Thoracic epidural anaesthesia and analgesia

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Thoracic epidural anaesthesia followed by postoperative epidural analgesia is increasingly being used for abdominal, major vascular and cardiothoracic surgery. The objective of thoracic block is not solely to block noxious afferent stimuli from the surgical site, but to impart a bilateral selective thoracic sympathectomy. Provision of pain relief and sympatholysis of such magnitude allows patients to cough, breathe deeply, drink and mobilise can contribute to enhanced postoperative outcomes such as improved respiratory function, reduction in ileus and protein sparing. The challenge is how to translate the encouraging results of randomised controlled studies and meta-analyses into clinical practice. Provision of sufficient intraoperative epidural analgesia such that the patient awakes pain-free and maintenance of a continuous bilateral sensory block covering the entire surgical site for several days is not an easy task. The aim of this review is to discuss the practical issues related to thoracic epidural analgesia.

Anatomy

Detailed knowledge of the differences between thoracic and lumbar anatomy is a prerequisite for successful thoracic epidural block. The kyphotic thoracic spine consists of 12 vertebral bodies forming, with the posterior longitudinal ligament, the anterior aspect of the neural arch. The anterolateral border is formed by the pedicles and the posterolateral border by laminae and ligamentum flavum. The spinous processes are aligned steeply in the high- and mid-thoracic regions but become less acutely inclined in the low-thoracic region.

Investigation of the anatomy and dynamics of the epidural space with techniques such as epiduroscopy have been supplanted by cryomicrotome sectioning, whereby, shortly after death, a cadaver is frozen and spine dissected in planes 100 µm thick. Artefact is minimized by immediate freezing, which allows relationships between structures to be readily identified. From this work, the lumbar epidural space has been anatomically described as segmented and discontinuous. This is attributable to dura being directly in contact with the lumbar bony canal and the predominance of lumbar epidural fat. In contrast, the thoracic epidural space contains less fat, thoracic dura is less adherent to the surrounding bony canal and the ligamentum flavum are less likely to meet in the midline. The anterior thoracic epidural space is filled predominantly with valveless veins, which connect to the basivertebral venous plexus and azygos vein.

After injection, local anaesthetic spreads as rivulets through many small channels not unlike fluid spreading between two sheets of polythene. The greater the volume of injectate, the more homogenous the spread as channels open up. Solution travels preferentially towards the nerve roots and through the intervertebral foramina, with some restriction exerted by the fascia of the posterior longitudinal ligament. Although cryomicrotome sectioning is unable to demonstrate any evidence of a dorsomedian fold nor fibrous tissue, posterior midline fat may still impede spread of local anaesthetic. Thus, it would seem that unilateral block is more likely a result of poor technique, inadequate volume or passage of the epidural catheter through an intervertebral foramen.

Nerve roots also differ in size between thoracic and lumbar regions. The diameter of thoracic roots is half that of lumbar roots, thus encouraging nerve block of the former, although much variability in root sizes occurs between individuals. Nerve roots are surrounded by arachnoid on entering the epidural space and separate into multiple fascicles passing to the dorsal root ganglion.

Sympathetic preganglionic fibres originate in the intermedio-dorsal cell column of the spinal cord and leave via nerve roots from T1 to L2. Although innervation of the heart is principally via sympathetic fibres from T1 to T4, other less direct pathways to and from the heart could still remain, as measurements of heart rate variability during high epidural thoracic anaesthesia suggest that complete cardiac sympathetic block does not always occur.

Key points
The aim of a thoracic epidural is to selectively block pain fibres from the surgical site and the thoracic sympathetic chain bilaterally. Selective sympathetic block is associated with respiratory, cardiac, gastrointestinal and metabolic benefits. Unilateral block is more likely to be caused by catheter malposition than anatomical barriers to spread. Lumbar epidural block is inappropriate for abdominal and thoracic surgery. Freedom from pain allows early mobilization and feeding.
Identification of epidural space

Although the decision to perform regional anaesthesia on a patient under general anaesthesia remains contentious, our preference is to perform thoracic epidural anaesthesia while the patient is lightly sedated. Insertion of a thoracic epidural in the awake patient has two principal benefits: lancinating pain warns the anaesthetist of any potential neurological damage; and the extent of sensory analgesia can be measured before inducing general anaesthesia.

