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Is non-invasive monitoring of intracranial pressure waveform analysis possible? Preliminary results of a comparative study of non-invasive vs. invasive intracranial slow-wave waveform analysis monitoring in patients with traumatic brain injury

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A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
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Summary

Background: An increasing body of evidence supports the concept that intracranial pressure (ICP) slow B waves represent the auto-regulatory response of spontaneous fluctuations of cerebral perfusion pressure. A relationship between cerebral auto-regulation and clinical outcome in patients with traumatic brain injury has also been established. The objective of our prospective clinical study was to compare the B slow ICP waves obtained invasively by standard ICP monitoring to those obtained non-invasively using a new ultrasound technology.

Material/Methods: In the participating institutions, over a period of six months, thirteen consecutive patients (8 males and 5 females) with severe closed head injuries (GCS <8) were included in our IRB-approved study. Intracranial pressure and B slow waves, as well as arterial blood pressure and waveforms, were evaluated by standard invasive techniques. Additionally, a new non-invasive ultrasound device, Vittamed (Telematics Scientific Laboratory, Kaunas, Lithuania), was employed for monitoring intracranial blood volume slow waves. Using these modalities, it was possible to compare the changes that occurred with invasive monitoring (Correlation factor RI) and the changes that occurred using non-invasive technology (Correlation factor RN).

Results: Bland Altman plot analysis showed positive correlation between the invasively and non-invasively obtained slow intracranial B waves (2σ=8.9%, $p<0.0001$) and cerebral auto-regulation indexes (RI and RN) (SD=5%, $p<0.0001$). Positive RI and RN values were correlated with poor clinical outcome.

Conclusions: Ultrasonographic technology (Vittamed) may have significant application in non-invasive continuous cerebrovascular auto-regulation monitoring in patients with severe head injuries.

key words: **arterial blood pressure • B waves • cerebral auto-regulation • cerebral perfusion pressure • ultrasound**

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BACKGROUND

It is widely accepted that the slow B waves of intracranial pressure (ICP), as originally described by Lundberg, represent the consequence of slow variations of intracranial blood volume [1]. These waves are characterized as repeatative alterations in the ICP at frequencies of 0.5 to 2 waves per minute [1]. Their duration ranges between 30 and 200 seconds [1]. The quantitative estimation of cerebral auto-regulation (CA) status is usually performed by calculating the correlation coefficient r(ICP; ABP) between the slow B waves of arterial blood pressure (ABP) and ICP [2].

Several recent clinical experimental studies have revealed that CA is a key factor in determining outcome in patients with traumatic brain injury [3,4]. This relationship between CA and outcome was found to be more significant than those previously established between outcome and physiological parameters, such as Glasgow Coma Scale (GCS) Score, mean arterial blood pressure (ABP), mean intracranial pressure (ICP), and mean cerebral perfusion pressure (CPP) [3–6].

Various innovative methodologies for non-invasive monitoring of ICP, CPP and CA in humans can be found in the literature [7–16]. Most of the proposed non-invasive technologies are based on ultrasound application, and have been used to monitor the physiological properties of various biophysical parameters, such as blood flow velocity of intracranial or intraocular vessels or pulsations of the cerebrospinal fluid in the ventricular system [9–11,14,16].

The main priniciple of non-invasive CA monitoring is based on the measurement of ultrasound propagation speed through the brain parenchyma [9,17,18]. Clinical investigation has revealed the similarity between invasively recorded ICP B slow waves and the same slow waves of ABP characterizing the cerebral vascular resistance, which determines intracranial cerebral blood flow and is responsible for CA [19]. The applicability of the non-invasive intracranial blood volume (IBV) slow wave monitoring technique for CA evaluation has been established by experimental studies on animal models [4,17,18].

Non-invasive intracranial blood volume and pressure measurement methodologies have been based on the assumption that idealized relationships exist between ultrasound propagation speed in the cerebral parenchymal acoustic pathway and cerebral blood volume (CBV), cerebrovascular resistance (CVR), CPP, ABP and ICP [9–11,14,16]. These relationships can be explained by the changes in the diameter of cerebral arterioles, which represent the consequence of cerebrovascular auto-regulation.

