Positive lumbar extradural space pressure

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Summary
Specially selected soft Macintosh balloon indicators were attached to needles during five extradural and five spinal punctures. When the needle point entered the extradural space, the mean balloon pressure decreased suddenly from 24.9 (range 14-37) to 12.3 (10-16) mm Hg in the five extradural punctures and from 22.3 (17-28) to 13.7 (10-17) mm Hg in the five spinal punctures. In the five spinal punctures, the balloon pressure did not alter when the needle was advanced from the extradural to the subarachnoid space. Contrary to expectation, none of the balloons deflated when the needle point entered the extradural or subarachnoid spaces. The balloon pressure varied rhythmically in synchrony with respiration and cardiac pulsations. The final balloon pressure, extradural space pressure and subarachnoid pressure were equal. The results suggest that the extradural pressure is positive and of the same magnitude as the prevailing lumbar cerebrospinal fluid pressure. Jugular venous compression, ventilation with carbon dioxide and positive end-expiratory pressure (PEEP) produce a rapid increase in cerebrospinal fluid (CSF) pressure. These stimuli also produced a measurable increase in the lumbar extradural pressure. Jugular venous compression increased the mean lumbar extradural pressure by 6.8 (3-10) mm Hg and ventilation with carbon dioxide increased it by 10 (5-12.5) mm Hg. PEEP values of 5, 10, 15 and 20 cm H₂O produced an immediate increase in extradural pressure of 1-2 mm Hg for every 5 cm H₂O of PEEP. The lumbar extradural pressure increased rapidly with stimuli known to increase CSF pressure. Changes in spinal CSF pressure may be detected by measuring extradural pressure. (Br. J. Anaesth. 1994; 73: 309-314)

Key words
Anaesthetic techniques, extradural.

The author routinely uses Macintosh balloons, made of latex rubber, to identify the extradural space. To identify the extradural space using a Macintosh balloon indicator, a needle is placed in the ligamentum flavum and the stilette removed. The balloon indicator is attached to the needle and inflated by injecting approximately 2 ml of air through its thick stem, using a 25-gauge needle. While the extradural needle is in the tough liga-
inflated with 1–1.5 ml of air. The pressure in the balloon was recorded continuously by attaching a pressure transducer (Statham Gould P50) between the needle and the Macintosh balloon, using a three-way connector and a 200-cm pressure cannula. Pressure signals were amplified (Electromed 4820 amplifier) and results recorded on a pen recorder (Linseis LS51).

A sudden decrease in balloon pressure and pulsation of the balloon indicated entry into the extradural space. When the needle point was in the extradural space, patients’ jugular veins were compressed manually and the effect on balloon pressure recorded.

After the study, a 19-gauge extradural catheter was inserted for extradural anaesthesia.

**PART 2**

Soft Macintosh balloons were attached to spinal needles in five male patients aged 66–84 yr undergoing urological or lower limb surgery under spinal anaesthesia. The puncture was performed at the L2–3 or L3–4 interspace, using a 25-gauge Sprotte needle.

After placing the spinal needle into the ligamentum flavum, a Macintosh balloon was attached to it and inflated with 1–1.5 ml of air. The balloon pressure was recorded continuously as the needle was advanced slowly from the ligamentum flavum, through the extradural space and into the subarachnoid space.

At the end of the study, after demonstrating free flow of cerebrospinal fluid, local anaesthetic was injected for spinal anaesthesia.

**PARTS 3 AND 4**

These studies were carried out in anaesthetized patients undergoing major gynaecological surgery. Their lungs were ventilated with a Manley MP3 ventilator. Under general anaesthesia, an extradural block was established at the L2–3 or L3–4 interspace with 0.25 % bupivacaine 20 ml using a 19-gauge (1.1 mm od) Portex extradural catheter. Monitoring of patients included electrocardiogram, non-invasive arterial pressure and end-tidal carbon dioxide measurements.

The studies were carried out at the end of surgery when at least 1 h had elapsed since the last injection of local anaesthetic into the extradural space. During the measurements of extradural pressure the patients were supine on a horizontal operating table. The extradural catheter was connected to the transducer by a pressure cannula delivering normal saline at a rate of 3 ml h⁻¹. The total amount of saline delivered into the extradural space during the study was less than 1 ml. The extradural pressure transducer was fixed 5 cm above the level of the operating table. Zero extradural pressure was referred to this point, that was assumed to be at the level of the extradural space in supine patients (depth of extradural space being approximately 5 cm from the skin). The total duration of the study was less than 15 min in all patients.

