

Spinal Anesthesia: Mechanisms, Physiological Effects, Contraindications, and Complications

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Abstract:

Spinal anesthesia is a widely used central neuraxial technique that produces temporary sensory, motor, and sympathetic blockade through the injection of local anesthetic, with or without adjunctive drugs, into the subarachnoid space. It provides effective anesthesia and analgesia while avoiding many of the respiratory complications associated with general anesthesia and is associated with high patient satisfaction. Its effects result primarily from blockade of neural transmission at the spinal nerve roots and spinal cord through mechanisms involving sodium and calcium channels as well as modulation of neurotransmitter activity. Spinal anesthesia can be performed using midline or paramedian approaches, with the latter being particularly useful in patients with difficult spinal anatomy. Despite its clinical advantages, spinal anesthesia produces important physiological effects, particularly sympathetic blockade that may reduce systemic vascular resistance, venous return, cardiac output, and heart rate. Potential complications include hypotension, hypothermia, urinary retention, nausea and vomiting, spinal hematoma, neurological complications, failed neuraxial block, total spinal anesthesia, and post-dural puncture headache. Therefore, appropriate patient selection, careful technique, adequate monitoring, and early recognition and management of complications are essential for the safe and effective use of spinal anesthesia.

Keywords: Spinal anesthesia; Neuraxial anesthesia; Subarachnoid block; Local anesthetics; Sensory blockade; Motor blockade; Sympathetic blockade; Spinal anesthesia complications.

Introduction:

Spinal anesthesia is a central regional block approach characterized by temporary sensory, motor, and sympathetic block generated by injection of local anesthetic into the subarachnoid space with or without additional drugs. Through the subarachnoid space, spinal anesthesia (SA) inhibits the nerve roots. SA is one of the most widely utilized anesthetic methods. It is chosen by both patients and anesthesiologists due to the benefits of giving anesthesia and analgesia simultaneously, avoiding major respiratory problems of general anesthesia (GA), and achieving high patient satisfaction(1).

Injecting local anesthetics into the subarachnoid space allows access to action sites within the spinal cord as well as the peripheral nerve roots. Local anesthetics have several potential activities within the spinal cord at various places. Local anesthetics, for example, can block sodium channels and limit the creation and propagation of electrical activity inside the dorsal and ventral horns. Other neuronal ion channels in the spinal cord, such as calcium channels, are also critical for afferent and efferent neural activity (2).

Spinal injection of N-type calcium channel blockers causes cell membrane hyperpolarization, resistance to electrical stimulation from nociceptive afferents, and severe analgesia. Local anesthetics may have similar effects on neuronal calcium channels, which may contribute to the analgesic effects of centrally given local anesthetics(3).

In the dorsal horn of the spinal cord, many neurotransmitters are involved in nociceptive transmission. Substance P is a neurotransmitter that affects nociception from C fibers and is released by dorsal root ganglion cells' presynaptic terminals. Local anesthetics in concentrations that occur during spinal and epidural anesthesia limit substance P release and binding to its receptor in the central neuroaxis in a noncompetitive manner(4).

Local anesthetics also influence inhibitory neurotransmitters that may be critical for nociceptive processing in the spinal cord, such as -aminobutyric acid. By inhibiting uptake and elimination, local anesthetics can enhance the effects of -aminobutyric acid. As a result, in addition to ion channel blocking, spinal anesthesia may be partially mediated by complicated interactions at brain synapses(5).

Although spinal local anesthetics can block sodium channels and electrical transmission in spinal nerve roots, other processes may also be at work. It is theorized that a considerable portion of the sensory information supplied via peripheral nerves is carried via coding of electrical signals in after depolarization and after oscillations. Studies revealing loss of sensory nerve function following incomplete local anesthetic blocking provide evidence for this idea. The sense of skin warmth, for example, can be lost despite unhindered conduction of tiny fibers(5).

Furthermore, a surgical depth of epidural and spinal anesthesia can be acquired with relatively small alterations in the anaesthetized area's somatosensory evoked potentials. Previous research has shown that sub-blocking concentrations of local anesthetic reduce naturally occurring after depolarization and after-oscillations without impacting action potential conduction appreciably. Thus, disruption of electrical information coding by local anesthesia may be a major mechanism for spinal nerve root block during spinal anesthesia (5).

