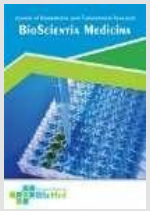
eISSN (Online): 2598-0580



Bioscientia Medicina: Journal of Biomedicine & Translational Research

Journal Homepage: www.bioscmed.com

Optimizing the Surgical Field via Multimodal Controlled Hypotension during Posterior Spinal Fixation for a T11 Burst Fracture: An Anesthetic Case Study

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ARTICLE INFO

Keywords:
Burst fracture
Controlled hypotension
Dexmedetomidine
Spine surgery
Thoracic vertebrae

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All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/bsm.v9i12.1455

A B S T R A C T

Background: Significant intraoperative blood loss is a major challenge in complex spinal surgeries, impairing surgical field visibility and increasing patient morbidity. Controlled hypotension is an established anesthetic technique to mitigate this challenge, yet the optimal combination of agents to ensure efficacy and safety remains an area of active investigation. This case study details the successful application of a multimodal anesthetic regimen, centered on the synergistic effects of dexmedetomidine and isoflurane, to achieve deliberate hypotension during posterior stabilization of a thoracic burst fracture. **Case presentation:** A 39-year-old male, classified as American Society of Anesthesiologists (ASA) physical status II, presented with a traumatic, unstable burst fracture of the eleventh thoracic vertebra (T11) following a high-energy fall. He was scheduled for a posterior decompressive laminectomy and T10-T12 pedicle screw fixation. Anesthetic management was initiated with a multimodal approach utilizing intravenous infusions of dexmedetomidine and morphine, supplemented by maintenance with isoflurane. This strategy was employed to maintain a target mean arterial pressure (MAP) of 60-65 mmHg. Throughout the 135-minute procedure, the patient's hemodynamics remained exceptionally stable within the target range. The estimated blood loss was minimal (approximately 350 mL), providing the surgical team with an excellent, clear operative field. The patient emerged smoothly from anesthesia with no neurological deficits and experienced a favorable postoperative recovery. **Conclusion:** This case demonstrates that a multimodal anesthetic strategy incorporating dexmedetomidine, a volatile agent, and opioid infusions is a highly effective and safe method for inducing and maintaining controlled hypotension in major spinal surgery. This approach successfully optimized the surgical conditions by minimizing blood loss and enhancing visibility, without compromising hemodynamic stability or vital organ perfusion, thereby contributing to a positive patient outcome.

1. Introduction

Traumatic injuries to the thoracolumbar spine, particularly burst fractures, represent a significant cause of morbidity and potential long-term disability, primarily affecting young, active individuals.[1] These high-energy injuries, often resulting from falls from height or motor vehicle collisions, are characterized by the failure of both the anterior and middle vertebral columns under axial compression. This mechanism frequently leads to retropulsion of bony fragments into the spinal canal, causing mechanical instability, progressive kyphotic deformity, and a high risk of catastrophic neurological injury to the spinal cord. The thoracolumbar injury classification and severity

score (TLICS), a widely adopted system, aids in standardizing treatment decisions.[2] A score greater than 4, often seen in burst fractures with neurological involvement or significant posterior ligamentous complex disruption, typically signifies instability necessitating surgical intervention.

The primary objectives of surgical management are threefold: decompression of the neural elements, restoration of spinal alignment, and achievement of immediate and long-term stability.[3] Posterior decompressive laminectomy combined with pedicle screw fixation has become the gold standard approach for many of these injuries. This technique provides robust, multi-segmental stabilization, allowing for the correction of deformity and creating an environment conducive to bony fusion. However, these procedures are anatomically complex and associated with substantial physiological challenges. The vertebral column is a highly vascular structure, surrounded by the rich epidural venous plexus and robust paraspinal musculature.[4] Surgical dissection in this region, especially in the context of acute traumatic inflammation, inevitably leads to significant intraoperative blood loss.