When auto-regulation is intact, the diameter of cerebral arterioles decreases with any increase of CPP within the linear range of CVR/CPP dependence. The cerebral blood volume also changes as a result of any change in the cerebral arteriole diameter. The results of a mathematical simulation show that the relationship between the ultrasound propagation speed and the cerebral blood volume is linear [4].

The concept of non-invasive monitoring of cerebral blood volume/ICP-CPP slow waves and trends using ultrasound can be summarized as follows:

a) the idea of measuring changes of intracranial blood volume non-invasively is based on the principle of transmitting a broadband ultrasonic signal through the scalp, meninges and brain parenchyma, while monitoring signal parameters, such as the time-of-flight [17,18]. Since the intracranial components (brain parenchyma, cerebrospinal fluid, blood) have different acoustic properties (ultrasound speed, frequency-dependent attenuation), changes of their content in the acoustic pathway directly influence the total acoustic characteristics of intracranial media and the monitored parameters of the ultrasonic signal. Our model is based on the hypothesis that the ultrasonic signal propagates through the 15 cm of human scalp in a straight line, and that the thickness of cranial components is as follows: 13.270 cm of brain parenchyma, 0.865 cm of CSF and 0.865 cm of blood. For evaluating the influence of the over-lying skin, skull and meninges, the thickness of extra-cranial and cranial layers on each side of the head was assumed to be equal to 0.8 cm;
b) the intraventricular or supraventricular parenchymal acoustic pathway crossing the human scalp is used in this case. The acoustic pathway consists of parenchymal tissue, relatively small blood vessels (arterioles, venules and capillaries), and a small amount of cerebrospinal fluid. The parenchymal arterioles determine the cerebrovascular auto-regulation. Blood volume in this pathway is the main factor determining ultrasound propagation speed. The ultrasound attenuation in this pathway depends mainly on the volume of parenchymal tissue;
c) it is known that dynamic CA can be reflected by the existence of latency of the cerebro-vascular auto-regulatory system [2]. Different latency is observed over a frequency range, higher for slow B waves and lower for spontaneous waves over the range of 0.1 Hz to 0.5 Hz. A convenient way to evaluate the latency of the auto-regulatory system is to measure the phase shift between the slow ABP and the slow ICP B waves and to calculate the correlation coefficient r(ICP, ABP) between them. In the case of intact CA, the phase of the slow ICP wave is nearly opposite to the phase of the slow ABP wave of the same frequency, and the value r(ICP, ABP) is close to –1.0 [2]. Thus any increase in the CPP (within physiological limits) is accompanied by an active constriction of the cerebral arterioles. In the case of impaired CA, however, any increase of ABP is accompanied by passive dilatation of cerebral arterioles. This results in the absence of phase shift between the slow ICP and ABP waves and gives positive values of r(ICP; ABP) up to +1.0 [2].

The implementation of the CA monitoring method requires a co-existent invasive monitoring of ICP and ABP slow waves. For the non-invasive implementation of the method, the data of the invasive ICP slow waves could be replaced by non-invasively measured slow waves of the relative ultrasound propagation speed attenuation $\Delta C/C_0$ in brain parenchyma, a fact that also reflects slow variations of the intracranial blood volume. Consequently, the estimation of CA can be performed by calculating the correlation coefficient between ABP and $\Delta C/C_0$ slow B waves. The same ABP slow B waves are used to calculate both the invasive estimate r(ICP; ABP) and the non-invasive estimate r($\Delta C/C_0$; ABP). In this case, r(ICP,ABP) is close to r($\Delta C/C_0$; ABP) when the correlation coefficient r($\Delta C/C_0$; ICP), between non-invasively