Positioning of spinal anesthesia: (Figure 1)

Lumbar puncture (LP) can be done with the patient sitting upright or resting on his or her left side or right side. If the lumbar puncture is done while the patient is sitting, make sure he is leaning forward and perpendicular to the bed/trolley. Place the patient's feet on a low stool and her elbows on her thighs to open the spaces between the spinous processes(6).

Make sure your hips and shoulders are square - in line with each other and perpendicular to the bed - for the left lateral position. Then, in fetal position, stretch the spine as much as possible to free up the spaces between the spinous processes. The line (Tuffier's line) that connects the highest point of both iliac crests is level with L4. Midway along this line, there is a gap between L3 and L4 that is usually used for lumbar puncture and is easily accessible.

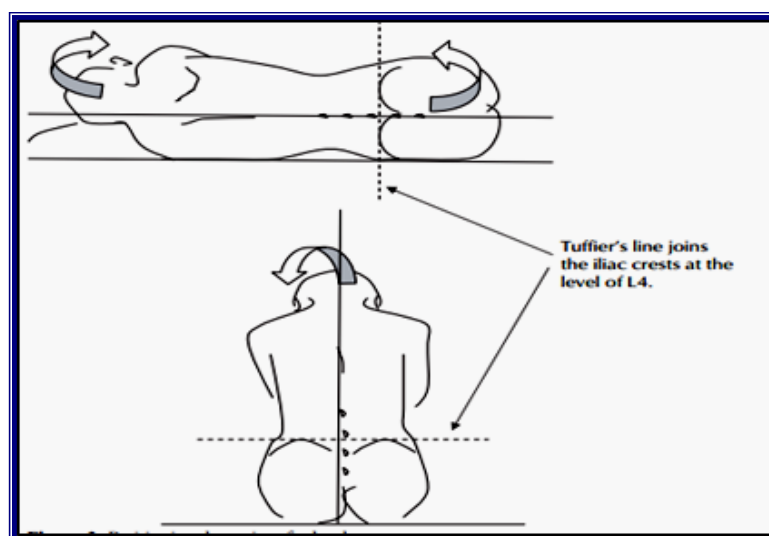


Figure (1): Positioning the patient for lumbar puncture(7).

Anatomic approach for spinal anesthesia: (Figure 2)

◆ **Midline approach:**

The spinal approach to the intrathecal area is midline with a straight line shot in the midline approach. Following lidocaine infiltration, the spinal needle is inserted into the skin, tilted somewhat cephalad. The needle passes through the skin, subcutaneous fat, supraspinous ligament, interspinous ligament, ligamentum flavum, epidural space, dura mater, and lastly the arachnoid mater (7).

◆ **Paramedian approach:**

It is the preferred approach in some individuals with complicated anatomy, such as kyphoscoliosis or severe arthritis. A skin wheel is raised one cm lateral and one cm caudad to the lower margin of the spinous process of the chosen lumbar vertebra using palpating fingers. The spinal needle is then inserted in a cephalo-medial orientation at 10 to 15 degrees of the sagittal plane. The needle will go through the skin, subcutaneous tissue, ligamentum flavum, epidural space, dura mater, and arachnoid mater before ending in the arachnoid mater (2).