After the study, residual neuromuscular block was antagonized, anaesthesia discontinued and patients sent to the recovery room. Extradural analgesia was continued after operation.

**PART 3**

The effects of jugular venous compression for 20–30 s and ventilation with 5 % carbon dioxide for 1 min on extradural pressure were studied in six patients aged 43–73 yr. Jugular venous compression of 20–30 s and ventilation with carbon dioxide for 1 min were chosen to assess the rapidity with which these stimuli altered the lumbar extradural pressure. The extradural pressure was measured using a calibrated transducer (Statham Gould P50). End-expired carbon dioxide concentration was measured with a Datex Normocap (CD 102) carbon dioxide analyser. The outputs from transducer and carbon dioxide analyser were recorded on the four-channel pen recorder. In all patients the neck was compressed manually (as in the Queckenstedt’s test) and the effect on extradural pressure recorded.

**PART 4**

The effect of PEEP on extradural pressure was studied in six patients aged 40–47 yr. Extradural and airway pressures were measured using transducers (Hewlett Packard Model Nos 78205A and 78206A). To measure airway pressure, an empty pressure cannula was passed through a side hole in the tracheal tube and connected to the transducer. In one patient undergoing extensive pelvic surgery, central venous pressure (CVP) was also measured via an internal jugular cannula.

PEEP, in increments of 5 cm H₂O (4 mm Hg) up to a maximum of 15 cm H₂O (three patients) or 20 cm H₂O (three patients) and lasting for approximately 1 min, was applied using a Manley PEEP valve. The duration of PEEP at each level was limited to approximately 1 min to avoid cardiovascular effects of PEEP. Between each increment, PEEP was returned to zero for 1–2 min to allow any minor cardiovascular effects to return to normal. The output from the pressure transducers was recorded on a multichannel tape recorder (Instrumentation Tape Recorder, Racal Store 7DS). The tape was replayed later and signals recorded on an X–Y plotter (Hewlett Packard X–Y Plotter, Model No. 7047A) for analysis.

To prevent damping of signals, the extradural catheter was flushed with 4–5 drops of saline before the study. Flushing produced a momentary increase in extradural pressure that returned to baseline within 1 min.

**Results**

**PART 1**

The Macintosh balloon pressure decreased suddenly when the needle point entered the extradural space in all patients (figs 1, 2). However, contrary to
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Figure 1  Pressure changes in a Macintosh balloon that did not deflate in the extradural space. Arrow indicates entry into the extradural space, the horizontal bars indicate duration of jugular venous compression. P = pinching to deflate the balloon.

Figure 2  Pressure changes in a Macintosh balloon that did not deflate in the extradural space. Arrow indicates entry into extradural space. P = pinching to deflate the balloon.

Figure 3  Pressure changes in a balloon that did not deflate in the subarachnoid space. X = entry into the extradural space, Y = entry into the subarachnoid space.

Figure 4  Effect of jugular venous compression on extradural pressure and pulse amplitude. Arrows indicate jugular venous compression. The curve should be read from right to left.

expectations, none of the balloons deflated. At the precise moment of entry into the extradural space, the balloons started to pulsate. The pulsations were synchronous with patients' heart beats. With the needle in the ligamentum flavum, the mean balloon pressure was 24.9 mm Hg. It decreased suddenly to 12.3 mm Hg when the needle point entered the extradural space.

The pressure recordings showed two types of periodic waves in the balloons: large and small. The large waves were in phase with positive pressure ventilation, the pressure increasing with inspiration and decreasing with expiration. The smaller waves were superimposed on the larger respiratory waves and were synchronous with arterial pulsations (fig. 1). Compression of the jugular vein produced an immediate increase in the baseline balloon pressure, which lasted until the compression was released (fig. 1).

In four patients in group 1, the pulsating balloons deflated when pinched between the fingers. On release of this pinching pressure, the balloons remained deflated. In the fifth patient, the difference between initial and final balloon pressures was only 5 mm Hg. It was possible to deflate this balloon by pinching, but it re-inflated on releasing pinching pressure. In all patients, transmitted arterial pulsations in the balloons confirmed that the needles were not blocked and were placed correctly in the extradural space (figs 1, 2).

