

Nerve damage following central neuraxial blockade: Is it avoidable?

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Permanent nerve damage following central neuraxial blockade is a devastating but fortunately rare event. This lecture will briefly summarize the possible causes of nerve damage followed by a more detailed discussion of factors that may increase the risk of nerve damage and how to avoid them.

Numerous mechanisms have been described that may result in major neurological complications. These can be subdivided as follows⁽¹⁾ (Table I, modified).

With the latter in mind, Greene defined the following criteria for establishing that spinal anesthesia caused neurological damage⁽²⁾:

- (a) The causative neurologic lesion is intradural
- (b) Temporally, it is at least possible that the onset of symptoms coincided with administration of anesthesia and that such symptoms did not antedate the anesthesia.
- (c) The intradural pathologic change is of a histologic type that can be associated with spinal anesthesia.
- (d) The pathologic change is actually due to the anesthesia.

Historically, the reported frequency of persistent sensory or motor deficits has ranged from 0.005% to 0.7%. Recent studies report the incidence of nerve injuries following spinal anesthesia at 0.13% (6/4,767)⁽³⁾, 0.12% (48/40,640)⁽⁴⁾, and 0.03% (12/35,439)⁽⁵⁾, and at 0% (0/5,561) for epidural anesthesia⁽⁵⁾.

Due to underreporting and medicolegal implications, the true incidence of neurological injury associated with central neuraxial blockade is unknown. Numerous studies have reported on the incidence of complications associated with regional anesthesia in general. Dahlgren and Tornebrandt report on 17 733 consecutive central blocks (8,501 spinal and 9,232 epidural anesthetics) performed during a three-year period⁽⁶⁾. Neurological complications related to anesthesia were reported in 17 cases of which 13 patients had

Table I. Mechanisms of neurological damage.

- (1) Cord trauma due to:
 - a. Direct injury to the spinal cord or nerve roots caused by the needle and/or catheter
 - b. Local anesthetic toxicity
- (2) Compression of the cord secondary to:
 - a. Hematoma formation as a result of:
 - i. Needle trauma
 - ii. Tumor
 - iii. Vascular anomaly
 - iv. Bleeding disorder
 - v. Patient receiving anticoagulant drugs
 - vi. "Idiopathic"
 - b. Abscess formation:
 - i. Exogenous infection via needle
 - ii. Hematogenous
- (3) Cord ischemia:
 - a. Anterior spinal artery syndrome
 - b. Vascular trauma
- (4) Intercurrent neurological disease that mimic neurological injury from epidural or spinal anesthesia:
 - a. Spinal meningioma
 - b. Spinal arteriovenous fistula, vascular malformation or hemangioma
 - c. Prolapsed intervertebral disc
 - d. Guillain-Barré syndrome
 - e. Multiple sclerosis
 - f. Spinal hematoma (spontaneous)
 - g. Tumor metastases
 - h. Thalassemia

persisting lesions after 3 spinal and 10 epidural blocks. In 7 epidural blocks, the connection between the neurological lesion and the anesthetic technique was questionable. In 5 of these cases, polyneuropathy or nonspecific neurological symptoms were present. Three complications after epidural

blocks resulted in paraplegias caused by spinal haematomas in patients with abnormal hemostasis, i.e. an incidence of 0.03% (3/9,232) and 0.04% (3/8,501) following spinal anesthesia.

Moen et al performed a retrospective study of severe neurologic complications after central neuraxial blockade performed in Sweden between 1990 and 1999⁽⁷⁾. During this period approximately 1,260,000 spinal and 450,000 epidural blocks were performed, including 200,000 epidurals for pain relief in labor. One hundred and twenty seven complications reported included spinal hematoma (33), cauda equina syndrome (32), meningitis (29), epidural abscess (13), and miscellaneous (20). Permanent neurologic damage was observed in 85 patients. The incidence of complications after spinal blockade was within 1:20-30,000 in all patient groups, after obstetric epidurals was 1:25,000, and in the remaining patients it was 1:3,600 ($P < 0.0001$).

