SINUS ARREST INDUCED BY TRIVIAL NASAL STIMULATION DURING ALFENTANIL–NITROUS OXIDE ANAESTHESIA

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SUMMARY
A case is reported of bradycardia and sinus arrest induced by insertion of a nasal temperature probe. Other possible causes of bradycardia and sinus arrest under anaesthesia are reviewed briefly. Evidence for the neurological basis of a nasocardiac reflex, similar to the oculocardiac reflex, is presented. A minor, trivial stimulus may elicit this reflex.

KEY WORDS

Several reports have described severe bradycardia and sinus arrest or asystole during opioid-based anaesthesia [1–7]. Although opioids are known to enhance vagal tone and promote, induce or cause bradycardia, in all reports of serious arrhythmias other drugs (β-adrenergic blockers, calcium entry blockers, neuromuscular blockers) have also been implicated. In addition, laryngoscopy and tracheal intubation may produce bradycardia, although sympathetic stimulation may also result. This report documents severe bradycardia and sinus arrest induced by trivial nasal stimulation in an otherwise healthy adult during alfentanil–nitrous oxide in oxygen anaesthesia.

CASE REPORT
A 19-yr-old, 1.65-m, 65-kg female was scheduled for dilatation of the cervix and curettage of the uterus for retained placenta. She had undergone uneventful vaginal delivery 2 days previously. She was afebrile and had a heart rate of 88 beat min⁻¹ and an arterial pressure of 95/55 mm Hg. She was otherwise healthy, but had a history of cigarette smoking. PCV was 30%. She did not appear anxious and was given no premedication. An 18-gauge i.v. cannula was inserted and, in the operating room, monitoring with a three-lead electrocardiograph, automatic arterial pressure cuff, precordial stethoscope and pulse oximeter was begun. The patient's lungs were preoxygenated with 100% oxygen using a circle system and face mask and she subsequently received vecuronium 1 mg and droperidol 0.625 mg i.v. Two minutes later the patient was given alfentanil 50 μg kg⁻¹ as an i.v. bolus over 15 s, followed by thiopentone 225 mg and suxamethonium 100 mg. Laryngoscopy and intubation were performed with a No. 3 Macintosh blade and a tracheal tube (7.5-mm i.d.) without difficulty. Breath sounds were present bilaterally and equal. Heart rate was 57 beat min⁻¹ (fig. 1: upper ECG trace) and arterial pressure 90/60 mm Hg after intubation. A nitrous oxide–oxygen mixture (2:1) was administered via a circle system for maintenance of anaesthesia in combination with an infusion of alfentanil 1 μg kg⁻¹ min⁻¹. Ventilation was controlled mechanically with a tidal volume of 700 ml and ventilatory frequency 8 b.p.m.

A temperature probe (Yellow Springs) protected by an American Safe Temp sleeve, was inserted approximately 2.0 cm in the left nares; this caused a sudden and profound decrease in heart rate to 33 beat min⁻¹ (fig. 1: second ECG trace). Heart rate returned to 60 beat min⁻¹ upon withdrawal of the temperature probe. Haemodynamically, the patient was otherwise stable (arterial pressure 94/60 mm Hg). The operation had not yet begun, and no signs of inadequate anaesthesia (tears or movement) were present.
SINUS ARREST INDUCED BY NASAL STIMULATION

FIG. 1. Electrocardiographic changes associated with alfentanil-nitrous oxide-oxygen anaesthesia and insertion of a nasal temperature probe. Upper trace: ECG after induction of anaesthesia and before arrhythmias. Second trace: bradycardia induced by insertion of nasal temperature probe. Third trace: near sinus arrest induced by insertion of nasal temperature probe. Fourth trace: rapid junctional rhythm produced by atropine 0.4 mg i.v.

Train-of-four stimulation with a peripheral nerve stimulator produced four twitches. Insertion of the temperature probe in a similar manner was repeated six times in order to verify causality and record the events on the electrocardiogram. Each time a significant bradycardia or sinus arrest of almost 3 s duration (fig. 1: third ECG trace) immediately ensued. Removal of the probe always led to a resolution of the arrhythmia. Atropine 0.4 mg i.v. was administered, causing a rapid junctional rhythm of 90 beat min⁻¹ (fig. 1: fourth ECG trace). Insertion of the temperature probe after atropine produced no further change in the electrocardiogram. The operation proceeded uneventfully and normal sinus rhythm returned during operation. The infusion of alfentanil was discontinued 15 min before surgery ended. Total surgical time was 30 min. Residual neuromuscular block was antagonized with edrophonium 60 mg and atropine 0.6 mg i.v. at the conclusion of the operation. Nitrous oxide was discontinued, spontaneous ventilation resumed and the trachea was extubated. The patient had an uneventful postoperative course.

