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Thomas Geeraerts
Yoann Launey
Laurent Martin
Julien Pottecher
Bernard Vigué
Jacques Duranteau
Dan Benhamou

Ultrasonography of the optic nerve sheath may be useful for detecting raised intracranial pressure after severe brain injury

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T. Geeraerts (✉) · Y. Launey · L. Martin · J. Pottecher · B. Vigué · J. Duranteau · D. Benhamou
University of Paris-Sud, Department of Anesthesiology and Critical Care Medicine, Hospital of Bicêtre, Assistance Publique-Hôpitaux de Paris (AP-HP), Faculty of Medicine,
Le Kremlin Bicêtre, 94275 Paris, France
e-mail: thomas.geeraerts@bct.aphp.fr
Tel.: +33-1-45213429
Fax: +33-1-45212875

Abstract *Objective:* To assess at admission to the ICU the relationship between optic nerve sheath diameter (ONSD) and intracranial pressure (ICP) and to investigate whether increased ONSD at patient admission is associated with raised ICP in the first 48 h after trauma. *Design and setting:* Prospective, blind, observational study in a surgical critical care unit, level 1 trauma center. *Patients and participants:* 31 adult patients with severe traumatic brain injury (TBI; Glasgow coma scale ≤ 8) requiring sedation and ICP monitoring, and 31 control patients without brain injury requiring sedation. *Measurements and results:* ONSD was measured with a 7.5-MHz linear ultrasound probe. Two TBI groups were defined on the basis of ICP profile. If ICP exceeded 20 mmHg for more than 30 min in the first 48 h (before any specific treatment), patients were considered to have high ICP; if not, they had normal ICP. The largest ONSD value (the highest value for the right and left eye) was significantly higher in high ICP patients (6.3 ± 0.6 vs. 5.1 ± 0.7 mm in normal ICP patients and 4.9 ± 0.3 mm in control patients). There was a significant relationship between the largest ONSD and ICP at admission ($r = 0.68$). The largest ONSD was a suitable predictor of high ICP (area under ROC curve 0.96). When ONSD was under 5.7 mm, the sensitivity and negative predictive values for high ICP were 100%. *Conclusions:* In the early posttraumatic period, ocular ultrasound scans may be useful for detecting high ICP after severe TBI.

Keywords Ocular ultrasound · Traumatic brain injury · Elevated intracranial pressure · Optic nerve sheath

Introduction

Raised intracranial pressure (ICP) is associated with poor outcome after traumatic brain injury (TBI) [1, 2]. In the early posttraumatic period there is a high risk of secondary ischemic damage to the brain, and ICP monitoring is highly recommended [3]. However, in emergency situations ICP monitoring remains difficult due to delays in obtaining blood coagulation test results or lack of surgical availability [4]. Noninvasive, simple, bedside methods have been developed for rapid assessment of the risk of high ICP. The transcranial Doppler (TCD) pulsatility index has been shown to reflect decreases in cerebral perfusion pressure (CPP) due to increases in ICP [5]. However, TCD is not always easy to perform even in expert hands as unsatisfactory images are obtained in about 5% of patients when a temporal window is used [6, 7].

Ocular ultrasonography has recently been used to detect elevated ICP. The optic nerve is part of the central nervous system and the intraorbital subarachnoid space surrounding the optic nerve is subject to the same pressure changes as the intracranial compartment [8–11]. The intraorbital part of the subarachnoid space is distensible and can therefore inflate if pressure increases. The optic nerve

sheath diameter (ONSD) displays predominantly anterior enlargement following injections into the orbital perineural subarachnoid space in cadavers [12]. In humans, following an intrathecal lumbar infusion of Ringer's solution, ONSD dilation reaches a maximum at peak cerebral spinal fluid (CSF) pressure, strongly suggesting a close relationship between CSF pressure and dilation of the orbital perineural subarachnoid space [13]. Several clinical studies have found that ONSD is correlated with elevated ICP [13–15]. ONSD values higher than the upper limit of the normal range have been recorded in children with hydrocephalus or hepatic failure with clinical signs of high ICP [14, 16]. In adults with moderate TBI ONSD is correlated with signs of high ICP on computed tomography (CT) [15, 17]. However, the correlation between ONSD and direct measurements of ICP has never been studied in trauma patients. Moreover, the significance of ONSD in patients on sedation and mechanical ventilation with severe TBI is unknown.

