Published in final edited form as:

Int J Obstet Anesth. 2014 May ; 23(2): 144–150. doi:10.1016/j.ijoa.2014.01.003.

Observational study of changes in epidural pressure and elastance during epidural blood patch in obstetric patients

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Abstract

Background—During an epidural blood patch, we inject blood until the patient describes mild back pressure, often leading to injection of more than 20 mL of blood. We undertook this study to measure the epidural pressures generated during an epidural blood patch and to identify the impact of volume on epidural elastance in obstetric patients.

Methods—This study was performed in postpartum patients who presented for an epidural blood patch with symptoms consistent with a postdural puncture headache. After identification of the epidural space using loss of resistance to air or saline, we measured static epidural pressure after each 5-mL injection of blood. Models were then fitted to the data and the epidural elastance and compliance calculated.

Results—Eighteen blood patches were performed on 17 patients. The mean final volume injected was 18.9 ± 7.8 mL [range 6 to 38 mL]. The mean final pressure generated was 13.1 ± 13.4 mmHg [range 2 to 56 mmHg]. A curvilinear relationship existed between volume injected and pressure, which was described by two models: (1) pressure = $0.0254 \times (\text{mL injected})^2 + 0.0297 \times \text{mL}$, or (2) pressure = $0.0679 \times \text{mL}^{1.742}$. The value for r^2 was approximately 0.57 for both models. We found no correlation between the final pressure generated and the success of the EBP.

Conclusions—We found a curvilinear relationship between the volume of blood injected during an epidural blood patch and the pressure generated in the epidural space. However, there was a large variation in both the volume of blood and the epidural pressure generated. The clinical importance of this finding is not known. A larger study would be required to demonstrate whether pressure is a predictor of success.

Keywords

Compliance; Epidural blood patch; Pressure

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Disclosure

The authors have no conflicts of interest to declare.

Introduction

The epidural blood patch (EBP) is considered standard for treatment of postdural puncture headache (PDPH).^{1,2} It is generally believed that the initial relief of PDPH symptoms following EBP is due to a tamponade effect of the injected blood on the lumbar cerebrospinal fluid (CSF), leading to normalization of CSF pressure.^{3,4} This mass effect on the lumbar thecal sac has been demonstrated by magnetic resonance imaging and appears to last for several hours.^{4,5} Intracranial pressure (ICP) has been shown to rise after EBP, supporting the hypothesis that an increase in lumbar CSF pressure leads to normalization of ICP and initial relief of the symptoms: permanent relief likely occurs from closure of the dural hole and CSF re-accumulation.^{6,7} However, it is not known what pressures are generated within the epidural space during the creation of this tamponade. Usubiaga et al. demonstrated that rapid injection of saline 20 mL into the epidural space increased both lumbar subarachnoid and epidural pressures to as high as 850 mmH₂O (equivalent to approximately 65 mmHg).⁸ Coombs and Hooper measured subarachnoid pressures following injection of up to 15 mL of blood for therapeutic EBP;⁹ using a slow injection, they found a maximum increase of 152 mmH₂O (approximately 11.2 mmHg) in subarachnoid pressure.

The appropriate volume of blood to inject during EBP has not been established. To date, epidural pressures generated when blood volumes >20 mL are injected for EBP and the relationship between injected volume and the resulting pressure have not been reported. Our usual practice when performing an EBP has been to inject blood into the epidural space until the patient describes mild back pressure, which frequently leads to the injection of volumes >20 mL. The purpose of this observational study was to measure epidural pressures during EBP in obstetric patients, and to determine the correlation between the injected volume and these pressures.