Excessive surgical bleeding poses a multifaceted problem. It obscures the surgeon's view of critical neural and bony landmarks, prolonging operative time and increasing the risk of iatrogenic injury. Furthermore, substantial hemorrhage can lead to hemodynamic instability, necessitate allogeneic blood transfusions with their attendant risks, such as transfusion reactions, immunomodulation, and the transmission of infectious diseases, and may contribute to postoperative anemia, coagulopathy, and delayed recovery.[5] The prone position, mandatory for posterior spinal approaches, can exacerbate this issue by increasing intra-abdominal pressure, which in turn impedes venous return and engorges the epidural venous plexus, further promoting surgical site bleeding.[6]

To counter these challenges, anesthesiologists have developed the technique of controlled (or deliberate) hypotension. First introduced in the 1940s, this strategy involves the intentional reduction of systemic blood pressure to a predefined target, thereby decreasing blood flow to the surgical field. The underlying principle is that by lowering the perfusion pressure within the incised vessels, the rate of hemorrhage is reduced, leading to a cleaner operative field, reduced need for transfusion, and potentially shorter surgical duration.[7] While the benefits are clear, the technique requires a sophisticated understanding of cardiovascular physiology and pharmacology to ensure that the reduction in systemic pressure does not compromise perfusion to vital organs, particularly the brain, heart, and kidneys.

Historically, controlled hypotension was achieved using high concentrations of potent volatile anesthetics or direct-acting vasodilators such as sodium nitroprusside or nitroglycerin. While effective, these methods can be associated with challenges like reflex tachycardia, rebound hypertension, and concerns over cyanide toxicity (with nitroprusside). Modern anesthetic practice has evolved towards a more nuanced, balanced, and multimodal approach. This involves the synergistic use of multiple agents acting via different physiological pathways to achieve the desired hemodynamic effect with lower doses of each individual drug, thereby minimizing side effects and enhancing stability.[8]

Central to this modern approach is the use of agents like dexmedetomidine, a highly selective alpha-2 adrenergic agonist. Through its action in the locus coeruleus and spinal cord, dexmedetomidine produces a centrally mediated sympatholysis, resulting in sedation, analgesia, and a decrease in both heart rate and blood pressure, without significant respiratory depression.[9] Its unique properties make it an attractive component of a hypotensive anesthetic, as it can blunt the tachycardic response often seen with other vasodilating agents and reduce the overall requirement for volatile anesthetics and opioids. When combined with a background of a volatile agent like isoflurane (which reduces blood pressure primarily through vasodilation and decreased systemic vascular resistance) and a

potent opioid (for blunting surgical stress response), a stable and easily titratable state of hypotension can be achieved.[10]

This case study presents a detailed account of the anesthetic management of a patient undergoing posterior spinal decompression and fixation for a traumatic T11 burst fracture. The primary aim of this report is to describe and analyze a specific multimodal protocol for controlled hypotension, centered on the co-administration of dexmedetomidine, isoflurane, and morphine. The novelty of this report lies in its detailed, step-by-step illustration of how this combination was used to effectively optimize the surgical field—as evidenced by minimal blood loss and surgeon satisfaction—while maintaining exquisite hemodynamic control and ensuring patient safety, thus providing a practical template for a sophisticated anesthetic technique in a high-risk surgical setting.

2. Case Presentation

A 39-year-old male (weight 65 kg, height 160 cm, BMI 25.3 kg/m²) was admitted to the emergency department of Dr. Moewardi Regional General Hospital following a fall from a height of approximately 6 meters at a construction site. He landed on his back and immediately experienced severe, non-radiating back pain, localized to the thoracolumbar junction. The patient did not lose consciousness and denied any associated headache, nausea, or vomiting. He reported no new-onset weakness, numbness, or tingling in his extremities and had no bowel or bladder incontinence. His past medical history was unremarkable for chronic illnesses such as hypertension, diabetes mellitus, asthma, or cardiac disease. He had no prior surgical history, no known drug allergies, and was not taking any regular medications. He reported smoking approximately 10 cigarettes per day for 15 years but denied regular alcohol or illicit drug use (Table 1).

