hemodynamic changes, to obtaining real-time information and make crucial and individualized therapeutic decisions to optimize CPP, as well as its usefulness in the post-DC follow-up [3–8].

DC is employed when intracranial hypertension is refractory to medical treatment, such as in cases of severe TBI, acute ischemic stroke (AIS), aneurysmal subarachnoid hemorrhage (aSAH), CNS infections, and venous sinus thrombosis [3, 4], and can be classified in two ways: chronological and initial vs add-on therapy [26, 27] (Table 55.1).

In general, there are two types of surgical techniques for DC (Table 55.2).

Table 55.1 Classification of decompressive craniectomy (DC)

Medical management	DC procedure time
Early primary: Carried out in patients with limited access to multimodal neuromonitoring and indicated as first line control of tissue damage, as long as the patient is under sedation and with availability of neuroimaging control [13] *Primary:* Also known as prophylactic, performed simultaneously with a cranial surgical procedure, such as post-traumatic mass-effect lesion evacuation, for the prevention of subsequent ICP increase [13, 26]; it is estimated that edema increases after 4-5 days, so the bone is removed before this timeframe *Secondary:* Also known as therapeutic, performed as second-line therapy in the management of medically-refractory increased intracranial pressure [3, 19, 20] Useful in centers with multimodal neuromonitoring resources [26]	*Early:* Carried out in the first hours after the trauma in ranges from 4 to 24 hours [27] *Late:* Carried out after 24–48 hours post-trauma [28]. DC is not recommended after 4-5 days of trauma

Table 55.2 Types of surgical techniques for DC

Bifrontal	Hemispheric
In frontal lobes edema with or without associated injury [21]	In the presence of unilateral focal lesion with mass-effect (i.e malignant MCA ischemic stroke) [4]
Diffuse brain edema with posterior basal cistern compression (quadrigeminal) [4]	Diffuse brain edema with basal cistern compression (ambiens and crural) [4]
Anterior-posterior trauma vector	Latero-lateral trauma vector
Technique: Frontal and temporal bones are removed up to 2–3 cm preceding the coronal suture bilaterally. The superior sagittal sinus and the falx cerebri can be ligated to allow anterior expansion [23]	*Technique*: The frontal, temporal and parietal bones are removed from one or both sides, obtaining a bone flap of at least 12–15 cm, with subsequent dural opening [24, 25]

55.4 Neurological Syndromes and Decompressive Craniectomy: The ¨Open Box¨ Concept

When a bone flap is removed, the skull or "closed box," becomes an "open box" [3, 5, 29, 31, 32, 36]. Therefore, once swollen brain tissue herniates through the craniectomy defect, ICP and midline shift (mass effect) are immediately reduced, responding to the Monro-Kellie doctrine [29, 32]. After DC, the neurosurgical team and ICU team should pay attention to the new "invisible" variable, atmospheric pressure [37].

The "open box" concept has particular pathophysiological features. The lateral ventricle can migrate to the craniectomy defect. However, it is not clear whether this represents a localized effect of ex-vacuo hydrocephalus, altered CSF dynamics, or a combination of both phenomena [32, 37]. Hence, a disturbance of CSF dynamics may occur, including a "siphon effect" and subsequent reduction of CBF. This may be due to both the venous return disturbance and the subarachnoid space obliteration due to direct pressure on the brain parenchyma that compromise regional CPP [32].

Different neurological syndromes (some early and some late) associated with DC has been described [29, 32, 37], resulting from external and/or internal forces, such as pathophysiological consequences on intracranial compartments (Tables 55.3 and 55.4, Figs. 55.2 and 55.3).

Table 55.3 Intracranial and extracranial forces. Open box concept (Fig. 55.2)

Intracranial forces	
Cerebral spinal fluid (CSF) pressure	
Gravitational forces	
Physical properties	Brain compliance
	Masses
	Brain-bone interfaces
	Sites of dural insertions
Extracranial forces	
Galea aponeurotica	
Subgaleal fluid	
Atmospheric pressure	1 atm = 14.7 PSI = 1.033 cmH_2O

Table 55.4 Neurological syndromes associated with DC

Syndrome	Key feature
Syndrome of the trephined or post-traumatic syndrome	Headache, vertigo, tinnitus, insomnia, memory disturbances, hemiparesis, gait disturbances, epilepsy, mood swings, cognitive and behavioral disorders [30, 31]
Sinking flap syndrome	Focal neurological deficit in hemicraniectomies [33]
Motor syndrome of the trephined	Term focused on motor disorders [34, 35]
True syndrome of the trephined (similarities with sinking flap syndrome)	Focal deficit [29] Ex: Hemiparesis (resolves after cranioplasty)
Neurological susceptibility to the skull defect	Neurological symptoms after decompressive craniectomy with subsequent recovery with cranioplasty [3]

