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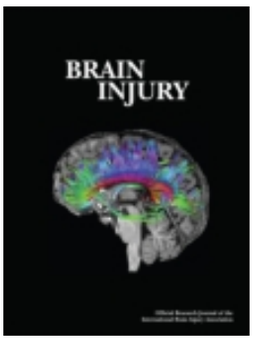

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Assessment of intra-cranial pressure after severe traumatic brain injury by transcranial Doppler ultrasonography

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Abstract

Primary objective: To investigate the potential of transcranial Doppler ultrasonography in estimating post-traumatic intra-cranial pressure early after severe traumatic brain injury.

Research design: The group of 24 patients was analysed for the observation of an early post-traumatic cerebral haemodynamic by middle cerebral artery blood velocity measuring.

Methods and procedures: The standard method of measuring the mean blood middle cerebral artery velocity by transcranial Doppler ultrasonic device was performed.

Main outcomes and results: The increased duration of intra-cranial hypertension correlated to the middle cerebral artery low blood velocity ($p = 0.042$; $r = -0.498$) ($n = 17$) and to elevated pulsatility indices ($p = 0.007$; $r = 0.753$) ($n = 11$) significantly. The increased duration of lowered cerebral perfusion pressure correlated to the middle cerebral artery low blood velocity significantly ($p = 0.001$; $r = -0.619$) ($n = 24$).

Conclusions: The significance of transcranial Doppler ultrasonography as a method to estimate an early post-traumatic intra-cranial pressure after severe brain injury was confirmed. This simple and non-invasive technique could be easily used in daily clinical practice and precede intra-cranial pressure monitoring in selected patients.

Keywords: *Ultrasonography, Doppler, brain injuries, intracranial pressure*

Introduction

The hypothesis of this study is that it is possible to record patterns of cerebral haemodynamic disturbances early after severe traumatic brain injury (TBI) and to evaluate an intra-cranial pressure (ICP) by an ultrasonic transcranial Doppler (TCD) device in patients suffering such an injury [1]. Therefore, it is probable to assess ICP indirectly by measuring the middle cerebral artery (MCA) blood velocity, as well as by estimating pulsatility indices (PI) [2–6].

There are multiple aetiologies of intra-cranial hypertension after severe TBI [7]. Cerebral blood flow instability following such an injury is strongly connected to the injury itself, as well as to the development of secondary mechanism of traumatic brain lesion resulting in brain tissue ischemia [8] and intra-cranial hypertension [9, 10]. The concept of secondary brain damage is related to the fact that the entire neuronal lesion does not happen at the instant of injury, but it extends progressively in hours and days after injury [11–13]. Brain oedema is the main consequence of such an injury [14], generating enlargement of brain tissue mass inside an anatomically unchangeable endocranial space [15, 16]. The result of elevated ICP that produces a compressive effect to cerebral blood vessels is a cerebral perfusion

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pressure (CPP) decrease as the most common cause of brain tissue ischemia [15, 17]. Thus, reducing ICP, as well as sustaining CPP values above critical level, is essential to evade ischemic brain lesions [18, 19]. Subsequently, there is a direct relationship between post-injury cerebral haemodynamics impairment and development of secondary brain tissue ischemia [19].

Marmarou et al. [20] stated that the proportion of hourly elevated ICP readings (i.e. duration of intracranial hypertension in hours per day for a sustained period of time) was also highly significant in explaining the outcome of severe brain injury. Concerning this, one has adopted their concept of duration of elevated ICP expressed in hours per day, to support the hypothesis and to interpret the results of this study.

The purpose of this article is to indicate the value of TCD ultrasonography in the early assessment of intra-cranial pressure following severe TBI. In such a way, TCD ultrasonography could be useful in the management of selected patients with severe head injury as a screening method to determine a specific group of patients in whom ICP monitoring is mandatory. In contrast to well known invasive routine methods of ICP monitoring that can only be successfully performed by skilled surgeons, this plain and non-invasive technique could be easily, accurately and commonly utilized in daily clinical practice by non-surgical medical personnel.

