

## ORIGINAL RESEARCH



# Sevoflurane versus desflurane on haemodynamics, arterial oxygenation and pulmonary mechanics in prone position during spinal surgery

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**Abstract**

**Background:** This study aimed to compare the effects of equipotent doses (one minimum alveolar concentration) of sevoflurane and desflurane on respiratory mechanics, haemodynamics and oxygenation in patients undergoing spinal surgery in the prone position. **Methods:** Fifty patients were randomised to receive either sevoflurane (n = 25) or desflurane (n = 25). Respiratory parameters (dynamic compliance (C<sub>dyn</sub>); peak (P<sub>peak</sub>), mean (P<sub>mean</sub>) and plateau (P<sub>plateau</sub>) airway pressures; driving pressure ( $\Delta$ P); tidal volume; and dead space), haemodynamic parameters (heart rate (HR) and systolic, mean and diastolic arterial pressures) and oxygenation parameters were recorded intraoperatively at baseline, after prone positioning, during surgery, and after returning to the supine position. **Results:** Prone positioning led to significant increases in P<sub>peak</sub> and reductions in C<sub>dyn</sub> in both groups ( $p < 0.05$ ). Although P<sub>peak</sub>, P<sub>mean</sub>, P<sub>plateau</sub> and  $\Delta$ P fluctuated intraoperatively, no intergroup differences were detected ( $p > 0.05$ ). After returning to the supine position, respiratory mechanics approached baseline in both groups. Oxygenation (arterial oxygen pressure), ventilation (arterial carbon dioxide pressure), end-tidal carbon dioxide, and pH remained stable and comparable. Sevoflurane was associated with slightly greater decreases in arterial pressures, whereas HR was similar between groups. **Conclusions:** Both desflurane and sevoflurane maintained stable intraoperative respiratory mechanics, oxygenation, and haemodynamics in patients without pulmonary disease. Prone positioning increased P<sub>peak</sub> and decreased C<sub>dyn</sub> similarly in both groups. Both agents appear safe for spinal surgery in the prone position. **Clinical Trial Registration:** This study was registered at [ClinicalTrials.gov](https://clinicaltrials.gov) (NCT06118489).

**Keywords**

Sevoflurane; Desflurane; Prone position; Spinal surgery; Haemodynamics; Respiratory mechanics; Oxygenation; Anaesthesia

## 1. Background

Volatile agents used in general anaesthesia are known to have various effects on the airways to different degrees. Sevoflurane, one of the most preferred agents, has been shown to reduce airway resistance [1] and peak airway pressure (P<sub>peak</sub>) more than many other agents, while also increasing dynamic compliance (C<sub>dyn</sub>) more effectively [2–5].

Desflurane, which features rapid onset, quick awakening and a fast return to normal orientation and pharyngeal reflexes [6, 7], has a sharp and pungent odour at room temperature, which may lead to breath holding or laryngospasm during anaesthesia induction [3]. It has also been demonstrated that desflurane, when administered at high concentrations, can cause airway irritation, as well as a temporary increase in bronchial secretions and airway resistance [8]. However, it

is also thought that this agent may induce bronchodilation through sympathetic stimulation [2, 9].

In spinal surgeries, patients are placed in the prone position to allow access to the surgical site. As a result, complications related to respiration and circulation may develop [10]. Prone positioning increases thoracoabdominal pressure and reduces venous return through the inferior vena cava, which in turn leads to a decrease in cardiac preload and cardiac index [11]. Moreover, the prone position causes changes in the respiratory system. Although the ventilation–perfusion mismatch decreases and arterial oxygenation improves, lung compliance is reduced and P<sub>peak</sub> increases [12–15].

The aim of this study was, therefore, to compare the effects of two different anaesthetic gases on respiratory mechanics, haemodynamics, oxygenation and gas exchange during spinal surgery performed in the prone position. Understanding how

desflurane and sevoflurane affect these parameters in the prone position is clinically relevant as altered respiratory mechanics and haemodynamic stability directly impact perioperative safety and postoperative recovery in spinal surgery patients.

## 2. Methods

### 2.1 Study design

This research was designed as a prospective, randomised, single-centre, single-blind experimental study. Ethical approval was granted by the Hamidiye Clinical Research Ethics Committee on 23 November 2023 (approval no: 21-108), and written informed consent was obtained from all participants. The study was registered at [ClinicalTrials.gov](https://www.clinicaltrials.gov) (NCT06118489) on 31 October 2023. Patients were recruited between December 2023 and February 2024.

### 2.2 Patient selection

A total of 50 patients aged 18–65 years with an American Society of Anaesthetists (ASA) physical status I–II were enrolled. Exclusion criteria included pulmonary disease, hepatic or renal dysfunction, Raynaud's or Buerger's disease, neuromuscular disorders, history of thoracic surgery, major surgical interventions, hypotension, failed Allen test, a body mass index (BMI)  $\geq 35$ , active smoking, and age below 18 or above 65 years. All patients were instructed to fast for at least eight hours preoperatively. Before surgery, demographic data, such as age, sex, height, weight, comorbidities and ASA status were recorded.

### 2.3 Anaesthesia protocol

Upon entering the operating theatre, a 20G intravenous cannula was inserted into the dorsum of the hand, and premedication was administered with intravenous midazolam (0.025 mg/kg).

Standard monitoring included noninvasive blood pressure, 3-lead electrocardiography (ECG), pulse oximetry, capnography, bispectral index (BIS; Covidien Inc., USA), and train-of-four (TOF; Draegerwerk AG & Co. KGaA) neuromuscular monitoring. Anaesthesia was induced with intravenous propofol (2–2.5 mg/kg), lidocaine (1.5 mg/kg), fentanyl (2  $\mu$ g/kg), and rocuronium (0.6 mg/kg) for muscle relaxation. Muscle relaxation was maintained with rocuronium, with additional doses administered as needed to maintain TOF 0/4 throughout surgery. TOF stimulation was performed at the adductor pollicis muscle, and measurements were recorded at specified intraoperative time points to ensure adequate neuromuscular blockade.

Endotracheal intubation was performed when BIS was between 40 and 60 and TOF showed complete neuromuscular blockade. Female patients were intubated using 7.0–7.5 mm tubes and males with 7.5–8.0 mm tubes. Mechanical ventilation was provided with a Draeger Primus™ ventilator. Maintenance of anaesthesia was achieved with 1 minimum alveolar concentration (MAC) desflurane or sevoflurane in a 50% oxygen/air mixture at a flow rate of 3 L/min. Remifentanyl infusion (0.05–0.2  $\mu$ g/kg/min) was administered continuously. No vasoactive or inotropic medications were administered

intraoperatively as all patients remained haemodynamically stable under the study protocol.

Desflurane 6% and sevoflurane 2% were administered, corresponding approximately to 1 MAC for adult patients aged 18–65 years. Although MAC decreases modestly with age, the chosen concentrations are within the clinically acceptable range for this age group. Depth of anaesthesia was continuously monitored using BIS and haemodynamic parameters.

