Bradycardia during cold ocular irrigation under general anaesthesia: an example of the diving reflex

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A case of bradycardia is reported which was precipitated by cold normal saline applied to the eye during general anaesthesia. The history and physiology of the diving reflex is discussed and we believe that these data suggest that this patient's bradycardia was induced by the diving reflex, and not by the oculocardiac reflex.

Nous rapportons un cas bradycardie provoquée par l'application oculaire de soluté physiologique froid. La discussion porte sur l'histoire et la physiologie du réflexe de plongée. Nous croyons que ces données supportent notre hypothèse d'un bradycardie provoquée par le réflexe de plongée et non par le réflexe oculocardiaque.

The oculocardiac reflex, well-described in the anaesthesia literature, results from a variety of stimuli including traction on the extraocular muscles, ocular manipulation, pressure or traction on the globe, or pressure applied to an empty orbit.[1-5] However, even in the absence of these stimuli, cold ocular irrigation may elicit bradycardia induced by the diving reflex. The oculocardiac reflex is active in awake and anaesthetized subjects and is manifested by a variety of dysrhythmias, which include bradycardia, bigeminy, ectopic beats, nodal rhythm, and asystole.[6-8] The diving reflex may have similar physiological mechanisms and clinical significance.

Key words
HEART: arrhythmia;
BRADYCARDIA: diving reflex.

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Case report

A 60-yr-old woman was scheduled for left scleral buckle. Her past medical history was unremarkable, with no history of cardiovascular, neurological, renal, haematopoetic, endocrinological, or pulmonary disease. She did not smoke or use alcohol or take medications. Previously she had undergone two gynaecological operations during uncomplicated general anaesthesia. Physical examination was normal and there was no syncopal history. The preoperative laboratory results were normal, with haemoglobin and haematocrit 13.7 gm · dl^{-1} and 39.7%, respectively. The preoperative 12-lead electrocardiograph (ECG) showed normal sinus rhythm with no suggestion of ischaemia. The vital signs the day before surgery were blood pressure 140/80 mmHg, pulse 88 beats · min^{-1} and respirations 18 breaths · min^{-1}. She weighed 55.7 kg and was 155 cm tall. Sixty minutes before her operation, the patient received diazepam 10 mg *po*, and a peripheral *iv* infusion of lactated Ringer's solution with 5% dextrose.

Intraoperative monitoring included a five-lead continuous ECG which displayed limb lead II. After preoxygenation, anaesthesia was induced with thiopentone 275 mg and lidocaine 100 mg *iv*. Tracheal intubation was facilitated with succinylcholine 80 mg *iv*, and was easily accomplished. Anaesthesia was maintained with 1% isoflurane and 50% nitrous oxide in oxygen. Ventilation was controlled so that end-tidal carbon dioxide concentration was 36 mmHg. Relaxation was maintained with 5 mg *iv* vecuronium bromide. Vital signs did not vary by more than 5% during anaesthetic induction and maintenance up to this point.

Fifteen minutes after induction, a watertight drape was placed around the left eye and the surgeon began to irrigate the eye liberally with sterile, room-temperature, normal saline. The temperature of the saline was 19.7°C. The heart rate promptly decreased from 78 beats · min^{-1} with normal sinus rhythm to a sinus bradycardia of 30 beats · min^{-1}. The surgeon stopped the irrigation and atropine 0.4 mg was given *iv*. The heart rate increased to 90 beats · min^{-1} with normal sinus rhythm. The episode of bradycardia was short-lived (lasting approximately 20 sec), and quickly returned to an acceptable rate with atropine administration and cessation of irrigation. During the episode, no ocular stimulus was present other than application of cold saline; the surgeon had placed no retractors, instruments, retracted the eye muscles or placed pressure on the eye. Pulse oximetry revealed that the patient was not hypoxic during the episode, neither did she become hypotensive, the blood pressure remaining at 110/80 as measured by auscultation before, during and after the episode. After the heart rate stabilized, the surgeon proceeded cautiously with ocular irrigation which caused no further change in vital signs. We were unable to record a rhythm strip during the episode. The drape was found to form a watertight seal, preventing the irrigation solution escaping from the field.

The remainder of anaesthesia was uneventful. The vital signs remained stable. At the end of the procedure muscle relaxation was reversed with glycopyrrolate and neostigmine with no change in heart rate. Emergence was uncomplicated and the tracheal tube was removed when the patient opened her eye. She was transferred to the recovery room and discharged when alert and awake. The postoperative visit the next day was unremarkable except for a mild sore throat.

