total volume of fluid replacement and the total nonblood fluid replacement were greater in the ION patients, and the administration of colloid as a percentage of the total nonblood replacement was less in the ION patients. The only remaining fluid in this analysis would be crystalloid. These findings directly support the concept that the crystalloid fluid volume was significantly greater in the ION patients, although a direct comparison of the volume of crystalloid administered in the two groups did not reach significance.

Other significant differences between the two groups included gender, obesity, use of the Wilson frame, duration of anesthesia, and estimated blood loss. Both the Study Group and Dr. Warner in his editorial[4] suggested that ION may be less common in women than men because of the protective effect of estrogen. A simpler and more reasonable explanation for the difference is that most anesthesia providers are more likely to give larger volumes of crystalloid fluid to men weighing 80–120 kg than they are to women weighing 60–80 kg. With respect to obesity, the Study Group suggested that positioning the obese patient prone may increase intraabdominal, intrathoracic, intraocular, and venous pressures and produce ischemia of the optic nerve by a variety of mechanisms. Another more plausible explanation would be that if prone positioning did increase venous pressure in the obese patients, it would be manifest most profoundly as blood loss at the operative site, which in turn, would necessitate greater fluid administration, including crystalloid fluid. Finally, the Study Group suggested that the reason that ION was more common with use of the Wilson frame was because the head is more dependent with its use. However, this explanation is only conjecture because the exact positioning of the head was not documented in all of the patients who experienced ION while on the Wilson frame. When using the Wilson frame, the head need not be dependent because it can be supported in the neutral position with pillows and head supports, and this may have been done in some of the ION patients on the Wilson frame. I do not believe that exactness in head position is necessary provided crystalloid fluid volume administration is limited. We do a large number of robotic-guided, laparoscopic, retropubic radical prostatectomies with the patients in a very steep Trendelenburg position for 4–6 h. The crystalloid fluid volume is limited to less than 1 l until the patient is returned to the level position to avoid fluid collection in the bladder, which will obscure the operative field when the bladder is opened. We have not had a case of ION in this population. Two things stand out in the reported cases of ION occurring after prostate surgery: the patients were in a Trendelenburg position for 4–6 h, and they received approximately 5–10 l crystalloid fluid.

The recent report of the American Society of Anesthesiologists Task Force on Perioperative Visual Loss[5] advocates the use of both colloid and crystalloid fluids but does not recommend any limit on the latter. Based on the evidence to date, which admittedly is mostly circumstantial, I would urge anesthesia providers to strongly consider limiting crystalloid fluid therapy to less than 40 ml/kg regardless of operative length. With this change alone, I believe that we will experience a measurable decrease in the incidence of ION.

C. Philip Larson, Jr., M.D.C.M., David Geffen School of Medicine at University of California, Los Angeles, California; Ronald Reagan UCLA Medical Center, Los Angeles, California. plarson@ucla.edu

References

1. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, Domino KB: The American Society of Anesthesiologists Postoperative Visual Loss Registry: Analysis of 93 spine surgery cases with postoperative visual loss. ANESTHESIOLOGY 2006; 105:652–9
2. Larson CP Jr: Excessive crystalloid infusion may contribute to ischemic optic neuropathy [letter]. ANESTHESIOLOGY 2007; 106: 1249
3. Postoperative Visual Loss Study Group: Risk factors associated with ischemic optic neuropathy after spinal fusion surgery. ANESTHESIOLOGY 2012; 116:15–24
4. Warner MA: Cracking open the door on perioperative visual loss. ANESTHESIOLOGY 2012; 116:1–2
5. American Society of Anesthesiologists Task Force on Perioperative Visual Loss: Practice advisory for perioperative visual loss associated with spine surgery: An updated report by the American Society of Anesthesiologists Task Force on Perioperative Visual Loss. ANESTHESIOLOGY 2012; 116:274–85

(Accepted for publication April 26, 2012.)

