The Use of Preoperative Stellate Ganglion Block for the Prevention of Postoperative Neurocognitive Disorders: A Possible Paradigm Shift



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INTRODUCTION

Postoperative neurocognitive disorders (PND), including postoperative delirium (POD) and postoperative cognitive dysfunction (POCD), are common complications following major surgery and are associated with prolonged hospitalization, functional decline, and long-term cognitive impairment. Surgical stress may contribute to PND through sympathetic and immune overactivation, leading to neuroinflammation, oxidative stress, endothelial dysfunction, and neuronal injury. Stellate ganglion block (SGB) has emerged as a potential preoperative intervention capable of attenuating these pathways through modulation of sympathetic activity.

Objective: To evaluate the biologic plausibility and existing evidence supporting preoperative SGB as a potential strategy for preventing postoperative neurocognitive disorders.

METHODS

A focused narrative review of recent translational and clinical literature was conducted using PubMed, Google Scholar, and OpenEvidence. Mechanistic investigations, randomized controlled trials, observational studies, and experimental models evaluating perioperative SGB were reviewed.

Primary outcomes included postoperative cognitive performance (MMSE and MoCA), incidence of POD and POCD, inflammatory biomarkers (IL-6), oxidative stress markers (MDA and SOD), endothelial function, cerebral perfusion, and perioperative hemodynamic stability. Findings were synthesized to evaluate the potential neuroprotective effects of sympathetic modulation with SGB.

RESULTS

Across the reviewed studies, preoperative SGB was associated with improvements in several outcomes and pathways implicated in postoperative neurocognitive dysfunction.

DISCUSSION

Current evidence supports a potential neuroprotective role for preoperative SGB through modulation of the sympathetic-inflammatory response to surgery. Across clinical and translational studies, SGB was associated with reduced inflammatory signaling and oxidative stress, improved cerebral perfusion and endothelial function, greater hemodynamic stability, and improved postoperative cognitive outcomes.

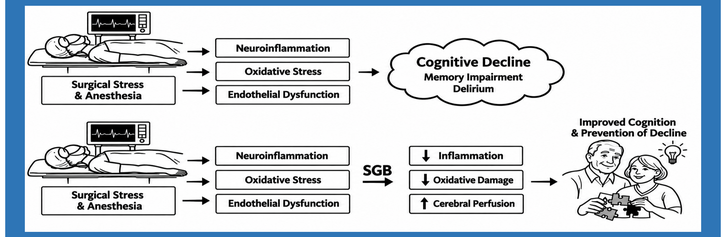


Figure 1. Mechanistic Effects of SGB on Postoperative Neurocognitive Disorders

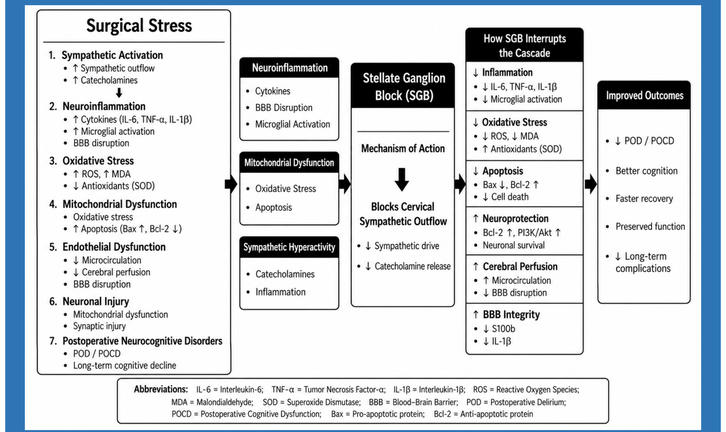


Figure 2. Proposed Mechanism by Which Preoperative SGB Interrupts the Sympathetic-Inflammatory Cascade to Reduce Postoperative Neurocognitive Disorders

Table 1. Summary of Clinical Studies Evaluating Preoperative Stellate Ganglion Block (SGB)

Study (Year)	Design	N (SGB / Control)	Population / Surgery	SGB Technique	Primary Outcomes	Key Findings
Xue et al. (2025)	Randomized Controlled Trial (RCT)	98 (49 / 49)	Elderly (≥65 yr) undergoing laparoscopic gastrointestinal surgery	Ultrasound-guided unilateral SGB before induction 0.5% ropivacaine 20 mL	• POCD (POD1) • IL-6, MDA, SOD • Hemodynamics • VAS pain (0–24 h) • Length of stay	• **POCD:** 14.3% vs **36.7%** (P=0.011) • IL-6: ↓ 23.7% (P=0.001) • MDA: ↓ 21.8% (P=0.002) • SOD: ↑ 57.4% (P=0.001) • MAP stability: ↓ (P=0.001) • LOS: ↓ 2.3 days (P=0.001)
Yang et al. (2025)	Prospective Comparative Study	80 (40 / 40)	Elderly (≥60 yr) undergoing gastrointestinal tumor surgery	Ultrasound-guided unilateral SGB before induction 0.5% ropivacaine 20 mL	• MMSE, MoCA (POD3) • IL-6, MDA, SOD • GI recovery • Time to first flatus • Length of stay	• MMSE: ↑ (P=0.001) • MoCA: ↑ (P=0.001) • IL-6: ↓ 28.2% (P=0.001) • MDA: ↓ 16.8% (P=0.001) • SOD: ↑ 16.2% (P=0.001) • Time to first flatus: ↓ 1.7 days (P=0.001) • LOS: ↓ 1.9 days (P=0.001)
Jin et al. (2024)	Animal Study	60 rats (30 / 30)	Rat myocardial infarction model with secondary cognitive impairment	Ultrasound-guided left SGB 0.5% ropivacaine 0.3 mL	• Morris Water Maze • Novel Object Recognition • Cerebral blood flow • Transcriptomic analysis (NOS3 mRNA)	• Learning latency: ↓ 32.1% (P=0.001) • Novel object recognition: ↑ (P=0.001) • CBF: ↑ 78.4% (P=0.001) • NOS3 mRNA: ↑ 3.1× (P=0.001) • TNF-α: ↓ 62.1% (P=0.001) • IL-1β: ↓ 31.5% (P=0.001) • Modulation of FKBP5, MYD88, IRAK1, NOS3 pathways

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