Hemodynamic and metabolic parameters in brain injury

Parâmetros hemodinâmicos e hemometabólicos na isquemia cerebral traumática

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Marco Antônio Stefani²
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ABSTRACT

Introduction: Indirect monitoring of brain metabolism through parameters such as cerebral perfusion pressure (CPP), intracranial pressure (ICP), arterial CO2 partial pressure (PaCO2) and mean arterial blood pressure (MABP) is necessary to guide treatment and to prevent secondary cerebral ischemia. The aim of the present study was to analyze the association between cerebral hemodynamic and metabolic parameters and the occurrence of traumatic brain ischemia.

Methods: Thirty-one patients were prospectively assessed in the pediatric or adult intensive care unit of Hospital de Pronto Socorro (HPS), in Porto Alegre, Brasil, from April to December, 2003. Patients were 23 adults (aged 17 to 66 years-old) and eight children (aged 3 to 13 years-old) with severe traumatic brain injury (TBI). Mean age was 24 years. The inclusion criteria were Glasgow coma scale (GCS) below 8 and abnormal cranial computed tomography (CT) results. Intracranial pressure, mean arterial blood pressure, arterial CO2 partial pressure and cerebral perfusion pressure were recorded.

Results: Cerebral ischemia was identified in 13 adults (56.5%) and in seven children (87.5%). High MABP was associated with mortality (P<=0.005) in children. High ICP (P=0.03) and low CPP (P=0.007) in adults were associated with cerebral ischemia. Fourteen patients (45.2%) died: 13 adults (56.5%) and one child (12.5%). Adult patients with low CPP had a worse outcome with higher mortality rate (P=0.045).

Conclusions: High ICP, high MABP and low CPP were associated with traumatic brain ischemia and higher mortality rate in these patients.

Keywords: brain injury, intracranial pressure, cerebral ischemia

RESUMO

Introdução: O estudo de medidas indiretas do metabolismo cerebral, como a pressão de perfusão cerebral (PPC), pressão intracraniana (PIC), pressão arterial de CO2 (PaCO2) e pressão arterial média (PAM), é necessário para guiar o tratamento e prevenir a ocorrência de dano isquêmico secundário. O objetivo deste estudo foi analisar a associação entre os parâmetros hemometabólicos e a ocorrência de isquemia cerebral traumática.

Métodos: Trinta um pacientes foram prospectivamente seguidos nas Unidades de Tratamento Intensivo Pediátrico e do Trauma do Hospital de Pronto Socorro de Porto Alegre de abril a dezembro de 2003. Vinte e três adultos (17 a 56 anos de idade) e oito crianças (3 a 13 anos de idade) com traumatismo craniano grave foram avaliados. Os critérios de inclusão foram escore < oito na Escala de Coma de Glasgow e tomografia computadorizada de crânio alterada. Foram monitorizados a PIC, PAM, PaCO2 e PPC.

Resultados: A idade média foi 24 anos. Isquemia Cerebral foi identificada em 13 (56.5%) adultos e em sete (87.5%) crianças. A queda da PAM foi associada com a mortalidade (p<=0.005) em crianças. A elevação da PIC (p=0.03) e a queda da PPC (p=0.007) em adultos foram associadas com isquemia cerebral. Doze pacientes (45.2%) morreram: 12 adultos (56.5%) e um (12.5%) paciente era criança. Pacientes adultos com baixa PPC apresentam maior índice de mortalidade (p=0.03). Conclusão: A elevação da PIC e da PAM e a queda da PPC estão associadas com a isquemia cerebral traumática nestes pacientes e com maior taxa de mortalidade.

Palavras-chave: trauma craniano, pressão intracraniana, isquemia cerebral

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INTRODUCTION

Traumatic brain injury (TBI) is the most frequent cause for brain surgery, and it is an important social problem due to its high morbidity and mortality. In Brazil, TBI accounts for 57,000 deaths every year, most of which resulting from car accidents.

Intracranial hypertension (ICH) has been regarded as one of the major factors influencing mortality and morbidity after TBI. Previous studies showed a 50% mortality rate for severe TBI; after the implementation of TBI management protocols, the Traumatic Coma Data Bank demonstrated that this rate may drop to 36%.