In Reply:

We would like to thank Drs. Kempen, Raw, and Larson for their interest in our study on determining risk factors for perioperative ischemic optic neuropathy (ION) after spinal fusion surgery in the prone position.[1] Dr. Kempen's suggestion to perform spine surgery in the lateral position, instead of the prone position, is intriguing. We have also considered this possibility in the past and queried our surgical colleagues. In special situations, such as second or third trimester pregnancy, when postponement of surgery is not feasible, spine surgery has been performed in the lateral position. However, the "up-down" manipulations required in the lateral position are technically more difficult than the more symmetric "right-left" manipulations in the prone position. Achieving ideal spinal alignment is much more challenging technically in the lateral position. Many surgeons rely on the lordosis imparted by some of the spinal frames in the prone position to provide optimal "anatomic" alignment for fusion.

We agree with Dr. Kempen that further study should be performed to examine the relative risks and benefits of staging very prolonged spine surgery with expected high blood loss, as we noted on pages 22 and 23 of our article.[1] The supposition that this injury may reflect the coincidental occurrence of spontaneously occurring ION in a general non-

surgical population is not supported by national incidence data. Data from the Nationwide Inpatient Sample published by Shen *et al.* demonstrates a greatly increased odds ratio of 6.96 for developing ION in spinal fusion surgery compared with the referent abdominal surgery.[2] Its occurrence in children and relatively healthy adults after spinal fusion surgery is not consistent with the high incidence of atherosclerotic risk factors found in the subpopulation of nonsurgical patients who develop nonarteric anterior ION.[3] In addition, the high percentage of cases with bilateral profound loss of vision in perioperative ION is not consistent with the clinical course of nonarteritic anterior ION, which typically presents with unilateral disease with less severe loss of vision.

Lastly, we also considered the possibility, like Dr. Kempen, that vasopressors may be a contributory factor in the development of ION. We did not find a significant association with vasopressor use in the univariate analysis, as noted in table 1 on page 18 of our article.[1]

Dr. Raw offers the interesting hypothesis that perioperative inflammation may contribute to the development of ION after spinal fusion surgery. Given the low incidence of perioperative ION, it may prove difficult to examine this hypothesis with prospective studies. Dr. Raw also notes that increased plasma homocysteine levels and lower vitamin B6 levels are independently associated with the occurrence of nonarteric anterior ION in the nonsurgical population. For this reason, nitrous oxide could play a contributory role. In our 2006 study, less than one-quarter of the 83 patients with ION after spinal fusion received nitrous oxide, making it unlikely that nitrous oxide administration is an important factor in perioperative ION.[4] But a systemic inflammatory syndrome may result from prolonged, complex surgery, and the notion that inflammation is a pathogenic factor in axonal injury or brain injury is supported by experimental studies in animals.[5,6]

We appreciate Dr. Larson's continued interest in perioperative ION and his efforts to provide precise limitations on the amount of crystalloid (40 ml/kg) administered. We remain curious about the 40 ml/kg crystalloid limit. It is not clear if he is encouraging a practice of intentional hypovolemia, or if he is advocating the use of colloid along with crystalloid to maintain euvolemia, as recommended in the American Society of Anesthesiologists' practice advisory.[7] The former practice of intentional hypovolemia in these cases with large blood loss and prolonged duration would subject patients to a high potential for end organ ischemia, or ultimately, cardiovascular collapse. The latter practice of using colloids along with crystalloids may reduce the incidence of ION, as suggested by the results of our multicenter case-control study. However, our studies and understanding of the current literature do not suggest that a specific limit to crystalloid administration, such as 40 ml/kg, will prevent perioperative ION. Of note, the mean crystalloid infusion for the control patients in our study was 4.6 l ± 2.3 l, well above Dr. Larson's limit of 40 ml/kg for most patients. The highest amount of crystalloid infused in a control patient was more than 18 l. Conversely, crystalloid limitation did not protect all patients from ION, as the lowest amount of crystalloid infused in an ION case was 2.2 l. Based on our observations, we do not believe that the 40 ml/kg crystalloid limit prevents ION, nor does it help predict those who might develop ION. Dr. Larson's supposition that the increased risk of ION seen in men was because men received more crystalloid than women was not supported by our data. There were no significant differences in the amount of crystalloid received between men and women, either in cases or controls.