Examination of the patient’s back, identification of bony landmarks and use of aseptic technique are essential before embarking on thoracic epidural block. Classical landmarks are root of spine of scapula at T3 and the inferior angle of scapula at T7. We always count up interspace from L3/4 as a check.

Two approaches exist to the thoracic epidural space – midline or paramedian. The midline approach is no more difficult in the low-thoracic region than lumbar epidurals because of the similar angulation of the spinous processes. However, in the mid- and high-thoracic regions, extreme upward angulation of the Tuohy needle directed through a small space makes insertion more difficult in the midline. Our preference is to use the paramedian approach, as the bony lamina of the vertebra below acts as a depth finder and there is a definite ‘rubbery’ feel as the Tuohy needle passes from bony lamina to ligamentum flavum.

The inferior tip of the spinous process corresponding to the vertebra above should be palpated and, 1 cm lateral to this point, local anaesthetic injected both to the skin and lamina of vertebral body below. The approach of the needle is about 15 degrees to the midline and 60–65 degrees from the coronal plane.

The thoracic epidural space is identified by two methods – loss of resistance to saline/air or hanging drop. Use of saline is associated with a reduced dural puncture rate and avoids the rare complications of venous air embolism and pneumocephalus. A popular misconception is that the pressure of the epidural space is negative. In fact, it is slightly positive, but large negative pressures are induced by tenting of the epidural space from the Tuohy needle and account for the rapid inward entry of saline using the hanging-drop method.

Combined epidural and general anaesthesia

Dermatomal sensory spread of local anaesthetics varies according to the site of injection. As illustrated in Figure 1, high-thoracic epidurals have minimal cranial but marked caudal spread. Conversely, more cranial spread occurs after low-thoracic epidurals than after high- and mid-thoracic epidurals. This phenomenon is attributable to a progressive increase in the width of the epidural space from 1–1.5 mm at C5 (as a result of cervical cord expansion) to 2.5–3 mm at T6 and 5–6 mm at L2. Thus, insertion of a low-thoracic epidural should be at a level corresponding to the midline of the surgical incision, whereas a high-thoracic epidural should be inserted at a relatively more cranial point in respect to the incision. Spread is also more extensive in the elderly and with higher volumes of injectate, although the relationship between volume

is not linear, as a stepwise increase in volume results in a relatively small increase in spread (Fig. 2). Higher concentrations of bupivacaine (0.75%) or its equivalent are preferred, as onset of sensory block is rapid and muscle relaxation is profound, thus reducing the need for intraoperative neuromuscular blocking agents.

Induction of general anaesthesia after the establishment of thoracic epidural anaesthesia can precipitate hypotension, particularly with propofol. Hypovolaemia after fasting, bowel preparation for surgery or both will accentuate any reduction in blood pressure. Therefore, our practice is to restore blood volume with intravenous fluids and establish general anaesthesia with intravenous etomidate under direct arterial pressure monitoring. When anaesthetized, fluids can be titrated according to central venous pressure or oesophageal Doppler monitor. Epidural top-ups of 3–5 ml bupivacaine 0.75% or equivalent are administered judiciously throughout the operation and on wound closure. Alternatively, a constant infusion of epidural solution can be run during the operation in order to minimize fluctuations in blood pressure. Little evidence exists regarding intraoperative management of epidural anaesthesia.
Sympathetic block

Selective sympathetic block is an integral part of thoracic epidural anaesthesia and analgesia. The recognition of the benefits and risks of sympathectomy with regard to cardiac, metabolic and gastrointestinal function is necessary in order to optimally manage patients during and after major surgery.

Cardiovascular

Sympathetic activation associated with surgery and postoperative pain manifests as tachycardia, hypertension and increased contractility, all of which serve to increase myocardial oxygen consumption. However, the response of patients with coronary atherosclerosis to surgical stress differs from that of healthy patients. Sympathetic stimulation may constrict post-stenotic coronary arteries and reduce blood supply to the subendocardium. The uncoupling of oxygen delivery and demand presents as postoperative silent myocardial ischaemia, the intensity of which predicts long-term severe myocardial events. Provision of a selective sympathectomy using thoracic anaesthesia in patients at risk of perioperative ischaemia has the potential to dilate constricted coronary vessels, reduce heart rate and improve cardiac function by reducing preload and afterload and optimizing myocardial oxygen delivery.