Stevens (2004) claims that currently available monitoring techniques used after severe TBI employ indirect measurements of cerebral oxygenation and perfusion, such as intracranial pressure (ICP) and cerebral perfusion pressure (CPP); moreover, proper information on cerebral blood flow (CBF) and oxygenation is necessary to appropriately guide treatment and to prevent secondary cerebral ischemia. Treatment protocols for the management of severe head injury should emphasize the immediate reduction of raised ICP to less than 20mmHg if possible. A CPP greater than 60mmHg appears to have little influence on the outcome of patients with severe head injury.

Previously reported neuropathological findings from cadavers have revealed a high rate of ischemic brain damage in severe non-missile brain trauma. The study hypothesis was that patients with severe TBI, metabolic and dynamic abnormalities (intracranial hypertension and cerebral hypoperfusion) presented with ischemic evolution.

The aim of this study is to evaluate cerebral ischemia occurrence in patients presenting with severe TBI and submitted to intracranial pressure monitoring. Also, we evaluated which hemodynamic and metabolic variables are associated with cerebral ischemia and death.

MATERIAL AND METHODS

An observational, follow-up cohort study of patients with severe closed traumatic brain injury was carried out at Hospital de Pronto Socorro (HPS) in Porto Alegre, Brazil, between March and December, 2003. The inclusion criteria were abnormal neuroimaging results and a Glasgow coma scale (GCS) equal to or less than 8. Those patients with GCS greater than 8, penetrating head trauma or firearm-related injuries and those with normal computed tomography (CT) scans were excluded from the study.

Thirty-one intensive care unit (ICU) patients who met the inclusion criteria were enrolled in the study. The patients were monitored 24 hours a day for an average of five days, for control of hemodynamic and metabolic parameters [ICP, arterial CO2 partial pressure (PaCO2), mean arterial blood pressure (MAPB), and CPP], as well as of CT scan findings. Evidence of cerebral ischemia was evaluated through abnormal neuroimaging results and autopsy results from Legal Medical Institute of Porto Alegre (Table 1). The study protocol was approved by the Research Ethics Committee of Pronto Socorro Hospital and Legal Medical Institute of Porto Alegre.

Intracranial pressure was assessed by the placement of a ventricular catheter and of a Codman® microsensor (Johnson & Johnson) by the attending brain surgeon. A digital ICP Express 82-6633ICP monitor was used. The catheter and the microsensor were placed separately through the same burr hole. The microsensor was placed intraparenchymally at a depth of approximately 4cm. The microsensor was zeroed prior to its placement.

The mean ICP measurements were analyzed separately for each monitoring day, and the mean ICP of each day was considered for the analysis.

The mean CPP was assessed by the difference between MAPB and ICP values. CPP values greater than 70 mmHg for adults and greater than 50 mmHg for children were regarded as adequate. Treatment was implemented according to the protocol for severe TBI management.

Table 1. Clinical, Tomography and Autopsy results

<table>
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<th>Case</th>
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<th>CH</th>
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CT: Cranial Tomography; ICH: Intracranial Hypertension; TI: Traumatic Ischemia; IH: Intracranial Hemorrhage; CF: Cranial Fracture; a: Nameless; F: Female; M: Male.

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Both systolic blood pressure (SBP) and diastolic blood pressure (DBP) were checked every hour, but in some patients, the catheter was placed in a radial artery for direct MABP monitoring. Among pediatric patients, MABP was measured by a catheter placed in a radial artery. Both adults and children were submitted to noninvasive SBP/DBP measurements every hour.

Arterial CO2 partial pressure was checked every day in all adult and pediatric patients, but in some of them, it was assessed twice or more times a day for the sake of therapeutic management. PaCO2 values were obtained by an AVL – Ominini® arterial blood gas analyzer after the collection of 2 ml of arterial blood. The therapeutic aim was to maintain hypocarbia at moderate (28-33 mmHg) or normal (35-40 mmHg) levels.

A cranial CT scan was performed on admission (a routine practice at HPS) and after 24 hours of the traumatic brain injury if the patient’s clinical status so allowed. Evidence of ischemic insults secondary to the trauma was expected to be found on the second CT scan. Abnormal CT scan results included effacement of perimesencephalic cisterns and of the third ventricle, midline shift, intracranial hemorrhage of any etiology, and focal or diffuse cerebral contusion.