Following induction, a 20G radial artery catheter was inserted to monitor invasive arterial pressure and collect arterial blood samples. Ventilation was managed in volume-control mode with an inspiratory-to-expiratory (I:E) ratio of 1:2, positive end-expiratory pressure (PEEP) of 5 cmH<sub>2</sub>O, respiratory rate of 12–14 breaths per minute, tidal volume (V<sub>t</sub>) set at 8 mL/kg of ideal body weight, and end-tidal carbon dioxide (EtCO<sub>2</sub>) maintained between 32 and 38 mmHg. The plateau phase (inspiratory hold) was set to 0.4 seconds for all patients to allow accurate measurement of plateau pressures and calculation of C<sub>dyn</sub>.

### 2.4 Randomisation and blinding

Patients undergoing spinal surgery were randomised in a 1:1 ratio to receive either desflurane or sevoflurane. Randomisation was conducted using a computer-generated sequence, with group allocations placed in sealed opaque envelopes opened immediately prior to induction.

Blinding was maintained throughout the study. While the anaesthetist managing the anaesthesia was aware of the inhalational agent for safety reasons, the patients and the data-collecting researchers remained blinded. The anaesthetic monitor did not reveal the agent used, preserving blinding during intraoperative and postoperative phases. This was a single-blind study with blinded outcome assessors: the patients and outcome assessors were blinded to group allocation, while the anaesthetist was not blinded due to safety concerns. No measures such as separate personnel for anaesthesia management and data collection were implemented; however, data collectors were unaware of group assignments, which helped reduce potential bias. Group D patients received 1 MAC desflurane (6%), and Group S received 1 MAC sevoflurane (2%).

### 2.5 Outcome measures and time points

The following parameters were recorded at specific time points: T0 (baseline, preoperative), T1 (15 min post-intubation, supine), T2 (15 min after prone positioning), T3 (30 min in prone), T4 (1 hour in prone) and T5 (15 min after returning to supine). Parameters included heart rate (HR; beats per minute), mean arterial pressure (MAP, mmHg), peripheral oxygen saturation (SpO<sub>2</sub>, %), EtCO<sub>2</sub> (mmHg), C<sub>dyn</sub> (mL/cmH<sub>2</sub>O), and airway pressures (peak, plateau, mean; cmH<sub>2</sub>O). Arterial blood gases (pH, arterial oxygen pressure (PaO<sub>2</sub>, mmHg), arterial carbon dioxide pressure (PaCO<sub>2</sub>, mmHg), bicarbonate (HCO<sub>3</sub><sup>-</sup>) (mmol/L) and arterial oxygen saturation (SaO<sub>2</sub>, %) were also collected at these time points. During data collection, TOF values were maintained at 0%. Driving pressure ( $\Delta$ P) was calculated as the difference between plateau pressure (P<sub>plateau</sub>) and PEEP

at each predefined measurement time point ( $\Delta P = P_{\text{plateau}} - P_{\text{EEP}}$ ).

Patients were placed in the standard prone position on the operating table with the head supported on a soft foam pillow and the chest, pelvis, and extremities padded with conventional surgical cushions, ensuring that the abdomen remained free. No special positioning frames (*e.g.*, Wilson, Andrews, Relton or Jackson table) were used.

After completion of the procedure, patients were returned to the supine position, and final measurements were recorded. Inhalational agents were discontinued, and 100% oxygen was administered. Neuromuscular blockade was reversed with sugammadex (2 mg/kg), and extubation was performed upon adequate spontaneous respiration. The duration of surgery and prone positioning time were documented.

The primary objective was to compare intraoperative pulmonary compliance (dynamic compliance,  $C_{\text{dyn}}$ ) between the desflurane and sevoflurane groups during spinal surgery in the prone position. Secondary objectives included comparisons of  $P_{\text{peak}}$  and  $P_{\text{plateau}}$  airway pressures,  $\Delta P$ , HR, MAP, arterial oxygenation, and  $\text{EtCO}_2$ , as well as the evaluation of the effects of positional changes on both respiratory and haemodynamic parameters. Surgical duration and prone positioning times were also recorded.

No patients required withdrawal from the study. Intraoperative safety was continuously monitored, including airway pressures and oxygen saturation. Although prone positioning may increase airway pressures, predefined safety thresholds were established (*e.g.*,  $\text{SpO}_2 < 90\%$  or  $P_{\text{peak}} > 40 \text{ cmH}_2\text{O}$ ). If these thresholds had been exceeded, appropriate rescue manoeuvres, including recruitment manoeuvres or adjustment of ventilatory parameters, would have been applied and the patient could have been withdrawn from the study if deemed necessary. Because no patients reached these criteria, all enrolled participants completed the study.

## 2.6 Sample size calculation

The sample size was estimated using G\*Power software (version 3.1.9; Heinrich-Heine-Universität Düsseldorf, Düsseldorf, NRW, Germany), guided by data from a previous

study by Eksi *et al.* [16], titled “The Comparison of Effects of Sevoflurane and Desflurane on Respiratory Mechanics in Smokers and Nonsmokers”. Post-intubation dynamic lung compliance ( $C_{\text{dyn}}$ ) values from that study were used as the primary parameter. A minimum of 21 participants per group was calculated to achieve 80% statistical power at a 5% significance level ( $\alpha = 0.05$ ), based on the assumption of normally distributed data and using a two-tailed, independent-samples *t*-test for between-group comparisons. To account for potential dropouts, each group was expanded to 25 patients. No participants were excluded during the study. The study follows the Consolidated Standards of Reporting Trials (CONSORT) guidelines for randomised controlled trials.

## 2.7 Statistical analysis

During the evaluation of the study findings, statistical analyses were performed using IBM SPSS Statistics software (version 22; IBM Corp., Armonk, NY, USA). The normality of the studied parameters was assessed using the Kolmogorov-Smirnov test, and it was determined that the parameters did not follow a normal distribution. Therefore, non-parametric tests were applied. All continuous variables are presented as medians with interquartile ranges (25th–75th percentiles) due to non-normal distribution. For comparisons between the two groups, the Mann-Whitney U test was used. Within-group comparisons over time were performed using the Friedman test (with *post hoc* Dunn’s test). Categorical data were compared using the Continuity (Yates) correction. Statistical significance was set at  $p < 0.05$ .

## 3. Results

A total of 50 patients were included in the study. Demographic data, duration of prone positioning and surgical time did not differ significantly between the desflurane and sevoflurane groups (Table 1; Fig. 1).

