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CASE REPORT

Non-invasive ventilation in the treatment of ventilatory failure following corrective spinal surgery

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Summary

Non-invasive positive pressure ventilation has previously been used successfully to treat both acute and chronic ventilatory failure secondary to a number of conditions, including scoliosis. We report two patients in whom it was used, on three separate occasions, to treat acute ventilatory failure following corrective spinal surgery. Non-invasive positive pressure ventilation may be useful postoperatively in high-risk patients undergoing major spinal surgery in an attempt to prevent intubation and its attendant complications.

Keywords *Scoliosis*; corrective surgery. *Ventilatory failure*; non-invasive ventilation.

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Non-invasive positive pressure ventilation (NIPPV) has been used in the treatment of both acute and chronic ventilatory failure secondary to a wide number of conditions, including chronic obstructive pulmonary disease (COPD) [1], neuromuscular disease [2], thoracoplasty and scoliosis [3]. When NIPPV is used in chronic ventilatory failure due to scoliosis, the benefits include improved arterial blood gases, improved quality of life and decreased hospital stay [4, 5]. Approximately 80% of patients are alive on treatment 5 years after the initiation of non-invasive ventilation [4, 5]. A number of studies have included small numbers of patients in whom NIPPV has been used to treat acute or chronic ventilatory failure secondary to scoliosis [6].

Acute ventilatory failure can develop following corrective surgery for scoliosis, especially in those patients with poor pre-operative pulmonary function. Surgical intervention may be deemed contraindicated for patients with extremely limited respiratory reserve. We describe two cases of ventilatory failure following corrective surgery, in whom treatment with NIPPV obviated the need for intubation and conventional mechanical ventilation.

Case history 1

An 11-year-old boy with severe congenital scoliosis, first noted when aged 6 months, was admitted for corrective spinal surgery. At the age of 4 years, he had undergone anterior disectomies and excision of the vertebral end plates of the mid-thoracic spine. Unfortunately, this attempt to arrest spinal growth did not succeed and his scolotic deformity progressed. He was otherwise well and was on no medication.

Pre-operatively, his main complaints were of back ache and exertional fatigue. Occasionally he experienced breathlessness on exertion but was able to play football. Examination revealed no signs of right heart failure or pulmonary hypertension and no neurological abnormality. His ECG and overnight oximetry were normal; his chest

Table 1 The pre-operative pulmonary function and arterial blood gases of each patient. FEV 1: forced expiratory volume in 1 s; FVC: forced vital capacity; Dlco: transfer factor for carbon monoxide; Kco: transfer coefficient.

	Patient 1	Patient 2
FEV 1; l (%)	0.55 (27)	0.98 (27)
FVC; l (%)	0.55 (25)	1.12 (30)
FEV 1/FVC ratio	100	88
Dlco; mmol.min^{-1}·kPa^{-1} (%)	1.6 (32)	3.6 (40)
Kco; mmol.min^{-1}·l^{-1}·kPa^{-1}	2.91	2.12
Analysis of blood gases on air		
pH	7.415	7.432
P_aCO_2; kPa	5.13	5
P_aO_2; kPa	13.7	11.33
HCO_3^-; mmol.l^{-1}	25	24.6

X-ray showed no parenchymal disease. Pre-operative lung function and arterial blood gases are shown in Table 1.

He underwent the first stage of a Leatherman anterior wedge resection, which consists of an anterior transthoracic excision of a congenital thoracic hemivertebra via a rib-excising thoracotomy. He was electively ventilated via a tracheal tube on the intensive care unit (ICU) for 6 h postoperatively. He was extubated and observed overnight before transfer to a high-dependency unit (HDU) facility the next day, at which time oxygen saturation was 96% on 40% oxygen by a face mask and his arterial carbon dioxide pressure (P_aCO_2) was 6.3 kPa. Over the next 2 days his arterial blood gases and conscious level deteriorated despite physiotherapy, attempts to increase his oxygen tension (F_iO_2) and good pain control with minimal use of opiates. There was no radiological evidence of a pneumothorax, effusion or consolidation to explain the deterioration. His respiratory rate rose to 50 breath.min^{-1} and arterial blood gas analysis showed a pH of 7.34, P_aCO_2 8.9 kPa, P_aO_2 21 kPa on 40% oxygen. NIPPV was applied using a pressure-cycled ventilator (BiPAP, Respironics Inc., Murrysville, Pennsylvania, USA) and a nasal mask. The inspiratory positive airway pressure (IPAP) was set at 12 cmH_2O and expiratory positive airway pressure (EPAP) at 4 cmH_2O with 0.5–1 l.min^{-1} of oxygen entrained to maintain his oxygen saturation above 90%. This rapidly improved alveolar ventilation and conscious level, with blood gases 4 h later showing pH 7.38, P_aCO_2 7.3 kPa, P_aO_2 16 kPa whilst on NIPPV (Fig. 1). Non-invasive ventilation was continued intermittently over the next 3 days. The patient's respiratory rate decreased to between 20 and 30 breath.min^{-1} and his blood gases improved further. NIPPV was stopped when the P_aCO_2 was in the normal range and oxygen saturation was at least 95% on an F_iO_2 of 28%. He continued to improve and underwent the second stage of the surgical procedure 4 weeks later (posterior hemivertebra excision and instrumented fusion). Postoperatively, he developed upper motor neurone signs consisting of spastic paraparesis with clonus and hyperreflexia. Following urgent surgery to revise the posterior instrumentation, he made a complete neurological recovery. He was then able to be extubated without the need for further ventilatory support.

Case history 2

An 18-year-old woman with a severe scoliotic deformity, first noted when she was 12 years old, was admitted for

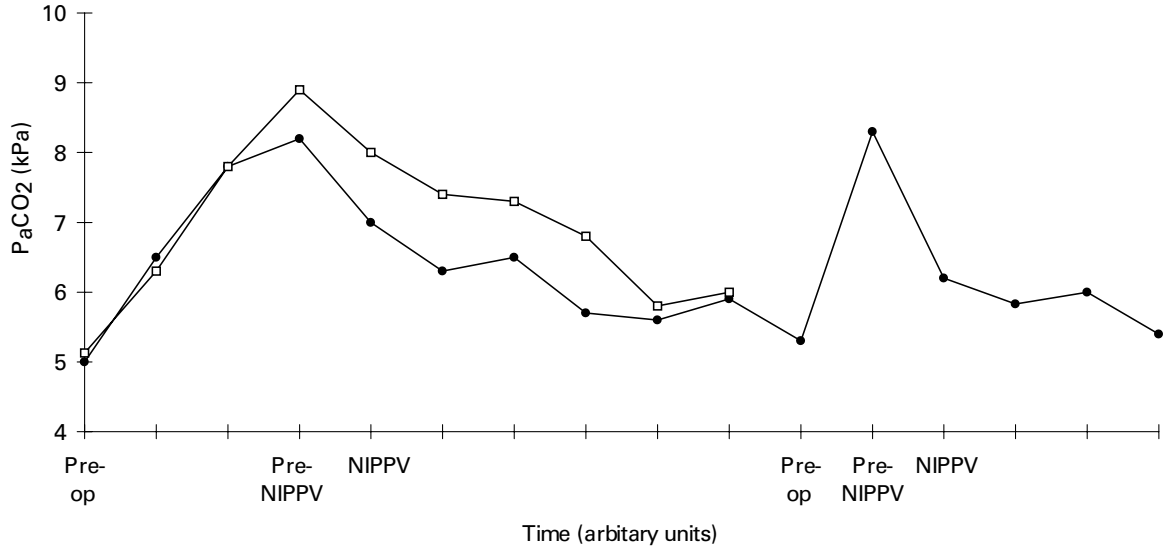

Figure 1 How P_aCO_2 for each patient varied over time and with NIPPV (squares: patient 1, circles: patient 2).