On physical examination in the emergency department, his vital signs were stable: blood pressure of 126/82 mmHg, heart rate of 80 beats per minute, respiratory rate of 20 breaths per minute, and SpO_2 of 99% on room air. The cardiopulmonary examination was normal. The abdominal examination was benign. The spinal examination revealed significant tenderness to palpation over the T11-T12 spinous processes, with associated paraspinal muscle spasm. There was a visible contusion in the area, but no step-off deformity. A comprehensive neurological examination was performed according to the American Spinal Injury Association (ASIA) standards. Motor examination revealed 5/5 strength in all myotomes of the upper and lower extremities. Sensory examination was intact to light touch and pinprick in all dermatomes. Deep tendon reflexes were 2+ and symmetric, and the Babinski sign was negative bilaterally. His ASIA impairment scale was graded as E (normal motor and sensory function). A thoracolumbar multi-slice computed tomography (MSCT) scan revealed an acute, severe burst fracture of the T11 vertebral body. Key findings included a loss of anterior vertebral body height of approximately 60%, retropulsion of a large bony fragment from the posterior wall of the vertebral body into the spinal canal, resulting in approximately 55% canal compromise at that level. The kyphotic angle at the fracture site measured 28°. The posterior ligamentous complex appeared disrupted on imaging. Fractures of the spinous processes of C4 and C5 and non-displaced fractures of the right 8th and 9th ribs were also noted. Magnetic Resonance Imaging (MRI) of the thoracolumbar spine was performed to evaluate the soft tissues and neural elements. It confirmed the severe T11 burst fracture and the significant canal stenosis due to the retropulsed fragment. T2-weighted images showed high signal intensity within the posterior ligamentous complex, confirming ligamentous disruption. There was no evidence of spinal cord edema, contusion, or hemorrhage at the level of compression.

Table 1. Summary of Anamnesis and Clinical Findings on Admission

PATIENT ASSESSMENT SUMMARY	
Demographics	39-Year-Old Male | 65 kg | 160 cm | BMI: 25.3 kg/m²
Present Illness	**Chief Complaint:** Severe, non-radiating back pain. **Mechanism of Injury:** Fall from a height of approximately 6 meters. **Associated Symptoms:** Denied loss of consciousness, nausea, vomiting, or neurological deficits.
Medical & Social History	**Past Medical History:** Unremarkable. No chronic illnesses. **Past Surgical History:** None. **Allergies:** No known drug allergies. **Social History:** Smoker (10 cigarettes/day for 15 years). Denied regular alcohol or illicit drug use.
Vital Signs & General Exam	**Blood Pressure:** 126/82 mmHg **Heart Rate:** 80 bpm (Normal Sinus Rhythm) **Respiratory Rate:** 20 breaths/min **SpO_2:** 99% on Room Air **General:** Awake, alert, no acute distress. Cardiopulmonary and abdominal exams were normal.
Spinal Examination	**Palpation:** Severe tenderness over T11-T12 spinous processes. **Inspection:** Visible contusion over thoracolumbar area; no step-off deformity. **Musculature:** Associated paraspinal muscle spasm noted.
Neurological Examination	**Motor Function:** 5/5 strength in all myotomes (Upper & Lower Extremities). **Sensory Function:** Intact to light touch and pinprick in all dermatomes. **Reflexes:** 2+ and symmetric. Babinski sign negative bilaterally. **ASIA Impairment Scale:** Grade E (Normal motor and sensory function).
Key Diagnostic Findings	**Diagnosis:** Unstable Burst Fracture of T11 Vertebra. **CT Scan Details:** 60% loss of anterior vertebral height, 55% spinal canal compromise, 28° kyphotic angle. **MRI Details:** Confirmed canal stenosis and posterior ligamentous complex disruption. No spinal cord edema.
Preoperative Assessment	**ASA Physical Status:** ASA II (due to smoking history and trauma). **TLICS Score:** Calculated > 4, indicating surgical intervention is required. **Planned Procedure:** T11 Decompressive Laminectomy and T10-T12 Posterior Spinal Fixation.

Based on these findings—fracture morphology (burst), posterior ligamentous complex injury, and neurological status (intact but at high risk)—the patient's TLICS score was calculated to be greater than 4, indicating a mechanically unstable fracture pattern warranting surgical fixation. The patient was diagnosed with an unstable T11 burst fracture and scheduled for T11 decompressive laminectomy and T10-T12 posterior spinal fixation.