Although we agree that increased venous pressure is likely to increase blood loss and fluid resuscitation, there are many types of surgery where arterial bleeding results in much greater blood loss and fluid resuscitation, but without an associated risk for ION. Therefore, we believe that the increased venous pressure – in the head – is one of the most important risk factors placing prone spinal fusion surgery patients at increased risk for developing ION. This same feature of increased venous pressure in the head is also present in other surgical procedures that carry a high risk for ION groups: bilateral radical neck dissections with ligation of bilateral external and internal jugular veins[8,9] and laparoscopic/robotic prostatectomies with the head placed in steep Trendelenburg for prolonged duration.[10] It is interesting that Dr. Larson has "specialized" fluid management plans for these types of procedures with increased venous pressure in the head and high risk for ION, yet dismisses venous congestion as a significant contributory factor for ION.

We are impressed by Dr. Larson's efforts to prevent ION in robotic prostatectomy patients who require steep Trendelenburg position for 4–6 h. It is not clear to us if cases of that duration for this procedure are at risk for ION. The duration of surgery for the five reported cases of ION after laparoscopic prostatectomy ranged from 6.5 to 9.9 h, with four of these cases lasting 7.9 h or more.[9,10] One additional case of ION occurring after a laparoscopic proctocolectomy also lasted greater than 6 h.[11] We are not aware of cases of ION after 4–6 h of robotic prostate surgery that were associated with Trendelenberg position and 5–10 l of crystalloid administration.

We applaud Dr. Kempen's, Dr. Raw's, and Dr. Larson's interest in this topic and their efforts to minimize the occurrence of perioperative ION. This is a devastating perioperative complication that deserves continued reflection and sound, methodical investigation.

Lorri A. Lee, M.D.,* Steven Roth, M.D., Michael M. Todd, M.D., Karen L. Posner, Ph.D., Nayak L. Polissar, Ph.D., Moni B. Neradilek, M.S., Nancy J. Newman, M.D., Robert A. Caplan, M.D., Frederick W. Cheney, M.D., Karen B. Domino, M.D., M.P.H., for The Postoperative Visual Loss Study Group. *University of Washington School of Medicine, Seattle, Washington. lorlee@uw.edu

References

1. Postoperative Visual Loss Study Group: Risk factors associated with ischemic optic neuropathy after spinal fusion surgery. Anesthesiology 2012; 116:15–24
2. Shen Y, Drum M, Roth S: The prevalence of perioperative visual loss in the United States: A 10-year study from 1996 to 2005 of spinal, orthopedic, cardiac, and general surgery. Anesth Analg 2009; 109:1534–45
3. Mathews MK: Nonarteritic anterior ischemic optic neuropathy. Curr Opin Ophthalmol 2005; 16:341–5
4. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, Domino KB: The American Society of Anesthesiologists Postoperative Visual Loss Registry: Analysis of 93 spine surgery cases with postoperative visual loss. Anesthesiology 2006; 105:652–9
5. Salgado C, Vilson F, Miller NR, Bernstein SL: Cellular inflammation in nonarteritic anterior ischemic optic neuropathy and its primate model. Arch Ophthalmol 2011; 129:1583–91
6. Terrando N, Monaco C, Ma D, Foxwell BM, Feldmann M, Maze M: Tumor necrosis factor-α triggers a cytokine cascade yielding postoperative cognitive decline. Proc Natl Acad Sci U S A 2010; 107:20518–22
7. American Society of Anesthesiologists Task Force on Perioperative Blindness: Practice advisory for perioperative visual loss associated with spine surgery: A report by the American Society of Anesthesiologists Task Force on Perioperative Blindness. Anesthesiology 2006; 104:1319–28
8. Pazos GA, Leonard DW, Blice J, Thompson DH: Blindness after bilateral neck dissection: Case report and review. Am J Otolaryngol 1999; 20:340–5
9. Weber ED, Colyer MH, Lesser RL, Subramanian PS: Posterior ischemic optic neuropathy after minimally invasive prostatectomy. J Neuroophthalmol 2007; 27:285–7
10. Lee LA, Posner KL, Bruchas R, Roth S, Domino KB: Visual loss after prostatectomy. Paper presented at: annual meeting of the American Society of Anesthesiologists; October 18, 2010; San Diego, California
11. Mizrahi H, Hugkulstone CE, Vyakarnam P, Parker MC: Bilateral ischaemic optic neuropathy following laparoscopic proctocolectomy: A case report. Ann R Coll Surg Engl 2011; 93:e53–4