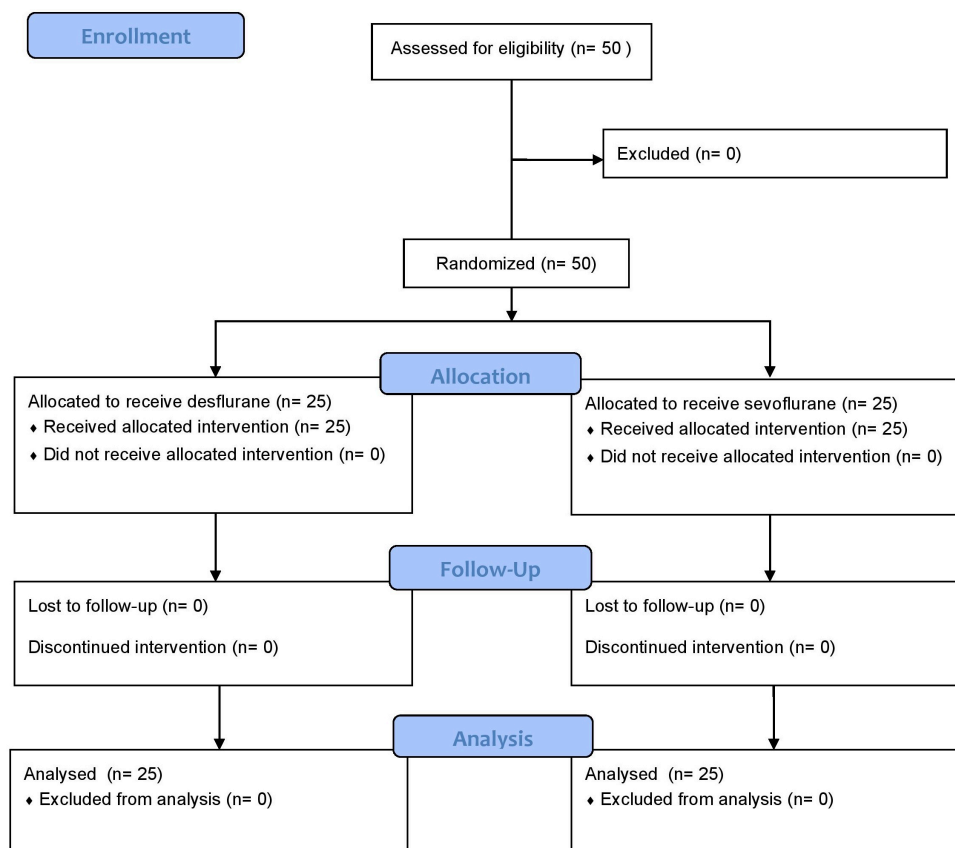
### 3.1 Respiratory mechanics

$C_{\text{dyn}}$  levels at T1–T5 did not differ significantly between the groups ( $p > 0.05$ ). In both groups,  $C_{\text{dyn}}$  decreased signifi-

TABLE 1. Demographic and clinical characteristics of patients in both groups.

	Desflurane		Sevoflurane		<i>p</i>
	Median	25th–75th percentile	Median	25th–75th percentile	
Age (yr)	52.0	46.5–58.0	59.0	43.0–63.5	0.071 <sup>1</sup>
Sex (n, %)					
Female	12.0	48.0%	11.0	44.0%	1.000 <sup>2</sup>
Male	13.0	52.0%	14.0	56.0%	
Weight (kg)	83.0	70.0–90.0	85.0	77.5–90.0	0.648 <sup>1</sup>
Height (cm)	168.0	160.0–178.0	170.0	160.0–175.0	0.800 <sup>1</sup>
BMI (kg/m <sup>2</sup> )	29.4	24.6–30.8	28.7	26.3–31.4	0.712 <sup>1</sup>
Prone time (min)	130.0	100.0–197.5	180.0	120.0–245.0	0.087 <sup>1</sup>
Operation time (min)	165.0	137.5–240.0	215.0	152.5–280.0	0.097 <sup>1</sup>

<sup>1</sup>Mann-Whitney U test; <sup>2</sup>Continuity (Yates) correction. BMI: body mass index.



**FIGURE 1. CONSORT flow diagram.** Diagram illustrating the enrolment, randomisation, allocation, follow-up, and final analysis of patients who underwent spinal surgery and were assigned to receive either sevoflurane or desflurane to compare their effects on haemodynamics, arterial oxygenation, and pulmonary mechanics. CONSORT: Consolidated Standards of Reporting Trials.

cantly at T2–T4 compared with T1 ( $p < 0.05$ ) and increased at T5 compared with T2–T4 ( $p < 0.05$ ). Ppeak, Pmean and Pplateau levels at T1–T5 were similar between groups ( $p > 0.05$ ). Within both groups, Ppeak and Pplateau increased significantly at T2–T4 compared with T1 ( $p < 0.05$ ) and decreased at T5 compared with T2–T4 ( $p < 0.05$ ). Pmean also increased significantly at T2–T4 compared with T1 ( $p < 0.05$ ) in both groups, with no significant changes at T5.  $\Delta P$  did not differ between groups at any time point ( $p > 0.05$ ). In both groups,  $\Delta P$  increased significantly at T2–T4 compared with T1 ( $p < 0.05$ ) and decreased at T5 compared with T2–T4 ( $p < 0.05$ ). Dead space ratio (Vd/Vt), Vt and dead space volume (Vd) did not differ significantly between groups ( $p > 0.05$ ), and no significant changes were observed over time ( $p > 0.05$ ) (Table 2, Fig. 2).

### 3.2 Haemodynamics

At baseline (T0), HR, systolic arterial pressure (SAP), and MAP were higher in the sevoflurane group ( $p = 0.022$ ,  $p = 0.001$ , and  $p = 0.003$ , respectively). At T4, SAP was also significantly higher in the sevoflurane group ( $p = 0.020$ ). HR at T1–T5, SAP at T1–T3 and T5, and MAP at T1–T5 did not differ between groups ( $p > 0.05$ ). Within both groups, HR and arterial pressures (SAP, MAP and diastolic arterial pressure (DAP)) decreased significantly over time compared with baseline ( $p < 0.05$ ) (Table 3).

### 3.3 Oxygenation and gas exchange

SpO<sub>2</sub> and PaO<sub>2</sub> did not differ between groups ( $p > 0.05$ ). In both groups, PaO<sub>2</sub> increased significantly over time compared with baseline ( $p < 0.05$ ). SpO<sub>2</sub> increased at later time points (desflurane: T3–T5; sevoflurane: T3–T4,  $p < 0.05$ ). The alveolar–arterial oxygen gradient (P(A–a)O<sub>2</sub>) did not differ between groups ( $p > 0.05$ ). In the sevoflurane group, P(A–a)O<sub>2</sub> at T5 was significantly lower than at T1, T3, and T4 ( $p < 0.05$ ), while the difference from T2 was not significant (Table 4). EtCO<sub>2</sub> and PaCO<sub>2</sub> did not differ between groups ( $p > 0.05$ ). In the desflurane group, EtCO<sub>2</sub> decreased at T2–T4 compared with T1 and increased at T5 compared with T2–T4 ( $p < 0.05$ ); PaCO<sub>2</sub> decreased at T4 compared with T1 ( $p < 0.05$ ). In the sevoflurane group, EtCO<sub>2</sub> decreased at T2–T3 compared with T1 ( $p < 0.05$ ), while PaCO<sub>2</sub> showed no significant changes (Table 4). The arteriovenous CO<sub>2</sub> difference (P(a–Et)CO<sub>2</sub>) did not differ between groups at T1–T4 ( $p > 0.05$ ). At T5, it was significantly higher in the sevoflurane group ( $p = 0.0029$ ). No significant intra-group changes were observed ( $p > 0.05$ ). pH did not differ between groups ( $p > 0.05$ ), but in both groups it decreased significantly at T5 compared with earlier time points ( $p < 0.05$ ), except in the desflurane group where no significant change was observed between T4 and T5 (Table 4).

