We are IntechOpen,
the world's leading publisher of
Open Access books
Built by scientists, for scientists

5,600
Open access books available

138,000
International authors and editors

170M
Downloads

154
Countries delivered to

Our authors are among the

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS
BOOK CITATION INDEX
INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Spinal Anesthesia for Lower Level Spine Surgery

W. Scott Jellish and Steven Edelstein

Additional information is available at the end of the chapter

1. Introduction

Lumbar discectomy is the most commonly performed spinal operation in the United States with more than half a million procedures performed annually. In addition, spinal anesthesia for surgical analgesia in these procedures has been established as an accepted technique for many years. With the refinement in surgical technique for lumbar discectomy, that has now made the procedure relatively non-invasive, spinal anesthesia plays an even more important role.

The original laminectomy and discectomy was performed by Mixter and Barr in 1934 [1]. Most surgeons perform a modified microdiscectomy originally described by Williams [2]. With the use of high-powered microscopes, the anatomy is better visualized and incisions are much smaller with less tissue and bone disruption. There are alterations to the standard microdiscectomy including laser disc removal, endoscopic discectomy and intradiscal electrothermal treatment. However, the microdiscectomy remains the procedure with the highest success rate. Lumbar laminectomy or discectomy is performed with the patient in the prone or lateral decubitus position. A midline paramedian incision is created and the lumbodorsal fascia is incised. Periosteal dissection exposes the laminae that are removed as necessary to provide access to the thecal sac and nerve roots. The nerve roots are retraced medially to expose the posterior longitudinal ligament that covers the intervertebral discs. The discectomy is performed by incising the ligament and removing disc material with a forceps. The laminar resection can also be extended to provide canal decompression in cases of spinal stenosis.

It is important to note that the lumbar spine has the largest vertebral bodies and bears the greatest weight. The center of gravity of the body is approximately 1 cm behind the sacral promontory that, in turn, places the entire weight of the body directly on L_{4-5} and L_{5-S1}. With aging, the discs tend to become less fluid and more fibrocartilagenous, with little difference

between nucleus and annulus. The discs are subject to pathologic changes that may lead to herniation of the nucleus pulposus and cause compression of the neural elements.

Subsequent removal of the disc or lamina with the assistance of loops or microscopes typically has a surgical duration of approximately two hours. As such, this has made spinal anesthesia an attractive choice for the anesthetic technique in these patients. This chapter will review the evidence supporting the utilization of this technique as well as the possible risks associated with neuraxial anesthesia and prone positioning.

2. Technique of spinal anesthesia for spine surgery

Briefly, once a decision has been made to proceed with spinal anesthesia several items must be performed in order to have a successful outcome. Knowing the level of surgical anesthesia required is extremely important since this will determine whether the patient can comfortably undergo the procedure and avoid the hemodynamic consequences of surgical stimulation. Of course, it is also essential that the area of coverage will provide relief from painful stimuli as well.

Anesthesia levels for lumbar surgery can be easily achieved with hyperbaric or isobaric local anesthetics. Typically, for L1-L5 surgery a dermatomal sensory level of at least T6-T8 will be required. Though this is higher than the level of the operative site, the higher level will allow for the surgery to take place and, depending on the local anesthetic selected, allow for a slow regression of surgical anesthesia coverage. In most instances, the patient is placed in the full prone position. The prone knee chest position and the horizontal side position have also been used. These positions are of importance since the spread of local anesthetic may be different depending on this position and also the baricity of the local anesthetic solution. After placing the spinal, the patient should be positioned supine with the level allowed to set before final positioning is achieved.

Bupivacaine appears to be the agent of choice since it provides adequate duration of coverage in comparison to other agents such as lidocaine. If lidocaine is selected, it is conceivable that regression of sensory coverage could occur shortly after positioning and draping of the patient. In addition, some practitioners will also select additives to the local anesthetic though the risk/benefits of these will be discussed later. A variety of agents have been used in lumbar surgery, all with varying degrees of success including opioids, epinephrine, phenylephrine, neostigmine and clonidine. Final selection of any and all additives will depend on the clinical situation and the physical status of the patient.

In most instances, the patient will have the spinal anesthetic placed prior to prone positioning. Usually the patient will be administered 400-600 ml of a balanced salt solution to expand intravascular volume prior to spinal placement. The preference for placement of the spinal block for many practitioners is to place the patient in the seated position. The seated position allows for better delineation of the overall spinal anatomy and helps to ascertain the midline, especially in larger individuals. In some instances, the patient can be placed in the lateral

decubitus position for spinal placement. The back is prepped and draped in a sterile fashion and the best interspace, either L2-3, 3-4 or 4-5, is identified and 2-4 ml of 2% lidocaine is injected to anesthetize the area where the spinal needle will be inserted. Most practitioners will use a 24g or 25g pencil-point spinal needle placed through an introducer and advanced until free flow of CSF is observed from the hub of the needle. The spinal anesthetic can also be accomplished with the use of a 22 gauge Quincke needle. Once subarachnoid placement is confirmed, either 2-3 ml of 0.5% plain bupivacaine or 1.5-2 ml of 0.75% hyperbaric bupivacaine is injected into the subarachnoid space. The patient is returned to the supine position, and once a T8-10 level is obtained, the patient is rolled into the prone position and either placed on chest rolls, a Wilson or Andrews frame and allowed to self position their upper body for comfort.

3. Baricity issues

There has been some controversy over the preferred baricity of the local anesthetic for spinal anesthesia in lumbar surgery. Jellish, et al, [3] in their prospective study effectively utilized hyperbaric bupivacaine 0.75% with dextrose 8.5% to achieve levels of T6-T10. The study patients were required to stay supine after placement of the local anesthetic for approximately 10 minutes to fix the spinal level. Fixation of a hyperbaric spinal is required since typically the patients are placed in prone position. This is of particular importance given the fact that there are times when the head-down position is transiently performed as the patient is positioned on a frame or in knee-chest position.

If a hyperbaric solution is selected and adequate time for fixation has not been performed, the solution could track cephalad and lead to a higher level than what is required. This is also accentuated since the frame and/or knee-chest position required for the surgery eliminates the lordotic curves of the spine. The fixation of a hyperbaric spinal occurs when the solution is taken up by the spinal tissue and blood, especially the dextrose solution. This results in a change in solution from hyperbaric to isobaric and subsequent positioning has little to no effect [4].

