of epidural anaesthesia is controversial. We would like to share our experience of a patient with increased pulmonary artery pressure. Consent from the patient has been obtained.

A 59-yr-old lady with a BMI of 46 and past medical history of asthma, type 2 diabetes, hypertension, arthritis, bilateral lymphoedema, and raised pulmonary artery pressure underwent laparoscopic-assisted left adrenalectomy for a non-functioning adrenal tumour. She had two previous uneventful general anaesthetics. Preoperative ECG showed sinus rhythm, normal axis with right ventricular strain pattern. Transthoracic cardiac ECHO was reported as, moderately dilated right heart with at least moderate systolic impairment, estimated elevated systolic pulmonary arterial pressure of 68 mm Hg and preserved left ventricular systolic function.

A thoracic epidural was inserted awake with full asepsis at the T6–7 level using a 16 G Tuohy needle with ease. A test dose of 0.25% bupivacaine 3 + 7 ml was given to ensure correct epidural position. General anaesthesia was commenced with remifentanil infusion, propofol, and atracurium. The trachea was intubated with an 8.0 COETT and anaesthesia maintained with remifentanil and isoflurane. The right radial artery and right internal jugular vein were cannulated. The patient was haemodynamically stable with normal arterial blood gases throughout the procedure. Four hours later, towards the end of procedure, an epidural bolus of 10 ml of 0.25% bupivacaine was given and neuromuscular block reversed with neostigmine and glycopyrrrolate.

The patient became bradycardic, hypotensive with subsequent asystolic cardiac arrest. One cycle of cardiopulmonary resuscitation was performed; adrenaline 1 mg and atropine 0.6 mg were given after which the patient had a return of spontaneous circulation. The arterial pressure was low, 72/33 mm Hg. There was a frank pulmonary oedema with decreased arterial oxygen saturation of 71% on ventilation with 100% oxygen. Noradrenaline and adrenaline infusion was commenced. The arterial pressure and oxygen saturation gradually improved.

Transthoracic cardiac ECHO was performed by the consultant cardiologist. ECHO showed dilated and impaired right ventricle with evidence of pulmonary hypertension, a good left ventricle, and no evidence of ventricular septal defect or significant mitral regurgitation. The ECG was reported as sinus rhythm with left axis deviation and intraventricular conduction defect. The patient was transferred to ITU; CTPA revealed no pulmonary embolism. Postoperative troponin-I was 0.45 ng ml⁻¹. The patient was ventilated for about 2 days, extubated without any neurological deficit, discharged from intensive care and from the hospital subsequently. The patient is being followed up by the cardiologist and pulmonary hypertension physician.

The safety of epidural anaesthesia in patients at risk of right ventricular pressure overload remains controversial. It has been showed in an animal model that thoracic epidural anaesthesia reduced the haemodynamic tolerance to acute increase in pulmonary artery pressure by inhibiting the right ventricular positive inotropic response. In our patient, we think that the bolus dose of local anaesthetic epidurally would have impaired the cardiac sympathetic response leading to right ventricular failure and subsequently to left ventricular failure, thereby causing haemodynamic collapse and cardiac arrest.

The clinical relevance of this observation is not an argument against the use of thoracic epidural analgesia. Instead, it suggests that patients with raised pulmonary artery pressure with right ventricular pressure overload may be at increased risk of cardiovascular collapse when neuroaxial anaesthesia extends to the cardiac sympathetic nerves. Awareness about this potential interaction could help clinicians optimize the application of epidural anaesthesia in these groups of patients. Although the patient tolerated the epidural bolus of local anaesthetic when awake, subsequent bolus under anaesthesia led to haemodynamic collapse and cardiac arrest. It may be that a bolus given after operation or by infusion rather than intraoperatively would have been better tolerated by the patient.

Conflict of interest

None declared.

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4 Santamore WP, Dell’Italia LJ. Ventricular interdependence: significant left ventricular contributions to right ventricular systolic function. Prog Cardiovasc Dis 1998; 40: 289–308

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Decompensation of undiagnosed spinal dural arteriovenous fistulae after lumbar epidural injection and spinal anaesthesia

Editor—Spinal dural arteriovenous fistulae (SDAVF) are vascular malformations present in the dura mater of the spinal cord that result in arteriovenous shunting.
Although rare, they account for 80% of all spinal vascular abnormalities. The pathophysiology of neurological deficit secondary to SDAVF is thought to involve venous hypertension in the spinal cord, progressing to venous congestion, ischaemia, and infarction. Spinal angiography is the gold standard investigation for SDAVF, although MRI, often with hindsight, may demonstrate increased cord oedema, flow voids, and medullary vein enlargement. Treatment aims to relieve symptomatology by occluding the primary draining vein and defunctioning the fistula via endovascular embolization or open laminectomy with fistula division.

We report on three patients where lumbar epidural injection (Cases 1 and 2) and spinal anaesthesia (Case 3) triggered acute decompensation of underlying SDAVFs. This phenomenon has been postulated secondary to lumbar epidurals but not following spinal anaesthesia. Other known precipitants include Valsalva-related activity and lumbar puncture.

