Cracking Open the Door on Perioperative Visual Loss

RARE events are nearly impossible to study. There often simply are not enough of them to consolidate into meaningful case series, and information from these scarce occurrences is not sufficient to allow anything except very minimal analyses. Therefore, we are typically left with only conjecture on etiologies that may have prompted the rare events. That conjecture causes all sorts of problems, with “experts” claiming their own versions of potential etiologies and, sad to say, chastising colleagues in medicolegal cases for not providing the experts’ theoretical standards of care that would have avoided the rare events. This rare event scenario has been particularly true during the past decade for blindness that develops after spine fusion surgery. Fortunately, in this issue of Anesthesiology, The Postoperative Visual Loss Study Group provide us with a novel application of several methodologies that allows them to detect important risk factors for ischemic optic neuropathy (ION) in spine fusion patients and to speculate on potential etiologies of this devastating perioperative complication.

The authors have taken events in the American Society of Anesthesiologists’ Postoperative Visual Loss Registry and used them as cases in a large 1:4 case-control study. In this study, controls were obtained from 17 medical centers that perform large volumes of spine fusion surgery. The blending of a case series (registry) and a multicenter case-control methodology is unique, allowing the authors to develop analyses for risk factors associated with perioperative ION and this type of surgery. The authors were careful in the construction of their methodology. For example, they matched out only one variable: cases and controls had to share the same year of surgery to avoid potential changes in practice over time influencing the outcomes. Their study design did, however, require similar criteria for inclusion; thus, all patients were adults, had anesthesia for longer than or equal to 4 h, and were placed in prone positions for at least a portion of their procedures. These inclusion criteria prevented analyses of several important questions (e.g., odd ratios for prone vs. other positions such as lateral or supine in spine fusion surgery) or limited the power of analyses for others (e.g., odd ratios for the full range of anesthetic durations).

While the authors found six risk factors associated with ION in the registry population, half of these strongly support their speculation that acute venous congestion of the optic canal is a potential etiology of ION in this setting. The use of a Wilson surgical bed frame, with its increased curvature resulting in the head being lower than the heart; obesity, with its potential elevation of intraabdominal pressure in prone-positioned patients; and long anesthetic durations can all contribute to increased venous congestion in the optic canal and potentially reduce optic nerve perfusion pressure. Unfortunately, the study’s retrospective methodology did not allow the authors to consider how patient tilt (e.g., head down vs. other positions) may have played a role.

The authors also found that increased estimated blood loss, male gender, and lower percent of colloid administration were independently associated with the development of ION after spinal fusion surgery. They offer insights as to how these may or may not be clinically important. Their speculation on why 69% of the blindness cases occurred in men, when there were similar proportions of men and women in the controls who underwent spine fusion surgeries, is partic-

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ularly interesting. They note that there is evidence estrogen may provide a neuroprotective effect.

A factor in perioperative neuropathies not studied extensively, but one that the authors tangentially mention in this blindness report, is systemic inflammation. A number of perioperative events trigger significant systemic inflammation and immunosuppression (e.g., venous congestion, blood transfusions, inhaled anesthetics). Until recently, no one linked perioperative neuropathies, including ION, to systemic inflammation. In 2010, however, Staff et al. reported on 33 patients with prolonged postoperative ulnar neuropathy. Sural nerve biopsies in 21 of these patients demonstrated epineuronal inflammation. Intensive immunotherapy in 17 of these 21 patients resulted in significant resolution of neurologic impairment ($P < 0.001$). Over time, investigators will have to learn what role, if any, an inflammatory response, either locally or systemically, plays in ION in this setting.

It is ironic that many “experts” in medicolegal cases involving perioperative blindness in spine surgery patients are absolutely sure that they know why these patients develop their vision loss. They often speak of standard care provided by the defendant anesthesiologist, frequently noting that intraoperative anemia or episodes of hypotension reflect poor anesthetic care. The Postoperative Visual Loss Study Group did not find an independent effect of intraoperative anemia or blood pressure more than 40% below baseline for 30 congruent or additive minutes. Case reports and case series, the only clinical data available on this rare event until the current study, do not provide sufficient data to allow analyses of these two oft-cited intraoperative variables. For now, we can be thankful to the authors that they have provided data that raise doubt as to the veracity of standard of care claims by “experts” on these two issues and have offered us constructive ideas to study as we search for ways to reduce the incidence of this catastrophic problem.

How should we use the information from the report by The Postoperative Visual Loss Study Group in our care of patients? The results suggest that the American Society of Anesthesiologist’s 2006 Practice Advisory on this issue is still relevant. Basically, it is prudent to attempt to reduce venous congestion in the optic canal. That is, we should consider using positions that allow the patients’ heads to be level with or higher than their hearts. It may be helpful to use colloids as well as crystalloids to maintain intravascular volume. Intraoperative positioning that helps reduce intraabdominal pressure and, therefore, venous congestion, may be useful. The use of the Wilson frame and other positioning devices should be assessed carefully, with a goal to reduce pressure on the abdomen and to keep the head level with or higher than the heart. Since the authors found duration of anesthesia to be an independent risk factor for ION in this population, it may be prudent to work with our spine surgeons to determine if there is merit to limiting the duration of surgeries that are anticipated to be prolonged, especially 6 h or longer. Staging these procedures may be helpful.

For clinical researchers, this report suggests many new questions for study. Until now, there have been no data to support speculation on etiologies of ION in this setting. Can we determine the role that inflammation may play in perioperative ION? What really happens in the optic canal during these surgeries? Does venous congestion occur, and does it reduce optic nerve perfusion? Can we develop radiologic techniques or biologic/physiologic markers to study the optic nerve and canal in prone-positioned patients? Is there an impact of estrogen or other hormones on the development (and therefore, potentially the prevention) of central or peripheral neuropathies? Can new spine fusion techniques and intraoperative positioning mechanics impact optic nerve perfusion or reduce operative times and blood loss? The lead authors and the many contributors to this study deserve our congratulations for creatively providing insights that finally allow us to move forward with additional studies. They have opened the door, even if only a crack. Their work offers hope that we may one day reduce or eliminate perioperative blindness in spine surgery patients.

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References


