

# The First 24 Hours after Severe Head Trauma

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*“One gram of first-hour treatment can avoid kilograms of treatment, later, in the intensive care unit”*

Paul E. Pepe, Oral presentation, ISICEM, 2011

## Introduction

Severe traumatic brain injury (TBI) is defined by a Glasgow Coma Scale (GCS) score of 8 or less during the first post-trauma day. It is recognized as a devastating pathology in terms of mortality and morbidity. Long-term recovery is poor with only 10 to 20 % patients returning to work after 1 year [1, 2]. If all severe TBI patients are taken into account (including those deceased before arrival at hospital), one-year mortality ranges between 35 to 45 % [3–6]. Mortality is dramatic in the first days with more than 40 % of all deaths occurring within the first 24 hours [1, 6].

Numbers of studies have demonstrated the importance of the initial hours after severe head trauma. After trauma, physiologic mechanisms against ischemia in cerebral tissue are impaired and traumatized brain highly vulnerable to ischemic injuries [7]. And the more recent the trauma, the more the brain is vulnerable [8]. Moreover, ischemic injuries are frequent during the first post-traumatic hours, notably in cases of multiple trauma. These ischemic injuries, even for a few minutes, have a dramatic negative impact on long-term outcomes. Thus, amelioration of initial care should greatly improve outcome.

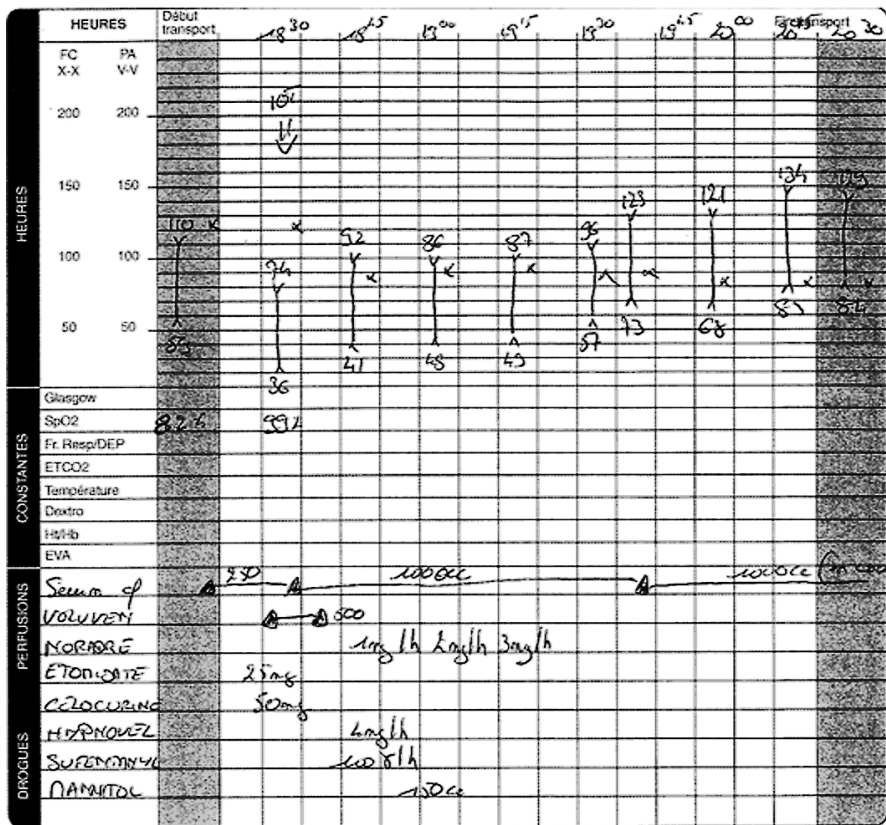
## Control of Arterial Pressure before Hospital Admission

Hypoxia and arterial hypotension, the “lethal duo” [9], decreasing oxygen and glucose transport, have been identified as major prognosis factors since the early nineties [10, 11]. Just one episode of arterial hypotension during the pre-hospital period increases mortality rates two- to four-fold [5, 10]. Ventilation and circulation control should thus be the two central axes of immediate management.