Baricity of the spinal anesthetic has also been shown to affect both the quality of the anesthetic and the level of the block. Isobaric procaine/tetracaine spinal anesthesia has the same success profile with minimal complication compared to general anesthesia for spine surgery [5]. If the sensory level is adequate and ventilation is not impaired by a high block, spinal anesthesia provides good surgical conditions for spine surgery. Subjective dyspnea associated with a high spinal level may be accentuated with the patient in the prone position. Some clinicians believe isobaric spinal anesthetics could be the best choice because the dense low thoracic block may be routinely achieved with minimal hemodynamic consequences. Also, the effect of the isobaric agent is not affected by other factors like gravity or prolonged position. As such, patients that are placed in the knee chest position can be turned prone immediately after placement of the spinal as opposed to wait times of 10 minutes or longer for the block to set with a hyperbaric technique.

Plain isobaric bupivacaine was compared to hyperbaric bupivacaine to determine quality of block and cephalad spread in patients undergoing spinal surgery [6]. A 3 mL solution of isobaric 0.5% bupivacaine was administered to one group and 2ml of 0.75% bupivacaine was administered to the second group. All injections were performed within 5 seconds with the needle bevel facing cephalad. After turning supine for 10 minutes, the patients were turned prone to begin surgery. Time of onset for sensory and motor block was more rapid with hyperbaric bupivacaine. In addition, the final level achieved was higher with hyperbaric bupivacaine, compared to isobaric solution. Maximum heart rate change was similar in both groups but maximum blood pressure change was greater with the hyperbaric solution and this required a greater need for blood pressure and heart rate treatments. The dependent movement of hyperbaric solutions, and the level of the block achieved was always several denervations higher than the equivalent dose of isobaric solution. Even though sensory block is higher with hyperbaric local anesthetics, sympathetic block could be even higher. This explains the alteration in blood pressure observed with hyperbaric spinal anesthesia that is accentuated by turning prone. Thus, when using hyperbaric bupivacaine, meticulous determination of block level must be made before positioning the patient to avoid hypotension and bradycardia.

More breakthrough pain during spinal surgery has been noted with hyperbaric bupivacaine solutions compared to isobaric. This is thought to be due to the superiority of plain bupivacaine in suppressing slow conducting repetitive stimuli that is characteristic of low back pain [7].

Rung and colleagues [8] have suggested the utilization of isobaric bupivacaine 0.5% for providing adequate anesthesia. The group felt that the isobaric nature of the medication would help avoid the issues regarding positioning and unwanted rises in anesthetic levels. In addition, they also felt that the utilization of isobaric agents would speed the procedure since the patient could first be placed in prone position and then have the anesthetic administered. This would decrease the amount of time required for preparation and speed the onset of surgery.

Another study examined the use of 15 mg of 0.5% plain bupivacaine injected at the L_{2-3} interspace and either placing the patient in the prone knee chest position before placement of the spinal or after spinal placement positioning the patient supine and allowing the spinal level to be obtained before positioning prone. [9] The mean drop in systolic blood pressure was 30 mmHg in prepositioned patients compared to 13 mmHg with spinal placed before positioning. More ephedrine was needed when the spinal was placed post positioning to maintain blood pressure compared to the patients who had the block placed in the horizontal side position. This same knee chest group of patients also needed more atropine or glycopyrrolate to maintain heart rate. The investigators believed that placing the spinal block in the lateral horizontal position and allowing the patient to lie supine for 20 minutes produced less hypotension and bradycardia when compared to patients who had the block placed in the prone knee chest position because these patients had more time to accommodate for vasodilation of the lower limbs. The controversy over the ideal baricity has not been settled and either agent may be appropriate for the procedure.

Typically during the insertion of the spinal anesthetic a pencil-point needle such as a Whitacre is used versus the standard cutting Quincke type. Obviously this is utilized to avoid undue trauma to the dura via the cutting needle and causing a potential dural tear that could interfere with surgery (cerebral spinal fluid (CSF) in the field) as well as lead to postdural puncture headaches. In addition, other studies suggest that the pencil-point needles lead to a local inflammatory response that help with rapid dural closure [10].

The appropriate level of needle insertion will obviously be determined by the procedure and what disc is affected. There have been concerns about the utilization of spinal anesthesia in patients with pre-existing spinal disease; however in a report by Hebl et al., [11] it was felt that the history of spinal surgery did not increase the risk of technical complications or block success, but did make placement potentially more difficult. The group felt that midline or lateral approach may be especially difficult if there were bone grafting or posterior fusions since success would only occur if the block was performed at areas that were unfused.

Prone (for isobaric only), sitting or lateral approaches for spinal anesthesia insertion have all been described, however is must be kept in mind that ultimately the spinal should be placed above the level of any lumbar stenosis (and below the level of the cord) since very tight stenotic lesions may affect spread of local anesthetic [12].

4. Additional issues regarding agent selection

Currently procaine, lidocaine, mepivacaine, tetracaine, ropivacaine, levobupivacaine, bupivacaine are all approved in the US for intrathecal use. As mentioned previously, bupivacaine is typically the choice of agent due to its duration of action. Ropivacaine and L-bupivacaine (S-enantiomer of bupivacaine) have a less cardiotoxic profile compared to bupivacaine; however, the overall volume utilized in spinal anesthesia is so small that this is of little concern. Tetracaine, which is an ester-based local anesthetic may also be utilized and may be in prepared in isobaric, hypobaric or hyperbaric solutions. Typically, because fixation for tetracaine takes a long time, it is not routinely utilized.

Lidocaine has a long history of safe use, but its association with transient neurologic symptoms (TNS) would make it a potentially poor choice for lumbar spine surgery. TNS, initially described in 1993[13] presents with the onset of back and leg pain post-procedure. It has been associated with positioning and can be found with all local anesthetics, but has been reported most frequently with lidocaine. Still there is no definitive proof that the local anesthetics are the source for TNS and some studies have strongly encouraged the discontinuation of this term to avoid linking the previous clinical symptoms with the use of lidocaine [14].

