Visual Loss in a Prone-Positioned Spine Surgery Patient with the Head on a Foam Headrest and Goggles Covering the Eyes: An Old Complication with a New Mechanism

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A patient developed central retinal artery occlusion during surgery in the prone position with eye protectors placed over his eyes. We discuss the potential hazards of using such a device (the Dupaco Opti-Gard) in this setting.
(Anesth Analg 2007;104:1185–7)

Eyes should be protected from injury during anesthesia. Although the most common intraoperative injury is corneal abrasion, the most serious risk, particularly when the patient is positioned prone, is blindness (1,2). Some have advocated eye goggles, also known as eye protectors, to prevent these injuries (*http://www.dupacoinc.com/main.htm*). However, eye protectors themselves also have the potential for injury. We report a case of perioperative blindness related to use of eye protectors in a patient who underwent spine surgery in the prone position. Review of the Food and Drug Administration (FDA)'s Medwatch Database revealed that other eye complications have occurred in patients when the same device was used with foam headrests. We discuss the implications of these reports for the anesthesia practitioner.

CASE REPORT

A 53-yr-old, 5′ 8″-tall, 175 lb man, positioned prone on an Andrews frame, underwent an L3–4 posterior lumbar interbody fusion. He had a history of diet-controlled diabetes mellitus and hypertension, and had previously undergone anesthesia without incident. His preoperative arterial blood pressure was 110/70, heart rate 72 bpm, electrocardiogram showed normal sinus rhythm, and his hematocrit was 45%. Toprol, used for blood pressure control, was taken on the morning of surgery. His blood glucose level was 80–110 by Accucheck. His preoperative vision was normal.

After induction of anesthesia and endotracheal intubation, a Dupaco Opti-Gard Eye Protector (catalog no. 28310, Dupaco, Oceanside, CA, Fig. 1) was placed over the taped eyes. The patient was then turned prone, and his head was placed on an OSI Gentle-touch foam headrest (Orthopedic Systems, Union City, CA). The procedure was completed uneventfully. Throughout the anesthetic, his arterial blood pressure was maintained at approximately 100/60 or more, with the exception of 25 discontinuous minutes of blood pressure in the range of 98/58–70. The patient received 2500 mL of lactated Ringer's solution, 225 mL of cell saver blood, estimated blood loss was 600 mL, and urine output was 390 mL. His hematocrit the morning after surgery was 35%. The anesthesia provider indicated that the patient's eyes were checked every 15 min during the case by palpating the edge of the foam backing of the device and the contact point with the patient's face, starting from the forehead and moving in an inferior direction. Throughout the case, the patient's head did not change position, and the Opti-Gard was still in place when he was returned to the supine position at the end of surgery.

Six hours postoperatively, the patient had no light perception in the left eye, a 5 mm fixed left pupil nonreactive to direct light, a corneal abrasion over 70% of the surface of the left eye, prominent ciliary injection, lower lid edema, a pale retina, and a cherry-red spot in the macula. His intraocular pressure was 13 in the left and 17 in the right eye. The examining ophthalmologist noted that visualization of the fundus was very difficult in the left eye due to pronounced corneal haziness. Extraocular movements were normal. Exotropia was present in the left eye. The right eye was normal. A small subcutaneous hematoma was present on the patient's forehead just above the nose. In a photograph of the patient taken two days later, a U-shaped abrasion (Fig. 2) was present over the left superior eyelid. Central retinal artery occlusion was confirmed by fluorescent angiography 2 wk later, showing slightly delayed retinal transit time (13 s, normal <11 s), retinal arteriolar narrowing, a residual cherry red spot, and a pale disk (Fig. 3). A computed tomography scan of the orbit did not reveal any swelling. He never regained vision in the left eye. This case was reported by the hospital's risk manager to FDA Medwatch.

DISCUSSION

There are numerous literature reports of visual loss in patients who have undergone surgery in the prone

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Accepted for publication January 29, 2007.

Conflict of interest statement: Dr. Roth has provided expert witness testimony for which he has been compensated, concerning postoperative visual loss, on behalf of hospitals, anesthesia providers, and patients, including the patient described in this case report. The litigation involving this case has been closed via a confidential settlement. The patient has given written permission for publication of the relevant clinical details, which includes photographs of his injuries.

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DOI: 10.1213/01.ane.0000264319.57758.55

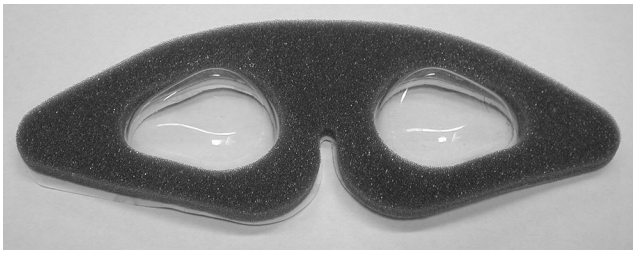

Figure 1. Front view of the Dupaco Opti-Gard.