Whereas pre-hospital intubation decreases mortality when performed by trained teams [4, 5], the frequency of pre-hospital arterial hypotension, observed in about one third of patients, has not decreased since the early nineties, even with pre-hospital medical teams [5, 12–14]. Given the dramatic effect of hypotension during this period, control of arterial pressure must be a major goal of pre-hospital medical teams. Causes of hypotension are multiple and interconnected: Hypovolemia related to extra cerebral hemorrhages, decreased venous return because of positive intrathoracic pressure during controlled ventilation [15], but also deep sedation [14, 16].

During the pre-hospital period, mechanical ventilation of severe TBI patients is recommended to prevent inhalation and hypoxic events, and to control PCO<sub>2</sub>.

Sedation is needed to limit causes of increased intrathoracic pressure, such as pain, cuff and ventilator asynchrony. All of these recommendations are aimed to limit risks of increased intracranial pressure (ICP). But if induction of anesthesia for tracheal intubation and sedation induce arterial hypotension, the net benefit on cerebral perfusion pressure (CPP) is lost (Fig. 1). Control of arterial pressure requires first efficient monitoring, which is not available during the pre-hospital period. Non-invasive techniques should be used to measure arterial pressure every 2 minutes, with particular attention during dangerous phases, such as the induction of anesthesia and sedation. Second, sedation must be cautiously titrated. Norepinephrine infusion (or phenylephrine bolus) should be used liberally, notably during induction of anesthesia to *prevent* hypotension.



**Fig. 1.** Example of a patient with severe TBI (GCS = 7) with a moderate scalp hemorrhage during the pre-hospital period. Induction of anesthesia, sedation and controlled ventilation induced a prolonged decrease in systolic arterial pressure (< 90 mmHg) for nearly one hour despite fluid resuscitation and norepinephrine (NORADRE) infusion.

## Immediate Care at Hospital Admission

As for the pre-hospital period, the most important goal on arrival at the hospital is control of peripheral events, such as hypoxia and hypotension [17]. Severe TBI patients must be admitted to a dedicated area by a team trained in polytrauma care. As for all multiple trauma patients, admission to hospital has therapeutic and diagnostic periods that allow ventilation and pressure control [18]. All polytrauma patients are managed following the same rules (**Fig. 2**):

- Respiratory control with  $\text{SaO}_2$  and end-tidal  $\text{CO}_2$  ( $\text{EtCO}_2$ ) monitoring, bedside chest x-ray and sonography; chest tube insertion if necessary.
- Arterial and 3-line venous femoral catheters for arterial monitoring, blood samples, fluid resuscitation and norepinephrine infusion.
- Circulation control: Close monitoring of invasive arterial pressure, fluid resuscitation and transfusion (with particular attention to coagulation abnormalities), search for occult blood loss, bedside chest and pelvis x-rays, and FAST (focused assessment with sonography for trauma). Surgical control of massive hemorrhage is, of course, an absolute priority [19]. Coagulation abnormalities are a major cause of worsening of even minor cerebral lesions. In cases of severe TBI, mean arterial pressure (MAP) should ideally be adjusted using transcranial Doppler (TCD) measurements to normalize cerebral blood flow (CBF) as soon as possible [20] (please see below).
- Information and coordination of surgical and radiological teams.

Many patients with severe TBI now benefit from standardized care in specialized trauma centers [21] and this is most probably related to the strong prognostic influence of hypotension and hypoxia events.

