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An Optic Nerve at Risk and a Prolonged Surgery in the Prone Position

Time to Reconsider?

PATIENTS who undergo prolonged, complex spinal surgery in the prone position may have a one in a thousand chance of developing some vision loss in the postoperative period. This morbidity typically manifests as permanent blindness that can be partial or total and is usually bilateral. Although rare, cases with incomplete recovery have been described, but vision never returns to the preoperative level. While most agree that anesthesia is extremely safe, it is not well understood in either the medical community or the public at large that what may be a fairly routine procedure may result in a major human tragedy. The most common etiology of vision loss reported in the postoperative period is the result of a direct ischemic event at the level of the optic nerve(s) resulting in ischemic optic neuropathy (ION) by a mechanism that is as yet poorly understood. In this issue of ANESTHESIOLOGY, Lee *et al.*[1] contributed invaluable information in helping us to understand this puzzling complication through study of blood flow and oxygen delivery to the optic nerve and brain in piglets.

Since the first review in the anesthesia literature addressing ION 13 yr ago,[2] the condition has only become more frequent, while little progress has been made in understanding its etiology. Anemia, hypotension, and some degree of orbital edema secondary to venous congestion are thought to be the primary culprits. One theory advanced to explain the mechanism of postoperative ION is the creation of a "compartmental syndrome" within the optic canal that would impede circulation and oxygen delivery to the optic nerve. This theory is based in part on the fact that in humans, the optic canal is a closed space through which the optic nerve travels. A significant increase in intraocular pressure has been recorded during the prone position, which is further exacerbated with time.[3] Also, the probable decrease in venous return from the eye and the subsequent venous stasis may contribute to an increase in pressure within the optic canal. Furthermore, the vascular supply to the optic nerve consists of two small end arteries without collaterals.

An analysis of the American Society of Anesthesiologists Postoperative Visual Loss registry reported by the same author,[4] despite its modest numbers, did allow us to draw a picture of the patient population in which this event has occurred. Overall, it was the description of a fairly "common" healthy, middle-aged population. The significant factors are blood loss in excess of 1 liter and prone positioning lasting more than 6 h. Of interest, more than 50% of the patients were obese. The other usual suspects, such as hypertension, diabetes, and coronary or cerebral vascular disease, were not strong factors, although 82% of cases had at least one of these risk factors. Nothing was found that indicates a possible link between these cases except for the surgical procedure. However, the question of why an ischemic event occurred more readily at the level of the optic nerve in these patients instead of usual targets for ischemia, such as the brain or the myocardium, remained unanswered.

One of the reasons for the difficulty in understanding ION is the lack of a suitable animal model that could mimic the conditions in which ION has been described in humans. Orbits in most mammals are different from humans in that they are not a closed circumferential bone structure. There is a gap between the frontal bone and the zygoma that is closed by a ligament. Furthermore, it is difficult to simulate an actual prone position in animals. While one may have some reservation with the model, especially regarding the role of venous congestion, this study is the first to reproduce the succession of events that may lead to ION, basically anemia, hypotension, and venous congestion, and analyze its effects on blood flow and oxygen delivery to the optic nerve and the brain. The results are fairly straightforward: "Compensatory mechanisms for porcine cerebral blood flow maintain stable oxygen delivery under specified conditions of hypotension and anemia, whereas optic nerve compensatory mechanisms were unable to maintain blood flow and to preserve oxygen delivery." The authors conclude that the porcine optic nerve is even more susceptible to physiologic perturbations than the brain. This is a very important piece of the puzzle, and one can be tempted to associate it with the compartmental syndrome theory to finally construct a solid hypothesis to explain the mechanism of ION: a highly ischemia-susceptible tissue subjected to increased surrounding pressures and decreased oxygen delivery.

However, not all patients undergoing spinal surgery develop ION, even after extreme blood loss and/or du-

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ration. Even more confounding, ION has been described after fairly short (3- to 4-h) surgery without major blood loss. This observation may suggest specific patient-based risk factors. ION is not a new entity, and ophthalmologists are aware that it happens spontaneously and acutely, usually in patients awakening from their natural sleep. This type of blindness has traditionally been attributed to the nebulous "small vessel disease" category of which hypertension, diabetes, and vascular disease are known risk factors. Of interest, a small cup/disk ratio on funduscopy, also dubbed "disk at risk," has been associated with spontaneous ION. Association with sleep apnea has also been reported. Here again, it is difficult to evaluate the prevalence of the spontaneous disease, computed to be approximately 1 in 10,000–16,000 adults, and some neuro-ophthalmologists are convinced that the incidence is on the rise. In the setting of ION as a postoperative complication, the "spontaneous" aspect of the disease should not be ignored.

Unless we are able to identify those at higher risk for ION and its etiology, we cannot generate a prevention scheme tailored to patient risk. More clinicians are explaining to their patients as part of the informed consent that blindness in the postoperative period is a possibility. Understandably, patients have a hard time associating blindness with back surgery, and it is unclear whether patients take the warning of potential blindness seriously.

Patients are facing a heavy price in terms of human cost for surgeries, some of which are not supported by strong evidence for long-term benefit. Although it is usually not the role of anesthesiologists to discuss surgical indications, this case places the anesthesiologist in an untenable position. We are aware that ION may happen and are very conscious that we cannot stratify patients preoperatively for risk of this devastating complication. Outcome studies for prolonged complex spinal surgeries need to be conducted, and their possible benefits should be weighed against the possibility of blindness.

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