Metabolic

Muscle wasting occurs after surgery because the rate of muscle protein breakdown exceeds the rate of synthesis. Amino acids provide the substrate for gluconeogenesis in the liver, resulting in an elevation in plasma glucose concentrations and increased insulin resistance. Thoracic sympathetic block of adrenal catecholamine release restores the balance between muscle synthesis and breakdown, reducing the increase in plasma glucose.

Gastrointestinal

The sympathectomy of thoracic epidural anaesthesia has been shown to benefit bowel function by reducing the duration of postoperative ileus, enhancing bowel blood flow and preventing reductions in gastric intramucosal pH in patients undergoing major abdominal surgery. The increase in bowel motility from unopposed parasympathetic activity is not associated with any significant increase in anastomotic dehiscence.

However, splanchnic block may also have deleterious haemodynamic consequences, as the splanchnic veins contribute substantially to the control of overall venous capacitance. Hence, sympathetic block during thoracic epidural anaesthesia vasodilates mesenteric vessels and is associated with hypotension because of reduced venous return. The degree to which blood pressure decreases is dependent on the relative extent of splanchnic sympathetic block and the degree of baroreceptor-induced vasoconstriction in unblocked regions of the body.

Hypotension can compromise gut mucosal integrity and myocardial blood flow. Translocation of endotoxin from the gut lumen and release of inflammatory mediators is the basis of the systemic inflammatory response leading to increases in capillary permeability and multi-organ failure. Therefore, it is important to prevent gut hypoperfusion.

The extent to which splanchic blood flow is flow- or pressure-dependent has been recently examined by measuring inferior mesenteric artery flow and serosal flux using laser Doppler imaging. From this work, it would appear that correction of mean perfusion pressure with inotropes is more likely to correct bowel blood flow than excessive fluid resuscitation. Use of ephedrine (mixed α- and β-agonist) or metaraminol (α-agonist) in small aliquots is often used to correct hypotension. A low-dose infusion of dopamine or dopexamine at 3–5 μg kg⁻¹ min⁻¹ may also be infused to increase mesenteric and hepatic blood flow.

Thoracic vs lumbar epidurals

Difficulties associated with needle insertion, uncertain and imprecise placement of catheters (particularly in the high- and mid-thoracic epidural space), persistent perioperative hypotension and a myriad of possible neurological problems may well be off-putting to the wary anaesthetist faced with an ill patient undergoing upper abdominal surgery. Insertion of a lumbar epidural catheter and upward extension of the block may be viewed as an easy compromise. Unfortunately, the consequences on patient outcome may well be worse than with a well-managed thoracic epidural.

Lumbar sensory block for abdominal surgery is difficult to maintain, rescue analgesia is required more often and motor block is inevitable. As sympathetic block is extended to the lower limbs, baroreceptor-mediated reflex vasoconstriction is limited to areas cephalad to the block, increasing the likelihood of coronary vasoconstriction and myocardial ischaemia. Furthermore, after large blood loss, decreases in mean arterial pressure, systemic vascular resistance and base excess are significantly larger in the presence of extensive thoracolumbar block than with selective thoracic block or general anaesthesia alone. The Bezold–Jarisch reflex, characterized by bradycardia, vasodilation and hypotension, is also more common with extensive lumbar epidural blocks. Only by restricting spread to the lumbar and low-thoracic regions can lumbar epidural block restrict splanchic sympathetic block, maintain venous return and lessen hypotension. Therefore, the evidence suggests that lumbar epidural anaesthesia should be avoided in patients undergoing abdominal or thoracic procedures.

Postoperative analgesia

The aim of thoracic postoperative analgesia is to provide optimal analgesia and minimal side-effects for several days without recourse to rescue medication. Sufficient freedom from pain to allow deep breathing and coughing without restriction goes some way towards attenuating the incidence of hypoxaemic episodes.
and pulmonary complications experienced after surgery. But is that sufficient to improve patient outcome? Opportunities provided by the pain-free state should be taken to remove drains and catheters, patients should be encouraged to mobilize and feeding should be started as soon as possible. Keeping patients pain-free requires that drug(s) administered to the epidural space are of sufficient concentration and volume that afferent input from the entire surgical field is blocked not just before incision and during surgery but for an indeterminate length of time so that when the epidural is stopped, wounds are healing, the intensity of pain is much less and the chances of fuelling the flames of central sensitization have subsided.