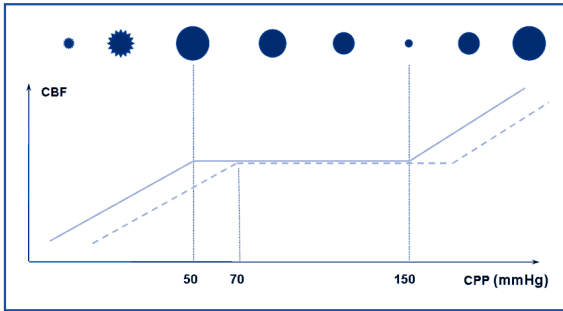
## Early Transcranial Doppler

The first step in the resuscitation of a patient with severe TBI is, thus, the standardized protocol of care for all multiple trauma patients; however severe TBI patients also require control of CBF. CBF is maintained in normal ranges by variations in cerebrovascular resistance above 50 mmHg of CPP ( $\text{CPP} = \text{MAP} - \text{ICP}$ ). After trauma, autoregulatory responses are depressed and higher CPP levels are needed to maintain CBF in normal ranges (**Fig. 3**). Thus, most experts recommend maintaining MAP at 80 mmHg. But, even with a mean MAP at 80 mmHg, up to 40 % of severe TBI patients have ischemic injuries at the time of implementation of invasive monitoring ( $\text{CPP} < 60$  mmHg or jugular venous oxygen saturation  $[\text{SjO}_2] < 55$  %) [20, 22]. Given that the mean reported delay before availability of invasive cerebral monitoring is about 7 hours after trauma [22, 23], we need other ways to evaluate and correct CBF earlier.

TCD permits rapid and non-invasive estimation of CBF and is particularly accurate at detecting low CBF [24]. Temporal windows are used for insonation of mean cerebral arteries that account for 70 % of the homolateral internal carotid flow. Peak systolic ( $V_s$ ), end-diastolic ( $V_d$ ) and time-averaged mean ( $V_m$ ) velocities are measured and the pulsatility index (PI) calculated as:  $\text{PI} = (V_s - V_d)/V_m$ . It has been shown, in experimental and clinical studies, that below the autoregulation range (i.e., when CBF decreases),  $V_d$  decreases with CPP more rapidly than  $V_m$  and  $V_s$ , with the strongest correlation observed between CPP and PI [24]

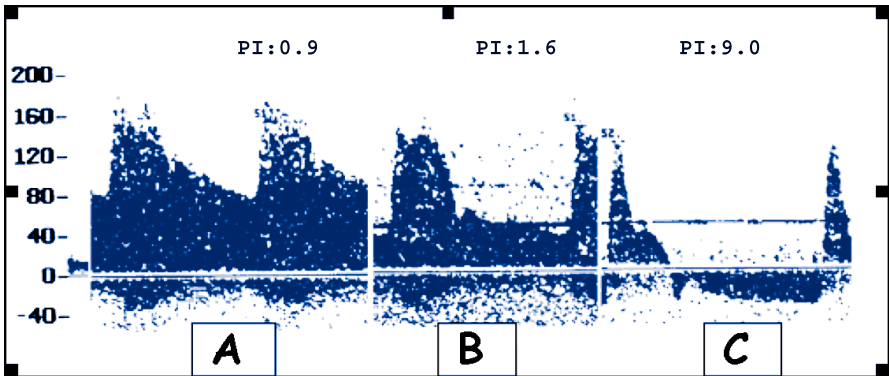


**Fig. 2.** Admission of a multiple trauma patient at our institution: A junior physician is responsible for arterial and venous catheterization, while, at the same time, the trauma leader performs FAST including, systematically, transcranial Doppler.



**Fig. 3.** Cerebral autoregulation: In normal conditions, cerebral blood flow (CBF) is maintained over a wide range of CPP (from 50 to 150 mmHg) by changes in cerebral vascular resistance. After traumatic head injury, autoregulatory responses are depressed and the autoregulation curve shifted to the right. This partially explains the importance of hypotension after TBI and the vulner-

ability of brain tissue. The state of vascular tonicity is symbolized by blue circles at the top of the figure. Small arteries are empty at low pressure and dilated when autoregulation begins. They then constrict progressively to maintain CBF constant. Vessels are stretched for high CPP after the plateau.



**Fig. 4.** Successive transcranial Doppler measurements during a progressive decrease in cerebral blood flow (CBF). A: normal. B: The pulsatility index (PI) is abnormally high ( $> 1.2$ ), indicating decreased CBF. C: Oscillating flow, cessation of cerebral perfusion has been reached when forward and reverse flow are nearly equal. Note that end-diastolic velocity (Vd) decreases far more than the peak systolic velocity (Vs) when CBF decreases, i.e., pulsatility increases.