We therefore studied the early posttraumatic period to determine the relationship between sonographic ONSD and intraparenchymal ICP. We also investigated whether a dilated ONSD at patient admission is be associated with raised ICP in the first 48 h after trauma. As "normal" values of ONSD have not been determined in sedated patients, we also studied a control group of ICU patients requiring sedation and mechanical ventilation but without head injury. Preliminary results of this work were presented in part at the 48th meeting of the Societé Française d'Anesthésie-Réanimation in Paris, France, 27–30 September 2006, and at the 35th meeting of the Société de Réanimation de Langue Française in Paris, France, 17–19 January 2007.

Methods

Study population

Between December 2005 and June 2006 49 adult patients were admitted to our surgical intensive care unit (ICU in a level 1 trauma center). Brain-injured patients with a postresuscitation Glasgow Coma Scale (GCS) score of 8 or less and abnormal head CT who required sedation, mechanical ventilation, and ICP monitoring were considered for inclusion in the TBI group. Patients with ocular trauma or a known history of ocular pathology (as glaucoma or cataract) or who died within the first 24 h after brain injury were excluded from the study. Patient care was in line with existing protocols and was not modified by this study. The study design was approved by the local research and ethics committee (Le Kremlin-Bicêtre, France). Given the observational nature of the study the committee waived the requirement for informed consent from the patient or his family (see Electronic Supplementary Material, ESM, 1).

Of the 49 patients 7 were excluded because no investigator was available, 8 because of ocular trauma,

Table 1 Distribution of general, hemodynamic and metabolic characteristics of patients (*ICU*, intensive care unit; *SAPS*, Simplified Acute Physiology Score; *MAP*, mean arterial pressure; *ICP*, intracranial pressure; *CPP*, cerebral perfusion pressure; $PaCO_2$, arterial carbon dioxide partial pressure)

	Brain-injured patients ($n=31$)			Controls ($n=31$)		
	Normal ICP ($n=16$)	High ICP ($n=15$)	*p*	Controls	*p* vs. normal ICP	*p* vs. high ICP
Age (years)	38 ± 19	38 ± 18	NS	55 ± 20	0.008	0.01
Weight (kg)	74 ± 9	75 ± 7	NS	77 ± 15	NS	NS
Sex male	63%	73%	NS	77%	NS	NS
Overall ICU length of stay (days)	17.6 ± 11	26.6 ± 34	NS	7 ± 10	NS	0.002
SAPS II score	38 ± 15	38 ± 11	NS	29 ± 15	NS	NS
Injury Severity Score	28 ± 13	27 ± 9	NS	–	–	–
Glasgow Coma Score, mode (range)	7 (5–8)	6 (3–8)	NS	–	–	–
20% mannitol infusion during prehospital transport	6%	13%	NS	0%	–	–
Data during first ocular sonography						
Heart rate (bpm)	83 ± 20	82 ± 11	NS	85 ± 25	NS	NS
MAP (mmHg)	90 ± 14	104 ± 22	0.01	74 ± 12	0.001	< 0.0001
ICP (mmHg) (first reading)	14 ± 3	26 ± 3	0.001	–	–	–
CPP (mmHg)	77 ± 13	73 ± 16	NS	–	–	–
Norepinephrine infusion	37%	53%	0.02	44%	NS	NS
Plasma sodium concentration (mmol/l)	141 ± 3.9	138 ± 3.8	NS	139 ± 4.6	NS	NS
$PaCO_2$ (Kpa)	4.8 ± 0.5	4.9 ± 0.5	NS	5.1 ± 0.4	NS	NS
$PaCO_2$ (mmHg)	37 ± 4	38 ± 4		39 ± 3		
Tympanic temperature (° C)	37.1 ± 0.9	37.2 ± 0.9	NS	36.9 ± 0.6	NS	NS