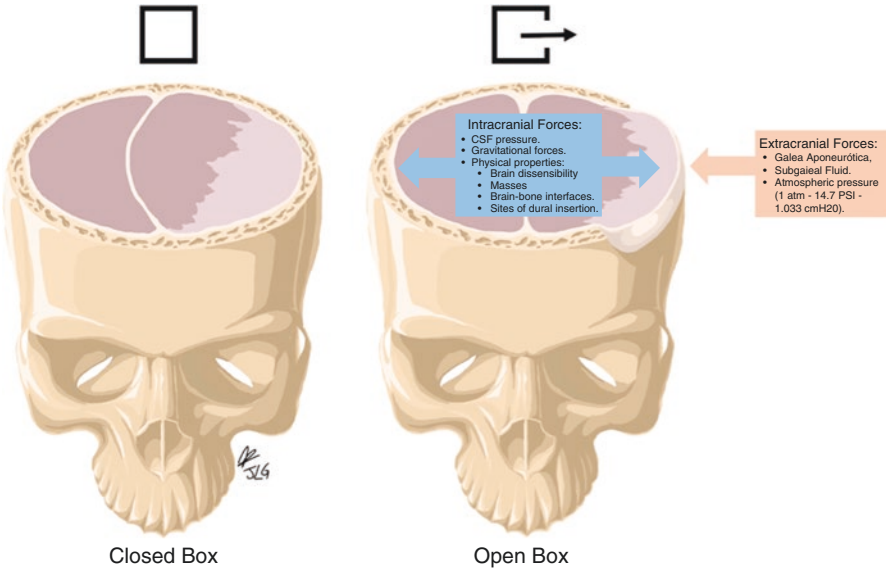


Fig. 55.2 Extracranial and intracranial forces in the "Open Box" concept

Consequently, the usefulness of TCD/TCCS in this clinical scenario, is real-time early detection of hemodynamic changes (CBFVs, spectral Doppler waveform, PI, etc.) to improve cerebral perfusion through individualized therapeutic decisions [30, 34] (Fig. 55.3).

55.5 TCD/TCCS: Hemodynamic Changes Associated with Decompressive Craniectomy

TCD/TCCS provides information for the evaluation of the hemodynamic changes of the "exposed" brain after bone window removal [5, 6], which facilitates the measurements.

After performing a DC, mechanical, systemic, and atmospheric pressure changes involved in cerebral perfusion must be considered [8, 29, 32, 42, 44]. The impact of cerebral hemodynamic changes (CBFVs and hemodynamic indexes) can be recorded by TCD/TCCS during post-DC follow-up in ICU (Table 55.5), as they are progressively restored.

Understanding the different cerebral hemodynamic patterns (pre- and post-DC), help the implementation of timely therapeutic decisions individualized to optimize CPP for each patient. Therefore, keep in mind that generalized treatment goals [17] can lead to hypo- or hyperperfusion phenomena, being deleterious for the patient [11, 12, 39, 40] (Figs. 55.4a–c and 55.5).