Occurrence of cerebral ischemia was defined by presence of ischemic findings on the CT scan and/or focal sensory/motor deficits. Ischemic findings were defined as focal and/or diffuse lesions shown by the CT scan 24 hours after TBI. Evidence of hypodensity and cerebral edema during the clinical outcome, associated with a reduction in CPP and focal sensory/motor deficits, was defined as a posttraumatic ischemic event. CT ischemia evidence was defined by occurrence of hypodensity after 24 hours of event. Autopsy and CT abnormalities were compared and ischemic findings were described in Table 1.

### Results

All of the 31 patients were referred to the ICU due to severe TBI. Mean age ± SD of patients was 24±14 years, with a range of 3 to 66 years. Twenty-six (83.9%) patients were male. TBI was caused by traffic accident in 77.4% (24) of the cases, and by work-related accidents in the remaining cases. The incidence of ischemia after TBI amounted to 64.5% (20 patients), and the mortality rate corresponded to 45.2% (14 patients) (Table 2).

| Table 2. Chart of 31 Patients with Severe Traumatic Brain Injury (TBI) |
|------------------------|------------------------|
| Age in years (media ± SD) | 24 ± 14 (3-66 years) |
| White race (%) | 27 (87.1) |
| Male Sex (%) | 26 (83.9) |
| Glasgow (1° day of ICP) a (median ± SD) | 5 ± 2 |
| Time TBI – ICP (in hours) b (media ± SD) | 24 ± 25 |
| Cause of TBI like traffic accident (%) | 24 (77.4) |
| Ischemia (%) | 20 (64.5) |
| Death (%) | 14 (45.2) |

a score in Coma Glasgow Scale in first day of ICP monitoring
b time between TBI and beginning of ICP monitoring

For analysis of hemodynamic and metabolic variables, except PaCO2, the patients were subdivided into adults and children, since baseline values vary with age. The hemodynamic and metabolic variables (ICP, MABP, CPP, PaCO2) were assessed for an average period of 5 days. Some patients were monitored for a longer period (mean of 7 days), whereas other died within the first hours of monitoring.

A total of 23 adult patients were admitted to the ICU. Mean age was 29±12 years, and 21 (93.3%) patients were male. A Glasgow coma scale of 5 was detected at the time of indication for ICP monitoring and the time between TBI and the monitoring corresponded to 23±25 hours (range of 4 to 120 hours). The incidence of ischemia and death after TBI was 56.5% (13 patients).

Eight patients were admitted to the pediatric ICU of HPS, five males (62.5%) and three females (37.5%). The incidence of is-
Ischemia and death versus intracranial pressure

The comparison of patients with posttraumatic ischemia with those without ischemic findings on the CT scan revealed that an increase in ICP on the fourth day was significantly associated with ischemia (35±16 x 19±6, P=0.037, Student’s t test) (Fig. 1). There were no statistically significant differences in intracranial pressure among nonsurvivors.

Figure 1. Increase in ICP on the fourth day was significantly associated with ischemia (35±16 x 19±6, P=0.037, Student’s t test)

Cerebral perfusion pressure and traumatic ischemia in adults

Figure 2. CPP values (38±16 x 65±19, P=0.007, Student’s t test) in adults on the fourth day showed significant differences.

Cerebral perfusion pressure (mmHg) and death in adults

Figure 3. CPP decrease in nonsurvivors comparatively to survivors on the fourth day (37±20 x 57±17, P= 0.045, Student’s t test).

Mean arterial blood pressure

There was a greater probability of fatal complications in the post-TBI clinical outcome, but the decrease in MABP may reduce mortality. Even though no statistical difference was observed on the fourth day of monitoring, an association between death and MABP decrease is likely to exist. By comparing nonsurvivors with survivors, MABP is probably lower among the former (65±17 x 80±12, P=0.05, Student’s t test). Comparison of patients with posttraumatic ischemic findings with those without any findings on the CT scan revealed that the decrease in MABP was not significantly correlated with ischemia.

Partial CO2 arterial pressure

By comparing non-survivors with survivors, mean PaCO2 was not significantly different in adult patients, or in those with ischemic findings relative to those without any findings on the CT scan.
**PEDIATRIC GROUP**

No statistically significant differences were found between CPP, ICP and PaCO2 in the pediatric group when the groups were compared in terms of ischemic findings and death. The child who died showed statistically significant difference in MABP increase (Fig. 4).