It is well known that determinants for level of spinal analgesia depend on the dosage administered as well as the baricity [15]. The total dose of bupivacaine administered is very important since the concentration of the medication changes after mixing with the CSF and the change in concentration has on the quality or level of the spinal anesthesia [16]. In addition, other studies have determined that there is non-homogenous spread of local anesthetics in the CSF

[4]. Typically there is an epicenter of local anesthetic concentration and subsequent spread away from the site with decreasing levels of local. The decreased concentration at these sites leads to a variation in uptake of the anesthetic in the level of the cord.

5. Additives

As with most spinal anesthetics, there may be the desire to place additives to enhance the quality of the block utilized for lumbar surgery. Opioids, vasoconstrictors, alpha-2 agonists and neostigmine [17] have all been described and each has associated risk and benefits. Opioids tend to work synergistically with local anesthetics and are known to enhance the quality of the block. However, these agents are also associated with urinary retention (a controversy that will be discussed later). Concern regarding the addition of vasopressors and spinal blood flow is unfounded and their overall mechanism of action is unclear and inconsistent depending on the agent chosen [18].

Clonidine, an alpha-2 agonist, has been utilized frequently in spinal surgery. It is known to block the motor and sensory affects associated with tetracaine, but sensory affects are much longer. The proposed mechanism is related to the vasoconstrictive properties and the antinociception associated with adrenergic stimulation and activation of the descending noradrenergic pathways [18, 19]. Other investigators noted that patients who underwent spinal surgery and received 150mg of clonidine epidurally displayed lower postanesthesia care unit pain scores and less demand for analgesics as well as improved postoperative hemodynamics [20]. These results were confirmed in a study by Farmery and Wilson-MacDonald who found that utilization of an epidural catheter with clonidine after spinal surgery (under general anesthesia) led to profound and prolonged postoperative pain relief along with a reduction in postoperative nausea and vomiting [21]. The use of clonidine will be discussed further in the section regarding pain control.

6. Benefits

Some of the benefits of performing spinal anesthesia for lumbar surgery include a perceived decrease in blood loss, lower rates of thromboembolism, less hypertension or tachycardia, and better postoperative pain control. In addition, during spinal anesthesia, the patient is only mildly sedated with a benzodiazepine or propofol. This allows for a more reliably assessment of potential positioning issues that will be discussed later.

7. Blood loss

It has been observed that there is a perception of less surgical blood loss associated with cases performed under spinal anesthesia. Preload is markedly reduced during spinal anesthesia and

there is a resultant drop in mean arterial pressure (MAP). This reduction will produce a decrease in vertebral interosseous pressure during neuraxial anesthesia which may lead to reduced blood pressure within the bone itself, considered the main source of bleeding during posterior lumbar spine surgery [22]. The mechanism in which spinal anesthesia may reduce blood loss may possibly be related to the fact that spinal anesthesia leads to a marked reduction in the high venous pressure that occurs in response to sympathetic activity provoked by pain produced by tissue damage during surgery [23]. On the contrary, inhalational anesthesia does not totally block these sensory signals but these signals are effectively inhibited with spinal anesthesia.

Spinal anesthesia permits spontaneous ventilation during surgery that in the prone position results in lower intrathoracic pressure compared with general anesthesia using positive pressure ventilation. The avoidance of positive pressure ventilation results in less distension of the epidural veins and a reduction in intrathoracic pressure. This reduction produces a better blood return through the vena cava and less blood flow and distention of the venous plexus for better surgical exposure [24]. The diminished blood loss observed during spinal anesthesia can facilitate removal of the disc or vertebral body and result in less surgical time observed because of reduced time to affect hemostasis.

It is also worth reviewing the hemodynamic effects of spinal anesthesia since they play a significant role in the reduction of blood loss. Spinal anesthetics are known to produce a sympathetic denervation that is more profound as the level of anesthesia progresses. When a partial sympathetectomy occurs, as is routine with a well-controlled spinal, the area of tissue above the level of sympathetic denervation displays a reflex increase in sympathetic tone. This helps to compensate for the peripheral vasodilation that subsequently occurs. Arterial and arteriole beds are affected but do not maximally vasodilate due to the maintenance of auton-omous tone. Thus, it is common to see a mild decrease in total peripheral vascular resistance of approximately 15-18% assuming cardiac output is maintained [4].The venous circulation, however, is profoundly affected and since, in spine surgery, the extremities lie below the level of the heart, there is a significant amount of pooling of the blood in the dependent capacitance vessels. If normovolemia is not maintained then a significant decrease in cardiac output is seen.

8. Blood pressure and coronary circulation

There have been numerous studies comparing spinal with general anesthesia, and in most instances there has been minimal intraoperative hemodynamic differences between the two techniques. In many of the comparisons, total anesthesia times were shorter with the use of spinal as compared to general anesthesia (GA) [3, 25, 26]. (Table 1) In all of these studies it was noted that mean arterial pressure and heart rate were lower in patients receiving spinal anesthesia. The incidence of bradycardia was lower in spinal anesthesia as well as the incidence of tachycardia. The observation that spinal anesthesia maintains hemodynamic stability with little effect on heart rate was noted in a recent study by Attari, et al [27]. In this study 72 patients underwent spine surgery with half assigned to general anesthesia and the other to spinal

anesthesia. Statistically significant reductions in MAP and heart rate changes were noted in the spinal group. In addition there was enhanced surgeon satisfaction as well as a reduction in postoperative pain. These results were supported in another study which compared sixty patients undergoing lumbar disk surgery [28]. This group noted like Attari, that there were less episodes of tachycardia, hypertension and better postoperative pain with less nausea/vomiting in patients undergoing spinal. However, in their study, they found that surgeon satisfaction was greater in the general anesthesia group.

	Spinal	General
Total anesthesia time (min)	106.6±3.2	131.0±4.3[*]
Surgical time (min)	67.1±2.8	81.5±3.6[*]
Blood loss (mL)	133±13	221±32[*]
Intravenous fluids (mL)	1329±60	1478±79
Bradycardia	14.0%	22.9%
Hypertension	3.3%	26.2%[*]
Tachycardia	14.8%	21.3%
Hypotension	54.1%	57.4%
Ephedrine required	36.1%	22.9%

Numeric dara expressed as mean ± SEM

Bradycardia and hypotension=decreases in heart rate (HR) and mean arterial pressure (MAP) to less than 80% of baseline values; tachycardia and hypertension=HR and MAP greater than 120% of baseline values.

[*]$P<0.05$ versus spinal anesthesia group.