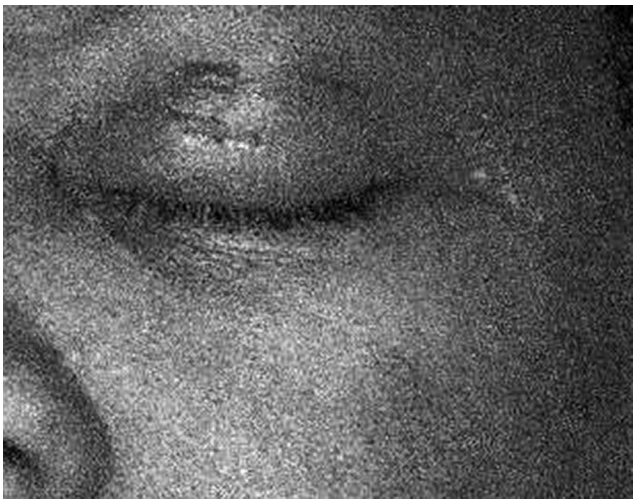

Figure 2. The patient's left eye has a U-shaped abrasion over the left superior eyelid in a photograph taken 2 days postoperatively.

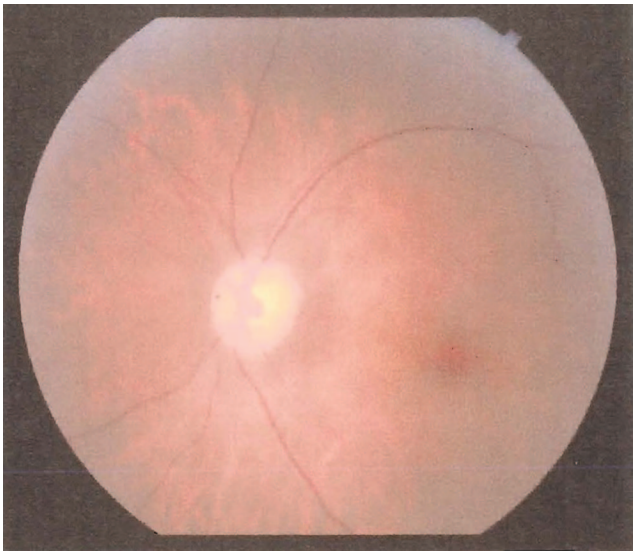

Figure 3. In a fundus photograph taken 2 wk after onset of visual loss, the left eye shows disk pallor, attenuated retinal arterioles, and a residual cherry-red spot.

position. In nearly all cases, the diagnosis has been either ischemic optic neuropathy or central retinal artery occlusion (CRAO) (3). In older case reports, CRAO was attributed to pressure on the eye exerted by the weight of the head compressing the eye against a horseshoe headrest (4). However, there are CRAO cases in patients positioned prone on other headrests wherein external pressure was inadvertently exerted on the eye (5). Nearly all reports of CRAO have involved unilateral injury. In addition, 10 spine surgery patients (11% overall) in the ASA Postoperative Visual Loss Registry have sustained unilateral CRAO, all presumed due to inadvertent compression of the eye (2). The mechanisms of retinal injury have been examined in animal models in various species (6–8).

To our knowledge, this case is the first report of visual loss in a patient in whom the eyes were protected by a specially designed eye protector. We reviewed the Medwatch MAUDE database (*http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfMAUDE/search.CFM*), using the "Advanced search" function for the terms: "Dupaco," "Opti-gard," "Eye protector," and "Shield, Ophthalmic." MAUDE data contain adverse event reports involving medical devices and consist of voluntary reports since June 1993, user facility reports since 1991, distributor reports since 1993, and manufacturer reports since August 1996. Three additional cases were found of patient injury when the Opti-Gard was used. In all three, patients were positioned prone with their faces in a foam headrest. The least serious injuries included postoperative keloid scarring from suspected pressure of the Opti-Gard on the side of the nose, and a superficial linear abrasion on the right upper cheek of another. A third patient sustained eyelid abrasions as in the above case report, and neuropraxia of the supraorbital nerve due to suspected pressure of the device on the supraorbital notch from which the nerve exits.

