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Intraoperative Bradycardic Reactions During Facial and Oral Surgery in the Office: The Role of the Trigemino-Cardiac Reflex (TCR)

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ABSTRACT

We present a series of 4 patients undergoing blepharoplasty or multiple dental implants who exhibited bradycardia and hypotension during the procedure attributable to the trigemino-cardiac reflex (TCR). The immediate administration of atropine, vasopressors, crystalloids solved the problem.

We advocate the need for a continuous monitoring of all vital signs plus the dedicated presence of the anesthesiologist even during minor surgery since the reaction occur at least in 2% of cases and my evolve into major cardiac events.

Abbreviations: Im: Involuntary movements; LA: Local Anestesia/Anesthetic; LOC: Loss of Consciousness; PONV: Postoperative Nausea and Vomiting

INTRODUCTION

Vasovagal reactions, resulting in bradycardia and/or hypotension in response to a number of stimuli (fear, pain, unusual sights and smells, anxiety, fatigue, fasting, venipuncture, local anesthesia, viscous distention, etc.) are frequently encountered in surgery and anesthesia.

Surgery can predispose patients to vasovagal episodes owing to the temporo-spatial coincidence of many stimuli; the reactions are usually self-limiting, but potentially life-threatening exceptions have been described up to asystole in different environments, starting with venipuncture [1], dentistry [2], Maxillo facial surgery [3], ophtalmic surgery [4], gastrointestinal endoscopy [5].

A complete review of this topic has been covered in the article [6]; but this excellent review failed to mention the possibility that perioperative bradycardia could originate from the trigeminal nerve. As a matter of fact the surgical procedures more frequently involved include, ophthalmology, maxillofacial, as mentioned above, neuro cranic [7-9] and dental, suggesting that the trigeminal nerve is responsible of the so called trigeminocardiac reflex (TCR) [10].

The TCR is the reflexive response of bradycardia, hypotension, and gastric hyper motility induced with mechanical stimulation in the distribution of the trigeminal nerve; the sensory nerve endings of the Vth cranial nerve arrive at the trigeminal nucleus and the efferent pathway passes through the reticular formation and connects to the motor nucleus of the vagus nerve; the efferent fibers arrive at the myocardium imposing the typical negative chronotropic and inotropic reponses on the heart.

This reflex is triggered usually by a strong stimulus applied in the area innervated by the trigeminal fibers, what is less known and common is the occurrence of vagal reactions intraoperatively, apparently without cause and not accompanied by pain or discomfort.

We present 4 cases of bradycardia and hypotension during conscious sedation done in the office for cosmetic and dental surgery; 3 reactions occurred in the middle of the case without any apparent discomfort: all were attributed to the TCR.

CASE REPORTS

All patients were premedicated 20-30 min before surgery; they were laying supine, ECG, SaO2, side stream etCO2 monitored continuously and NIBP every 5 min: vital signs were collected every 5 min. A peripheral vein was in cannulated and normal saline infused at a speed of 100-150 ml/h. Midazolam and fentanyl were administered 5-10 min

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before LA; sedation depth assessed according to the Ramsay scale maintaining a score between 2 and 3 at all times.

In all cases local anesthesia was successful and patients did not complain of pain, did not lose consciousness (except perhaps FS), pallor and sweating were not noticed (**Table 1**).

Table 1. Patients and their surgery details.

Name	Age	Weight (kg)	Height (cm)	Asa	Surgery	Premedication (mg)	Sedation	Time of occurrence (min) from start of surgery and till last
SV	51	54	160	2 (micro citemia	Blefaroplasty sup and inf	Diaz 5+halop 0.6	Midaz 2+Fent 50	30 min
AC	39	60	162	1	Blefaroplasty sup and inf	diaz 5+halop 1	Midaz 2+Fent 50+Clon 60	65 min
FS	68	75	149	2, hypert ension, obesity	Multiple dental implants	diaz 5+ halop 0.6	midaz 3+mep 30	60 min
CP	55	83	180	1	Multiple dental implants	diaz 8	midaz 4+fent 80+halop 0.5	85 min

Diaz: Diazepam (mg); fent: fentanyl (µg); clon: clonidine (µg); mep=meperidine (mg); halop=haloperidol (mg)

FS was receiving an additional dental block when the reaction started; in the remaining patients surgery and sedation was proceeding smoothly.

SV: BP drop from 102/55 to 69/35, HR from 55 to 35. Therapy Atropine 0.5 mg, ephedrine 5 mg. SaO2 100: after 3 min BP 85/52, HR 82. NO pain, no loc, no IM. SaO2 and et CO2 stable, AC: BP drop from 103//69 to 74/45, HR from 65 to 45: atropine 0.7 mg, no loc, no pain, no IM, SaO2 and etCO2 stable.