Many comparisons have been made of local anaesthetic alone, opioid alone or the combination. Overall findings suggest that the combination of local anaesthetic and opioid provides best analgesia on movement, has less hypotension than with local anaesthetic alone and halves the duration of ileus compared with epidural opioid alone or patient-controlled analgesia (PCA). Our combination of choice is bupivacaine 0.1% and diamorphine 25 µg ml⁻¹ at a variable nurse-controlled rate of 8–20 ml h⁻¹.

Knowledge of the optimal mix of local anaesthetic and opioid would improve pain relief and minimize side-effects associated with both drugs. One study has so far attempted this in 190 patients receiving thoracic epidural analgesia for 48 h after major abdominal surgery. A stepwise optimization model investigated combinations of bupivacaine, fentanyl and infusion rate until there was the best balance of analgesia and side-effects. The optimal combinations were (i) bupivacaine 8 mg h⁻¹ plus fentanyl 30 µg h⁻¹ at an infusion rate of 9 ml h⁻¹ and (ii) bupivacaine 13 mg h⁻¹ plus fentanyl 25 µg h⁻¹ at an infusion rate of 9 ml h⁻¹.

Alternative adjuvants include clonidine and epinephrine. Clonidine is an α-2 agonist associated with hypotension and sedation. Using the same optimization model, the best dose of clonidine was 5 µg h⁻¹ when combined with bupivacaine 9 mg h⁻¹ and fentanyl 21 µg h⁻¹ at an infusion rate of 9 ml h⁻¹. Addition of epinephrine to mixtures of local anaesthetic and fentanyl improves pain relief (probably by a spinal cord α-2 agonist effect), attenuates sensory block regression and reduces plasma fentanyl concentrations.

**Administration**

Continuous infusion, despite being the most popular means of administration, is associated with sensory block regression, particularly with local anaesthetic alone. Addition of opioid increases the time to first analgesic rescue. The continued popularity of infusion stems from the perception that it is has less cardiovascular and respiratory side-effects than with bolus alone.

Patient-controlled epidural analgesia, usually with a background infusion, allows patient self-titration and sparing of local anaesthetic consumption. However, this technique is dependent on an awake, cooperative patient and is not suitable for patients sedated after general anaesthesia. Furthermore, the technique is dependent on the perception of pain, thus allowing novocain stimuli into the spinal cord.

Continuous bolus administration at fixed time intervals has been shown to minimize block regression when compared with the same concentration of local anaesthetic given as an infusion. Although recent work with local anaesthetic opioid showed similar results for obstetric analgesia, this methodology is still to be applied to major abdominal surgery.

**Side-effects**

Compared with lumbar epidurals or PCA, thoracic epidural anaesthesia confers many advantages to patients. However, do the feared neurological complications of thoracic epidurals tip the balance of benefits vs risks towards less invasive methods of pain relief?

One of the most thorough studies of neurological damage after thoracic epidural analgesia complications has been conducted by Giebler and colleagues. No permanent neurological sequelae were reported in 4,185 patients, although unsuccessful catheter placement occurred in 1 in 93 patients and self-limiting peripheral nerve lesions in 1 in 174 patients. The incidence of dural tap was 1 in 140 patients but was less common at higher insertion sites.

Epidural management should be heavily protocol-driven by trained nurses performing hourly recordings in order to detect the early signs of permanent neurological damage such as epidural haematoma or abscess and immediate life-threatening events such as respiratory and cardiovascular depression. The daily problems encountered while managing an epidural service limit the potential benefits and are highlighted by the continuous audit of thoracic epidurals conducted in our department since 1993. More than one-fifth of patients still experience severe pain and hypotension, particularly in the recovery area soon after surgery, technical failure is not uncommon and 1 in 8 patients still have their epidurals removed at night by ward staff because of pressure on beds. The response to technical failure is often to resort to PCA, exposing the patient to inferior pain relief and the side-effects of morphine. Lack of sufficiently trained staff, not high-dependency beds, prevents expansion of epidural services.

**Key references**


See multiple choice questions 16–19.