corrective spinal surgery. She also had an Ebstein's anomaly, which is a combination of displacement of an abnormal tricuspid valve downwards into the right ventricle and thinning of the right ventricular wall. This condition is often associated with other abnormalities such as an atrial septal defect or, as in this case, pulmonary stenosis. Her cardiac condition was treated in infancy, when she underwent an open pulmonary valvotomy and a right ventricular outflow tract resection. Asthma had been diagnosed 2 years prior to her spinal surgery. Her regular medication included inhaled budesonide 200 μg twice daily and beta-agonists as required.

She complained of breathlessness and wheeze on exertion, which was helped by inhalers. This breathlessness was thought to be due to a combination of asthma and her chest wall deformity. Pre-operatively, she had a severe scoliosis, a loud pansystolic murmur and a split second heart sound. Her respiratory rate was 20 breath.min^{-1} and there was no audible wheeze. Her ECG showed long-standing right bundle branch block. Echocardiography revealed considerable right ventricular dilation; the tricuspid valve was displaced towards the right ventricular apex and was markedly regurgitant. There was moderate pulmonary regurgitation but left ventricular function was preserved. Pre-operative lung function and arterial blood gases are shown in Table 1.

She underwent anterior transthoracic discectomies and end plate excisions from $T_{4/5}$ to $T_{8/9}$ inclusive via a right 6th rib-excising thoracotomy. Two chest drains and a paravertebral, subpleural catheter were inserted. Post-operatively whilst on a surgical HDU, she became increasingly breathless and her oxygen saturation decreased. Two days postoperatively, her respiratory rate was 40 breath.min^{-1}; there was no wheeze on auscultation nor was there any evidence radiologically of consolidation, a pneumothorax or an effusion. Arterial blood gas tensions on 50% oxygen revealed pH 7.34, P_aCO_2 7.0 kPa and P_aO_2 17 kPa. At this stage she was already on regular nebulised bronchodilators and opiates had been reduced to the lowest level consistent with good pain relief. Non-invasive ventilation was initiated using a pressure-cycled ventilator (NIPPY II, Friday Electronics Ltd, London, UK) with a full-face mask. Ventilator settings were IPAP 10 cmH_2O, EPAP 4 cmH_2O, inspiratory time 0.8 s and expiratory time 2.2 s with 3 l.min^{-1} of oxygen entrained.

Her dyspnoea, respiratory rate and blood gases all improved rapidly (Fig. 1) and she was weaned from NIPPV over the next 3 days. Two weeks later she underwent the second stage of the procedure (posterior Harrington Instrumentation and fusion of thoracic spine with intra-operative scoliosis correction) and again developed postoperative ventilatory failure (pH 7.21, P_aCO_2 8.2 kPa, P_aO_2 15 kPa on 50% oxygen). A second episode of treatment with NIPPV resulted in a rapid improvement in her clinical condition and blood gases. She was discharged from hospital 2 weeks after her second operation.

Discussion

We describe two cases in which NIPPV was used successfully to treat ventilatory failure following corrective surgery for severe scoliosis. In both cases (three separate episodes), the need to return to ICU for intubation and conventional mechanical ventilation was avoided. The increased costs and possible complications of intubation and conventional ventilation, including nosocomial pneumonia [7], were thus avoided.

The mechanisms involved in postoperative ventilatory failure are likely to be multifactorial and include atelectasis, the respiratory depressant effects of opiates, infection, pain leading to decreased chest wall movement and decreased lung compliance. Pulmonary function does not usually improve following anterior spinal surgery and in the longer term it tends to decrease slightly, though this decrease is not thought to be clinically relevant [8]. However, our patients had much worse pre-operative lung function than those reported in the above study, where the mean (standard deviation) pre-operative FVC was 3.64 (0.64) l.

Although one patient was asthmatic and both were receiving opiates via paravertebral catheters, neither of these mechanisms is likely to be the cause of the ventilatory failure. Although lung function was not measured immediately postoperatively in the second patient, there was no wheeze on auscultation and the presence of tachypnoea in both cases makes opiate excess improbable. In neither case was there an apparent cause for the deterioration on the chest radiograph.

Respiratory complications are common following both cardiothoracic and abdominal surgery and result in significant morbidity, mortality and expense. Non-invasive respiratory support has been used to try to decrease the incidence of postoperative complications. Continuous positive airway pressure (CPAP) has been shown to decrease radiological atelectasis and to produce more rapid recovery in FVC after abdominal surgery [9, 10], but these studies did not show any other clinically relevant benefits. Non-invasive pressure support ventilation has been shown to slow respiratory rate and improve oxygenation in a group of 110 mainly postoperative patients who had acute respiratory failure [11]. This study suggested that non-invasive ventilation prevented the need for intubation and formal conventional mechanical ventilation in this group of patients. Another smaller but again uncontrolled study reported benefit in a more

mixed group of surgical and medical patients [12]. Neither study included patients who had undergone spinal surgery. The use of NIPPV to treat acute ventilatory failure due to scoliosis in nonsurgical patients has been reported [6]; all these patients subsequently went on to home ventilation on discharge from hospital.

Although the use of NIPPV can prevent the complications of intubation and conventional mechanical ventilation, it is not without its own drawbacks. The majority of non-invasive ventilators can give only a relatively low inspired oxygen concentration (about 45%). It is therefore unsuitable for patients in whom the main problem is hypoxia; in both our cases the main problem was ventilatory rather than hypoxic failure. Complications of NIPPV include aerophagia and pressure sores from the mask with up to 20% of patients not being able to tolerate the technique.

In summary, we report two patients with severe scoliosis in whom NIPPV was used successfully to treat ventilatory failure that followed corrective spinal surgery so avoiding the need for intubation and its complications. Neither patient has required further ventilatory support 38 and 18 months later. If postoperative NIPPV can reduce complications of intubation and conventional mechanical ventilation in high-risk spinal surgery patients, its use could improve outcome. These observations need to be confirmed in prospective controlled trials.

References

1 Bott J, Carroll MP, Conway JH *et al*. Randomised controlled trial of nasal ventilation in acute ventilatory failure due to chronic obstructive airways disease. *Lancet* 1993; **341:** 1555–7.