PART 2

Entry of the spinal needle into the extradural space was indicated by a sudden decrease in mean balloon pressure from 22.3 to 13.7 mm Hg and visible pulsations of the balloon. When the needle was advanced from the extradural into the subarachnoid space, the balloon pressure did not alter and the balloon did not deflate (fig. 3). Pressure signals were considerably damped in this group because of the narrow spinal needles and respiratory waves were not seen. In all patients, there was a free flow of CSF from the spinal needle when the balloon was disconnected.

Injection of local anaesthetic produced predicted regional blocks in all patients. There were no adverse effects in any patient.

PARTS 3 AND 4

During the study, the baseline extradural pressure remained relatively constant. Flushing the pressure cannula with 4-5 drops of saline increased the extradural pressure by 3-5 mm Hg, but the baseline was regained within 1 min. Respiratory and cardiac
waves similar to those in part 1 of the study were present in the extradural space.

PART 3

Jugular venous compression produced an immediate increase in lumbar extradural pressure that lasted until the compression was released. The mean increase in extradural pressure was 6.8 (range 3–10) mm Hg. During the increase, the amplitude of the ventilatory waveform transmitted to the extradural pressure remained constant, but the amplitude of cardiogenic oscillations increased considerably (fig. 4).

Ventilation with carbon dioxide produced a mean increase in the extradural pressure of 10 (5–12.5) mm Hg. The increase occurred approximately 1 min after the administration of carbon dioxide, the delay being caused by the wash-in of carbon dioxide through the breathing system. The increase in extradural pressure was associated with an increase in the amplitude of the pulse wave. The extradural pressure decreased rapidly to the baseline value when carbon dioxide administration was discontinued (fig. 5).

PART 4

The extradural pressure increased in parallel with PEEP. The increase followed the ventilatory cycle and occurred immediately after the application of PEEP. The increase in extradural pressure was related directly to the amount of PEEP applied (fig. 6). The response to PEEP varied among patients and ranged between 1 and 2 mm Hg for every 5-cm H₂O increment in PEEP (fig. 7). In one patient
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A pressurized Macintosh balloon normally deflates suddenly when the needle point enters the extradural space. A preliminary study [2] showed that Macintosh balloons exerting a pressure of less than 50 mm Hg may not deflate when the needle point enters the extradural space.

The extradural space is a potential space between the rigid vertebral canal and the dura, and the pressure of the CSF must keep the dura opposed to the vertebral canal. During extradural puncture, the pressure of the advancing needle indents the dura [3, 4] and creates a sub-atmospheric pressure in the extradural space. Identification of the extradural space by “the hanging drop” technique [5] relies on this sub-atmospheric pressure. When the needle is removed, the indented dura should straighten out because of the outward pressure of CSF on the dura and again render the extradural space a potential one.

Injection of fluid into the extradural space must push the dura inwards. The inward movement of the dura would be opposed by the CSF pressure. Therefore, extradural injection requires a force to overcome the outward pressure of CSF. The pressure of air in the soft balloons was insufficient to push the dura inwards and very little air entered the potential extradural space. Therefore, the soft balloons did not deflate in the extradural space.

In part 1 of the study, a small amount of air from the balloon may have entered into the extradural space. The amount of air entering the extradural space should depend on the compliance of the balloon and the resistance of the extradural space. One may speculate that the air is contained in a pocket between the dura and the rigid vertebral canal.

Fluid injected into extradural space may remain under pressure as it is compressed between the dura and the vertebral canal. The fluid would act as a cushion through which the pulsatile waves of the dura and vasculature would be transmitted. The arterial pressure wave in the spinal CSF originates from the choroid plexus [6]. Pulsations of the balloons seen in this study are likely to be the arterial pressure waves of spinal CSF, transmitted through the dura into the extradural space.

In part 2, the balloon pressure decreased suddenly when the needle entered the extradural space. However, there was no further change in the balloon pressure when the needle pierced the dura and entered the subarachnoid space. Therefore, the final balloon pressure must be in equilibrium with the extradural pressure and the spinal CSF pressure; or the extradural and spinal CSF pressures are equal.

As the spinal CSF imparts its pressure to the potential lumbar extradural space, changes in spinal CSF pressure should be reflected in extradural space pressure. Compression of jugular veins increases spinal CSF pressure [7-9] and extradural pressure [10-12]. In this study, compression of jugular veins produced a square wave increase in extradural pressure for the duration of compression. This increase is almost identical to the increase in spinal CSF pressure produced by jugular venous compression [8, 9].

