

Use of Near Infrared Transillumination / Back Scattering Sounding (NIR-T/BSS) to assess effects of elevated intracranial pressure on width of subarachnoid space and cerebrovascular pulsation in animals

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The objective was to assess changes in the width of the subarachnoid space (SAS) and amplitude of cerebrovascular pulsation (CVP) during acute elevation of intracranial pressure (ICP) using Near Infrared Transillumination/Back Scattering Sounding (NIR-T/BSS). Changes in the width of the SAS and amplitude of CVP were observed in rabbits during experimental ICP elevation induced by: (1) quick injections of saline into the subdural space of the spinal cord, and (2) distension of a surgical catheter balloon placed intracranially in the subdural space. The amplitude of CVP was also assessed during acute elevation of blood pressure in the intracranial portion of the internal carotid artery (ICA) induced by adrenaline. Each of the injections of saline caused a transient rise in the width of the SAS and amplitude of CVP. The amplitude of the increase in CVP was dependent on changes in blood pressure in the ICA ($r=-0.82$, $P<0.01$). Distension of the intracranial balloon resulted in elimination of the respiratory oscillations in the CVP and increased its systolic–diastolic amplitude. An increase in the amplitude of CVP was evoked by adrenaline without an increase in the carotid blood flow. We demonstrated that during elevation of ICP the amplitude of CVP depends on blood pressure rather than on blood flow in large cerebral vessels. Elimination of the respiratory oscillations by a minute (“sub-critical”) ICP increase may be used as an early indicator of rising ICP. The direction of changes recorded using NIR-T/BSS was consistent with changes recorded using tensometric transducers.

Key words: cerebrovascular pulsation, infrared radiation, intracranial pressure monitoring, NIR-T/BSS, subarachnoid space

INTRODUCTION

Raised intracranial pressure (ICP) develops in 50–75% of patients with severe head trauma (Chan et al. 1992). A parallel rise in the mortality indices with the increase in magnitude of ICP elevation has been observed (Czosnyka and Pickard 2004). Early detection of ICP elevation could prevent irreversible changes in the function and structure of the brain.

Unfortunately, from the diagnostic point of view, the modest increases of ICP are almost always asymptomatic. Symptoms become visible in the later phase when ICP rise more quickly (Langfitt et al. 1965). The lack of clinical symptoms in the early phase means that detection of ICP elevation is not an easy task and in many cases is done with a certain delay. Of importance is the fact that despite continuous improvements, ICP monitoring is still an invasive procedure. Therefore, a non-invasive technique for monitoring of changes in the width of the subarachnoid space (SAS) and cerebrovascular pulsation (CVP), would allow for the initial assessment of increasing ICP, and support moni-

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toring of the efficacy of treatment over an extended period of time (Frydrychowski and Plucinski 2007).

Cerebral perfusion pressure (CPP) maintenance in patients with traumatic brain injury remains a matter of controversy (Coles et al. 2004, White and Venkatesh 2008). The infusion of vasopressors may result in beneficial changes in regional cerebral blood flow (CBF) without improving the early neurological outcome (Kroppenstedt et al. 2002). Furthermore, the optimal CPP threshold required to start therapy needs to be established (Steiner et al. 2002, Howells et al. 2005). The optimal CPP may vary from patient to patient and depends on age, ICP, plasma oncotic pressure and cerebrovascular resistance (Coles et al. 2004). Cerebrovascular resistance, in turn, depends on autoregulatory mechanisms that significantly alter the elastic properties of the cerebral arteries, therefore changing their compliance and the characteristics of CVP (Frydrychowski et al. 2002a). Further understanding of interdependencies between CPP, CBF and CVP may help to find individual thresholds of vascular reactivity and improve management of the traumatized human brain (Czosnyka et al. 2002).