**Case 1**

A 78-yr-old male presented with an insidious onset back pain and leg weakness. A clinical diagnosis of lumbar canal stenosis was made, apparently confirmed by lumbar MRI, which showed a disc protrusion and canal stenosis at L3/4 (Fig. 1A). He developed temporary left leg weakness, bladder dysfunction, and saddle paraesthesia 12 h post day-case epidural injection (for symptom relief; L3/4, 16 G, 5 cm depth, 8 ml 1% lidocaine, 80 mg triamcinolone). Three months, and a repeat MRI (again reported as lumbar stenosis) later, his mobility had deteriorated to 5 m with a frame. Review of his previous MRI raised the possibility of a dural fistula and a further MRI and spinal angiography were performed (Fig. 1B and C), confirming an SDAVF. The patient underwent thoracic hemi-laminectomy with surgical disconnection of the SDAVF.

**Case 2**

A 41-yr-old male presented with an 8 month history of lower back pain with intermittent left leg weakness and
paraesthesiae. Lumbar MRI showed L4/S and L5/S1 degenerative disease and canal narrowing. He developed bilateral lower limb weakness and altered sensation 24 h post-epidural injection (for symptom relief; L3/4, 5 cm depth, 7 ml 1% lidocaine, 80 mg triamcinolone). Symptoms persisted and repeat MRI (Fig. 1e) and spinal angiography confirmed the SDAVF. Embolization resulted in significant symptom reduction.

Case 3

A 54-yr-old male, with no pre-existing neurological symptoms, underwent transurethral resection of prostate under spinal anaesthesia (L2/3, 25 G catheter, 3.5 ml bolus 0.5% bupivacaine). He developed right leg weakness and paraesthesia 24 h post-procedure. Urgent lumbo-sacral MRI showed no spinal haematoma but demonstrated a congenitally tethered spinal cord, significant oedema, and a cyst-like area within the cord at L3/4 (Fig. 1e). Spinal angiography confirmed an S3 dural fistula. Symptoms did not improve with conservative management and 6 months post-TURP, he underwent an L5/S1 laminectomy with division of sacral fistula.

The onset of neurological symptoms post-procedure in all three cases strongly suggests a causal effect, especially as MRI demonstrated no acute pathology such as epidural haematomas. In Case 1, SDAVF was diagnosed 15 weeks post-epidural injection. Persistence of symptoms prompted a re-evaluation of previous imaging, culminating in diagnosis. Cases 2 and 3 were diagnosed within 1 week due to earlier consideration of SDAVF. Outcome in all three cases was favourable, but evidence shows that diagnostic delay is associated with increasing morbidity.

Acute or subacute neurological deterioration after epidural corticosteroid injection or spinal anaesthesia should prompt consideration of an underlying SDAVF after procedural complications have been excluded. MRIs should be scrutinized for cord expansion and flow voids on the dorsum of the cord. If an SDAVF is suspected, additional imaging in the form of spinal angiography is indicated. Early recognition and review of imaging may prevent morbidity associated with SDAVF.

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Caesarean section and brain tumour resection

Editor—Life-threatening neurological disorders caused by space-occupying tumours are rare during pregnancy. We report on a 27 yr old woman, height 1.69 m, weight 64 kg, in the 34th week of her third pregnancy. She suffered from bifrontal headache since the beginning of pregnancy. Five weeks before hospital admission, the headache shifted towards the upper cervical spine and she became sensitive to light and noise. Nausea and vomiting occurred and movement became ataxic. A magnetic resonance imaging scan showed a 5 × 4 × 4.6 cm space-occupying median infratentorial tumour compressing the fourth ventricle, with hydrocephalus and shift of the median line. The cerebellar tonsils were herniated into the foramen magnum.

It was agreed that Caesarean section and neurosurgical resection of the tumour should be done during the same anaesthetic.

The preoperative assessment was essentially normal. She was positioned left side down and Ringer solution 500 ml was infused. During preoxygenation, she had a generalized epileptic seizure. Anaesthesia was induced with fentanyl 0.5 mg, thiopental 400 mg, and succinylcholine 100 mg. Sellick’s manoeuvre was used to prevent regurgitation. Intubation was successful at the first attempt. No antiepileptic drug was administered.

The baby was delivered without any operative complication and the umbilical cord was cut 150 s after intubation with an Apgar score of 6/10/10 (weight 2380 g). Oxygen was administered by mask ventilation initially. Naloxone 0.03 mg was given i.v. after 4 min, resulting in crying and adequate respiration within 1 min. Glucose (10%) 6 ml h⁻¹ and caffeine citrate 40 mg were administered i.v. Oxygen 1.5 litre min⁻¹ was applied via nasal cannula. A further dose of naloxone 0.03 mg was given 15 min later. The newborn was supported by nasal continuous positive airway pressure breathing without additional oxygen for 3 days for respiratory distress.