Discussion

This case is an example of the diving reflex in an adult during general anaesthesia. Classically the diving reflex is described as bradycardia without hypotension during the application of cold irrigants to the distribution of the ophthalmic division of the fifth cranial nerve. Bradycardia is mediated by the vagus nerve.[9,10] This reflex appears to be more prominent in children than in adults and has been used in both awake adults and children to treat refractory supraventricular tachycardia.[11-14] It is a primal reflex and appears to be present in most vertebrates. It is more potent than a Valsalva manoeuvre, carotid massage or ocular compression in inducing vagally mediated bradycardia.[15]

The diving reflex was first described by Bert in 1879 while investigating duck asphyxiation. He found that water applied to the ducks' eyes and nostrils produced bradycardia, and ducks tolerated underwater asphyxiation longer than chickens.[5,17] In 1894, Richet concluded that the duck's ability to tolerate long periods underwater was a reflex physiological adaptation, conserving available oxygen stores and allowing the animal to tolerate long periods without oxygen. He speculated that blood was preferentially shunted to more oxygen-dependent organs.[16]

The diving reflex was characterized in diving and non-diving animals years after the initial work of Bert and Richet. The diving reflex is present in man, seals, and dogs. It is a highly integrated reflex consisting of initiating events which temporally summate to protect against asphyxiation under water.[10,16,18] Clinically, the reflex has been induced using ice water. However, in man, the reflex is also initiated by facial application of 0–20°C cold saline.[19] In our patient, the temperature was 19.7°C and fell within the 15–20°C range, the most frequently studied temperature range.[20,21] Further general anaesthesia does not abolish the reflex in dogs and seals,[22] but *im* or *iv* anticholinergic agents may block or antagonize the reflex.[9] In seals, and perhaps in man, afferent information from the carotid body and sinus, mediated by the glossopharyngeal nerve, is important in sustaining the later portions of the reflex which includes vasoconstriction and apnea.[22,23]

Both the parasympathetic and sympathetic nervous systems are integral parts of the diving reflex. In dogs, bradycardia results from increased vagal tone,[24,25] as the reflex is abolished with atropine. The sympathetic nervous system causes intense peripheral vasoconstriction which diverts blood flow from nonvital organs and maintains cardiac output, blood pressure and venous tone.[23,24] Blood flow to the kidneys, muscles, and gut is minimized. Cardiac blood flow and cardiac output may decrease by 90% but the reflex protects blood flow to the heart, lungs, and brain.[26] The diving reflex begins when temperature receptors in the ophthalmic division of cranial nerve five are excited by cold water.[26] The sensory information proceeds to the sensory nucleus of the fifth cranial nerve and then to the vagal motor nucleus.[26] Fifth cranial nerve sensory input also enters the brainstem cardioinhibitory centre and mediates the sympathetic response.[26] The vagal motor nucleus and the cardioinhibitory centre also communicate with the respiratory centre.[26] This produces a more intense reflex during exhalation.[19,28] Only the brain maintains a normal or increased blood supply.[9,27]

The diving reflex may have clinical importance. Death from arrhythmias due to the oculocardiac reflex are well known.[6] The diving reflex is mediated by similar reflex arcs. In awake humans, the diving reflex appears to be more intense than the oculocardiac reflex.[15,21] Bradycardia during the diving reflex is more profound than the maximal bradycardia of the oculocardiac reflex.[15,21] The diving reflex also involves intense sympathetic nervous system activity altering myocardial repolarization as evidenced by T-wave and ST segment changes.[29] The combination of slowed myocardial conduction, altered ventricular repolarization, and the propensity of the oculocardiac reflex to produce life-threatening arrhythmias implies that the diving reflex also may precipitate a potentially dangerous situation.[29,30] The risk may be compounded in the elderly ophthalmology patient with multiple medical problems. In experienced pearl divers, two cases of idioventricular rhythms and other dysrhythmias during routine dives were attributed to the diving reflex.[30] Wolf suggested that the diving reflex may be important as a possible mechanism of sudden death.[31] The diving reflex may cause death in the immersion syndrome, but also enhances survival during long periods of cold-water immersion.[32]

The oculocardiac reflex and diving reflex are separate reflexes. Examination, however, reveals many similarities. Both reflexes are initiated by stimuli delivered in the distribution in the ophthalmic division of the fifth cranial nerve, are more intense during expiration, are mediated partially by the vagus nerve, and initiate physiological mechanisms to conserve oxygen.[24] A variant of the diving reflex is even present in some fish to prevent asphyxiation in air.[33] The diving reflex initiates bradycardia by sensing cold water, while the oculocardiac reflex initiates bradycardia by sensing pressure. The diving reflex may not be uncommon during anaesthesia but it is reported as the oculocardiac reflex because of a temporal relationship between the use of cold ocular irrigants and ocular manipulation. These reflexes, which may be evolutionary reflexes to protect against drowning, perhaps should be called "asphyxial protective reflexes." In mammals, the reflexes may prevent birth asphyxiation and explain their prominence in infants and children.[17]

Conclusion

This case demonstrates a possible diving reflex during general anaesthesia in a healthy, elderly woman. The clinical presentation was consistent with the diving reflex; none of the inciting stimuli required by the oculocardiac reflex were present. The response was classic: bradycardia without hypotension shortly after initiation of eye irrigation with cold 19.5°C saline. Irrigants of this temperature has produced bradycardia in awake humans and in diving and non-diving animal models during general anaesthesia.[2,22,25,29] The bradycardia was reversed by administering *iv* atropine and discontinuing the eye irrigation. The atropine prevented bradycardia recurrence.

The diving reflex is uncommon during ophthalmological surgery.[10] In the elderly patient the diving reflex may present a similar risk to the patient as the oculocardiac reflex as they share similar pathways and physiological mechanisms. It is prudent to prevent the diving reflex by avoiding cold ocular irrigants.[10] However, we do not recommend the routine use of anticholinergics because of the risk of tachycardia.

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