Postoperative Visual Loss Following Spinal Surgery: Causes and Mechanisms of Injury

Postoperative visual loss (POVL) is a rare but devastating complication that can occur following spinal surgery, particularly those performed in the prone position. Despite its infrequency, the profound impact on patients' quality of life makes understanding its causes and mechanisms a critical concern for surgeons, anesthesiologists, and perioperative care teams.

Incidence and Clinical Significance

POVL after spinal surgery is reported with an incidence ranging from 0.013% to 0.2%, with higher rates in extensive or prolonged procedures. The condition can manifest as transient or permanent vision impairment, including complete blindness. Given the elective nature of many spinal surgeries, prevention of POVL is paramount.

Primary Causes of Postoperative Visual Loss

The three most prominent causes of POVL after spinal surgery are ischemic optic neuropathy (ION), central retinal artery occlusion (CRAO), and cortical blindness.

Ischemic Optic Neuropathy

Ischemic optic neuropathy, particularly the posterior form (PION), is the leading cause of POVL in spine surgery. It results from insufficient blood flow to the optic nerve, causing ischemia and subsequent infarction. Two main subtypes exist:

- Anterior ischemic optic neuropathy (AION): Involves the optic nerve head and is often associated with visible optic disc swelling.
- **Posterior ischemic optic neuropathy (PION):** Affects the retrobulbar portion of the optic nerve, typically without optic disc swelling, making diagnosis more challenging.

Central Retinal Artery Occlusion

CRAO results from obstruction of the central retinal artery, leading to acute retinal ischemia. It is commonly linked to direct external pressure on the globe during surgery, especially when the patient is in the prone position without adequate eye protection or positioning.

Cortical Blindness

This rare form arises from ischemia or infarction of the occipital cortex in the brain, secondary to hypotension or embolic events during surgery. Unlike optic nerve-related causes, pupillary reflexes remain intact, distinguishing cortical blindness clinically.

Mechanisms of Injury

Understanding the mechanisms behind POVL is essential to mitigate risk.

Hypotension and Anemia

Prolonged intraoperative hypotension and significant blood loss leading to anemia can reduce oxygen delivery to the optic nerve. The optic nerve is vulnerable to ischemia due to its limited collateral circulation, especially in PION.

Increased Intraocular Pressure and Venous Congestion

Positioning in the prone posture can increase intraocular pressure (IOP) due to venous congestion, especially if the head is positioned below the heart level or compressed against surgical supports. Increased IOP reduces perfusion pressure in the optic nerve and retina, precipitating ischemic damage.

External Compression

Direct pressure on the eyes from headrests or surgical equipment can occlude the central retinal artery or compress the globe, causing CRAO. Meticulous avoidance of pressure on the eyes during positioning is critical.

Embolic and Vascular Events

Embolism or thrombosis affecting the posterior circulation can cause cortical blindness. Additionally, systemic vascular disease, atherosclerosis, or prolonged hypotension heighten this risk.

Risk Factors

Certain patient and surgical factors increase susceptibility to POVL, including:

- Prolonged surgery duration (>6 hours)
- Large volume blood loss and transfusions
- Prone positioning with improper head support
- Pre-existing vascular disease, hypertension, or diabetes
- Use of vasopressors causing vasoconstriction

Conclusion

POVL following spinal surgery is a complex, multifactorial complication primarily driven by ischemic injury to the optic nerve or retina. Vigilant intraoperative monitoring of hemodynamics, meticulous patient positioning to avoid ocular compression, and minimizing blood loss are

crucial strategies to reduce risk. Heightened awareness among surgical teams and prompt recognition can improve outcomes for affected patients.

Continued research into hemodynamic optimization and protective positioning techniques remains essential to further decrease the incidence of this profound complication.