In our previous papers we presented the theoretical analysis of the feasibility of Near Infrared Transillumination/Back Scattering Sounding (NIR-T/BSS) for the assessment of changes in the width of the SAS and amplitude of CVP (Plucinski et al. 2000, Plucinski and Frydrychowski 2007), and the results of our experiments on a mechanical-optical model (Frydrychowski et al. 2001a,b, 2002a,b,c, 2007). In the experimental design described in this paper, we focused on the effects of minute (“sub-critical”) and significant elevations of ICP, as well as the acute increase in blood pressure in the internal carotid artery (ICA) on the NIR-T/BSS tracings in an animal model. The aim of the study was to assess changes in the width of the SAS and amplitude of CVP during acute elevation of ICP *in vivo* using NIR-T/BSS method.

METHODS

The experiments were performed on 23 crossbred male rabbits, with a body weight ranging from 3.0 to 4.0 kg. The animals were anaesthetized with urethane and α -chloralose (2.5 g urethane was mixed with 0.5 g α -chloralose and dissolved in 25 ml saline). α -chloralose was used because of limited effect on the autonomic system or autonomic reflexes (MacGowan et al. 2005). The

mixture was administered intravenously to the marginal auricular vein at the dose of 2.5 ml kg⁻¹ body weight. Heparin was given as an i.v. injection (500 IU×kg⁻¹ body weight). The study protocol was approved by the ethical committee of the Medical University of Gdansk.

Tracheotomy was performed and a breath sensor was mounted in the tracheal tube for continuous monitoring of respiration. The sensor was connected to a respirotachometer. The external carotid arteries (ECA) were ligated in all animals. Two catheters were inserted into the right common carotid artery (CCA): one in the headward direction used for measurement of blood pressure in the cerebral arteries and the other directed upstream towards the heart for measurement of blood pressure in the systemic circulation. As the ECAs were occluded, the pressure measured in the intracranial portion of the right ICA was a directly proportional estimate of that in the circle of Willis. The same catheter was used for the intraarterial administration of adrenaline. An electromagnetic flow transducer was placed on the left CCA, which, due to ECA occlusion, measured real flow in the left ICA. The flow transducer (type C 2.5 mm) was connected to a Narcomatic electromagnetic flow meter (Hugo Sachs Elektronik – Harvard Apparatus GmbH, March-Hugstetten, Germany). Prior to NIR-T/BSS recording performed with the sensor unit placed on the shaved scalp, a laminectomy was performed in the lumbo-sacral region and two catheters were introduced into the subdural space of the spinal cord for monitoring of the ICP and administration of quick injections of saline (1 mL bolus). All pressures were monitored using Statham P23 ID tensometric transducers (Gould Statham Instruments; Oxnard, CA, US). In those animals in which NIR-T/BSS monitoring was to be carried out directly through the skull bones (with the scalp removed), the frontal bones were revealed via a sagittal incision, and the IR sensor unit was placed on their surface. In each animal an ECG was recorded throughout the experiment (2nd bipolar lead, needle electrodes). Simultaneous recording of all collected signals was carried out using a multichannel Mingograf 82 recorder (Siemens, Munich, Germany). Selected variables were also recorded using an IBM PC-compatible microcomputer and an analogue–digital converter.

After preparation procedures, animals were left until blood pressure, heart rate and respiration stabilised. In all experiments the body temperature of the animals was maintained constant with an electric heat-



ing cushion. Experiments were commenced not earlier than 60 minutes after completion of the initial preparation procedures. The animals were divided into 3 groups. In each group only one experimental procedure was performed. To facilitate reading each of experimental procedures: (1) distensions of the intracranial balloon with 0.3 mL of saline ($n=6$), (2) administration of adrenaline ($0.5 \mu\text{g} \times \text{kg}^{-1}$) into the right internal carotid artery ($n=8$), (3) infusions of 1.0 mL of saline into the subdural space of the spinal cord ($n=9$) is described in the results section.