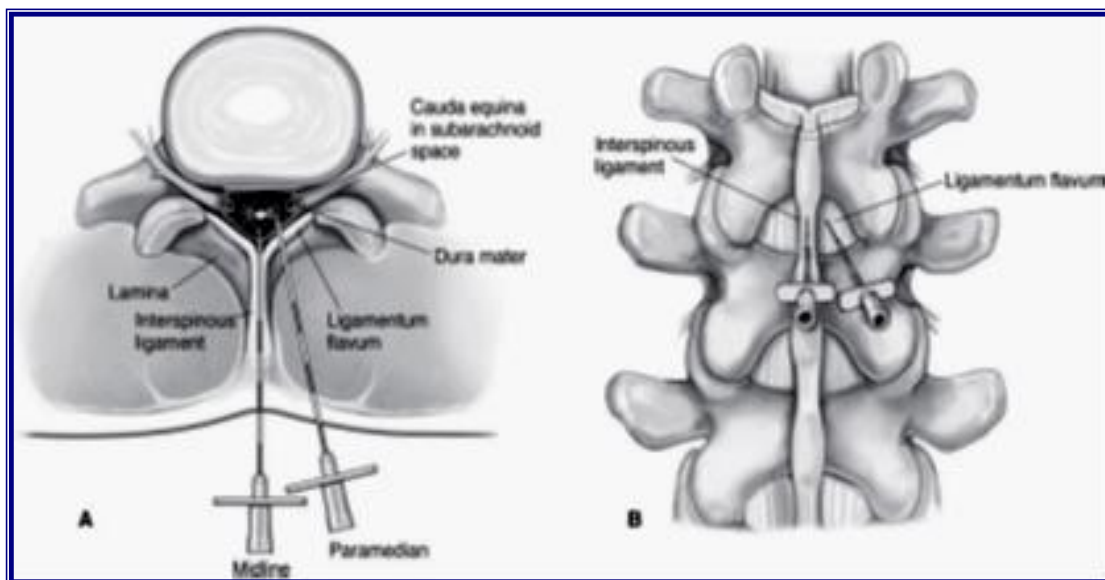


Figure (2): Spinal anesthesia via the midline and paramedian approaches.
(A): Transverse view. (B): Posterior view (8).

Assessing Level of Blockade:

A blunted needle or a piece of ice might be used to determine the amount of sensory block. T4 dermatome levels are normally enough.

The modified Bromage scale has traditionally been used to determine the degree of motor block associated with spinal anesthesia(9):

- 1 = Complete block (unable to move feet or knees).
- 2 = Almost complete block (able to move feet only).
- 3 = Partial block (just able to move knees).
- 4 = Detectable weakness of hip flexion (between scores 3 and 5).
- 5 = No detectable weakness of hip flexion while supine (full flexion of knees).
- 6 = Able to perform partial knee bend

Effects of Spinal Anesthesia (7,10):

A-Somatic blockade:

Central sensory and motor blockade is commonly used to relieve pain and relax skeletal muscles.

B-Autonomic blockade:

The disruption of autonomic signals to various organ systems mediates the majority of the visceral symptoms of central blockage.

1. Cardiovascular effect

Spinal anesthesia reduces cardiac output and systemic vascular resistance in the cardiovascular system. Sympathetic fiber blockade prevents vasoconstriction and causes blood pooling in the splanchnic system and lower extremities. A reduction in venous return (preload) leads to a reduction in cardiac output. If local anesthetic spread reaches T1-T4 levels, the cardiac accelerator fibers are blocked, resulting in a drop-in heart rate, which further reduces cardiac output.

In addition to those changes, three reflexes are associated with spinal anesthesia which can worsen bradycardia and even lead to cardiac arrest:

- A-** The pacemaker reflex involves cells in the sinoatrial node that depolarize in accordance to the degree of strain. An abrupt drop in venous return causes sinoatrial cells to stretch and depolarize, reducing the heart rate.
- B-** The baroreceptor reflex involves receptors in the right atrial wall and the vena cava-atrial junction. Increases in venous return send signals to the cardiac accelerator fibers via the vagus nerve, resulting in an increase in heart rate. Conversely, a decrease in venous return decreases stimulation of the baroreceptors.
- C-** Bradycardia is caused by the Bezold-Jarisch reflex, which links a decrease in left ventricular volume to an increase in vagal output via the vagus nerve.

2. Respiratory effects:

Overall, spinal anesthesia has little direct influence on the respiratory system. Even at mid- to high-thoracic anesthesia levels, respiratory indices such as minute ventilation, tidal volume, and mean inspiratory flow rate remain unaltered. The same can be said for pulmonary gas exchange. A slight decrease in vital capacity, maximal expiratory pressure, and flow has been seen, which has been linked to abdominal muscle weakness.

3. Central nervous system:

Spinal anesthesia has frequently been shown to lessen hypnotic and sedative requirements via processes that are still unknown. De-afferentiation phenomenon (blockade of spinal nerves reduces afferent input to the brain, making the reticular activating system more sensitive to sedative/hypnotic drugs), rostral spread of local anesthetics within the CSF (with direct blocking effects of local anesthetics on brain centers), and absorption of local anesthetics leading to increased systemic levels are theories to explain this finding.

