Clinical Reports

Trigeminocardiac reflexes: maxillary and mandibular variants of the oculocardiac reflex

Scott Lang BSc MD CRCPC Dennis T. Lanigan DMD MD, Mike van der Wal MB ChB

Three case reports are presented to illustrate the existence and importance of reflex bradycardic responses that can occur during maxillofacial surgical procedures. All three patients were healthy young adults undergoing operations which did not include any manipulation of orbital structures. After the patients had been anaesthetized for some time and were haemodynamically stable, profound bradycardia or ventricular asystole occurred suddenly in response to manipulations of the bony structures of the maxilla or mandible, or dissection of, or traction on, the attached soft tissue structures. The parasympathetic supply to the face is carried in the trigeminal nerve. Alternative afferent pathways must exist via the maxillary and/or mandibular divisions, in addition to the commonly reported pathway via the ophthalmic division of the trigeminal nerve in the classic oculocardiac reflex. The efferent arc involves the vagus, regardless of which branch of the trigeminal nerve transmits the afferent impulses. All patients undergoing maxillofacial procedures should be monitored carefully for reflex bradycardia and ventricular asystole.

Voici trois cas mettant en lumière l'existence d'une importante bradycardie réflexe pouvant survenir pendant une intervention chirurgicale maxillo-faciale. Alors que l'anesthésie durait depuis un bon moment, que leur hémodynamie était stable, et en l'absence de manipulation des structures de l'orbite, nos trois patients, de jeunes adultes, devinrent sévèrement bradycardes ou asystoliques pendant la mobilisation du maxillaire supérieur ou inférieur ou la dissection/traction des tissus s'y rattachant. Le trijumeau assure l'innervation parasympathique de la face. On connaît bien le réflexe oculo-cardiaque qui passe par la branche ophtalmique du trijumeau. Les branches maxillaires et/ou mandibulaires du nerf peuvent donc aussi transporter des afférences initiant un réflexe similaire avec efférence vagale. Soyons dorénavant aux aguets d'une bradycardie réflexe ou d'une asystolie ventriculaire lors des chirurgies maxillo-faciales.

Key words
COMPLICATIONS: arrhythmia;
HEART: arrhythmia, bradycardia;
SURGERY: maxillofacial.

From the Departments of Anaesthesia and Oral and Maxillofacial Surgery, Royal University Hospital, University of Saskatchewan, Saskatoon, Saskatchewan, Canada.

Address correspondence to: Dr. S. Lang, Department of Anaesthesia, Royal University Hospital, Saskatoon, Saskatchewan, Canada, 57N 0X0.

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The oculocardiac reflex was first described in 1908 by both Bernard Ascher[1] and Guiseppe Dagnini.[2] Anaesthetists are well aware of the potential for disaster due to unrecognized or untreated bouts of severe bradycardia or asystole caused by this reflex.[3-5] It is not well recognized, however, that reflex bradycardia can occur during facial surgery not involving the orbit. Three case reports and a review of the literature will illustrate the existence of trigeminocardiac pathways, other than the classic oculocardiac reflex, that demand equal respect and vigilance. These reflexes are therefore better termed trigeminocardiac reflexes as has been suggested by Shelly and Church.[6]

Case #1

A 28-yr-old Caucasian female, 50 kg in weight, was admitted with a left unilateral cleft lip and palate deformity. Past medical history included previous surgery to repair her cleft lip and palate. She was known to have a congenital heart defect, and cardiac consultation confirmed a small haemodynamically insignificant ventricular septal defect. She had a right bundle branch block pattern on her preoperative ECG. She was otherwise healthy. Premedication consisted of clonidine 150 μg orally. Anaesthesia was induced with thiopentone 275 mg

and sufentanil 40 μg An uneventful intubation was facilitated with 30 mg of *iv* atracurium.

