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Increase in optic nerve sheath diameter induced by epidural blood patch: a preliminary report

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Editor's key points

- Post-dural puncture headache appears to be related to cerebrospinal fluid hypotension.
- Optic nerve sheath diameter (ONSD) measured by ultrasonography reflects intracranial pressure (ICP).
- In a preliminary study of 10 subjects, successful epidural blood patch increased ONSD.
- This supports an increase in ICP as the therapeutic mechanism.

Background. Post-dural puncture headache (PDPH) might be related to cerebrospinal fluid hypotension. Studies in brain-injured patients have shown a good relationship between optic nerve sheath diameter (ONSD) measured by ocular sonography and invasively measured intracranial pressure (ICP). The aim of this study was to evaluate changes in ONSD after lumbar epidural blood patch (EBP).

Methods. Consecutive subjects receiving an EBP for PDPH were included. ONSD and pain measurements were performed before (T_0), 10 min (M_{10}), 2 h (H_2), and 20 h (H_{20}) after the EBP.

Results. Ten subjects were included. ONSD [median (inter-quartile range)] increased with time after EBP, from 4.8 mm (4.5–5.1) at T_0 to 5.2 mm (4.9–5.7) at M_{10} (P=0.005 *vs* T_0), 5.5 mm (5.1–6.0) at H_2 (P=0.007 *vs* T_0), and 5.8 mm (5.2–6.3) at H_{20} (P=0.02 *vs* T_0). EBP was clinically successful in nine of 10 subjects. In subjects in whom EBP was successful, ONSD significantly increased at M_{10} and T_2 compared with T_0 (P=0.004 and 0.008, respectively) but did not reach statistical significance at H_{20} (P=0.06). In the subject in whom EBP failed, a small increase in ONSD was observed over time.

Conclusions. In this preliminary report, EBP was followed by ONSD enlargement in subjects with successful EBP, but not in the subject with EBP failure. Since ONSD is a surrogate marker of ICP, this suggests that a sustained increase in ICP is associated with successful EBP.

Keywords: epidural blood patch; intracranial hypotension; intracranial pressure; optic nerve sheath diameter; post-dural puncture headache

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Post-dural puncture headache (PDPH) is a rare complication of spinal and epidural anaesthesia. The incidence of accidental dural puncture is low, estimated at 0.5% in obstetric anaesthesia in a recent study of 17 198 epidural blocks.[1] Many treatments have been proposed, but the most effective remains lumbar epidural blood patch (EBP).[2]

Two mechanisms have been proposed to explain the efficacy of EBP in PDPH.[3] The first suggests that the blood patches the hole in the dura mater which stops the cerebrospinal fluid (CSF) leak. The second suggests that relief of symptoms are relieved due to an increase in intracranial pressure (ICP) induced by injection of blood around the lumbar dura mater. Both hypotheses suggest that CSF hypotension is involved in PDPH pathophysiology. However, modifications of ICP after EBP have been studied only in rats,[4] with no studies available in humans.

The optic nerve, as part of the central nervous system, is surrounded by a dural sheath. The intraorbital subarachnoid space surrounding the optic nerve is subject to similar pressure changes to those in the intracranial and lumbar compartments.[5 6] The retrobulbar part of the perioptic subarachnoid space is distensible and can therefore inflate as pressure increases. Hayreh[7] showed in monkeys and humans that the subarachnoid space surrounding the optic nerve communicates with the intracranial cavity and that changes in CSF pressure can be transmitted along the optic nerve sheath. In humans, after an intrathecal lumbar infusion of Ringer's solution, optic nerve sheath diameter (ONSD) dilation reaches a maximum at peak CSF pressure, strongly suggesting a close relationship between CSF pressure and dilation of the orbital perineural subarachnoid space.[8] Recent studies have shown that the ONSD measured by ultrasonography correlates with ICP in different clinical situations.[9]

We hypothesized that changes in ONSD after EBP correlate with symptom relief associated with correction of CSF hypotension.