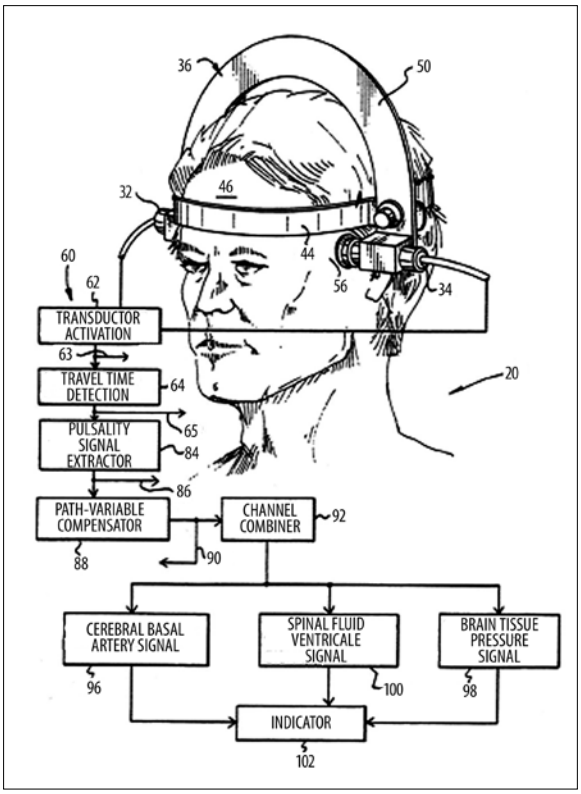

Figure 1. Schematic diagram of the Vittamed device.

recorded parenchymal blood volume slow waves and invasively recorded ICP slow waves is close to +1.0.

Volume changes of intracranial components were set at ±8 ml because such levels of increase of the intracranial volume can cause an increase of ICP up to the critical level of 25 mmHg. This volume change (±8ml) is also typical for slow B waves, the appearance of which might be indicative of possible impairment of cerebrovascular auto-regulation.

The objective of this prospective clinical study was to compare non-invasively obtained data on intracranial pressure slow B waveform analysis and cerebral auto-regulation indexes with the data obtained invasively by employing standard ICP monitoring.

Material and Methods

A new, non-invasive brain physiological monitoring technology (Vittamed) has been developed in the Telematics Scientific Laboratory of the Kaunas University of Technology (Kaunas, Lithuania) (Figure 1). This new technology is based on the non-invasive measurement of acoustic properties of the intracranial media reflected by ultrasound propagation speed and attenuation in the parenchymal acoustic pathway.

The previously described non-invasive ultra-sonic monitoring device (Vittamed) was employed in a neuro-intensive care unit setting in the participating institutions, for non-invasive slow-wave ABP monitoring. A standard invasive ICP monitor (INTEGRA NeuroSciences, NJ, USA) and the invasive ABP monitor (Datex, Helsinki, Finland) were also em-

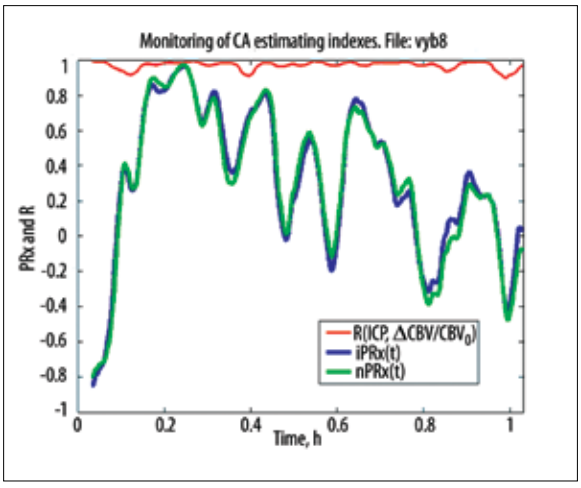

Figure 2. Simultaneous monitoring of cerebral auto-regulation estimating indices for 1-hour monitoring session.

ployed for all of our patients. Simultaneous monitoring of ICP, ABP and $\Delta C/C_0$ was performed, and the recorded data were used for the estimation of slow waves, slow trends, and selected indices [$r(\Delta C/C_0;ABP)$, $r(ICP;ABP)$, and $r(\Delta C/C_0;ICP)$] of the CA status (Figure 2).

Thirteen patients (8 males and 5 females) with severe traumatic brain injury (GCS<8) were simultaneously monitored invasively and non-invasively. The patients' mean age was ranged between 18 and 64 years, with a mean age of 31.2±0.5 years. Fifty-six one-hour sessions of invasive and non-invasive CA monitoring and 87 one-hour sessions of ICP and $\Delta C/C_0$ simultaneous monitoring were performed during this clinical trial.

Results

The time span when the correlation coefficient $r(\Delta C/C_0;ICP)$ exceeded 0.9 was 40% of the total monitoring time (34.8/87hrs). The value of the correlation coefficient $r(\Delta C/C_0;ICP)$ was above 0.6 during 80% (69.6/87 hrs) of the total monitoring time.

