Transmural Pressure of Epidural Veins in the Thoracic and Lumbar Spine of Pigs

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Background: The aim of this study was to determine the transmural pressure gradient of epidural veins located within the spinal column and to investigate the effects of increased intra-abdominal and intrathoracic pressure on this gradient.

Methods: Fourteen pigs were sedated with ketamine and anesthetized with halothane. Ventilation was controlled after tracheotomy. Needles were inserted into the epidural and subarachnoid space, and a catheter was threaded into an epidural vein. Pressures in these structures were measured during controlled and spontaneous ventilation, increased abdominal pressure, and thoracic compression. Measurements were made in both the thoracic and lumbar regions.

Results: The pressure gradient between the epidural vein and the surrounding epidural space was low—1 or 2 mmHg—in both the lumbar and thoracic areas. This gradient was not affected by the mode of ventilation or increased abdominal or thoracic pressure (or both), even though the absolute intramural pressure increased.

Conclusion: The pressure gradient between the epidural vein and the surrounding space is low and does not change when abdominal or thoracic pressures are increased. (Key words: Anatomy; anesthetic techniques; Batson’s venous plexus; epidural space; physiology; positive end-expiratory pressure; spine; ventilation.)

BLOOD is encountered frequently when needles or catheters are placed for epidural and spinal anesthesia.

For example, Horlocker et al. found frank blood in 7% and minor hemorrhage in 15% of 1,000 blocks performed for orthopedic procedures. However, the incidence of subarachnoid or epidural hematomas that disturb nerve function after these procedures is very low even in patients who have anticoagulation therapy as part of vascular surgery. The overall risk for a spinal hematoma after central nerve block has been estimated to be 1:150,000 after epidural anesthesia and 1:220,000 after spinal anesthesia. But what accounts for this apparent paradox? Don’t injured vessels bleed?

One possible explanation for minimal bleeding despite vascular trauma is a low pressure gradient between epidural veins and the epidural space. We tested this hypothesis in pigs by simultaneously measuring cerebrospinal fluid (CSF), epidural vein, and epidural space pressures in both lumbar and thoracic areas and by determining their variation with changes in intra-abdominal and intrathoracic pressures.

Materials and Methods

General Preparation

Fourteen farm-bred male pigs (20–25 kg) were sedated with ketamine (16 mg/kg given intramuscularly) and anesthetized with halothane (0.5 to 0.8%, end-tidal). Body temperature was maintained at 39°C with a heating pad. After skin infiltration with 2% lidocaine, a tracheotomy was performed and the trachea was intubated. The lungs were ventilated with oxygen delivered by a positive-pressure respirator (Harvard, S. Natick, MA) with 5 cm H₂O positive end-expiratory pressure to maintain an end-tidal carbon dioxide level of approximately 30 mmHg. The protocol was approved by the Animal Care and Use Committee of the University of Pittsburgh.

A catheter was inserted into the proximal aorta via the right carotid artery to measure systemic blood pressure. Heart rate was derived from the arterial pressure waveform with a cardiotachometer. A central venous

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catheter was inserted into the right external jugular vein. Correct placement was confirmed by pressure waveform analysis. Normal saline (5 ml·kg⁻¹·h⁻¹) was infused intravenously via an ear vein.

The animal was turned to the left lateral position. A 22-gauge spinal needle was inserted into the subarachnoid space in the lumbar region using a paramedian approach in 12 pigs and into the cisterna magna in four pigs. When free flow of CSF was obtained, the needle was attached to a saline-filled transducer (Gould, Cleveland, OH).

Two 17-gauge Touhy needles were inserted into the epidural space at adjacent interspaces using the loss-of-resistance technique. One was filled with saline, connected to a transducer, and used to measure epidural space “pressure.” We confirmed the needle position using a nerve stimulator attached to the metal stylet of a shortened epidural catheter. The catheter (Abbott model 15497-01, 20 gauge, Chicago, IL) was made of Teflon and had an end hole but no side holes. The stylet was advanced 2 mm beyond the tip of the catheter after the catheter was advanced 1 or 2 cm into the epidural space. Electric stimulation (5 mA, 1 Hz; Medtronic 5880A, Minneapolis, MN) of the wire produced vigorous motor activity in the chest wall (or lower extremities) if the catheter was positioned correctly in the thoracic (or lumbar) epidural space and only a vague, localized twitch of the paraspinal muscles if it was not.