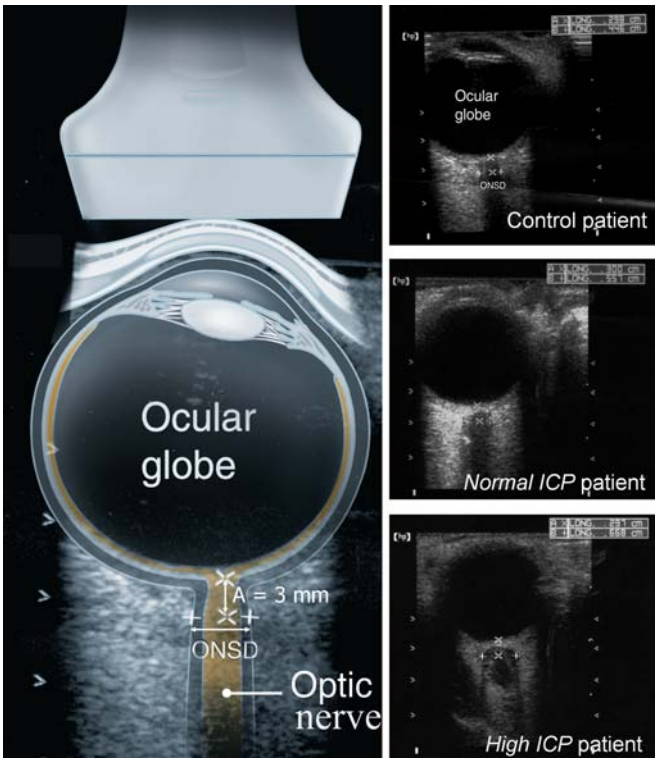

Fig. 1 Two-dimensional ocular sonography. *Left* A diagram showing the ultrasound probe, the ocular globe and the optic nerve with the optic nerve sheath diameter (ONSD) together with the ultrasound settings is presented; ONSD was measured 3 mm behind the globe, using an electronic caliper (A X LONG measure), and an axis perpendicular to the optic nerve (B + LONG measure). *Right* Typical results for a control patient, a patient with traumatic brain injury (TBI) and normal ICP, and a patient with TBI and high ICP

and 2 because their TCD signals were suboptimal. One patient died within the first 24 h (brain death) and was not included in the final results presentation; nevertheless as this case could have given extremely useful information, data pertaining it are presented in the discussion section. Data are thus presented for 31 TBI patients: 16 with normal ICP and 15 with high ICP during the first 48 h after injury (Table 1). The two groups did not differ in demographic characteristics, severity scores, or initial GCS score. Six patients in the high-ICP group underwent urgent neurosurgery (two acute extradural hematomas, three acute subdural hematomas performed during the first 48 h, one decompressive craniectomy performed on day 4). No patient in the normal ICP group was operated on for neurosurgical purposes. Norepinephrine infusions were administered to 53% of the high-ICP patients and to 37% of the normal-ICP patients. The groups did not differ significantly in plasma sodium concentration, $PaCO_2$, or tympanic temperature. The initial ICP value was significantly lower in normal-ICP than in high-ICP patients, but CPP did not differ significantly between the two groups.

The values of TCD and of ocular sonography taken into consideration for the present study were not the first values obtained immediately after patient arrival (during FAST) but rather those obtained after initial CT and the decision to place ICP device. These measurements were performed before ICP monitoring device placement. The ICP monitoring device was implanted no more than 1 h after ultrasound evaluation, and the first values were recorded 10 min after device insertion. Thereafter ICP was monitored continuously until at least 48 h after injury.

Control patients were also included. These patients were admitted to the ICU during the same period, also required sedation and mechanical ventilation, but were free of TBI. These patients were admitted for septic shock, prolonged postoperative mechanical ventilation, multiple trauma without brain injury, acute renal failure or acute respiratory failure. They were also sedated with intravenous midazolam and sufentanil. Ocular sonography and TCD were performed the day after admission, but no ICP monitoring device was implanted in these patients. Of the 32 control patients initially enrolled, the data for one were subsequently not included because the TCD signals were suboptimal (Table 1).

Ocular sonography

Ultrasound examination of the eye was performed by investigators trained in ocular sonography (T. G. or Y. L.) and according to previously described protocols [15, 18–20]. Investigators were blinded to cerebral CT results, and ultrasound results were obtained before ICP monitoring device placement. Patients were placed in a supine position at 20° to the horizontal. A thick layer of gel was applied over the closed upper eyelid. The 7.5-MHz linear probe (HP Sonos 5500, Hewlett Packard, Les Ulis, France) was placed only on the gel in the temporal area of the eyelid, not on the eyelid itself, to prevent pressure being exerted on the eye. The placement of the probe was adjusted to give a suitable angle for displaying the entry of the optic nerve into the globe [13]. The field was reduced to a depth of 4 cm (predetermined for "small organs"), and the optic nerve was not zoomed. The two-dimensional mode was used, and ONSD was measured 3 mm behind the globe using an electronic caliper and an axis perpendicular to the optic nerve (Fig. 1). For each optic nerve two measurements were made—one in the sagittal plane and the other in the transverse plane—by rotating the probe clockwise.

