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Asystole and Bradycardia During Maxillofacial Surgery

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A Chinese female undergoing maxillary osteotomy developed asystole when the maxillary tuberosity was cut. Surgery was stopped. After about 10 sec and before instituting cardiac massage, sinus rhythm and bradycardia ensued. Atropine was administered intravenously, resulting in an increase in heart rate. No further episodes of asystole or bradycardia were encountered.

Asystole is a rare complication of maxillofacial surgery. We report a case of asystole during such an operation and review the literature on stimulation of the trigeminovagal reflex during extraorbital maxillofacial surgery.

CASE REPORT

A 35-yr-old, 47-kg, Chinese female presented for elective LeFort I and Hofer osteotomies. She had no history of previous anesthetics or any significant illnesses. Physical examination was normal. Preoperative investigations, including a chest X-ray, electrocardiogram (ECG), complete blood count, and blood biochemistry, were within normal limits.

For premedication, the patient received 15 mg of midazolam orally 1 hr preoperatively. On arrival in the anesthetic room, her blood pressure was 105/65 mm Hg and her pulse was 60 beats/min. Following 5 mg dexamethasone given intravenously to reduce postoperative swelling, anesthesia was induced with 50 μg fentanyl and 250 mg thiopental. Succinylcholine (75 mg) was given to facilitate intubation. Her nares were sprayed with a 4% cocaine solution, and nasoendotracheal intubation was carried out uneventfully. Anesthesia was maintained with 2% enflurane in a mixture of 40% oxygen and 60% nitrous oxide. Tubocurarine (20 mg) was administered for muscle relaxation, and the patient's lungs were mechanically ventilated. The ventilation was adjusted to maintain an end-tidal carbon dioxide concentration ($EtCO_2$) of 30 to 35 torr. The ECG, pulse oximetry, and $EtCO_2$ were measured continuously, and the blood pressure was measured noninvasively at 3-min intervals.

Surgery of the mandible commenced 25 min after induction of anesthesia. Three boluses of labetalol, totaling 40 mg, were given at this time to induce a hypotensive blood pressure of 70/50 mm Hg. A further 25 μg of fentanyl was injected 1 hr after induction.

Surgery of the maxilla began 1.5 hr after induction of anesthesia. During the next 30 min the patient's pulse and blood pressure remained stable with a heart rate around 60 beats/min and a blood pressure of about 70/50 mm Hg. At this time the surgeon, using a 10-mm straight osteotome, began to cut the right maxillary tuberosity at an oblique angle via a transoral route. A sudden asystole with a flat ECG and oximetry waveform was detected. The surgeon was informed immediately, and the surgical stimulus was stopped. The inspiratory oxygen concentration was changed to 100%, and the enflurane vaporizer was switched off. After approximately 10 sec of asystole, a sinus bradycardia developed. Atropine (0.6 mg) was given intravenously, after which the heart rate rapidly increased to 104 beats/min and the blood pressure increased to 104/65 mm Hg. Nitrous oxide and enflurane were restarted, and the operation was completed uneventfully. The pulse remained stable at 80 to 96 beats/min. The same osteotomy technique was carried out on the left side uneventfully. At the end of the procedure, muscle relaxation was reversed with 2.5 mg neostigmine and 0.6 mg atropine. The patient woke up 20 min after surgery had finished and recovered uneventfully.

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DISCUSSION

Bradycardia during facial surgery has been described on several occasions for different operations of the head and neck.[1–14] The cause of the bradycardia is considered by most commentators to be due to stimulation of structures innervated by the trigeminal nerve, a mechanism that is well documented in eye surgery, where it is known as the oculocardiac reflex. The terms *trigeminocardiac* and *trigeminovagal reflex*[2,3] have been proposed to include the additional, nonocular sources of this reflex.

Reflex Pathway

The afferent pathway of the trigeminovagal reflex is via one of the branches of the trigeminal nerve. The pathway continues to the Gasserian ganglion and then to the main sensory nucleus of the trigeminal nerve in the floor of the fourth ventricle. In the reticular formation, short internuncial fibers connect to the efferent pathway, which originates in the motor nucleus of the vagus nerve. Cardioinhibitory efferent fibers arising from the motor nucleus of the vagus nerve are carried by that nerve to end in the myocardium.