Table 2. Preoperative Laboratory and Ancillary Tests

TEST CATEGORY	TEST NAME	RESULT	STATUS / NORMAL RANGE
Hematology	Hemoglobin	14.5 g/dL	Normal (13.5-17.5 g/dL)
	Hematocrit	43 %	Normal (41-50 %)
	Platelet Count	250,000 /µL	Normal (150-450 x10³/µL)
	White Blood Cell Count	8,500 /µL	Normal (4.5-11.0 x10³/µL)
Coagulation Profile	Prothrombin Time (PT)	12.5 seconds	Normal (11-13.5 sec)
	International Normalized Ratio (INR)	1.0	Normal (0.8-1.1)
	Activated Partial Thromboplastin Time (aPTT)	30 seconds	Normal (25-35 sec)
Serum Chemistry	Sodium (Na^+)	140 mEq/L	Normal (136-145 mEq/L)
	Potassium (K^+)	4.1 mEq/L	Normal (3.5-5.1 mEq/L)
	Creatinine	0.9 mg/dL	Normal (0.74-1.35 mg/dL)
	Blood Urea Nitrogen (BUN)	15 mg/dL	Normal (7-20 mg/dL)
Ancillary Tests	Electrocardiogram (ECG)	Normal Sinus Rhythm	No Ischemic Changes
	Chest X-ray (PA/Lat)	No Acute Infiltrates	Clear Lungs

Overall Assessment: *All laboratory values are within normal limits, indicating no underlying hematologic, coagulopathic, or metabolic derangements that would contraindicate major surgery. Ancillary tests confirm no acute cardiopulmonary comorbidities.*

A comprehensive preoperative evaluation was conducted to ensure the patient's fitness for major spinal surgery and to identify any potential risks (Table 2). Laboratory investigations confirmed the patient's physiological stability. Hematologic parameters were reassuring, with a normal hemoglobin of 14.5 g/dL and a hematocrit of 43%, precluding preoperative anemia that would lower the threshold for transfusion. The platelet count of 250,000/µL and a white blood cell count of 8,500/µL were also well within normal limits. Crucially, the coagulation profile, including a Prothrombin Time (PT) of 12.5 seconds, an International Normalized Ratio (INR) of 1.0, and an Activated Partial Thromboplastin Time (aPTT) of 30 seconds, was entirely normal, indicating no intrinsic coagulopathy and a low risk of excessive surgical bleeding. The serum chemistry panel revealed balanced electrolytes, with a sodium of

140 mEq/L and potassium of 4.1 mEq/L, and confirmed normal renal function, evidenced by a creatinine of 0.9 mg/dL and a Blood Urea Nitrogen (BUN) of 15 mg/dL.

Ancillary testing further affirmed the patient's low-risk profile. A 12-lead electrocardiogram (ECG) demonstrated a normal sinus rhythm at a rate of 78 beats per minute, with no evidence of underlying ischemia, arrhythmia, or conduction abnormalities. A chest X-ray confirmed the absence of any acute cardiopulmonary processes like pneumothorax, hemothorax, or pulmonary contusion, showing only the previously noted healing rib fractures. Based on this comprehensive assessment, which revealed a healthy individual with the exception of his traumatic injuries and smoking history, the patient was classified under the American Society of Anesthesiologists (ASA) physical status II. Following a detailed discussion of the proposed surgical and anesthetic plans, including the risks and benefits, the patient provided full informed consent to proceed.