Hemodynamic and intracranial monitoring

Arterial pressure was monitored continuously, via a femoral catheter (4 F, Seldicath, Plastimed, Saint Leu

La Forêt, France). ICP was measured continuously via an intraparenchymal catheter (Neuromonitor-Microsensor kit, Codman, Chatenay Malabry, France) inserted into the frontal lobe by a neurosurgeon. CPP was calculated as the difference between mean arterial pressure (MAP) and ICP. Elevated ICP was defined as ICP of 20 mmHg or higher for more than 30 min during the first 48 h after injury (before any specific treatment such as hypothermia, mannitol, or decompressive craniectomy). Such episodes were detected using the trend data from the patient monitor (IntelliVue MP90, Philips Medical Systems, Eindhoven, The Netherlands; see ESM 2).

Results

Optic nerve sheath diameter

As the normal value of ONSD in sedated patients was unknown at the start of the study the control group was used as a reference. ONSD in control patients was 4.9 ± 0.3 mm in the right eye and 4.8 ± 0.5 mm in the left (Table 2). Right and left ONSD in normal ICP patients were, respectively, 5.1 ± 0.7 and 5.0 ± 0.7 mm (n.s. vs. control). High-ICP patients had a significantly larger ONSD than patients of the other two groups (right ONSD 6.2 ± 0.4 mm and left ONSD 6.3 ± 0.6 mm, $p < 0.0001$ vs. the control group and normal-ICP group).

Transcranial Doppler

End diastolic velocities of both middle cerebral arteries (MCA) were significantly lower in high-ICP patients. The left MCA pulsatility index value was significantly higher in high-ICP patients than in normal-ICP patients (1.45 ± 0.67 vs. 0.99 ± 0.09 cm/s, $p = 0.002$; Table 2). No significant difference was found for the right MCA pulsatility index.

Correlation between ONSD and baseline ICP

At patient admission a significant relationship was found between the largest ONSD (the highest value obtained for the right or left ONSD) and baseline ICP (linear regression, $r = 0.68$, $p < 0.0001$, $n = 31$; Fig. 2).

Prediction of elevated ICP in the first 48 h

Baseline ICP accurately predicted elevated ICP in TBI patients in the first 48 h (area under the curve, AUC, 0.94; 95% confidence interval, CI, 0.79–0.99; Fig. 3). The best cutoff value was 18 mmHg (sensitivity 80%; specificity 100%). The largest ONSD also accurately predicted elevated ICP in the first 48 h (AUC 0.96; 95% CI 0.83–0.99), with a best cutoff value of 5.9 mm (sensitivity 87%; specificity 94%). The positive predictive value of this cutoff

Table 2 Ocular ultrasound, transcranial Doppler, and CT results (*ONSD*, optic nerve sheath diameter; *MCA*, middle cerebral artery; *ICP*, intracranial pressure)

	Brain-injured patients ($n = 31$)			Controls ($n = 31$)		
	Normal ICP ($n = 16$)	High ICP ($n = 15$)	*p*	Controls	*p* vs. normal ICP	*p* vs. high ICP
Right ONSD (mm)	5.1 ± 0.7	6.2 ± 0.4	< 0.0001	4.9 ± 0.3	NS	< 0.0001
Left ONSD (mm)	5.0 ± 0.7	6.3 ± 0.6	< 0.0001	4.8 ± 0.5	NS	< 0.0001
Transcranial Doppler						
Right MCA						
Systolic velocity (cm/s)	119 ± 37	90 ± 26	0.02	84 ± 26	0.0003	NS
Mean velocity (cm/s)	73 ± 25	50 ± 21	0.001	51 ± 14	0.0005	NS
End diastolic velocity (cm/s)	49 ± 20	29 ± 9	< 0.0001	34 ± 11	0.0005	NS
Pulsatility index	1.01 ± 0.34	1.29 ± 0.49	NS	0.96 ± 0.25	NS	0.005
Left MCA						
Systolic velocity (cm/s)	102 ± 29	99 ± 41	NS	86 ± 25	NS	NS
Mean velocity (cm/s)	63 ± 20	50 ± 22	NS	51 ± 13	NS	NS
End diastolic velocity (cm/s)	42 ± 15	28 ± 15	0.003	33 ± 8	NS	NS
Pulsatility index	0.99 ± 0.09	1.45 ± 0.67	0.002	0.99 ± 0.22	NS	0.008
Head CT						
Marshall's category						
I–II	94%	33%	0.001	–	–	–
III	6%	27%	NS	–	–	–
IV	0%	0%	NS	–	–	–
V	0%	33%	0.01	–	–	–
VI	0%	7%	NS	–	–	–
Traumatic subarachnoid hemorrhage	50%	80%	NS	–	–	–
Compressed cysterns	25%	80%	0.001	–	–	–