Invasive and non-invasive monitoring of slow waves was comparable in 80 of the total 87 hrs of monitoring time (91.9%), while CA showed agreement between invasive and non-invasive results – $r(\Delta C/C_0;ABP) \sim r(ICP;ABP)$ – in 78/87 hrs (89.6%) of monitoring time. The mean amplitude of CPP slow waves was above 3 mmHg during all these monitoring sessions. The correlation coefficient $r(\Delta C/C_0;ICP)$ was close to +1.0 when the state of CA was changing between intact ($r<0$) and impaired ($r>0$) (Figure 3A,B). The Bland Altman plot of invasively and non-invasively measured slow waves and the distribution of differences between these waves revealed strong correlation between invasively and non-invasively measured diagnostic data, with clinically insignificant negligible dispersion.

Statistical analysis

Statistical analysis was employed to assess the similarity between the invasively monitored ICP B slow-waves and the non-invasively monitored $\Delta C/C_0$ slow waves. Two types of

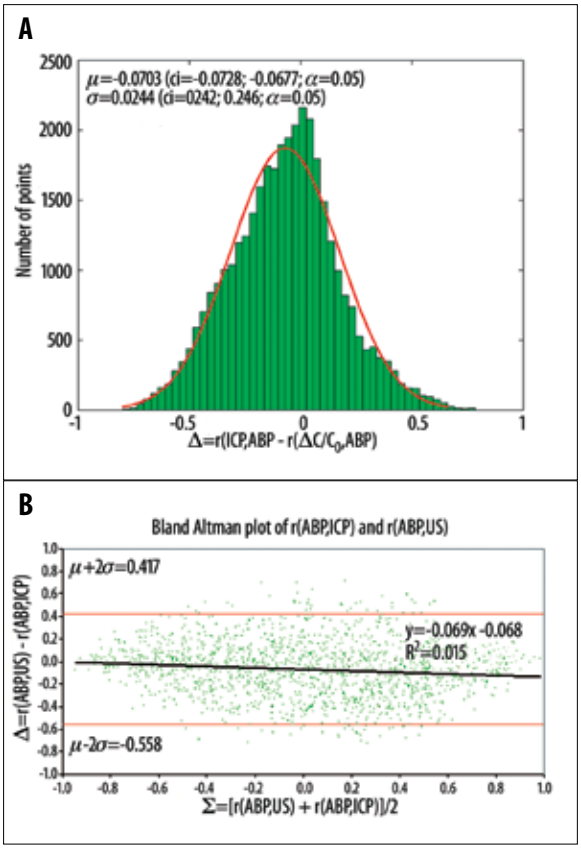

Figure 3. Estimating the similarity between the normalized slow-waves of invasive ICP and non-invasive $\Delta C/C_0$. (**A**) Distribution of differences between the non-invasive and invasive data; (**B**) Bland Altman plot of non-invasive and invasive data.

data sets were evaluated for each session of monitoring. The first type of data sets, which included all measurement points, were checked by calculating the standard deviation and the mean of differences between the normalized invasive and non-invasive data and the correlation coefficient between these data. The second type of data sets, including randomly selected data points, were statistically evaluated by performing a paired *t*-test. The values of *t*-criterion corresponding to each data set were calculated using normalized data. Normalization was performed by dividing the original measurement data by standard deviation. The calculated values of *t*-criterion were checked by the following formula:

$$|t|<T_{crit}(\alpha=0.05,\ n=50\ldots70), \qquad (1)$$

where:

- *t* is the calculated value of *t*-criterion of paired samples;
- *Tcrit* is the critical value of *t*-statistics;
- α is the significance level of the test;
- n is the number of randomly selected points.

The investigated number of samples was N=87. The obtained proportion of samples with positive results in terms of the *t*-criterion was higher than the proportion of interest (p=0.95), which confirms the hypothesis for the coincidence of invasively and non-invasively measured slow waves:

$$\pi_{N+}=N_+/N=0.954. \qquad (2)$$

It was also determined that the coefficient $r(\Delta C/C_0; ICP)$ exceeded 0.9 when the amplitude of the CPP B waves was above 3 mmHg. Therefore, it would be useful to implement CPP slow wave amplitude measurements to assess the measurement reliability of non-invasive slow waves.

Discussion

The extensive use of invasive ICP monitoring in modern neurosurgery has been well justified in the literature [20–23]. Its importance in the management of patients, mostly but not exclusively those with severe head injuries, is widely accepted [20–27]. The accurate knowledge of the ICP and CPP is important in predicting the overall outcome of these patients [28,29].