Methods

This was a prospective, observational study performed between November 1996 and December 1997. The Committee for Clinical Investigations at Beth Israel Hospital provided full ethical and scientific review, and final approval of the protocol and informed consent document. Written informed consent was obtained from a convenience sample of obstetric patients who had symptoms consistent with a PDPH after neuraxial anesthesia and requested an EBP. As this was a purely observational study, no power calculation was determined. The study was arbitrarily terminated after 14 months. The EBP was performed in our standard fashion, with sterile preparation and draping of the arm for phlebotomy and of the back for the epidural procedure. The phlebotomist initially obtained 20 mL of blood, withdrew an additional 20 mL while the first aliquot was injected, and withdrew a final 20 mL in case pressure sensation in the back was not reached within 40 mL. All procedures were performed with the patient in the left lateral decubitus position. After identification of the epidural space with a 17-gauge Tuohy needle (Smiths Medical, Keene, NH, USA) using loss of resistance to saline or air, a sterile three-way stopcock placed on the end of the Tuohy needle. An attending anesthesiologist or senior resident under direct supervision of an

attending anesthesiologist performed all EBP procedures. One port of the stopcock was connected to a strain-gauge pressure transducer using sterile non-compressible tubing flushed with non-heparinized saline (Transpac IV, Hospira, Lake Forest, IL, USA) (Fig. 1). Initial pressure in the epidural space for each subject was defined as the zero point before the injection of any blood. The stopcock was closed to the transducer during blood injection and was then switched open to the transducer and epidural space, but closed to epidural syringe, to measure epidural pressure following injection of each 5-mL aliquot of blood. The pressure tracing was allowed to equilibrate until no perceptible changes in the pressure tracing could be seen, which generally occurred within several seconds. This was recorded as the steady state pressure. This was repeated for every 5 mL injected until the patient experienced mild back pressure or discomfort. If this discomfort occurred in the middle of a 5-mL injection, a final pressure reading was obtained and the total volume noted. After the completion of the EBP, the patient was asked to sit up in bed for several minutes to assess the initial efficacy of the injection. This was categorized as “complete initial relief”, “partial initial relief”, or “no improvement”.

Statistical analysis

The relationship between the final volume injected or the epidural pressure generated, and the initial efficacy of the EBP was determined using logistic regression. The resulting epidural pressure as a function of volume for all patients were combined in aggregate, and fitted to the quadratic relationship:

$$P(V)=AV^2+BV \quad (1)$$

and to the power law relationship:

$$P(V)=CV^D \quad (2)$$

where P denotes the epidural pressure referenced from baseline, and V is the cumulative volume of injected blood. The coefficients for Equation 1 (A and B) were estimated using a linear least squares technique, while those of Equation 2 (C and D) were obtained using a nonlinear gradient search algorithm (SigmaPlot version 11.0, Systat Software, Inc., Chicago, IL, USA). For both equations, we computed the coefficient of determination (r^2). The correlation was considered statistically significant for $P < 0.05$. Statistical comparison between the two models was made using the corrected Akaike Information Criterion (AICc).¹⁰ The Intraclass Correlation Coefficient (ICC) using the volume injected as the class was determined in order to assess the degree of consistency in the grouping of pressures generated at each volume.

The epidural elastance E (mmHg.mL⁻¹) as a function of injected volume V (mL) was determined according to the derivative of pressure with respect to volume for each model (Equations 3 and 4, respectively):

$$E(V)=\frac{dP}{dV}=2AV+B \quad (3)$$

$$E(V) = \frac{dP}{dV} = CDV^{D-1} \quad (4)$$

Epidural compliance ($\text{mL} \cdot \text{mmHg}^{-1}$) for each model was determined as the reciprocal of elastance ($C = 1/E$).

Results

The delay between patient enrolment and manuscript preparation was due largely to misplacement of the original data collection sheets when one of the authors (SDP) changed offices; the data sheets were found after a considerable delay following a second office move. A total of 17 patients who received 18 EBPs were enrolled between November of 1996 and December of 1997. Patient demographics are shown in Table 1. Nine patients had a documented accidental dural puncture with an epidural needle, seven likely had a PDPH from spinal anesthesia (either spinal or combined-spinal-epidural anesthesia), and two developed symptoms consistent with a PDPH after epidural anesthesia without a documented dural puncture.

