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ORIGINAL RESEARCH

Preferential Flow Patterns of Injectate in Epidural and Inadvertent Subdural Anesthesia: Exploring the Hemodynamic Stability of High-Level Epidural, Subdural and Combined Epidural-Subdural Blocks in Relation to ASA Class

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Purpose: This study examines the hemodynamic responses elicited by epidural, subdural, and combined epidural-subdural anesthesia during spinal surgery, with a focus on anesthetic levels and ASA classifications. It integrates image findings to enhance understanding of the anesthetic impact on hemodynamic stability.

Patients and Methods: A retrospective analysis was conducted involving patients who underwent endoscopic, open, or fusion spine surgeries with epidural anesthesia and monitored anesthesia care (MAC) between March 2018 and September 2023. Comprehensive demographic data, details regarding anesthetic levels, ASA class and hemodynamic measurements were systematically collected. Additionally, fluoroscopic images were assessed to investigate the distribution patterns of anesthetics and their relationship to hemodynamic outcomes.

Results: In patients undergoing epidural, subdural, and combined epidural-subdural anesthesia with high-level blocks above T5 and classified as ASA class III or higher, no significant differences were observed in hypotensive events or vasopressor usage compared to those with lower-level blocks or ASA classifications. The mean duration of surgery was 90.6 ± 40.9, 105.4 ± 42.5, and 100.8 ± 46.6 minutes, respectively, across the three groups. Subdural anesthesia exhibited a similar hemodynamic profile, with milder blood pressure decreases. Imaging analysis indicated distinct anesthetic distribution patterns primarily in the posterior epidural and subdural spaces, which helped preserve anterior sympathetic and motor functions, suggesting a relationship between fluoroscopic imaging features and hemodynamic stability.

Conclusion: Hemodynamic stability was maintained in the subdural and combined epidural-subdural groups compared to the epidural group in ASA I to III patients. However, epidural anesthesia showed better hemodynamic outcomes for ASA class above III. High-level epidural and subdural anesthesia primarily induced posterior diffusion, resulting in minimal anterior sympathetic block while preserving stability. These findings suggest that epidural anesthesia may be a viable alternative for spinal surgeries and applicable to other procedures for patients with high ASA classifications.

Keywords: epidural anesthesia, subdural anesthesia, hemodynamic, high level block, ASA class

Introduction

Regional anesthesia reduces blood pressure (BP = CO x SVR) mainly by blocking preganglionic sympathetic nerves from T1 to L2.[1–4] Subdural anesthesia was traditionally known as a dangerously unstable anesthetic technique due to its potential to cause vital instability.[5–7] However, our recent observations indicate that hemodynamically, subdural

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anesthesia can maintain blood pressure with minimal use of vasopressors. Additionally, in approximately 40% of subdural anesthesia cases, a motor block involving the L3, L4, L5, and S1 nerves (knee extension and flexion, big toe extension, ankle dorsiflexion and plantar flexion) was observed, persisting beyond the typical duration of epidural anesthesia (4 hours).[8] This led us to question why sympathetic and motor nerves, which both traverse anteriorly at the T1-L2 preganglionic level, exhibited differential block effects. Specifically, why somatic motor nerves were blocked while the sympathetic, which follow a similar anterior path, remained largely unaffected, maintaining hemodynamic stability. To explore this further, we reanalyzed our imaging data and collected information.

Spinal anesthesia employs a single-layer compartment similar to a water-filled conduit, allowing for simultaneous anesthetic effects on both posterior and anterior nerve roots at corresponding dermatomal levels. Typically, variability in anesthetic distribution spans no more than 2 spinal levels.[9] In contrast, the lateral view of epidural anesthesia reveals a dual (anterior and posterior)-layer structure resembling a thin, cylindrical tube, with lateral pathways visible on both sides in the anteroposterior view (Figure 1). When employing a posterior approach to epidural anesthesia, this technique promotes the longitudinal spread of anesthetic along the posterior aspect and circumferential diffusion around the anterior, ultimately reaching the centrally located spinal nerves. Additionally, the anesthetic may flow bilaterally through the exiting nerve roots, affecting multiple each dermatomal levels. Consequently, epidural anesthesia may result in uneven distribution of the anesthetic agent, potentially leading to patchy blocks if the spread is non-uniform. These characteristics are also applicable to subdural blocks.

Compared to general anesthesia, epidural anesthesia with monitored anesthesia care (MAC) may not always achieve complete surgical anesthesia, but usually provides sufficient analgesia to allow safe and motionless surgery without deep sedation. Motor block is uncommon with epidural anesthesia, which facilitates postoperative neurologic exams. Subdural anesthesia may produce delayed motor block, often developing after the incision, suggesting a slower intraoperative onset. These characteristics are relevant for assessing safety in longer procedures and cases where deep sedation is avoided. Based on our five-year experience, epidural anesthesia in ASA III or higher patients generally causes mild and gradual sympathetic block, supporting stable hemodynamics. Although prone positioning under MAC carries inherent risks, epidural anesthesia may reduce the need for deep sedation. However, in patients with airway risk—such as sleep apnea—general anesthesia may still be preferable.

In the previous study, we examined hemodynamic changes associated with epidural, subdural, and combined epidural-subdural anesthesia without specific condition considerations.[8] In this current analysis, we conduct a comprehensive investigation of hemodynamic variations based on ASA classification within each type and explore differences linked to anesthesia above T5 in each area. Furthermore, this study will assess blood pressure medication usage, fluid management specifics, and lengths of hospital stays in each anesthesia type. We also analyzed the relationship between hemodynamic changes and fluoroscopic imaging characteristics across epidural, subdural, and combined anesthesia.

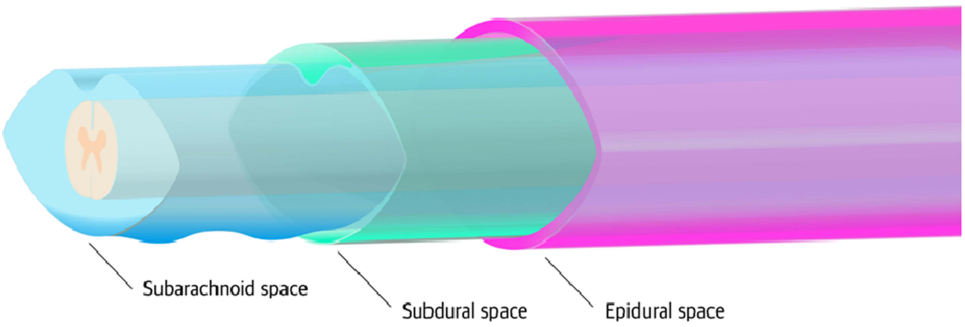

Figure 1 The 3D schematic provides an anatomical illustration of the meningeal spaces from the outermost to innermost layers: the epidural, subdural, and subarachnoid spaces. The epidural space is depicted as a long, thin, hula-hoop like structure. In contrast, the subdural space exhibits a variable contour, resembling a long, irregular, bumpy structure due to the presence and extent of iatrogenic fissures. The innermost layer, the subarachnoid space, is represented as a continuous tubular structure similar to a water-filled tube. Following the posterior injection of radiocontrast media, the lateral view shows the epidural and subdural spaces as either one or two distinct layers, while the subarachnoid space consistently appears as a single, continuous tubular entity.

