Anesthetic Management of the Trigemino-cardiac Reflex During Mesiodens Removal—A Case Report

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We describe a case in which reflection of a palatal flap for removal of a mesiodens is presented as the triggering factor for bradycardia caused by stimulation of the trigemino-cardiac reflex. The management of the case, as well as the reflex arc, is discussed.

Key Words: Trigemino-cardiac reflex; Bradycardia.

CASE REPORT

A 5-year-old white girl with a noncontributory medical history was seen for emergency dental treatment after falling at a skating party. Clinical examination revealed fractures of the maxillary central and lateral incisors. There was also a cast on the right leg because of a fracture sustained at the time of the dental trauma. There were no other injuries to the maxilla or mandible noted; however, there was an incidental finding of a mesiodens between the primary central incisors. Caries were also noted on the primary maxillary cuspids. Because of the presence of the mesiodens, general anesthesia was presented to the parents as an option for treatment so that this could be removed at the time of the extractions and restorations. The parents agreed and the patient was scheduled for general anesthesia.

Preoperative evaluation of the patient revealed no significant medical problems. The patient did have a history of necrotizing ulcerative colitis as an infant and the previously noted fracture of the right leg. The right leg was no longer in a cast and no orthopedic fixation was present. The patient did have a preoperative history and physical examination by a physician who did not find any system problems. On the day of the general anesthetic the patient weighed 24 kg. She was not taking any medication and had no drug allergies and no family history of anesthesia problems. Previous anesthesia for

casting of her right leg was without incident. A pre-anesthesia physical examination revealed a normal airway examination. Her chest was clear to auscultation bilaterally with a regular heart rhythm. Preoperative vital signs including heart rate, respiration rate, and blood pressure were within normal limits.

The patient was taken to the operating room and monitoring consisting of a pulse oximeter, noninvasive blood pressure, electrocardiogram, precordial stethoscope, and temperature probe was applied and activated. Smooth inhalation induction of anesthesia was accomplished with sevoflurane, nitrous oxide, and oxygen. Twenty micrograms of fentanyl was given and the patient was intubated with a 5.0 uncuffed nasal endotracheal tube. Intubation was easy under direct-vision laryngoscopy. The tube was secured and placement was confirmed by auscultation and the presence of sustained end tidal carbon dioxide. The eyes were taped, pressure points were padded, and the patient was turned over to the surgeon. Maintenance of anesthesia was with isoflurane, oxygen, and nitrous oxide. The heart rate (beats per minute) was in the 90s and systolic blood pressure was 90–100 mm Hg.

The caries on the canines were restored with composite and 0.9 mL of 2% lidocaine with 1:100,000 epinephrine was administered via infiltration by the surgeon. The maxillary primary central and lateral incisors were removed. As the surgeon started to reflect a palatal flap to gain access to the mesiodens, the patient experienced sinus bradycardia with a heart rate of 45 beats per minute. The electrocardiogram tracing did not appear to be a type of heart block. The blood pressure was 60/20. The surgeon was told to stop the procedure and atropine 0.4 mg was immediately given IV. The in-

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travenous fluids were opened all the way. The heart rate increased to 140 beats per minute and the systolic blood pressure increased to 110 mm Hg. The procedure continued without further complication. The mesiodens was removed, sutures were placed, and hemostasis was obtained. The patient was awakened and extubated without complication. There were no postoperative anesthetic complications and the patient was discharged to home later that day.

DISCUSSION

The trigeminocardiac reflex is a trigeminal-vagal arc that can result in a significant decrease in heart rate. It is theorized that the reflex mechanism is that sensory nerve endings of the trigeminal nerve send impulses via the Gasserian ganglion to the sensory nucleus of the trigeminal nerve. This forms the afferent pathway of the reflex arc. These neurons continue in the reticular formation to connect with the efferent pathway in the motor nucleus of the vagus nerve. Efferent fibers that are cardioinhibitory come from the motor nucleus of the vagus nerve and terminate in the myocardium.¹ Clinically, stimulation of the trigeminocardiac reflex can include sudden-onset sinus bradycardia, bradycardia terminating in asystole, asystole with no preceding bradycardia, and hypotension.

In dentistry, severe bradycardia has occurred during reduction of zygomatic arch fractures and manipulation of midface fractures. Asystole has been reported during downfracture of the maxilla during a Le Fort I osteotomy. Repositioning of the maxilla showed a return to sinus rhythm. Treatment with both atropine and glycopyrrolate allowed the downfracture to continue without further occurrence of a dysrhythmia.² The reflex has also been induced by a displaced zygomatic fracture and resolved when the fracture was reduced.³

The case presented is a unique example of the trigeminovagal reflex where the afferent impulses are caused by stimulation of the nasopalatine nerve and the efferent pathway is the vagus nerve. Because children are more prone to vagal stimulation, bradycardia should be a consideration when palatal flaps are reflected for procedures such as removal of a mesiodens or exposure of an impacted cuspid. This would especially be true if

sympathetic reflexes are blunted by the administration of general anesthesia or narcotics.

In this case the nasopalatine nerve was infiltrated to achieve local anesthesia, which should theoretically have blocked the response. However, there may have been insufficient local anesthetic used to prevent the reflex from occurring because of traction on the nerve.

A dose of 0.4 mg atropine was given to treat the bradycardia that was thought to be attributable to parasympathetic stimulation caused by reflection of the palatal flap. The recommended dose for atropine in children is 0.02 mg/kg, which would calculate to a dose of 0.48 mg in this child. Because atropine is not universally successful in preventing the bradycardia, the procedure should be halted immediately until there is a return of normal sinus rhythm.² In a child who has symptomatic bradycardia that does not respond to atropine, 0.01 mg/kg of epinephrine would be indicated. Atropine would be given before epinephrine only if, as in this case, the bradycardia was thought to be attributable to vagal stimulation and not some other cause such as hypoxia.⁴

CONCLUSIONS

A case of bradycardia thought to be attributed to the trigeminovagal reflex is presented. Management includes halting the procedure until normal sinus rhythm returns and administration of atropine and/or glycopyrrolate.

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