Iatrogenic Oculocardiac Reflex in a Patient with Head Injury

Panagiotis Stathopoulos, MD, DDS, PhD¹ Michael Mezitis, DDS¹ George Kostakis, MD, DDS, MSc, PhD¹
George Rallis, MD, DDS, PhD¹

¹ Department of Oral and Maxillofacial Surgery, General Hospital of Attica "K.A.T.," Kifissia, Athens, Greece

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Address for correspondence and reprint requests Panagiotis Stathopoulos, MD, DDS, PhD, Department of Oral and Maxillofacial Surgery, General Hospital of Attica "K.A.T.," 98, Tatoiou Street, Nea Erythrea, Attica, Greece 14671 (e-mail: pan_stath@yahoo.gr).

Abstract

A 16-year-old girl with a history of a recent fall from the third floor was transferred to the emergency room. On presentation, the patient, who had sustained multiple facial fractures, was in clinical shock with a blood pressure 80/40 mm Hg, heart rate 130/min, tachypnea (>30/min), Po₂ 50 mm Hg, and So₂ 82%, and she was intubated for airway protection. Severe nasal hemorrhage was detected (hematocrit: 22%), therefore a bilateral anteroposterior balloon nasal catheter was inserted and inflated with air. Bleeding was controlled. A few minutes later, her heart rate dropped to 40/min. Atropine was administered intravenously and the rate increased to 60/min. Computed tomography of the head revealed brain and subarachnoid hemorrhage, multiple fractures of the facial skeleton, and a round foreign body, full of air, compressing the left eye. The medial wall and the floor of the ipsilateral orbit were also fractured, establishing a naso-orbital communication. The left catheter was immediately removed. Heart rate was restored to normal. Facial fractures were addressed surgically. Patient's vision is intact.

Keywords

- oculocardiac reflex
- orbital wall fractures
- balloon nasal catheter

Several case reports and reviews describe the oculocardiac reflex in ophthalmologic, anesthetic, and maxillofacial surgery literature. The increase of the parasympathetic tone produced by pressure applied on the globe or orbital and periorbital tissues may lead to nausea, vomiting, bradycardia, and even asystole.^{1,2} The incidence of fatal cardiac arrhythmias in patients with an oculocardiac reflex is 1 per 3500.¹ The occurrence of the reflex during reduction of zygomatic³ and nasal fractures,⁴ midface disimpaction,⁵ orbital floor fractures,^{6,7} maxillary osteotomy,² and insufflation of the temporomandibular joint⁸ has been well reported. We describe a case of the oculocardiac reflex leading to severe bradycardia in a young patient after compression of the eye caused by a balloon nasal catheter inserted into the orbit as a result of a traumatic naso-orbital communication.

Case Report

A 16-year-old girl with a history of a recent fall from the third floor was transferred to the emergency room. On presentation, the patient was in clinical shock. Her blood pressure was 80/40 mm Hg, heart rate 130, and respiratory rhythm >30/min. Clinical examination revealed a left zygomatico-orbital complex fracture, comminuted nasoethmoidal fractures, and severe nasal hemorrhage (hematocrit: 22%). The patient's blood gas values were Po₂ 50 mm Hg and So₂ 82% due to upper airway obstruction, and therefore she was intubated for airway protection. After resuscitation, a bilateral anteroposterior balloon nasal catheter was inserted and inflated with air. Bleeding was controlled and the vital signs of the patient were restored to normal. Few minutes later, heart rate dropped to 40/min. Atropine was given intravenously and the rate increased to 60/min. Computed tomography of the head, which was performed

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Figure 1 Axial computed tomography of the head (bone window): balloon nasal catheter compressing the left eye.

15 minutes later, revealed brain and subarachnoid hemorrhage, multiple fractures of the facial skeleton, and a round foreign body, full of air, compressing the left eye (► **Fig. 1**). The medial wall and floor of the ipsilateral orbit were also fractured, establishing a naso-orbital communication (► **Fig. 2**). The left catheter was removed at once. Heart rhythm was restored to normal. Facial fractures of the patient were addressed surgically with open reduction and internal fixation 8 days later. Ophthalmologic examination disclosed that the patient's vision was intact.

Figure 2 Fracture of medial wall of orbit, with balloon nasal catheter compressing the left globe.

Discussion

Stimulation of the ophthalmic nerve, first division of the trigeminal, by compression of the eye leading to inhibition of heart rate due to excitation of the vagus nerve is believed to be the mechanism of the oculocardiac reflex.⁹ The afferent pathway commences with pressure receptors in the ocular and periocular tissues, follows the ciliary nerves to the gasserian ganglion along the ophthalmic nerve, and ends in the trigeminal sensory nucleus, located in the floor of the fourth ventricle. The impulses reach the visceral motor nuclei of the vagus nerve through the reticular formation. The efferent limb travels via the vagus nerve to the myocardium. Stimulation of the reflex arc results in negative inotropic and chronotropic effects.¹⁰

In our patient, sinus bradycardia developed after left eye compression owing to a balloon nasal catheter inserted and inflated with air. A pathological communication had been already established between the nasal cavity and the orbit as the result of the severe trauma sustained. As the lamina papyracea is the most vulnerable part of the medial orbital wall, most fractures occur around it.¹¹ It is most likely that the probe of the catheter passed through this deficit and when the balloon was inflated with air, the globe was compressed, generating the oculocardiac reflex. This hypothesis is further supported by the fact that when the catheter was removed, heart rate was restored to normal.

It has been reported that the force and type of the trigeminal nerve stimulation seem to affect the occurrence of the oculocardiac reflex. Interestingly enough, it is supported that the more acute the onset and the more powerful the pressure applied on the eye, the more likely the reflex is to appear.³ In our case, both the rapid onset and the intensity of the force produced inside the orbit by the nasal catheter seem to have activated the trigeminovagal reflex. It is also widely accepted that surgical intervention should be performed early in patients with severe and persistent oculocardiac reflex.^{12,13} As soon as a sudden bradycardia is noted, any compression applied on the eye should be immediately eliminated. Communication with the anesthetist and monitoring of the cardiac rhythm during maxillofacial surgery are essential measures for the detection and treatment of the oculocardiac reflex. Usually, as soon as the stimulus cessation is achieved, the patient will regain sinus rhythm. As the reflex is initiated by parasympathetic stimulation, the use of anticholinergic agents is logical. In the case presented, intravenous administration of atropine together with the nasal catheter removal resulted in restoration of the heart rate to normal. To the best of our knowledge, this is the first case in the English literature of an oculocardiac reflex occurrence as a result of a direct compression of the eye by a balloon nasal catheter. This case indicates that extreme care and vigilance should be demonstrated when a nasal catheter is inserted to control hemorrhage in a patient with head injury.

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