**TABLE 2. Intraoperative comparison of respiratory mechanics and volumes between desflurane and sevoflurane groups.**

Group	T1 Median (25th–75th percentile)	T2 Median (25th–75th percentile)	T3 Median (25th–75th percentile)	T4 Median (25th–75th percentile)	T5 Median (25th–75th percentile)	Mann- Whitney $p$ <sup>1</sup>	Friedman $p$ (within- group) <sup>2</sup>	Significant <i>post hoc</i> comparisons <sup>3</sup>
Cdyn (mL/cmH <sub>2</sub> O)								
Desflurane	42.00 (36.80–54.50)	35.20 (30.30–37.60)	35.80 (29.50–38.70)	34.30 (31.60–39.00)	45.00 (37.40–52.90)	NS	0.001*	T1–T2, T1–T3, T1–T4, T2–T5, T3–T5, T4–T5
Sevoflurane	49.00 (43.50–56.50)	38.00 (33.90–46.00)	38.10 (33.00–43.20)	37.00 (31.20–45.40)	50.40 (42.90–57.00)		0.001*	T1–T2, T1–T3, T1–T4, T2–T5, T3–T5, T4–T5
Ppeak (cmH <sub>2</sub> O)								
Desflurane	17.00 (15.00–20.50)	21.00 (20.00–22.00)	22.00 (20.50–23.00)	22.00 (21.00–23.50)	18.00 (16.50–20.50)	NS	0.001*	T1–T2, T1–T3, T1–T4, T2–T5, T3–T5, T4–T5
Sevoflurane	18.00 (16.00–19.00)	20.00 (18.00–23.50)	20.00 (19.00–23.50)	21.00 (19.00–24.00)	18.00 (15.00–19.00)		0.001*	T1–T2, T1–T3, T1–T4, T2–T5, T3–T5, T4–T5
Pmean (cmH <sub>2</sub> O)								
Desflurane	8.00 (8.00–9.00)	9.00 (8.50–10.00)	9.00 (9.00–10.00)	9.00 (9.00–10.00)	9.00 (8.00–9.00)	NS	0.001*	T1–T2, T1–T3, T1–T4
Sevoflurane	8.00 (8.00–9.00)	9.00 (9.00–10.00)	9.00 (8.50–10.00)	9.00 (9.00–10.00)	8.00 (8.00–9.00)		0.001*	T1–T2, T1–T3, T1–T4
Pplateau (cmH <sub>2</sub> O)								
Desflurane	16.00 (14.50–19.00)	20.00 (18.00–21.00)	21.00 (18.00–21.50)	20.00 (18.00–21.50)	17.00 (15.00–19.00)	NS	0.001*	T1–T2, T1–T3, T1–T4, T2–T5, T3–T5, T4–T5
Sevoflurane	16.00 (14.50–18.00)	19.00 (16.50–22.00)	19.00 (17.00–21.50)	20.00 (17.00–22.00)	16.00 (13.50–17.50)		0.001*	T1–T2, T1–T3, T1–T4, T2–T5, T3–T5, T4–T5
ΔP (cmH <sub>2</sub> O)								
Desflurane	11.00 (9.50–14.00)	15.00 (13.00–16.00)	16.00 (13.00–16.50)	15.00 (13.00–16.50)	12.00 (10.00–14.00)	NS	0.001*	T1–T2, T1–T3, T1–T4, T2–T5, T3–T5, T4–T5
Sevoflurane	11.00 (9.50–13.00)	14.00 (11.50–17.00)	14.00 (12.00–16.50)	15.00 (12.00–17.00)	11.00 (8.50–12.50)		0.001*	T1–T2, T1–T3, T1–T4, T2–T5, T3–T5, T4–T5

