



Anaesthesiology

A RARE CASE OF SUDDEN ASYSTOLE IN A PATIENT WITH NO CO-MORBIDITIES DURING STAPEDOTOMY UNDER LOCAL ANAESTHESIA AND SEDATION WITH IV DEXMEDETOMIDINE

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ABSTRACT **INTRODUCTION** The Vagus nerve (CN X), the major parasympathetic output of the ANS, is attributed to various protective reflexes due to its extensive distribution in the thorax and abdomen. The reflex system consists of a somatic afferent, parasympathetic and sympathetic efferent fibres which are responsible for the deceleration- asystole. **CASE PRESENTATION** We report a case of a 46-year-old male, ASA-1, with no history of cardiac disease, diagnosed with right ear otosclerosis, undergoing stapedotomy under local anaesthesia. He was sedated with iv Dexmedetomidine infusion and maintaining stable vitals. Intraoperatively, when footplate of stapes was manipulated, patient initially complained of giddiness. He was reassured as it is commonly encountered due to the stimulation of oval window causing vertigo. In the next few minutes, when stapes piston was being inserted, patient suddenly jerked and developed intense bradycardia and asystole. Iv Atropine was administered, surgical stimulation was stopped and chest compressions were started with Return of Spontaneous Circulation (ROSC) in less than 20 seconds. Ventilation was assisted for a few minutes. Patient regained consciousness and surgery was completed without any further incidents. **CONCLUSION** Although most middle ear surgeries can be done under local anaesthesia and sedation, these vagal reflexes should be anticipated as they can be life-threatening.

KEYWORDS : Asystole, bradycardia, Dexmedetomidine, stapedotomy, vagal reflex

INTRODUCTION

Ear surgeries most commonly done are tympanoplasty, tympanostomy, myringotomy, stapedectomy, stapedotomy and mastoidectomy. Most of these surgeries can be done under local anaesthesia with the help of sedation. Surgical stimulation in this area can cause vagal stimulation causing severe bradycardia, and sometimes even asystole in the presence of other precipitating factors. We hereby report such a case of sudden asystole precipitated by IV Dexmedetomidine infusion.

CASE REPORT

We report a case of a 46-year-old male, weighing 60 kg, with no prior history of cardiac disease, no other co-morbidities, diagnosed with right ear otosclerosis, planned for stapedotomy under local anaesthesia. His pre-operative blood investigations, ECG and chest X-ray were normal.

On the day of surgery, standard ASA monitors were applied, initial HR-72/min, BP- 110/70mmHg and IV line was placed. He was sedated with IV Fentanyl (60mcg), midazolam(1mg) and started on IV Dexmedetomidine infusion (0.5 mcg/kg/hr). The patient was arousable and maintaining stable vitals. Within 20 minutes of starting the surgery, when footplate of stapes was manipulated by the operating surgeon, patient initially complained of giddiness. He was reassured as it is commonly encountered due to the manipulation around the footplate of stapes which lies over the oval window causing vertigo. HR: 65/min and BP: 100/60mmHg. In the next few minutes, when stapes piston was placed over the fenestra on the footplate and crimped to the long process of uncus, HR dropped from 60/min to <40/min with hypotension. Patient suddenly jerked and developed intense bradycardia and asystole.

Dexmedetomidine infusion was stopped, IV Atropine 0.6mg was administered, simultaneously surgical stimulus was removed and chest compressions were started, mask ventilation with 100% O₂ for a few minutes with Return of Spontaneous Circulation (ROSC) in less than 20 seconds. Patient regained consciousness, hemodynamically stable and surgery was completed successfully without any further incidents. A 12-lead ECG was taken post operatively and found to be normal.

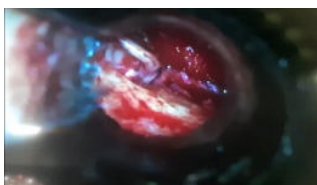


Fig 1. Intraoperative view of middle ear

DISCUSSION

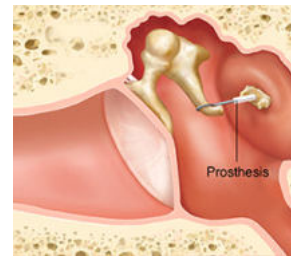


Fig 2. Stapedotomy

Stapedotomy is a common surgery done for the treatment of otosclerosis under conscious sedation. The common nerves encountered in the middle ear are the tympanic branch of the glossopharyngeal nerve (CN IX), the mandibular and maxillary branches of the trigeminal nerve (CN V), the mandibular, submandibular, chorda tympani and tympanic plexus of the facial nerve (CN VII) and Arnold's branch of the vagus nerve (CN X).

Dexmedetomidine is a potent, highly selective alpha-2 adrenergic receptor agonist with sedative, analgesic and anxiolytic properties. It causes dose dependent decrease in heart rate and blood pressure without clinically relevant respiratory depression.

Increase in parasympathetic outflow resulting from Dexmedetomidine, as well as the patient's autonomic response to abrupt surgical stimulation could be one of the contributing factors. The bradycardic response observed with dexmedetomidine can be augmented by the concurrent use of other medications with negative chronotropic and/or vagal effects.

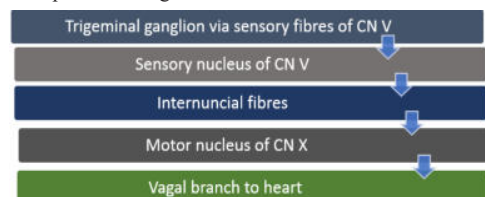


Fig 3. Trigemino-cardiac reflex

The bradycardia could be due to the Trigemino-cardiac reflex (TCR) where the afferent pathways may be through the mandibular/maxillary

divisions of the Trigeminal nerve and the efferent via the vagus nerve. It could also be the result of a Vasovagal reflex where efferent component results in an initial increase in sympathetic tone, followed by bradycardia and peripheral vasodilation.

Asystole in this scenario cannot be attributed to a specific cause. The bradycardia caused by Dexmedetomidine was further exaggerated by the vagal nerve stimulation.

CONCLUSION

Asystole in a patient receiving iv dexmedetomidine alone is rare though the incidence of bradycardia has been reported to be as high as 42% in patients who receive dexmedetomidine. On the other hand, vagal response due to ear stimulation is also quite common.

In summary we report a case of severe bradycardia progressing to asystole in the presence of a surgical manipulation in a patient receiving IV Dexmedetomidine for sedation. Prompt removal of the triggers resulted in complete recovery with no evidence of cardiovascular or neurologic compromise. Several factors combined to cause asystole in this patient, so caution must be exercised when using Dexmedetomidine in the presence of vagal/negative chronotropic stimuli.

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