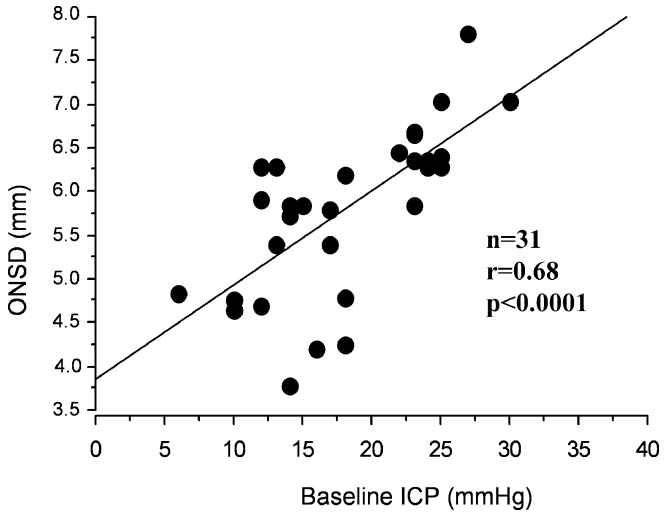

Fig. 2 Relationship between parenchymal ICP and largest ultrasonographic ONSD (the highest value obtained for the right or left side) on admission after initial CT. Linear regression analysis identified a significant relationship between ICP and ONSD

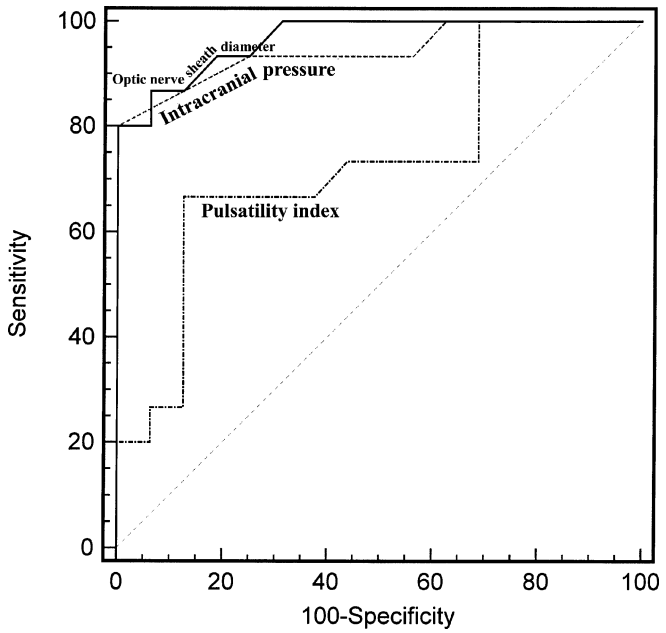

Fig. 3 Receiver operating characteristic curves for ICP, largest ONSD, and worst pulsatility index value for the two middle cerebral arteries on admission to the intensive care unit after initial CT with respect to high ICP (ICP ≥ 20 mmHg for more than 30 min in the first 48 h after injury)

value for the prediction of elevated ICP was 93% and its negative predictive value 88%. The sensitivity and negative predictive values were 100% for ONSD cutoff of 5.7 mm. The area under the receiver operating characteristic (ROC) curves for ICP and ONSD were similar ($p = 0.63$). The worst pulsatility index value (the highest value obtained for the right or left MCA) was found to predict increased ICP less efficiently than baseline ICP or largest ONSD (AUC 0.73; 95% CI 0.54–0.87, $p = 0.02$ vs. ICP ROC curve and $p = 0.009$ vs. ONSD ROC curve).

Reproducibility of ONSD measurements

Three repeated measurements (in less than 1 h) of ONSD were performed in 12 TBI patients by T. G. and Y. L. (6 normal ICP, 6 high ICP). The mean standard deviation of ONSD for a single patient was 0.27 mm.

Discussion

Our results suggest that sonographic measurement of the ONSD in the early posttraumatic phase is potentially useful for detecting elevated ICP in patients with severe brain injury.

