Case Report
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Cardiac Dysrhythmia During Superficial Parotidectomy

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Bradycardia and asystole due to the trigeminocardiac reflex can occur during several maxillofacial, skull base and ophthalmic procedures.

We describe a case where severe bradycardia occurred during superficial parotidectomy. The case report is presented to show a possible existence and importance of reflex bradycardic responses that may occur during surgical procedures involving the parotid gland and their management.

Keywords: trigeminocardiac reflex, anesthesia, bradycardia, parotidectomy

Introduction

The trigeminal nerve is the largest of the cranial nerves, and it provides sensory supply to the face, scalp, and mucosa of the nose and mouth. Bradycardia and asystole due to the trigeminocardiac reflex (TCR) can occur during several maxillofacial, skull base and ophthalmic procedures. [1] Stimulation of any sensory branch of the trigeminal nerve is thought to provide an important stimuli for the initiation of the trigeminorespiratory reflex, parasympathetic dysrrhythmias, sympathetic hypotension, apnea, gastric hypermotility etc. [2] In fact, the term TCR was coined by the anesthetists Shelly and Church. They also described the first occurrence of central TCR in humans during the surgery of cerebellopontine angle and brainstem. According to Schaller's experience, the TCR occurs in 10-18% of the patients undergoing maxillofacial, skull base and ophthalmic surgery. [3]

Presented case highlights the importance of reflex bradycardic responses, which may occur during parotidectomy, and the significance of perioperative management of TCR.

Case report

A 44 years old heavy smoker normotensive male, weighed 85 kg and with height 185 cm, was scheduled for left superficial parotidectomy. His medical history and physical examination were unremarkable. Results of the routine clinical investigations, including electrocardiogram (ECG), were within the normal limits. Premedication included 5mg of intravenous dormikum. His baseline heart rate (HR) was 85 beats per min and his blood pressure (BP) measured 128/80 mmHg. The anesthesia was induced with intravenous injection of propofol (160 mg) and fentanyl. [100 μg] Orotracheal intubation was facilitated with suxamethonium. The anesthesia was maintained with sevoflurane (2.5%) and oxygen, and controlled ventilation. The HR and BP remained stable at induction (75-85 beats per min and 140/80 - 110/70 mmHg, respectively). The surgeon proceeded with left superficial parotidectomy (facial nerve was preserved). For the initial 50 minutes, the patient's condition was stable, with ETCO2 (end tidal carbon dioxide) ranging between 28 and 35 mmHg, SpO2 (peripheral capillary oxygen saturation) 98-100%, BP between 120/80 and 100/60 mmHg, and sinus rhythm of 70-80 beats per min. During dissection of the superficial lobe for the mobilization of facial nerve, there was a little rise in BP up to 145/100 mmHg and in HR up to 90 beats per min. A dose of fentanyl (100 μg intravenously) was administered and concentration of sevoflurane was increased up to 4%. About 30 minutes later, sudden bradycardia occurred. The

BP and HR records were 80/46 mmHg and 42 beats per min correspondently (Fig. 1). The surgery was immediately halted, oxygenation and normocapnia were confirmed, and inhalational anesthetic agents were discontinued. A dose of atropine (1 mg intravenously) was administered and repeated after two minutes, as HR decreased further to 39 beats per min. Because HR remained unresponsive to atropine, a dose of dopamine (10 μg/kg/min intravenously) was administered. Then HR promptly increased to 94 beats per min with normal sinus rhythm, BP and SpO2 became 110/76 mmHg and 99% respectively. 5 min following administration of dopamine, BP constituted 120/81 mmHg and HR was 80-90 beats per min. In order to stop transmission of the afferent signals topical lidocaine (2%) was applied around the gland.

The entire episode lasted 10 min; throughout it systolic BP (recorded every 1 min) and SpO2 remained >90 mmHg and >99% respectively. Then the anesthetics were re-instituted and the surgery was continued. During the surgery there were several episodes of bradycardia, which reversed after withdrawal of the mechanical stimuli and a dose of dopamine.

The total duration of surgery was 3 hours. The patient made safe recovery with no untoward events in the postoperative period. His postoperative serial 12 lead ECG, serum electrolytes and other common biochemical parameters were within normal limits. The patient was discharged on postoperative day 2 to continue follow up an outpatient basis.