Even though Opti-Gard was originally designed to prevent corneal abrasion from drying of the eyes, foreign bodies, or other unintended materials from contacting the eye during surgery (US Patent no. 4,122,847, Craig), Dupaco markets it as a Class I FDA device categorized as an "Ophthalmic Shield." Along with the diagnosis of unilateral CRAO, a fixed pupil, the large corneal abrasion and haziness, lid edema, and the eyelid injury strongly suggest a compression-induced injury to the eye with trauma to the anterior structures and ischemia in the retina, effectively rendering any other mechanism of injury implausible in this patient. In addition, exotropia suggests extraocular muscle dysfunction secondary to compression. Because the anesthesia provider indicated that the Opti-Gard was in proper position at the conclusion of the surgery, we believe that the injury was due either to compression of the eye by the plastic lens of the device, which can be deformed by downward or sideward pressure, or to loosening of the glue attaching the flange of the device to the face, resulting in displacement of the lens into the eye when the anesthesia provider blindly palpated the edge of the protector. Clearance between the clear plastic lens of the device and the edge of the foam opening for the eyes in the OSI Gentle-touch is limited (Fig. 4). Blind palpation of the edge of the foam backing of the Opti-Gard during the anesthetic may have deformed

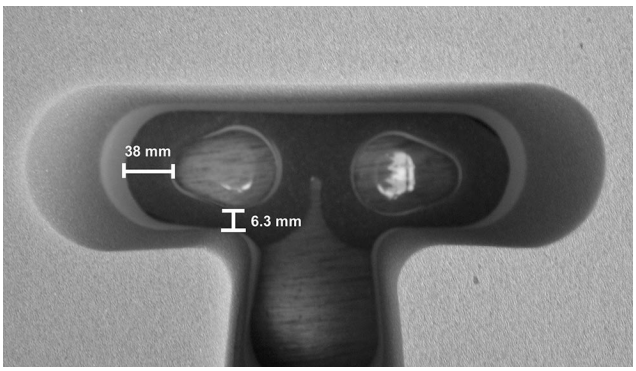

Figure 4. Position of the Opti-gard in an OSI Gentle-touch headrest showing the clearance from the foam to the edge of the lens.

the lens into the eye, or the glue adhesive might have loosened due to moisture accumulating on the face. The force required to pull Opti-Gard off the face, particularly with the patient positioned prone and subjected to typical anesthetic conditions, such as moisture on the face and ambient temperature changes, has not been tested.

In this case, the Opti-Gard inadvertently became a hazard to the patient rather than a protective device. The additional case reports to the FDA described above suggest that compressive injury is also a risk when the Opti-Gard is used in conjunction with foam headrests. One patient in the Medwatch reports also sustained apparent compression of the supraorbital nerve, which exits the orbit at the supraorbital ridge above the eye. The mechanism of this injury may be related to pressure of the plastic lens on the supraorbital ridge, or improper placement of the device over the eyes.

In summary, we report an unfortunate case of central retinal artery occlusion and subsequent blindness associated with use of the Dupaco Opti-Gard eye protector in a prone-positioned patient whose head was placed on an OSI Gentle-touch headrest. This event occurred because of loosening and/or compression of the Opti-gard plastic lens, followed by direct compressive contact between the plastic lens and the eye. There may be a greater margin of safety with other headrests that allow more room between the eye opening of the headrest and the edge of the plastic lens of the device, or where the anesthesia provider is able to visualize and intermittently assess the position of the Opti-Gard. However, because of the risk of inadvertent eye compression, despite palpation of the foam backing or even of the plastic lens itself, we feel there is no added safety advantage of the Opti-Gard for patients positioned prone. Taping of the eyes, intermittent palpation and visualization of the eyes should be adequate to detect compression and reduce the risk of direct eye injury (3).

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limited. Previous studies in adults show that the optimal intrathecal morphine has a much wider range, and that the dose is dependent on the anticipated degree of postoperative pain for the type of operation. For example, the suggested effective dose of intrathecal morphine is 500 μg for knee surgery (2), whereas it is 50 μg for transurethral prostate resections (3). Pediatric "high" spinal morphine dose used for oncology, spinal surgery, cardiac surgery, and frontal encephalocele repair suggests a threefold range: from 10 to 30 μg/kg (4,5). In a recent unpublished pediatric study, we found that 2 μg/kg intrathecal morphine provides excellent postoperative analgesia in patients undergoing hypospadias repair. We therefore believe that pediatric effective "low" morphine dose needs to be determined independently for each type of surgery.

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DOI: 10.1213/01.ane.0000278123.09637.b7

In Response:

We agree with the authors (1) that different doses of morphine may be administered intrathecally. However, our study (2) simply describes our experience with the use of intrathecal morphine in a dose of 4–5 μg/kg. We have also listed the surgical procedures where this dose was administered. Larger doses of intrathecal morphine tend to have a greater incidence of side effects (3) and usually require monitoring of the patient in an intensive care unit (ICU). About 90% of the patients in our study (2) were monitored on the regular floor and the others were admitted to the ICU as per the institutional policy of admitting all neurosurgical patients to the ICU and not due to the fact that intrathecal morphine was administered. Using a uniform dose also helps to formulate the timing and dosage of rescue analgesics.