2 Ellis ER, Bye PTB, Bruderer JW, Sullivan CE. Treatment of respiratory failure during sleep in patients with neuromuscular disease. *American Review of Respiratory Diseases* 1987; **135:** 148–52.

3 Ellis ER, Grunstein RR, Chan S *et al*. Non-invasive ventilatory support during sleep improves respiratory failure in kyphoscoliosis. *Chest* 1988; **94:** 811–15.

4 Leger P, Bedicam JM, Cornette A *et al*. Nasal intermittent positive pressure ventilation; Long term follow up in patients with severe chronic respiratory insufficiency. *Chest* 1994; **105:** 100–5.

5 Simmonds AK, Elliott MW. Outcome of domiciliary nasal intermittent positive pressure ventilation in restrictive and obstructive disorders. *Thorax* 1995; **50:** 604–9.

6 Finlay G, Concannon D, McDonnell TJ. Treatment of respiratory failure due to kyphoscoliosis with nasal intermittent positive pressure ventilation (NIPPV). *Irish Journal of Medical Science* 1995; **164:** 28–30.

7 Meduri GU, Abou-Shala N, Fox RC *et al*. Non-invasive face mask mechanical ventilation in patients with acute hypercapnic respiratory failure. *Chest* 1991; **100:** 445–54.

8 Wong CA, Cole AA, Watson L *et al*. Pulmonary function before and after anterior spinal surgery in adult idiopathic scoliosis. *Thorax* 1996; **51:** 534–6.

9 Lindner KH, Lotz P, Ahnefeld FW. Continuous positive airway pressure effect on functional residual capacity, vital capacity and its subdivisions. *Chest* 1987; **92:** 66–70.

10 Stock MC, Downs JB, Gauer PK *et al*. Prevention of postoperative pulmonary complications with CPAP, incentive spirometry and conservative therapy. *Chest* 1985; **87:** 151–7.

11 Pennock BE, Crawshaw L, Kaplan PD. Non-invasive nasal mask ventilation for acute respiratory failure; Institution of a new therapeutic technology for routine use. *Chest* 1994; **105:** 441–4.

12 Wysocki M, Tric L, Wolff MA *et al*. Noninvasive pressure support ventilation in patients with acute respiratory failure. *Chest* 1993; **103:** 907–13.

CASE REPORT

Damage to the conus medullaris following spinal anaesthesia

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Summary

Seven cases are described in which neurological damage followed spinal or combined spinal-epidural anaesthesia using an atraumatic spinal needle. All patients were women, six obstetric and one surgical.

All experienced pain during insertion of the needle, which was usually believed to be introduced at the L_{2-3} interspace. In all cases, there was free flow of cerebrospinal fluid before spinal injection. There was one patchy block but, in the rest, anaesthesia was successful. Unilateral sensory loss at the levels of L_4–S_1 (and sometimes pain) persisted in all patients; there was foot drop in six and urinary symptoms in three. Magnetic resonance imaging showed a spinal cord of normal length with a syrinx in the conus ($n = 6$) on the same side as both the persisting clinical deficit and the symptoms that had occurred at insertion of the needle. The tip of the conus usually lies at L_{1-2}, although it may extend further. Tuffier's line is an unreliable method of identifying the lumbar interspaces, and anaesthetists commonly select a space that is one or more segments higher than they assume. Because of these sources of error, anaesthetists need to relearn the rule that a spinal needle should not be inserted above L_3.

Keywords *Anaesthetic techniques*, *regional*: spinal; combined epidural-spinal. *Complications*, *neurological*. *Anatomy*: spinal cord; conus medullaris.

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This report documents seven cases in which single-shot spinal or combined spinal-epidural (CSE) anaesthesia using an atraumatic spinal needle was followed by neurological symptoms involving more than one segmental nerve root. One has previously been reported in detail elsewhere [1]. Five of these cases were encountered in medicolegal practice and two in a survey [2]. Summaries of clinical and anaesthetic data are given in Table 1 and of the neurological outcome in Table 2.

Case 1

A woman who had undergone two previous Caesarean sections was booked for elective Caesarean section under spinal anaesthesia. A 27-G Whitacre spinal needle was inserted at L_{2-3}, with slight difficulty because the patient was restless. When the needle was inserted she complained of pain down her right leg, but there was a good flow of cerebrospinal fluid (CSF) from the needle. Hyperbaric bupivacaine was injected without pain and sensory block ensued as expected. Later the same day she complained of throbbing pain radiating down the right leg, with weakness in her right leg, pain in the right thigh and buttock, paraesthesia in the right big toe and numbness over the whole of the right leg and lateral border of her right foot. Reflexes were absent and motor power was reduced uniformly round all three joints in the right leg. A magnetic resonance imaging (MRI) scan of the lumbar spine was initially reported to show no abnormality, but on review showed a small syrinx to the right of the midline, at about the level of the body of the 12th thoracic vertebra (Fig. 1).

Some months later, she still complained of persistent pain and numbness in the right leg and foot. On examination, light touch, pin prick and vibration sense were all reported to be absent below the level of T_{8-9}. All reflexes were brisk except the right knee, ankle and plantar reflexes which were absent, and there was a persistent foot drop. There was also some difficulty initiating micturition, with poor bladder sensation. Electromyographic (EMG) changes suggested motor and sensory deficit at the levels of L_{4-5}–S_1 and that the damage was central, in the spinal cord or roots, rather than peripheral. The extent of authenticated damage could not be explained by damage to a single root.

Case 2

A primipara required an emergency Caesarean section when not in labour because of unprovoked decelerations in the fetal heart trace. It was decided that this should be under spinal anaesthesia. A 25-G pencil-point needle was inserted at L_{2-3}. Insertion was easy but was associated with left hip pain. Clear CSF was obtained and hyperbaric bupivacaine injected without problem, producing a satisfactory block.

On the following day, she stayed in bed as her leg felt weak, but micturition was normal. Numbness, weakness and 'pins and needles' in the left leg persisted but with some improvement over the next few days. Neurological examination showed that sensation was reduced over the lateral side of the left lower calf, dorsum of the foot and the outer three toes. There was weakness of all movements in the left ankle joint and great toe, and the left ankle jerk was reduced.