After this test, the catheter was advanced in short jabs until a telltale “give” was felt, indicating puncture of an epidural vein. The stylet was completely withdrawn at this point, and venous blood could be aspirated if the cannulation was successful. The catheter was connected to a saline-filled transducer. The position of the catheter in an epidural vein was verified by injecting a 2-ml bolus of normal saline into the epidural space. A prompt increase in pressure in the epidural space and CSF (fig. 1) occurred in all animals except one, in which a vein outside the space presumably was cannulated. Data from this animal were excluded. The intravascular position was checked intermittently by injecting dobutamine (3–5 µg bolus). A prompt increase in arterial pressure and heart rate constituted a positive test dose.

Blood pressure, heart rate, central venous pressure, CSF pressure, epidural vein pressure, epidural space pressure, and the end-tidal carbon dioxide level were recorded on an oscillograph (model 3800; Gould). All transducers were set at zero atmospheric pressure at the level of the spinous processes in the lateral decubitus position and calibrated against a mercury column.

Fig. 1. The original record showing the test used to confirm the intraspinal location of the epidural vein catheter. A 2-ml bolus of normal saline was injected into the epidural space at the mark. If the catheter tip was intraspinal, a prompt and synchronous increase in vein, space, and cerebrospinal pressure occurred. Pressure in the vein did not increase in one animal in which the catheter was located in a vein outside the spinal column. Each upward tic on the bottom trace represents 1 s. The traces were electronically averaged.

**Experimental Protocol**

After recording a steady baseline, measurements were made during abdominal compression, thoracic compression, and simultaneous thoracic and abdominal compression. Firm manual pressure was applied for about 15 s. The interval between compressions was approximately 30–45 s, and sufficient time was allowed for the pressures to return to baseline before the next maneuver. Compressions were repeated to permit recording of both phasic and electronically averaged traces.

We also investigated the influence of different modes of ventilation. We observed the influence of spontaneous breathing by disconnecting the animal from the ventilator, of zero end-expiratory pressure by disconnecting the (5 cm H₂O) positive end-expiratory pressure.
valve during mechanical ventilation, and then of positive end-expiratory pressure by imposing a 10 cm H$_2$O valve.

Data Collection and Analysis
Data were recorded with a polygraph (Gould) and stored on magnetic tape for later analysis. Pressure values at baseline and peak were taken from the electronically averaged trace, and the gradients between pressures were calculated from these values in each animal and then averaged. Data were analyzed using a standard statistical package (SPSS-PC, version 1.0, Chicago, IL).

Changes in measured variables resulting from abdominal or chest compression and positive end-expiratory pressure were analyzed using a paired $t$ test. Probability values <$0.05$ were considered significant, and no correction for the multiple testing problem was made. Two-way analysis of variance was used to test the hypotheses that the site of measurement (lumbar vs. thoracic region) or type of compression (abdominal, chest, or both) affected the magnitude of the transmural pressure gradient of epidural veins. Data are presented as the mean, and variance is presented as the standard deviation.

Results
Measurements were made in the lumbar epidural space ($n = 6$), the thoracic space ($n = 5$), and in both spaces ($n = 2$). Systolic blood pressure and heart rate did not change during compressions (table 1). The central venous pressure was 2 or 3 mmHg lower than the epidural venous pressure. Baseline CSF, epidural venous, and epidural space pressures were similar in the lumbar and thoracic areas. No subatmospheric pressures were found in the epidural space of any animal. Venous, epidural space, and CSF pressure increased (all $P < 0.001$) together and became more phasic during the three compression maneuvers (fig. 2). The average transmural gradient of the epidural vein was about 1 or 2 mmHg, although negative values sometimes were observed. Although the absolute pressure within the vein and in the surrounding space increased with compression, the transmural pressure gradient was unchanged by compression maneuvers. Results of the analysis of variance revealed that this gradient was independent of the site of measurement (lumbar vs. thoracic) and type of compression (table 1, fig. 3).

Increasing the airway pressure from zero end-expiratory pressure to 10 cm H$_2$O positive end-expiratory pressure increased all pressures by about 1 mmHg both in the lumbar and thoracic area (fig. 4). Decreased airway pressure during apnea (fig. 5) and spontaneous respiration (data not shown) had a minimal effect on the pressures.

Discussion
The results show that the pressure gradient between the inside of an epidural vein and the surrounding space is small, approximately 1 or 2 mmHg, during spontaneous and controlled ventilation and during increased abdominal or thoracic pressure (or both).

Assumptions
We assumed that the epidural vein catheter was positioned in an epidural vein. We developed criteria to ensure correct venous placement but did not confirm the location of the catheter at autopsy.