Participants and methods

During a 1-year period, between January and December 2004, 24 consecutive patients suffering severe traumatic non-penetrating brain injury who were treated at the Division of Neurosurgery, Osijek Clinical Hospital, Osijek, Croatia, were selected for the study.

There were 21 men and three women in the series. The mean age of the group was 35.9 years (SD $\pm$ 17.23 years) and the median was 29.5 years, ranging from 18–65 years. The mechanisms of injury were traffic accidents in 21 and falls in three cases. Associated injuries were recorded in 13 out of 24 patients in this series. There were five patients with skeletal bone fracture, two with thoracic injury and two with spinal injury. Four patients sustained multiple life threatening associated injuries.

Eleven patients underwent early surgery due to mass lesions, i.e. acute intra-cranial haematoma, while the remaining 13 were treated without surgery. Out of 11 surgical patients, six underwent unilateral left side craniotomy and five were operated on at the right side of the head. The bone flap was removed in five out of 11 surgically treated patients.

Regarding the extent of injury, diffuse axonal bihemispheral injury was recorded in nine out of 13 conservatively treated patients, while unilateral focal damage up to 3 cm in diameter was noticed for the remaining four. In all patients cerebral lesion was located supratentorially, while in eight out of 24 patients post-traumatic subarachnoidal haemorrhage was recorded.

The inclusion criteria were patients' state of conscience assessed by the Glasgow Coma Scale Score (GCS) of equal to or less than 8, recorded intra-cranial hypertension of ICP higher than 25 mm Hg, as well as the patients' age between 18–65 years.

The patients who satisfied the inclusion criteria, but in whom ultrasonographic bone window was not possible, and therefore it was unable to obtain TCD measurements, were excluded from the study. The patients who underwent surgery immediately upon admission due to their life threatening condition were also excluded. The patients who suffered severe brain injury but whose ICP values recorded via the external venticulostomy were normal were not included too. Therefore, 24 patients remained for the final analysis according to these criteria.

Following hospital admission and computed tomography (CT) brain scanning, ICP monitoring was performed in all 24 patients. Intra-cranial pressure was measured continuously every hour per day by the external ventriculostomy, from the start of the ventricular catheter placement until it was removed. The highest number of ICP recordings in a given hour was assigned as an hourly measure of intra-cranial hypertension. Data were collected directly from a bedside monitor by the use of an interfaced computer. The mean length of permanent ICP monitoring was 5.92 days (SD $\pm$ 2.22 days) and the median was 5.00 days, ranging form 3–12 days after admission.

All the patients were admitted to the Intensive Care Unit and mechanically ventilated and antimicrobial prophylaxis was applied in all. Intracranial pressure was maintained below 25 mm Hg by moderate hyperventilation ($pCO_2 > 30$ mm Hg), intermittent 20% mannitol intravenous infusion (up to four time a day) and by the external ventriculostomy. Cerebral perfusion pressure was maintained above 70 mm Hg by the intensive fluid resuscitation and the administration of vasopressor agents when necessary (dopamine, administered as a continuous 24-hour intravenous infusion in a dosage of 20 ml h^{-1}).

The standard technique of insonating the middle cerebral artery and measuring the mean blood velocity by TCD ultrasonic device of 2 MHz (Trans Scan-3D Scanner, EME GmbH, Überlingen, Germany) has been applied in all patients within the

first 24 hours following hospital admission. The initial TCD measurements were taken as early as possible, immediately after the patient was haemodynamically stabilized and the diagnostics was completed. The precise interval between injury and time of TCD recordings was not specified. Measurements were always taken prior to surgery in the group of patients who were operated on. Doppler tests were obtained once during day 1 post-injury and discontinued afterwards. All the measurements in every patient were the responsibility of the same two-person team of operators.

The following parameters were recorded from TCD measurements: Normal MCA blood velocity (50–74 cm s^{-1}); decreased the MCA blood velocity (40 ± 10 cm s^{-1}); normal PI values ($PI \leq 1$); pathological PI values ($PI > 1$). The pulsatility index was calculated from the difference between end systolic and diastolic the MCA velocities divided by the mean MCA velocity.