Methods

The study was approved by the Institutional Review Board of Nanoori Hospital Medical Research Center (NR-IRB 2023-005), with informed consent waived due to its retrospective nature. As the Institutional Review Board of Nanoori Hospital waived the requirement for patient consent to review medical records, all data were either anonymized or handled with strict confidentiality to ensure the privacy of the participants. Conducted according to the Ethical Principles for Medical Research Involving Human Subjects from the Helsinki Declaration,[10] the study included patients who underwent endoscopic, open, or fusion spine surgery with epidural anesthesia with monitored anesthesia care (MAC) at Nanoori Hospital in Gangnam between March 2018 and September 2023. Patients were excluded if they received either spinal (subarachnoid) anesthesia or combined subdural and subarachnoid anesthesia or if records were incomplete or unverifiable.

Anesthetic Technique

The anesthetic technique employed in this study is consistent with methods in previous literature; however, a brief overview will be provided herein.[8,11,12]

Upon arrival in the operating room, patients were placed in the prone position on a Wilson frame, and standard monitoring for blood pressure, oxygen saturation, and electrocardiography was initiated. Each patient received supplemental oxygen and their faces were supported by a hollowed-out rectangular face protection cushion.

Tuohy needle was introduced into the epidural space using fluoroscopic guidance, targeting the nearest interlaminar space and the region with a thickened ligament flavum that has not been previously operated on, as indicated by preoperative MRI findings. The correct placement of the needle was confirmed via the loss-of-resistance technique and fluoroscopic imaging with contrast media. A single injection of either 7.5–15 mL of 0.75% Ropivacaine with 1:200,000 epinephrine diluted in 2.5–5 mL of radiocontrast dye (BONOREX®) was administered (0.5% Ropivacaine). Epidural infusion was administered to the lower to mid-thoracic epidural space to ensure adequate coverage of the surgical field. In instances of significant epidural adhesions or severe stenosis, supplementary injections were performed to facilitate appropriate dye distribution (Video S1).

Following the placement of the epidural, capnogram monitoring was initiated to evaluate respiratory status, provided that the patient's vital signs and comfort levels were stable. Sedatives were carefully selected based on their minimal respiratory impact, rapid onset, short duration, and the availability of reversal agents.[13,14] The MAC protocol was subsequently commenced, involving a loading dose of dexmedetomidine (Precedex®) at 0.5–1 mcg/kg infused over 10–20 minutes, followed by a maintenance dose of 0.5–0.7 mcg/kg/h. Additionally, remifentanil was administered at a concentration of 20 mcg/mL, infused at a rate of 5–7 mL/h, and Midazolam was administered in doses of 1 to 2 mg, tailored to the patient's age, sex, and calculated adjusted body weight. An extra dose of 1 mg of midazolam was administered each hour. The dosages of these medications were fine-tuned based on continuous evaluations of the patient's respiratory function and sedation level. In instances where the patient became unresponsive to noxious stimuli, the oxygen flow was increased to 6 L. A small pillow was positioned beneath the rectangular face-protection cushion to facilitate the sniffing position and thus improve airway patency. If the capnogram indicated unstable breathing, an oral or nasal airway was inserted, the oxygen delivery method was switched to a simple mask, and the oxygen flow was increased to approximately 10 L to maintain an FiO2 of about 0.6. Furthermore, we temporarily discontinued or reduced the dosage of remifentanil, noted for its short context-sensitive half-time.[15] Dosages of midazolam, dexmedetomidine, and remifentanil were adjusted according to the patient's age, weight, and level of sedation.

During surgery, hypotension, defined as a mean arterial pressure (MAP) reduction of more than 25% from baseline, was addressed by administering fluid boluses. To address the reduction in cardiac output (CO) resulting from preoperative fluid deficits associated with an 8-hour fasting period, a more liberal fluid administration strategy was implemented rather than a restrictive approach. Patients experiencing blood loss greater than 200 mL or classified as ASA III or higher were preferentially managed with hydroxyethyl starch 130/0.4 colloid for hemorrhage control rather than crystalloids, ensuring that the dosage did not exceed 20 mL/kg per day, in accordance with updated guidelines established after the Boldt scandal.[16] Interventions such as ephedrine (4–8 mg) or phenylephrine (50 mcg) were utilized as needed. Additionally, a vasopressin analog (terlipressin, 1 mg) was administered in instances where previous measures were inadequate, particularly for patients on long-term angiotensin receptor blockers (ARB) or angiotensin-converting enzyme (ACE) inhibitors. Hypertension, defined as MAP exceeding 125% of baseline, was managed with nicardipine (500 mcg) or diltiazem (3 mg). Bradycardia, characterized by a heart rate of less than 40 beats per minute, was treated with atropine (0.5 mg).

Postoperatively, patients were transferred to the Post-Anesthesia Care Unit (PACU) for monitoring of vital signs, neurological assessments including the motor function of the lower extremities (mainly L3 to S1), and pain evaluation using a numerical rating scale (NRS). Patients were discharged to the ward once awake and responsive with assessable motor function, followed by regular evaluations of NRS scores, neurological assessments and vital signs.

Data Collection

We differentiated between patients undergoing fusion surgery at 1–2 levels and those receiving simple decompression at 1–3 levels. A retrospective analysis was conducted on radiographic images, medical records, and clinical data retrieved from the hospital's electronic medical record (EMR) system. The dataset included demographic details such as gender, age, height, American Society of Anesthesiologists (ASA) physical status, history of previous spine surgeries, the types and number of surgical levels treated, the frequency of instances where mean arterial pressure (MAP) changed by more than 25% from baseline, the associated treatments administered, and the quantity of medications and fluids given.

Furthermore, we differentiated between the imaging characteristics related to epidural, subdural, and combined epidural-subdural configurations were assessed based on stored fluoroscopic radiographs, affected sensory dermatomes from anesthetic records, and the occurrence and duration of any motor block. Hemodynamic differences associated with high-level blocks were analyzed, as well as the number of patients with a high-level block above T5, alongside their immediate and 10-minute post-injection stored fluoroscopy images. Additionally, the hemodynamic differences related to ASA classification were evaluated.