(Fig. 4). Thus, an increase in PI ( $> 1.4$ ) is the first sign of decreased CBF, whatever the cause: Hypocapnia [25] or low CPP (because of high ICP and/or low MAP) [24]. The thresholds of PI and Vd have not yet been fully elucidated. It has been shown that, in severe TBI patients, a PI  $> 1.4$  coupled with a Vd  $< 20$  cm/sec identifies patients with the highest ICP and lowest SjO<sub>2</sub> at admission [20] and is significantly correlated with poor outcome [26].

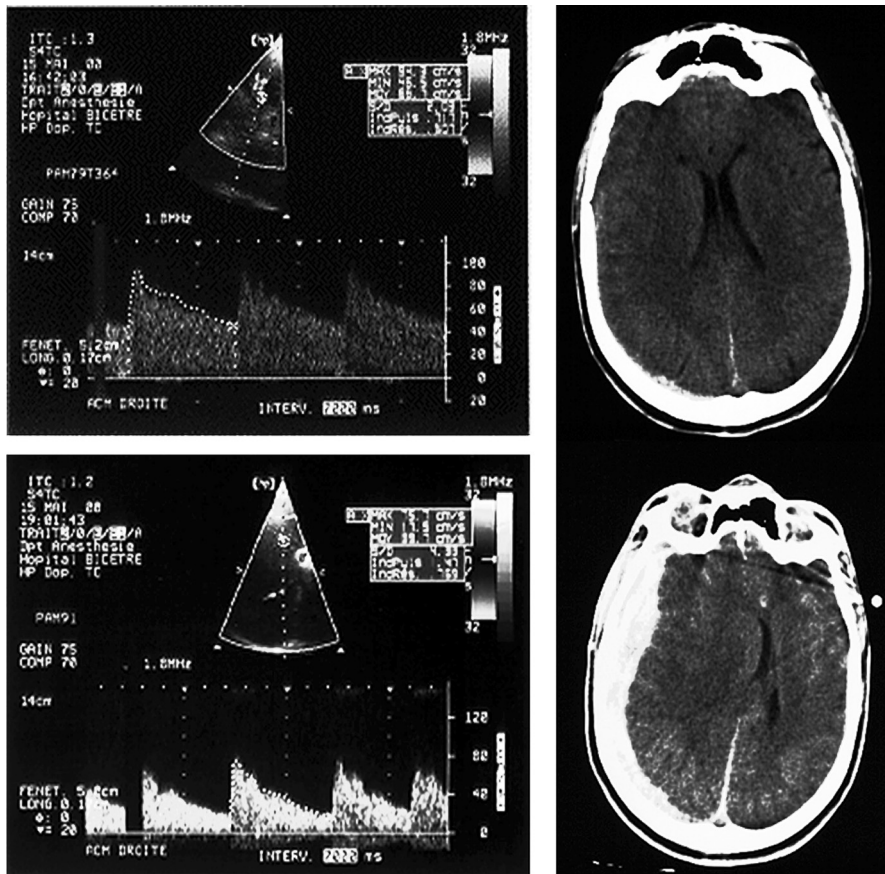
When CBF is impaired as diagnosed on TCD measurements, PaCO<sub>2</sub> must first be controlled because hypocapnia decreases CBF by a direct vasoconstrictive effect. Cerebral perfusion may then be restored by increasing MAP and/or use of osmotherapy [20]. Osmotherapy usually increases CBF in 15 minutes [24] for 4 hours. If necessary, it provides time for a computed tomography (CT) scan to be performed, then other indicated therapies (such as hypothermia or surgery) with restored cerebral perfusion.

Given the ease of use of TCD, it may even be considered before hospital admission to further reduce the duration of cerebral ischemic injuries. The feasibility of TCD measurements in pre-hospital settings has been demonstrated [27, 28].