For NIR-T/BSS recording directly through the bones of the skull, a simplified version of the IR sensor unit was used, consisting of: a) an emitter (E), i.e. infrared light-emitting diode (LED), and b) a receptive element (proximal sensor, PS) located away from the emitter. Recording of NIR-T/BSS signals through the intact scalp requires the use of a different technique for measurement and a more complicated version of the IR sensor unit, because of the strong modulation of the signals received by the pulsatile blood flow in the arteries of the skin. Therefore, for NIR-T/BSS recording through the intact scalp, different IR sensor construction as well as data acquisition and processing methods were used. The IR sensor unit consisted of: (a) an emitter (E), i.e. infrared light-emitting diode (LED; SFH-484, Siemens, Munich, Germany), (b) a proximal sensor (PS), and (c) a distal sensor (DS) diode. The IR sensors utilized BPW-34 photodiodes (Siemens, Munich, Germany). A pulsatile stream of infrared radiation generated by the emitter propagates across the scalp, skull bones and a layer of cerebrospinal fluid (CSF) in the SAS. The energy of radiation propagated to the adjacent deeper layer decreases at each level as a result of scattering, absorption, reflection and longitudinal propagation in a given layer. A large portion of the radiation emitted is propagated in the superficial layers of the head (skin, bone) and reaches the PS. A relatively small amount of energy is carried by the deep IR stream, which manages to reach the surface of the brain and travels within the SAS as in an optical waveguide (Plucinski et al. 2000, Plucinski and Frydrychowski 2007, Frydrychowski et al. 2002a, Frydrychowski and Plucinski 2007). This deep stream propagates easily in the CSF with only minor reductions. A certain portion of the radiation reaches the DS, crossing the aforementioned tissue layers in the reverse sequence. Electric signals generated by the photosensors (PS and DS) undergo analogue–digital conver-

sion in a special data acquisition module, are recorded by an IBM PC-compatible microcomputer running dedicated software and stored on the computer hard disk for further analysis.

The opposite effects of changes in the width of the SAS on the magnitude of the PS and DS signals are used in the signal processing procedure to assess the direction and magnitude of these changes. The key concept in the signal processing method consists of reduction of the proportional factors, which affect both signals in the same manner, through division of the DS signal by that of the PS. The quotient of the two signals will herein be referred to as the Transillumination Quotient (TQ). Modulation of the PS and DS signals, resulting from pulsation of the cutaneous arteries, exerts an identical effect on both signals, and therefore is reduced to a constant in the process of division. An increase in the width of the SAS magnifies the DS signal and does not influence that of the PS, which causes an increase in the TQ. A decrease in the width of the SAS, on the other hand, diminishes the DS signal and does not influence that of the PS, which results in a decrease in the TQ. The TQ is not affected by pulsation of the cutaneous arteries, and therefore allows for non-invasive monitoring of the width of the SAS.

The deep IR stream propagating in the SAS is modified by a slight arterial modulation, which does not affect the more superficial IR streams. As the total IR stream reaching the PS is composed almost exclusively of the radiation propagated in the skin and bone, with the deep stream constituting a minute part of it, CVP exerts almost no influence on the PS signal. Conversely, as the DS signal depends on the deep IR stream propagated in the SAS, it is strongly affected by amplitude–frequency modulation, whose source is the CVP. That is why the TQ also exhibits this modulation, visible as long- and short-period oscillations around a certain base level. The mean level at which the oscillations occur depends on the mean width of the SAS over a period of time, while the long-period oscillations result from the respiratory and other low-frequency physiological cycles, and the short-period oscillations are the consequence of CVP transmitted to the whole of the brain from the cerebral arteries. The amplitude of this CVP is dependent on the amplitude of pulsation of the intracranial arteries, while its frequency is equal to the heart rate (HR).



Thus, in the transillumination quotient (TQ) in mode B, as in the individual signal recorded in mode A, we can identify three main components: (1) constant or non-pulsatile component – further referred to as the sas component (sas-TQ) – its value depending on the width of the CSF-filled SAS, (2) slow-variable pulsation, further referred to as the subcardiac component (scc-TQ) – with a frequency significantly lower than the HR frequency, (3) fast-variable pulsation, further referred to as the cardiac component (cc-TQ) – resulting from heart-generated arterial pulsation which is the cause of fast oscillations in the width of the SAS.