Data for the EBPs are shown in Table 2. The mean volume injected was 18.9 ± 7.8 mL with a range 6 to 38 mL. The mean final epidural pressure generated was 13.1 ± 13.4 mmHg, with a range 2 to 56 mmHg. Only two patients developed epidural pressure >30 mmHg, the others were <25 mmHg. By design, all patients described mild lumbar or radicular symptoms at the final injection volume.

There was a strong correlation between the volume injected and the pressure generated (Fig. 2). The quadratic fit to the relationship between the volume (mL) injected and the dependent pressure yielded coefficients of $A = 0.0254 \text{ mmHg} \cdot \text{mL}^{-2}$ and $B = 0.0297 \text{ mmHg} \cdot \text{mL}^{-1}$, with $r^2 = 0.567$. The power law fit yielded coefficients of $C = 0.0679 \text{ mmHg} \cdot \text{mL}^{-2}$ and $D = 1.742^*$ with $r^2 = 0.577$. Both models yielded statistically significant fits to the data ($P < 0.001$), with AICc demonstrating no difference in their corresponding qualities of fit. Based on these estimated parameters, the predicted epidural elastance demonstrated a linear (Equation 3) or near-linear (Equation 4) dependence on injected volume, as shown in Fig. 3. Likewise, epidural compliance demonstrated a hyperbolic relationship with injected volume. The ICC was 0.83 (95% CI 0.75 to 0.89, $P < 0.001$).

The initial relief of symptoms following EBP was partial or complete in 15/18 (83.3%) women. One had no relief and the success was not recorded in the final two. While there was a strong positive correlation between the volume injected and the pressure generated, there was no correlation between either parameter or the initial efficacy of the EBP. The one patient with no initial relief had her EBP five days after a documented dural puncture with the Tuohy needle; she received 12 mL of blood and the final pressure generated was 6 mmHg. No complications were reported with EBP in any patients.

*Since the exponent D in Equation 2 was not constrained to an integer value, the actual units of the parameters C and D depend on their values. For simplicity, we assumed that the units of the coefficient C in Equation 2 were $\text{mmHg} \cdot \text{mL}^{-2}$ and that of elastance as predicted by Equation 4 were $\text{mmHg} \cdot \text{mL}^{-1}$.

Discussion

We found a strong, curvilinear relationship between the amount of blood injected into the epidural space during an EBP and the static pressure generated. A quadratic pressure-volume relationship implies an expandable epidural space that stiffens as its volume fills. This is consistent with linearly increasing elastance (or, equivalently, a hyperbolically decreasing compliance) with injected blood volume (Fig. 3). The epidural pressure generated during EBP, and a curvilinear pressure-volume relationship have not been previously reported. Grocott et al. measured the change in ICP associated with epidural injection of lidocaine in normal pigs and pigs with increased ICP.¹¹ They calculated an elastance of $8.3 \pm 3.1 \text{ mmHg.mL}^{-1}$ in normal pigs. They did find that the change in ICP increased as the baseline ICP increased, suggesting increasing elastance, but this was not calculated. Similarly, Hilt et al. described ICP changes during epidural injections of local anesthetic in two patients with baseline increased ICP.¹² They found a linear correlation between the baseline ICP and the change in the ICP ($r = 0.864$), suggesting a possible binomial relationship between injected volume and the final ICP. However, they did not calculate the elastance. Hirabayashi et al. calculated the resistance and compliance of the epidural space in humans during rapid injection of a fixed volume (10 mL) of local anesthetic.¹³ They calculated a mean compliance of $0.39 \pm 0.13 \text{ mL.mmHg}^{-1}$. This converts to an elastance of $2.6 \pm 0.9 \text{ mmHg.mL}^{-1}$. However, because they injected the same volume into each subject, they could not determine the impact of injectate volume on compliance/elastance. They did find that increased compliance was associated with a greater number of blocked segments, while increased resistance to injection decreased block height.