In 35 adult patients with TBI assessed in the emergency department Blaivas et al. [15] found that signs of elevated ICP on brain CT were closely related to dilation of the optic nerve sheath on ocular ultrasound scans. Mean ONSD was 6.3 mm in patients with radiological signs of elevated ICP and 4.4 in patients with no such signs. The authors considered 5 mm to be the upper limit of normal values. The specificity of this cutoff value for detecting tomographic elevated ICP was 93% and its negative predictive value 100%. These results were recently confirmed by the same team [17]. We obtained similar results using intraparenchymal ICP. The gold-standard method for ICP measurement remains intraventricular drain; nevertheless intraparenchymal probes appears to be an acceptable alternative with a small drift to the zero reference and a low infection rate [21]. Using the ROC curve we were able to improve the definition of the cutoff value of ONSD predicting elevated ICP. The best cutoff value was 5.9 mm. As this test is likely to be used for detection, its sensitivity (probability of positive tests for sick patients) and negative predictive value (probability of the patient being healthy if the test is negative) must be excellent. Both these values were 100% when largest ONSD was under 5.7 mm.

TCD has been used for detecting secondary neurological deterioration in the first few hours after injury in patients with mild to severe TBI [22, 23]. Prolonged pulsed TCD may induce a significant thermal effect that should be prevented by minimizing the duration of exposure [24]. Pulsatility index values reflect CPP more accurately than ICP [5]. In this study, in line with local protocols, some patients received aggressive treatment in the shock/trauma room—use of vasoactive agents to maintain MAP. The pulsatility index recorded after this initial treatment may have been artificially low due to CPP normalization. In this study the poor ability of pulsatility index to predict elevated ICP may be related to catecholamine-induced MAP support, as shown by

the absence of difference in CPP between the two TBI groups.

The head CT classification described by Marshall et al. [25] was designed to predict the risk of elevated ICP and mortality rate. Subarachnoid hemorrhage on CT is also significantly correlated with the risk of elevated ICP [26]. Nevertheless, the initial CT is found to be normal in about 20% of patients with severe TBI [27, 28], principally because it is carried out very soon after injury. In this study head CT was always performed less than 4 h after TBI, and the observation that more than 30% of high ICP patients had a Marshall's classification of category I or II is therefore not surprising. Furthermore, the proportion of patients with traumatic subarachnoid hemorrhage on initial CT was similar in both the normal-ICP and high-ICP groups. This highlights the limitations of using the initial CT as a predictor of elevated ICP and the need for other indices.

One of the limitations of this study is that ocular ultrasound scans were not carried out in patients with ocular trauma. Facial trauma with orbital fractures and optic nerve injury may occur in 10% of patients with severe TBI [29]. The effects of eye trauma on ultrasonographic ONSD measurement are unclear, but the interpretation of such scans would probably be very difficult.

Ocular ultrasound is regarded as safe when Doppler frequency analysis is not used for prolonged duration [30]. B-mode with ultrasound equipment as used today is not capable of producing harmful temperature rises [24]. In the present study the two-dimensional mode was used without Doppler analysis. Variation in ultrasonographic measurement of ONSD seems to be limited as the median intraobserver and interobserver variations were shown to be, respectively, 0.1 and 0.3 mm [31]. Our results also show limited variations during repeated measurements (mean standard deviation 0.27 mm). Nevertheless, the two investigators were trained in ocular sonography. Inexperience with sonography may be an important limitation for the use of this method, and study of the learning curve for critical care physicians would certainly be of interest. Moreover, these variations could also be affected by the quality of the ultrasound machine used.

The limited correlation between ONSD and ICP obtained at the patient's admission may be due to the delay between ultrasound examination and ICP monitoring. Due to the "blinded to ICP" study design, ultrasonographic evaluation and TCD were carried out before ICP placement. Time lag to the first ICP results could not be predicted exactly but was consistently less than 1 h. The lack of strictly simultaneous measurements may have decreased the correlation between ICP and ONSD. When only strictly simultaneous measurements of ICP and ONSD are considered (this was the case in 13 of our patients with one measure on day 1 and one on day 2 posttrauma), a better relationship was found between the largest ONSD (the highest value obtained, for the right or left ONSD) and ICP (Fig. 4, linear regression, $r = 0.74$; $p < 0.0001$).