Jellish et al. Spinal vs General Anesthesia for Spinal Surgery. Anesth Analg 1996;83:559-64

Table 1. Intraoperative Data for Spinal versus Genera; Anesthesia Groups

Another recent comparative study also found the incidence of tachycardia to be higher with general anesthesia [29]. They found the incidence of bradycardia to be similar, as well as intravenous fluids and operative times. They did note a higher incidence of hypotension with spinal anesthesia compared to the other studies. This may reflect the importance of the fluid preload prior to the placement of the spinal block which was not used in that study.

Patients undergoing lumbar procedures under spinal anesthesia seemed to have similar or better hemodynamic variables than patients having the procedure under general anesthesia. Less intraoperative hypertension is noted and less tachycardia is observed with spinal anesthesia. Tetzloff, et al. [30]. using power spectral heart rate data which included low frequency, high frequency and the ratios of low/high frequency demonstrated that with spinal dermatomal levels below T_8, the prone position resulted in a significant increase in heart rate with spinal anesthesia and a significant decrease in blood pressure with general anesthesia. Low frequency and low frequency/high frequency ratios were unchanged in the spinal

anesthesia group. The preservation of low frequency heart rate variation may reflect better presentation of cardiac sympathetic activity with spinal anesthesia. Low thoracic levels of spinal anesthesia preserve the sympathetic efferent signals to the myocardium more than general anesthesia. Placing a patient in the prone position may reduce venous return and preload which is better tolerated with a spinal anesthetic.

Given the fact that many of the patients presenting for spinal surgery may have co-morbidities such as coronary artery disease, one may be concerned regarding the presence of hypotension. It has been noted that the decrease in MAP results in a significant decrease in coronary blood flow. One investigator found that there was a 48% decrease in myocardial oxygen supply during spinal anesthesia but there was also a 53% decrease in myocardial oxygen requirements [31]. There are three reasons for the decrease in myocardial oxygen requirement that include the reduction in afterload, preload and heart rate. Heart rate reduction is related to both the vagal predominance that occurs after sympathetectomy as well as the decrease in right atrial pressures and pressures in the great veins (via intrinsic chronotropic stretch receptors) which leads to bradycardia [4].

9. Pain control

Improving postoperative analgesia in spine surgery patients is also a challenge. Though many of the patients who receive spinal anesthesia for their spine procedure have reduced pain scores and analgesia requirements in the immediate postoperative period, their analgesic requirements are similar to general anesthesia patients 24 hours after surgery. Several studies comparing the two anesthetics demonstrated that patients who had spinal anesthesia had lower pain scores and analgesic requirements [3, 25, 29]. In many of the studies the lower pain scores may result from two different mechanisms. Patients who received spinal anesthesia had much lower initial pain scores than general anesthesia patients. There may be a preemptive effect in which spinal anesthesia attenuates pain by inhibiting afferent nociceptive pathways [32]. Also, since sensory recovery will lag behind motor recovery after spinal block, the patients receiving neuroaxial anesthesia likely had residual blockade even though motor function had returned.

Pain after spine procedures is a combination of musculoskeletal, usually derived from surgical trauma and neuropathy that is radiating and burning in nature and is secondary to the nerve compression or injury that required the laminectomy or discectomy. This type of pain responds poorly to opioids but has been shown to be relieved with the administration of epidural clonidine [33].

Sympathetic hyperactivity is reduced from the administration of epidural clonidine through three mechanisms. It may inhibit nociceptor neurotransmitter release in the dorsal horn and sympathetic outflow in the spinal cord intermediolateral column. In addition, it may inhibit norepinephrine release from sympathetic terminals in the periphery. Clonidine may also be absorbed into the systemic circulation where it reaches alpha 2 adrenoreceptors of the dorsal horn and provides analgesia by increasing the antinoceptive threshold of the spinal cord which

activates the descending noradrenergic pathway to inhibit small diameter afferent induced substance P release [19].

The addition of epidural clonidine to spinal anesthetics for spine surgery has been found to reduce pain in patients receiving rescue analgesics to increase the time to the first rescue dose of analgesics for pain. Clonidine prolongs sensory and motor block associated with intrathecal bupivacaine [34]. Patients who received epidural clonidine along with their spinal anesthetic required their first analgesia dose 3.7 hours after surgery [20]. Another study showed that by using a small dose combination of epidural morphine and clonidine for postoperative analgesia after lumbar disc surgery reduced pain with movement after surgery[35]. These patients experienced a frequent incidence of difficult micturition not observed when epidural clonidine was administered without added opioids.

The infiltration of local anesthetics into the surgical wound has also been noted to prolong postoperative analgesia after lumbar spine surgery. The infiltration of 0.375% bupivacaine subcutaneously has been noted to produce an analgesic effect which lasted approximately 13 hours [36]. With the use of newer local anesthetics that have a timed release, this type of analgesia could be even more prolonged [37].

The success rate of the spinal anesthetic in patients with spinal pathology is also a consideration. Some practitioners have noted ineffective spread or patchy block with spinal anesthesia after previous spine surgery. There are a number of problems that could affect the spread of the local anesthetic including altered anatomy which may make placement of the spinal more difficult. Insertion of a spinal needle through the site of a fusion may be complicated by scar tissue and bone graft material. Intradural scarring commonly referred to as arachnoiditis, characterized by an inflammation of the pia arachnoid membrane surrounding the spinal cord may alter the anatomy of the subarachnoid space and limit the spread of local anesthetics [38]. Most investigators have noted a high success rate of spinal anesthesia after previous spinal surgery with failure rates of less than 1% [26].

10. Venous thromboembolism

Finally, spinal anesthesia for lumbar spine surgery also decreases the incidence of lower extremity thromboembolic complications [39]. The most likely explanation is the modulation of the hypercoagulable state that occurs after surgery. Neuraxial anesthesia with local anesthetics has been shown to enhance fibrinolytic activity, reduce antithrombin III activity to normal levels and attenuate increases in postoperative platelet activity [40].

11. Postoperative nausea

Many studies have noted a reduced incidence in postoperative nausea and vomiting. The increased need for narcotic analgesics in patients receiving GA may be a contributing factor

to the higher amount of emetic symptoms with GA. In addition, anesthetic factors such as the use of N_2O (nitrous oxide) or the administration of certain pungent inhalational anesthetics could produce more nausea after surgery. The incidence of nausea and vomiting has also been demonstrated to be less with low level T-8 or bolus spinal anesthesia compared to GA because of improved gastric emptying.