Sites of Stimulation

Maxillary osteotomy can obviously activate the trigeminovagal reflex. In the case reported here, stimulation occurred when one of the maxillary tuberosities was being sectioned. The afferent pathway was probably via the posterior superior alveolar nerve or the greater palatine nerve of the maxillary division of the trigeminal nerve (Figure 1). Several other episodes of stimulation have been reported during downfracture of the maxilla.[4–6] Complete mobilization and advancement of the maxilla causes traction on the maxillary soft-tissue pedicle containing the palatine neurovascular bundles, which may stimulate this reflex. The trigeminovagal reflex can also be activated during:

(1) midface disimpaction[1] and elevation of zygomatic arch fractures[7–9,14];
(2) insufflation of the temporomandibular joint for temporomandibular arthroscopy[10];
(3) the use of a mouth prop to mobilize an ankylosed temporomandibular joint, or a ramus stripper to detach muscular tendon fibers from the anterior portion of the coronoid process in an ankylosed temporomandibular joint[6];
(4) the use of a channel retractor subperiosteally along the medial aspect of the mandibular ascending ramus[4]; and
(5) procedures on structures related to the nose, namely dissection over the frontonasal region for access to LeFort II and nasoethmoidal fractures and traction on the nasal portion of the bitemporal flap.[13]

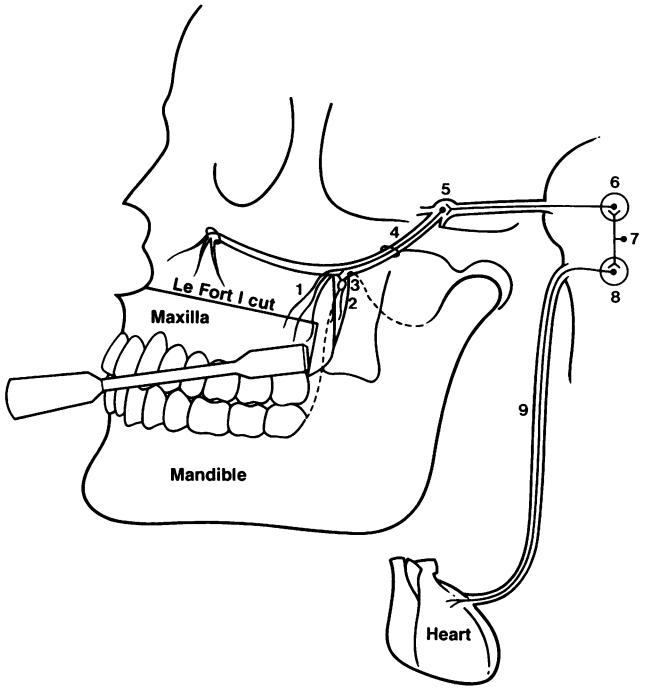

Figure 1. Probable pathway of the trigeminovagal reflex in this patient. **1,** Posterior superior alveolar nerve; **2,** palatine nerve; **3,** sphenopalatine ganglion; **4,** maxillary nerve; **5,** trigeminal ganglion; **6,** sensory nucleus of the trigeminal nerve; **7,** internuncial fibers; **8,** motor nucleus of the vagus nerve; **9,** vagal branch to the heart.

However, simple rotational disimpaction of the maxilla using Tessier spreaders appears not to activate the trigeminovagal reflex.[6]

Effects

Activation of the trigeminovagal reflex has been sudden and unexpected. It has resulted in bradycardia,[1,3,4,7,9,12,13] bradycardia terminating in asystole,[4,6–8,11] and asystole with no preceding bradycardia.[4,5,14] Asystole has been followed by bradycardia and has reverted directly to sinus rhythm.

Morbidity and Mortality

In several cases the simple stoppage of surgery resulted in the recovery of a normal rhythm.[1,4–7,9,12] In other instances, anticholinergic drugs were used to increase the heart rate.[3–8,10,12] Some cases have been reported in which patients had recurrent bradycardia for a prolonged period despite injection of anticholinergic drugs.[4,10,12] One operation was halted due to a recurrent severe bra-

dycardia.[13] There is also one reported death probably following this reflex.[9]

Predisposing Factors

A number of factors have been postulated to predispose patients to the trigeminovagal reflex. High sympathetic activity with parasympathetic stimulation, known as "accentuated antagonism," can produce a severe bradycardia by eliciting a variety of negative chronotropic and inotropic cardiac responses.[6] Hypoxemia, hypercarbia, and light anesthesia may aid in precipitating the reflex by elevating the background sympathetic tone. However, in the majority of cases reported there is no evidence of high sympathetic activity at the time of reflex precipitation.[1,3–12]

Hypercarbia may accompany spontaneous ventilation during general anesthesia. In eye surgery, a high $PaCO_2$ more than doubled the percentage of significant bradycardia in one series. Surprisingly, a prospective trial of controlled ventilation also indicated a small increase in the incidence of oculocardiac reflex.[15] Modern neuromuscular blocking agents have been implicated in facilitating bradycardic responses to surgery due to their lack of a protective action on the heart.[16] The majority of cases reported have involved controlled ventilation.