In Finland the Patient Injury Act (PIA) has been in effect since May 1987. This legislation is a no-fault compensation scheme and implies that if a patient during the course of medical treatment suffers any injury as a result of that treatment he or she may file a claim to the PIA. Between 1 May 1987 and 31 December 1993, 23,500 claims for compensation were made. Aromaa et al. reviewed all claims made through the PIA involving spinal and epidural anesthetics during the above period⁽⁸⁾. The total number of anesthetics given during this period was estimated by sending questionnaires to every hospital in the country. They report that 86 claims were associated with spinal and/or epidural anaesthesia out of a total number of 550,000 spinals and 170,000 epidural. These included 25 serious complications associated with spinal anaesthesia, including the following neurological complications: paraplegia (5), permanent cauda equina syndrome (1), peroneal nerve paresis (6), and neurological deficits (7). The 9 serious complications associated with epidural anaesthesia included paraparesis (1), permanent cauda equina syndrome (1), peroneal nerve paresis (1), and neurological deficit (1). Thus, the incidence of serious neurological complications was 19/550,000 (0.003%) following spinal and 4/170,000 (0.002%) following epidural anaesthesia. They concluded that an atraumatic technique, careful patient selection and the early diagnosis and treatment of complications are essential in avoiding permanent injury.

Horlocker et al retrospectively reviewed 4,767 consecutive spinal anesthetics performed between June 1987 and June 1990⁽³⁾. Mean patients age was 65 ± 15 years and included 3,560 (74.7%) men and 1,207 (25.3%) women. A preexisting neurologic condition was present in 481 (10.1%) cases. Paresthesia was elicited during needle placement in 298 (6.3%) cases. Six patients reported pain upon resolution of the spinal anesthetic (persistent paresthesia). Four

persistent paresthesias resolved within 1 week; the remaining two resolved in 18-24 months. The presence of a paresthesia during needle placement significantly increased the risk of persistent paresthesia ($P < 0.001$), however none of the cases resulted in permanent neurological damage. The authors conclude that these results are similar to those of previously published reviews and demonstrate the continued safety of spinal anesthesia.

Auroy et al reported in 2 separate studies, one in 1997 and the second in 2002, on the incidence of serious complications related to regional anesthesia^(4,5). In the first study, out of a total of 103,730 regional anesthetics, 40,640 were spinals, 30,413 epidurals, 21,278 peripheral nerve blocks, and 11,229 intravenous regional anesthetics. Of 34 neurological complications (radiculopathy, cauda equina syndrome, paraplegia), 21 were associated either with paresthesia during puncture (19) or with pain during injection (2), suggesting nerve trauma and/or intraneural injection. Twelve patients who had neurologic complications after spinal anesthetics had no paresthesia during needle placement and no pain on injection. Of these 12 patients (7 with radiculopathy and 5 with cauda equina syndrome), 9 received intrathecal hyperbaric lidocaine, 5%. The incidence of neurologic injury was significantly greater after spinal anesthesia (6 ± 1 per 10,000 cases; $P < 0.05$) than after each of the other types of regional procedures (1.6 ± 0.5 per 10,000 cases for the weighted average). The authors drew the following conclusions: (1) Two-thirds of the patients with neurologic deficits had either a paresthesia during needle placement or pain on injection. (2) Seventy-five percent of the neurologic deficits after nontraumatic spinal anesthesia occurred in patients who had received hyperbaric lidocaine, 5%. In the second study all French anesthesiologists were invited to participate in a 10-month prospective survey based on (1) voluntary reporting of major complications related to regional anesthesia occurring during the study period using a telephone hotline service available 24 hours a day and managed by three experts, and (2) voluntary reporting of the number and type of regional anesthesia procedures performed using pocket booklets. The 487 participants reported 56 major complications in 158,083 regional anesthesia procedures performed (3.5/10,000). Pertinent to this discussion, lidocaine spinal anesthesia was associated with more neurologic complications than bupivacaine spinal anesthesia (14.4/10,000 vs 2.2/10,000). Most neurologic complications were transient.

As mentioned above, spinal cord injury may occur through numerous mechanisms. This lecture however will concentrate on the mechanical causes as well as the maneuvers that may limit the incidence of this complication. Mechanical injury may be direct, due to needle or catheter placement or indirect as a result of spinal cord ischemia secondary

to hematoma and abscess formation, or anatomical abnormalities such as spinal stenosis. It should be stressed that once mechanical damage has occurred, the spinal cord is more susceptible to further damage by agents that would under normal circumstances not be regarded as noxious, e.g. local anesthetics and epinephrine⁽⁹⁾.

Direct trauma to the spinal cord and/or spinal nerves can occur during the placement of the spinal or epidural needle as well as an epidural catheter. A number of issues have been addressed that, if not taken into account, may increase the risk of nerve damage (Table II).

A common and frequently overlooked mechanism is misidentification of the vertebral interspace. The intercrestal line (Tuffier's Line) is assumed to pass through either the body of L4 or the L4-5 interspace whereas the lower end of the spinal cord, or conus medullaris is assumed to reach the lower border of L1. With these two assumptions in mind, it is common practice to insert the spinal or epidural needle at either L4-5, L3-4, or L2-3 vertebral interspaces with the assumption that the needle is being inserted below the termination of the spinal cord.