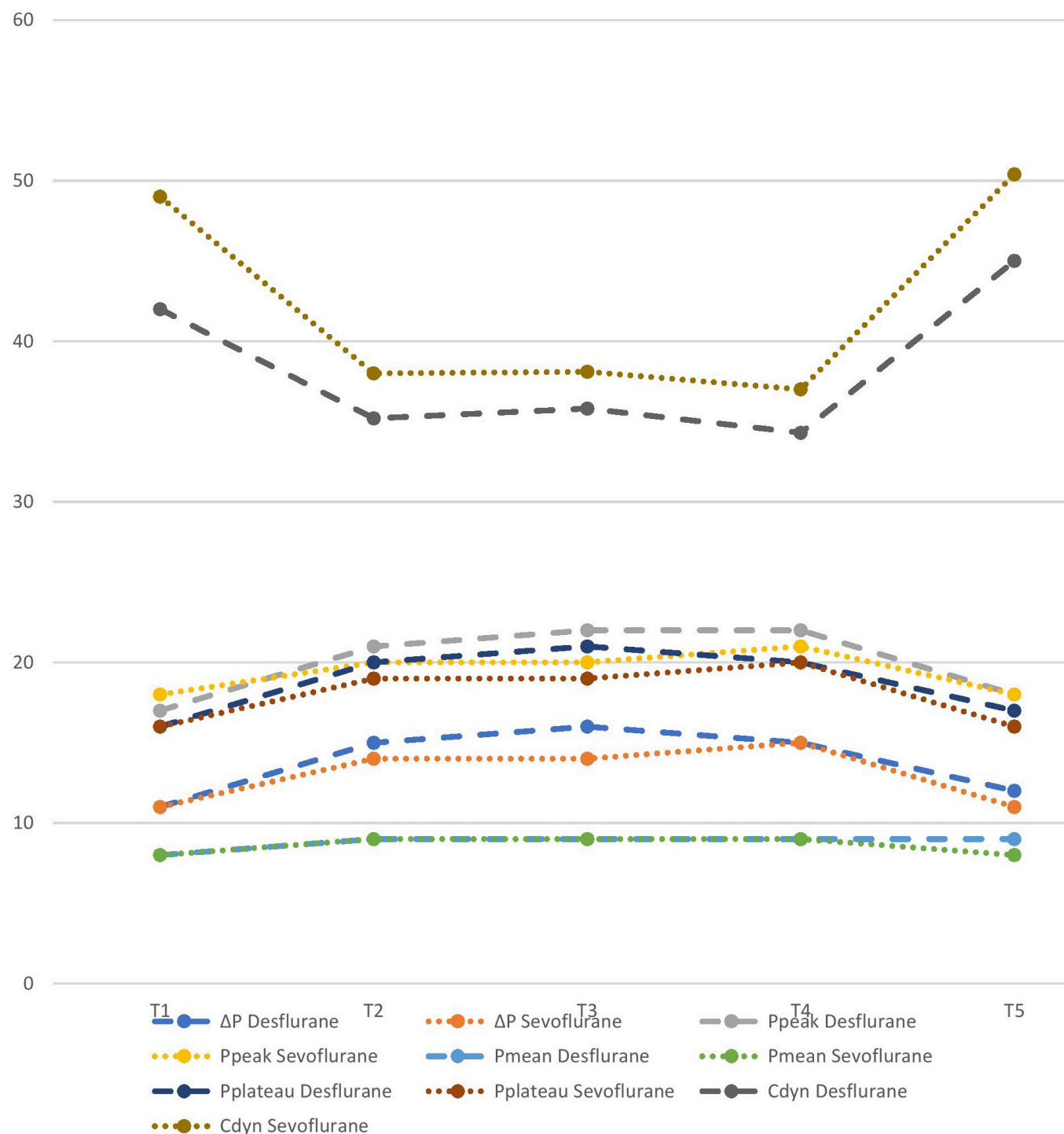


TABLE 2. Continued.

Group	T1 Median (25th–75th percentile)	T2 Median (25th–75th percentile)	T3 Median (25th–75th percentile)	T4 Median (25th–75th percentile)	T5 Median (25th–75th percentile)	Mann- Whitney $p^1$	Friedman $p$ (within- group) <sup>2</sup>	Significant <i>post hoc</i> comparisons <sup>3</sup>
Vt								
Desflurane	506.00 (471.00–539.50)	501.00 (480.00–542.50)	504.00 (466.00–548.00)	510.00 (475.00–562.00)	511.00 (477.50–542.50)	NS	0.236	–
Sevoflurane	509.00 (481.50–550.00)	500.00 (468.00–539.50)	500.00 (477.50–555.50)	507.00 (483.50–548.00)	500.00 (471.50–555.00)		0.538	–
Vd								
Desflurane	62.00 (43.50–77.00)	72.00 (51.00–81.50)	73.00 (48.00–82.50)	69.00 (43.00–96.50)	66.00 (52.50–74.50)	NS	0.470	–
Sevoflurane	62.00 (44.00–115.50)	91.00 (45.50–116.00)	78.00 (55.50–108.50)	91.00 (56.00–104.00)	76.80 (58.50–107.50)		0.816	–
Vd/Vt								
Desflurane	0.12 (0.09–0.17)	0.15 (0.09–0.17)	0.15 (0.11–0.16)	0.13 (0.08–0.16)	0.12 (0.10–0.15)	NS	0.376	–
Sevoflurane	0.14 (0.08–0.22)	0.15 (0.09–0.23)	0.15 (0.11–0.21)	0.16 (0.11–0.20)	0.15 (0.11–0.20)		0.787	–
MV								
Desflurane	6.50 (5.38–7.71)	6.42 (4.81–7.92)	6.48 (5.16–7.84)	6.49 (5.40–7.72)	6.42 (5.58–7.43)	NS	0.287	–
Sevoflurane	6.72 (5.40–8.00)	6.69 (5.23–9.22)	6.72 (5.30–8.96)	6.73 (5.11–8.58)	6.57 (5.25–8.03)		0.771	–

Median (25th–75th percentile) values are shown for C<sub>dyn</sub>, P<sub>peak</sub>, P<sub>mean</sub>, P<sub>plateau</sub>,  $\Delta P$ , V<sub>t</sub>, V<sub>d</sub>, V<sub>d</sub>/V<sub>t</sub>, and MV at T1–T5. Group comparisons: Mann-Whitney U test; within-group: Friedman test with post hoc Dunn's test (only significant comparisons reported). All other  $p$ -values are provided in **Supplementary Table 1**.

<sup>1</sup>Mann-Whitney U test: desflurane vs. sevoflurane; <sup>2</sup>Friedman test; <sup>3</sup>Post hoc Dunn's test; \* $p < 0.05$ . NS: not significant; C<sub>dyn</sub>: dynamic compliance (mL/cmH<sub>2</sub>O); P<sub>peak</sub>: peak airway pressure (cmH<sub>2</sub>O); P<sub>mean</sub>: mean airway pressure (cmH<sub>2</sub>O); P<sub>plateau</sub>: plateau pressure (cmH<sub>2</sub>O);  $\Delta P$ : driving pressure (P<sub>plateau</sub> – PEEP, cmH<sub>2</sub>O); V<sub>t</sub>: tidal volume (mL); V<sub>d</sub>: dead space volume (mL); V<sub>d</sub>/V<sub>t</sub>: dead space/tidal volume ratio; MV: minute ventilation (L/min).



**FIGURE 2. Intraoperative respiratory mechanics in prone position under desflurane and sevoflurane anaesthesia.** Comparison of dynamic compliance (Cdyn), peak airway pressure (Ppeak), mean airway pressure (Pmean), plateau pressure (Pplateau), and driving pressure ( $\Delta P$ ) at five time points (T1–T5) in patients receiving desflurane or sevoflurane.

TABLE 3. Intraoperative haemodynamic parameters in patients receiving desflurane or sevoflurane.

Group	T0 Median (25th–75th percentile)	T1 Median (25th–75th percentile)	T2 Median (25th–75th percentile)	T3 Median (25th–75th percentile)	T4 Median (25th–75th percentile)	T5 Median (25th–75th percentile)	Mann- Whitney <i>p</i> <sup>1</sup>	Friedman <i>p</i> (within- group) <sup>2</sup>	Significant <i>post hoc</i> comparisons <sup>3</sup>
HR									
Desflurane	71.0 (64.5–78.0)	75.0 (65.5–86.0)	63.0 (57.5–73.0)	64.0 (55.5–67.5)	62.0 (60.0–65.0)	64.0 (59.0–68.0)	T0: 0.022*; NS: (others)	0.001*	T0–T2, T0–T3, T0–T4, T0–T5, T1–T2, T1–T3, T1–T4, T1–T5
Sevoflurane	80.0 (73.0–87.0)	82.0 (75.0–87.0)	69.0 (64.0–77.0)	63.0 (56.5–66.0)	60.0 (56.5–69.5)	60.0 (56.5–65.0)		0.001*	T0–T2, T0–T3, T0–T4, T0–T5, T1–T2, T1–T3, T1–T4, T1–T5
SAP									
Desflurane	134.0 (127.0–146.5)	116.0 (107.0–133.0)	106.0 (91.5–117.5)	99.0 (86.0–103.5)	96.0 (87.0–103.0)	100.0 (90.0–118.0)	T0: 0.001*; T4: 0.020*; NS: (others)	0.001*	T0–T2, T0–T3, T0–T4, T0–T5, T1–T3, T1–T4
Sevoflurane	155.0 (149.0–167.0)	123.0 (105.5–143.0)	110.0 (100.5–127.0)	103.0 (94.5–108.0)	103.0 (97.0–107.5)	103.0 (96.5–113.0)		0.001*	T0–T1, T0–T2, T0–T3, T0–T4, T0–T5, T1–T3, T1–T4, T1–T5
MAP									
Desflurane	104.0 (101.5–110.0)	94.0 (78.0–105.0)	71.0 (63.5–93.5)	70.0 (63.5–80.5)	72.0 (63.5–82.5)	70.0 (66.0–88.0)	T0: 0.003*; NS: (others)	0.001*	T0–T2, T0–T3, T0–T4, T0–T5, T1–T2, T1–T3, T1–T4, T1–T5
Sevoflurane	117.0 (106.0–121.0)	92.0 (76.5–100.0)	86.0 (76.0–91.0)	75.0 (64.5–83.0)	76.0 (69.0–81.5)	77.0 (69.5–85.5)		0.001*	T0–T2, T0–T3, T0–T4, T0–T5, T1–T3, T1–T4, T1–T5
DAP									
Desflurane	83.0 (77.0–88.0)	75.0 (54.5–84.0)	67.0 (51.0–80.5)	60.0 (50.0–67.0)	58.0 (49.0–66.5)	56.0 (50.5–69.0)	NS	0.001*	T0–T2, T0–T3, T0–T4, T0–T5, T0–T1, T1–T3, T1–T4, T1–T5
Sevoflurane	89.0 (78.5–96.0)	70.0 (60.0–86.0)	68.0 (60.0–75.0)	60.0 (54.5–72.0)	60.0 (52.0–66.5)	57.0 (53.0–67.5)		0.001*	T0–T1, T0–T2, T0–T3, T0–T4, T0–T5, T1–T3, T1–T4, T1–T5