FS, BP drop from 103/57 to 89/47, HR from 73 to 43, SaO2 93; LOC for 1 min; tonicoclonic movements of the right hand and head: atropine 0.7 mg+etilephrine l, oxygen 2 L/min+dexamethasone 4 mg; BP to 108/51 and hr 89, within 2 min; SaO2 98: crystalloids 250 ml in 5 min.

Patient returned for completion of surgery 3 months later, premedicated with diazepam 6 mg+atropine 0.5 per os, +atropine 0.3 at the beginning of surgery, midazolam 3 mg+fentanyl 50 µg, procedure uneventful.

CP: bradycardia+hot flush, fast response to atropine 0.8 mg and 250 ml crystalloids.

DISCUSSION

The occurrence of the vaso-vagal reactions during the operation, excluding cases were this type of reaction happens at the moment of venipuncture or during infiltration local anesthesia, could be estimated at around 0.8-1% since these cases were collected in the last 2 years of a private practice limited to dental and cosmetic surgery encompassing roughly 500 cases. This incidence is similar to that reported in larger series like Shalom et al. [11] who reported an incidence around 2% of syncopal episodes over 2600 minor dermatological procedures and Edmondson et al. [12] who described vasovagal episodes in dental surgery.

The etiology of the above reactions surely attributable to the TCR remain unknown, since in all cases the pharmacological sedation provided by the anesthesiologist and the local/truncal analgesia provided by the surgeon were deemed good and in no instances the patients complained of pain, with the exception of patient FS where the reaction occurred during a LA top up in the palate.

The link between the stimulation of any sensory part of the V^{th} cranial nerve with the hemodynamic irregularities has

been frequently reported in the field of maxillo-facial and neurosurgery [13,14].

The physiology of this reflex has been intensively studied in the most recent years with the aim to allow a more precise definition and classification [15,16]. More recently a chronic form of the reflex has been described [17] so that we may divide the TCR into acute and cronic variants and into peripheral or central according to the site of origin: oculocardiac and maxilla-mandibular being peripheral and any TCR episode due to the stimulation of the fifth nerve pathway beyond the Gasserian ganglion considered as the central TCR.

The primary pathophysiological hemodynamic mechanism involved in our patients remain unknown since the intermittent measurement of blood pressure as opposed to the continuous monitoring of ECG and pulse oximetry could not give any clue as far as the etiology, since the slowing of the ECG gave the first sign of the reaction and prompted the immediate injection of atropine, blood pressure being checked immediately after and supported with vasopressors and crystalloids if found low. Only in the patient FS the reaction could be attributed to sudden stimulation in the area supplied by the greater palatine nerve (V).

Haloperidol, a potent neuroleptic used in the treatment of severe agitation [18] and then in the treatment of schizophrenia and other psychotic disorders [19] was administered at a low dosage to reduce the possibility of PONV [20] in this outpatient high risk population: moreover his protective effect was enhanced by the administration od dexamethasone at the end the procedure according to a multimodal strategy [21,22].

It is possible that the administration of haloperidol, could have been contributory to the onset of hypotension since the drug possesses a moderate alpha adrenergic blocking effect, thereby reducing peripheral vascular resistance and hence inducing a moderate hypotension [23,24]: unfortunately the more specific antiemetic droperidol is not available in Italy outside the hospital.

Atropine remains the milestone for the treatment of acute bradycardia and its advantages have been emphasized during manipulation of percutaneous compression of the trigeminal ganglion for trigeminal neuralgia albeit at low dosage in order to avoid rebound tachycardia [25]; however the clinical situation of our cases is far different from neurosurgery and we did not observe any dangerous rebound increase in HR following its injection, recommending the administration of a full dose, at least 0.5 mg. In the context of conscious sedation we cannot try to abolish the reflex by deepening anesthesia with a bolus of propofol as suggested by Chowdhury et al. [26]

It since might impair the respiration and hemodynamic stability of the patient and cause a shift toward general anesthesia with the loss of the protective reflexes of the airways.

In order to obtain the best outcome we suggest that beside the drugs to be used for sedation, the diligent anesthesiologist should always have a few prefilled labeled syringes at hand with at least 1 mg of atropine and a vasopressor like ephedrine 30-50 mg, diluted in 10 ml of NS. It is also important that no time should be wasted looking around for the injection port of the intravenous line: a 3 way stopcock situated in the extension set is the best choice for speed of access and prevention of needle stick injuries: at the same time the fully release of the roller clamp maximize the infusion of the crystalloid in use.

CONCLUSION

In conclusion, the sudden appearance of bradycardia and hypotension during the maintenance phase of any procedure around the face emphasizes the need for the dedicated and continuous vigilance from the part of the anesthesiologist; treatment must be immediate in order to prevent he cardiocirculatory collapse.

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