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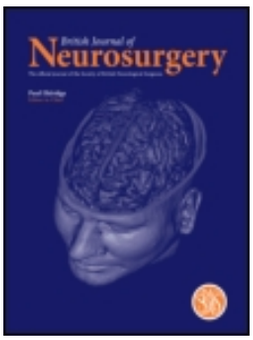

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ORIGINAL ARTICLE

Transcranial Doppler ultrasonography as an early outcome forecaster following severe brain injury

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Abstract
Knowledge of post-traumatic cerebral haemodynamic disturbances might be beneficial for predicting the management outcome when measuring the basal cerebral arteries blood flow velocity by ultrasonic transcranial Doppler device immediately after severe head injury. Thirty patients who sustained severe brain injury underwent an early blood velocity measuring by transcranial Doppler ultrasonography during a 1-year period of study. The standard technique of measuring the mean blood flow velocity in the middle cerebral artery was applied. The outcome was assessed at 6-month follow-up by the Glasgow Outcome Score. The middle cerebral artery low blood flow velocity, and the increased values of the pulsatility index significantly correlated to an unfavourable outcome. Transcranial Doppler ultrasonography for measuring the middle cerebral artery blood flow velocity has been proved worthy as a possible predictor of severe head injury management outcome. This non-invasive and simple procedure could be engaged in the daily management of severely brain-injured patients.

Key words: *Severe head injury, transcranial Doppler, outcome.*

Introduction

The hypothesis of this study is that it is possible to predict the management outcome in severely head-injured patients by transcranial Doppler (TCD) ultrasonography. By measuring the middle cerebral artery (MCA) blood flow velocity, as well as by estimating pulsatility indices (PI), it is probable to assess the outcome since low flow velocities and the increased PI values are a direct result of post-traumatic cerebral haemodynamics disorder that could well affect the prognosis of such an injury.

Cerebral blood flow disturbances that occur after severe head injury are robustly linked to the injury *per se*, as well as to the development of a secondary mechanism of traumatic brain lesion. The type of lesion is well recognized as an important determinant of outcome after severe head injury.[1] Frequently, post-traumatic contusions are accompanied by cerebral haemorrhage. Traumatic contusions could be enlarged,[2] causing intracranial hypertension and secondary brain tissue ischaemia,[3–5] which may describe the further deterioration that is often seen in these patients. Nevertheless, the ultimate result of such a lesion is brain tissue ischaemia[6] and intracranial pressure (ICP) hypertension.[7,8] The result of elevated ICP, which produces a compressive effect to cerebral blood vessels, is a cerebral perfusion pressure (CPP) decrease that is the most common cause of brain tissue ischaemia.[9] Among patients who sustained severe traumatic brain injury, elevated ICP was the main cause of death.[10] Consequently, there is a direct relationship between postinjury cerebral circulation impairment and the advancement of secondary brain tissue ischaemia[11] that affects the management outcome.[12]

The aim of this paper is to point out the value of an early TCD ultrasonography[13] in the outcome assessment of severe brain injury.

Material and methods

Between January and December 2004, 30 patients who suffered severe head injury were treated at the Division of Neurosurgery, Osijek University Hospital, Osijek, Croatia. The group consisted of 24 (80%) males and six (20%) females. The mean age was 33.9 years (SD $\pm$ 16.74 years) and the median was 25.5 years, ranging from 18 to 65 years. Traffic accidents were the most common mechanism of injury (80% in

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24 patients), followed by falls in three (10%) cases. Associated injuries were recorded in 17 out of 30 (56.7%) patients in this series. There were eight patients with skeletal bone fractures, two had thoracic injuries and two had injuries of the spine. Five of them (29.4%) sustained multiple life threatening associated injuries.

Thirteen patients (43.3%) underwent early surgery due to mass lesions, i.e. acute intracranial haematoma, while the remaining 17 (56.7%) were treated without surgery. Out of 13 surgical patients, eight underwent unilateral left side craniotomy and five were operated on the right side of the head due to epidural haematoma. Five patients had acute subdural haematoma, where three had intracerebral haematoma. The bone flap was removed in seven out of 13 surgically treated patients.

Regarding the extent of injury, diffuse axonal bihemispheral injury was recorded in 13 out of 17 conservatively treated patients, while in the remaining five, unilateral focal damage up to 3 cm in diameter was noticed. In all patients cerebral lesions were located supratentorially, while in 20 out of 30 post-traumatic subarachnoidal haemorrhage was recorded.