Table 1 Subject characteristics. PDPH, post-dural puncture headache; EBP, epidural blood patch

Subject	Gender	Age (yr)	Weight (kg)	Cause of PDPH	Volume injected in EBP (ml)	Outcome
1	Female	36	70	Epidural anaesthesia	23 (Blood)	Success
2	Female	29	85	Epidural anaesthesia	25 (Blood)	Failure
3	Male	28	65	Lumbar puncture	25 (Blood)	Success
4	Male	35	68	Lumbar puncture	22 (Blood)	Success
5	Male	42	59	Epidural infiltration	32 (Blood)	Success
6	Male	35	63	Lumbar puncture	16 (Blood)	Success
7	Female	38	75	Epidural anaesthesia	17 (Blood)	Success
8	Female	39	81	Epidural anaesthesia	26 (Blood)	Success
9	Female	23	70	Lumbar puncture	25 (Blood)	Success
10	Female	34	86	Lumbar puncture	30 (Colloids)	Success

Methods

Human subjects, control group, and ethics approval

This study was conducted in two teaching hospitals (University hospitals Antoine Béclère, Clamart, and Bicêtre, Kremlin-Bicêtre, France) and was approved by the Ethics Committee. For the control group of healthy volunteers, informed consent was collected before subjects were studied. This study is registered at Afssaps.fr (Number ID RCB: 2010-A01352-37). The diagnosis of PDPH was made by the physician in charge of the subject according to the criteria defined by the International Headache Society.[10] All subjects had clinically documented symptomatic PDPH that had failed to respond to medical treatment. After informed consent, subjects were included when referred to the anaesthetist for lumbar EBP. During the EBP, blood injection was stopped when the subject felt low back pain or drowsiness.

Conduct of the study

Ultrasound measurement of ONSD was performed as previously described[8 9 11] by two investigators trained in ocular ultrasonography (C.D. or A.L.G.). Subjects were placed in the supine position. A thick layer of gel was applied over the closed upper eyelid. A 7.5 MHz linear probe (Micromaxx, Sonosite Inc., Bothell, WA, USA) was placed on the lateral area of the closed eye, the hand lying on the forehead of the subject, to prevent excessive pressure being exerted on the eye. The placement of the probe was adjusted to view the entry of the optic nerve into the globe. The field was reduced to a depth of 40 mm. Two-dimensional mode was used to measure ONSD 3 mm behind the globe using an electronic calliper and an axis perpendicular to the optic nerve. For each optic nerve, two measurements were made, one in the transverse plane and one in the sagittal plane. The reported ONSD corresponds to the mean of the four values obtained for each subject (transverse and sagittal plane for both eyes).

Data handling

Severity of headache was assessed using numerical pain score (NPS) (from 0 to 10) collected before the blood patch (T_0), 10 min (M_{10}), 2 h (H_2), and 20 h (H_{20}) after the EBP whenever possible. Success of EBP was defined by relief of pain with an NPS $<4/10$.

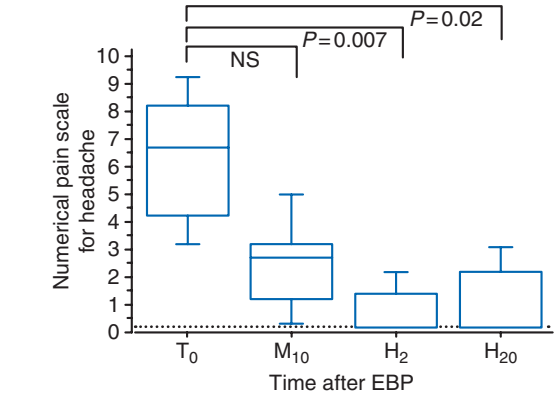

Fig 1 Changes in numerical pain scale for headache after EBP for subjects with successful blood patch (T_0, before blood patch; M_{10}, 10 min after; H_2, 2 h after; H_{20}, 20 h after). 10th, 25th, 75th, 90th percentiles and median values are represented.

Statistical analysis

Assuming a non-Gaussian distribution, the Wilcoxon matched pairs test was used to compare ONSD and pain scales at the different times (Prism 4.0, GraphPad Software, Inc., La Jolla, USA). A *P*-value of <0.05 was considered statistically significant. Values are expressed as median and inter-quartile range (IQR).

Results

Ten subjects were included. Epidemiologic characteristics are presented in Table 1. Measures at H_{20} were possible in only five subjects due to early hospital discharge of other subjects.