The effect of ventilation with carbon dioxide on lumbar extradural pressure has not been reported previously. Carbon dioxide is known to produce an increase in CSF pressure by cerebral vasodilatation. The vasodilatation induced by carbon dioxide should produce a shift of CSF from the intracranial to the vertebral canal and thus increase the lumbar extradural pressure. Ventilation with 5% carbon dioxide produces an increase in ventricular fluid and intracranial extradural pressures in dogs [13]. In this study, ventilation with 5% carbon dioxide produced an almost identical increase in the lumbar extradural pressure in humans.

Pulsation of the brain is commonly seen during intracranial surgery. The pulsatile vibration of the brain parenchyma derived from the blood flow in the cerebral arteries may have enough energy to generate the spinal CSF pulse [6]. The amplitude of the pulse wave varies directly with intracranial pressure [6]. The amplitude increases when the intracranial pressure is increased by jugular venous compression [8, 9], induced hypercapnia [14] or by injection of fluid into the CSF [15].

An increase in the amplitude of the pulse wave in the extradural space has not been reported previously. This study shows that jugular venous compression and ventilation with carbon dioxide that are known to increase CSF pressure also produce an increase in lumbar extradural pressure. The increase in extradural pressure was associated with an increase in the amplitude of the extradural pulse wave.

Discussion

In whom CVP was also measured, application of PEEP produced an immediate increase in both CVP and extradural pressure. The extradural pressure waveform mimicked the CVP waveform and both increased in parallel with each ventilatory cycle (figs 8, 9). When PEEP was removed, CVP and extradural pressure returned to the pre-PEEP baseline level within 3 or 4 ventilatory cycles.
Mechanisms have been suggested for transmission of abdominal cavity. Blood is forced into the extradural and vertebral veins and veins of the thoraco-pressure [17]. Nerves and loose fatty and areolar tissue into the extradural space and the pleural cavities has been demonstrated [16]. Increased intrathoracic pressure may also push small amounts of venous blood, spinal pressure during coughing [10,11]. Several recordings have shown an increase in extradural pressure via a common mechanism. It is well known that thoracic pressure is transmitted to the extradural space. Macintosh demonstrated this by showing that a balloon connected to the extradural space could be partially reinflated when the patient coughed [1]. Manometric recordings have shown an increase in extradural pressure during coughing [10,11]. Several mechanisms have been suggested for transmission of intrathoracic pressure to the extradural space. The intrathoracic pressure may be transmitted directly to the extradural space through the intervertebral foramina; free communication between the thoracic extradural space and the pleural cavities has been demonstrated [16]. Increased intrathoracic pressure may also push small amounts of venous blood, spinal nerves and loose fatty and areolar tissue into the vertebral canal and thus increase the extradural pressure [17].

There is a rich anastomosis between the extradural and vertebral veins and veins of the thoraco-abdominal cavity. Blood is forced into the extradural veins when pleural and abdominal pressures are increased [18]. The close correlation between the increase in central venous pressure and extradural pressure seen in one patient indicates that PEEP may increase the extradural pressure by impeding venous return and increasing the blood volume in the vertebral and extradural veins.

Spinal CSF pressure increases with increasing amounts of PEEP [19]. In this study PEEP produced a similar increase in extradural pressure. This study did not elucidate if the increase in lumbar extradural pressure during PEEP ventilation was caused by an increase in spinal CSF pressure or by an increase in extradural venous blood volume. It is likely that PEEP increased the spinal CSF pressure and the lumbar extradural pressure via a common mechanism.

The observations in part 2 of this study demonstrated that the lumbar extradural pressure was in equilibrium with the prevailing spinal CSF pressure [10]. It is suggested that the lumbar extradural pressure is positive and derived from the spinal CSF pressure. The spinal dural sac acts as a movable membrane and pressure on its opposing sides is rapidly equilibrated [10]. Thus changes in spinal CSF pressure may be detected by the less invasive measurement of lumbar extradural pressure.

It is concluded that the soft balloons did not deflate in the extradural or subarachnoid spaces because the final balloon pressure was in equilibrium with the extradural and subarachnoid pressures. The pulsations in the balloons were derived from the pulse waves in the spinal CSF. The extradural space pressure was positive and of the same magnitude as the prevailing spinal CSF pressure.

References
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