

Asystole during Vitrectomy Secondary to Increasing Intraocular Infusion Pressure Transmitted via Sclerotomy Infusion Cannula

Brandon A Van Noord^{1*} and Manxu Zhao²

¹Department of Anesthesiology, Keck Medical Center of the University of Southern California, Los Angeles, CA, USA

²Department of Anesthesiology, Cedars-Sinai Medical Center, Los Angeles, CA, USA

Abstract

The case of a 61 year-old male with recurrent retinal detachment who experienced a systole after increasing intraocular pressure through the sclerotomy infusion cannula during vitrectomy is reported. While the oculocardiac reflex is well-known, this case is unique as neither direct ocular compression nor extraocular muscle traction was present when the incident occurred. When any structure innervated by the trigeminal nerve is manipulated, vigilance should be maintained as this potentially fatal reflex can successfully be treated and prevented.

Introduction

Oculocardiac reflex (OCR) stimulation results in a parasympathetic response after extraocular muscle (EOM) traction or ocular compression. The afferent loop of the OCR pathway involves the ophthalmic division of the trigeminal nerve with the efferent loop as the vagus nerve [1]. OCR incidence is difficult to establish as the definition is inconsistent and patient susceptibility varies according to surgical and anesthetic technique. The OCR has been reported in up to 90% of pediatric strabismus surgeries as pediatric vagal tone is higher and the EOMs are directly manipulated [Gupta]. Asystole secondary to the OCR is rare: a study of 822 ophthalmic operations performed under general anesthesia documented an incidence of 0.24% [2].

Case Report

A 61 year-old male underwent right eye vitrectomy with endolaser photocoagulation and gas tamponade. Pre-operatively, the patient's blood pressure was well controlled with benazepril, he had good exercise tolerance, and his electrocardiogram was normal. One year prior to surgery, the patient underwent uncomplicated general anesthesia for cataract extraction, pars plana vitrectomy, and scleral buckle placement in the same eye.

The patient received 2 mg midazolam and 0.2 mg glycopyrrolate IV prior to surgery. After sedation, the ophthalmologists placed a retrobulbar block. Anesthesia was induced with 100 mg lidocaine, 100 mcg fentanyl, 200 mg propofol, and 50 mg rocuronium IV. Subsequent to endotracheal intubation, anesthesia was maintained with isoflurane. Initially, the patient was hemodynamically stable with heart rate, blood pressure, and oxygen saturation within normal limits. An hour after induction, paralysis resolved and the patient was spontaneously breathing at 9-12 bpm with tidal volume 438-466 ml and end-tidal CO₂ 43-50 mmHg. 83 minutes after incision, the high pressure alarm on the infusion device began to sound. Subsequent to increasing intraocular pressure through the infusion cannula, the patient experienced asystole. There was no direct ocular compression or EOM traction immediately prior to asystole. The ophthalmologist was immediately instructed to stop the procedure, the infusion pressure decreased, and asystole resolved spontaneously after 30 seconds without intervention.

During asystole, the SpO₂ and ETCO₂ began to drop and the waveforms became flat. After cardiac activity resumed, ETCO₂ was elevated and the patient coughed/bucked, likely stimulated by CO₂ retention. Patient bucking/coughing ultimately dislodged the infusion cannula, displaced the infusion to the suprachoroidal space, and created a choroidal detachment. The ophthalmologist successfully treated the de-

tachment by changing the infusion site and draining the effusion while removing the cannula. The choroidal detachment did not recur and the patient remained hemodynamically stable throughout the remainder of the case. The heart rate range after induction and prior to asystole was 69-82. There were no episodes of bradycardia either prior to or subsequent to asystole. This case report met IRB exemption criteria.

Discussion

OCR manifestations include bradycardia, sinoatrial block, bigeminy, atrioventricular nodal escape, wandering pacemaker, and syncope [3]. While symptoms are generally mild and transient, untreated OCR has been fatal [4,5].

In this case, scleral distension secondary to increasing ocular pressure likely stimulated the long and short ciliary nerves. The afferent stimulus was carried to the visceral motor nucleus of the vagus in the brainstem reticular formation via the ciliary and geniculate ganglia. From there, the efferent pathway proceeded along the vagus nerve via the cardiovascular center of the medulla to the SA node (Ripart). The OCR can be thought of more broadly as part of the trigeminocardiac reflex as the maxillary and mandibular divisions of the trigeminal nerve can serve as alternative afferent reflex pathways. Manipulation of any central or peripheral structure innervated by the trigeminal nerve can result in a vagally mediated, parasympathetic response. Published examples include asystole after puncture of the cavernous sinus during embolization [7], bradycardia following orbital floor fractures without EOM entrapment [8], complete atrioventricular block after zygomatic fracture [9], and repeated asystole during mandibular advancement surgery [1].