EBP was successful in nine of 10 subjects. Median NPS for headache decreased with time from 6.5 (4–8) at T_0 to 2.5 (1–3) at M_{10}, 0 (0–1.25) at H_2, and 0 (0–2) at H_{20} (Fig. 1).

In the overall data set of 10 subjects, ONSD increased with time after EBP, from 4.8 mm (4.5–5.1) at T_0 to 5.2 mm (4.9–

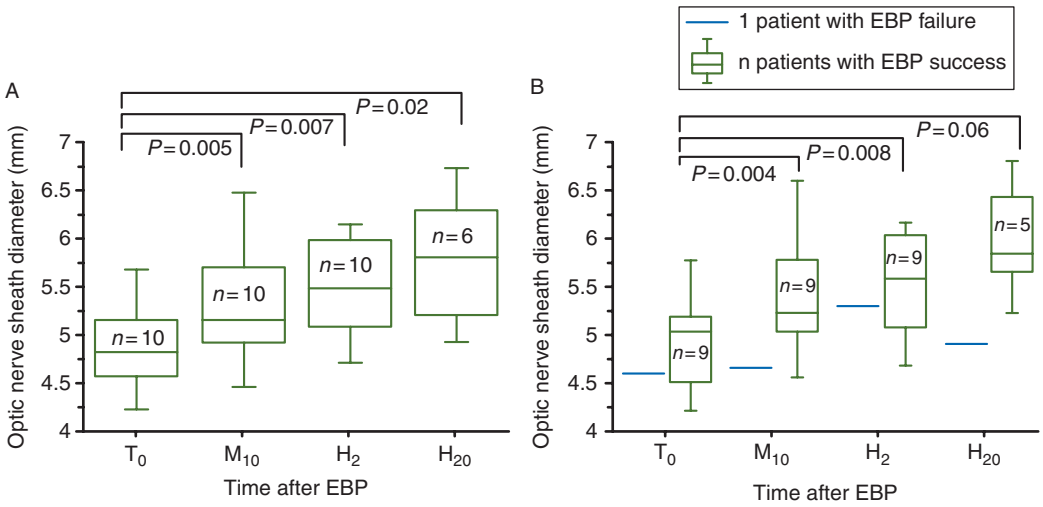

Fig 2 Changes in ONSD measured with ocular sonography after EBP (T_0, before blood patch; M_{10}, 10 min after; H_2, 2 h after; H_{20}, 20 h after). (A) Values in the overall data set of 10 subjects, and (B) according to the success or failure of EBP. 10th, 25th, 75th, 90th percentiles and median values are represented. The number of subjects included at each time is represented in the boxes.

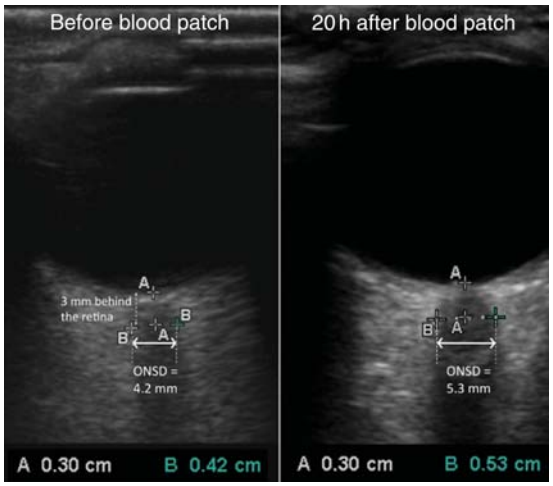

Fig 3 Example of ONSD measurement using sonography before and after EBP. The calliper is used to perform the measurement 3 mm behind the ocular globe (retina).

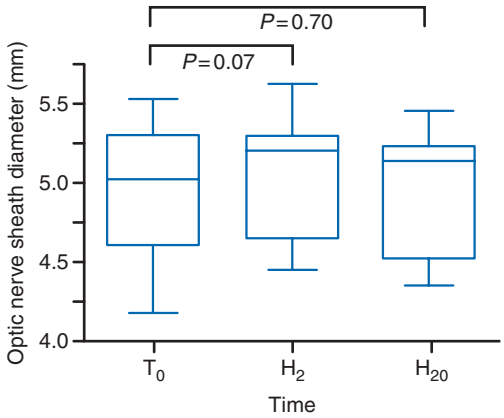

Fig 4 Changes in ONSD measured with ocular sonography in 10 healthy volunteers (T_0, first measurement; H_2, 2 h after; H_{20}, 20 h after). 10th, 25th, 75th, 90th percentiles and median values are represented.

