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Oculocardiac Reflex Caused by Midface Disimpaction

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The oculocardiac reflex has been reported to cause bradycardia. The following case report describes a possible instance of the oculocardiac reflex occurring with repair of midface impaction.

REPORT OF A CASE

A healthy, 70-kg, 22-year-old man was admitted six days prior to operation following trauma to the head and face caused by blows inflicted with a lead pipe. Injuries included basilar skull fracture with cerebrospinal fluid otorrhea and rhinorrhea and a mild cerebral concussion. The neurologic abnormalities had resolved prior to operation. Also present were extensive facial fractures, scheduled for correction. These included a mandibular fracture, right zygomatic fracture, intraorbital fractures (including a blowout fracture of the right orbital floor), comminuted fracture of the right maxillary sinus, fracture of the left maxillary sinus, and a nasoethmoid complex fracture with a posteriorly displaced midface.

After premedication with diazepam, 15 mg, administered orally at 8:00 AM, anesthesia was induced 70 minutes later with thiopental, 300 mg, and succinylcholine, iv. The trachea was easily intubated with an 8.0-mm orotracheal tube and anesthesia was maintained with halothane, nitrous oxide, and oxygen. The patient received no atropine or other anticholinergic drug for premedication or induction. Monitors included an electrocardioscope with digital readout of heart rate. A tracheostomy was performed and the anesthetic administered via a tracheostomy tube with spontaneous ventilation. The second part of the procedure, open reduction and internal fixation of the facial fractures, was then begun. About 1 hour, 45 minutes after induction, the Rowe impaction forceps were inserted into the nasal cavity and mouth to grasp the posteriorly displaced maxilla and pull it forward. At this time the patient's pulse rate was 90/min. It had been stable since induction of anesthesia. With application of anteriorly and caudally directed force to the maxilla, sinus bradycardia to 54/min abruptly developed. Blood pressure, which had been 98 torr systolic, fell to 90 torr. When traction was released after about 20 seconds the heart rate rapidly returned to normal and stabilized at about 86/min. The remainder of the anesthetic course was uneventful.

DISCUSSION

The oculocardiac reflex has been reported to occur with pressure on the globe,[1–3] retrobulbar block,[1] pressure due to post-enucleation hemorrhage,[4] intraorbital hematoma,[5] pressure applied in the orbit after enucleation,[2] and massage of the eye immediately after retrobulbar block,[1] as well as manipulation of the extraocular muscles.[1,2,6] The event in this case was similar, but a maneuver not directly involving the eye probably was the cause of the unexpected oculocardiac reflex. During reduction, the retrodisplaced midface was pulled anteriorly, putting traction on the floor and medial aspect of the orbit as well as the maxilla. This, in turn, undoubtedly put tension on the orbital contents and stimulated the terminal branches of the trigeminal nerve.

The afferent limb of the oculocardiac reflex is via branches of the ophthalmic division of the trigeminal nerve in the orbit to the gasserian ganglion. The vagus nerve provides the efferent limb to the heart, producing bradycardia by increasing parasympathetic tone. Other arrhythmias may be seen due to the autonomic imbalance that results from the increased vagal tone.[3,7,8]

The reflex may be prevented with intravenous administration of atropine[6,7] or retrobulbar block.[2,9] However, intramuscular administration of atropine in usual doses has been shown to be ineffective.[1,7] Because of the brief, nonrecurrent nature of the oculocardiac reflex in this case, the bradycardia was not treated.

The oculocardiac reflex is known to be a potential hazard of ophthalmic surgery. That this may also occur with reduction of facial fractures is less well known. The oculocardiac reflex should be recognized as a potential hazard of midface disimpaction.

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