4. Urinary system:

The sacral spinal nerves (S2-S4) control the bladder and urethral sphincters. Blocking such segments eliminates the urge to urinate, increasing the risk of bladder overdistention and postoperative urine retention in the presence of long-acting local anesthetics. Overdistention of the bladder can cause discomfort and hypertension, while stimulation of vagal afferents can cause bradycardia and hypotension. As a result, the implantation of an indwelling urinary catheter or ultrasonography bladder volume monitoring may be considered.

5. Thermoregulation:

In general, general anesthesia impairs the body's thermo-regulatory capacity more than spinal anesthesia; nevertheless, vasodilation from sympathetic blocking promotes a re-distribution of heat from core tissues (head, trunk, internal organs) to skin, upper and lower extremities. Furthermore, in the blocked segments, shivering thresholds are reduced, limiting the ability to create heat.

6. Gastrointestinal effects:

When sympathetic inputs to the gastrointestinal tract are blocked at T6-L1 levels, hyperperistalsis results from unopposed parasympathetic stimulation. Nausea and vomiting may occur as a result of this effect (or, in other situations, as a result of hypotension and hypoperfusion).

7. Renal effects:

The blood supply to the renal system is unaffected by spinal blockage. As long as perfusion pressure is maintained, autoregulation provides enough blood flow to the kidneys. Urinary retention can develop when autonomic bladder control is lost. Local anesthetics inhibit the bladder's detrusor function. Normal function will not be restored until sensory function is restored to S3. Urinary retention is more likely in patients who have had an enlarged prostate in the past. Urinary retention should be checked in patients (2).

8. Metabolic and endocrine effects:

The hypothalamic neuroendocrine response is exacerbated by surgery, pain, and anxiety. Catabolic hormones such as catecholamines, adrenocorticotrophic hormone (ACTH), cortisol, antidiuretic hormone (ADH), glucagon, and aldosterone are reduced by spinal anesthesia (2).

Contraindications of spinal anesthesia:

Table (1): Showing contraindications of spinal anesthesia(2).

1.Absolute	2. Relative	3.Controversial
■ Infection at the site of injection. ■ Patient refusal. ■ Coagulopathy or other bleeding diathesis. ■ Severe hypovolemia. ■ Increased intracranial pressure. ■ Severe aortic stenosis. ■ Severe mitral stenosis.	■ Sepsis. ■ Uncooperative patient. ■ Severe spinal deformity. ■ Demyelinating lesions. ■ Stenotic valvular heart lesions. ■ Preexisting neurological deficits	■ Prior back surgery at the site of injection. ■ Inability to communicate with patient. ■ Maneuvers that compromise respiration. ■ Prolonged operation. ■ Major blood loss. ■ Complicated surgery.

Complications of Spinal Anesthesia:

◆ Hypotension:

When the sympathetic chain becomes blocked, especially when greater dermatome levels are required, hypotension is an unavoidable consequence of spinal anesthesia. A reduction in blood pressure may cause nausea and vomiting, indicating spinal cord ischemia, resulting in an unfavorable state for the patient and operating personnel(11).

◆ **Hypothermia:**

Following neuraxial anesthesia, it is typical to experience a drop in body temperature. Patients are unable to control the reduction in core temperature because subarachnoid local anesthetic injection inhibits all signals of cutaneous temperature. Vasodilation caused by sympathetic blocking improves epidermal blood flow, allowing for consistent cooling of the body's core temperature. When prepping the skin for surgery with antiseptic solutions, evaporation from the surgical field and irrigation solutions, or rapid fluid infusion, may also contribute to hypothermia during surgery (12).

Shivering can be triggered by a drop in core temperature, especially during the postoperative phase, which increases oxygen demand. Special care should be taken to reduce this physiologic stress, especially in pediatric, obstetric, and elderly patients, because it can have catastrophic repercussions such as inadequate perfusion to essential organs, coronary ischemia, and infection. To reduce the severity of this problem, it is critical to warm the patient with blankets, surgical thermal beds, forced air heaters and prior warming of irrigation solution, intravenous treatments and blood products (12).

◆ **Urinary retention:**

Bladder distension during the postoperative period causes discomfort in patients and, if not relaxed, leads to more serious consequences, including irreversible detrusor muscle injury. Because spinal anesthesia blocks all afferent nerve fibers, the patient is unable to sense bladder distension or urine urgency(13).