In mode B, for further analysis, the first harmonic of the arterial pulsation-dependent oscillations of TQ is extracted through appropriate filtering, along with its modulation. Modulation of that harmonic is a compound amplitude-frequency one. In the tracings of signals recorded in NIR-T/BSS, this harmonic with its modulation is shown as the fast-variable component (cc-TQ), also referred to as the “cardiac component” of TQ. Also presented in the results is the constant component (sas-TQ). A detailed description of the method of signal analysis is presented in other papers (Plucinski et al. 2000, Plucinski and Frydrychowski 2007, Frydrychowski et al. 2002a, Frydrychowski and Plucinski 2007).

For mode B experiments *W*-Shapiro-Wilk, *U*-Mann-Whitney and ANOVA tests were used to analyse the differences between average values. Changes in ICP, blood pressure, sas-TQ and cc-TQ responses were compared versus baseline values. Correlation and regression analysis was performed to assess the interdependences between ICP, blood pressure, sas-TQ and cc-TQ. All statistical calculations were performed using the Statistica for Windows 6.0 commercial package.

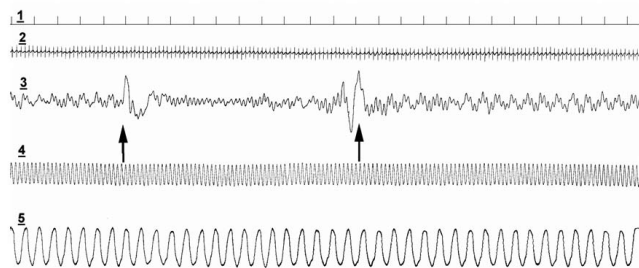


Fig. 1. Effect of minute distention (0.3 mL) of the intracranial balloon on the recorded variables: (1) time marker (interval 1 second), (2) ECG, (3) pulsatile component of the sensor signal (NIR-T/BSS), (4) systemic arterial blood pressure (mm Hg), (5) respiration (thermosensor). Vertical arrows indicate distention and release of the intracranial balloon.

RESULTS

Mode A – NIR-T/BSS recording with the IR sensor unit placed directly on the frontal bone

In the first series of experiments, a minute increase in ICP was achieved through volume additions by inflating and deflating an intracranial balloon. Distension of the intracranial balloon was achieved with an infusion of 0.3 mL of saline. Procedure was performed in each rabbit ($n=6$) twice with 10 seconds interval. Noteworthy is the cessation of respiratory oscillations of the pulsatile component (scc-TQ) of the sensor signal as the only change in NIR-T/BSS tracings. Sub-critical distension of the intracranial balloon did not cause any changes in the respiratory pattern and systemic arterial pressure, but resulted in elimination of the respiratory oscillations of the pulsatile component of the sensor signal and increased its systolic–diastolic amplitude in all six animals. Figure 1 is representative of the results obtained from all six experiments.

Changes in the amplitude of the NIR-T/BSS signal during and after acute elevation of systemic arterial pressure were assessed via intra-arterial administration of adrenaline ($0.5 \mu\text{g} \times \text{kg}^{-1}$) into the right internal carotid artery. This procedure was performed only once in each of rabbits from the experimental group ($n=8$). The results are presented in Figures 2 and 3 and are representative of all eight experiments. After 15–20 heartbeats (approx. 3 seconds), adrenaline caused an elevation in the systemic arterial pressure. Equally