Table 1 Clinical and anaesthetic details of seven patients who suffered damage to the conus medullaris following spinal anaesthesia

Case no.	Weight (kg)	Type of block*	Procedure	Position of patient	Size of needle (G)	Presumed level and details of insertion†	Dose of hyperbaric bupivacaine 0.5%	Outcome of block
1	62	SSS	Caesarean section	Left lateral	27	L_{2-3}; free flow of CSF	2.5 ml with fentanyl 12.5 μg	Good
2	47	SSS	Caesarean section	Sitting	25	L_{2-3}; free flow of CSF	2.6 ml	Good
3	86	CSE	Caesarean section	Left lateral	25	L_{2-3}; CSF after needle withdrawn	2.0 ml with fentanyl 25 μg	T_4
4	66	SSS	Breech delivery	Unknown	25	L_{2-3};‡ CSF after needle withdrawn; pain on injection	3.0 ml	Incomplete on left side
5	65	CSE	Caesarean section	Sitting	26	L_{2-3}; free flow of CSF	2.5 ml	T_{4-6}
6	102	CSE	Caesarean section	Sitting	25	L_{2-3}/L_{3-4} (uncertain); no recorded difficulty with insertion	2.3 ml with fentanyl 25 μg	T_4
7	102	CSE	Incisional hernia repair	Sitting	27	L_{1-2}/L_{2-3};¶ difficult insertion	3.0 ml	Good

*SSS, single shot spinal; CSE, combined epidural–spinal. †All patients complained of pain on insertion of the spinal needle. CSF, cerebrospinal fluid. ‡T_{11-12} later confirmed. ¶L_{1-2} later confirmed.

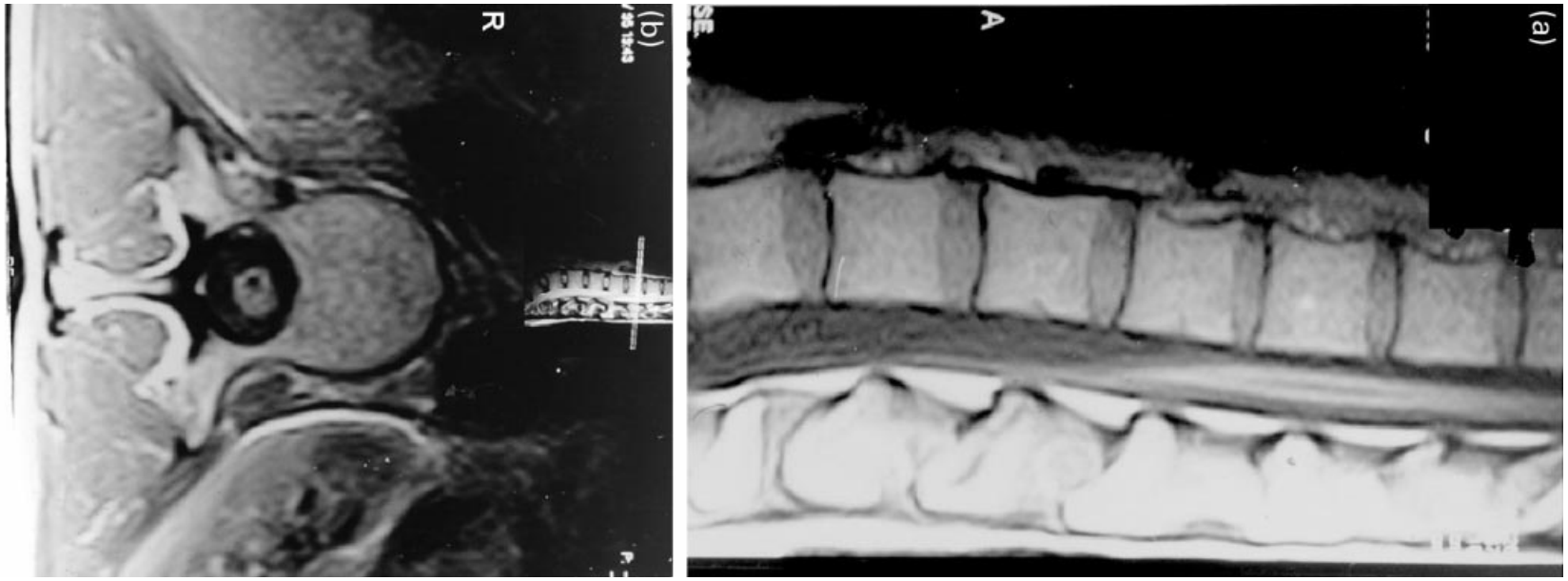


Table 2 Neurological outcome in seven patients who suffered damage to the conus medullaris following spinal anaesthesia

Case no.	Pain on insertion	MRI appearance of conus medullaris	Neurological outcome		
			Urinary problems	Sensory	Motor
1	Right leg	Syrinx right side (Fig. 1)	Yes	L_4-S_1 on right	Right foot drop
2	Left hip	Syrinx left side	No	L_5-S_1 on left	Left foot drop
3	Back, left leg	Syrinx left side (Fig. 2)	Yes	L_4-S_1 on left	Left foot drop
4	Right side	?Normal	No	T_4-S_2 on right	Only lasted one week
5	Right leg	Syrinx right side (Fig. 3)	Yes	L_4-S_3 on right	Right foot drop
6	?Left leg	High signal in conus at L_1	No	L_4-S_1 on left	Left leg weakness
7	Right leg	Syrinx right side	No	L_5-S_1 on right	Right foot drop

The MRI scan showed no epidural abnormality or disc protrusion, but a cavity within the cord on the left side from the lower thoracic region to the conus, which ended at L_{1-2}. The appearance was not consistent with a congenital abnormality and there was no evidence of vertebral damage to suggest a traumatic syrinx. She went home, after some improvement, on the sixth postpartum day. Mild foot drop and some abnormality of sensation persisted on the left side.

Case 3

A parous woman was booked for Caesarean section after failed induction of labour for mild pre-eclampsia at 41 weeks' gestation. A CSE technique was used, and although the anaesthetist stated that it was inserted in 'the lower back' the patient recalled that it was at about the level of her bra strap. During insertion of the spinal needle she reported severe pain radiating throughout her back and down the left leg. Cerebrospinal fluid emerged only after the needle was withdrawn slightly. She was given hyperbaric bupivacaine with fentanyl intrathecally with only transient slight pain, and a bilateral block to T_4 resulted. The operation passed uneventfully and the epidural catheter was not used.

After the Caesarean section it soon became clear that her left leg was not recovering normally. She had persistent left leg weakness and pain, numbness from the groin downwards, headache and difficulty passing urine. On examination, she had sensory loss from L_1 downwards in the left leg, severe weakness (slight movement only) round all joints and absent reflexes in the left leg. The MRI scan later that day showed no epidural abnormality, but a swollen conus with increased signal from T_{10} to T_{12}, mainly on the left side (Fig. 2). This was diagnosed as a small haematoma within the cord.

Headaches, foot drop, toe deformities, pain and some numbness in the left leg persisted, with sphincter disturbance which showed some improvement.

Case 4

This patient received a single-shot spinal anaesthetic during labour for assisted vaginal breech delivery. The spinal needle was sited by an experienced anaesthetist, who reported using the L_{2-3} interspace, although later examination suggested it was T_{11-12}. On insertion of the needle, the patient screamed and reported a burning sensation on the right side. The needle was withdrawn and CSF aspirated. Hyperbaric bupivacaine was injected but there was pain on injection. The block was incomplete on the left side.

Initially on the first postpartum day there was sensory loss up to T_4 on the right and T_{10} on the left. Motor function was grossly impaired but recovered completely in a week. Sensation returned to normal on the left over weeks, but remained abnormal on the right from T_4 to S_2 with dysaesthesia requiring medical treatment. There were no bladder symptoms. The MRI scan was reported as normal but the signs were believed to relate to damage to the spinothalamic tract.