◆ **Nausea and vomiting:**

Because of a drop in blood pressure, which causes hypoperfusion of the medulla or cephalad, opioids travel to the chemoreceptor trigger zone. Because morphine tends to move cephalad within CSF, the incidence of nausea and vomiting after neuraxial opioid is substantially higher with the comparatively poorly lipid soluble morphine compared to more lipid soluble drugs such as fentanyl. The use of an opioid antagonist such as naloxone or the opioid agonist-antagonist nalbuphine is the most effective treatment for opioid-induced nausea. Nausea and vomiting caused by hypotension are addressed by treating the hypotension with vasopressors and using antiemetics(14).

◆ **Hematologic complications:**

Spinal hematoma after spinal anesthesia is a serious complication that necessitates immediate surgical surgery to prevent lasting neurological damage. The incidence of this condition has traditionally been considered as 1 in 220,000 patients undergoing spinal anesthesia, however the real prevalence remains unclear and is thought to be increasing. As risk factors, advanced age, female gender, patients receiving coagulation-inhibiting medicines, difficulty completing block and installation of the indwelling epidural catheter are mentioned(15).

When there is an unexpected increase in the duration of motor block or a delay in recovery, the presence of hematoma is commonly considered. Neurosurgery must be performed within eight hours of the epidural hematoma to restore motor functions without causing neurologic damage. If neurologic injury is feared, imaging examinations, such as computed tomography or, preferably, MRI, should be performed as soon as possible (13).

◆ **Neurological complications:**

The occurrence of significant neurological impairments after spinal anesthesia is uncommon. A prospective analysis of approximately 40000 cases found that the incidence of significant neurological impairments was around 0.05%. Despite the low prevalence, many patients refuse spinal anesthesia because they are afraid about this consequence. These neurological impairments are(1) :

- A. Aseptic meningitis: This is the most innocuous neurologic consequence. Fever, nuchal stiffness, and photophobia are typical symptoms that appear within 24 hours of spinal anesthesia. CSF microscopic examination reveals polymorphonuclear leukocytosis; bacterial CSF cultures are negative. Aseptic

meningitis is solely treated symptomatically and usually cures within a few days.

- B. Cauda equina syndrome: This occurs after the neuraxial blockage has been resolved. This syndrome may be permanent, or it may recede gradually over a period of weeks or months. It is distinguished by a sensory deficiency in the perianal area, urine and fecal incontinence, and various lower extremity deficiencies.
- C. Adhesive arachnoiditis: The most significant neurologic problem. This reaction usually manifests itself several weeks, if not months, after spinal anesthesia. The syndrome is distinguished by a progressive progression of sensory impairments and lower-limb motor restriction.
- D. There are a meningeal proliferation and vasoconstriction of the spinal cord vasculature. After a protracted period of arterial hypotension, spinal cord ischemia and infarction may occur. The presence of epinephrine in anesthetic solutions has been linked to a decrease in blood flow to the spinal cord. Traumatic spinal cord and nerve root injury is a rare cause of neurologic impairment.
- E. Transient neurological effects following 5% lidocaine spinal anesthesia. It is characterized by significant radicular back pain that is not accompanied by sensory or motor impairments. These symptoms went away on their own within a few days. The incidence was higher with lidocaine than with bupivacaine, and it was more common in individuals undergoing hip or knee surgery in the flexed position.

◆ Failed neuraxial block:

Inadequate analgesia/anesthesia following epidural, spinal, or mixed spinal epidural anesthesia is considered a failed neuraxial block. Inadequate drug dosing, technical issues such as needle movement during injection, incomplete entry of the needle opening into the subarachnoid space, injection of anesthesia solution into a nerve root sleeve, or patient factors such as the patient having soft spinal ligaments that affect the ability to detect the sense of loss of resistance or curvature spine abnormalities that make ducting the midline and the interlaminar spaces more difficult (2).

◆ Total Spinal Anesthesia:

As a result of a large distribution of local anesthetics enclosing the entire spinal cord and, in some cases, the brain, acute cardiopulmonary collapse (hypotension, bradycardia, respiratory arrest) occurs. The goal of treatment is to support the circulatory disturbance with vasopressors, inotropics, chronotropic, and positive pressure ventilation as a backup for the pulmonary embarrassment(16).

◆ Post-Dural-puncture headache [PDPH]:

Post-Dural puncture headache is a painful consequence that primarily affects middle-aged women and the obstetric population. Other risk factors include a lower BMI, a history of PDPH, and the existence of recurrent headaches(13).