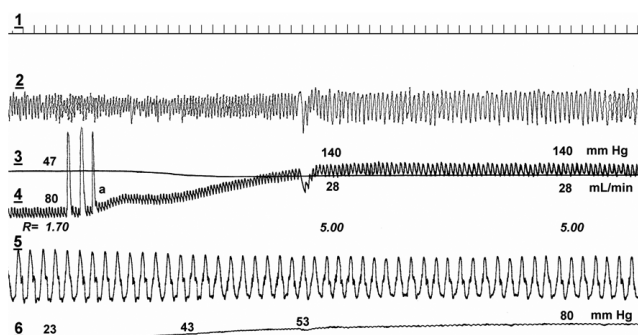


Fig. 2. Influence of intraarterial injection of adrenaline on the recorded variables (ascending phase): (1) time base, (2) pulsatile component of the sensor signal (NIR-T/BSS), (3) blood flow in the left common carotid artery (mL/min), (4) mean systemic arterial pressure (mm Hg), (5) respiration, (6) blood pressure in centripetal portion of the right common carotid artery (mm Hg).



prompt was the decrease in amplitude of the NIR-T/BSS signal observed synchronously with the rise in systemic arterial pressure and the distinct decrease in blood flow in the left ICA. After a short (approx. 4 seconds) plateau in the systemic arterial pressure, a further marked rise in this pressure was observed. This rise in systemic arterial pressure restored the amplitude of the NIR-T/BSS signal to the initial level, despite the cerebral vasoconstriction induced by adrenaline, as demonstrated by the low carotid flow. The rise in systemic pressure evoked a baroreflex, whose effect was seen as a deceleration of the heart rate. A decrease in the tidal volume was also observed. The systemic arterial pressure remained elevated with concomitant bradycardia for about 30 seconds. An increase in pulse pressure is clearly visible in tracing 4 throughout that period. Noteworthy was a marked rise in the amplitude of the NIR-T/BSS signal accompanying this increase in pulse pressure, until the beginning of the descending phase of the arterial pressure. A decrease in arterial pressure from 140 to 95 mm Hg (Fig. 3, tracing 4) was accompanied by a decrease in pulse pressure and in a parallel decrease in the amplitude of NIR-T/BSS pulsation. This can be explained by a decrease in the force stretching the arterial walls with continuing cerebral vasoconstriction. As systemic arterial pressure assumed the baseline value of 80 mm Hg the pulse pressure increased. A progressive rise in pulse pressure was reflected in a parallel increase in the amplitude of pulsation in the NIR-T/BSS signal.

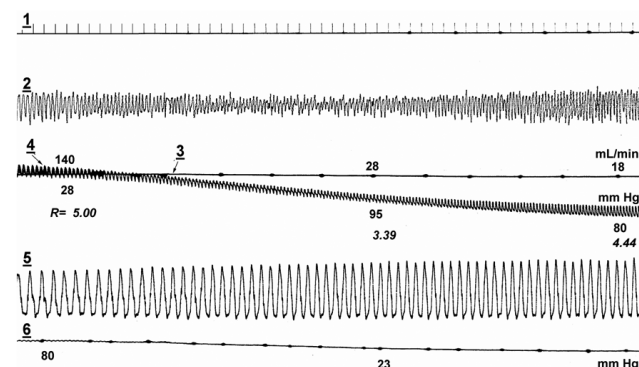


Fig. 3. Influence of intraarterial injection of adrenaline on the recorded variables (descending phase): (1) time base, (2) pulsatile component of the sensor signal (NIR-T/BSS), (3) blood flow in the left common carotid artery (mL/min), (4) mean systemic arterial pressure (mm Hg), (5) respiration, (6) blood pressure in centripetal portion of the right common carotid artery (mm Hg).

Mode B – NIRT recording with the IR sensor unit placed on the surface of the smooth-shaved intact scalp in the frontal region

Use of the NIR-T/BSS method in human clinical practice would obviously require the method to be non-invasive. Therefore we performed a series of experiments in the last group of rabbits ($n=9$) with the IR sensor unit placed on the surface of a smooth-shaved intact scalp. Figure 4 is representative of all nine experiments. Six subsequent infusions of 1.0 mL of saline were injected into the subdural space of the spinal cord of each animal. Each of the injections of saline caused a marked, transient rise in the width of the SAS. Due to the short intervals between subsequent injections (less than two minutes), the width of the SAS did not assume the initial value in between the injections, so a rising trend in the width of the SAS was observed (Fig. 4,