It is generally believed that the initial effect of the EBP is predicated on an increase in CSF pressure caused by the mass effect of the injected blood.³ While a mass effect of the injected blood on the lumbar thecal sac has been demonstrated,^{4,5} little is known about the epidural pressures generated by this effect. Our data suggest that there is a wide range of pressures generated by the EBP and that only a small increase in pressure might be necessary to achieve success. In nine of 18 patches, the final pressure increase was $<10 \text{ mmHg}$ and in five it was 5 mmHg or less. Patients three and 17 had complete resolution of their symptoms with final pressures of only 4 and 3 mmHg, respectively.

The clinical implications of the rise in epidural pressure are unknown. If the elevation in epidural pressure, and thus cerebrospinal pressure, is required to normalize ICP and provide immediate relief of headache, then perhaps measuring the pressure generated would be a better indicator of likelihood of success than volume injected. Recent findings indicate that an EBP may be associated with only a 60–75% permanent cure rate after PDPH.^{14,15} Increasing the volume of blood injected from 20 to 30 mL does not appear to improve this rate.¹⁵ Perhaps the failure of increased volume to improve success is due to the large individual variability in the pressures generated at any given volume. Based on the r^2 for either model in the current study, we found that only 57% of the variation in pressure could be attributed to the volume injected. Likely because of the small sample size, we did not find an association between the volume of blood injected or the final pressure generated and the initial EBP success. However, one could postulate that the final pressure generated during EBP would be a better predictor of success than volume. In support of this hypothesis,

Higuchi et al. found that injection of 5, 10, or 15 mL of saline into the epidural space produced variable degrees and pattern of compression of the thecal sac between individuals, and large differences in the amount that flowed out the intervertebral foramina.¹⁶ Measurement of the epidural pressure may better reflect the impact of the injectate on the epidural space and thus initial success. More research would be required to determine this.

While the maximum epidural pressure reached was <25 mmHg in most of the patients in our series, surprisingly high pressures (34 and 56 mmHg) were observed in two women. Although this was associated with injection of >20 mL of blood (38 and 32 mL, respectively), the volumes were not exceedingly high and not dissimilar from that used in a recent randomized trial.¹⁵ Based on our results, one would expect that these high epidural pressures could also be reached in normal clinical practice.

Injection into the epidural space is not without potential risk. Usubiaga et al. described headache in patients after rapid injection of saline into the epidural space.⁸ They reported peak epidural pressures similar to that in our study (up to 85 cm H₂O); this pressure was short-lived because they injected saline. Gill et al. reported 12 cases of blindness after epidural fluid injection during epiduroscopy.¹⁷ The patients generally received epidural injection of 41 to 120 mL (although one received only 20 mL) and all cases were associated with retinal hemorrhage. The authors postulated that the rise in ICP associated with epidural injection likely led to retinal venous obstruction and subsequent venous hemorrhage. They recommended an epidural injection rate of no more than 1 mL per 1–2 seconds. While blindness is a very rare complication of epidural procedures, it has been reported after epidural steroid¹⁸ and local anesthetic injection,^{19,20} and even after the injection of 30 mL of saline to treat a dural puncture.²¹ Routine measurement of the epidural pressure during EBP might have a clinical advantage by helping to avoid the generation of excessively high pressure that may occur even at relatively low volumes, and thus may prevent the consequent complications associated with rapid increases in CSF pressure described above.