Median (25th–75th percentile) values are shown for HR, SAP, MAP, and DAP at T0–T5. Group comparisons: Mann-Whitney U test (only significant differences reported); within-group: Friedman test with post hoc Dunn's test (only significant comparisons reported). All other  $p$ -values are provided in **Supplementary Table 1**.

<sup>1</sup>Mann-Whitney U test: desflurane vs. sevoflurane; <sup>2</sup>Friedman test; <sup>3</sup>Post hoc Dunn's test; \* $p < 0.05$ . NS: not significant; HR: heart rate (beats/min); SAP: systolic arterial pressure (mmHg); MAP: mean arterial pressure (mmHg); DAP: diastolic arterial pressure (mmHg).



**TABLE 4. Comparison of oxygenation, carbon dioxide, and pH parameters between desflurane and sevoflurane groups.**

Group	T0 Median (25th–75th percentile)	T1 Median (25th–75th percentile)	T2 Median (25th–75th percentile)	T3 Median (25th–75th percentile)	T4 Median (25th–75th percentile)	T5 Median (25th–75th percentile)	Mann- Whitney <i>p</i> <sup>1</sup>	Friedman <i>p</i> (within- group) <sup>2</sup>	Significant <i>post hoc</i> comparisons <sup>3</sup>
PaO <sub>2</sub>									
Desflurane	88.200 (82.600– 94.400)	211.000 (150.500– 243.500)	210.000 (171.000– 235.000)	224.000 (193.000– 236.500)	213.000 (196.000– 230.100)	220.000 (181.500– 241.000)	NS	0.001*	T0–T1, T0–T2, T0–T3, T0–T4, T0–T5
Sevoflurane	91.300 (80.000– 95.000)	207.000 (175.000– 240.400)	229.000 (189.500– 242.000)	210.000 (191.000– 241.000)	210.000 (185.000– 245.000)	230.000 (211.600– 252.000)		0.001*	T0–T1, T0–T2, T0–T3, T0–T4, T0–T5, T1–T5
SaO <sub>2</sub>									
Desflurane	97.700 (97.000– 98.100)	99.400 (98.800– 99.500)	99.400 (98.800– 99.600)	99.400 (99.200– 99.700)	99.400 (99.200– 99.700)	99.300 (99.000– 99.700)	T4: 0.026*; NS: (others)	0.001*	T0–T1, T0–T2, T0–T3, T0–T4, T0–T5
Sevoflurane	98.000 (96.100– 98.500)	99.100 (98.300– 99.700)	99.200 (98.900– 99.500)	99.200 (98.900– 99.600)	99.200 (98.700– 99.500)	99.300 (98.800– 99.400)		0.001*	T0–T1, T0–T2, T0–T3, T0–T4, T0–T5
SpO <sub>2</sub>									
Desflurane	99.000 (98.000– 99.000)	99.000 (98.000– 99.000)	99.000 (98.500– 100.000)	99.000 (99.000– 100.000)	99.000 (99.000– 100.000)	99.000 (99.000– 100.000)	NS	0.010*	T0–T3, T0–T4, T0–T5
Sevoflurane	99.000 (98.000– 99.000)	99.000 (98.000– 100.000)	99.000 (98.000– 100.000)	100.000 (99.000– 100.000)	99.000 (99.000– 100.000)	99.000 (98.000– 100.000)		0.001*	T0–T3, T0–T4
P(A–a)O <sub>2</sub>									
Desflurane	–	96.000 (60.500– 153.500)	101.125 (72.800– 139.100)	86.500 (67.700– 115.600)	94.125 (80.100– 119.400)	89.000 (63.600– 126.700)	NS	0.078	–
Sevoflurane	–	97.625 (70.100– 130.700)	78.125 (68.700– 120.500)	96.625 (65.600– 113.900)	99.375 (62.200– 110.800)	73.500 (56.000– 99.800)		0.002*	T1–T5, T3–T5, T4–T5
pH									
Desflurane	7.420 (7.410–7.440)	7.410 (7.400–7.440)	7.420 (7.410–7.440)	7.420 (7.400–7.450)	7.400 (7.390–7.440)	7.390 (7.370–7.410)	NS	0.001*	T0–T5, T1–T5, T2–T5, T3–T5
Sevoflurane	7.420 (7.410–7.450)	7.420 (7.400–7.450)	7.420 (7.390–7.450)	7.420 (7.390–7.460)	7.400 (7.380–7.450)	7.390 (7.370–7.420)		0.001*	T0–T5, T1–T5, T2–T5, T3–T5, T4–T5

TABLE 4. Continued.