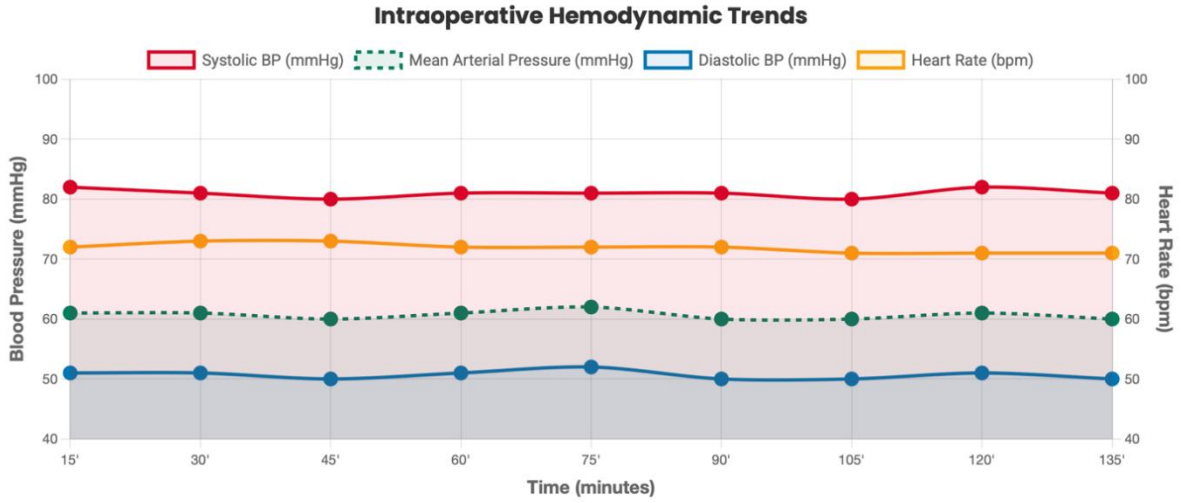
The primary anesthetic goals were to ensure hemodynamic stability, facilitate a smooth induction and emergence, provide adequate analgesia and amnesia, and, critically, to implement controlled hypotension to minimize intraoperative blood loss and optimize surgical conditions. Upon arrival in the operating room, standard ASA monitors were applied (ECG, non-invasive blood pressure cuff, pulse oximeter, and temperature probe). Two large-bore (16-gauge) intravenous catheters were secured. After local anesthesia with 1% lidocaine, a 20-gauge arterial catheter was placed in the left radial artery for continuous, beat-to-beat blood pressure monitoring. A triple-lumen central venous catheter was placed in the right internal jugular vein for central venous pressure monitoring and as a reliable route for vasoactive infusions if needed. A Foley catheter was inserted to monitor urine output. Neuromonitoring with somatosensory evoked potentials (SSEP) and motor evoked potentials (MEP) was planned. The patient was preoxygenated with 100% oxygen for 5 minutes. Premedication was administered intravenously with Midazolam 2 mg and Fentanyl 100 µg. Anesthesia was induced with Propofol 70 mg. After confirming loss of consciousness, neuromuscular blockade was achieved with Rocuronium 50 mg. The patient was intubated uneventfully on the first attempt with a size 8.0 cuffed endotracheal tube, which was secured at 22 cm at the teeth. Bilateral chest auscultation confirmed correct placement. Following induction, the patient was carefully turned from the supine to the prone position onto a Jackson spinal table. Careful attention was paid to ensure all pressure points were padded, the abdomen was hanging free to minimize intra-abdominal pressure, and the head and neck were maintained in a neutral position. The integrity and positioning of the endotracheal tube and all invasive lines were reconfirmed.

Anesthesia was maintained with Isoflurane in an air/oxygen mixture (FiO_2 0.5), with the end-tidal concentration adjusted to 0.8-1.2% (approximately 0.7-1.0 MAC) to contribute to the desired level of hypotension. Mechanical ventilation was initiated using a pressure-controlled ventilation-volume guaranteed (PCV-VG) mode, with a tidal volume of 8 mL/kg, a respiratory rate of 12 breaths per minute to maintain an end-tidal CO_2 ($ETCO_2$) of 35-40 mmHg, and a positive end-expiratory pressure (PEEP) of 5 cmH_2O. The multimodal controlled hypotension regimen was initiated as follows: (1) Dexmedetomidine: A loading dose of 1 µg/kg was administered over 10 minutes, followed by a continuous infusion at a rate of 0.4 µg/kg/hour; (2) Morphine: A continuous infusion was started at 10 µg/kg/hour for baseline analgesia; (3) Isoflurane: The concentration was titrated to achieve the target MAP. The target MAP was set to 60-65 mmHg. This target was chosen to be low enough to significantly reduce surgical bleeding but high enough to remain above the lower limit of autoregulation for vital organs in a healthy young adult. The combination of central sympatholysis from dexmedetomidine and peripheral vasodilation from isoflurane allowed for a rapid and stable achievement of this target without causing significant reflex tachycardia; the heart rate remained

between 60-70 beats per minute throughout the hypotensive period.