Gall et al. (3) concluded that intrathecal morphine in a dose of 5 μg/kg can be a useful adjunct in the management of postoperative pain after spine surgery (very painful) for idiopathic scoliosis.

Hypospadias surgery in our institution is almost always performed as a day surgery procedure, which makes the use of intrathecal morphine not feasible.

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DOI: 10.1213/01.ane.0000278124.52700.c6

Etiology of Postoperative Visual Loss Not Always as Obvious as It Appears to Be

To the Editor:

A recent case report by Roth et al. (1) described unilateral central retinal artery occlusion in a patient in a prone position with a Dupaco Opti-Gard eye shield in place to "protect the eyes." Although inadvertent mechanical compression of the globe would seem to be the most likely etiology for blindness in this case, an alternative mechanism cannot be excluded. Optic nerve perfusion pressure is equal to the mean arterial pressure minus intraocular pressure (IOP) or venous drainage pressure, whichever is greater. There was only a modest decrease in mean arterial pressure reported during the procedure, but perfusion pressure could have been substantially decreased by changes in IOP. Direct pressure upon the globe is just one potential cause of dramatic increases in IOP.

Several studies have suggested that profound (as much as 10-fold) increases in IOP during general anesthesia in patients in the prone position even when the head is secured by pins and the eyes are presumably protected from mechanical compression (2,3). The absence of increased IOP postoperatively does not reliably exclude the possibility of significant intraoperative perturbations in IOP. Although less likely, the corneal abrasion and bruising noted postoperatively could be caused if a sedated patient were rubbing at a blind eye. External compression of the globe is an infrequent cause of visual loss for patients having surgery in the prone position (4). The risk factors for postoperative visual loss have only been partially identified and when these complications occur, the etiology often cannot be precisely identified.

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DOI: 10.1213/01.ane.0000278615.26380.71

In Response:

Drs. Heitz and Grunwald (1) are correct that inadvertent mechanical compression of the globe is the most likely etiology for unilateral blindness in our case report (2), and that the risk factors for postoperative visual loss have only been partially identified (3). However, in the context of the central retinal artery occlusion found in this patient, it is somewhat misleading to suggest that risk factors have only been partially identified. The mechanisms of central venous artery occlusion have been described in animal models (4–7). Although intraocular pressure (IOP) increases without external compression in prone-positioned patients, such increases are bilateral; thus, unilateral central venous artery occlusion is difficult to attribute to increased IOP alone in this setting (7–8). External compression is suggested by a number of findings in this patient. The constellation of symptoms and signs we described in this patient effectively negate Drs. Heitz and Grunwald's notion that increased IOP from prone positioning was responsible for central venous artery occlusion, accompanied by a "coincidental" corneal abrasion. An external eyelid injury shaped in the form of the edge of the eye protectors, a hazy cornea (probably due to compression and anterior chamber ischemia and edema), and a large (70% surface area) corneal abrasion (also probably due to widespread corneal hypoxia from compression) are beyond the typical signs and symptoms of postoperative corneal abrasion. Moreover, extraocular muscle dysfunction (exotropia) also suggests external compression. Explanations of the injury as anything other than external compression remain unlikely.

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DOI: 10.1213/01.ane.0000278622.30303.f1

Pneumocephalus and Headache After Epidural Analgesia: Should We *Really* Still Be Using Air?

To the Editor:

A recent case report describes pneumocephalus with headache after loss of resistance to air technique during a combined spinal epidural for labor analgesia (1). Nafiu and Urquhart (2) recently described a similar case following an unidentified dural puncture in a parturient.

The loss of resistance to air technique in epidural analgesia is associated with more complications including a greater rate of dural puncture rate, patchy block, and pneumocephalus (3). The current controversy between whether saline or air is the better medium for identifying the subdural space leads us to believe that it may be time to abandon the loss of resistance to air technique (3).

We are unaware of any study showing that using air is superior to using saline. Valickovic et al. suggest that pneumocephalus following loss of resistance to air is a rare complication; yet, this simply reflects the paucity of studies addressing this issue. The best data to date are from Aida et al. (4) in nonobstetric patients showing, via computerized tomography, evidence of pneumocephalus in over 80% of patients with either clear or occult meningeal perforation. No patient with headache following use of loss of resistance to saline developed pneumocephalus.

Aida et al. also propose a passive "wait and see" management of pneumocephalus. Other "proactive" articles (2,5) recommend administration of 100% oxygen to the patient to expedite resorption of the air bubble. Our main concern with passive management of pneumocephalus is that should the patient undergo a nitrous oxide based general anesthetic while an air bubble is present; there is increased potential for expansion of the air bubble with resultant tension pneumocephalus (5).

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