The inclusion criteria were the patients' state of conscience assessed by the Glasgow Coma Scale Score (GCS)[14] of equal or less than eight, along with the patients' age ranging between 18 and 65 years. All patients in whom the TCD bone window was not possible making it unable to obtain TCD measurements, were excluded from the study.

The standard technique of insonating the middle cerebral artery and measuring the mean blood flow velocity by TCD ultrasonic device of 2 MHz was performed in all patients within the first 24 h after hospital admission. The signal was usually taken from the right temporal bone window, except for five patients who underwent unilateral right-side craniotomy. In all patients, initial TCD measurements were taken as early as possible, immediately after the patient was haemodynamically stabilized and the diagnostics was completed. In the group of patients who underwent surgery, initial measurements were always taken preoperatively.

The following parameters were recorded from Doppler measurements:

- normal middle cerebral artery blood velocity ($50-74$ cm/s);
- decreased blood velocity (40 ± 10 cm/s);
- normal PI values ($PI \leq 1$);
- pathological PI values ($PI > 1$).

The pulsatility index[15] was calculated from the difference between end systolic and diastolic MCA velocities divided by the mean MCA velocity. The same team of operators was responsible for all the measurements in every patient.

Intracranial pressure monitoring was performed in the majority of patients (24 out of 30, 80%) following hospital admission and computed tomography (CT) brain scanning. Intracranial pressure was measured continuously every hour per day by external ventriculostomy from the insertion of the ventricular catheter until its removal. Data were collected directly from a bedside monitor using an interfaced computer. The highest number of ICP recordings in a given hour was assigned as an hourly measure of intracranial hypertension. The mean duration of permanent ICP monitoring was 5.9 days ($SD \pm 2.22$ days) and the median was 5.00 days, ranging from 3 to 12 days after admission. In those patients, CPP was individually calculated from the difference between mean arterial blood pressure and ICP.

All patients were admitted to the Intensive Care Unit and mechanically ventilated. Intracranial pressure was maintained below 25 mmHg by moderate hyperventilation ($pCO_2 > 30$ mmHg), intermittent 20% mannitol infusion, and by external ventriculostomy. Cerebral perfusion pressure was maintained above 70 mmHg by intensive fluid resuscitation and the administration of vasopressor agents when necessary. The treatment protocol was based on the intensity to preserve euvolemia, normothermia and euglycemia.

The outcome was assessed at 6-month follow-up after hospital discharge by control clinical examination of the survived patients using the Glasgow Outcome Score (GOS).[16] It was considered favourable when GOS $4-5$ (moderate disability and complete recovery) was recorded and unfavourable when GOS $1-3$ (death, vegetative state and severe disability) was recorded.

Eight patients succumbed to severe brain injury and the early fatality rate was 26.7%. Various complications were recorded in nine out of 22 (40.9%) surviving patients. The most common complication was pneumonia in five cases. There were two cases each of hydrocephalus and deep venous thrombosis.

A statistical data analysis was performed to obtain the results. The correlations between TCD variables and the outcome were investigated by Pearson's coefficient of correlation (r), the χ^2-test and the Fisher exact test when necessary. The Fisher test was used to analyse the data from Table I because there was a low frequency in the sample. The chi-square test was employed for the analysis of the data from Table II to test the interdependence of the investigated variables. The linear correlations between haemodynamic and TCD quantitative variables were calculated by Pearson's coefficient of correlation (r).

Statistical significance of the correlation between the investigated quantitative variables was achieved by the p level. The significance level was set at $p < 0.05$. The multiple regression model was applied to test the independence between TCD and haemodynamic variables.

TABLE I. Division of patients according to normal or pathological MCA velocity and their correlation to outcome

	MCAv 50–74 cm/s	MCAv <50 cm/s	Total
GOS 4–5	15	3	18
GOS 1–3	6	6	12
Total	21	9	30

Fisher exact test: $p = 0.102$.
Normal MCA velocity was recorded in the majority of patients (83.3%) with a satisfactory outcome.

TABLE II. Division of patients according to normal or pathological PI values and their correlation to outcome

	PI ≤ 1	PI > 1	Total
GOS 4–5	15	3	18
GOS 1–3	2	10	12
Total	17	13	30

$\chi^2 = 10.46$; df = 1; $p < 0.01$.
Satisfactory PI values (PI ≤ 1) correlate to a favourable outcome, while the increased PI values PI (>1) predict an unfavourable outcome. The variables are independent. Correlation between the PI and the GOS values is significant.