Parameters recorded from cerebral haemodynamic measurements (ICP monitoring) were as follows: Number of hours of the elevated ICP per day ($ICP > 25$ mm Hg); number of hours of the decreased CPP per day ($CPP < 70$ mm Hg).

For the purpose of this study, the elevated ICP is defined as a pressure higher than 25 mm of mercury, while the decreased CPP is described as a perfusion pressure lower than 70 mm Hg. Cerebral perfusion pressure was individually calculated from the difference between the mean arterial blood pressure and ICP.

Eighteen patients survived, while six succumbed to severe head injury. Different complications were recorded in seven patients who survived. The most common complications were pneumonia in five cases, followed by hydrocephalus and deep venous thrombosis in one case each.

Doppler and cerebral haemodynamic parameters were compared to reach the results. Correlations between haemodynamic and TCD variables were calculated by Pearson's coefficient of correlation (r). Statistical significance of correlation between the investigated quantitative variables was obtained by the p-level. The level of significance was set at $p < 0.05$. The linear regression equations were calculated in the regression analysis of haemodynamic and TCD variables.

Commercially available software (SPSS for Windows, release 9.0.0, by SPSS Inc., Chicago, IL) was utilized for data processing and analysis.

Results

The normal MCA velocity (50–74 cm s^{-1}) was recorded by ultrasonic TCD device in 16 out of 24 patients, while the decreased MCA blood velocity (40 ± 10 cm s^{-1}) was recorded in the remaining eight patients. Normal PI values ($PI \leq 1$) were calculated in 14 out of 24 patients, while the remaining 10 had the pathological PI values ($PI > 1$) (Table I).

Table I. Distribution of patients according to the measured middle cerebral artery blood velocity, calculated values of the pulsatility index and daily duration of intra-cranial hypertension.

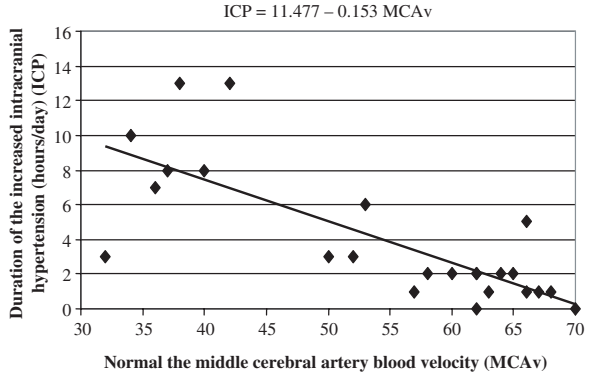
MCA blood velocity (cm/s) ($n = 24$)	
<50	8
50–74	16
Pulsatility Index ($n = 24$)	
≤1	14
>1	10
Duration of elevated ICP (hours per day) ($n = 24$)	
<6	17
≥6	7
Total	24

The considerable correlations between the TCD parameters and the daily duration of the elevated ICP, as well as the decreased CPP were established.

A statistically significant negative correlations ($p = 0.042$; $r = -0.498$) between the MCA blood velocities (MCAv) and the duration of the elevated intra-cranial pressure ($ICP > 25$ mm Hg) per day were noticed in the group of patients with normal blood velocities (50–74 cm s^{-1}) ($n = 17$). The increased duration of intra-cranial hypertension was followed by the decrease in the MCA velocity. Therefore, the increased number of hours of intra-cranial hypertension correlated to low blood velocity significantly. The relation was generalized by the linear regression equation $ICP = 11.477 - 0.153\,MCAv$, which means that the increase of the MCA blood velocity for one unit expectedly decreases ICP for 0.153 units (Figure 1).

