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Postoperative Visual Loss

Still No Answers—Yet

POSTOPERATIVE visual loss (PVL) is a devastating and poorly understood injury. Although rare, there are certain operative procedures, especially spinal surgery, in which the incidence seems to be significantly higher. Ischemic optic neuropathy (ION), which affects the anterior or posterior portions of the optic nerve, is the most common cause of PVL.[1] Visual loss may also be caused by retinal arterial occlusion and cortical blindness. Awakening with visual impairment may be one of the most frightening and catastrophic postanesthetic complications that a patient could sustain. It is also an enormous medical–legal liability problem. PVL has evoked controversy. Is it a preventable injury? If so, is it possible or even desirable to change our practice in an attempt to prevent it? Should we routinely inform patients of this risk?[1,2] Lee and Lam[3] report in this issue of ANESTHESIOLOGY yet another case of postoperative blindness after spinal surgery. Although the report does not contain new information, it is disturbing as one more vivid reminder of how PVL can follow a seemingly uncomplicated anesthetic administration without the commonly cited risk factors, which include hypotension, anemia, and external compression of the eye.[1,4] There have been many other cases reported that lack these risk factors as well.[4] We need to look beyond a simple approach to PVL and consider how we might try to prevent this adverse event by taking a systems approach. Because of the impression that the incidence of PVL is increasing, it is essential that we learn more about its causes.

To achieve our goals of consistently good outcomes, an environment fostering a rich reporting culture must be created and supported to capture accurate data with details of clinical care.[5] However, many obstacles prevent adequate reporting of rare but serious events of this type. Disincentives to reporting include extra work, skepticism, lack of trust, fear of reprisal, no effective means to report, and an "organizational culture" that discourages reporting.[6,7] The majority of our knowledge about PVL derives from case reports by ophthalmologists and surgeons, and it is only in the past 5 yr that cases have begun to be reported in the anesthesia literature.[8–10] The incidence of PVL in a general surgical population is 1 in 61,000.[9] This low incidence renders a prospective study difficult if not impossible. In a recent study of nearly 225,000 anesthetics over a 15-yr period at our institution, the incidence of PVL after spinal surgery was 1 in 1,100 (3 patients of 3,351), a 50-fold higher rate compared with all other procedures. The result after spinal surgery is in accordance with estimates derived from survey studies.[11–13] Open heart surgery, head and neck surgery, and sinus surgery are also believed to be associated with a higher risk of PVL.[4] The medical–legal implications, underreporting, and the absence of an animal model have hampered achievement of an adequate understanding of the mechanisms of PVL.

Anesthesiologists may not be responsible for this injury in many instances, but we are in the best position to gather data to begin to understand this complication. There are a number of means to capture adverse event data reliably as demonstrated, *e.g.*, by the Australian Incident Monitoring System.[14] The American Society of Anesthesiologists (ASA) Closed Claims Project has established a Post-Operative Visual Loss Registry.* Since June 1999, 35 cases have been submitted anonymously to the Registry. Preliminary results were reported at the last ASA annual meeting[15] and in the *ASA Newsletter*.[16] The goal is to accumulate data from 100 patients with PVL. When completed, this project will provide by far the largest and most detailed characterization of patients that have sustained PVL. This is an important step toward beginning to achieve an understanding of PVL. To submit cases, the patient's medical record must be available, and data must be entered on standardized forms available from the Registry. Although the Registry has its limitations, *e.g.*, it cannot definitively establish the mechanisms of PVL, it is currently the only organized data gathering tool for PVL. With anonymity assured, anesthesiologists should not be reluctant to submit cases. Anonymity will help to ensure trust and confidentiality while incident reporting systems continue to evolve.

Another possibility would be to conduct a large, multicenter case control study to compare patients with case-matched controls. This type of study at our hospital did not show any differences in intraoperative factors, such as blood loss, blood pressure, hematocrit, or the quantities of fluid administered intraoperatively, but our results were limited by the small sample size of visual loss patients (four patients at a single institution). A larger study encompassing at least 15–20 affected patients undergoing the same surgical procedure might

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yield sufficient statistical power to examine at least some of the suspected factors. This study would begin to yield insight into the mechanisms of PVL.

A prospective study would be ideal but is especially difficult because of the low incidence. However, a look at some "intermediate" variables, such as blood pressure, intraocular pressure, or tests of visual function, may be of value. Another direction is to use an animal model, but none have been developed. The monkey's ocular circulation is closest to that of humans,[17] but expense is a limitation. The rat, whose optic nerve is not dissimilar from the human,[18] would be a potential alternative.