Group	T0 Median (25th–75th percentile)	T1 Median (25th–75th percentile)	T2 Median (25th–75th percentile)	T3 Median (25th–75th percentile)	T4 Median (25th–75th percentile)	T5 Median (25th–75th percentile)	Mann- Whitney <i>p</i> <sup>1</sup>	Friedman <i>p</i> (within- group) <sup>2</sup>	Significant <i>post hoc</i> comparisons <sup>3</sup>
PaCO <sub>2</sub>									
Desflurane	38.800 (36.900– 41.000)	39.500 (38.400– 40.300)	38.300 (36.700– 39.700)	38.100 (36.800– 39.200)	37.900 (35.800– 40.000)	38.900 (36.800– 40.900)	NS	0.010*	T1–T4
Sevoflurane	40.200 (36.200– 44.800)	39.300 (37.800– 45.600)	38.200 (36.800– 44.500)	38.200 (37.200– 42.500)	39.700 (37.600– 42.000)	39.600 (38.200– 45.400)		0.127	–
EtCO <sub>2</sub>									
Desflurane	–	34.000 (32.500– 36.500)	32.000 (32.000– 33.000)	32.000 (32.000– 33.000)	32.000 (32.000– 33.500)	34.000 (32.000– 35.000)	NS	0.001*	T1–T2, T1–T3, T1–T4, T2–T5, T3–T5, T4–T5
Sevoflurane	–	34.000 (32.000– 35.500)	32.000 (32.000– 34.500)	32.000 (32.000– 33.000)	33.000 (32.000– 34.000)	33.000 (32.000– 35.500)		0.002*	T1–T2, T1–T3
P(a–Et)CO <sub>2</sub>									
Desflurane	–	4.800 (3.600–5.800)	5.900 (3.600–6.900)	5.800 (4.000–6.200)	5.000 (3.000–7.100)	5.000 (4.000–5.900)	T5: 0.029*; NS: (others)	0.685	–
Sevoflurane	–	5.400 (3.300–10.300)	6.000 (3.500–10.400)	6.000 (4.300–9.000)	7.000 (4.300–8.300)	6.000 (4.500–9.100)		0.959	–

Median (25th–75th percentile) values are shown for EtCO<sub>2</sub>, PaCO<sub>2</sub>, P(a–Et)CO<sub>2</sub>, PaO<sub>2</sub>, SpO<sub>2</sub>, SaO<sub>2</sub>, pH, and P(A–a)O<sub>2</sub> at T1–T5. Group comparisons: Mann-Whitney U test; within-group: Friedman test with post hoc Dunn's test (only significant comparisons reported). All other *p*-values are provided in **Supplementary Table 1**.

<sup>1</sup>Mann-Whitney U test: desflurane vs. sevoflurane; <sup>2</sup>Friedman test; <sup>3</sup>Post hoc Dunn's test; \**p* < 0.05. NS: not significant; EtCO<sub>2</sub>: end-tidal carbon dioxide; PaCO<sub>2</sub>: arterial carbon dioxide; P(a–Et)CO<sub>2</sub>: arterial to EtCO<sub>2</sub> gradient; PaO<sub>2</sub>: arterial oxygen; SpO<sub>2</sub>: peripheral oxygen saturation; SaO<sub>2</sub>: arterial oxygen saturation; P(A–a)O<sub>2</sub>: alveolar–arterial oxygen gradient.

See **Supplementary Table 1** for full statistical results.

## 4. Discussion

In this study, we compared the effects of equipotent doses (1 MAC) of sevoflurane and desflurane on haemodynamics, respiratory mechanics, and oxygenation in patients undergoing spinal surgery in the prone position. Our findings demonstrated that both anaesthetic agents were generally safe and effective under the study conditions.

Inhalational anaesthetics have varying effects on airways and respiratory mechanics. Volatile agents may reduce lung resistance and elastance through smooth muscle relaxation and suppression of vagal reflexes, resulting in bronchodilation [17–19]. However, as reported by Sivaci *et al.* [20], desflurane in low-flow anaesthesia during laparoscopic surgery caused a significant decrease in C<sub>dyn</sub> compared with sevoflurane, which maintained stable compliance throughout the procedure, indicating a potential negative effect of desflurane on pulmonary mechanics under certain conditions.

Animal studies [17, 18] have also reported bronchodilatory effects of desflurane, although some *in vivo* studies suggested paradoxical increases in airway resistance at higher concentrations [2, 21, 22].

Clinically, sevoflurane, desflurane and isoflurane at 1–1.5 MAC reduce airway resistance and peak pressures while improving C<sub>dyn</sub> in most settings [19]. At higher concentrations, desflurane may induce irritant effects and increased resistance, whereas sevoflurane maintains bronchodilation [19, 23]. In our study, both agents at 1 MAC showed comparable airway pressures and C<sub>dyn</sub>, supporting similar effects on pulmonary mechanics under standardised conditions.

The prone position, commonly used in spinal surgery to optimise surgical access and patient safety, induces several physiological changes that affect respiratory function [24]. In clinical studies, prone positioning has been reported to improve lung compliance by promoting alveolar recruitment in the dorsal regions, which can enhance oxygenation, particularly in patients with severe respiratory failure, such as Acute Respiratory Distress Syndrome (ARDS) or under general anaesthesia [25, 26].

However, in respiratory physiology, transitioning a patient to the prone position may lead to increased airway pressures and decreased pulmonary and thoracic compliance, significantly affecting chest wall expansion and abdominal cavity compression [13, 14]. As a result, elevated intrathoracic and intra-abdominal pressures can restrict diaphragmatic and abdominal movements, leading to a reduction in lung C<sub>dyn</sub> and an increase in P<sub>peak</sub>, as demonstrated in several studies [13, 15, 27, 28]. In our study, at baseline in the supine position (T1), C<sub>dyn</sub> and airway pressures (P<sub>peak</sub>, P<sub>mean</sub>, P<sub>plateau</sub>) were comparable between groups, indicating similar pulmonary mechanics prior to positioning. Transitioning to the prone position (T2–T4) resulted in a significant reduction in C<sub>dyn</sub> and an increase in P<sub>peak</sub> in both groups, with values returning towards baseline at T5. P<sub>mean</sub> and P<sub>plateau</sub> showed similar trends. These findings are consistent with prior studies demonstrating that prone positioning can increase airway pressures and reduce lung compliance due to altered thoracic and abdominal mechanics. These results highlight the key role of positioning in modulating pulmonary mechanics, with

desflurane showing a slightly greater impact on airway pressures, although no clinically significant bronchoconstriction was observed at 1 MAC (Fig. 2; Table 2).

$\Delta P$  is a clinically relevant parameter that reflects lung stress by integrating T<sub>v</sub> and system compliance. According to recent meta-analyses,  $\Delta P$  is a stronger predictor of mortality than tidal volume or PEEP, and lower  $\Delta P$  levels have been associated with improved survival in patients with ARDS [29].

Further, intraoperative ventilation strategies based on  $\Delta P$  have been shown to improve gas exchange and reduce post-operative pulmonary complications [30].

In our study, although PEEP was constant, making  $\Delta P$  changes closely mirrored P<sub>plateau</sub>,  $\Delta P$  increased during prone positioning and it decreased after returning towards baseline, reflecting changes in lung stress during positioning, in line with its use as a predictor of mechanical stress in intraoperative ventilation (Table 2).