Statistical Analysis

Patients were divided into three groups (epidural, subdural, and combined epidural-subdural anesthesia) based on the stored fluoroscopic radiographic images. The data distribution for continuous variables was first evaluated for normality using the Shapiro–Wilk test. Normally distributed data were compared using a one-way analysis of variance with the Scheffe post-hoc test and presented as the mean ± standard deviation (SD). Abnormally distributed data were compared using the Kruskal–Wallis test followed by the Mann–Whitney *U*-test with Bonferroni correction and presented as mean ± standard devia-tion, the median (P25–P75). Descriptive variables were analyzed using the χ^2-test or Fisher's exact test, as appropriate, followed by Bonferroni correction to adjust the P-value, considering the potential false-positive rate from multiple comparisons. All statistical analyses were performed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA), and statistical significance was set at P < 0.05.

Results

Of the 628 patients, 308 received epidural anesthesia, 123 received subdural anesthesia, and 197 received combined epidural–subdural anesthesia, accounting for approximately 49%, 19.6%, and 31.4% of the sample, respectively. The mean duration of surgery was 90.6 ± 40.9, 105.4 ± 42.5, and 100.8 ± 46.6 minutes, respectively, across the three groups (Table 1).

Table 1 Demographics of All Patients

		Type of Anesthesia			P value
		Group E (n=308)	**Group S (n=123)**	**Group E+S (n=197)**	
Sex	Male Female	173 (56) 135 (44)†	46 (37) 77 (63) *	97 (49) 100 (51)*,†	0.002
Age (years)		58.23 ±14.3†	70.63±11.2*	64.84±12.7*,†	<0.001
Height (cm)		163.9±9†	157.9±10.1*	161±8.7*,†	<0.001

(*Continued*)

Table 1 (Continued).

	Type of Anesthesia			P value
	Group E (n=308)	Group S (n=123)	Group E+S (n=197)	
ASA physical status I II III	 3 (1) 251 (81) 54 (18)	 – 93 (76) 29 (24)	 3 (1.5) 152 (77.6) 41 (20.9)	0.213
Previous spine surgery	71 (23)†	72 (59)*	54 (27)†	<0.001
Surgical procedure				
OPEN	156 (51)	55 (44.7)	97 (49.2)	0.315
Endo	125 (40.7)	54 (43.9)	73 (37.1)	0.315
OPEN fusion	25 (8)	12 (9.8)	26 (13.2)	0.315
Endo fusion	1 (0.3)	2 (1.6)	1 (0.5)	0.315
Operation time (min)	90.55±40.9†	105.41±42.5*	100.81±46.6	0.002
Anesthesia time (min)	134.98±43†	150.24±45.8*	146.29±48.6	0.002

Notes: *P<0.05 compared with group E, †P<0.05 compared with group S. Group E: epidural anesthesia, Group S: subdural anesthesia, Group E+S: combined epidural and subdural anesthesia. Data are presented as numbers of patients (%) or mean ± SD.
Abbreviations: OPEN, Open discectomy from 1 to 3 level and/or decompression; Endo, Endoscopic discectomy from 1 to 3 level and/or decompression; ASA, American Society of Anesthesiologists.

In a cohort of patients undergoing 1–3 level open or endoscopic discectomy, unilateral laminectomy, or unilateral to bilateral laminectomy, the epidural (E) group consisted of 281 patients, the subdural (S) group had 109 patients, and the combined epidural + subdural (E+S) group included 170 patients. This corresponds to approximately 50.1%, 19.5% and 30.4% of the sample respectively. The combined E+S group exhibited a significantly higher incidence of bleeding compared to the other groups, resulting in more frequent episodes of hypotension relative to the epidural group. However, the frequency of vasopressor use did not show a significant difference when compared to the epidural group. In the subdural group, the volume of colloid used was higher than that of the epidural group, suggesting a anesthesiologists' preference for colloid over crystalloid for volume resuscitation particularly in the subdural group. Nonetheless, the degree of hypotension and the frequency of vasopressor use did not exhibit significant differences compared to the epidural group. Notably, among the three groups, only the epidural group demonstrated a significantly shorter mean Hospitalization Duration (HOD) of 4.5 days (Table 2).

In a cohort of patients undergoing 1–2 level open or endoscopic fusion surgeries, there were 26 patients in the epidural group, 14 in the subdural group, and 27 in the combined epidural + subdural group, accounting for

Table 2 Demographic and Clinical Characteristics for Patient Cohort Undergoing Open or Endoscopic Decompression Surgery at 1 to 3 Levels

	Type of Anesthesia			P value
	Group E (n=281)	Group S (n=109)	Group E+S (n=170)	
ASA physical status (No) I II III or IV	 3 (1.1%) 230 (81.8%) 48 (17.1%)	 – 81 (74.3%) 28 (25.7%)	 3 (1.8%) 132 (77.6%) 35 (20.6%)	0.164

(*Continued*)

Table 2 (Continued).

	Type of Anesthesia			P value
	Group E (n=281)	Group S (n=109)	Group E+S (n=170)	
High block (No)	48 (17.1%)	16 (15.1%)	27 (15.9%)	0.878
Hypotensive episode (No)	1.6±1, 2 (1–2)	1.8±1, 2 (1–2)	1.9±1.16, 2 (1–2)*	0.027
Hypertensive episode (No)	0.7±1, 0 (0–1)	0.8±1.1, 0 (0–1)	0.9±1.17, 1 (0–2)	0.132
Use of vasopressor (No)	0.2±0.62, 0 (0–1)	0.4±0.88, 0 (0–0)	0.3±0.75, 0 (0–0)	0.125
Use of vasodilator (No)	0.5±0.93, 0 (0–1)	0.7±1.14, 0 (0–1)	0.7±1, 0 (0–1)	0.236
Crystalloid (mL)	1479.5±425.29†	1306.4±432.11*	1527.6±399.46*,†	<0.001
Colloid (mL)	51.1±138.24†, 0 (0–0)	98.8±192.54*, 0 (0–50)	74.4±192.06, 0 (0–10)	0.034
Blood loss (mL)	128.8±103.65	162.2±157.4	173.7±139.28*	<0.001
HOD (day)	4.5±3.21, 3 (2–6)	6.2±4.18, 5 (3–8)*	5.6±4.57, 4 (3–7)*	<0.001

Notes: *$p < 0.05$ compared with group E, $^{\dagger}p < 0.05$ compared with group S. Group E: epidural anesthesia, Group S: subdural anesthesia, Group E+S: combined epidural and subdural anesthesia. Data are presented as numbers of patients (%) or mean ± SD, medians (P_{25}–P_{75}).
Abbreviations: ASA, American Society of Anesthesiologists; No, number; HOD, hospital day.

approximately 62%, 13% and 25% of the sample respectively. No significant differences were observed among the three groups in terms of blood pressure variability, fluid consumption, frequency of vasopressor use, or Hospitalization Duration (HOD) (Table 3).