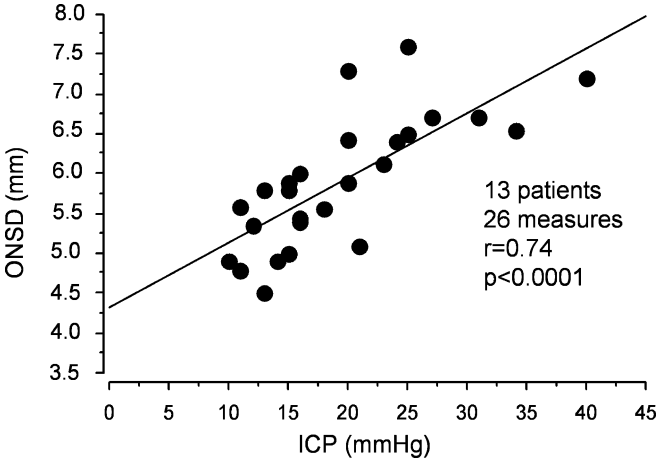

Fig. 4 Relationship between simultaneous parenchymal ICP and largest ultrasonographic ONS (the highest value obtained for the right or left side) during the 48 first hours after admission (one measure on day 1 and one on day 2 posttrauma)

Initial data obtained for the patient who died within the first 24 h (brain death) already showed very high values of ONSD at admission (7.7 mm). The TCD pulsatility index value was 6.6 (end diastolic velocity 0 cm/s) and first reading ICP was 75 mmHg. Refractory elevated ICP rapidly occurred despite aggressive treatment including large dose of mannitol (500 ml) and decompressive craniectomy. Unfortunately, data from ONSD during brain death could not be obtained.

This study did not consider the necessity for invasive ICP monitoring in patients with severe TBI. There is a strong need for reliable ICP monitoring in severe TBI patients because such monitoring may improve outcome [32, 33]. Nevertheless, ICP monitoring is not routinely used in many centers, principally because no neurosurgeon is available to implant the monitoring device. This study provides new information on the utility of ultrasonographic ONSD measurement in the first few hours after brain injury to predict increases in ICP. Ultrasonographic ONSD measurement with standard probes can easily be taught. Our results suggest that when a patient with brain injury is admitted to a hospital with no available neurosurgeon, ultrasonographic ONSD measurement may be useful, together with the determination of neurological status, TCD, and CT, to assess the urgency of ICP monitoring device placement and transfer to a specialized center.

Conclusion

This study shows that ocular ultrasound imaging in the early posttraumatic period may be useful for predicting elevated ICP after severe TBI. There was no case of elevated ICP in the first 48 h when initial optic nerve sheath diameter was lower than 5.7 mm. There was a significant relationship between ONSD and ICP. This simple, rapid, noninvasive method can be used at the patient's bedside to detect high ICP, but these data should be confirmed before proposing to include routinely ONSD in the multimodal monitoring of patients presenting with severe head trauma.