The final potential utility of measuring the pressure during EBP would be to help prove, or disprove, that an acute elevation in CSF pressure is the mechanism for the initial relief during EBP. Five of our patients demonstrated only a small rise in epidural pressure. The need for only a small increase in pressure is supported by Gormley's original description of the EBP, in which he achieved rapid and complete resolution of PDPH in six patients with the injection of only 2 to 3 mL of blood.²² Fichtner et al. found no change in ICP after injection of 15 mL into the lumbar epidural space during a successful EBP.²³ Franzini et al. suggested a novel mechanism for spontaneous intracranial hypotension (SIH); they speculated that SIH may be caused by profound lowering of the epidural pressure caused by the excessive drainage of the epidural venous plexus.²⁴ This creates a large pressure gradient between the CSF and epidural space and promotes CSF movement to the epidural space, and even epidural tearing. They further postulated that the goal of the epidural blood patch is thus merely to bring epidural pressure back to normal in order to reestablish the usual pressure gradient. This would require a smaller increase in epidural pressure than might be needed to create a shift of CSF from the lumbar space to the cranium as was postulated by Carrie.³ The degree to which this would be relevant during PDPH after a dural puncture is not clear. However, if large studies were to fail to find a correlation between the pressure

generated and initial success, as was the case in our series, it would be difficult to argue that this pressure is important in the mechanism of EBP.

We have assumed that the pressure measured in the epidural space at the end of the Tuohy needle is representative of pressures throughout the rest of the central nervous system. This seems reasonable based on available data, but we cannot prove this assumption as we did not measure ICP or pressures in other parts of the epidural space. Usubiaga et al. demonstrated that intrathecal (IT) pressures paralleled epidural pressures with the injection of 20 mL of epidural saline, and that the IT pressures were consistently *higher* than epidural pressure.⁸ Coombs and Hooper⁹ and Grocott and Mutch¹¹ found rises in lumbar CSF and intracranial epidural pressures with injections into the lumbar epidural space, but they did not measure the lumbar epidural pressures *per se*, and thus could not directly correlate the two pressure sets. We can think of no reason that the lumbar epidural pressure would not be transmitted to the CSF and cranium as long as the blood remained even partially liquid (a requirement for ongoing injection). The Monro-Kellie doctrine, which dictates that the sum of the volumes of the brain, CSF and intracranial blood is constant,²⁵ is dependent on the fact that changes in the pressure from one of these liquids is transmitted to the others and their volumes impacted by this pressure change. As long as the blood in the epidural space remains liquid (and thus deformable), the mean pressure in the blood (as measured by our system) should be transmitted to the CSF and thus to the brain.

We can identify several limitations to this study. First, the small sample size makes it difficult to draw clinical conclusions from our findings. A larger study might have been able to establish a relationship between the final volume or pressure and the initial efficacy of the blood patch. Second, the anesthesia providers who performed the blood patch were not blinded to the pressure generated, and they also did the assessment of the initial efficacy of the EBP. The amount of blood injected might have unduly influenced the observer's assessment of EBP success, as clinicians would likely believe that more volume is likely to be more effective. As we did not find a correlation between volume injected and efficacy, we doubt this had a large impact. Neither the volume injected nor the pressure generated was an interpreted result, and thus they were unlikely to be influenced by individual bias. Third, much of the procedure was not standardized (using the endpoint of patient back pressure, the amount injected using the loss of resistance technique, the rate of injection, the time at which the steady state pressure was determined). It is possible that some clinicians injected more blood than others after the patient described back pressure. Our experience of using this technique for >20 years has demonstrated that once the patient describes back pressure it is unlikely that more than about five additional mL can be injected. More importantly, "forcing" additional blood volume into the epidural space would not invalidate our results. By design, all patients experienced subjective back pressure during the EBP. However, there was a large inter-individual variation in the measured pressure at which these symptoms occurred. There was no cut-off value at which the symptoms appeared. In addition, variation in the rate of injection is unlikely to have influenced our results as we were obtaining static pressure reading of epidural space elastance, not dynamic pressure readings of epidural space resistance. With regards to the steady state pressure, it is certain that the pressure in the epidural space was continuously and slowly falling until it returned