Prior to surgery she received penicillin G 2 million units and dexamethasone sodium phosphate 8 mg *iv*, and the surgeon infiltrated the left maxillary buccal sulcus and the alveolar cleft site with 5 ml of 0.5% lidocaine with 1:200,000 epinephrine for haemostasis. Anaesthesia was maintained with 50% nitrous oxide in oxygen, isoflurane (end-tidal (ET) isoflurane concentration 0.4–1.1%; measured by mass spectography) and intermittent doses of sufentanil and atracurium. During the first 1½ hr the patient remained stable with a $PETCO_2$ ranging between 26 and 32 mmHg (3.46–4.26 kPa), oxygen saturations >97%, a mean arterial pressure of 70 mmHg (9.31 kPa), and sinus rhythm at 70 beats · min^{-1}.

The patient had a satisfactory anteroposterior and transverse relationship between the maxillary greater segment and the mandible. The surgery planned was to perform a left unilateral maxillary osteotomy with anterior advancement and transverse expansion of the left lesser segment. Using vertical incisions, a unilateral maxillary osteotomy was performed, the segment was downfractured, and a pterygomaxillary osteotome was then utilized posteriorly to push the left maxillary segment forward. Every time the osteotome was used a sudden decrease in the heart rate was observed resulting, eventually, in three seconds of ventricular asystole. The asystole resolved when the surgeon released the forward pressure on the pterygomaxillary osteotome. Subsequently, 0.6 mg of *iv* atropine was given and the procedure was completed without any further episodes of bradycardia or asystole. Muscle relaxants were reversed with edrophonium 50 mg and atropine 0.6 mg, and extubation occurred uneventfully in the awake patient. The patient was monitored in the recovery room for two hours without recurrence of any arrhythmias and was discharged to the ward.

Case #2

A 26-yr-old, 66 kg Caucasian female was admitted due to problems of vertical maxillary excess, an open bite malocclusion, mandibular retrognathia, and a long-standing history of temporomandibular joint dysfunction. Her planned surgery included a Le Fort I maxillary osteotomy with intrusion, a mandibular advancement via bilateral sagittal mandibular ramus osteotomies, and an advancement genioplasty. She had had no previous operations and was healthy. Her preoperative ECG was normal. Premedication included ranitidine 150 mg and nadolol 40 mg orally one hour before surgery. Anaesthesia was induced with thiopentone 250 mg and fentanyl 250 μg. Intubation was facilitated with succinylcholine 100 mg. She then received dexamethasone sodium phosphate 8 mg and cefazolin 1 g *iv*. Anaesthesia was maintained with 50% nitrous oxide in oxygen and isoflurane (ET isoflurane concentration between 0.5–1.0%). Intermittent doses of atracurium and fentanyl were used as indicated. Prior to starting surgery the surgeon infiltrated the maxillary buccal sulcus with 10 ml 0.5% lidocaine with 1:200,000 epinephrine. Following completion of her maxillary surgery, and prior to beginning the mandibular sagittal split ramus osteotomies, the mandibular posterior buccal sulcus area was infiltrated with 10 ml 0.5% lidocaine with 1:200,000 epinephrine. For the initial 2½ hr the patient's condition was stable, with $PETCO_2$ ranging between 28 and 37 mmHg (3.7–4.9 kPa), SaO_2 > 98%, blood pressure between 80/50 and 90/60 mmHg (10.6/6.65–11.97/7.98 kPa), and heart rate in sinus rhythm at 45–55 beats · min^{-1}. No problems were encountered during the Le Fort I osteotomy, the genioplasty, or the right mandibular sagittal split ramus osteotomy. During the soft tissue dissection to gain access to do the osteotomy cuts for the left sagittal split ramus osteotomy, problems were encountered when placing a channel retractor subperiosteally along the medial aspect of the mandibular ascending ramus. The patient developed brief ventricular asystole, but the cardiac rhythm returned to normal after the procedure was immediately stopped. The asystole resumed, however, when the surgeon again attempted to place the channel retractor along the medial aspect of the mandibular ramus. The procedure was stopped and the surgeon administered 4 ml of 2% lidocaine with 1:100,000 epinephrine for an inferior alveolar nerve block while the anaesthetist simultaneously gave 0.6 mg of atropine *iv*. No further arrhythmias were encountered and the rest of her surgery was carried out without incident. At the end of the procedure, the patient was given droperidol 1 mg and metoclopromide 10 mg *iv* as nausea prophylaxis. Residual muscle relaxation was reversed with edrophonium 40 mg and atropine 0.6 mg and the trachea was extubated when the patient was awake. She had no episodes of bradycardia or asystole while monitored in the recovery room for 2½ hr. She was transferred to an observation unit. However, subsequently she experienced episodes of bradycardia and irregular rhythm and was sent to the surgical intensive care unit overnight for more comprehensive monitoring. The ECG in the ICU showed an intermittent nodal rhythm (sinus arrhythmia).