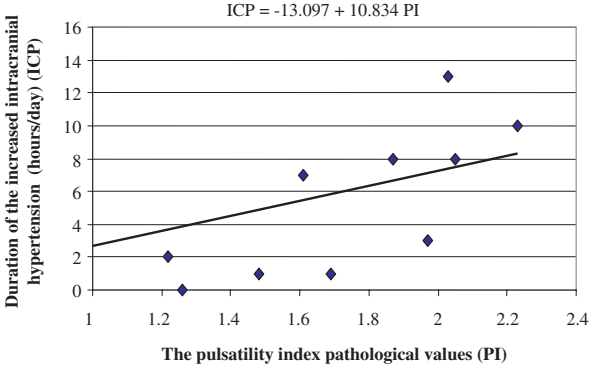
There was also a statistically significant correlation ($p = 0.007$; $r = 0.753$) between the PI values and the duration of intra-cranial hypertension recorded in the group of patients with pathological PI values ($PI > 1$) ($n = 11$). Longer duration of the elevated ICP per day was followed by a considerable increase of the PI values. Consequently, the increased number of hours of intra-cranial hypertension correlated to the increased PI values significantly. The relation was generalized by the linear regression equation $ICP = -13.097 + 10.834$ PI, which means that the increase of PI value for one unit expectedly increases ICP for 10.834 units (Figure 2).

Correlation between duration of the decreased CPP (<70 mm Hg) per day and the MCA blood velocity was negative and also statistically significant in this series ($p = 0.001$; $r = -0.619$) ($n = 24$). Longer duration of the decreased CPP was followed by the decrease in blood velocity. Thus, the



ICP: Duration of the increased intracranial pressure
MCAv: The middle cerebral artery blood velocity

Figure 1. Correlation between the middle cerebral artery velocity and duration of intra-cranial hypertension in the patients with normal blood velocity ($n = 17$). Correlation to duration of intra-cranial hypertension was negative ($r = -0.498$) and statistically significant ($p = 0.042$).



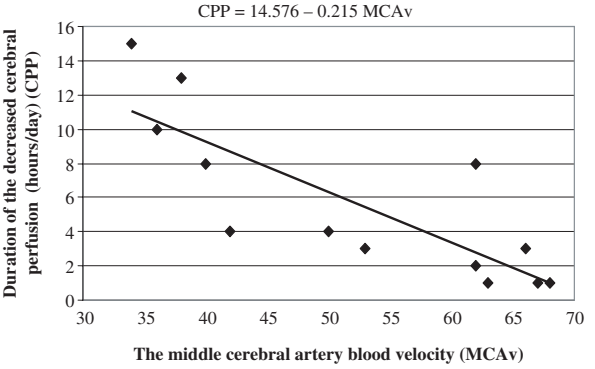
ICP: Duration of the increased intracranial pressure
PI: The pulsatility index

Figure 2. Correlation between values of the pulsatility index and duration of intra-cranial hypertension in the patients with pathological values of the pulsatility index ($n = 11$). A statistically highly significant correlation ($p = 0.007$; $r = 0.753$) between duration of intra-cranial hypertension and values of the pulsatility index was observed.

increased number of hours of cerebral hypoperfusion correlated to low blood velocity significantly. The relation was generalized by the linear regression equation $CPP = 14.576 - 0.215\,MCAv$, which means that the increase of the MCA blood velocity for one unit expectedly decreases CPP for 0.215 units (Figure 3).

Discussion

An insufficient level of cerebral blood flow is the main origin of secondary ischemic brain lesion [21].



CPP: Duration of the decreased cerebral perfusion pressure
MCAv: The middle cerebral artery blood velocity

Figure 3. Correlation between the middle cerebral artery velocity and duration of the decreased cerebral perfusion pressure ($n = 24$). A statistically highly significant negative correlation ($p = 0.001$; $r = -0.619$) between duration of the decreased cerebral perfusion and blood velocities was observed.

Instantly after severe brain injury cerebral blood flow is extremely low and near the ischemic threshold [8]. In the settings of intra-cranial hypertension after such an injury a reduction in cerebral perfusion may occur, presuming that the patient is haemodynamically stable with normotensive mean arterial pressure (MAP).