to normal (likely hours to days after the procedure). We measured pressures in mmHg and thus could not identify small changes in pressure. Thus our results were based on an apparent steady-state in the observed measurement. The extrapolation of these data to the non-pregnant population may be limited. Pregnancy is associated with a decrease in lumbar CSF volume (at least when supine), a decrease in the amount of inter-foraminal leak of epidural injectate and an increase in the accumulation of epidural injectate into the posterior portion of the epidural space.²⁶ This is associated with a larger decrease in lumbar CSF volume during epidural injection than that seen in the non-pregnant state.²⁶ These changes might be expected to influence the pressure-volume relationship during EBP, and thus our findings might not be relevant to non-pregnant patients. Our patients were a mean of 2.5 days postpartum, so the degree to which these pregnancy-related changes were still in effect is not known. Finally, we pooled data from all measurements in all patients, rather than assessing the fit for each patient. We did this in part because there were only few measurements for some of the patients. The assessment of the ICC helped to demonstrate that there was statistically highly significant grouping of the pressure generated at each volume group across patients, but the r^2 of 0.57 suggests that factors other than injected volume play a role in the pressure generated.

The system to measure the epidural pressure was clumsy and not easy to use, potentially limiting its use in clinical practice. Also, any movement of the epidural needle after initial placement might change the reference pressure. This possibly occurred in two patients where the pressure jumped to 16–18 mmHg after the first 5-mL injection and rose very slowly after that. In retrospect, measuring dynamic pressures (as done by Grocott et al.¹¹) would have aided in the measurement of elastance using a stress-relaxation model. Unlike Grocott et al. however, we measured pressure after multiple volumes, and were able to calculate elastance curves based on these multiple measurements. This would have also precluded the need to manipulate the stopcock during the procedure. Finally, the wide range in measured pressure at which patients experience symptoms would limit the use of subjective sensations as a surrogate for epidural injection pressure. It is possible that two separate mechanisms are responsible for the rise in pressure and the symptoms felt.

In conclusion, we found a positive, monotonic, curvilinear, concave relationship between the volume of blood injected during epidural blood patch and the pressure generated in the epidural space. This could be expressed as a quadratic or power relationship, each with a fair (r^2 approximately 0.57) correlation. The clinical importance of this finding is not known.

Acknowledgments

This study was partially supported by NIH Grant HL089227.

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Fig. 1.
Schematic diagram of experimental arrangement. See text for details.

Fig. 2.

Relationship between injected volume and epidural pressure during epidural blood patch.

Symbols denote data from individual patients. Thick solid line represents quadratic fit to all data, while dashed line represents two-parameter power law fit.

Fig. 3.
Predicted epidural compliance and elastance curves simulated from quadratic fit (solid lines) and two-parameter power law fit (dashed lines).

Table 1

Patient demographics

Age (years)	33.5 ± 3.3
Height (cm)	164.9 ± 6.9
Weight (kg)	80.8 ± 13.8
Body mass index (kg/m ²)	29.8 ± 4.4
Procedure (total n = 18)	
Epidural	11 (61.1%)
Documented dural puncture	9 (50%)
No documented dural puncture	2 (11.1%)
Spinal	2 (11.1%)
Combined spinal-epidural	5 (27.8%)

Data are mean (SD) or number (%).

Table 2

Epidural blood patch data

Days post-procedure	
Mean $\pm$ SD	2.5 $\pm$ 1.2
Median [range]	2 [0–5]
Blood volume injected (mL)	
Mean $\pm$ SD	18.9 $\pm$ 7.8
Median [IQR]	19 [12–21]
Range	6–38
Final pressure (mmHg)	
Mean $\pm$ SD	13.1 $\pm$ 13.4
Median [IQR]	9 [5.8–15.5]
Range	2–56
Initial success	
Complete	9 (50%)
Partial	6 (33.3%)
None	1 (5.6%)
Not reported	2 (11.1%)

Data are mean $\pm$ SD, median [IQR], range and number (%)