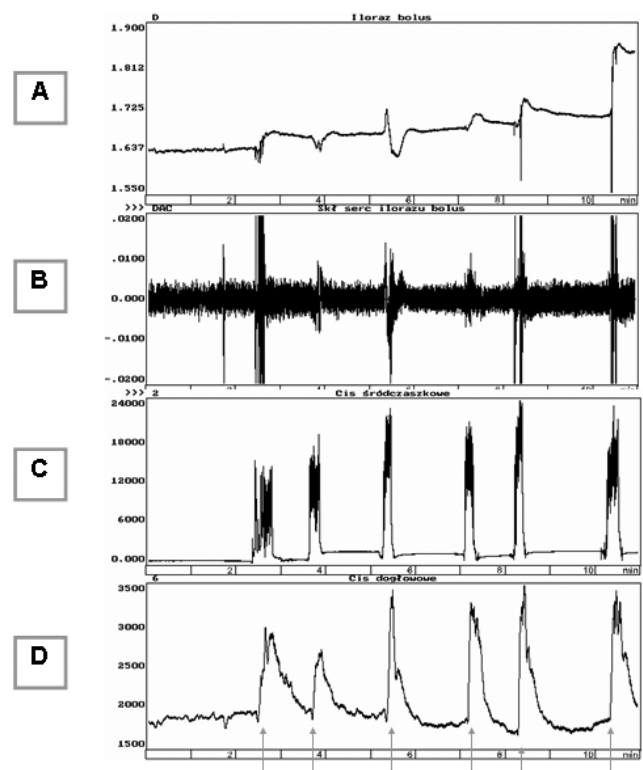


Fig. 4. Effects of single boluses of saline into the subdural space of the spinal cord in the lumbar region on the recorded variables: (A) sas-TQ (NIR-T/BSS): an elevation of the tracing indicates an increase in the width of the SAS, (B) cc-TQ (NIR-T/BSS): an increase in the amplitude of cc-TQ indicates an increase in the amplitude of pulsation of the cerebral resistant arteries, (C) ICP (mm Hg), (D) pressure in intracranial portion of the ICA (mm Hg). Boluses are indicated with vertical arrows.

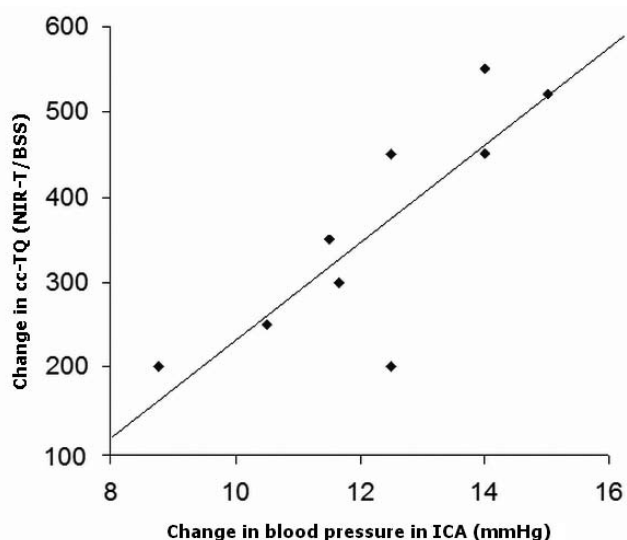


Fig. 5. Linear correlation analysis of changes in blood pressure in the ICA and cc-TQ (the amplitude of CVP). All values of changes in blood pressure in ICA and cc-TQ obtained from 9 rabbits were included in the analysis of linear correlation (Spearman $r=0.82$, $P<0.01$).

tracing A, peaks at the climbing baseline level). Large increases in ICP in response to each of the boluses are also clearly visible (Fig. 4, tracing C). These changes were accompanied by marked increases in the amplitude of cc-TQ (Fig. 4, tracing B), i.e. amplitude of CVP, and also transient rises in the pressure in the ICA (Fig. 4, tracing D). Mean \pm SE values describing the changes evoked by the first boluses of saline are given in Table I. Correlation and regression analysis revealed that increases in cc-TQ (the amplitude of CVP) after

injection of the first boluses were dependent on changes in blood pressure in the ICA (Fig. 5). Subsequent boluses were not analysed to avoid bias related to different baseline values.