Broadbent et al examined the anaesthetists' ability to identify correctly a marked lumbar interspace in 100 patients undergoing spinal magnetic resonance imaging scans⁽¹⁰⁾. Using ink, one anaesthetist marked an interspace on the lower spine and attempted to identify its level with the patient in the sitting position. A second anaesthetist attempted to identify the level with the patient in the flexed lateral position. A marker capsule was taped over the ink mark and a routine scan performed. The actual level of markers ranged from one space below to four spaces above the level at which the anaesthetist believed it to be. The marker was one space higher than assumed in 51% of cases and was identified correctly in only 29%. Accuracy was unaffected

by patient position (sitting or lateral), although it was impaired by obesity ($p = 0.001$) and positioning of the markers high on the lower back in non-obese patients ($p < 0.001$). The spinal cord terminated below L-1 in 19% of patients. This, together with the risk of accidentally selecting a higher interspace than intended for intrathecal injection, implies that spinal cord trauma is more likely when higher interspaces are selected. Based on these findings, Reynolds, in an editorial in Anaesthesia, suggests that the L3-4 interspace, one that is popular for lumbar epidural catheter insertion in labour, is the highest interspace "that all good anaesthetists should aim for (when performing) spinal anaesthesia"⁽¹¹⁾. This suggestion is particularly important as, in Broadbent's study, *experience did not improve accuracy of identification*.

Hogan used data aggregated from cadaver dissections and radiographic studies to determine to what extent Tuffiere's Line was a true indicator of the L4-5 interspace as well as the relationship between Tuffiere's Line and the conus medullaris⁽¹²⁾. He demonstrated that Tuffiere's line may lie at the modal level of the L4-5 interspace in 35% of individuals, but in the remainder its distribution extends from L3-4 to the L5-S1 interspace. Broadbent and her colleagues have pointed out that anatomists have long established that the tip of the conus may lie below the body of L1 in up to 58% of adults and below L1-2 in about 42%⁽¹⁰⁾. Thus, even if Tuffiere's Line is correctly identified, there are still a small percentage of patients in whom the needle may be inserted at a level that is above the termination of the spinal cord.

Hamandi et al reported five patients with damage to the distal spinal cord following spinal anesthesia⁽¹³⁾. The patients developed leg weakness and sensory disturbance. MRI of the lumbosacral spine showed an abnormal area of high signal within the conus medullaris in all patients. Symptoms and signs persisted at 1- to 2.5-year follow-ups. The authors conclude that possible factors leading to spinal cord injury include *incorrect needle placement* and type of needle used (Table III).

Kim et al analyzed the position of the conus medullaris and Tuffier's line in the same patient population, to correlate this position with age and sex, and to determine an objective guide for the selection of a safe intervertebral space during spinal block⁽¹⁴⁾. The conus medullaris and Tuffier's line (median [range]) were positioned at L1-lower (T12-upper to L3-upper) and L4-5 (L3-4 to L5-S1), respectively. The distance between the conus medullaris and Tuffier's line (mean \pm SD [range]) was 12.6 ± 1.9 [7-18] segments, which corresponded to the height of approximately three vertebral bodies and intervertebral spaces. In no case did Tuffier's line overlap with the conus medullaris. During spinal block, there seems to be a safety margin of 2-4 vertebral bodies and intervertebral spaces between the conus

Table II. Potential causes of mechanical damage to the spinal cord/nerve secondary to needle and/or catheter trauma.

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| <ol style="list-style-type: none"> (1) Misidentification of vertebral interspace <ul style="list-style-type: none"> • Normal weight patients • Magnified in obese patients (2) Termination of the spinal cord below the expected level (3) Incomplete fusion of the ligamentum flavum (4) Off-midline needle insertion (spinal nerve injury) <ul style="list-style-type: none"> • Paravertebral or lateral approach • Unrecognized during midline approach (5) Misinterpretation and/or mismanagement of paresthesia (6) Lack of paresthesia/pain in awake patients (7) Vascular injury (8) Anterior spinal artery syndrome |
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Table III. Spinal cord damage following spinal anesthesia⁽¹³⁾.