12. Post-anesthesia care unit (PACU)

Hemodynamics in the PACU have been noted to be better with spinal anesthesia compared to GA. Both heart rate and blood pressure have been noted to be higher in GA patients upon admit to PACU. (Figure 1) This may be due to the increased sympathetic activity during emergence from anesthesia and possibly undertreated pain with opioids or other analgesics prior to emergence. Patients who had spinal anesthesia were much less hypertensive throughout their recovery room stay.
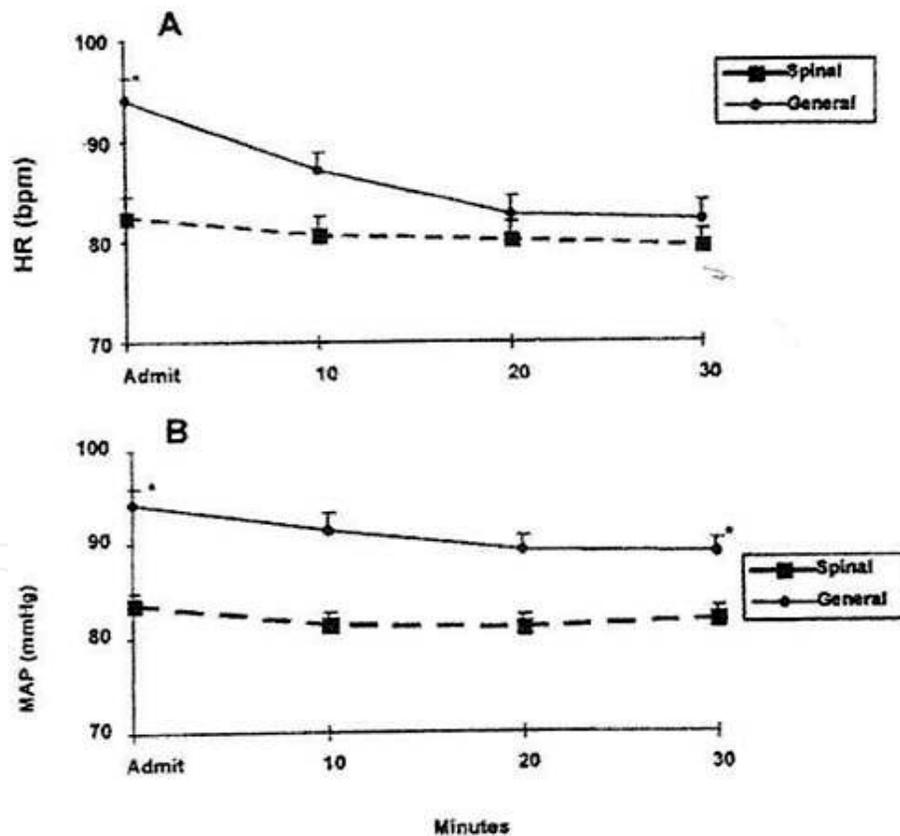
Jellish et al. Spinal vs General Anesthesia for Spine Surgery. Anesth Analg 1996;83:559-64

Figure 1. Heart rate (HR) and mean arterial pressure (MAP) values at admission (admit) and at 10, 20 and 30 min after adimission to the postanesthesia care unit (PACU). Intergroup differences in HR (A) were noted at PACU admission but did not persist through 20-min time point. Intergroup differences in MAP (B) were also observed at PACU admission and were still present 30 min after admission. *Significant difference compared to spinal group at a $P<0.001$ level.

13. Rare complications

Complications associated with spinal anesthesia for lumbar surgery have been relatively rare. There have been no reports of post-dural puncture headache even when a dural tear occurred during surgery [9].A possible explanation is that surgery near the spinal cord elicits inflammatory responses that help seal any small puncture site. In addition, the presence of small amounts of post-procedural blood may serve to seal the site similar to applying a blood patch. Other complications associated with spinal anesthesia may play a role in these lumbar cases and will be discussed further.

14. Neurological complications

In general, spinal anesthesia has a long history of safety. In the widely quoted study by Dripps and Vandam, properly performed spinal anesthesia is safe. A study which reviewed over 10,000 spinal anesthetics failed to find any major neurologic sequelae [41]. However, in a retrospective study by Hebl et al, [11], one of the major findings was that the patient population with pre-existing spinal stenosis or disk disease had an increased risk of worsening pre-existing deficits or development of new deficits after neuraxial blockade. In addition, those patients with multiple neurologic diagnoses have even higher risk. It was noted that the frequency of persistent postoperative neurologic deficits was approximately 1.1% (95% CI 0.5-2%) with prior epidemiological investigations being somewhere between 1:1000 to 1:10,000.

The group went on to propose that the neurological problems seen may have been the result of a "double-crush" phenomenon [42].In double-crush syndrome there is a pre-existing lesion (proximal) and distal to the lesion there is another compression that renders the nerve vulnerable to further injury. Neuraxial anesthesia may add insult by the additive effects of neural ischemia and local anesthetic toxicity. In spinal anesthesia, local anesthetic toxicity resulting from maldistribution and high concentrations is well known. This toxicity has resulted in cauda equina syndrome seen with microcatheters utilized for continuous spinal anesthesia [43]. Though these studies are worrisome, there have yet to be reports of neurological complications from spinal anesthesia used for lumbar spine surgery.