CSF seeping from the Dural hole may cause PDPH. The following mechanisms explain this: A reduction in intracranial pressure induces traction of pain sensitive cranial structures, a decrease in CSF volume may promote compensatory cerebral vasodilatation, and adenosine receptor activation may generate cerebral vasodilatation (13).

The therapy protocol is determined by the severity of the PDPH. Bed rest and oral or intravenous hydration replenishment are used as conservative treatments. Analgesics, vasoconstrictors, and medicines that stimulate CSF production are examples of pharmacological therapy. Gabapentin has also been used to treat PDPH with success. Epidural morphine has also been shown to be useful; however it can leak into the intrathecal space through the Dural hole and has well-known side effects such as itching, nausea, and vomiting. Despite controversy, the epidural blood patch [EBP] remains the gold standard for treating PDPH (13).

REFERENCES:

1. Di Cianni S, Rossi M, Casati A, Cocco C, Fanelli G. Spinal anesthesia: An evergreen technique. Vol. 79, *Acta Biomedica de l'Ateneo Parmense*. 2008. p. 9–17.
2. Butterworth J. Teaching Method: On Campus Text Book Title Morgan and Mikhail's Clinical Anesthesiology Edition 6th Edition.
3. Olschewski A, Hempelmann G, Vogel W, Safronov B V. Blockade of Na⁺ and K⁺ currents by local anesthetics in the dorsal horn neurons of the spinal cord. *Anesthesiology*. 1998;88:172–9.
4. Hodgson PS, Neal JM, Pollock JE, Liu SS. The neurotoxicity of drugs given intrathecally (Spinal). Vol. 88, *Anesthesia and Analgesia*. Lippincott Williams and Wilkins; 1999. p. 797–809.
5. Liu SS, McDonald SB. Current issues in spinal anesthesia. Vol. 94, *Anesthesiology*. Lippincott Williams and Wilkins; 2001. p. 888–906.
6. Hewson DW, Bedfordth NM, Hardman JG. Spinal cord injury arising in anaesthesia practice. Vol. 73, *Anaesthesia*. Blackwell Publishing Ltd; 2018. p. 43–50.
7. Salinas F V., Sueda LA, Liu SS. Physiology of spinal anaesthesia and practical suggestions for successful spinal anaesthesia. Vol. 17, *Best Practice and Research: Clinical Anaesthesiology*. 2003. p. 289–303.
8. Ng KT, Lim WE, Teoh WY, Shariffuddin II, Ti LK, Abidin MFBZ. Paramedian versus midline approach of spinal anesthesia: a systematic review and meta-analysis with trial sequential analysis. *J Anesth*. 2024 Feb;38:65–76.
9. Craig D, Carli F. Bromage motor blockade score—a score that has lasted more than a lifetime. *Canadian Journal of Anesthesia/Journal canadien d'anesthésie*. 2018;65:837–8.
10. Operater. <https://www.nysora.com/techniques/spinal-anesthesia-2/>. Spinal Anesthesia - NYSORA.
11. Wong CA. Spinal anesthesia-induced hypotension: is it more than just a pesky nuisance? Vol. 223, *American Journal of Obstetrics and Gynecology*. Mosby Inc.; 2020. p. 621–3.
12. Crowley LJ, Buggy DJ. Shivering and Neuraxial Anesthesia. Vol. 33, *Regional Anesthesia and Pain Medicine*. 2008. p. 241–52.
13. Apan A, Cuvaş Apan Ö. Complications in Spinal Anaesthesia. In: *Topics in Spinal Anaesthesia*. InTech; 2014.
14. Ebrie AM, Woldeyohanis M, Abafita BJ, Ali SA, Zemedkun A, Yimer Y, et al. Hemodynamic and analgesic effect of intrathecal fentanyl with bupivacaine in patients undergoing elective cesarean section; a prospective cohort study. *PLoS One*. 2022 Jul;17.
15. Horlocker TT. Regional anaesthesia in the patient receiving antithrombotic and antiplatelet therapy. *Br J Anaesth*. 2011 Dec;107 Suppl 1:i96-106.
16. Cook TM, Counsell D, Wildsmith JAW. Major complications of central neuraxial block: Report on the Third National Audit Project of the Royal College of Anaesthetists. *Br J Anaesth*. 2009;102:179–90.