(Accepted for publication April 26, 2012.)

Author-created Surrogate Time Intervals Misrepresents Actual Times

To the Editor:

We are constantly amazed when the performance of anesthesiologists practicing in the anesthesia care team model (including both private practice and academic settings) is judged by administrators, operating room (OR) managers, and surgeons on "first-case starts." Unfortunately, too many of these nonanesthesiologists use magical thinking and demand that all the ORs start at the same time and without delay. The reality is that in an anesthesia care team model, the anesthesiologist cannot be at two places at once. Therefore, it should be obvious that when starting more than one room first thing in the morning, surgeons and OR teams may have to wait for the anesthesiologist to become available to attend to each patient.

With this understanding of reality, we read Epstein and Dexter's recent publication with great interest.[1] But unfortunately, instead of looking at the first-case starts, the authors chose to also look at other portions of anesthesia care as well. We were dismayed over this methodology because the authors utilized retrospective data that lacked a critical data element necessary to accurately determine anesthesiologist presence. Because their retrospective data from the single center studied did not include the actual time and duration of demanding portions of anesthesia care, the authors had to develop surrogate time interval definitions that would capture the critical portions of anesthesia team care. This deserves emphasis. The authors do not know from the electronic health record data when the actual demanding portions of anesthesia occurred, the duration of those occurrences, and the role the attending anesthesiologist played in managing those events. These surrogate definitions are found in table 2 of their publication.

To illustrate how broad these surrogate time intervals are and how they include not only the critical portion but also many noncritical portions, one only has to look at the first definition: induction of general anesthesia. The authors chose to define this time period as when the patient enters the OR to intubation (or the equivalent) + 3 min. Therefore, they include within their definition of the induction the following events: transportation into the OR, movement of the patient from the stretcher to the bed, placement of the IV (if not done in holding), placement of standard monitors, and waiting for the surgeon to arrive. This overly broad definition creates artificial "conflicts," where none in fact occur. For example, if the anesthesiologist is present in OR A for extubation, and the nonphysician anesthesia provider brings the patient into OR B, then, by the authors' definition of induction, the anesthesiologist is not available for a critical portion and there is a "lapse" identified by the simulation.

One could apply this definition of induction to the surgeon. If the critical portion of the anesthetic begins when the patient arrives in the OR and includes the preoperative briefing (authors' definition), then similarly, a critical portion of surgery should include the time from the patient's arrival into the OR to the briefing. If a surgeon is not present during this period for probably justifiable reasons (*e.g.*, rounding on inpatients, meeting with the family of previous patient, and so on), the surgeon would be found in "lapse" of care by the authors and would contribute to avoidable inefficiencies.

This one example illustrates how using retrospective data and surrogate time intervals will result in exaggeration of so-called lapses. Similar problems exist for all the other definitions in their table 2.

Furthermore, electronic health records do not document the timing, duration, and content of every communication between anesthesiologist and nonphysician anesthesia provider (anesthesiology assistant, nurse anesthetist, or anesthe-