15. Cardiac arrest

Though it has been mentioned previously that there is less observed bradycardia during spinal anesthesia for spine surgery, there still exists the concern for profound bradyarrhythmias and cardiac arrest. In a review of studies about cardiac arrest during spinal anesthesia, investigators found an overall incidence of 0.07% (7 for every 10,000 patients) [44]. More than half were in patients under the age of thirty and this may explain the paucity of events during lumbar surgery with an older patient population. The mechanism proposed is a result of the blockade

of sympathetic efferents that leads to bradyarrhythmias via vagal predominance. The presence of vagal mediated bradycardia and decreased venous return from venodilation combines to cause further issues. It is well known that right atrial pressures are decreased in low spinals (36%) and high spinals (up to %53) [45]. This decrease in preload elicits reflexes that cause severe bradycardia [46]:

- Pacemaker stretch → decrease venous return → decrease atrial stretch → decrease heart rate

- Firing of low pressure baroreceptors in the right atrium

- Bezold-Jarisch reflex

The Bezold-Jarisch reflex (BJR) is triggered by the stimulation of intracardiac mechanoreceptors that subsequently lead to bradycardia, hypotension and vasodilation [47]. According to Mackey [46] and Kinsella [48], the mechanoreceptors associated with BJR are usually triggered by distention, but when there is a decrease in venous return (as seen with prone position and spinal anesthesia), along with an increase in inotropic state (compensatory response to decreased preload), the walls of the ventricle may deform and trigger the mechanoreceptors similar to what is seen during distention. This results in a paradoxical vasodepressor response. This vasodepressor response along with the pre-existing bradycardia may lead to cardiac arrest. It is interesting to note that the BJR is also triggered by spinal anesthesia via $5\text{-}HT_3$ receptors in the vagal nerve endings. The $5\text{-}HT_3$ trigger can be abolished by the administration of ondansetron, an antagonist to $5\text{-}HT_3$.

Risk factors identified by Pollard [44] that are associated with cardiac arrest include the following:

- Baseline heart rate <60

- ASA status I

- Use of beta blocking drugs

- Sensory level above T6

- Age <50 years

- Prolonged PR interval

Recommendations include the maintenance of preload whenever possible, followed by a step-wise escalation of pharmacological intervention starting with atropine (0.4-0.6 mg), then ephedrine (25-50 mg) and finally, if still not responsive, epinephrine (0.2-0.3 mg) intravenously.

16. Urinary retention

Urinary retention has also been associated with spinal anesthesia. However, in several studies the incidence of urinary retention was not different with spinal or general anesthesia. In most situations when spinal anesthesia is associated with urinary retention, opioids were added to

the local anesthetic [49]. Subarachnoid opioids clearly increase the incidence of urinary retention, as well as respiratory depression, drowsiness and pruritis. We tend not to utilize opioids as part of the spinal anesthetic.

17. Other potential problems

17.1. Prone positioning issues

Positioning related neurologic injury has also been noted to occur more frequently during spinal surgery in the prone positioned patient [50].The most prevalent is injury to the brachial plexus. Injury to the brachial plexus is attributed to its long and superficial course in the axilla and its attachment to two firm points of fixation, the vertebrae proximally and the axillary fascia distally in the arm. The plexus also passes directly beneath the clavicle and above the first rib (Figure 2). This close proximity to freely moving bony structures makes this nerve bundle prone to stretching and compression from arm malposition. Brachial plexus injury occurs most frequently when the patient is in the prone position, especially when the arms are adducted more than 90°. In this position traction on the plexus and compression between the clavicle and first rib is responsible for the neurologic deficit. If patients are placed in the lateral decubitus position, they may be subject to brachial plexus injury from compression when the dependent arm and shoulder are positioned between the thorax and the table.
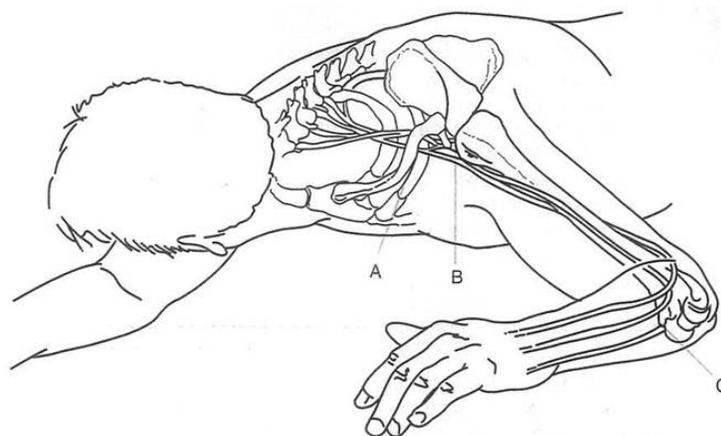
Jellish et al. Spinal Anesthesia for Spinal Surgery. Best Practices & Research. Clinical Anaesthesiology 2003;17(3): 323-334
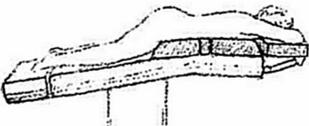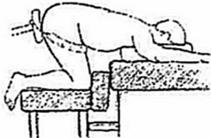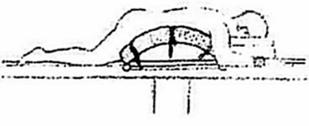
Figure 2. Possible areas of injury to the brachial plexus: (A) neck rotation away from arm may cause stretch and compression between clavicle and first rib; (B) injury to plexus at humeral head; (C) compression or ulnar nerve in cubital tunnel.

The eyes and ears are also of concern in prone positioned patients. Pressure on the globe or hypotension, with venous congestion, could result in increased intraocular pressure and possible blindness related to ischemic injury to the optic nerves. There have been reports of increased extraocular pressure resulting from using a cushion or horseshoe head-rest to

position the face [51, 52].In addition, ECG monitoring wires or oral gastric tubes, if present, could migrate under the head during prone positioning. The face could lie directly on these objects causing pressure induced ischemia to the face or eyes. These problems are avoided with the use of spinal anesthesia. The patient may be only mildly sedated and using their upper body can help to self-position with their head on a pillow or cushion. If abnormal positioning occurs, the patient will feel discomfort and alert the practitioner to the problems. They can also move their arms and head to avoid prolonged abnormal or awkward position that could produce injury.

17.2. Sedation issues

A spinal anesthetic with an awake or sedated patient who is spontaneously breathing may not be ideal for all spine surgeries. Prone positioning on different positioning systems can affect cardiac output with the possibility of a significant decrease in stroke volume and cardiac index in conjunction with the development of increased vascular and pulmonary resistance [53] (Figure 3). Patients with normal cardiac status can usually tolerate these changes. However, patients with compromised cardiac status might not be able to tolerate supine to prone positioning, especially with decreased sympathetic tone. A large drop in blood pressure or cardiac output could affect consciousness and spontaneous breathing.