5.7) at M_{10} (P=0.005 *vs* T_0), 5.5 mm (5.1–6.0) at H_2 (P=0.007 *vs* T_0), and 5.8 mm (5.2–6.3) at H_{20} (P=0.02 *vs* T_0) (Fig. 2A).

In subjects in whom EBP was successful, ONSD significantly increased at M_{10} and H_2 *vs* T_0 (P=0.004 and 0.008, respectively) but did not reach significance at H_{20} compared with T_0 (P=0.06). In the subject in whom EBP failed, a small increase in ONSD was observed from 4.6 mm at T_0 to 4.7 mm at M_{10}, 5.3 mm at H_2, and 4.9 mm at H_{20} (Fig. 2B). Moreover, baseline ONSD values (before EBP) were not different from those obtained in subjects in whom EBP was successful. An example of ONSD changes after EBP is shown in Figure 3.

To assess the stability of ONSD measurements with time, ocular sonography was also performed in a control group of 10 healthy volunteers at T_0, H_2, and H_{20}. In this control group, there was no significant change in ONSD over time (Fig. 4).

Discussion

In this preliminary report, we showed that in nine subjects suffering from PDPH, the optic nerve sheath enlarged early after successful EBP. Interestingly in the one subject, in whom EBP failed to resolve the PDPH, ONSD did not increase after EBP.

Usubiaga and colleagues[12] showed that injection of 20 ml of saline solution in the lumbar epidural space in humans

caused an immediate increase in epidural and subarachnoid pressures lasting from 3 to 10 min. Magnetic resonance imaging (MRI) performed before and after lumbar puncture showed that reductions in intracranial CSF volume were frequently related to PDPH.[13] In a porcine model, lumbar epidural anaesthesia increased ICP.[14] MRI changes and reduced ONSD after a dural tap indicate CSF hypovolaemia.[15] More recently, Kroin and colleagues[4] found that epidural injection of either a saline solution or whole blood caused a transient increase in ICP of 7.8 mm Hg. Whole blood and fibrin glue were able to maintain the cisternal pressure elevation for 4 h.

EBP usually produces immediate relief of headache.[16] This 'tamponade' effect is probably transient and the long-term efficacy of the EBP could result from sealing the dural defect by the injected blood, stopping the CSF leak. The immediate increase in ONSD observed in our study could reflect the tamponade effect of EBP, and the sustained increase in ONSD the progressive correction of ICP with CSF production within the first 20 h. Interestingly, in our subject in whom EBP failed to resolve headache, ONSD did not increase significantly, suggesting that neither the tamponade effect nor the CSF sealing effect had occurred.

Our study has some potential limitations. Lack of experience with ocular ultrasonography is a potential limitation of this method. However, the learning curve is rapid (between 10 and 25 scans depending on previous ultrasonography experience),[17] and the two investigators in the present study were well trained in ocular ultrasonography. Variability in ultrasonographic measurement of ONSD is also a potential limitation, but has been shown to be limited to <0.2 and 0.3 mm for median intra- and inter-observer variations, respectively.[18 19] In the present study, the difference in median ONSD was 0.7 mm between T_0 and H_2 and 0.9 mm between T_0 and H_{20}. Thus, it is unlikely that these differences were solely related to intra- or inter-observer variations. We acknowledge that our series is small and does not explore all clinical situations that might be encountered (such as cases in which EBP fails to resolve PDPH or in which PDPH recurs after initial efficacy).

To our knowledge, this is the first study in humans using a simple non-invasive method to estimate ICP changes induced by EBP. We found that success of EBP was accompanied by a substantial increase in ONSD, whereas in a single case, failure was not. However, further larger studies are needed to evaluate the value of ONSD measurement to predict EBP efficacy.

Conflict of interest

None declared.

Funding

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