DISCUSSION

The results of experiments presented in this study clearly show that elevation of ICP affects both pulsatile (cc- and scc-TQ) and non-pulsatile (sas-TQ) components of the NIR-T/BSS method. Here, we wish to underline the difference between changes in the sas-TQ component related to changes in the width of the SAS in mode A (bone, emitter and one IR sensor) and mode B (intact scalp, emitter and two IR sensors). In mode A, the non-pulsatile component (sas-TQ) of the sensor signal decreases with the increase in the width of the SAS, and increases with narrowing of that space. In mode B, sas-TQ increases with the increase in the width of the SAS, and decreases with the width of that space, assuming lower values.

Small volume additions by inflating and deflating a balloon inserted within the cerebrospinal space can be used to assess the volume pressure response, and predict brain compliance (Piper et al. 1999, Yau et al. 2002). Absence of changes in systemic blood pressure and respiration in response to balloon distension may indicate that the elevation of ICP in that procedure was minute ("sub-critical"). Thus, detected by NIR-T/BSS suppression of respiratory oscillation potentially constitutes an indicator of very early rises in ICP. Injections

Table I

Changes evoked by the first boluses of saline (1.0 mL) injected into the subdural space of the spinal cord (mean \pm SE values)				
	Number	Baseline	Bolus (1.0 mL)	Change of mean
TQ (NIR-T/BSS)	9	1643.54 \pm 63.11	1722.54 \pm 87.56	79.33* \pm 13.04
cc-TQ (NIR-T/BSS)	9	117.23 \pm 19.72	480.56 \pm 61.93	363.33* \pm 44.85
ICP (mmHg)	9	5.3 \pm 1.13	24.36 \pm 1.23	19.06* \pm 0.61
ICA (mmHg)	9	103.34 \pm 5.7	115.61 \pm 6.3	12.27* \pm 0.65

* $P<0.01$ change versus baseline value



of 1.0 mL saline into the subdural space of the spinal cord resulted in transient, abrupt and very high increases in the ICP (Fig. 4, tracing C), reflecting sudden extracerebral CSF volume gains. These simple experiments demonstrated that an increase in the CSF compartment, i.e. the “optical channel” or propagation duct for the IR stream, leads to an increase in sas-TQ (Fig. 4, tracing A). Although increases in the width of SAS (sas-TQ) were fully consistent and synchronous with ICP increases (Fig. 4, Table I), correlation and regression analysis did not reveal any direct interdependencies. This may reflect the fact that NIR-T/BSS monitors only changes in the width of the SAS, not the volume gain in the whole CSF compartment. Experiments in subjects suffering from traumatic brain injury confirmed the high sensitivity of sas-TQ measurements during ICP changes (Frydrychowski and Plucinski 2007) or brain oedema. Further studies are warranted to clarify the relationship between ICP, sas-TQ, cc-TQ and respiratory oscillations.

Rises in ICP were accompanied by increases in blood pressure in the intracranial vessels (Fig. 4, tracing D). These, in turn, were accompanied by synchronous rises in the amplitude of cc-TQ (Fig. 4, tracing B; Table I). We found that changes in the pulsation of resistant arteries (cc-TQ) were directly dependent on changes in blood pressure in ICA (Fig. 5). A crucial observation made during the experiment with adrenaline was that the amplitude of CVP increased when there was a rise in the systemic arterial pressure without an increase in carotid blood flow, or even when the carotid flow decreased (Fig. 2). In our previous experiments we proved that the amplitude of CVP increased when there was a rise in the carotid blood flow at constant systemic arterial pressure (Frydrychowski et al. 2002a). The presented results provide further evidence that the relationship between blood flow in large brain vessels and the pulsatile blood volume in small resistant arteries is highly variable and non-linear. The equation becomes even more complex during ICP elevation due to direct interactions between the cerebral vasculature and CSF space (Haubrich et al. 2007, Carrera et al. 2010). To date the pressure–flow velocity relationship in the human cerebral circulation has been determined from arterial blood pressure and non-invasive monitoring of blood flow in large cerebral vessels (Aaslid et al. 2003). However, the cerebral circulation is extremely complex (Panerai 2009), non-linear (Panerai et al. 2006) and after brain injury, is asym-