Type	Picture	Chest Support	Abdomen	Pelvis	Legs
Siemens Positioning System		Yes	Partial compression	Supported	At heart level
Andrews Frame		Yes	Hangs free	Partial support	Below heart
Wilson Frame		Yes	Partial compression	Partial support	Slightly below
Jackson Spine Table		Yes	Hangs free	Supported	Supported at thigh At heart level
Longitudinal Bolster		Partial	Partial compression	Partial support	At heart level

Jellish et al. Effects of Prone Positioning Systems on Hemodynamic and Cardiac Function During Lumbar Spine Surgery: An Echocardiographic Study. Spine 2006:31(12) 1388-1393

Figure 3. Description of the five different positioning systems used in this study. The type of body support each positioner provides and lower extremity position in relation to the heart are also described.

In addition, surgeries that may last more than 2-3 hours may not be conducive for spinal anesthesia. The tolerance for prone positioning on a frame in an awake or mildly sedated patient is approximately 2 hours. Patients become restless and tend to begin to adjust position in order to relieve the strain of maintaining one position for a prolonged period of time. A cooperative surgeon who can perform the procedure in a reasonable amount of time is imperative.

We also believe one to two level laminectomies or discectomies are ideal for this technique. Larger laminectomies for multi-level fusions would be too prolonged to be well tolerated in the spontaneously breathing sedated patient. Body habitus must also be considered in selecting the appropriate patient for spinal anesthesia for spine surgery. Large, obese patients with protuberant abdomens may not tolerate prone positioning well, especially if breathing spontaneously. Their ability to breathe against the restrictive effects of a large abdomen, especially if not adequately decompressed by the positioning system, could cause the patient undue anxiety and intolerance because of the inability to adequately deep breath.

18. Summary

Spinal anesthesia is an appropriate technique for lumbar spine procedures of two to three hours duration. An appropriate patient and cooperative surgeon will also facilitate the use of this anesthetic technique. The ability of the patient to self-position and guard against position related injury is of major benefit. A better postoperative experience with less pain, nausea and hemodynamic stability make this technique superior to general anesthesia for overall patient satisfaction and reduced morbidity. Short term pain control is definitely improved with spinal anesthesia and new and improved methods for providing longer term analgesia may make this anesthetic technique even more beneficial, especially if contemplating same day discharge and reduced hospital stay.

Author details

W. Scott Jellish[*] and Steven Edelstein

*Address all correspondence to: wjellis@lumc.edu

Department of Anesthesiology, Loyola University Medical Center, Maywood, USA

References

[1] Mixter WJ, Barr JS. Rupture of the intervertebral disk with involvement of the spinal cord. NEJM 1934;211:210-15.

[2]   Williams RW. Microlumbar discectomy: A conservative surgical approach to a virgin herniated lumbar spine. Spine 1978;3:175-82.

[3]   Jellish WS, Thalji Z, Stevenson K, et al. A prospective randomized study comparing short and intermediate term perioperative outcomes after spinal or general anesthesia for lumbar disk and laminectomy surgery. Anesth Analg 1996; 83: 559-64.

[4]   Bridenbaugh PO, Greene NM. Spinal (subarachnoid) neural blockade. In: Cousin MJ, Bridenbsaugh PO. (ed.) Clinical anesthesia and pain management 3rd Ed. Philadelphia: 1998 p202-241.

[5]   Silver DJ, Dunsmore Rh, Dickson Cm. Spinal anesthesia for lumbar disc surgery. Anesth Analg 1976; 550-54.

[6]   Tetzloff JE, O'Hara J, Bell G, et al. Influence of baricity on the outcome of spinal anesthesia with bupivacaine for lumbar spine surgery. Reg Anesth 1995;20: 533-37.

[7]   Gissen AJ, Covino BG, Gregus J. Differential sensitivies of mammalian nerve fibers to local anesthetic agents. Anesthesiology 1980;53:467-74.

[8]   Rung GW, Williams D, Gelb DE, et al. Isobaric spinal anesthesia for lumbar disk surgery. Anesth Analg 1997;84:1165-166.

[9]   Laasko E, Pitkanen M, Kytta J, Rosenberg DN. Knee chest vs horizontal side position during induction of spinal anesthesia in patients undergoing lumbar disc surgery. Br J Anaesth 1997;79:609-11.

[10] Reina MA, de Leon-Cassola OA, Lopez A, et al. An scanning electron microscopy in vitro study of dural lesions produced by 25-gauge Quincke and Whitacre needles evaluated by electron microscopy. Reg Anesth Pain Med 2000;25:393-402.

[11] Hebl JR, Horlocker TT, Kopp SL, et al. Neuraxial blockade in patients with preexisting spinal stenosis, lumbar disk disease, or prior spine surgery: Efficacy and neurologic complications. Anesth Analg 2010;111:1511-19.

[12] Goddard M, Smith PD. Spinal anaesthesia for spinal surgery. Anaesth 2006;61:723-24.

[13] Schneider M, Ettlin T, Kaurfmann M, et al. Transient neurologic toxicity after hyperbaric subarachnoid anesthesia with 5% lidocaine. Anesth Analg 1993;76:1154-71.

[14] Zaric D, Christiansen C, Pace NL, et al. Transient neurologic symptoms (TNS) following spinal anaesthesia with lidocaine versus other local anaesthetics. Cochrane Database Syst Rev 4 D003006 2005.

[15] Greene NM. Distribution of local anesthetics in the subarachnoid space. Anesth Analg 1985;64:715-30.

[16] Shesky MC, Rocco AG, Bizzarri-Schmidt M, et al. A Dose-response study of bupivacaine for spinal anesthesia. Anesth Analg 1983;63:931-5.

[17] Bouaziz H, Tong C, Eisenach JC. Postoperative analgesia from intrathecal neostigmine in sheep. Anesth Analg 1995; 80:1140-44.

[18] Brown DL. Spinal, epidural, and caudal anesthesia. In: Churchill Livingstone Elsevier Miller's Anesthesia 7th Ed. Philadelphia: 2010: 1611-1637.

[19] Kuraishi Y, Hirota N, Sato Y, et al. Noradrenergic inhibition and the release of substance P from the primary afferents in the rabbit spinal dorsal horn. Brain Res 1985;359: 177–82.

[20] Jellish WS, Abodeely A, Fluder EM, Shea J. The effect of spinal bupivacaine in combination with either epidural clonidine and/or 0.5% bupivacaine administered at the incision site on postoperative outcome in patients undergoing lumbar laminectomy. Anesth Analg 2003;96:874-80.