Figure 1 Magnetic resonance imaging scan using T1 weighting, in case 1. (a) The sagittal view shows cord and epidural fat as white and fluid as dark. The cord ends at the lower border of L_1. A dark cleft is visible in the substance of the cord, which also appears as a dark spot on the right of the midline in (b), the axial image at T_{12}.

Case 5

This patient had pre-eclampsia but with normal clotting. She had a CSE inserted reportedly at L_{2-3}. During insertion of the spinal needle she experienced transient pain shooting down her right leg, but there was free flow

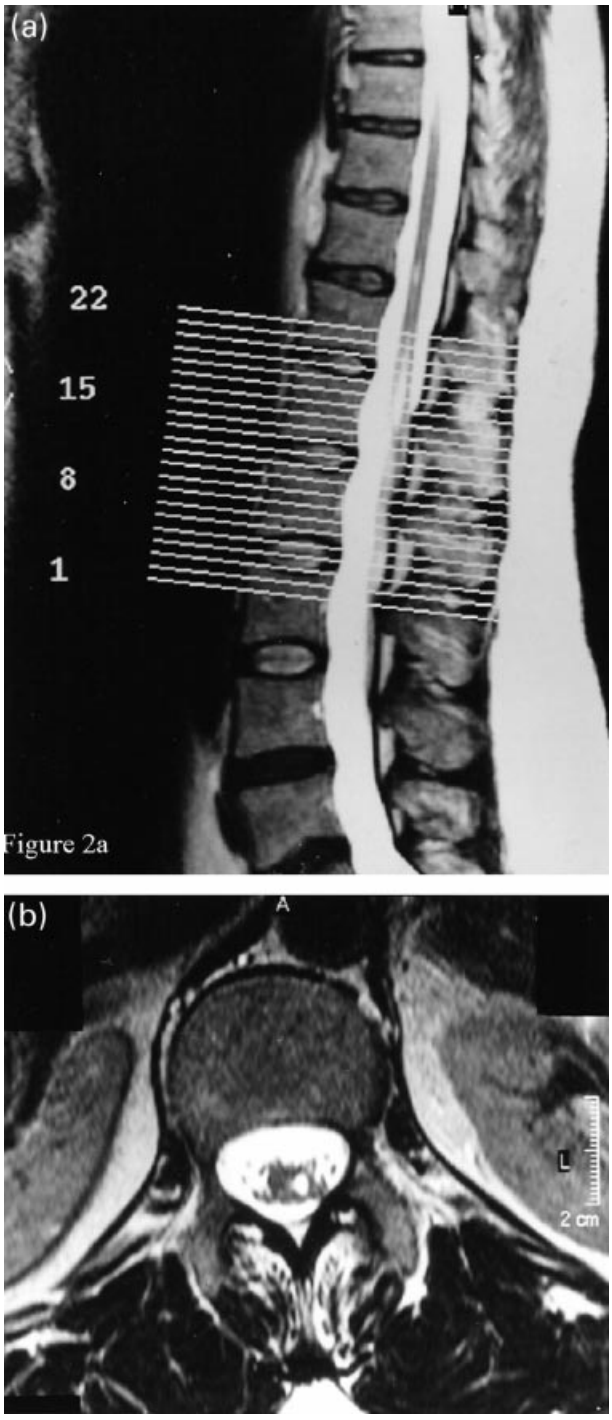


Figure 2 Magnetic resonance imaging scan using T2 weighting, in case 3. The end of the cord is less clear in this sagittal image (a), which appears to show several vacuoles, but the syrinx is clearly visible on the left side in (b), the axial view at cut 17 in (a).

of CSF and no pain on injection of bupivacaine. The resulting block height was as expected.

Postpartum recovery was incomplete in the right leg, and initial neurological examination revealed diminished sensation on the right from L_4 to S_3, with weak dorsal and plantar flexion at the ankle but normal reflexes. She had urinary retention requiring prolonged catheterisation. The MRI scan (Fig. 3) showed what was reported as a cleft within the right side of the conus at T_{12}–L_1. This was diagnosed as a conus infarct.

She was reviewed 8 months later when her urinary problems had recovered, but dysaesthesia and foot drop continued in the right leg. On external examination, the site of skin puncture was found to be consistent with L_{2-3}. It was considered impossible for the spinal needle to have reached the site of the lesion.

Case 6

This patient required Caesarean section under CSE because of deteriorating renal disease early in the third trimester of pregnancy. The level of CSE insertion was thought to be L_{2-3} or L_{3-4}. The patient recalled pain on insertion, although no problems were recorded by the anaesthetist at the time. The level of block was T_4 and surgery was uneventful.

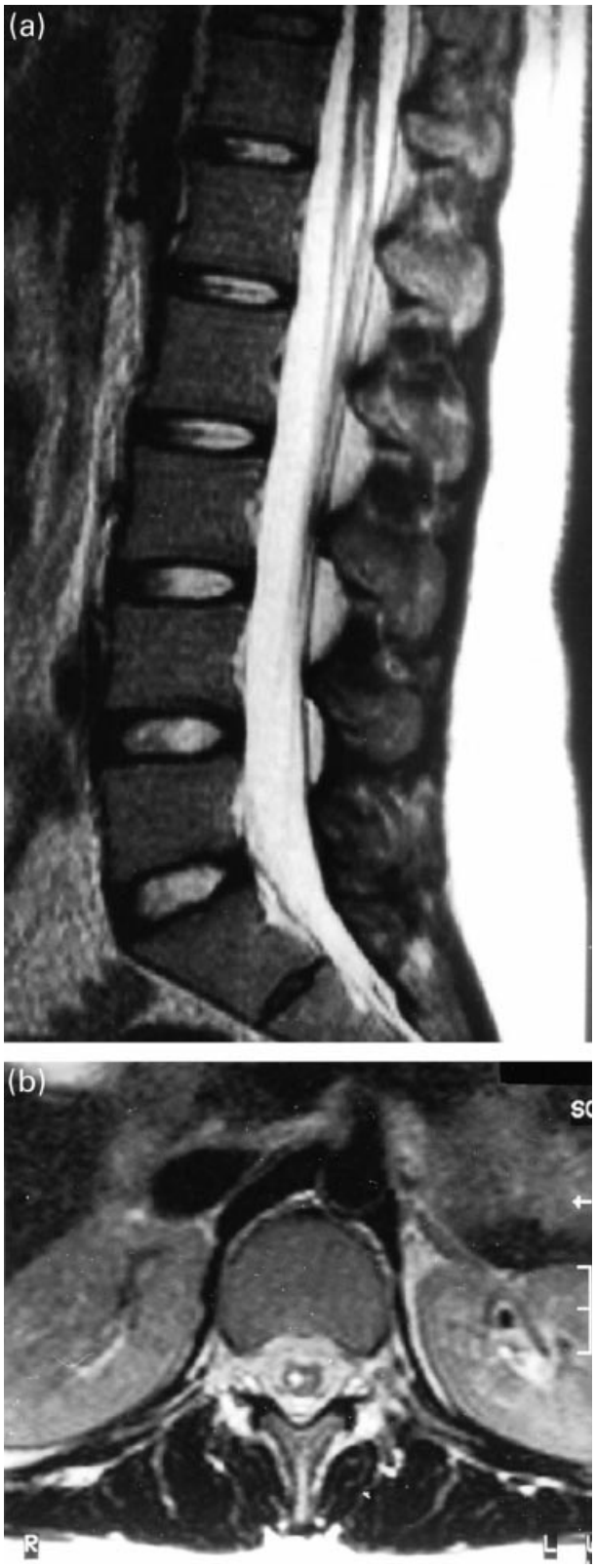
Post partum, the patient complained of pain in the left leg as soon as the block began to wear off. The MRI scan showed a high signal on T2-weighted spin echo in the conus at the vertebral level of L_1.