According to the results of this study, the longer duration of intra-cranial hypertension immediately after severe TBI was followed by a statistically significant decrease of the MCA blood velocity. The authors would also like to stress that intra-cranial hypertension was recorded via the external ventriculostomy in all the patients in the series, while the patients with normal ICP levels were excluded from the analysis. In such a way, TCD measurements were limited to the patients with elevated ICP. Therefore, only the duration of intra-cranial hypertension ($ICP > 25$ mm Hg) was correlated to the MCA blood velocity.

Since cerebral blood velocity after severe TBI is strongly correlated to cerebral blood flow itself [16, 22], it can be concluded that the elevation of ICP corresponds to the decrease of cerebral blood flow [15]. Therefore, cerebral blood flow after severe brain injury is drastically decreased when marked duration of intra-cranial hypertension is present. Considering this, it is possible to notice a particular elevation of ICP after such an injury by measuring the MCA velocity [15, 23, 24]. In such a way, measuring blood velocity by a TCD device is a valuable early indicator of intra-cranial hypertension, that could precede ICP monitoring [24–27].

By measuring the MCA velocity, this study has recorded that the diastolic velocities were much more decreased than the systolic, which was directly correlated to the increase of the PI values. Normal PI values are $PI = 0.90 \pm 0.24$ [28]. The pulsatility index is an important concept, which makes the measurement dimensionless and unitless, as well as independent of the angle of insonation and the need to get the most precise velocity [29].

The values of PI are potential indicators of a disturbed cerebral perfusion, as well as of a development of intra-cranial hypertension [4]. Correlating the PI values to the duration of elevated ICP in this series, it was recorded that longer duration of intra-cranial hypertension was followed by the increase of the PI values, which was highly statistically significant.

Therefore, it can be concluded that intra-cranial hypertension corresponds to the increased PI values. Consequently, the decrease of blood velocity corresponds to the increase of the PI values. Concerning this, cerebral blood flow immediately after severe brain injury is decreased when the PI values are increased [26, 30]. Thus, the PI values are also strong indicators of post-traumatic cerebral blood flow and cerebral ischemia, as well as ICP and CPP [29, 31].

A statistically highly significant negative correlation between the duration of the decreased cerebral perfusion and the MCA blood velocities was recorded in this series. Regarding this, the decreased cerebral perfusion is correlated to low blood velocity [2, 19, 32].

Since blood velocity corresponds to a post-traumatic cerebral blood flow itself [33–35], it can be concluded that an early post-traumatic cerebral blood flow is decreased when low CPP is recorded for a longer period of time. Therefore, blood velocity is also an indicator of post-traumatic cerebral perfusion [2, 15, 27, 36].

The probable differences in variation of the increased ICP levels between the sub-groups of surgically vs. non-surgically treated patients; patients with focal vs. diffuse brain injury; survived patients vs. those who died; and patients with more severe vs. less severe brain injury were not possible to investigate due to a limited number of patients in this series which would make the sub-groups too small to fit the statistical analysis.

The fundamental finding in this study is the fact that intra-cranial hypertension after severe brain injury creates cerebral haemodynamic disturbances that are transferred into slower blood velocity and higher pulsatility. An early low blood velocity was recorded in one third of patients in this series and was mostly accompanied by high pulsatility indices.

Considering the results of this study, it has been concluded that TCD ultrasonography for measuring the MCA blood velocity after severe TBI has been proven to be a worthy tool to estimate ICP, as well as to evaluate an early post-traumatic cerebral haemodynamics. It significantly correlates to ICP and CPP when performed within hours of injury.

Contrary to well known invasive routine methods of ICP monitoring [9] that can only be successfully carried out by a surgical expert, this technique is simple and non-invasive. Therefore, it could be easily and frequently performed in everyday clinical practice by non-surgical medical personnel as a screening method to precede ICP monitoring in selected patients. In an emergency, it can be utilized as a method of choice alone, if ICP monitoring is not available.

Concerning these results, it is still hard to tell whether it is possible to use this method as a sufficiently accurate substitute for ICP monitoring in the early course of severe brain injury on a regular basis. It is obvious that more prospective studies based on much more extensive material are required to maintain these findings and to provide further supportive evidence of this method.

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