Table 3 Patient Cohort Undergoing Open or Endoscopic Fusion Surgery at 1 to 2 Levels

	Type of Anesthesia			P value
	Group E (n=26)	Group S (n=14)	Group E+S (n=27)	
ASA physical status (No)				0.661
1	–	–	–	
2	21 (80.8)	12 (85.7)	20 (74.1)	
3 or 4	5 (19.2)	2 (14.3)	7 (25.9)	
High-level block (≤ T5) (No)	7 (26.9)	1 (7.7)	6 (22.2)	0.378
Hypotensive episode (No)	1.9±1.58, 1.5 (1–2.25)	2.3±1.07, 2 (1.75–3)	2.2±1.28, 2 (1–3)	0.576
Hypertensive episode (No)	1.1±1.13, 1 (0–1.25)	0.7±0.95, 0 (0–1.25)	1.1±1.07, 1 (0–2)	0.541
Use of Vasopressor (No)	0.5±0.86, 0 (0–1)	0.4±0.63, 0 (0–1)	0.9±1.33, 0 (0–1)	0.179
Use of Vasodilator (No)	0.7±1.09, 0 (0–1)	0.3±0.61, 0 (0–1)	0.5±0.8, 0 (0–1)	0.392
Crystalloid (mL)	1800±620.16	1921.4±1002.06	2125.9±754.9	0.302
Colloid (mL)	373.7±295.64, 300 (100–550)	514.3±295.76, 600 (150–700)	402.6±315.4, 510 (100–550)	0.370
Blood loss (mL)	496.4±133.29	464.3±172.57	485.7±183.36	0.839
HOD (day)	11.6±5.54, 10 (7.75–15)	11.4±3.23, 12 (8.75–13)	12.7±6.09, 11 (8–15)	0.697

Notes: Group E: epidural anesthesia, Group S: subdural anesthesia, Group E+S: combined epidural and subdural anesthesia. Data are presented as numbers of patients (%) or mean ± SD, medians (P_{25}–P_{75}).
Abbreviations: ASA, American Society of Anesthesiologists; No, number; HOD, hospital day.

Patients with epidural, subdural, and combined epidural + subdural anesthesia for 1 to 3 level open or endoscopic decompression and fusion were compared for high-level block (above and including T5) and non-high-level block (below T6). There was no evidence of difference with regard to the number of hypotensive episodes, the use of vasopressors, or the amount of crystalloid and colloid used in all groups (Table 4).

In the cohort with high-level blocks at or above T5, 48 patients (17.1%) had epidural anesthesia, 16 patients (15.1%) had subdural anesthesia, and 27 patients (15.9%) had combined epidural and subdural anesthesia. There were no significant differences among these three groups in terms of the frequency of blood pressure fluctuations, vasopressor usage, or fluid administration (Table 5).

In the epidural anesthesia group, a total of 233 patients were classified as ASA II or lower, while 48 patients were classified as ASA III or higher. Those in the ASA III or higher category exhibited greater blood loss and longer hospital duration (HOD); however, no significant differences were observed in the frequency of blood pressure drops, vasopressor requirements, or fluid administration based on ASA classification.

Table 4 Comparison Between ≤ T5 High-Level Block and Non-High-Level Block Across Each Anatomical Space

		Hypotensive Episode Number (No)	Use of Vasopressor Number (No)	Crystalloid (mL)	Colloid (mL)
Open/endo					
Group E	> T5 level block (233)	1.67±1, 1 (0–2)	0.24±0.61, 0 (0–0)	1482.8±416	49.1±135.54
	≤ T5 level block (48)	1.52±1.19, 1 (0–1)	0.23±0.69, 0 (0–0)	1463.8±472.29	60.6±151.85
	P value	0.379	0.91	0.778	0.6
Group S	> T5 level block (90)	1.78±0.96, 1 (0–2)	0.39±0.91, 0 (0–0)	1312.8±440.03	98±189.5
	≤ T5 level block (16)	2.13±1.31, 2 (0–2)	0.44±0.73, 0 (0–1)	1300±391.58	78.1±167.3
	P value	0.211	0.84	0.914	0.695
Group E+S	> T5 level block (143)	1.85±1.07, 1 (0–2)	0.29±0.78, 0 (0–0)	1511.2±410.16	67.13±194.81
	≤ T5 level block (27)	2.22±1.5, 2 (0–2)	0.22±0.51, 0 (0–0)	1614.8±330.16	113.1±175.07
	P value	0.232	0.682	0.217	0.255
Fusion					
Group E	> T5 level block (19)	2.21±1.72, 2 (0–2)	0.58±0.96, 0 (0–1)	1805.3±642.24	384.7±317.34
	≤ T5 level block (7)	1±0.58, 0 (0–2)	0.29±0.49, 0 (0–0)	1785.7±603.96	343.6±246.22
	P value	0.083	0.452	0.945	0.76
Group S	> T5 level block (12)	2.33±1.16, 2 (0–2)	0.42±0.67, 0 (0–1)	1941.7±909.5	491.7±314.67
	≤ T5 level block (1)	2	0	400	700
	P value	0.787	0.561	0.132	0.538
Group E+S	> T5 level block (21)	2.43±1.33, 2 (0–2)	1.1±1.45, 1 (0–1)	2228.6±802.27	431.4±325.87
	≤ T5 level block (6)	1.5±0.84, 1 (0–2)	0.33±0.52, 0 (0–1)	1766.7±435.51	301.7±277.14
	P value	0.119	0.056	0.192	0.385

Note: Data are presented as mean ± SD.

Abbreviations: OPEN, Open discectomy from 1 to 3 level and/or decompression; Endo, Endoscopic discectomy from 1 to 3 level and/or decompression; No, number.