metric (Aaslid et al. 2007). NIR-T/BSS is the only method that enables continuous, non-invasive monitoring of the amplitude of CVP, which reflects the state of small resistant arteries.

Regardless of the recording mode (A or B), the power of the IR stream received by all IR sensors depends on the following factors: the thickness and structure of the skull bones, the optical density of the CSF and the width of the SAS. In mode B, the thickness and density of the scalp are also important. In these experiments, the only variable parameter, which affects the power of the IR stream reaching the distal sensor, was the width of the SAS. Within and between subject high reproducibility and repeatability of NIR-T/BSS measurements were demonstrated earlier (Frydrychowski et al. 2001a, 2002a). NIR-T/BSS, alike NIRS allows for direct within subject comparisons (Frydrychowski et al. 2002a, Wagner et al. 2003). Presented NIR-T/BSS tracings make possible quantitative comparisons before and after each experimental procedure. The exact values of NIR-T/BSS signals (sas-TQ and cc-TQ) can be read from each tracing. Directions and magnitudes of changes are consistent across different procedures, can be easily explained basing on current physiological and/or pathophysiological knowledge and were validated with routinely used tensometric recordings. So far the measurements with the use of IR light (NIRS and NIR-T/BSS) does not allow for direct between subjects or animals comparisons due to differences in skull bones parameters (Frydrychowski et al. 2002a, Wagner et al. 2003).

Changes in arterial CO₂ tension (PaCO₂) markedly alter CBF in all known mammalian species. We did not measure PaCO₂ in this study, so we are not able to assess the potential impact of changing PaCO₂ on the recorded responses. Nevertheless we observed high between animals reproducibility of results, thus we can make an assumption that even if the results were influenced by altered PaCO₂ all rabbits were affected in a similar way. Furthermore we investigated the effects of acute ICP changes. Increased PaCO₂ shifts but does not change the direction of responses evoked by ICP increase (Ursino et al. 2000). Further studies are planned to define the precise relationship between PaCO₂, cc-TQ and sas-TQ. The ICP was measured in lumbo-sacral region by means of two catheters introduced into the subdural space of the spinal cord for monitoring of the CSF pressure, and administration of injections of saline (bolus). Although we recognise that



the ICP transmitted to lumbo-sacral space may suffer various influence, we do believe that our recordings closely reflected the ICP on subdural brain space (Martins et al. 1972). The experiments were carried out in rabbits, whose skull bones were relatively thin. A new and improved numerical/mathematical model for use on human subjects has been proposed (Plucinski and Frydrychowski 2007) and verified (Frydrychowski and Plucinski 2007).

CONCLUSIONS

We have demonstrated that the amplitude of CVP does not depend directly on blood flow in large vessels. On the contrary pulsation of pial arteries may increase during significant elevation of blood pressure, or ICP, when there is no change or even a decrease in blood flow in the ICA. We have shown that during acute elevation of ICP changes in the amplitude of CVP are directly related to the changes in blood pressure in large cerebral vessels. Furthermore we have indicated that minute (“sub-critical”) ICP elevation affects NIR-T/BSS respiratory oscillations. Suppression of respiratory oscillation potentially constitutes an indicator of very early rises in ICP. Further research is warranted to verify the feasibility of the use of NIR-T/BSS in clinical practice in patients with diseases and post-traumatic pathologies involving elevated intracranial pressure.

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