[21] Farmery AD, Wilson-MacDonald J. The Analgesic effect of epidural clonidine after spinal surgery: A randomized placebo-controlled trial. Anesth Analg 2009;108:631-4.

[22] Kakiuchi M. Reduction of blood loss during spinal surgery by epidural blockade under normotensive general anesthesia. Spine 1997;22:89-94.

[23] Cousins MJ, Bromage PR. Epidural neural blockage. In: Cousins MJ, Bridenbaugh PO (ed). Clinical Anesthesia and Management of Pain. Phildelphia: 1988 p 253-360.

[24] Goddard M, Smith PD. Spinal anaesthesia for spinal surgery. Anaesth 2006;61:723-24.

[25] McClain RF, Kalfus I, Bell GR, et al. Comparison of spinal and general anesthesia in lumbar laminectomy surgery: A case controlled analysis of 400 patients. J Neurosurg Spine 2005;2:17-22.

[26] Tetzloff JE, Dilger JA, Kodsy M et al. Spinal anesthesia for elective spine surgery. J Clin Anesthesia 1998;10:666-69.

[27] Attari MA, Mirhosseini SA, Honarmad A, et al. Spinal anesthesia versus general anesthesia for elective lumbar spine surgery: A randomized clinical trial. J Res Med Sci 2011;16:524-29.

[28] Kara I, Celik JB, Bahar OC, et al. Comparison of spinal and general anesthesia in lumbar disc surgery. J Neuro Sci (Turkish) 2011;28:487-96.

[29] Sadrolsadat SH, Mahdavi AR, Moharari RS, et cl. A Prospective randomized trial comparing the technique of spinal and general anesthesia for lumbar disk surgery: A study of 100 cases. Surgical Neurology 2009;71:60-5.

[30] Tetzloff JE, O'Hara JF, Yoon JH, et al. Heart rate variability and the prone position under general vs spinal anesthesia. J Clin Anesth 1998;10:656-9.

[31] Hackel DB, Sancetta SM, Kleinerman J. Effect of hypotension due to spinal anesthesia on coronary blood flow and myocardial metabolism in man. Circulation 1956;13:92-7.

[32] Covino BG. Rationale for spinal anesthesia. Int Anesthesiol Clinics 1989;27:8-12.

[33] Eisenach J, Detweiler D, Hood D. Hemodynamics and analgesic actions of epidurally administered clonidine. Anesthesiology 1993;78:277-87.

[34] Lund C. Quitzau S, Greulich A, et al. Comparison of the effects of extradural clonidine with those of morphine on postoperative pain, stress response cardiopulmonary function and motor and sensory block. Br J Anaesth 1989;643:516-9.

[35] Bonhomme V, Doll A, Dewandre PY, et al. Epidural administration of low dose morphine combined with clonidine for postoperative analgesia after lumbar disc surgery. J Neurosurg Anesth 2002;14:1-6.

[36] Cherian MN, Matthews MP, Chandry MJ. Local wound Infiltration with bupivacaine in lumbar laminectomy. Surg Neurol 1997;47:120-5.

[37] Gorfine SR, Onel E, Patou G, Krivokapic. Bupivacaine extended release liposome injection for prolonged postsurgical analgesia in patients undergoing horrhoidectomy: A multicenter randomized, double blind, placebo controlled trial. Dis Colon Rectum 2011;54 1552-9.

[38] Sun Ko. Spinal anaesthesia following previous spinal surgery. European J Anaesth 1994;11: 321-33.

[39] McDaniel MD, Pearce WH, Yao JS, et al. Sequential changes in coagulation and platelet function following femorotibial bypass. J Vasc Surg 1984;26:1-8.

[40] Rosenfeld BA, Beattie C, Christopherson R, et al. The effects of different anesthetic regimens on fibrinolysis and the development of post-operative arterial thrombosis. Anesthesiology 1993;79:435-43.

[41] Dripps RD, Vandam LD. Long term follow-up of patients who received 10,098 spinal anesthetics. I. Failure to discover major neurological sequelae. JAMA 1954; 156:1486-91.

[42] Upton AR, McComas AJ. The double crush in nerve entrapment syndromes. Lancet 1973;302:359-62.

[43] Rigler M, Drasner K, Krejcie TC, et al. Cauda equina syndrome after continuous spinal anesthesia. Anesth Analg 1991;72;275-81.

[44] Pollard JB. Cardiac arrest during spinal anesthesia: Common mechanisms and strategies for prevention. Anesth Analg 2001;92:252-6.

[45] Sancetta SM, Lynn B, Simeone FA, et al. Studies of hemodynamic changes in humans following induction of low and high spinal anesthesia: I. General considerations of the problem. The changes in cardiac output, brachial arterial pressure, peripheral and pulmonary oxygen contents and peripheral blood flows induced by spinal anesthesia in humans not undergoing surgery. Circulation 1952;6:559-71.

[46] Mackey DC, Carpenter RL, Thompson GE, et al. Bradycardia and asystole during spinal anesthesia: A report of three cases without morbidity. Anesthesiology 1989;70:866-8.

[47] Campagna JA, Carter R. Clinical relevance of the Bezold-Jarisch reflex. Anesthesiology 2003;98:1250-60.

[48] Kinsella SM, Tuckey JP. Perioperative bradycardia and asytole: A relationship to vasovagal syncope and the Bezold-Jarisch reflex. Br J Anaesth 2001;86:859-69.

[49] Young DV. Comparison of Local, Spinal and general anaesthesia for inguinal herniorrhaphy. Am J Surg 1987;153:560-3.

[50] Uribe JE, Koller J. Omar H, et al. Brachial plexus injury following spine surgery. J Neurolog Spine 2010;13:552-8.

[51] Bekar A, Tureyen K, Aksoy K. Unilateral blindness due to patient positioning during cervical syringomyelia surgery, unilateral blindness after prone positioning. Neurosurg Anesth 1996;8:227-9.

[52] Grossman W, Ward WT. Central retinal artery occlusion after scoliosis surgery with a horse shoe headrest: Case report and literature review. Spine 1993;18:1226-28.

[53] Dharmavarm S, Jellish WS, Nockels RS. Effect of prone positioning on hemodynamic and cardiac function during lumbar spine surgery. The Spine Journal 2006;31:1388-93.