Table 5 Comparison of Hemodynamic Variances in Patients with ≤ T5 High-Level Block Across Each Anatomical Space in Those Undergoing Decompression Surgery

	Type of Anesthesia			P value
	Group E (n=48)	**Group S (n=16)**	**Group E+S (n=27)**	
High block (≤ T5)	(17.1)	16 (15.1)	27 (15.9)	0.878
Hypotensive episode number	1.52±1.19, 1 (1–2)	2.13±1.31, 2 (1–2)	2.22±1.5, 2 (1–3)	0.057
Hypertensive episode number	0.79±1.07, 0.5 (0–1)	1.31±1.74, 1 (0–2)	1.44±1.37, 1 (0–2)	0.104
Use of Vasopressor number	0.23±0.69, 0 (0–0)	0.44±0.73, 0 (0–1)	0.22±0.51, 0 (0–0)	0.501
Use of Vasodilator number	0.58±1.03, 0 (0–1)	0.94±1.61, 0 (0–1)	1.22±1.53, 1 (0–2)	0.124
Crystalloid (mL)	1463.75±472.29, 1500 (1162–1775)	1300±391.58, 1250 (1074–1675)	1614.81±330.16, 1650 (1400–2000)	0.062
Colloid (mL)	60.63±151.85, 0 (0–0)	78.13±167.3, 0 (0–50)	113.15±175.07, 0 (0–150)	0.405
Blood loss (mL)	144.38±114.48, 100 (50–200)	195.31±135.74, 135 (100–300)	223.15±143.79*, 200 (100–350)	0.034
HOD	4.44±3.07, 3.5 (2–6)	7.25±4.85, 5.5 (4–10.5)	6.11±5.29, 4 (3–8)	0.043

Notes: *$p < 0.05$ compared with group E. Data are presented as means + SD, medians (P_{25}–P_{75}), or numbers of patients (%).
Abbreviation: HOD, hospital day.

In the subdural anesthesia group, 81 patients were classified as ASA II or lower, and 28 patients as ASA III or higher. There were no significant differences in the frequency of blood pressure drops, vasopressor requirements, or fluid administration between the ASA classifications.

In the combined epidural and subdural anesthesia group, 135 patients were classified as ASA II or lower, and 35 patients as ASA III or higher. Although there was no significant difference in the frequency of blood pressure fluctuations between the ASA classifications, patients with ASA III or higher experienced greater blood loss, had higher colloid solution usage, and required more frequent vasopressor administration compared to those with ASA II or lower (Table 6).

Table 6 Comparison of Hemodynamic Variances in Patients with Different ASA Classifications Across Each Anatomical Space in Those Undergoing Decompression Surgery

	≤ ASA 2	**≥ ASA 3**	**P value**
EPIDURAL	N=233 (82.9)	N=48 (17.1)	–
Hypotensive episode (No)	1.6±0.97, 2 (1–2)	1.85±1.27, 2 (1–2.75)	0.116
Hypertensive episode (No)	0.66±0.96, 0 (0–1)	0.98±1.23, 1 (0–2)	0.045*
Use of Vasopressor (No)	0.21±0.54, 0 (0–0)	0.4±0.92, 0 (0–0)	0.171
Use of Vasodilator (No)	0.47±0.88, 0 (0–1)	0.73±1.14, 0 (0–1)	0.147
Crystalloid (mL)	1466.7±429.63, 1500 (1200–1750)	1541.88±402.01, 1650 (1300–1800)	0.266
Colloid (mL)	50.99±140.13, 0 (0–0)	51.46±130.1, 0 (0–8.75)	0.983
Blood loss (mL)	121.65±103.46, 100 (50–150)	163.65±98.33, 162.5 (65–250)	0.01*
HOD (day)	4.22±2.87, 3 (2–5)	5.79±4.34, 4 (3–7.75)	0.02*

(*Continued*)

Table 6 (Continued).

	≤ **ASA 2**	≥ **ASA 3**	**P value**
SUBDURAL	N=81 (74.3)	N=28 (25.7)	–
Hypotensive episode number (No)	1.79±1.05, 2 (1–2)	1.89±0.92, 2 (1–2.75)	0.645
Hypertensive episode number (No)	0.7±1.1, 0 (0–1)	1±1.12, 1 (0–1.75)	0.224
Use of Vasopressor number (No)	0.4±0.82, 0 (0–0)	0.43±1.07, 0 (0–0)	0.864
Use of Vasodilator number (No)	0.54±1.04, 0 (0–1)	1±1.36, 0.5 (0–1.75)	0.067
Crystalloid (mL)	1349.38±425.77, 1400 (1075–1625)	1182.14±433.81, 1300 (850–1437.5)	0.077
Colloid (mL)	78.02±174.36, 0 (0–5)	158.93±230.56, 25 (0–300)	0.098
Blood loss (mL)	150.43±149.07, 100 (50–200)	196.07±177.9, 135 (72.5–250)	0.187
HOD (day)	5.96±4.08, 5 (3–7.5)	6.89±4.47, 6 (4–8.75)	0.313
Epidural + Subdural	N=135 (79.4)	N=35 (20.6)	–
Hypotensive episode number (No)	1.93±1.14, 2 (1–2)	1.86±1.22, 2 (1–2)	0.755
Hypertensive episode number (No)	0.88±1.1, 1 (0–2)	1.09±1.42, 1 (0–2)	0.36
Use of Vasopressor number (No)	0.33±0.82, 0 (0–0)	0.09±0.28, 0 (0–0)	0.005*
Use of Vasodilator number (No)	0.55±0.84, 0 (0–1)	1.09±1.42, 1 (0–2)	0.038*
Crystalloid (mL)	1518.48±402.56, 1500 (1250–1800)	1562.86±390.95, 1600 (1250–1800)	0.56
Colloid (mL)	58.74±188.66, 0 (0–0)	135±195.75, 0 (0–250)	0.044*
Blood loss (mL)	153.19±119.25, 100 (70–200)	252.71±179.66, 200 (100–350)	0.003*
HOD (day)	5.43±4.47, 4 (3–7)	6.4±4.93, 5 (3–8)	0.264

Notes: Data are presented as means + SD, medians (P_{25}–P_{75}), or numbers of patients (%), *$p < 0.05$.
Abbreviations: ASA, American Society of Anesthesiologists; HOD, hospital day.

Discussion

Regional anesthesia leads to a reduction in blood pressure (BP = CO x SVR) primarily through the blockade of preganglionic sympathetic nerves from T1 to L2, in conjunction with a reduced release of endogenous catecholamines from the adrenal glands. This blockade induces vasodilation and hence decreases systemic vascular resistance (SVR, afterload).[1–4] Secondly, blockade from T6 to L1 results in dilation of the splanchnic veins, leading to reduced venous return (preload) and consequently decreased cardiac output (CO).[17,18] Additionally, a high-level blockade involving the cardiac accelerator fibers from the T1–T4 spinal segments, or through modulation by the reverse Bainbridge reflex, leads to a decline in heart rate (HR) and myocardial contractility. This cascade effect precipitates a significant decrease in CO, accompanied by a marked reduction in arterial blood pressure due to compounded reductions in SVR.[1–4] Furthermore, a higher level of sympathetic blockade proportionally diminishes the engagement of compensatory mechanisms via baroreceptor activity, thereby increasing susceptibility to cardioinhibitory reflexes such as the Bezold-Jarisch reflex,[19–21] which may ultimately lead to cardiac arrest and mortality.[22] In conclusion, sympathetic block below the L1 level does not have a significant impact on blood pressure.