Case #3

A 52 kg 38-yr-old Caucasion female was admitted for correction of a severe class III malocclusion secondary to a midfacial retrusion and mandibular prognathism. Planned surgery included a Le Fort I osteotomy with advancement and bone grafting, and a mandibular set-

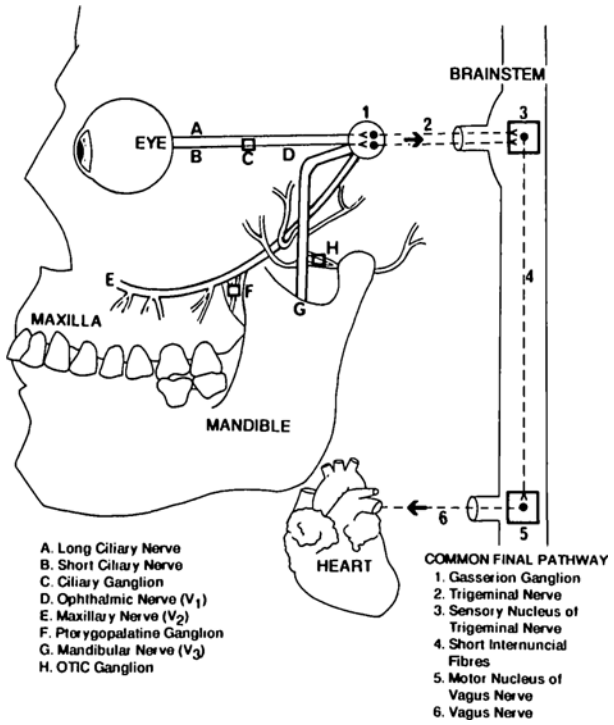

FIGURE Trigeminovagal reflexes.

back. She had had three previous general anaesthetics without complications and had no relevant medical history. Her preoperative ECG was normal. Anaesthesia was induced with thiopentone 250 mg and alfentanil 250 μg; nasotracheal intubation was facilitated with succinylcholine 100 mg. Prior to starting surgery she received dexamethasone sodium phosphate 8 mg and cefazolin 1 g *iv*, and her maxillary buccal sulcus was infiltrated with 10 ml of 0.5% lidocaine with 1:200,000 epinephrine. Anaesthesia was maintained with 50% nitrous oxide in oxygen and halothane (ET halothane concentration between 0.9–1.0%). Intermittent doses of vecuronium and alfentanil were given as required. For the first 70 min, the patient was stable with $P_{ET}CO_2$ between 29 and 31 mmHg (3.86–4.12 kPa), $SaO_2 > 98\%$, blood pressures between 90/60 and 110/60 mmHg (11.97/7.98–14.63/7.98 kPa), and sinus rhythm at 75–95 beats · min^{-1}.