| Case # | Needle Type | Intended level | MRI abnormality | Termination of conus medullaris |
|---------|---------------------|----------------|-----------------|-----------------------------------|
| 1 (C/S) | 24G Pencil point | L2-3 | T12-L1 | Lower border L1 vertebral body |
| 2 (TKA) | 26G Unknown | L3-4 | T12 | Middle L1 vertebral body |
| 3 (TKA) | 25G CSE | L3-4 | T12-L1 | Lower border L1 vertebral body |
| 4 (C/S) | 25G Pencil point | L3-4 | L1-T9 | Lower border L1 vertebral body |
| 5 (THA) | 26G Unknown | L2-3 | T12 | T12-L1 interspace |

medullaris and Tuffier's line, which is consistent regardless of sex or presence of transitional vertebra. However, because the conus medullaris and Tuffier's line become closer with age and the clinical use of Tuffier's line requires palpation through subcutaneous fat, *caution must be exercised regarding selection of the intervertebral space, especially in the aged and obese population.*

The accuracy of clinical determination of the lumbar spinal interspace level, using surface ultrasound imaging as control in obstetric anaesthesia was assessed by Schlotterbeck et al⁽¹⁵⁾. At the anaesthesia follow-up visit, women who had received lumbar neuraxial anaesthesia during labour were prospectively included. The intervertebral level of needle insertion, located by the needle scar position, was identified by ultrasonography and compared with the clinical level reported on the chart by the anaesthetist who performed the block. *The clinical puncture level was accurate in 36.4% of the 99 patients studied.* Ultrasound examination showed the puncture level to be more cephalad than the level noted in the anaesthetic record in almost 50% of patients. In 15% of patients, the puncture level was more caudad than the anaesthetist had assessed. Factors including type of anaesthesia, indication, time period, level of anaesthetic experience, BMI, and spinal pathology did not seem to influence the frequency of errors. The authors that these findings highlight the potential for serious complications associated with the performance of neuraxial blocks above the spinous process of L3 in the parturient.

Nerve damage may be caused by the local anaesthetic infiltration needle if inserted above the termination of the spinal cord. Absalom et al. describe a case of spinal cord injury caused by direct trauma from a local anaesthetic in-

filtration needle⁽¹⁶⁾. During local anaesthetic infiltration before placement of an epidural catheter, the patient suddenly rolled over onto her back, causing the infiltrating needle to advance all the way to its hub. She immediately showed signs of spinal cord injury, confirmed by MRI scan. However, her neurological status gradually improved, and on discharge she was able to walk, with a sensory deficit localized to her left foot.

Anatomical variations may further complicate the issue and increase the risk of nerve damage. These include incomplete fusion of the ligamentum flavum in the cervical and thoracic region^(7,18), as well as a decrease in the distance between the ligamentum flavum and the dura from 4-5 mm in the lumbar region to 1-2 mm in the thoracic area⁽¹⁹⁾. In the former situation the lack of tactile feedback during epidural needle placement may be misleading while in the latter case the margin of safety is significantly reduced.

Paresthesia elicited during performance of spinal and epidural anesthesia is common and may be associated with an increased risk of persistent paresthesia^(4,5,8,20). However, it is a common misconception that in an awake or mildly sedated patient, nerve damage cannot occur without a significant paresthesia on needle and/or catheter insertion. While these symptoms are indicators of possible nerve trauma, they are not always perceived and/or reported by the patient^(21,22). On the other hand, direct injections into the spinal cord will invariably result in severe pain^(13,23).

The performance of regional anesthesia, in adult patients under general anesthesia is a controversial topic⁽²⁴⁻²⁶⁾ particularly with the advent of ultrasound-guided regional anesthesia⁽²⁷⁻³⁰⁾, but is beyond the scope of this discussion.

Neal published suggested guidelines to prevent, (or at least limit the risk of) mechanical spinal cord injury. The importance of meticulous attention to detail coupled with a cautious approach to the performance of central neuraxial blockade cannot be overstressed.

Guidelines to prevention of mechanical spinal cord injury⁽⁹⁾. (Modified).

1. Meticulous technique
 - a. Strict asepsis
 - b. Careful identification of spinal vertebral level
 - c. Slow and controlled needle and catheter advancement
2. Placement of blocks in awake or minimally sedated patients, i.e. avoid placing in anesthetized or heavily sedated patients

3. Realization that radiographic or ultrasound guidance may aid precise needle placement in some blocks but not always prevent injury
4. Avoidance of neuraxial techniques in patients with compromised coagulation⁽³¹⁾
5. Consideration of risk-to-benefit ratio before placing neuraxial blocks in patients with known epidural tumor mass and spinal stenosis
6. Realization that mechanical spinal cord injury is possible despite exacting technique and vigilance. Many conditions that theoretically predispose a patient to neuraxis injury are unknown to the patient or to the anesthesiologist.

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