The spinal nerves in the central nervous system are encased in three protective membranes, creating distinct spaces known as the epidural, subdural, and subarachnoid spaces. Anesthesia can theoretically be administered in each of these spaces. Deeper within, the spinal nerves encased in the pia mater float in cerebrospinal fluid (CSF). Anteriorly, these nerves carry somatic motor fibers along with autonomic motor fibers, which can be either parasympathetic or sympathetic, depending on the spinal level. Posteriorly, somatic and visceral sensory fibers travel together (Figure 2).

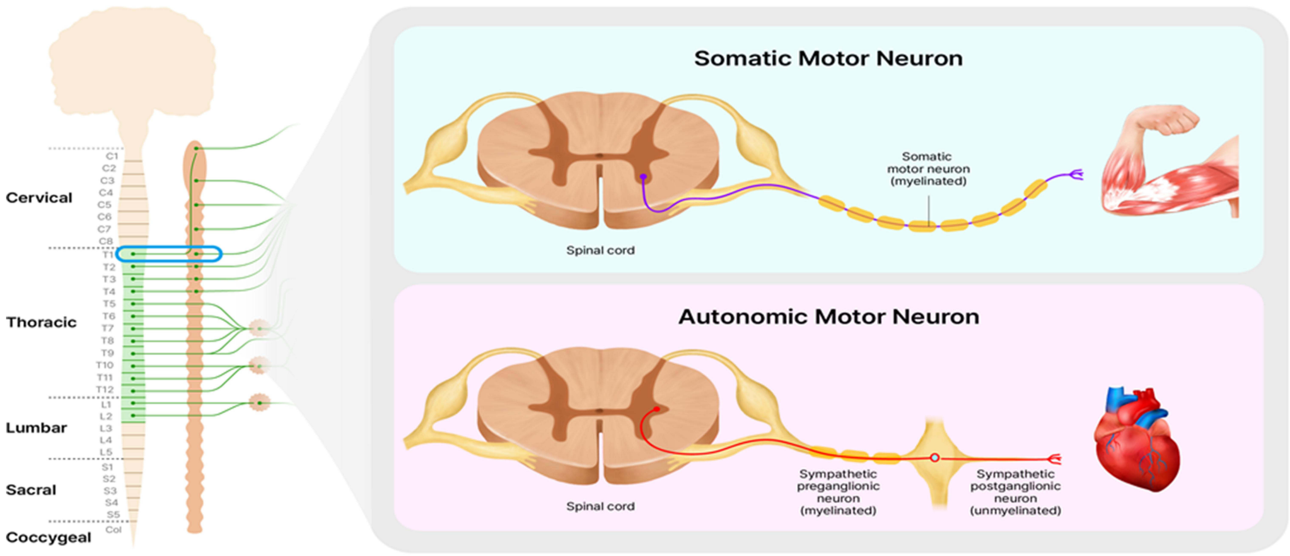

Figure 2 At T1-L2 levels, the anterior nerves contain both motor fibers (myelinated A-fiber), which are responsible for movement, and preganglionic sympathetic fibers (myelinated, B-fiber). Beyond the paravertebral ganglion, the sympathetic nerve fibers continue as unmyelinated C postganglionic sympathetic fibers.

Consequently, spinal (subarachnoid) anesthesia has the potential to block the entire nerve, impacting both anterior and posterior fibers in a similar manner from the injection site.

Epidural Anesthesia

Compared to subarachnoid anesthesia, where the local anesthetic injection is administered directly near the spinal nerves, larger volume of local anesthetics in the epidural space can be significantly diminished as they traverse through fat and vessels before reaching both the spinal nerve roots and the central spinal nerve. Consequently, the actual dosage of anesthetic that reaches the nerves is limited, leading to a less dense effect on the posterior sensory nerves, anterior motor nerves, and sympathetic nerves.[23] This leads to a smaller reduction in CO, SVR, and, consequently, blood pressure. In this study, patients undergoing decompression or fusion surgery with epidural anesthesia and MAC demonstrated hemodynamic stability even in patients with a high-level block above T5 or those classified as ASA 3 or higher. Hypotensive episodes were recorded as 1.6 ± 1 in decompression surgery and 0.9 ± 1.58 in fusion surgery. These hypotensive episodes were effectively managed with fluid administration and vasopressor administration, preventing additional adverse events.

Four factors explain the hemodynamic stability observed in our study: 1) Anesthetic distribution: Contrary to concerns about the anesthetic migrating toward the anterior portion of the epidural space in the prone position, it instead spreads exclusively toward the posterior region. This minimizes its effect on the anterior sympathetic and motor nerves, thereby helping to maintain stable blood pressure (Figure 3). Traditionally, epidural anesthesia is performed with the patient in a sitting or lateral decubitus position, followed by repositioning to supine to assess sensory dermatomes.[24,25] However, our method involves administering anesthesia with the patient already in the prone position on a Wilson frame, where they remain throughout the procedure which may promote anterior spread due to the gravitational effect on the anesthetic and contrast medium. Despite concerns about blocking anterior motor and preganglionic sympathetic nerves, our observations indicate that, in the absence of previous surgical adhesions or severe stenosis, the anesthetic primarily spreads longitudinally toward the posterior region, then bilaterally, and eventually circumferentially toward the anterior,[8] consistent with findings from Yokoyama et al, on epidural solution spread.[26] 2) Differential nerve block: Different nerves exhibit varying sensitivities to local anesthetics. While unmyelinated pain and postganglionic sympathetic nerves (C fibers) are the most resistant to local anesthetics, posterior pain fibers (A-delta fibers) are more sensitive to anesthetics than anterior motor fibers (A-alpha fibers) and preganglionic sympathetic fibers (B fibers). This differential sensitivity contributes to the preservation of motor and sympathetic functions while effectively blocking pain.[27] 3) Ropivacaine