The Le Fort I osteotomy and maxillary downfracture were accomplished without difficulty. However, on advancing the maxilla forward, using only finger pressure, the patient's heart rate suddenly decreased from 95 to 65 beats · min^{-1}. It returned to baseline on discontinuing the traction. The bradycardia recurred when forward maxillary traction was repeated, but once again was corrected immediately after discontinuance of the traction. No active intervention was required and the remainder of the surgery was completed without further arrhythmias. Muscle relaxation was reversed with atropine 1.2 mg and neostigmine 3.0 mg *iv* at the end of the procedure, and the trachea was uneventfully extubated when she was awake.

Discussion

The oculocardiac reflex is a well-recognized phenomenon. It occurs most commonly during strabismus surgery[3,4] but can also occur during other operations in and about the orbit.[7–12] In all these situations the afferent limb of the reflex arc is via the ophthalmic division of the trigeminal nerve (Figure). The afferent 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 nerve fibres connect with the efferent pathway, which originates in the motor nucleus of the vagus nerve. Depressor fibres of the vagus nerve end in the myocardium. However, the ophthalmic division is not the only branch of the trigeminal nerve which can carry the afferent stimuli in the reflex arc which is responsible for bradycardia or asystole. Loewinger *et al.*[13] and Shearer *et al.*[14] reported episodes of bradycardia during elevations of zygomatic fractures. Roubideaux[11] documented a case of a sudden decrease in heart rate (from 90–54 beats · min^{-1}) consonant with surgical disimpaction of a fractured maxilla in a 22-yr-old healthy male. Ragno *et al.*[15] described several episodes of ventricular asystole in a single patient during the downfracturing of a maxilla as part of Le Fort I osteotomy. Precious *et al.*[16] reported a 1.6% incidence (8/502 cases) of either asystole or bradycardia in patients undergoing maxillofacial orthognathic or temporomandibular surgery. Although it might be argued that the ophthalmic nerve could be the afferent limb of the reflex arc when maxillary osteotomies are performed, the same claim cannot be made for mandibular osteotomies or temporomandibular joint surgery. Alternative reflex pathways must occur via the maxillary (second) and/or mandibular (third) divisions of the trigeminal nerve.

Several factors are known to increase the risk of the oculocardiac reflex occurring.[4] These include: hypercarbia, hypoxaemia, light anaesthesia, age (more pronounced in children, perhaps due to higher resting vagal tone), and the nature of the provoking stimulus (stimulus strength, duration, and waveform). Pharmacological agents such as potent narcotics like sufentanil[17] and alfentanil,[18] beta blockers, and calcium channel blockers[19] may predispose to this reflex. Narcotics may augment vagal tone via their inhibitory action on the sympathetic nervous system.

The oculocardiac reflex normally fatigues with repetitive stimuli. The initial baseline heart rate has no influence on the incidence of the oculocardiac reflex:

tachycardia is therefore not protective.[4] Recognized therapeutic manoeuvres include the avoidance of factors known to predispose to the development of the reflex, cessation or modulation of the surgical stimulus, *iv* atropine or glycopyrrolate, and local anaesthetic blockade of the afferent nerves.

Three cases have been presented where combinations of bradycardia and ventricular asystole were encountered in response to surgical stimuli in the distribution of the maxillary and mandibular divisions of the trigeminal nerve in otherwise healthy individuals undergoing maxillary or mandibular osteotomies. Certain factors may have predisposed the patients in this series to the development of reflex bradycardic episodes. These include: conduction abnormalities and a ventricular septal defect in case #1, preoperative nadolol in case #2, and the use of intraoperative narcotics in all three patients.

If continuous ECG monitoring is employed and the anaesthetist and surgeon are appropriately vigilant, these reflex bradycardic/asystolic events can be recognized early and managed effectively by stopping the surgical stimulus and administering *iv* atropine or glycopyrrolate. If the reflex persists, local anaesthetic infiltration or block of the nerve responsible for the afferent stimuli should mitigate any further problems.

Prevention of trigeminovagal reflexes requires awareness of the anatomical structures involved, cooperation between the surgeon and the anaesthetist, and knowledge of factors known to favour or evoke these reflexes.

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