![Figure 4: The only patient who died had a greater MABP on the first and second days of ICP monitoring, respectively (87 x 59 ±5, P=0.005, Student’s t test) (90 x 59 ±6, P=0.006, Student’s t test).](image)

By comparing the only patient who eventually died with survivors, the former had a greater MABP on the first and second days of ICP monitoring, respectively (87 x 59 ±5, P=0.005) (90 x 59 ±6, P=0.006) (Fig. 4).

**DISCUSSION**

Traumatic brain injury is a major public health problem. Since it often victimizes young people of working age, it causes tremendous human and socioeconomic losses in Brazil. Over 50,000 deaths from TBI occur every year, and neurological morbidity is observed in about 5-10% of these cases.

The mean age of patients with severe TBI was compatible with that described in the literature. The overall mean age of patients was 24 years, with a range of 3 to 66 years. Of these patients with TBI, 77% of traumas arose from traffic accidents. Andrade et al (2001) describe that the highest incidence of TBI is found in the 15-24 year age group, and that 50% of these cases result from traffic accidents.

According to the National Center for Health Statistics, the male/female ratio is 2:1. Males are usually more often affected than females, with a rate of 84% (5:1 male/female ratio). The higher male preponderance in this study is likely due to the large number of men who commute to work by car every day.

The management of patients with severe TBI starts with primary care, with appropriate support and transportation to specialized health care facilities. Initial treatment consists in maintaining the patient’s physiological status and treating ICH, thus preserving CBF. Ghajar and Hariri (1992) state that response to trauma is similar between adults and children, and that the self-regulating mechanism is maintained.

Initial treatment should be aggressive in order to prevent hypoxia and hypotension. Maintenance of adequate arterial blood pressure preserves CBF and, consequently, cerebral perfusion. ICP monitoring plays a crucial role in the neurological assessment of traumatically brain-injured patients. Increases in ICP are a leading cause of death among neurosurgical patients, and are frequent in cases of TBI. Miller et al reported that 40% of patients with severe TBI had an increase in ICP and, of these, 50% eventually died. Jull et al showed that the reduction of ICP to less than 20 mmHg is mandatory in adult patients with severe TBI. Pfenninger and Santi describe a remarkable association of sustained ICP levels equal to or greater than 20 mmHg with dismal prognosis in children.

Based on our data, we can observe mean intracranial pressures greater than 20 mmHg during the clinical outcome of adult and pediatric patients (Table 3 and 4). Mean ICP in children was higher on the first days following TBI, dropping thereafter. In adults, ICP remained stable with a slight increase after the development of trauma. In patients with a fatal outcome and with ischemic findings on the CT scan, mean ICP was 35 mmHg, and tended to be associated with posttraumatic ischemia (P=0.06, Student’s t test). All of these patients were diagnosed with intracranial hypertension by ICP monitoring.

![Table 3: Hemodynamic and Metabolites Parameters in Adults (n=23)](image)

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<td>MABP (mean ±SD)</td>
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<td>78±14</td>
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<td>33±6</td>
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![Table 4: Hemodynamic and Metabolites Parameters in Pediatric patients (n=8)](image)

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<td>MABP (mean ±SD)</td>
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Jull et al showed that an ICP greater than 20 mmHg is associated with neurological deterioration. The statistical significance of posttraumatic ischemia in patients with sustained ICP increase (35 ± 16 mmHg, P=0.037, Student's t test) may contribute to neurological deterioration, as described in the literature. By analyzing Figure 1, we note that mean ICP was persistently greater than 30 mmHg among nonsurvivors and equal to 21 mmHg among survivors, without statistical significance. This corroborates the assumption that a mean ICP of 20 mmHg is the acceptable threshold, as proposed by some authors, and also shows that ICP was more refractory to treatment in nonsurvivors.

Adelson et al suggest that a CPP of 50-65 mmHg is appropriate for the pediatric group and that a CPP less than 40 mmHg is consistently associated with an increase in mortality. In the pediatric group, mean CPP was 40 mmHg and mean ICP amounted to 32 mmHg, whereas in adults, mean CPP was 52 mmHg and mean ICP was 31 mmHg (Table 3 and 4). Although the pediatric group had a slightly higher mean ICP and a lower mean CPP, mortality was higher among adult patients.