DISCUSSION

Rivard and Lebowitz [6] have reported bradycardia and asystole after induction of anaesthesia with alfentanil and suxamethonium. Laryngoscopy, tracheal intubation and ventilation with 100% oxygen restored sinus rhythm without other pharmacological therapy [6]. Maryniak and Bishop [2] also reported two cases of sinus arrest associated with induction of anaesthesia with alfentanil and suxamethonium that occurred when the larynx was sprayed with lignocaine [2]. In both patients, sinus rhythms returned spontaneously. Explanations for the occurrence of these rhythm disturbances include vagomimetic effects of opioids, especially potent and rapid acting agents such as alfentanil [8]; enhanced vagal tone induced by suxamethonium, because of breakdown products, ganglionic stimulation, or both; and stimulation of vagally innervated structures such as the larynx.

The significant bradycardia and brief sinus arrest sustained by our patient differed from those mentioned above. The arrhythmias occurred in our patient approximately 10-15 min after the initial injection of alfentanil 50 μg kg⁻¹. Maryniak and Bishop [2], and others [9], suggest that peak plasma concentrations of alfentanil and peak effect would have occurred sooner. Thus alfentanil probably did not cause the arrhythmias in our patient. Similarly, suxamethonium was not related temporally as a direct cause. Other possible mechanisms, for example surgical stimulus, were not present, as surgery had not yet begun. In addition, our patient had a stable haemodynamic state until the insertion of the nasal temperature probe which repeatedly and consistently caused the problem described.

Sensory innervation of the nasal mucosa is provided by the maxillary and ophthalmic divisions of the trigeminal nerve, which have their cell bodies located in the Gasserian ganglion. The anterior portion of the nose, the part most likely to have been stimulated in our patient, is innervated by the anterior ethmoid nerve, which arises from
the nasociliary branch of the ophthalmic division of the trigeminal nerve. Sympathetic and parasympathetic nerve fibres also accompany the anterior ethmoid nerve [10]. Interestingly, the ophthalmic division of the trigeminal nerve and Gasserian ganglion are involved also in the oculocardiac reflex. Bradycardias and other arrhythmias are well known to occur during eye surgery. Atropine-induced arrhythmias have been observed also during treatment of oculocardiac reflex related bradycardias [11]. Atropine also induced a junctional rhythm in our patient. In atropine moderate doses, may inhibit vagal influence on the atrio-ventricular node before affecting the sinus node, allowing for junctional escape rhythms [12]. Thus it appears that there exists a nasocardiac reflex similar to the oculocardiac reflex.

A search of the literature reveals reports describing various nasocardiac reflexes with a neurological basis in the trigeminovagal arc [10, 13–16]. These reports document that stimulation of the nasal mucosa may elicit a host of responses including apnoea, closure of the larynx, bradycardia, arrhythmias, vasomotor changes and sudden death [10, 13]. Death after trivial nasal stimulation, for example sneezing, has been reported in patients with myocardial disease [14, 15]. Baxandall and Thorn [16] have reported profound bradycardia in an anaesthetized patient during turbinate bone manipulation. These authors describe the maxillary division of the trigeminal nerve (the innervation of the turbinates) as the afferent limb of this nasocardiac reflex. In our patient, the anterior part of the nose was stimulated, implicating the anterior ethmoidal nerve and ophthalmic division of the trigeminal nerve as the afferent limb of the nasocardiac reflex [17]. In addition, our report is highlighted by the trivial nature of the stimulus producing bradycardia and sinus arrest.

In conclusion, we have observed that an apparently minor stimulus, the insertion of a nasal temperature probe, may induce significant bradyarrhythmias and sinus arrest. The role that anaesthesia with alfentanil and suxamethonium played in our patient is unknown. A neurological basis for a nasocardiac reflex exists and may be similar to the oculocardiac reflex in terms of mechanism and treatment.

REFERENCES