We assumed that the measurements of epidural space pressure were reliable, although it is difficult to measure pressure in a space filled with fat, blood vessels, and loose connective tissue. Several lines of reasoning support this assumption. We found that the phasic trace closely resembled that measured in the subarachnoid space and in the epidural veins (fig. 5). All these traces had a small upward deflection that occurred shortly after the arterial pulse, and all varied with respiration. Bolus injections of saline (1 or 2 ml) into the epidural space through a separate needle resulted in a prompt and synchronous increase in all pressures (fig. 1).

Calculation of a transmural gradient assumes that the measured vein and epidural space pressures are representative of those found throughout these structures. This may not be a valid assumption for the space pressure measurement because the epidural space is really a series of discrete compartments rather than a uniform longitudinal sac. There is little assurance that pressure measured in one compartment represents that in all others. On the other hand, the assumption is likely valid for the venous system because pressure is transmitted equally in all directions in a fluid such as blood, and there are no valves in the epidural veins. Thus for our calculation to be valid, it is only necessary that an epidural vein run through the epidural “compartment” that contains the tip of the epidural needle. Such anatomic congruity was likely in our study because pigs have large epidural veins in the posterolateral epidural space.
Table 1. Hemodynamics and Local Pressure Values in Lumbar and Thoracic Regions of the Spine

<table>
<thead>
<tr>
<th>Variable</th>
<th>Condition</th>
<th>Lumbar Region</th>
<th>Thoracic Region</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Baseline</td>
<td>Peak</td>
</tr>
<tr>
<td>BPS (mmHg)</td>
<td>A</td>
<td>83 ± 15</td>
<td>88 ± 13</td>
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<tr>
<td></td>
<td>T</td>
<td>83 ± 15</td>
<td>82 ± 18</td>
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<tr>
<td></td>
<td>B</td>
<td>82 ± 12</td>
<td>86 ± 12</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>A</td>
<td>122 ± 25</td>
<td>122 ± 24</td>
</tr>
<tr>
<td></td>
<td>T</td>
<td>123 ± 26</td>
<td>123 ± 26</td>
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<tr>
<td></td>
<td>B</td>
<td>123 ± 26</td>
<td>124 ± 26</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>A</td>
<td>6.0 ± 2.1</td>
<td>13.2 ± 3.0</td>
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<tr>
<td></td>
<td>T</td>
<td>5.9 ± 2.2</td>
<td>8.8 ± 3.0</td>
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<tr>
<td></td>
<td>B</td>
<td>6.3 ± 2.8</td>
<td>13.2 ± 3.4</td>
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<tr>
<td>CSF pressure (mmHg)</td>
<td>A</td>
<td>8.2 ± 2.3</td>
<td>15.8 ± 2.4</td>
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<tr>
<td></td>
<td>T</td>
<td>8.0 ± 2.1</td>
<td>10.8 ± 2.5</td>
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<tr>
<td></td>
<td>B</td>
<td>7.9 ± 2.0</td>
<td>14.0 ± 2.3</td>
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<tr>
<td>Epidural vein pressure (mmHg)</td>
<td>A</td>
<td>8.0 ± 2.0</td>
<td>15.0 ± 2.6</td>
</tr>
<tr>
<td></td>
<td>T</td>
<td>8.0 ± 1.9</td>
<td>11.0 ± 2.1</td>
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<tr>
<td></td>
<td>B</td>
<td>8.1 ± 2.0</td>
<td>15.0 ± 2.5</td>
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<tr>
<td>Epidural space pressure (mmHg)</td>
<td>A</td>
<td>7.1 ± 2.1</td>
<td>15.1 ± 2.3</td>
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<tr>
<td></td>
<td>T</td>
<td>7.5 ± 2.5</td>
<td>10.6 ± 2.8</td>
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<tr>
<td></td>
<td>B</td>
<td>7.3 ± 2.6</td>
<td>14.3 ± 2.8</td>
</tr>
<tr>
<td>Epidural vein transmural pressure (mmHg)</td>
<td>A</td>
<td>0.8 ± 0.8</td>
<td>0.2 ± 1.5</td>
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<tr>
<td></td>
<td>T</td>
<td>0.6 ± 1.1</td>
<td>0.5 ± 1.2</td>
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<tr>
<td></td>
<td>B</td>
<td>0.8 ± 1.1</td>
<td>0.8 ± 1.2</td>
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</tbody>
</table>

A = abdominal compression; T = thoracic compression; B = both abdominal and thoracic compression; BSP = systolic blood pressure; HR = heart rate; CVP = central venous pressure; CSF = cerebrospinal fluid; Transmural = epidural vein pressure - epidural space pressure.

Data are mean ± SD.

*P < 0.001, n = 6 for lumbar region measurements; n = 7 for thoracic region measurements.