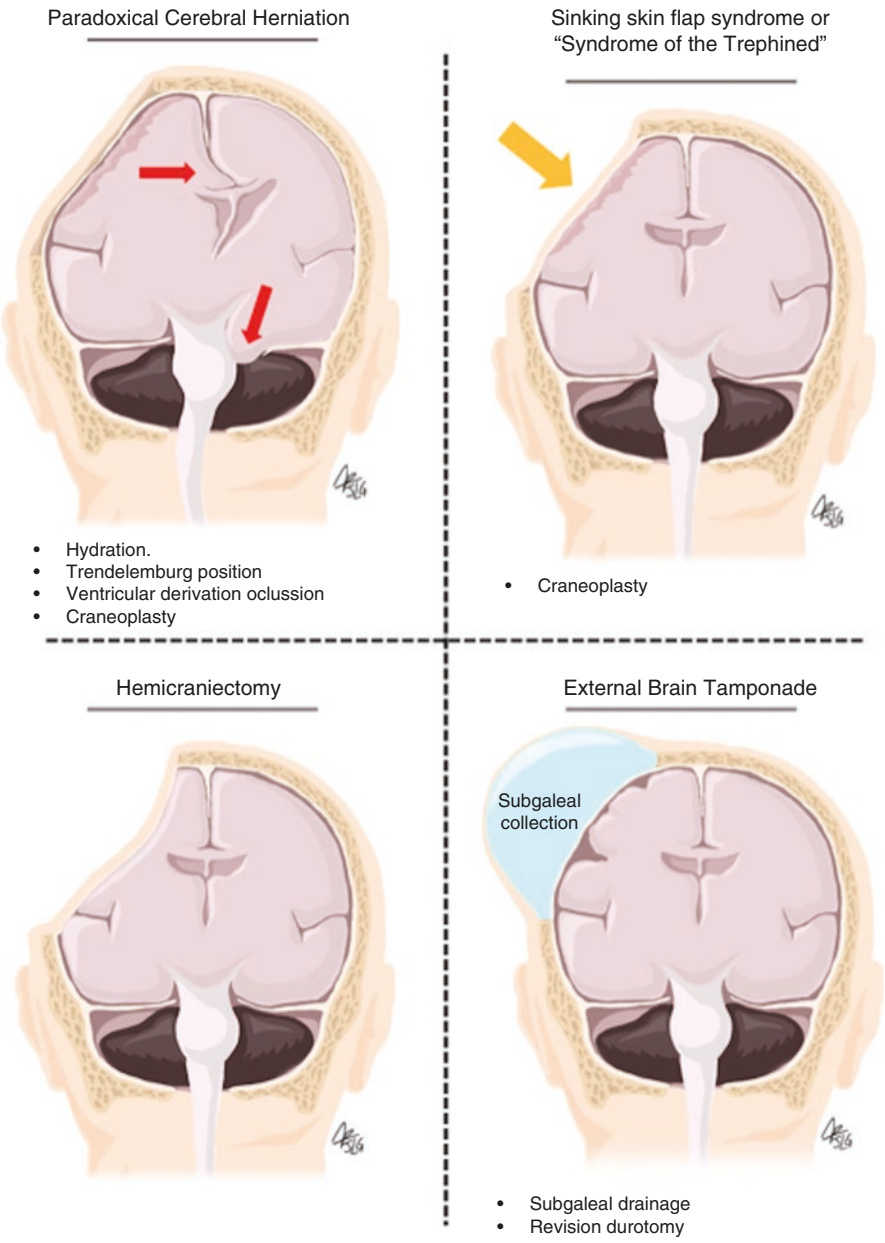


Fig. 55.3 Neurologic syndromes and potential treatments

Table 55.5 TCD/TCCS Patterns: Post-decompressive craniectomy

	TCD pattern				
	Normal	Intermediate /Transition	Oligemic	"Pure"Hyperemic (LI < 3)	Vasospasm (LI < 3)
PI	=	Normal/ ↑	Normal/ ↑	Normal/ ↑	Normal/ ↑
Wave morphology	=	Normal/ ↑	Hight resistance	↓	↓
MFV	=	Normal	↓	↑ ↑	↑ ↑ ↑
Congestion	=	Normal	Normal/	Normal/ ↓	Normal/ ↓