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

Results

The substantial correlation between TCD parameters and the outcome was established. A positive, statistically significant although weak correlation between flow velocity and the outcome was observed ($r = 0.136$; $p < 0.01$). The relation was generalized by the linear regression equation GOS = 0.929 + 0.041 MCAv, which means that the increase of the middle cerebral artery blood velocity for one unit expectedly increases the GOS value for 0.041 units (Fig. 1).

Low MCA blood flow velocity was correlated to the unfavourable outcome. In six out of nine (66.6%) patients with low MCA velocity, GOS 1–3 was recorded. Normal MCA blood velocities values were recorded in the majority of the patients (in 15 out of 18–83.3%) with the satisfactory outcome. The blood velocity value of 62 cm/s was associated with the favourable outcome, while the MCA velocity of 40 cm/s was connected to the unfavourable outcome (Table I).

When the PI values were correlated to the management outcome, a statistically significant negative, strong correlation was found ($r = -0.722$; $p < 0.01$). The relation was generalized by the linear regression equation GOS = 6.477 – 2.613 PI, which means that the increase of the PI value for one unit expectedly decreases the GOS value for 2.613 units (Fig. 2). Distributions were completely reversed in a way that pathological PI values (PI > 1) were

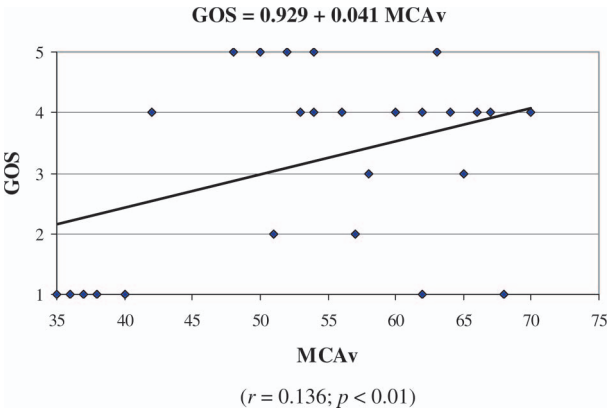

FIG. 1. Correlation between MCA velocity and outcome. Positive, statistically significant, although weak correlation between the MCA blood flow velocity and the outcome ($r = 0.136$; $p < 0.01$). Normal MCA blood velocity values correlate to a satisfactory outcome. Low MCA blood flow velocity correlates to an unfavourable outcome.

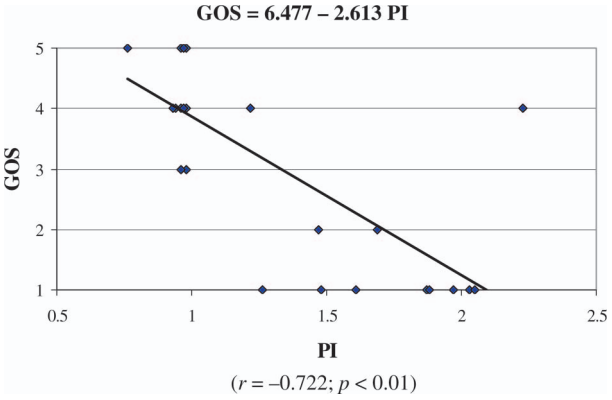

FIG. 2. Correlation between PI values and outcome. Statistically significant negative, strong correlation between the pulsatility index values and the outcome ($r = -0.722$; $p < 0.01$). The increased PI values accompanied by the decreased GOS.

connected to the unfavourable outcome (in 10 out of 13 patients, 76.9%), while normal PI values (PI ≤ 1) were strongly linked to the favourable outcome (in 15 out of 17 patients, 88.2%; Table II).

Thus, normal PI values were predictors of favourable, while pathological PI values were indicators of unfavourable outcomes. The increased PI values were followed by the decreased GOS values in our series, which was statistically significant.

Additionally, the MCA blood flow velocity was correlated to the daily duration of ICP hypertension for the patients who were ICP monitored. Low MCA flow velocity was significantly correlated to the increased number of hours of the elevated ICP in the group of patients with normal MCA velocity (50–74 cm/s; $p = < 0.05$; $r = -0.498$; $n = 17$; Table III). Longer duration of intracranial hypertension immediately after severe traumatic brain injury was followed by a considerable decrease of the MCA velocity in our series. When the multiple regression model was applied, the correlation between ICP and blood flow velocity variables was negative and therefore dependent on the PI variables.