Usually, CBF remains constant at CPP levels between 60 and 160 mmHg. However, in TBI this self-regulating mechanism can be affected by several factors. Jull et al describe that a CPP greater than 60 mmHg seems to play a minor role in the prognosis of patients with severe TBI. We found a significant association between mean CPP values greater than 60 mmHg (65 mmHg) and lack of posttraumatic ischemic findings (P=0.007, Student's t test). A significant association was also noted between CPP decrease and the fatal outcome (P=0.045, Student’s t test).

CPP in survivors was initially similar to that of other patients and that it worsened with time among ischemic patients (Fig. 3). The same occurred among non-survivors. This also testifies to the notion that ideal CPP is that above 60 mmHg. In fact, controversy exists over whether it should be greater than 60 or greater than 70 mmHg, but in this study we observed that when CPP was around 60 mmHg (57 mmHg), survival rate increased.

The only child who eventually died had a higher MABP on the first 2 days of ICP monitoring (P<0.05, Student’s t test).

Hypercarbia increases intracranial pressure, whereas hypocarbia has the opposite effect; desirable PaCO₂ levels are around 32 mmHg, but a PaCO₂ less than 28 mmHg should be avoided during hyperventilation, especially on the first day of trauma, unless other therapeutic measures have failed. Cruz recommends a PaCO₂ of 20-30 mmHg for 3-4 days until hyperventilation is optimized. This author also points out the necessity for gradual stabilization of PaCO₂ at 30 mmHg when the patient’s ICP levels tend to normalize and after at least 4 days.

The five-day analysis of mean PaCO₂ levels in patients with fatal outcome and posttraumatic ischemia allows us to assert that there was no statistically significant difference between the study groups. We believe this variable was not statistically significant due to the use of a standard protocol for the management of severe TBI. In this protocol, we did not test for hypercarbia or hypocarbia, but we assessed the associations of CPP and ICP with ischemia and mortality.

We found statistically significant differences between ICP increase and CPP decrease in adults with ischemia. In children, despite no statistically significant difference, we perceived that mortality was lower, even at a CPP level around 40mmHg, but there was a larger incidence of ischemic findings. The increase in MABP in children results from a reactive phenomenon, and the increase in ICP is the major cause of death in this group. This evidence probably suggests that children are more tolerant of ICP increases than adults, which therefore explains the lower mortality rate.

Posttraumatic ischemia in patients with severe TBI submitted to ICP monitoring showed an incidence of 64.5% (20 patients). In adult patients, this incidence amounted to 56.5% (13 patients). The diagnosis of ischemia was more common in children (85.5%, 7 patients) and among patients that eventually died (87.7%, 12 patients).

Traumatic brain injury is the most common cause of death and disability in young adults living in industrialised countries, in which 180-250 people per 100 000 per year die or are hospitalised as a result. Mortality rates found in the present study are consistent with those described in the literature, amounting to 45% (14 deaths). We observed that 13 deaths (56.5%) occurred among adults, with only one death (12.5%) occurring among children. The increase in ICP and in MABP and the decrease in CPP are associated with mortality and posttraumatic cerebral ischemia in our patients. Interestingly enough, children tolerate higher ICP than adults and have a lower mortality rate, but higher incidence of ischemic findings on CT scans.

There is no question that primary prevention of severe traumatic brain injury is the desired approach to this common and devastating problem. Nevertheless, advances in the prevention of secondary injury are of great importance for cases in which primary prevention has failed. The goal of clinicians and scientists in this field is to identify the sequence of events in secondary injury with the aim of developing targeted, specific interventions to improve the outcomes of severe and mild traumatic brain injury.

Secondary TBI can be prevented by maintaining homeostasis during ICP monitoring. Although the greatest impact on survival and outcome to date may be attributed to systemic and intracranial physiologic management (e.g., fluid resuscitation, intracranial pressure monitoring), future mitigation of the progression of secondary injury will likely be through molecular, gene and pharmacologic interventions.
We highlight the importance of ICP monitoring for the acute management of severe TBI in both children and adults. Based on this variable, we can check CPP and MABP values in order to control for abnormal cerebral hemodynamic and metabolic parameters and to intervene whenever necessary.

REFERENCES


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