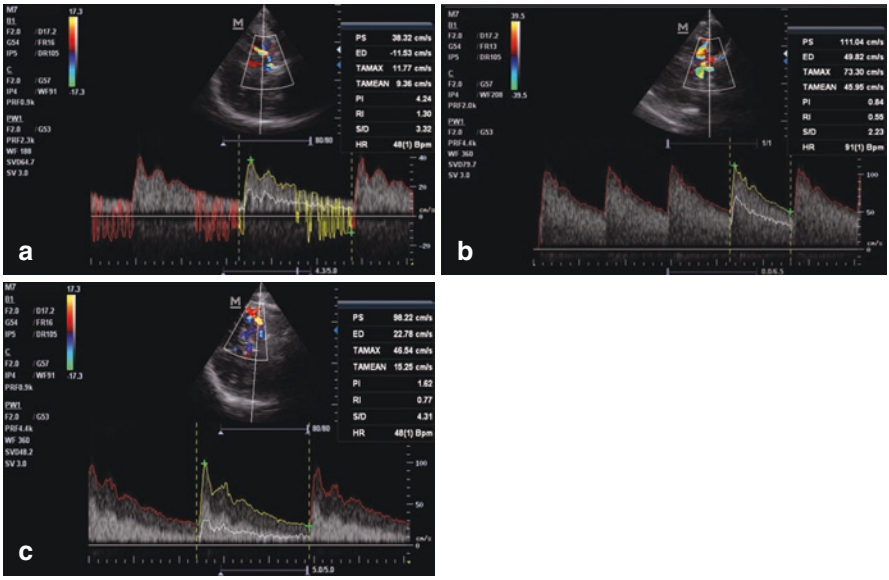


Fig. 55.4 (**a**) "Oligemic / hypoperfused" pattern in post-DC patient. This highlights an abnormally elevated PI and a very low amplitude of spectral Doppler wave, which in addition to low mean flow velocities (MFV, shown in the device screen as "TAMAX") in MCA (<40 cm/s) suggest severe intracranial hypertension. *ED* End-diastolic volume, *RI*Resistance index. (**b**) "Hyperemic/ hyperperfused" pattern in the patient of Fig. A, this time post-DC (48 hr). Although literature [40] has defined "hyperemia" with MCA MFV >100 cm/s, there is an increase in MCA MFV >30% in comparison with pre-DC values. Turbulent flow, vascular congestion, increasing spectral Doppler waveform amplitude and a marked decrease in PI are also shown, which suggests a possible "exacerbated" reperfusion phenomena of the vascular territories secondary to intracranial pressure reduction after DC. (**c**) "Intermediate" pattern post-DC (6 hr). Although it has been described as "non-specific" by some authors [40], in this particular context of early post-DC, we propose a transition pattern between oligemia and hyperemia, with a tendency to PI improving (but still increased), a normal spectral-waveform morphology and stable values of MFV, reflecting the importance of "normoperfusion" as a neuroprotective measure and treatment goal in the neurocritical patient

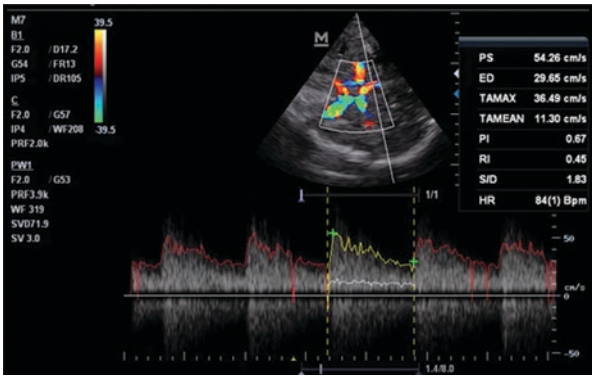


Fig. 55.5 Insonation of the posterior cerebral artery (P1) and associated findings. The PI normalization, spectral-waveform morphological changes, and also MFV normalization after DC had been also reflected in a global manner, as a response to intracranial pressure management and control

55.6 Conclusion

During follow-up of cerebral hemodynamic changes in patients with acute brain injury (ABI), it is important to prevent and identify disturbances that extend their hospital stay and determine unfavorable long-term outcomes [38]. In the context of refractory intracranial hypertension, DC is a crucial intervention to restore adequate cerebral perfusion and brain oxygenation delivery. TCD/TCCS is a noninvasive monitoring tool that provides useful information for hemodynamic parameters analysis and estimate cerebral prognosis of the critically ill patients with ABI [6, 22].

TCD/TCCS is an useful monitoring method to assess indirectly CBF in neurocritical patients to demonstrate its optimization after therapeutic interventions such as DC or external ventricular drain (EVD), providing data to follow-up and control of CPP and ICP changes derived from medical and surgical therapeutic interventions [8, 9]. However, the measurement of the hemodynamic variables by TCD/TCCS (CBFVs, spectral Doppler waveform, PI, etc.) is an operator- and/or acoustic window-dependent technique [6, 10]. Hence, it is vitally important to have high-expertise staff to reduce as much as possible the error rate on clinical interpretation of data [10, 38]. Although there is increasing knowledge about the hemodynamic and vascular perfusion changes during ABI after a DC (Table 55.5), the heterogeneous therapeutic results in this clinical scenario cannot be overlooked. Therefore, a careful, detailed and specific analysis of each clinical situation is required to make individualize decisions [5, 6, 8, 9, 10–12, 39, 40].