For a study of PVL to produce meaningful results, what should we be looking for? Indices of ocular perfusion, blood loss, and fluid administration would seem to be relevant, but for reasons we will explain, the role of these is currently controversial. Looking at blood flow in the optic nerve would be highly relevant but is not yet possible on a routine clinical basis. There are patient-specific factors as well that should be considered. The vascular supply to the optic nerve differs between the anterior and the posterior portions of the nerve. The anterior optic nerve head is supplied primarily by the end-arterial posterior ciliary arteries, and the posterior is supplied by penetrating pial vessels.[4] Some individuals are more susceptible to ischemia in the anterior optic nerve because of variation in the number of posterior ciliary arteries, with resultant "watershed" zones.[19] Up to 20% of healthy patients have abnormal autoregulation (undetectable clinically) in the anterior optic nerve.[20] Vessels in the posterior optic nerve are easily compressible and, together with changes in perfusion pressure, may render some patients more susceptible to ION.[21,22] Decreases in perfusion are tolerated within the autoregulatory limits in the retina and optic nerve in healthy patients, but atherosclerosis may decrease blood flow in the optic nerve.[23] Therefore, patients with vascular disease may be at higher risk for ION.

Decreased perfusion pressure in the retina or optic nerve may be caused by decreased mean arterial pressure, or increased pressure in the venous drainage of the retina or optic nerve. Therefore, prolonged decreases in blood pressure, especially in patients with disturbed autoregulation, might be deleterious. Increased venous pressure, *e.g.*, with internal jugular vein compression or ligation, prolonged head-down position, or large quantities of fluid infusion, also decrease perfusion. With prone positioning, increases in intraocular pressure might be potentiated by these factors. The combination of decreased blood pressure and increased venous pressure seems to pose the greatest risk.[4] External compression of the eye could increase intraocular pressure, decreasing perfusion pressure, and cause retinal ischemia[24]; nonetheless, it is not likely that compression can cause an isolated ION without retinal ischemia.[2]

The optic nerve seems highly sensitive to the effects of acute blood loss,[1] but there is no convincing evidence that isovolumic hemodilution exposes the optic nerve to risk. This is an important issue because of the general trend to reduce blood usage intraoperatively.[1,8] Hemodilution did not significantly decrease oxygen delivery to the choroid while increasing retinal oxygen delivery in cats.[25,26] Arguing further against an exclusive role for hypotension and hemodilution is their routine occurrence during cardiopulmonary bypass; the low incidence after bypass suggests the involvement of other factors. Moreover, for patients sustaining PVL after spinal surgery, hematocrit and intraoperative blood pressure were no different compared with unaffected controls. Affected patients underwent longer operations with greater blood loss, similar to findings in cardiac surgical patients sustaining ION.[11] In addition, ION patients who had undergone cardiac surgery were more likely to be fluid overloaded and to require vasopressors postoperatively.[27] Hayreh[28,29] has suggested from monkey studies that vasoconstrictors, such as angiotensin and serotonin, may in fact play a role in the development of ION.

In the absence of obvious external compression of the eye, PVL seems to be a multifactorial problem with no consistent underlying mechanism. Anatomical variation in the blood supply to the optic nerve is undetectable by the anesthesiologist but could explain why only a small proportion of patients sustain this complication, or perhaps this variation, taken together with major shifts in fluid balance and in blood pressure, may be responsible. In summary, the study of PVL is at this time akin to that of trying to understand malignant hyperthermia in the 1960s. Given the heightened awareness of the potential impact of improvements in patient safety[30] and the large national increase in funding for patient safety research in the last year,† we would hope this devastating disorder will be recognized as an important field to fund and study. Ultimately, a larger epidemiologic study as well as research at a basic level will be necessary to determine the underlying causes and means to prevent PVL.

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References

1. Williams EL, Hart WA, Tempelhoff R: Postoperative ischemic optic neuropathy. Anesth Analg 1995; 80:1018–29
2. Roth S, Roizen MF: Ischemic optic neuropathy: Role of the anesthesiologist? (letter). Anesth Analg 1996; 82:435–6
3. Lee LA, Lam AM: Unilateral blindness after prone lumbar spine surgery. ANESTHESIOLOGY 2001; 95:793–5
4. Roth S, Gillesberg I: Injuries to the visual system and other sense organs,

† Available at: www.ahrq.org. Accessed July 2001

Anesthesia and Perioperative Complications, 2nd edition. Edited by Benumof JL, Saidman LJ. St. Louis, Mosby, 1999, pp 377–408