The Vittamed device has been tested in large animal experimental studies, as well as in a previous clinical study performed in our institution [17,18]. These studies showed a significant correlation between invasively and non-invasively measured ICP [17,18].

The complications related to the application of invasive ICP monitoring devices, though rare, render the idea of employing non-invasive ultrasonographic technology very appealing [18,26,30–32]. The Vittamed device is definitely advantageous compared to the existent ICP monitoring methods, since no intervention is necessary. The patients, particularly children, tolerate the application of the monitoring device significantly better, since no skin penetration is necessary for applying the Vittamed helmet and there is no discomfort associated with the monitoring process [18]. Invasive ICP monitoring has been associated with infection, which has been reported in different series at a frequency up to 22%, depending on the nature of the monitoring device, the insertion technique, the patients' underlying pathology, amd the duration of monitoring [33], and with intra-cranial hemorrhage (which has been reported in only 1–2% of the cases but with unfortunately devastating results) [34,35]. These risks do not arise in connection with the use of the Vittamed device, since no intervention is necessary [18,32,36]. The monitored patients do not need to be placed in the intensive care unit for monitoring, a fact which dramatically minimizes the cost of the monitoring. Additionally, the non-invasive character of the ultrasonic device used makes to possible to monitor ICP, CPP, CBV and CSF volume even in patients with clotting abnormalities, a clinical situation not at all infrequent in patients with traumatic brain injuries. Moreover, in patients for whom invasive ICP monitoring has to be discontinued either due to increased risk of infection, prolonged monitoring, or patient's temporary improvement and a newly observed deterioration, the Vittamed technology gives the opportunity for prolonged monitoring.

Although the concept that ICP monitoring maximizes the patient's outcome is gaining popularity, it has remained controversial even so. Indeed, the exclusive monitoring of ICP misses important neurophysiological events and ignores several other physiological parameters that have been implicated in the pathophysiological mechanisms occuring during and after a traumatic brain injury. It is widely accepted that impaired cerebral auto-regulation is only indirectly reflected in ICP al-

terations. Importantly, the Vittamed technology appears to have the ability to offer major insights into such neurophysiological changes. The Vittamed device allows the neurosurgeon to monitor and record easily real time multi-parameter information, such as variation of CBF, CA, CVB and CSF. The accurate knowledge of the cerebral blood and cerebrospinal fluid volumes is valuable direct data indicating the presence or absence of edema and brain parenchyma compliance. This significant information could dramatically alter the management of patients with intracranial hypertension.

The customary invasive methods provide information about the patient's brain metabolic activity, such as regional measurements of the pH, pO2, pCO2 and intra-cranial temperature, which are definitely irreplaceable, particularly in critically ill patients. In these circumstances, however, the Vittamed device can reasonably act as a screening non-invasive method for determining the necessity to implement such invasive, traditional monitoring.

Unfortunately, the invasive character of the customary ICP monitoring modalities and its associated complications limit the use of such methods only to patients with severe intracranial pathology. Our non-invasive method minimizes these limitations and could possibly expand the application of ICP, CPP, CBV and CA monitoring in the evaluation of patients with hydrocephalus, pseudotumor cerebri, minor head injuries, spinal cord injuries and chronic headaches. A multi-institutional, prospective clinical study is currently underway to collect data from use of the Vittamed monitoring device in patients with traumatic brain injury, and another study in patients with spinal canal compromise of traumatic etiology.

CONCLUSIONS

The non-invasive monitoring of ABP and cerebral blood volume slow wave, as well as CA status, using a Vittamed ultrasonographic device, is a safe and promising technology, which could in some cases replace – and in others, enhance, and possibly expand – the use of the existent invasive technology in the evaluation of several different intracranial pathological entities. The implementation of non-invasive human cerebrovascular auto-regulation monitoring is possible by measuring non-invasively slow $\Delta C/C_0$ waves (instead of ICP) and by calculating the correlation coefficient between the slow waves of $\Delta C/C_0$ and ABP. The non-invasive character of this modality becomes more important when the method is applied to pediatric patients. Further multi-institutional prospective clinical studies are necessary to validate the accuracy of this advanced technology in estimating cerebral auto-regulation.

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