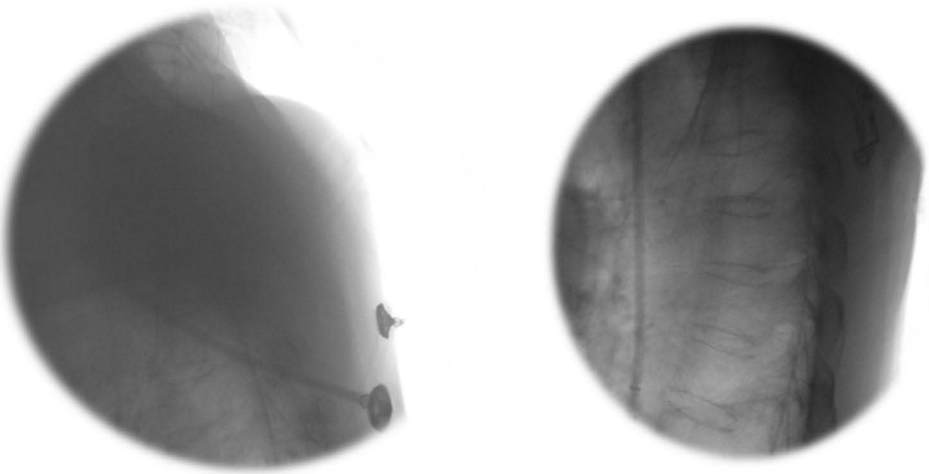

Figure 3 10 min post-anesthesia in the prone position: in the case of an epidural high-level block above T5, the radiocontrast media administered alongside the anesthetic agent migrates longitudinally in a cephalad direction exclusively within the posterior epidural space.

properties: Ropivacaine, the local anesthetic used, preferentially blocks sensory nerves over motor nerves, which helps maintain motor function and overall hemodynamic stability during surgery.[28,29] 4) Dexmedetomidine property: In contrast to spinal anesthesia, which frequently results in a rapid decline in blood pressure, epidural anesthesia is characterized by a delayed and gradual reduction in blood pressure. The biphasic hemodynamic effects of dexmedetomidine appear to be partially attenuated the hypotensive effects of epidural anesthesia by its initial hypertensive effect.[30] Additionally, the analgesic properties of dexmedetomidine provide an opioid-sparing effect, potentially reducing the need for remifentanil, which may offset the hypotensive effects of remifentanil.[31]

Subdural Anesthesia

Subdural anesthesia, proposed by Reina in 2002 as arising from iatrogenic traumatic fissures rather than from a naturally existing space, depends on the pressure and volume of the injectate to create tearing and separation between the dura and arachnoid mater.[32] Depending on the location and extent of the tearing, this separation results in patchy anesthesia patterns rather than a uniform nerve blockade.

In this study, patients undergoing decompression or fusion surgery with subdural or combined epidural-subdural anesthesia generally maintained stable blood pressure. Significant upward extension of the break to higher thoracic / cervical levels is unlikely, unless fluids are injected at very high volumes and pressures. In our study, high level blocks occurred in 16 of 109 patients (15.1%) who underwent decompression surgery and 1 of 14 patients (7.7%) who underwent fusion surgery. High-level subdural blocks predominantly display posterior subdural images with small or no anterior subdural images visibility (Figure 4) consistent with Agalwar's findings.[33] Subarachnoid images in the lateral view reveal a larger anterior radiolucent space in the lower lumbar region, suggesting a larger anterior epidural/subdural space in this area that decreases as it ascends.[34,35] This suggests that the potential anterior subdural space, notable in the lower lumbar region for motor block induction, significantly diminishes towards the thoracic area (Figure 5), limiting anterior anesthetic spread (Figure 4 arrow). Furthermore, if we delve into a more microscopic level of anatomy, there are separate attachments of the dura mater and the arachnoid mater on the dorsal nerve root; the arachnoid being fixed proximal to the ganglion, the dura distal to it. The arachnoid and dura are attached together on the ventral root and so the potential space is much greater over the dorsal root ganglion (Figure 6), and theoretically there may be preferential pooling of the local analgesic solution at this point (posterior).[33,36] If this were the case, sensory loss could be profound, with sparing of the pre-ganglionic sympathetic fibers and the motor fibers carried in the ventral roots. Therefore, it is plausible to infer that fissure formation within the subdural space occurs more readily in the longitudinal (posterior) direction rather than circumferentially (anterior).

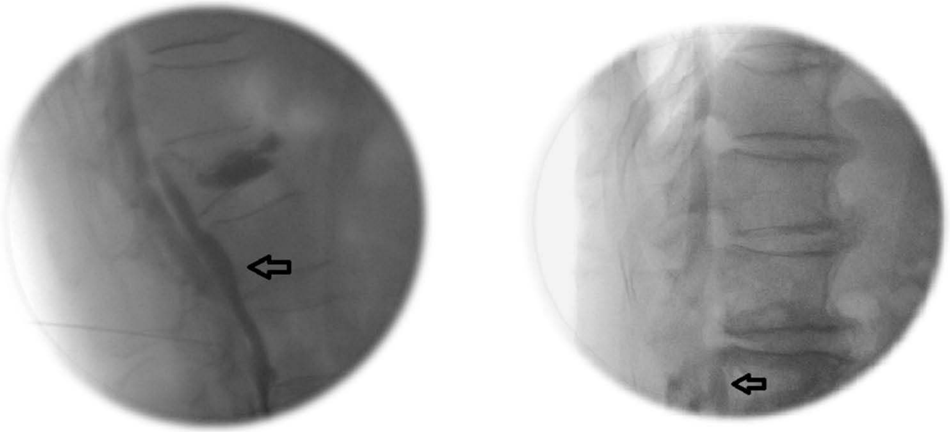

Figure 4 High-level subdural blocks predominantly display posterior subdural images with no or small anterior subdural (arrow) image visibility.

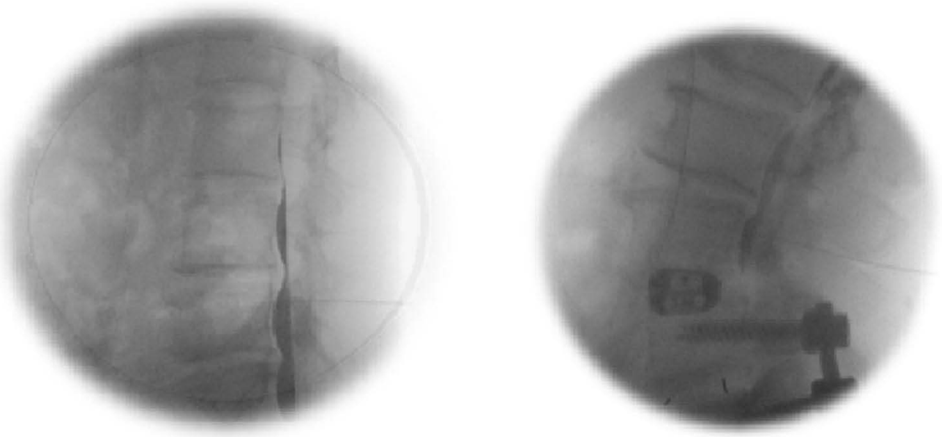

Figure 5 Subarachnoid image in lateral view: a single-layer image shows a pattern of hyperbaric contrast medium in the most ventrally dependent region featuring ventral undulations attributable to the arachnoid mater when in the prone position. A line of lucency between the contrast spread and the posterior vertebral body suggests the area within the anterior epidural and subdural space. In the lower lumbar area, the corresponding size is larger, and this area decreases in size as it ascends.