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References

1. Becker DP, Miller JD, Ward JD, Greenberg RP, Young HF, Sakalas R (1977) The outcome from severe head injury with early diagnosis and intensive management. J Neurosurg 47:491–502
2. Marshall LF, Smith RW, Shapiro HM (1979) The outcome with aggressive treatment in severe head injuries. II. Acute and chronic barbiturate administration in the management of head injury. J Neurosurg 50:26–30
3. Chesnut RM, Marshall LF, Klauber MR, Blunt BA, Baldwin N, Eisenberg HM, Jane JA, Marmarou A, Foulkes MA (1993) The role of secondary brain injury in determining outcome from severe head injury. J Trauma 34:216–222
4. Ghajar J (2000) Traumatic brain injury. Lancet 356:923–929
5. Hassler W, Steinmetz H, Gawlowski J (1988) Transcranial Doppler ultrasonography in raised intracranial pressure and in intracranial circulatory arrest. J Neurosurg 68:745–751
6. Aaslid R, Huber P, Nornes H (1986) A transcranial Doppler method in the evaluation of cerebrovascular spasm. Neuroradiology 28:11–16
7. Kirkpatrick PJ, Chan KH (1997) Transcranial Doppler. In: Reilly P, Bullock R (eds) Head injury. Chapman & Hall, London
8. Liu D, Kahn M (1993) Measurement and relationship of subarachnoid pressure of the optic nerve to intracranial pressures in fresh cadavers. Am J Ophthalmol 116:548–556
9. Villain MA, Candon E, Arnaud B, Hamard H, Adenis JP (2003) Optic nerve sheath decompression in optic neuropathy complicating idiopathic intracranial hypertension: a new focus. J Fr Ophtalmol 26:191–197
10. Hayreh SS (1968) Pathogenesis of oedema of the optic disc. Doc Ophthalmol 24:289–411
11. Lichtenstein D (1992) In: Lichtenstein D (eds) L'echographie générale en réanimation. Springer, Berlin Heidelberg New York
12. Hansen HC, Helmke K (1996) The subarachnoid space surrounding the optic nerves. An ultrasound study of the optic nerve sheath. Surg Radiol Anat 18:323–328
13. Hansen HC, Helmke K (1997) Validation of the optic nerve sheath response to changing cerebrospinal fluid pressure: ultrasound findings during intrathecal infusion tests. J Neurosurg 87:34–40
14. Newman WD, Hollman AS, Dutton GN, Carachi R (2002) Measurement of optic nerve sheath diameter by ultrasound: a means of detecting acute raised intracranial pressure in hydrocephalus. Br J Ophthalmol 86:1109–1113
15. Blaivas M, Theodoro D, Sierzenski PR (2003) Elevated intracranial pressure detected by bedside emergency ultrasonography of the optic nerve sheath. Acad Emerg Med 10:376–381
16. Helmke K, Burdelski M, Hansen HC (2000) Detection and monitoring of intracranial pressure dysregulation in liver failure by ultrasound. Transplantation 70:392–395
17. Tayal VS, Neulander M, Norton HJ, Foster T, Saunders T, Blaivas M (2007) Emergency department sonographic measurement of optic nerve sheath diameter to detect findings of increased intracranial pressure in adult head injury patients. Ann Emerg Med 49:508–514
18. Helmke K, Hansen HC (1996) Fundamentals of transorbital sonographic evaluation of optic nerve sheath expansion under intracranial hypertension. I. Experimental study. Pediatr Radiol 26:701–705
19. Helmke K, Hansen HC (1996) Fundamentals of transorbital sonographic evaluation of optic nerve sheath expansion under intracranial hypertension. II. Patient study. Pediatr Radiol 26:706–710
20. Blaivas M, Theodoro D, Sierzenski PR (2002) A study of bedside ocular ultrasonography in the emergency department. Acad Emerg Med 9:791–799
21. Steiner LA, Andrews PJ (2006) Monitoring the injured brain: ICP and CBF. Br J Anaesth 97:26–38
22. Jaffres P, Brun J, Declety P, Bosson JL, Fauvage B, Schleiermacher A, Kaddour A, Anglade D, Jacquot C, Payen JF (2005) Transcranial Doppler to detect on admission patients at risk for neurological deterioration following mild and moderate brain trauma. Intensive Care Med 31:785–790
23. Ract C, Le Moigno S, Bruder N, Vigue B (2007) Transcranial Doppler ultrasound goal-directed therapy for the early management of severe traumatic brain injury. Intensive Care Med 33:645–651
24. Barnett SB, Ter Haar GR, Ziskin MC, Rott HD, Duck FA, Maeda K (2000) International recommendations and guidelines for the safe use of diagnostic ultrasound in medicine. Ultrasound Med Biol 26:355–366
25. Marshall LF, Marshall SB, Klauber MR, Van Berkum Clark M, Eisenberg H, Jane JA, Luerssen TG, Marmarou A, Foulkes MA (1992) The diagnosis of head injury requires a classification based on computed axial tomography. J Neurotrauma [Suppl] 9(1):S287–S292
26. Eisenberg HM, Gary HE Jr, Aldrich EF, Saydjari C, Turner B, Foulkes MA, Jane JA, Marmarou A, Marshall LF, Young HF (1990) Initial CT findings in 753 patients with severe head injury. A report from the NIH Traumatic Coma Data Bank. J Neurosurg 73:688–698
27. Lobato RD, Sarabia R, Rivas JJ, Cordobes F, Castro S, Munoz MJ, Cabrera A, Barcena A, Lamas E (1986) Normal computerized tomography scans in severe head injury. Prognostic and clinical management implications. J Neurosurg 65:784–789
28. Stein SC, Spettell C, Young G, Ross SE (1993) Delayed and progressive brain injury in closed-head trauma: radiological demonstration. Neurosurgery 32:25–30

29. Holt GR, Holt JE (1983) Incidence of eye injuries in facial fractures: an analysis of 727 cases. Otolaryngol Head Neck Surg 91:276–279
30. Williamson TH, Harris A (1996) Color Doppler ultrasound imaging of the eye and orbit. Surv Ophthalmol 40:255–267
31. Ballantyne SA, O'Neill G, Hamilton R, Hollman AS (2002) Observer variation in the sonographic measurement of optic nerve sheath diameter in normal adults. Eur J Ultrasound 15:145–149
32. Cremer OL, van Dijk GW, van Wensen E, Brekelmans GJ, Moons KG, Leenen LP, Kalkman CJ (2005) Effect of intracranial pressure monitoring and targeted intensive care on functional outcome after severe head injury. Crit Care Med 33:2207–2213
33. Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care (2000) Recommendations for intracranial pressure monitoring technology. J Neurotrauma 17:497–506