The total duration of the surgery was 135 minutes. Hemodynamic parameters were recorded every 5 minutes and remained remarkably stable (Figure 1). The MAP was consistently maintained within the 60-65 mmHg range. Urine output was maintained above 0.5 mL/kg/hour throughout the procedure. The patient's core temperature was kept at 36.5°C using a forced-air warming blanket. The surgeon reported an excellent, dry surgical field with minimal diffuse bleeding, allowing for clear identification of anatomical planes and precise placement of the T10, T11, and T12 pedicle screws. The total estimated blood loss (EBL) for the entire procedure was 350 mL. Total intravenous fluids administered were 1500 mL of lactated Ringer's solution. No blood products were required. Neuromonitoring signals (SSEP/MEP) remained stable at baseline throughout the surgery.

Intraoperative Hemodynamic & Physiological Data



Supplementary Physiological Data

PARAMETER	15'	30'	45'	60'	75'	90'	105'	120'	135'
SpO_2 (%)	99	99	99	99	99	99	99	99	99
$ETCO_2$ (mmHg)	38	37	38	36	37	38	37	39	38

Figure 1. Intraoperative hemodynamic trends and physiological data.

At the conclusion of the surgical procedure, the dexmedetomidine and morphine infusions were discontinued. The isoflurane concentration was reduced, and 100% oxygen was administered. Reversal of neuromuscular blockade was achieved with Sugammadex 200 mg. The patient began breathing spontaneously, and upon meeting standard extubation criteria (awake, following commands, adequate tidal volume, and respiratory rate), the trachea was extubated smoothly.

The patient was transferred to the Post-Anesthesia Care Unit (PACU) for close monitoring. His initial vital

signs in the PACU were: blood pressure 110/70 mmHg, heart rate 75 bpm, respiratory rate 16 breaths per minute, and SpO_2 100% on 2L of oxygen via nasal cannula. He was awake, alert, and oriented. A postoperative neurological examination confirmed his baseline ASIA E status with no new deficits. His pain was well-controlled, with a reported score of 3/10 on the Numeric Rating Scale (NRS). Postoperative analgesia was managed with patient-controlled analgesia (PCA) using morphine, supplemented with intravenous paracetamol 1g every 6 hours. Ondansetron 4 mg was administered for nausea prophylaxis. The patient was transferred to the surgical ward after 2 hours in the PACU. His postoperative course was uneventful. He was mobilized with a spinal brace on postoperative day 1 and discharged home on postoperative day 5 with a comprehensive rehabilitation plan.

3. Discussion

This case study successfully demonstrates the clinical utility and safety of a multimodal anesthetic strategy for inducing controlled hypotension in a patient undergoing complex posterior spinal surgery. The primary finding was the achievement of a stable, targeted level of hypotension that resulted in a significantly optimized surgical field, as evidenced by minimal intraoperative blood loss (350 mL) for a procedure of this magnitude.[11] This outcome underscores the profound impact that careful anesthetic management can have on surgical conditions and overall patient safety. The discussion will focus on the underlying pathophysiological and pharmacological principles that contributed to this success.

Posterior spinal surgery is inherently associated with a high risk of bleeding due to the unique vascular anatomy of the vertebral column.[12] The procedure requires extensive dissection of the highly vascular paraspinal muscles and periosteum. More significantly, it involves exposure of the vertebral elements and potential entry into the spinal canal, which contains Batson's plexus—a large, valveless network of epidural veins. This plexus is particularly susceptible to engorgement. In the prone position, any compression of the abdomen can increase intra-abdominal pressure, which is transmitted directly to the venous system via the inferior vena cava, leading to significant and persistent venous oozing that can be difficult to control surgically. Arterial bleeding occurs from segmental arteries and nutrient vessels supplying the bone and surrounding tissues.[13]

The rate of surgical bleeding is governed by fundamental principles of fluid dynamics, primarily described by Poiseuille's law, which states that flow is directly proportional to the pressure gradient across the vessel.[14] By intentionally reducing the systemic mean arterial pressure, the driving pressure for arterial hemorrhage is lowered. Similarly, by reducing central venous congestion through careful positioning (freeing the abdomen) and lowering systemic pressure, the pressure within the epidural plexus is decreased, reducing venous hemorrhage. The goal of controlled hypotension is to lower this perfusion pressure into a range where bleeding is minimized but tissue oxygen delivery to critical organs remains adequate.[15] This requires a delicate balance, as excessive hypotension can breach the lower limits of organ autoregulation, leading to ischemic injury.