## Computed Tomography Scans

CT scans are central to diagnosis of surgical situations. About 10 % of patients with severe TBI need neurosurgery [3, 6, 13]. The timing of a CT scan is important, because hemorrhagic lesions increase in the first post-trauma hours. When the CT scan is done in the first 2 hours after trauma, Oertel et al. observed growing lesions in 50 % of cases with neurosurgery indicated in 30 % [29] (Fig. 5). Major factors of progressive lesions were age and coagulation abnormalities. Some studies have also shown that the second CT scan is more predictive of outcome than the first [30]. Nevertheless, a CT scan does not give us a real view of ischemic risks. Although CT scan classification from a “trauma data bank” was related to cerebral hypertension [31], ICP cannot be estimated based on CT scan results [32].



**Fig. 5.** Example of a 32-year old man with head trauma after a traffic accident. One hour after trauma, he was conscious with a normal transcranial Doppler (pulsatility index [PI] = 0.72). CT scan showed a minor subdural hematoma. After 3 hours, the patient became unconscious (Glasgow Coma Scale [GCS] = 5) with an abnormal transcranial Doppler (PI = 1.45, end-diastolic velocity [Vd] = 19 cm/s). Osmotherapy was performed before a second CT scan, which demonstrated an urgent surgical situation.

## Intracranial Monitoring

Guidelines for ICP monitoring are clear [17]. Insertion of an intracranial monitoring device for continuous ICP is recommended for severe TBI (GCS $\leq$ 8) in all cases when the CT scan is abnormal, and also when the vulnerability of the brain is suspected (age more than 40, hypotension or local signs) even with a normal CT scan [17]. ICP monitoring is urgent for rapid control of ischemic risks. However, it is first important to have a real evaluation of the GCS without any sedation or intoxication (alcohol can be present in 60 % of TBI patient [33]). Moreover, insertion of an intracranial monitoring device also requires hemostasis correction (international normalized ratio [INR]  $\leq$  1.5 [34], platelet count  $\geq$  100000). Therefore, intraparenchymal ICP monitoring, implemented by neurosurgeons or intensivists at the bedside, is not available until several hours after trauma [6, 23]. Intraventricular catheters must be placed in the operating room by neurosurgeons and are at risk of becoming infected.

A jugular catheter has been proposed to measure SjO<sub>2</sub> on admission [22] but insertion load and technical difficulties have limited expansion of this method. Brain tissue oxygen monitoring (PbtO<sub>2</sub>) could be an elegant additional monitoring technique. PbtO<sub>2</sub> probes can be inserted through the same burr-hole as an ICP probe and secured with a multiple-lumen bolt. PbtO<sub>2</sub>-guided management seems to be more informative than CPP monitoring alone [35] with some promising results on outcome [36].

## Discussion about Intracranial Monitoring

Several studies have shown that use of invasive monitoring greatly reduces mortality and morbidity of severe TBI patients using historical comparisons [37] or invasive versus non-invasive centers comparisons [38]. However, these results have been debated because of the lack of randomized studies and the conflicting results observed these last ten years [39]. Some publications, excluding patients who died during the first 48 hours, found a better outcome and shorter ICU stay *without* ICP monitoring [40].

We conducted an observational study including all patients with severe TBI in the Paris area during a 22-month period, and following them from field to hospital discharge (Paris-TBI study). An ICP device was placed in 51 % of the 504 severe TBI patients included. This rate is similar to or higher than that published in other countries (28 % [3] to 45 % [13]). We observed that ICP was less often inserted in patients older than 45 years or those with a GCS score of 3. Multivariate analysis with a propensity score analysis of ICP monitoring at admission showed a two-fold increase in early mortality (one week) for patients without ICP monitoring [6]. Other parameters independently associated with increased early mortality, with or without ICP monitoring, were: At least one episode of areactive mydriasis (odds ratio [OR]=2.8 [1.9–4.2]) or hypotension (OR = 2.8 [1.9–4.1], hemorrhagic shock (OR = 1.9 [1.1–3.1]), age more than 75 years (OR = 2.9 [1.5–5.2]) and GCS score less than 6 (OR = 2.1 [1.3–4.0]). Admission to a specialized center was an independent factor for favorable outcome (OR = 0.6 [0.4–0.9]). Early mortality was significantly increased in patients aged more than 45 years or with GCS at 3 *only* in the absence of ICP monitoring.

These results show that patients with some factors of poor prognosis benefit less from invasive monitoring and that the decision to use ICP monitoring inde-

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