Introduction

Vagally mediated reflex bradycardic responses such as trigemino-cardiac reflex and oculocardiac reflex during maxillofacial,[1] nasal[2] and ocular surgeries[3] have been previously reported. These disturbances occur due to profound parasympathetic stimulation and can cause intense bradycardia which can rarely culminate in asystole. The afferent pathway of these reflexes is mediated by any of the branches of the trigeminal nerves, and the efferent pathway is via the vagus nerve. Elicitation of this reflex is commonly seen during surgical manipulation and is manifested as bradycardia or even asystole. We report a case where nasocardiac reflex was unusually observed in a patient when aspiration and injection were done through a nasogastric tube.

Keywords: Bradycardia, nasocardiac reflex, nasogastric tube

Case Report

A 52-year-old female patient was admitted in the Intensive Care Unit (ICU) following an episode of exacerbation of chronic obstructive pulmonary disease with elevated PaCO₂ levels (85 mmHg) in the arterial blood gases (ABG) and obtunded mental state in the evening. She was immediately put on noninvasive ventilation (NIV) and treated with antibiotics, bronchodilators and steroids. After 2 h of NIV support, the patient did not show any clinical benefit and had to be intubated for mechanical ventilation. The patient responded favorably to the mechanical ventilation and treatment overnight. Subsequent ABG’s revealed a decreasing trend of PaCO₂ which reduced to 53 mmHg on the 2nd day morning with an overall improvement in the general and mental condition. As the patient was still on mechanical ventilation, for initiation of alimentary feeding, a NGT was decided to be inserted. A 16G silicone NGT was inserted smoothly after adequate lubrication (water based jelly) in a single attempt through her right nostril up to the 50 cm mark. To confirm the proper placement of the NGT in the stomach, aspiration through the NGT was attempted with a 50 ml syringe. As about 30 ml bilious gastric
contents came out in a gush, an episode of sudden and profound drop in the heart rate (94 to 42 beats/min) was observed (confirmed by manually palpating the radial pulse). The heart rate normalized as soon as the aspiration was stopped. Further aspiration did not yield any gastric contents and neither did the heart rate drop. Now as a test feed, when 30 ml of normal saline at room temperature was injected with a 50 ml syringe, through the NGT a similar drop in heart rate (90 to 45 beats/min) was observed midway during the injection. The heart rate returned to the baseline when the injection was discontinued. However throughout this episode, sinus rhythm was maintained, and other vital parameters (systolic and diastolic blood pressures, oxygen saturation) remained stable. Anticipating similar disturbances, injection atropine was loaded in a syringe and kept at the bedside. The patient underwent an urgent naso-gastric aspiration to manage the hemodynamic changes. After the initial episodes, drop in the heart rate was not observed whenever aspiration and injection were attempted through the NGT, thereafter. The patient’s clinical status improved further the next day, and she could be extubated on the 3rd day and recovered sufficiently to be discharged from the ICU by 4th day.

Discussion

Nasocardiac reflex has been previously described by Baxandall and Thorn wherein bradycardia was observed when nasal speculum was introduced into nares under general anesthesia.[4] 25% ammonia, when used to stimulate the medial turbinate, has been reported to cause a significant drop in heart rate in subjects.[5] The nasal cavity (nasal septum and lateral wall) receives extensive sensory supply through the trigeminal nerve via its ophthalmic (V1) and maxillary (V2) branches as explained below.

a. Nasal septum [Figure 1]
  - Anterosuperior part: Through internal nasal branch of anterior ethmoidal nerve (branch of V1)
  - Posteroinferior part: Through sphenopalatine branch of the pterygopalatine ganglion (branch of V2)
  - Mobile part: Through external nasal nerve (branch of V1).

b. Lateral wall [Figure 2]
  - Anterosuperior quadrant: Through anterior ethmoidal nerve (branch of V1)
  - Anteroinferior quadrant: Through anterosuperior alveolar nerve (branch of V2)
  - Posterosuperior quadrant: Through posterosuperior lateral nasal nerve of the pterygopalatine ganglion (branch of V2)
  - Posteroinferior quadrant: Through anterior palatine nerve from the pterygopalatine ganglion (branch of V2).

Through the V1 or V2 branches of trigeminal nerve, the afferent limb of a reflex arc is formed involving the pterygopalatine ganglion, gasserian ganglion, trigeminal nerve, sensory nucleus of trigeminal nerve, short internuclear fibers, motor nucleus of the vagus nerve and finally the vagus nerve which supplies the heart [Figures 1 and 2]. Stimulation of branches of trigeminal nerve or the areas they innervate, can provoke a complex physiological response characterized by apnea’ bradycardia, vasoconstriction and inhibition of respiratory drive which involves the integration of afferent impulses carried on fibers innervating somatic receptors, baroreceptors and chemoreceptors.[6] Such reflexes are known as trigeminovagal reflexes and include the oculocardiac reflex and the comprehensively discussed trigeminocardiac reflex[7] which are elicited during surgical manipulations. Clinically trigeminocardiac reflex has been reported to occur during craniofacial surgeries, manipulation of trigeminal nerve/ganglion, surgeries of cerebellopontine angle, cavernous sinus and pituitary fossa.[8] The probable explanation of this event is that during the process of initial aspiration of gastric contents (to confirm proper placement) and subsequent injections of liquid feed through the NGT the liquid columns would have raised the pressures inside the NGT which were conducted to the circumference of the NGT effecting in a minor circumferential increase. Since the NGT was snugly fitting (16 G) the conformational changes of its circumference would have stimulated the nasal mucosa of the septum or turbinates where the wall of the NGT was abutting against. These regions had trigeminal nerve innervations which were stimulated triggering nasocardiac reflex and the consequent bradycardia.

Various factors can predispose to the occurrence of trigeminovagal responses. High sympathetic activity with concomitant parasympathetic stimulation known as “accentuated antagonism” can produce severe bradycardia by eliciting a variety of negative
chronotropic or ionotropic cardiac responses. Hypoxemia, hypercarbia or lighter depth of anesthesia can potentially precipitate such reflexes by increasing the baseline sympathetic tone. Due to the acute critical nature of the illness, the patient was in a state of sympathetic stress. The associated hypercarbia would have accentuated the sympathetic activity and aided the occurrence of this reflex. Classically these reflexes are known to “fatigue” following repeated stimulation.

Presumably the insignificant changes in the heart rate during the later episodes of injection and total absence thereafter was the result of such fatigue.

Through this report, we attempt to emphasize that trigeminoovagal responses (oculocardiac or trigeminocardiac reflexes) which were till now attributed mainly to surgical stimulation could occur following seemingly safe and routine exercises like aspiration and injection through NGT (nasocardiac reflex). Clinicians should, therefore, be aware of the existence of such a phenomenon and should anticipate its occurrence under rare circumstances.

References


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