Pain, dysaesthesia and weakness in the left leg continued. Eighteen months later she had reduced temperature and vibration sensation below the left knee, allodynia in L_5 and S_1, and a numb great toe. There was some weakness at the hip, knee and ankle, with eversion the most severely affected.

Case 7

This obese patient with a history of difficult intubation presented for re-repair of an incisional hernia. It was decided to use spinal anaesthesia, but insertion was not easy and several attempts produced 'pins and needles' down the left leg. Eventually, and with difficulty, a CSE approach was tried on what felt to the patient like the right side. The subarachnoid space was entered successfully at a level that was thought to be either L_{1-2} or L_{2-3} (later confirmed by image intensifier as L_{1-2}). This produced a sharp pain on the right side, but spinal anaesthesia produced a good block and surgery was successfully accomplished.

Postoperatively, she complained of pain and weakness in the right leg, and was found to have foot drop. This was



initially attributed to tight antithromboembolism stockings, but symptoms persisted and 2 weeks later MRI revealed a syrinx within the right side of the conus. Electromyography confirmed an L_5–S_1 lesion. Her foot drop persisted.

Discussion

The seven patients whose cases are reported here all suffered damage to more than a single nerve root, three after single-shot spinals and four after CSEs. Their ages ranged from teens to fifties and their weights from 47 to 102 kg. That six of them were obstetric patients probably reflects the author's sphere of practice. In all cases an atraumatic spinal needle, usually a 25 or 27 G Whitacre, was used. In all cases the anaesthetist believed the needle was being inserted at L_{2-3} or thereabouts. In three cases there was free flow of CSF without any need for needle adjustment, yet all patients reported pain on insertion of the needle, and in only one was there also pain on injection of the anaesthetic. All patients received bupivacaine rather than lidocaine, the former having considerably less propensity to neurotoxicity [3, 4], while some but not all received fentanyl. In all but one case, spinal anaesthesia produced a block such as would be expected from the dose of bupivacaine that was given (Table 1). Case 4 might have been expected to suffer the most severe symptoms as the only case in which the local anaesthetic appeared to have been injected into the cord, with a resulting incomplete block. Indeed, sensory symptoms were the most extensive, but there was no sphincter disturbance and motor loss was short-lived. Symptoms were believed to relate to the spinothalamic tract, although such damage would be unlikely from a spinal needle as the spinothalamic tract is of course on the anterolateral aspect of the spinal cord. Uniquely in this case, no changes were said to be visible on MRI.

In all cases sensory and motor deficit of lower motor neurone distribution relating to one leg, usually with unilateral MRI changes in the conus, followed pain on spinal needle insertion on the same side (Table 2). In no case did the spinal cord appear to be unduly long.

Aetiology of conus lesions

When major neurological symptoms follow neuraxial blockade, the worried clinician turns to imaging techniques to exclude an epidural space-occupying lesion which would require urgent decompression. Epidural haematoma, abscess, prolapsed disc and spinal stenosis

Figure 3 Magnetic resonance imaging scan using T2 weighting, in case 5. (a) Sagittal view. The syrinx shows to the right of the midline in (b), the axial view at the level of the T_{12}–L_1 disc.

have all been reported in such circumstances [5, 6]. Subdural haematoma is also reported after spinal anaesthesia [7]. Compression in the lumbar region, as it is often exerted below the cord, usually gives rise to cauda equina syndrome. No such features were visible on MRI in any of the present cases.

Signs and symptoms relating to the conus medullaris may also result from compression by tumours [8, 9], syrinx [10], congenital cysts [11, 12] or vascular malformations, or from trauma [13–15], infarction [16–19] and tethered cord [20, 21].

Mathew & Todd [8] analysed the presentation in 62 patients with tumours in either the cauda equina or the conus. The commonest symptom in both groups was back pain, with bilateral leg pain being more common with conus and unilateral with cauda equina tumours. Unilateral or bilateral leg weakness, usually lower motor neurone, could occur in both groups, with bladder involvement present in 36% of conus and 26% of cauda equina tumours. Anal sphincter involvement was uncommon.

A congenital cyst differs from a syrinx in that the former is a congenital dilatation of the ventriculus terminalis (the terminal portion of the central canal, not normally visible on MRI) and is lined with ependyma [11]. Presenting symptoms are said to be non-specific and include low back pain, sciatica, leg weakness and bladder dysfunction [11, 12]. The MRI appearance of such a cyst is much larger than in the present series, and suggests that it virtually fills the vertebral canal, causing considerable compression of nervous tissue and accounting presumably for the common occurrence of back pain, a symptom that was prominent in case 6 in the present series.

Trauma may result from vertebral fracture [15], a shearing injury (as seen in leg injuries from motor cycle accidents) causing nerve root avulsion [13], or occasionally even manipulation [22] causing vascular damage. Pain and bladder symptoms are prominent [14].

Infarction of the conus medullaris is uncommon, because the blood supply to the conus is normally secure, being derived from the artery of Adamkiewicz with generous anastomoses. Occlusion of this artery may result in paraplegia, not cauda equina or conus syndrome. However, in a minority of individuals, perhaps 20%, the blood supply to the conus comes from sacral radicular arteries with fewer anastomoses, and may be more vulnerable. The usual victims are the elderly with arterial pathology [16–18]. Pre-eclampsia might have been a possible contributory factor in cases 3 and 5.

Tethered cord syndrome results from a congenital anomaly; it may occur in isolation, in some forms of scoliosis and typically diastematomyelia. It commonly presents with urinary symptoms [20, 21] and may be exacerbated by the lithotomy position. Although typically the conus is abnormally low, it may occasionally be at a normal level but tethered by a tight filum terminale. Tethered cord should represent a contraindication to neuraxial anaesthesia. None of the cases reported here showed evidence of it.