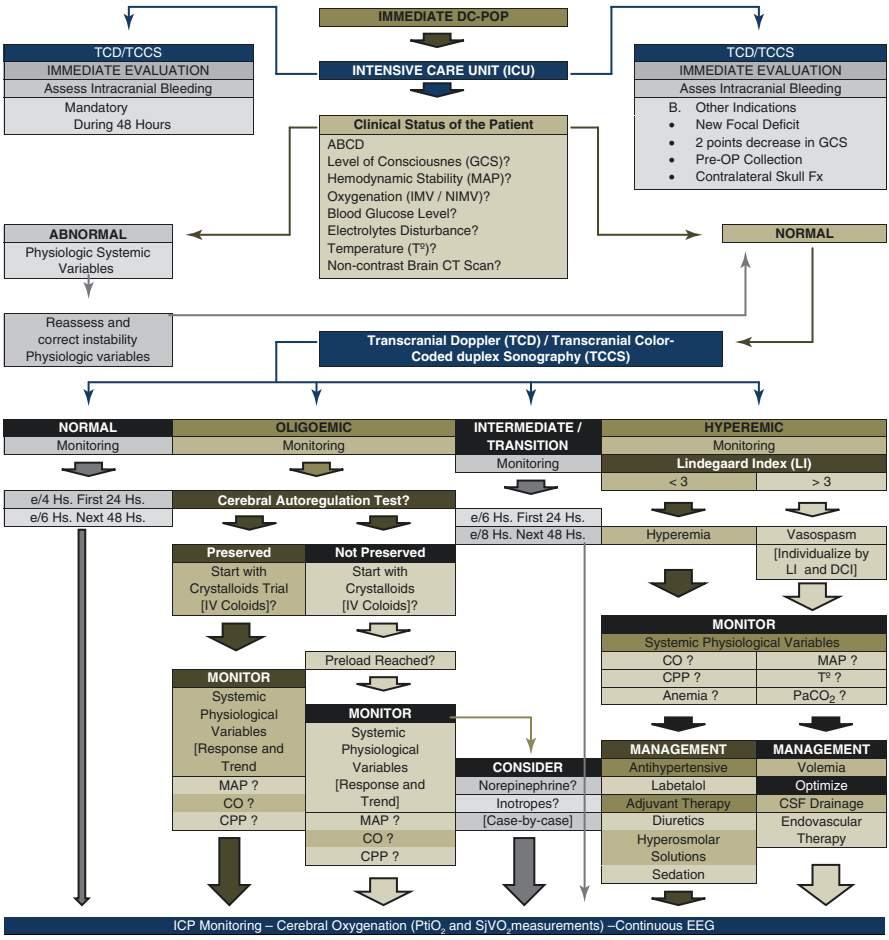
DC is currently considered as a "definitive" management to control intracranial hypertension. This data assure that this procedure is effective for the management of cerebral oligemia without necessarily implying a post-DC cerebral hemodynamic equilibrium [41, 42, 46]. This surgical procedure may cause secondary harmful effects by hyperemia (paradoxical increase of ICP and/or risk of bleeding) or oligemia (hypoperfusion, ischemia). These cerebral hemodynamic events are possible causes of perpetuous secondary brain injury [18].

In consequence, the need for early and protocolized management of these probable neurological worsening variables, (1) arterial hypertension, (2) hypervolemia status, (3) hyperdynamic states (exacerbate hyperemia), (4) dehydration status and ICP increases (worsen the oligemia), which are considered commonly as late causes of clinical deterioration. Hence, the cerebral hemodynamic repercussion of these physiological/pathophysiological variables can be assessed indirectly by TCD/TCCS through pre-post DC cerebral flow patterns analysis [6–8, 38–40]. In the specific case of patients with TBI, the metabolic crisis is not only the result of cerebral ischemia and hypoperfusion [39, 40] but even can occur under apparently normal conditions of cerebral blood flow assessed by TCD/TCCS.

Therefore, multimodal neuromonitoring (MMM) in ICU is crucial because microvascular alteration [41], cerebral excitotoxicity, electrical pathologic activity, and brain dysoxia [18, 39, 41] require the integration of different type of devices to arrive at a diagnosis, where information provided in real-time by TCD/TCCS at bedside is very useful to make-directed therapeutic decisions.

Finally, it is important to consider the skull multicompartmental theory at time to perform cerebral hemodynamic assess by TCD/TCCS [41, 46]. This theory refers to the fact that a patient with (or without) DC can develop hyperemia in one hemisphere and vasospasm in the contralateral hemisphere. These clinical conditions challenge classical management strategies, rendering them contradictory. However, bedside assessment by TCD/TCCS allows us to better understand these cerebral hemodynamic phenomena, motivating a more individualized treatment [37–41].

Algorithm



DC-POP Decompressive craniectomy postoperative, *ABCD* Airway-breathing-circulation-disability, *MAP* Mean arterial pressure, *IMV* Invasive mechanical ventilation, *NIMV* Non-invasive mechanical ventilation, *GCS* Glasgow coma scale, *Fx* Fracture, *IV* Intravenous, *CO* Cardiac output, *CPP* Cerebral perfusión pressure, *DCI* Delayed cerebral ischemia, *e/* Every, *Pre-OP* Pre-Operative

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