Fig. 2. The original record of one animal. The effects of manual compression of the abdomen, thorax, and both on epidural vein pressure (VEIN), epidural space pressure (SPACE), cerebrospinal fluid pressure, and central venous pressure are shown. Intraspinal pressure was measured in the thoracic region. During compression, pressures increase synchronously and to a similar degree. Each compression was made twice to allow for phasic and averaged recordings. The "rounded humps" are electronically averaged signals.
It could be argued that the small boluses of saline (1 or 2 ml) used to identify the epidural space as a part of the loss-of-resistance technique may have altered the pressure within the space, making it impossible to record the correct ("negative") pressure. We assume that this was not the case because space pressure returned to baseline in 1 or 2 min after the catheter and needle system were flushed. A similar time course was found recently by Grocott and Mutch.\textsuperscript{3} It seems likely that the fluid supplied in this manner was rapidly dispersed within the space or that it compressed adjacent veins. On the other hand, a small amount of fluid is necessary at the tip of the needle to "couple" the transducer with the space and produce an accurate measurement.

**Interpretation**

The primary finding in our study is that the transmural pressure of veins within the spinal epidural space is low and not affected by maneuvers that increase abdominal or thoracic pressure.

Pressure within the epidural veins was 2 or 3 mmHg higher than central venous pressure, which makes sense because this pressure difference drives venous...
return from the spinal column. Vein pressure increased simultaneously and to a similar degree as central venous pressure during abdominal and chest compression, preserving the gradient favoring venous return. The concordance in pressures corresponds with anatomic studies showing multiple connections between the veins in the spinal column and those in the adjacent body cavities.  

Pressure outside the epidural veins was positive, and only 1 or 2 mmHg less than intraluminal pressure. These pressures were closely linked during chest and abdominal compression. Our results conflict with those of previous studies showing subatmospheric pressure in the epidural space, 7-9 but they correspond with work by other investigators. 5,10,11 Our animals were in the lateral recumbent position and well hydrated. These factors may contribute to the positive epidural space pressure we observed. This explanation is suggested by the previous work of Bengis and Guyton, 9 who made careful measurements in anesthetized dogs in the ventral recumbent position. Their initial pressures were subatmospheric, but rapid infusion of intravenous fluids led to positive pressure within the epidural space, suggesting that filling of the epidural veins occurred and that there is a link between vein pressure and space pressure. This observation implies that the position and venous pressure of the subject are crucial determinants of the measured epidural space pressure. Our study confirms and extends these earlier results.

The parallel changes of CSF pressure with epidural space and vein pressures lends further weight to the concept of dynamic interactions between the flexible structures within the rigid spinal canal. Abdominal and chest compression presumably increased the volume of blood in epidural veins, thus increasing pressure on the other structures. Another plausible explanation is that increased intrathoracic pressure reduced cerebral venous outflow and increased CSF pressure. 12 Increases in CSF pressure in the spinal canal would act to increase epidural space pressure. Whatever the explanation, the consequence is that intramural pressure in epidural veins can be increased by these compression maneuvers without increasing the transmural pressure.

Our data provide a possible explanation for the curious observation that blood is frequently encountered during spinal and epidural anesthesia yet the incidence of a significant epidural hematoma is low after these procedures. Extravasation of even a small amount of blood should provide sufficient back pressure to tamponade the bleeding, given that the pressure driving blood out of the vein is only 1 or 2 mmHg. Our experimental maneuvers may be relevant to the clinical occurrence of ascites, pregnancy, or obesity, which result in increased abdominal pressure, and coughing or straining, which result in increased intrathoracic pressure.

**Study Limitations**

Because the study was done in pigs, the results may not be applicable to humans. There are important differences in the anatomy of the spinal canal between pigs, in which the epidural veins are posterolateral, and in humans, in whom the epidural veins are most frequently found in the anterior epidural space. 4 All animals were studied in the lateral recumbent position.

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**Fig. 5.** Pressure recorded from a systemic artery (BPS) as well as the epidural space, vein, and cerebrospinal fluid are shown during controlled ventilation and during a short period of apnea, during which recorder speed was increased 25 times. All intraspinal pressures showed muted arterial pulsations. Each upward tic on the time line represents 1 s.
and were well hydrated. Measurements made in other positions (such as sitting) or in poorly hydrated subjects might yield different results.

In conclusion, the pressures in the epidural space, the subarachnoid space, and the epidural veins are closely related and are dynamically linked. With increased abdominal or intrathoracic pressure (or both), the epidural vein, CSF, and epidural space pressures all increase in parallel. The pressure gradient between the epidural vein and the surrounding space is low — 1 or 2 mmHg — and does not change if abdominal or thoracic pressures are increased.

References


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