Opioids can make the vagal tone the predominant tone. It has been suggested that the use of opiods, in particular fentanyl, may foster initiation of the oculocardiac reflex.[3,4,11,17] Alfentanil, like fentanyl, also has been reported to suppress the stress response and make the vagal tone dominant.[17]

In one series the trigeminovagal reflex was reported more frequently in men than in women,[6] though the number of osteotomies performed has been more in women. The incidence of oculocardiac reflex is reported to be high in children,[18] probably because of their higher resting vagal tone. However, possibly due to the small number of children undergoing maxillofacial surgery, the trigeminovagal reflex has not been reported in children undergoing such treatment.

β-Adrenergic blockers may accentuate the reflex. One patient who developed bradycardia via the trigeminovagal reflex was on nadolol and developed repeated episodes of bradycardia.[4]

Prevention

A preoperative dose of intramuscular atropine or glycopyrrolate has proved to be unreliable in preventing the oculocardiac reflex and similarly may not prevent the trigeminovagal reflex.[19] Intravenous atropine and glycopyrrolate have been shown to be effective in abolishing the oculocardiac reflex in children.[19] Thus, many investigators have suggested the use of an intravenous anticholinergic drug at induction.[1,3,5,8–12,14] However, much of the surgery done in the maxillofacial region is of greater duration than ophthalmic surgery, and the protection afforded by the anticholinergic may be lost as its blood concentration falls during the course of the operation. A case has been reported of bradycardia during elevation of the zygomatic arch where intravenous atropine had been given at induction.[9] Further, the use of anticholinergic drugs may cause other dysrhythmias during maxillofacial surgery.[20] Hypotensive anesthesia has been frequently used during maxillofacial surgery. Due to the resultant increase in heart rate, many clinicians may prefer to avoid giving anticholinergics at induction.

Conduction nerve blockade has been found to be protective against the oculocardiac reflex.[21] Local anesthetics with epinephrine are frequently used during osteotomy surgery to decrease hemorrhage, to improve the quality of the surgical field, and to prevent blood loss. Local anesthetics may be infiltrated to provide pain control and improve recovery. In addition, some use it specifically to protect against the trigeminovagal reflex.

Treatment

Monitoring and observation of the cardiac rhythm during orthognathic surgery is of prime importance in the detection and treatment of the resulting bradycardia or asystole. As soon as sudden bradycardia or asystole is detected, the surgery should be stopped immediately and any traction on soft tissues released. Usually cessation of the stimulus will enable the patient to regain a sinus rhythm. Often it is all that is necessary. Anticholinergics are nonetheless frequently given; their use is logical, as the reflex is precipitated by parasympathetic stimulation, and the drugs may prevent further episodes of the reflex. When used, time should be allowed for the drug to take effect. At least one recurrence was ascribed to the fact that the drug had not had time to act before a fresh stimulus was given.

Cardiac massage should be reserved for cases where conservative measures have failed to reestablish cardiac output, as there is serious morbidity associated with it.[22] Cardiac massage has been reported twice in the treatment of asystole associated with the trigeminovagal reflex.[6]

Treating the initial episode with anticholinergics does not guarantee protection from further episodes. Even after treatment of the initial bradycardia one should be vigilant for further episodes of bradycardia, as they have been shown to recur with further stimulation. Additional anticholinergics may be necessary to treat these episodes. If the bradycardia recurs, local anesthetic blockade of the nerve concerned may stop these episodes.

Maxillofacial surgery is becoming increasingly popular. Bradycardia reported during this type of surgery should be taken seriously, as there is at least one anecdotal report of a death due to this reflex during facial surgery and, in one report alone, at least 60 deaths due to the oculocardiac reflex.[23] By continuous and meticulous monitoring of the ECG and pulse waveform, by knowing the procedures during which the trigeminovagal reflex may be precipitated, and by exercising extra vigilance during these procedures, bradycardia or asystole may be recognized immediately and managed effectively.

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