In our examination of radiocontrast images from cases with motor blockade, we observed that the anesthetic sometimes spreads below the L1 level, reaching the anterior subdural space. This is possibly due to the larger capacity of the anterior subdural space only in the lower lumbar region, where the sympathetic block does not significantly impact blood pressure. Conversely, there have been only two documented cases—excluding instances where prior dura tears led to obliteration of the post-subdural space after dural repair—where the anesthetic extended above the L1 level into the anterior subdural space (Figure 7).

However, a greater number of the images of subdural anesthesia predominantly show posterior bulging, resembling a sausage-like shape. This indicates that, in these cases, the anesthetic is confined within the posterior subdural space. These findings support previous reports,[37,38] confirming that the anesthetic generally does not migrate anteriorly within the subdural space. Consequently, motor blockade is typically absent in such cases, consistent with our observations.

Combined Epidural-Subdural Anesthesia

In patients with combined epidural-subdural anesthesia, fluoroscopic radiocontrast imaging demonstrates that the anesthesia pattern shifts from the subdural to the epidural space as the anesthetic ascends, which helps maintain

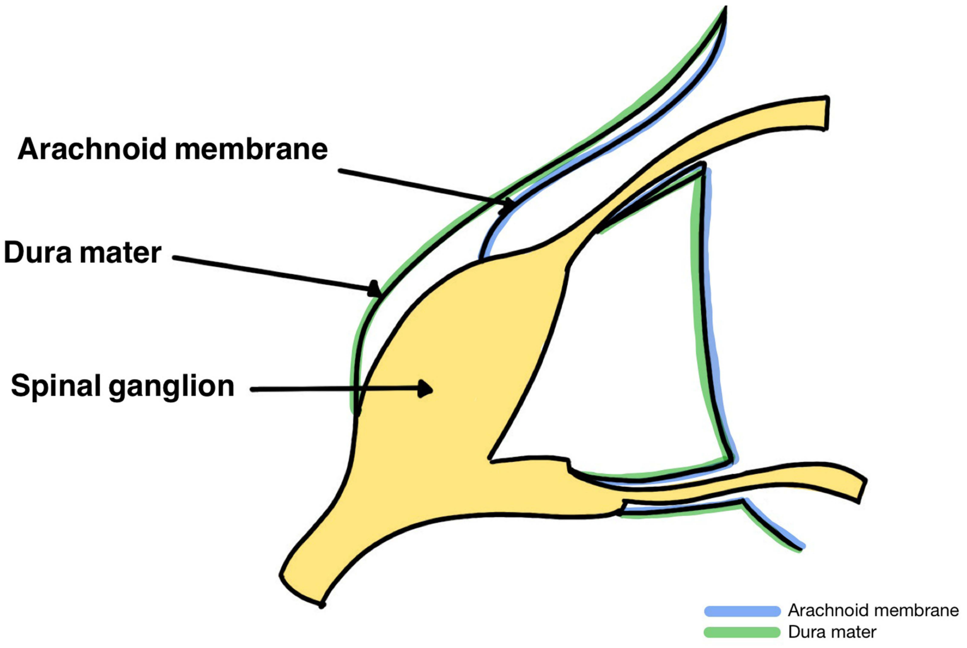

Figure 6 The size difference between the posterior and anterior subdural spaces is caused by the different attachment sites of the dura mater and arachnoid mater.

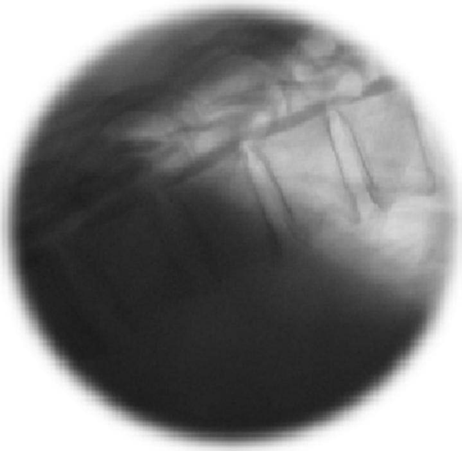

Figure 7 If a patient has a history of dural tear with repair from a previous spinal surgery, the posterior subdural space may be obliterated, increasing the possibility of anesthetic spread to the anterior subdural space.

hemodynamic stability. Consequently, even with high-level blocks above the T5 level, the anesthesia behaves similarly to an epidural block, resulting in minimal hemodynamic effects, even in patients with more severe health conditions classified as ASA III or higher.

Our findings should be interpreted cautiously due to several limitations. First, the retrospective nature of the study prevented routine postoperative CT scans necessary to confirm needle placement and precise spatial localization. Second, the hemodynamic effects of sedatives such as dexmedetomidine, remifentanil, and midazolam also compound the results, making it difficult to assess the hemodynamic outcomes attributable solely to regional anesthesia. Third, retrospective studies like ours may not capture the full array of unmeasured variables influencing outcomes, and potential confounding factors could skew results. Finally, the reliance of the study on data from a single institution may limit the generalizability of our findings to broader populations. This underscores the need for a prospective, large-scale, multi-center study to validate these results and provide robust guidance for applying epidural anesthesia in various surgical contexts.

Conclusion

In patients receiving high-level epidural and subdural anesthesia, the anesthetic predominantly spreads posteriorly, leading to a more effective sensory block while producing a comparatively lesser sympathetic block anteriorly, thereby reducing the impact on blood pressure. In high-level subdural blocks, similar to epidural anesthesia, fissures tend to form mainly in the posterior region, with anterior fissures occurring primarily below the L1 level. Consequently, despite the presence of motor block, there were minimal fluctuations in blood pressure.

Hemodynamic stability was maintained in the subdural and combined epidural–subdural groups compared to the epidural group in patients with ASA status I to III. Performing monitored anesthesia care (MAC) in the prone position involves certain risks. In particular, patients with airway issues or sleep apnea should undergo careful assessment of the risks and benefits.

Although our observations are not directly compared to general anesthesia and are limited by the retrospective, single-center design, we propose that epidural anesthesia may be a feasible option for spinal surgeries and may have potential applicability in other procedures for patients with high ASA classifications. However, given the unconventional and unintended nature of subdural anesthesia, these findings should be interpreted with caution, and further prospective studies are warranted.

Disclosure

The authors declare that there is no conflicts of interest regarding the publication of this paper.

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