Trigeminovagal Reflex Caused by Stimulation of the Mandibular Division of the Trigeminal Nerve under General Anesthesia: A Case Report

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Abstract: We report a rare case of trigeminovagal reflex (TVR) resulting from stimulation of the mandibular division of the trigeminal nerve under general anesthesia. Although general anesthesia reportedly prevents TVR, it should be part of the differential diagnosis in all cases of unexplained sinus arrest during oral and maxillofacial surgery.

Key words: general anesthesia, sagittal split ramus osteotomies, trigeminovagal reflex

Introduction
The oculocardiac reflex was first reported in 1908 by both Aschner and Dagnini. Since its afferent and efferent pathways are the trigeminal and vagus nerves, respectively, it is also called the trigeminovagal reflex (TVR). TVR resulting from stimulation of the ophthalmic or maxillary (II) divisions of the trigeminal nerve has frequently been reported as a serious complication under general anesthesia. However, TVR caused by stimulation of the mandibular (III) division occurs very rarely. We report a case in which TVR caused by stimulation of the mandibular division of the trigeminal nerve was observed during sagittal split ramus osteotomies (SSRO) under general anesthesia.

Case Report
A 31-year-old female (weight, 47 kg; height, 155 cm) with progenia presented herself for elective bilateral SSRO under general anesthesia. She had no history of circulatory or autonomic nervous system diseases, and her physical examination was unremarkable. Preoperative investigations, including a chest X-ray, electrocardiogram (ECG), blood chemistry examinations and pulmonary function tests, were within normal parameters.

The patient was not premedicated prior to surgery. Blood pressure and heart rate (HR) on entering the operating room were 120/74 mmHg and 68 beats/min, respectively. Anesthesia was induced using 50 mg of propofol, and intubation was facilitated by administration of 40 mg of rocuronium, following which nasotracheal intubation was performed uneventfully. The patient was mask-ventilated with 5% sevoflurane in 100% oxygen until the onset of muscle relaxation. Thereafter, anesthesia was maintained with 1.5 to 2.0% sevoflurane in 33% oxygen together with 0.15 to 0.25 μg/kg/min remifentanil.

For the initial 2 hr after commencement of SSRO, the patient’s condition remained stable with an ETCO2 of 36 to 40 mmHg, SpO2 of 99 to 100%, blood pressure of 80/50 to 90/60 mmHg, and HR of 60 to 70 beats/min with sinus rhythm.
Right SSRO was performed uneventfully. However, when soft tissue was dissected to gain access for the left SSRO osteotomy cuts, sinus arrest suddenly occurred. At that time, manual pressure was only being applied over the mandible, and at no time was there pressure over the eye. The surgeon was informed immediately, and surgical stimulation was discontinued. Sinus rhythm, although with bradycardia (HR of 30 – 35 beats/min), returned immediately after the procedure was stopped. According to the ECG monitor, the period of asystole lasted for approximately 8 sec. To counteract the bradycardia, 0.5 mg of atropine sulfate was administered, following which HR increased to 90 beats/min within 1 min. Prior to restarting the surgery, the surgeon performed an inferior alveolar nerve block with 2 ml of 1% lidocaine with 1:100,000 adrenaline. The remaining intra- and postoperative period proceeded uneventfully with no further episodes of arrhythmia.

**Discussion**

In the present patient, sinus arrest occurred when the soft tissue was dissected to gain access for performance of the osteotomy cuts for SSRO, and sinus rhythm was restored by temporarily stopping the surgical procedure. We, therefore, surmised that TVR was activated by stimulation of the mandibular division of the trigeminal nerve as a result of the surgical stimulus. In our patient, sinus arrest suddenly occurred when soft tissue was dissected to gain access for the left SSRO osteotomy cuts, despite the right SSRO being performed uneventfully during the first 2 hr of surgery. Classically, the TVR is reported to “fatigue” with repeated application of the triggering stimulus. Therefore, it is possible that surgical stimulation during the right SSRO might have resulted in such fatigue and therefore a lack of activation of the TVR during right-sided surgery.

General anesthesia reportedly prevents TVR. However, Precious et al. reported a 1.6% incidence (8/502 cases) of either asystole or bradycardia in patients during maxillofacial orthognathic or temporomandibular surgery under general anesthesia. Although several factors are known to increase the risk of occurrence of TVR, the reflex under general anesthesia is strongly affected by ventilatory conditions. Bosomworth and Ziegler reported that TVR tends to occur under conditions of hypercapnia and hypoxemia. However, intraoperative SpO₂ and ETCO₂ in the present case remained normal throughout the procedure. Thus, adverse ventilatory conditions can be eliminated as possible contributing factors to the occurrence of TVR in our patient. Seo et al. stated that pharmacological agents may predispose patients to the occurrence of this reflex. Remifentanil is known to strongly augment vagal tone via autonomic or central nervous system inhibition. In this case, we suspect that remifentanil induced set the stage for vagal predominance, thus making this reflex more easily inducible.

In summary, we describe a case in which TVR, caused by stimulation of the third division of the trigeminal nerve, was observed during SSRO under general anesthesia. TVR should be part of the differential diagnosis in all cases of unexplained sinus arrest during oral and maxillofacial surgery. Further, oral and maxillofacial surgeons should remember that temporary cessation of the precipitating stimulus is the most important therapeutic step for terminating TVR.

**Acknowledgements**

We would like to thank Dr. Hiroki Son (Hirakata City Hospital) for his valuable advice and suggestions. No financial support was received for this paper.

**Reference**

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