5. Blumenthal D: Making medical errors into "medical treasures." JAMA 1994; 272:1867–8

6. Barach P, Small SD: Reporting and preventing medical mishaps: Lessons from non-medical near miss reporting systems. Br Med J 2000; 320:753–63

7. Leape LL: Error in medicine. JAMA 1994; 272:1851–7

8. Brown RH, Schauble JF, Miller NR: Anemia and hypotension as contributors to perioperative loss of vision. ANESTHESIOLOGY 1994; 80:222–6

9. Roth S, Thisted RA, Erickson JP, Black S, Schreider BD: Eye injuries after nonocular surgery: A study of 60,965 anesthetics from 1988–1992. ANESTHESIOLOGY 1996; 85:1020–7

10. Roth S, Nunez R, Schreider BD: Visual loss after lumbar spine fusion. J Neurosurg Anesthesiol 1997; 9:346–8

11. Myers MA, Hamilton SR, Bogosian AJ, Smith CH, Wagner TA: Visual loss as a complication of spinal surgery. Spine 1997; 22:1325–9

12. Stevens WR, Glazer PA, Kelley SD, Lietman TM, Bradford DS: Ophthalmic complications after spinal surgery. Spine 1997; 22:1319–24

13. Cheng MA, Sigurdson W, Tempelhoff R, Lauryssen C: Visual loss after spine surgery: A survey. Neurosurgery 2000; 46:625–31

14. Runciman WB, Sellen A, Webb RK, Barker L: Errors, incidents, and accidents in anesthetic practice. Anesth Intensive Care 1993; 21:506–19

15. Uppal M, Posner K, Roth S: Visual loss complicating surgery: Review of 21 cases (abstract). ANESTHESIOLOGY 2000; 93(suppl 3A):A1136

16. Lee LA: Postoperative visual loss data gathered and analyzed. ASA Newsletter 2000; 64:25–7

17. Alm A, Bill A: Ocular circulation, Adler's Physiology of the Eye, 8th edition. Edited by Moses A, Hart C. St. Louis, CV Mosby, 1987, pp 183–203

18. Morrison JC, Johnson EC, Cepurna WO, Funk RH: Microvasculature of the rat optic nerve head. Invest Ophthalmol Vis Sci 1999; 40:1702–9

19. Hayreh SS: In vivo choroidal circulation and its watershed zones. Eye 1990; 4:273–89

20. Pillunat LE, Anderson DR, Knighton RW, Joos KM, Feuer WJ: Autoregulation of human optic nerve head circulation in response to increased intraocular pressure. Exp Eye Res 1997; 64:737–44

21. Hayreh SS: Posterior ischemic optic neuropathy. Ophthalmologica 1981; 182:29–41

22. Isayama Y, Hiramatsu K, Asakura S, Takahashi T: Posterior ischemic optic neuropathy: I. Blood supply of the optic nerve. Ophthalmologica 1983; 186:197–203

23. Hayreh SS, Bill A, Sperber GO: Effects of high intraocular pressure on the glucose metabolism in the retina and the optic nerve in atherosclerotic monkeys. Graefes Arch Clin Exp Ophthalmol 1994; 232:745–52

24. Walkup HE, Murphy JD: Retinal ischemia with unilateral blindness: A complication occurring during pulmonary resection in the prone position. J Thorac Cardiovasc Surg 1952; 23:174–5

25. Neely KA, Ernest JT, Goldstick TK, Linsenmeier RA, Moss J: Isovolemic hemodilution increases retinal oxygen tension. Graefes Arch Clin Exp Ophthalmol 1996; 234:688–94

26. Roth S: The effects of isovolumic hemodilution on ocular blood flow. Exp Eye Res 1992; 55:59–63

27. Shapira OM, Kimmel WA, Lindsey PS, Shahian DM: Anterior ischemic optic neuropathy after open heart operations. Ann Thorac Surg 1996; 61:660–6

28. Hayreh SS: Anterior ischemic optic neuropathy: VIII. Clinical features and pathogenesis of post-hemorrhagic amaurosis. Ophthalmology 1987; 94:1488–502

29. Hayreh SS: Retinal and optic nerve head ischemic disorders and atherosclerosis: Role of serotonin. Prog Retin Eye Res 1999; 18:191–221

30. Kohn LT, Corrigan JM, Donaldson MS: Committee on Quality of Health Care in America, Institute of Medicine. To Err Is Human: Building a Safer Health Care System. Washington, DC, National Academy, 2000