The success of the technique described in this case lies in the synergistic interaction of the chosen anesthetic agents, which allowed for balanced and stable control of the patient's hemodynamics.[16] As the cornerstone of our intravenous regimen, dexmedetomidine played a pivotal role. Its primary mechanism of action is the agonism of α_2-adrenergic receptors in the central nervous system. In the locus coeruleus, this activation leads to an inhibition of norepinephrine release, producing sedation and anxiolysis. In the vasomotor center of the medulla, it reduces sympathetic outflow to the periphery, resulting in decreased heart rate (chronotropy) and reduced systemic vascular resistance (SVR), leading to a dose-dependent decrease in blood pressure.

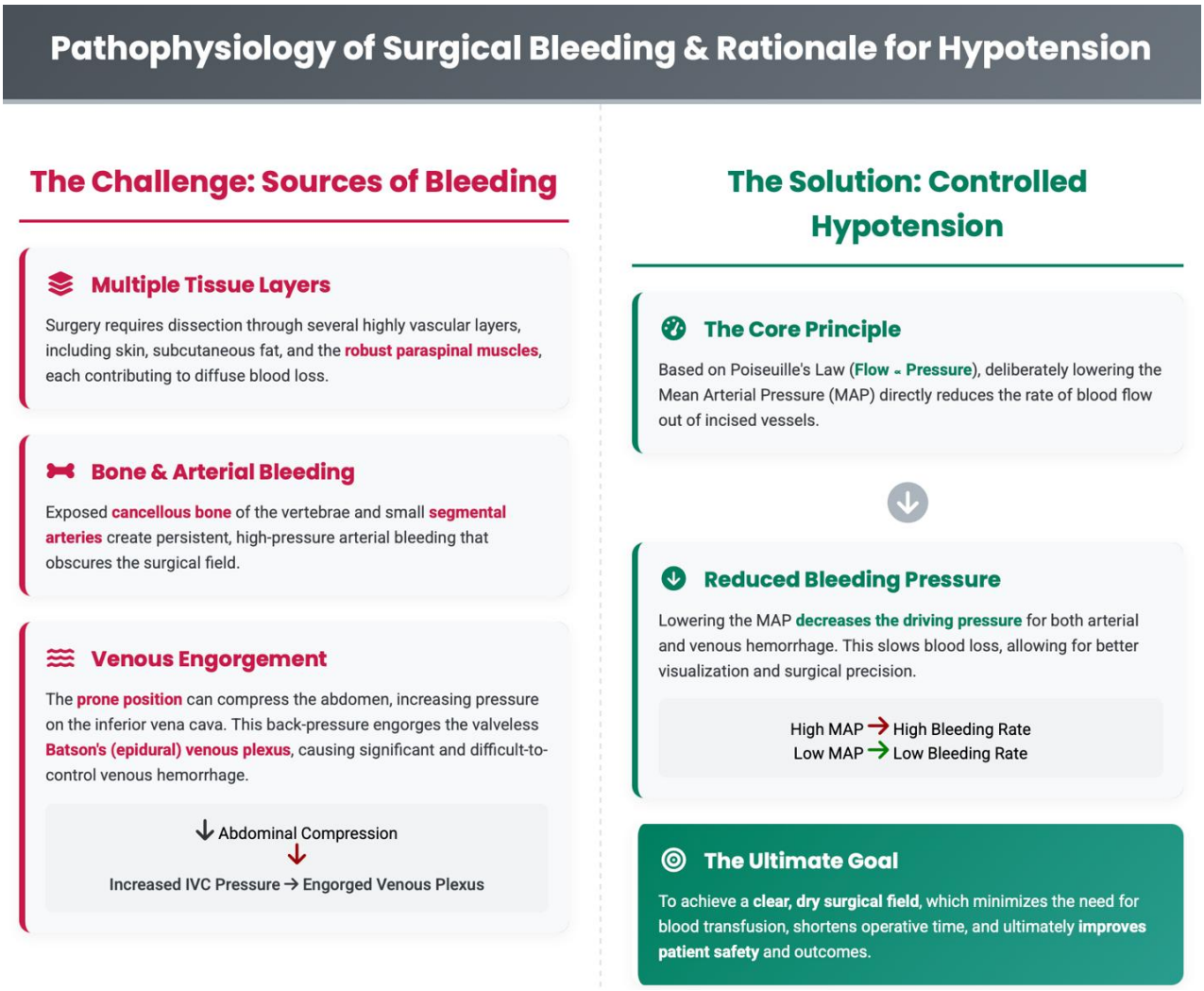


Figure 2. Pathophysiology of bleeding in spine surgery and the rationale for hypotension.