Discussion

The TCR has been described during many neurosurgical, ophthalmologic and maxillofacial procedures. It is defined as the sudden onset of parasympathetic dysrhythmia, sympathetic hypotension, apnea, or gastric hyper-motility during stimulation of any sensory branch of trigeminal nerve. The proposed mechanism for the development of the TCR is that the sensory endings of trigeminal nerve send neuronal signals via the Gasserian ganglion to the sensory nucleus of the trigeminal nerve, forming the afferent pathway of the reflex arc. This afferent pathway continues along the short internuncial nerve fibers in the reticular formation to connect with the efferent pathway in the motor nucleus of the vagus nerve (Diagram 1). [4] Several experiments on animal models demonstrate that trigeminally induced cardiovascular reflexes could be mediated initially in the trigeminal nucleus caudalis and subsequently in the parabrachial nucleus, the rostral ventrolateral medulla oblongata, the dorsal medullar reticular field, and the paratrigeminal nucleus.

There has been a lot of discussion about the best and more effective management of TCR. Without any doubt, one can say that application of atropine is not the only modality of the treatment. To the authors' opinion, the first and the most important management option for the TCR is to be aware of its existence and to minimize any mechanical stimulation of the nerve that leads to its occurrence.

According to the clinical experience on this topic [5], the management of patients with TCR can be classified into the following categories which are illustrated in the flow-chart (Diagram 2):

1. Risk factor identification and modification.
2. Prophylactic treatment with either vagolytic agents or peripheral nerve blocks in case of peripheral manipulations on trigeminal nerve.
3. Careful cardiovascular monitoring during anesthesia especially in those with risk factors for TCR.
4. Treatment of the condition when it occurs:
   a. cessation of the manipulation, and;
   b. administration of vagolytic agents or adrenaline.

If a TCR is elicited, the surgeon must stop the stimulus and wait until the pulse recovers its normal rhythm. There is also important issue concerning choice of pharmacological agent. Prabhakar et al. reported a 48-year-old female who developed severe bradycardia and hypotension during craniotomy for parietal convexity meningioma; she was unresponsive to atropine and successfully managed with epinephrine. [6] This important case report underlines the fact that TCR may be refractory to atropine and other vagolytic agents should be considered instead.

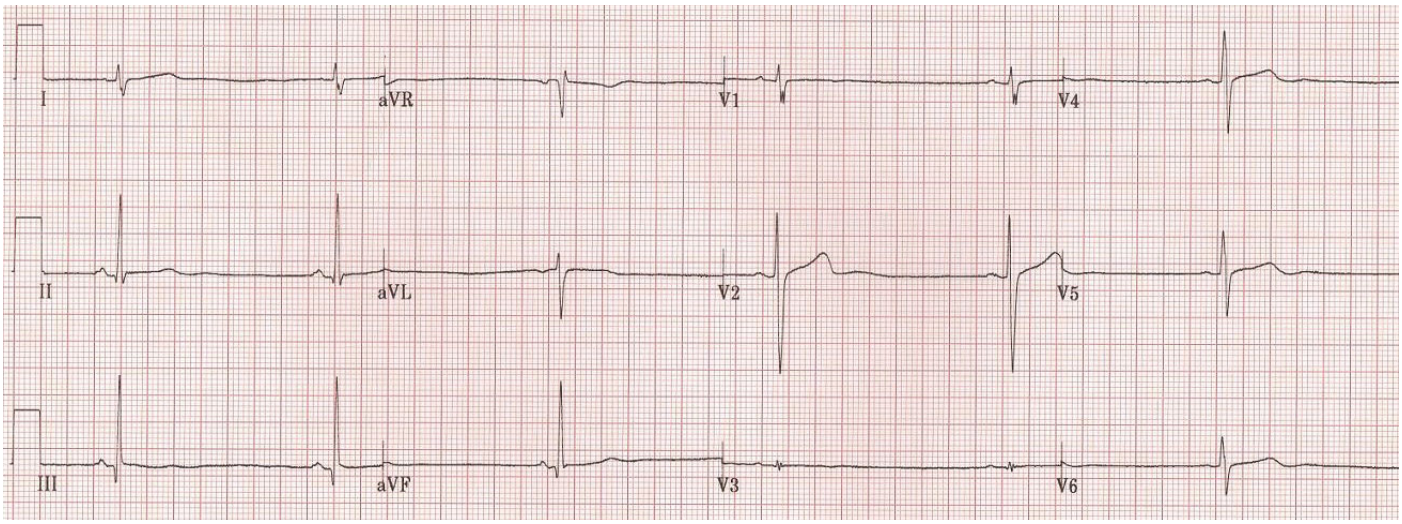
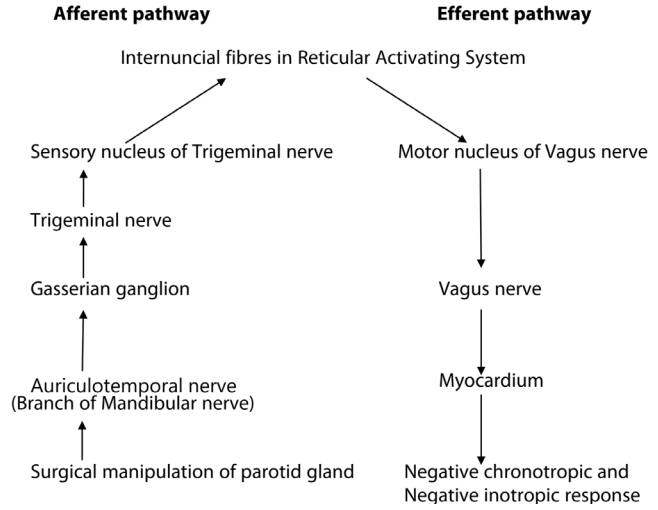