Intrathecal injection of a neurotoxic substance, such as irritant agents that were once used for spinal anaesthesia, can produce cauda equina syndrome, because the sacral roots are poorly myelinated and particularly vulnerable to chemical damage. Although Waters *et al.* [3] reported 'conus medullaris injury' following spinal anaesthesia using tetracaine and lidocaine in sequence, the cauda equina was the more likely site of such injury. Cauda equina syndrome is indeed described following continuous spinal anaesthesia using lidocaine [23]. Tedeschi *et al.* [24] reported a case of true conus injury following spinal anaesthesia in a 62-year-old diabetic woman, with the MRI appearance of gas within the cord and oedema surrounding it. Katz & Hurley [25] described the case of a parturient who was given repeated painful top-ups via an epidural catheter embedded within the conus. The resulting syrinx appeared to fill the vertebral canal, an MRI appearance similar to that of a congenital cyst. Greaves [26] reported a case of haematomyelia following attempted spinal anaesthesia believed to be at L_{3-4}. The patient, another elderly woman, had a hip replacement under general anaesthesia following failed spinal anaesthesia. She had suffered severe pain reportedly in the left leg on injection of an estimated 0.3 ml of hyperbaric bupivacaine. An atraumatic spinal needle had not been used. Postoperatively, she had sensory deficit from T_{12} to L_3 and a dense motor paralysis in the right leg. She died on the tenth postoperative day of pulmonary embolus, and at autopsy the needle track, identified at T_{12}–L_1, penetrated the conus and was associated with a haemorrhage within the cord extending 4.5 cm cephalad, on the right of the midline. The anomalous side of the initial pain was apparently unnoticed by the author, editor and all referees of this paper. This case is important because, although there was no MRI, the nature of lesion was verified at postmortem examination, and also because of the proven error of perhaps three segments in the level of spinal insertion.

Three anatomical factors

1 Although the cord commonly ends opposite the lower border of L_1 or the L_{1-2} interspace, it may extend as low as L_3. Thomson [27] found that it reached L_2 in 43% of women but only 27% of men. The frequency distribution of the segmental level at which the spinal cord ends was assessed by Reimann & Anson [28] in 129 cadavers, and by Saifuddin *et al.* [29] in a more recent MRI study of 504 adults. Their results are summarised in Fig. 4. Considering the angle of entry of a spinal needle, if inserted at L_{1-2}

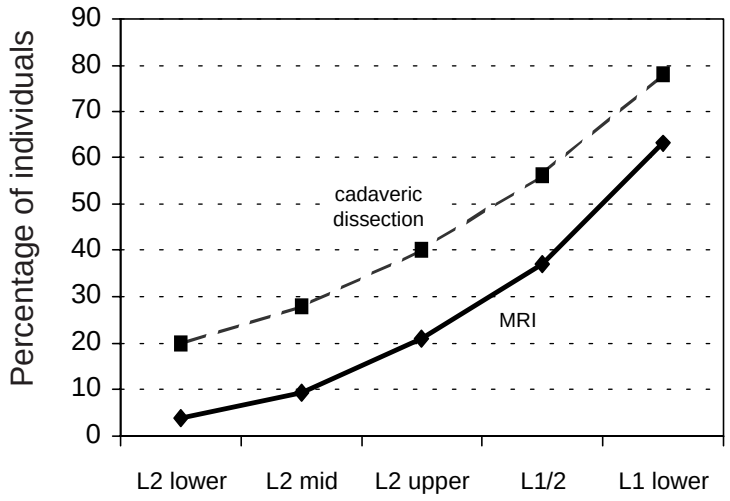


Figure 4 Proportion of adults in whom the cord would be present at each spinal level. Cumulative data derived from Reimann & Anson [28] (■) and Saifuddin *et al.* [29] (◆); L2 lower = the lower third [29] or half [28] of the body of L_2; L2 mid = middle of the body of L_2 (interpolated in upper curve); L2 upper = upper third [29] or half [28] of the body of L_2; L1/2 = L_{1-2} interspace; L1 lower = the lower third [29] or half [28] of the body of L_1.

it might reach a conus that ended at the lower border of L_1, which according to Fig. 4 would encompass between 63 and 78% of individuals. Using the same argument, if inserted at L_{2-3} it might be possible to reach the conus in 4–20% of people.

2 Tuffier's line, that joining the iliac crests, while commonly used to identify lumbar interspaces, does not bear a constant relationship to them. Although the mode is the lower border of L_4 to the L_{4-5} interspace [30, 31], the level may vary from L_{3-4} to L_5–S_1, hence a major source of error.

3 At the level of the conus the nerve roots form a highly organised overlapping pattern in close proximity to the cord, and bound to it by an intricate web of arachnoid membrane [32].

Possible mechanisms in the cases reported here

The consistent histories in these seven cases, with damage to more than one root, strongly suggest that the needle-tip alone can cause conus damage. In all cases the symptoms were mainly unilateral, unlike with more severe conus lesions, but this is perhaps not surprising because the MRI lesions appeared small and unilateral (Figs 1–3). The MRI changes that were observed are consistent with fluid collection, intramedullary haemorrhage or a small infarct. Yet most of those involved did not believe the needle could have reached the spinal cord. This may be a misapprehension (see below) but it is possible, perhaps, that a needle inserted among the tightly knit terminal roots could tear the surrounding membrane causing a small haemorrhage.

Why this cluster of cases? Can they be attributed to the current fashion for atraumatic needles? It is true that an atraumatic spinal needle has at least 1 mm of blind tip beyond the hole and there may be a tendency to insert it further into the subarachnoid space than is necessary with a Quincke needle. Yet this 1 mm is unlikely to be the whole answer.

For many years spinal anaesthesia was in the doldrums, while epidural blockade flourished, and anaesthetists learnt to site epidural needles at all manner of levels. Then, with the re-introduction of atraumatic needles and the use of less noxious local anaesthetic solution, the practice of spinal anaesthesia made a comeback, particularly in obstetric practice, and it is now the most popular form of anaesthesia for Caesarean section [33]. So now, many anaesthetists with liberal attitudes to lumbar interspaces that are, moreover, condoned by many textbooks [34], have taken to using spinal and CSE anaesthesia. It should be emphasised, however, the cephalad tilt given to the spinal needle at CSE using the needle-through-needle technique is not the only problem, as three of the cases reported here related to single-shot spinal anaesthesia.

Although many anaesthetists are confident that they can identify lumbar interspaces accurately, van Gessell *et al.* [35] demonstrated that 59% of dural punctures were performed one or two spaces higher than assumed. More recently, Broadbent *et al.* [36] found that when a group of experienced anaesthetists believed they had identified L_{3-4}, in 85% of observations the space selected was one to four segments higher than this. Given the inaccuracy of methods of identifying lumbar interspaces, and the variability of the position of the conus, it cannot be logical to aim to insert a needle intrathecally above the spinous process of L_3.

If a spinal needle causes pain, it is obviously correct to avoid injection although it may be too late to prevent nerve damage. It would also be wise to establish by radiological means the interspace that has been used. The

moral of these stories is not to avoid atraumatic needles but to avoid upper lumbar interspace at all times, exercising particular care in women.