This central sympatholysis is particularly advantageous for controlled hypotension. Unlike direct vasodilators that can provoke a compensatory reflex tachycardia, dexmedetomidine intrinsically produces bradycardia, contributing to a stable heart rate and reduced myocardial oxygen consumption.[17] Furthermore, its analgesic properties reduce the requirement for opioids, and its anesthetic-sparing effect allows for lower concentrations of volatile agents, further enhancing hemodynamic stability. In our patient, the dexmedetomidine infusion provided a stable baseline of sympatholysis, preventing sharp hemodynamic responses to surgical stimulation.

Volatile halogenated anesthetics, including isoflurane, induce hypotension primarily by causing direct relaxation of vascular smooth muscle, leading to a reduction in SVR.[18] They also cause a dose-dependent depression of myocardial contractility, although this is less pronounced with isoflurane compared to older agents. By combining a low concentration of isoflurane with the dexmedetomidine infusion, we were able to leverage its potent vasodilatory effects while minimizing its negative inotropic effects. This combination allows for rapid, breath-to-breath adjustments in the depth of anesthesia and level of hypotension, providing a fine-tuning capability that is difficult to achieve with

intravenous agents alone. The volatile agent ensures amnesia and immobility, while the dexmedetomidine provides hemodynamic stability and analgesia.

Opioids are essential for blunting the profound sympathetic and neuroendocrine stress response to intense surgical stimuli, such as periosteal stripping and pedicle screw insertion.[19] This stimulation, if unblocked, would cause a surge in catecholamines, leading to hypertension and tachycardia that would counteract the intended hypotensive state. The initial bolus of fentanyl during induction and the subsequent morphine infusion provided a continuous level of potent analgesia, preventing such breakthroughs and contributing to the overall smoothness of the anesthetic course. The synergy of these three classes of drugs created an ideal state: central sympatholysis and bradycardia from dexmedetomidine, peripheral vasodilation from isoflurane, and potent analgesia from the opioid. This allowed the MAP to be gently guided into the target range and held there with minimal fluctuation, a hallmark of a well-controlled and balanced anesthetic.

The paramount concern with any hypotensive technique is the risk of iatrogenic ischemia to vital organs.[20] The brain, heart, and kidneys possess autoregulatory mechanisms that maintain constant blood flow across a wide range of systemic blood pressures. For a normotensive individual, the lower limit of cerebral autoregulation is typically a MAP of 50-60 mmHg. Our target MAP of 60-65 mmHg was deliberately chosen to respect this physiological boundary. In a young patient with no history of chronic hypertension (which can shift the autoregulatory curve to the right), this target provides a buffer of safety. Continuous monitoring of surrogate markers of organ perfusion is non-negotiable. In this case, stable neuromonitoring signals (SSEP/MEP) provided real-time assurance of adequate spinal cord perfusion. Consistent urine output (>0.5 mL/kg/hr) indicated sufficient renal perfusion, and the absence of ST-segment changes on the ECG suggested adequate myocardial perfusion. The stability of the heart rate, without tachycardia, further indicated that the myocardial oxygen supply-demand ratio was well-balanced. This comprehensive monitoring is essential to confirm that the systemic hypotension is well-tolerated at the tissue level.

4. Conclusion

This case study provides a detailed illustration of a modern, multimodal approach to controlled hypotension for complex spine surgery. The judicious and synergistic combination of a central sympatholytic agent (dexmedetomidine), a volatile anesthetic (isoflurane), and an opioid infusion proved to be a remarkably safe and effective strategy. It allowed for the precise maintenance of a target hypotensive state, which translated directly into an optimized, near-bloodless surgical field. This optimization facilitated the surgical procedure, minimized iatrogenic risks, and obviated the need for blood transfusion. The stable intraoperative hemodynamics and favorable postoperative outcome reinforce the concept that a well-planned, physiologically-sound anesthetic technique is not merely an adjunct to surgery, but a critical determinant of its success.

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