Animal [31] and human [32] studies have shown that desflurane increases HR through parasympathetic inhibition. Its sympathetic stimulatory effects have also been demonstrated in healthy young volunteers [9, 33, 34]. One study reported that inhalation of 1 MAC of desflurane after endotracheal intubation elevated HR [34], while another showed a transient rise in HR following a brief increase in desflurane concentration [9]. Desflurane has also been associated with vascular depression, significantly reducing MAP and Systemic Vascular Resistance [33]. Both desflurane and sevoflurane were shown to suppress baroreceptor activity at equivalent doses. However, Ebert *et al.* [35] found that sevoflurane did not suppress baroreceptor reflex activity more than desflurane. Similarly, Lee *et al.* [34] reported a greater HR increase with desflurane compared with sevoflurane in response to MAP reduction, indicating stronger baroreflex suppression.

In our study, baseline HR and arterial pressures were higher in the sevoflurane group, which may reflect interindividual variability rather than drug-specific effects. Over time, both anaesthetics led to a decline in HR and arterial pressures, consistent with their known cardiovascular depressant properties. Although transient fluctuations were observed, the overall trend was a reduction in both groups. However, the more pronounced decrease in SAP with sevoflurane, particularly after prone positioning, suggests that desflurane may provide slightly greater haemodynamic stability during prolonged surgery (Table 3). These findings align with previous reports indicating that sevoflurane exerts stronger negative inotropic and vasodilatory effects compared with desflurane, whereas desflurane's sympathetic activation may contribute to better preservation of blood pressure in the intraoperative setting [34, 35]. These findings are consistent with known reductions in venous return and cardiac index due to increased intrathoracic pressure in the prone position [10].

The reductions in HR and SAP may have decreased CO<sub>2</sub> production. Under conditions of a fixed I:E ratio and volume-controlled ventilation, this could prolong inspiratory time and potentially influence peak and plateau airway pressures. In line with our results, these haemodynamic changes did not lead to significant differences in PaCO<sub>2</sub>, EtCO<sub>2</sub>, or dead space ventilation indices (V<sub>d</sub>, V<sub>d</sub>/V<sub>t</sub>) between the two groups. These effects did not alter the overall trends observed between groups

(Tables 2,4).

Although prone positioning has been reported to improve oxygenation [2], in our cohort of ASA I–II patients without lung disease, no significant changes in oxygenation indices ( $\text{SpO}_2$ ,  $\text{PaO}_2$ ,  $\text{P(A-a)O}_2$ ) were observed. This suggests that ventilation–perfusion alterations induced by prone positioning did not significantly affect oxygenation in this patient population. Other studies, including Jo *et al.* [27] and Lee *et al.* [36], have similarly reported no significant changes. Our data also indicated no significant improvement in oxygenation following the position change. Given the increased  $\text{Ppeak}$  and decreased compliance observed, it appears that ventilation–perfusion alterations [2] do not significantly affect oxygenation, which aligns with the conclusions of Yucepur *et al.* [37]. Furthermore, no differences in oxygenation or compliance changes were found between the desflurane and sevoflurane groups. Although both  $\text{SpO}_2$  and arterial oxygen saturation were monitored to ensure patient safety [38], these parameters did not show clinically meaningful differences between groups (Table 4).

From a clinical standpoint, these findings indicate that both sevoflurane and desflurane may be used safely in ASA I–II patients without pulmonary disease undergoing spinal surgery in the prone position. However, sevoflurane may be preferable in patients with reduced pulmonary reserve, given its lower impact on airway pressures, whereas desflurane may provide more stable haemodynamic parameters during prolonged procedures. These observations highlight the importance of tailoring anaesthetic choice to individual patient characteristics and surgical context.

## 5. Conclusions

In conclusion, during spinal surgeries performed in the prone position, 1 MAC sevoflurane and desflurane exhibit comparable safety profiles in terms of haemodynamic stability, oxygenation, and respiratory mechanics. However, both desflurane and sevoflurane showed similar increases in  $\text{Ppeak}$ ,  $\text{Pmean}$ ,  $\text{Pplateau}$ , and  $\Delta\text{P}$  following prone positioning, which should be interpreted with caution in patients with underlying pulmonary disease. These observations emphasise that the prone positioning itself, rather than the anaesthetic agent alone, is a major determinant of changes in pulmonary mechanics, including the observed reductions in dynamic compliance, during spinal surgery. In our study, desflurane appeared to provide more stable haemodynamic parameters during prolonged surgical procedures. For patients without lung pathology, both agents can be used safely.

These findings may assist anaesthetists in selecting inhalational agents based on individual patient characteristics, particularly regarding respiratory function and expected surgical duration. Future studies may further explore the effects of these agents in patients with pre-existing pulmonary conditions or during extremely prolonged surgeries.

## 6. Limitations

The main limitation of this study was the absence of invasive monitoring, which may have reduced the precision of assessing

the respiratory and cardiovascular effects of the anaesthetic agents. Subtle physiological responses could, therefore, have been underestimated, and unmeasured perioperative factors might have influenced the outcomes, thereby reducing generalisability.

Although MAC values were not individually adjusted for age, all participants were between 18 and 65 years, where MAC variation is minimal. Continuous BIS and haemodynamic monitoring ensured adequate anaesthetic depth. Nevertheless, the lack of age-adjusted MAC values may limit applicability to elderly or paediatric patients.

This study focused solely on the intraoperative and early postoperative periods, preventing evaluation of late complications or long-term pulmonary function, which was not assessed via spirometry. Thus, our findings cannot address long-term outcomes or postoperative recovery patterns. Postoperative haemodynamic and respiratory parameters were also not analysed, confining conclusions to intraoperative findings. Despite strict inclusion criteria—non-smokers, ASA I–II, and BMI <35—other variables such as surgical duration and intraoperative fluid shifts may have affected respiratory and haemodynamic outcomes.

As a single-centre investigation, the results may not be generalisable to other institutions with differing practices, surgical techniques, or patient populations. Finally, desflurane’s environmental impact—related to its production, use and atmospheric release—may influence the long-term relevance and clinical acceptance of studies involving this agent.

## 7. Future directions

Future research may explore the effects of anaesthetic agents in elderly and paediatric populations and evaluate long-term postoperative outcomes. Studies including additional agents beyond desflurane and sevoflurane could offer a broader perspective. The use of advanced monitoring methods may help detect subtle physiological changes not captured in this study. Multicentre designs involving diverse patient populations and surgical procedures could also improve the generalisability of findings. Moreover, investigating the environmental impact of anaesthetic gases may contribute to more sustainable clinical practices.

## AVAILABILITY OF DATA AND MATERIALS

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

## AUTHOR CONTRIBUTIONS

YY, DM, EY and FYI—were responsible for design and conduct of study, data analysis and manuscript preparation; accept public responsibility for this study and its conclusions. YY and FYI—have revised it for important intellectual content and have approved the final version.

## ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was granted by the Hamidiye Clinical Research Ethics Committee on 23 November 2023 (approval no: 21-108), and written informed consent was obtained from all participants. This study was conducted in accordance with the principles of the Declaration of Helsinki.

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## CONFLICT OF INTEREST

The authors declare no conflict of interest.

## SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found, in the online version, at <https://oss.signavitae.com/mre-signavitae/article/2019693451451219968/attachment/Supplementary%20material.docx>.

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