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References

1 Rajakulendran Y, Rahman S, Venkat N. Long-term neurological complication following traumatic damage to the spinal cord with a 25 gauge Whitacre spinal needle. *International Journal of Obstetric Anesthesia* 1999; **8**: 62–6.

2 Holloway J, Seed PT, O'Sullivan G, Reynolds F. Paraesthesiae and nerve damage following combined spinal epidural and spinal anaesthesia. *International Journal of Obstetric Anesthesia* 2000; **9**: 151–5.

3 Waters JH, Watson TB, Ward MG. Conus medullaris injury following both tetracaine and lidocaine spinal anesthesia. *Journal of Clinical Anesthesia* 1996; **8**: 656–8.

4 Lambert LA, Lambert DH, Strichartz GR. Irreversible conduction block in isolated nerve by high concentrations of local anesthetics. *Anesthesiology* 1994; **80**: 1082–93.

5 Loo CC, Dahlgren G, Irestedt L. Neurological complications in obstetric regional anaesthesia. *International Journal of Obstetric Anesthesia* 2000; **9**: 99–124.

6 Reynolds F. Maternal sequelae of childbirth. *British Journal of Anaesthesia* 1995; **75**: 515–17.

7 Pryle BJ, Carter JA, Cadoux-Hudson T. Delayed paraplegia following spinal anaesthesia: spinal subdural haematoma following dural puncture with a 25-G pencil point needle at T12–L1 in a patient taking aspirin. *Anaesthesia* 1996; **51**: 263–5.

8 Mathew P, Todd NV. Intradural conus and cauda equina tumours: a retrospective review of presentation, diagnosis and early outcome. *Journal of Neurology, Neurosurgery and Psychiatry* 1993; **56**: 69–74.

9 Hida K, Iwasaki Y, Cho K, Imamura H, Abe H. Gliomas of the conus medullaris. *Paraplegia* 1994; **32**: 5258.

10 Sridhar K, Ramamurthi R, Vasudevan MC. Isolated eccentric syrinx of the conus medullaris simulating a tumour. *British Journal of Neurosurgery* 1999; **13**: 423–5.

11 Sigal R, Denys A, Halimi P, Shapeero L, Doyon D, Boudghene F. Ventriculus terminalis of the conus medullaris: MR imaging in four patients with congenital dilatation. *American Journal of Neuroradiology* 1991; **12**: 733–7.

12 Matsubayashi R, Uchino A, Kato A, Kudo S, Sakai S. Cystic dilatation of ventriculus terminalis in adults: MRI. *Neuroradiology* 1998; **40**: 45–7.

13 Moossy JJ, Nashold BS Jr, Osborne D, Friedman AH. Conus medullaris nerve root avulsions. *Journal of Neurosurgery* 1987; **66**: 835–41.

14 Sampson JH, Cashman RE, Nashold BS Jr, Friedman AH. Dorsal root entry zone lesions for intractable pain after trauma to the conus medullaris and cauda equina. *Journal of Neurosurgery* 1995; **82**: 28–34.

15 Taylor TK, Coolican MJ. Injuries of the conus medullaris. *Paraplegia* 1988; **26**: 393–400.

16 Anderson NE, Willoughby EW. Infarction of the conus medullaris. *Annals of Neurology* 1987; **21**: 470–4.

17 Andrews BT, Kwei U, Greco C, Miller RG. Infarct of the conus medullaris simulating a spinal cord tumor: case report. *Surgical Neurology* 1991; **35**: 139–42.

18 Ohbu S, Ishimoto A, Honda M, Fukuda H, Hata Y, Tada S. Infarction of the conus medullaris. *European Neurology* 1990; **30**: 343–4.

19 Herkes GK, Selby G, Sorby WA. Infarction of the conus medullaris – clinical and radiographic features. *Clinical and Experimental Neurology* 1989; **26**: 205–11.

20 Selcuki M, Coskun K. Management of tight filum terminale syndrome with special emphasis on normal level conus medullaris (NLCM). *Surgical Neurology* 1998; **50**: 318–22.

21 Warder DE, Oakes WJ. Tethered cord syndrome: the low-lying and normally positioned conus. *Neurosurgery* 1994; **34**: 597–600.

22 Balblanc JC, Pretot C, Ziegler F. Vascular complication involving the conus medullaris or cauda equina after vertebral manipulation for an L4–L5 disk herniation. *Revue Du Rhumatisme, English Edition* 1998; **65**: 279–82.

23 Rigler ML, Drasner K, Krejchie TC, *et al*. Cauda equina syndrome after continuous spinal anesthesia. *Anesthesia and Analgesia* 1991; **72**: 275–81.

24 Tedeschi E, Marano I, Savarese F, *et al*. Neurological deficit following spinal anaesthesia: MRI and CT evidence of spinal cord gas embolism. *Neuroradiology* 1999; **41**: 275–8.

25 Katz N, Hurley R. Epidural anesthesia complicated by fluid collection within the spinal cord. *Anesthesia and Analgesia* 1993; **77**: 1064–5.

26 Greaves JG. Serious spinal cord injury due to haematomyelia caused by spinal anaesthesia in a patient treated with low-dose heparin. *Anaesthesia* 1997; **52**: 150–4.

27 Thomson A. Fifth annual report of the committee of collective investigation of the Anatomical Society of Great Britain and Ireland for the year 1893–94. *Journal of Anatomy and Physiology* 1894; **29**: 35–60.

28 Reimann AF, Anson BJ. Vertebral level of termination of the spinal cord with report of a case of sacral cord. *Anatomical Record* 1944; **88**: 127–38.

29 Saifuddin A, Burnett SJ, White J. The variation of position of the conus medullaris in an adult population. A magnetic resonance imaging study. *Spine* 1998; **23**: 1452–6.

30 Hogan QH. Tuffier's line: the normal distribution of anatomic parameters (letter). *Anesthesia and Analgesia* 1994; **78**: 194–5.

31 Render CA. The reproducibility of the iliac crest as a marker of lumbar spine level. *Anaesthesia* 1996; **51**: 1070–1.

32 Wall EJ, Cohen MS, Abitbol JJ, Garfin SR. Organization of intrathecal nerve roots at the level of the conus medullaris.

Journal of Bone and Joint Surgery: American Volume 1990; **72**: 1495–9.

33 Shibli KU, Russell IF. A survey of anaesthetic techniques used for caesarean section in the UK in 1997. *International Journal of Obstetric Anesthesia* 2000; **9**: 160–7.

34 Reynolds F. Logic in the safe practice of spinal anaesthesia. *Anaesthesia* 2000; **55**: 1045–6.

35 Van Gessel EF, Forster A, Gamulin Z. Continuous spinal anesthesia: where do spinal catheters go? *Anesthesia and Analgesia* 1993; **76**: 1004–7.

36 Broadbent CR, Maxwell WB, Ferrie R, Wilson DJ, Gawne-Cain M, Russell R. Ability of anaesthetists to identify a marked lumbar interspace. *Anaesthesia* 2000; **55**: 1106–26.