OCR risk factors include hypercarbia, hypoxemia, light anesthesia, and the strength and duration of the inciting stimulus. Medications such as beta blockers, calcium channel blockers, and narcotics can also

*Corresponding author: Department of Anesthesiology, University of Southern California, USA, Tel: 626-354-3412; E-mail: brandon.vannoord@gmail.com

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increase OCR likelihood through sympathetic inhibition and vagal augmentation [1]. Prevention strategies that have been documented in prospective, randomized trials include anticholinergic pretreatment and regional anesthesia. Bradycardia incidence was reduced from 70% to 10% in strabismus surgery in patients aged 2-30 following IV atropine pretreatment [10]. As atropine may precipitate more dangerous arrhythmias than the OCR itself, prophylaxis is typically reserved for pediatric patients. In pediatric strabismus surgery, bradycardia incidence decreased from 94% to 13% when regional anesthesia was added to general anesthesia alone [11]. The OCR is typically managed by releasing ocular pressure, increasing anesthetic depth, and rescue atropine. If the OCR results in prolonged asystole, then chest compressions may be required for medication circulation. The OCR typically fatigues.

Volatile anesthetics (VA) appear to inhibit the OCR more successfully than propofol. In a meta-analysis of pediatric strabismus surgery patients, OCR incidence was 21.5% with VA and 49% with propofol [12]. Lower BIS values directly correlated with lower OCR incidence during sevoflurane general anesthesia in a prospective trial [13]. In adults undergoing strabismus surgery, while a trend of decreased OCR incidence was observed with VA, it was not statistically significant [14]. OCR incidence was 12.5% during isoflurane and 18.5% during propofol anesthesia (OR 1.5, 16.8 NNH). In a prospective study, rocuronium also decreased OCR frequency during pediatric strabismus surgery (29% rocuronium vs. 53% succinylcholine vs. 44% control, $p=0.049$) [15].

Conclusion

The case of a 61 year-old male with recurrent retinal detachment who experienced asystole after increasing intraocular pressure through the sclerotomy infusion cannula during vitrectomy is presented. This unique oculocardiac reflex presentation occurred despite no extraocular muscle traction or direct ocular compression at the time of asystole. A retrobulbar block was placed, the patient received glycopyrrolate pretreatment, anesthesia was maintained with volatile anesthetics, and asystole resolved spontaneously after cessation of the eliciting stimulus. However, this case highlights the need to maintain muscle relaxation, appropriate anesthetic depth, and vigilance in situations and cases not typically associated with the oculocardiac reflex. This is important as steps may be taken to prevent this potentially fatal reflex.

References

1. Lang S, Lanigan DT, van der Wal M (1991) Trigemino-cardiac reflexes: maxillary and mandibular variants of the oculocardiac reflex. *Can J Anaesth* 6: 757-760.
2. Galkowska I, Nizienska E (1975) Cardiac arrest during ophthalmological operations. *Anaesth Resusc Intensive Ther* 3: 197-200.
3. Moonie GT, Rees DL, Elton D (1964) The oculocardiac reflex during strabismus surgery. *Can Anaesth Soc J* 11: 621-623.
4. Sorenson EJ, Gilmore JE (1956) Cardiac arrest during strabismus surgery. *Am J Ophthalmol* 41: 748-752.
5. Bietti OB (1966) Problems of anesthesia in strabismus surgery. *Int Ophthalmol Clin* 6: 727-737.
6. Ripart J, Mehri K, Della Rocca R (2012) Local & Regional Anesthesia for Eye Surgery. *The New York School of Regional Anesthesia*.
7. Amiridze N, Zoarski G, Darwish R, Obuchowski A, Solovevchic N (2009) Embolization of a Cavernous Sinus Dural Arteriovenous Fistula with Onyx via Direct Puncture of the Cavernous Sinus through the Superior Orbital Fissure: Asystole Resulting from the Trigemino-cardiac reflex. A Case Report. *Interv Neuroradiol* 15: 179-184.
8. Joseph JM, Rosenberg C, Zoumalan CI, Zoumalan RA, White WM et al. (2009) Oculocardiac reflex associated with a large orbital floor fracture. *Ophthalm Plast Reconstr Surg* 25: 496-498.
9. Kosaka M, Asamura S, Kamiishi H (2000) Oculocardiac reflex induced by zygomatic fracture; a case report. *J Craniomaxillofac Surg* 28: 106-109.
10. Gilani SM, Jamil M, Akbar F, Jehangir R (2005) Anticholinergic premedication for prevention of oculocardiac reflex during squint surgery. *J Ayub Med Coll Abbottabad* 17: 57-59.
11. Gupta N, Kumar R, Kumar S, Sehgal R, Sharma KR (2007) A prospective randomized double blind study to evaluate the effect of peribulbar block or topical application of local anaesthesia combined with general anaesthesia on intra-operative and postoperative complications during paediatric strabismus surgery. *Anaesthesia* 62: 1110-1113.
12. Tramer M, Moore A, McQuay H (1995) Prevention of vomiting after pediatric strabismus surgery: a systematic review using the numbers-needed-to-treat method. *Br J Anaesth* 75: 556-561.
13. Yi C, Jee D (2008) Influence of the anaesthetic depth on the inhibition of the oculocardiac reflex during sevoflurane anaesthesia for paediatric strabismus surgery. *Br J Anaesth* 101: 234-238.
14. Tramer MR, Fuchs-Buder T, Sansonetti A, Rifat K (1997) Low incidence of the oculocardiac reflex and postoperative nausea and vomiting in adults undergoing strabismus surgery. *Can J Anaesth* 44: 830-835.
15. Karanovic N, Jukic M, Carev M, Kardum G, Dogas Z (2004) Rocuronium attenuates oculocardiac reflex during squint surgery in children anesthetized with halothane and nitrous oxide. *Acta Anaesthesiol Scand* 48: 1301-1305.