When the same ICP variables were correlated to the PI values, an increased number of hours of elevated ICP significantly correlated to the increased PI in the group of patients with pathological PI values ($PI > 1$; $p < 0.01$; $r = 0.753$; $n = 11$; Table III). Correlation between ICP and the PI variables was independent of blood flow velocity variables on the multiple regression model.

When the multiple regression model was applied, both flow velocity and the PI variables were positively independently correlated to ICP variables (duration of intracranial hypertension; $r = 0.741$; $p < 0.01$).

Discussion

Immediately after traumatic brain injury, cerebral blood flow (CBF) is extremely low and near the ischaemic threshold.[6] Simultaneously, cerebral blood velocity following such an injury is strongly correlated to CBF itself.[17] Accumulating interstitial oedema with increasing mass effect may further compromise CBF and produce a secondary ischaemic insult.[18] Accordingly, an inadequate level of CBF is an important cause of secondary ischaemic brain damage.[19,20] Therefore, early ischaemia after such an injury may be an important factor in determining outcome[6] since, instantly after the injury, patients are more vulnerable to secondary insults. Consequently, no less than 30% of severely brain-injured patients sustain episodes of early ischaemia.[21] According to the results of this study, TCD parameters (the MCA blood flow velocity and the PI) may offer consistent insight into early post-traumatic cerebral haemodynamics, since postinjury, low CBF corresponds to low MCA blood flow velocity and the raised PI values.

However, exceeding thresholds of ICP and CPP may also be harmful to severe brain injury outcome.[22,23] Yet, it seems that the most powerful predictor of neurological worsening after severe brain damage is the presence of intracranial hypertension.[24] Concerning this, the duration of ICP elevation may be as significant in determining the quality of survival as absolute ICP extent.[25] Longer duration of elevated ICP immediately after severe traumatic brain injury was followed by a significant decrease of the MCA velocity in our series (Table III). Therefore, recordings of post-traumatic pathophysiological cerebral haemodynamics by TCD ultrasonography proved to be indispensable in foreseeing the management outcome, provided that it is applied within 24 h after hospital admission of severe brain injured patients.

Considering the results of this study, a major correlation between TCD parameters and outcome was noticed (Tables I and II, Figs. 1 and 2) and a potential prognostic value of TCD parameters to the management outcome was confirmed. Regarding this, we have also concluded that an early insight in post-traumatic pathophysiological cerebral haemodynamics using the TCD ultrasonic device is important in predicting the management outcome of severe head injury.[11]

The essential finding in this study is the fact that cerebral haemodynamic disturbances created soon after severe brain injury are transferred into slower blood flow velocity and higher pulsatility, which could serve as a valuable predictor of the outcome.

Obvious correlation between the MCA velocity and the outcome, as well as statistically significant negative correlation between the PI values and the outcome that was noticed in our series, confirms TCD measuring as a strong indicator of the management outcome.[11,26–30] We have observed that the PI is a particularly strong predictor of the outcome. Use of dimensionless PI has the advantage of reducing measurement errors due to the angle of insonation.[31] Chan *et al.*[19] have also notified the interdependence between low MCA blood flow velocity following severe head injury and bad outcome. Therefore, low MCA velocities and the increased PI values are early and accurate indicators of the unfavourable outcome.

Taking the results into consideration, one should keep in mind that certain restraints of this paper arise from its retrospective nature and a relatively small number of patients in our series.

TABLE III. Correlation between haemodynamic and TCD parameters

	Coefficient of correlation (r)	Risk level p
Duration of intracranial hypertension (ICP > 25 mmHg)		
MCA blood flow velocity – normal values ($n = 17$)	−0.498	0.042
Pulsatility index (PI) – pathological values ($n = 11$)	0.753	0.007

Longer duration of elevated ICP followed by significant decrease of the MCA velocity ($r = -0.489$; $p = 0.042$). Postinjury low CBF and decreased CPP correspond to the raised PI values ($r = 0.753$; $p = 0.007$).

Conclusions

When applied within the first 24 h following hospital admission, TCD ultrasonography is valid as a predictor of the management outcome at 6 months after severe head injury. Therefore, this non-invasive and simple technique could be well engaged in the daily treatment of severely head-injured patients.

Considering our results, it appears that TCD ultrasonography is a reliable early forecaster of the outcome. Therefore, the implementation of TCD monitoring in patients who sustained severe brain injury is well justified. The potential benefit of this study for severely head-injured patients in terms of improving management strategies may be the fact that the prognostic value of this method might influence the management protocol and improve

patient care systems, as well as the treatment of such an injury.

Clearly, additional prospective studies on a larger number of patients are required to maintain these findings.

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