Figure 1.
Intraoperative ECG record of patient.

Diagram 1.
The trigeminocardiac reflex (TCR) [8]

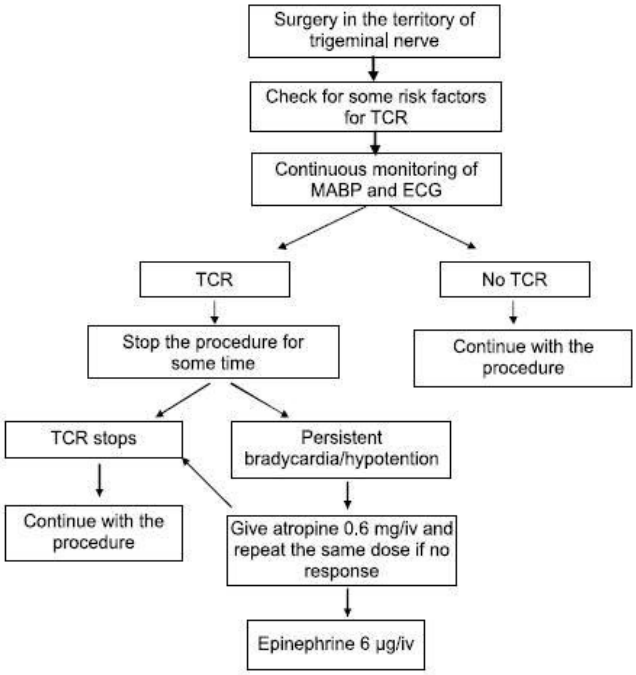


Our patient was not hypertensive and had no structural heart disease; however, his intraoperative blood pressure was labile. The patient initially developed hypertension (145/100 mmHg) and tachycardia (100 beats per min) which were considered due to pain and light anesthesia. So, this event was managed satisfactorily by increasing the concentration of sevoflurane (up to 4%), and administration of fentanyl. [100 μg] Dysrhythmias and severe bradycardia occurred suddenly in response to dissection, manipulation and traction of the superficial lobe of parotid gland. Cessation of the surgical stimuli and topical application of lidocaine did disrupt the dysrhythmia. A dose of atropine was administered but failed to control the bradycardia, which was subsequently corrected by dopamine.

The effect of dopamine can be explained by its indirect β- and α-adrenergic influences through stimulation of norepinephrine release. Dopamine is a second line drug for symptomatic bradycardia, and should be used after atropine. [7] At doses of 5 to 10 μg/kg/min, it shows positive inotrope and chronotrope effects. This dose of dopamine (a cardiac dose) acts on the sympathetic nervous system and increases cardiac output together with blood pressure.

Here we would like to underline the possibility of TCR during surgery, involving parotid gland and successful resolution of anticholinergic-resistant bradycardia with the use of appropriate dose of dopamine.

Diagram 2.
Common managment of trigeminocardiac reflex (TCR) [2]



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