Sevoflurane-induced arrhythmia in an adult and a child

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Abstract

When inhalational induction is indicated, sevoflurane is the most popular choice in both children and adults. Higher inspired concentrations of sevoflurane can cause adverse cardiac arrhythmias that are seen to disappear once the agent is discontinued. We report the occurrence of isorhythmic atrioventricular dissociation and junctional rhythm with absent P waves during sevoflurane anaesthesia in an adult and a child, respectively.

Key words: Anaesthesia, general, complications, arrhythmia, volatile agent, sevoflurane

Introduction

Among the inhalational agents, sevoflurane has the least myocardial depressant effect.[1] Sevoflurane does not sensitise the myocardium to arrhythmogenic effects of catecholamines[2] and has the least effect on cardiac conduction system.[3] Despite all the research demonstrating its safety, adverse cardiac arrhythmias such as nodal rhythm with bradycardia,[4] prolongation of QTc interval,[5] precipitation of torsade de pointes[6] and isorhythmic atrioventricular dissociation (ISRAVD)[7] have been reported with sevoflurane anaesthesia. Use of higher concentrations during induction may precipitate arrhythmias in susceptible individuals.

Case Reports

Case 1

A 60-year-old woman weighing 60 kg with carcinoma in the right breast was scheduled for elective modified radical mastectomy. She was a known hypertensive since 4 years, on treatment with oral metoprolol 50 mg twice daily and amlodipine

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10 mg once daily, with no other comorbidities. Preoperative pulse rate (PR) was 60/min and blood pressure (BP) was 140/70 mmHg. The patient had short neck with modified Mallampatti grade 4 and protruding incisors. Head extension was restricted, but mentohyoid and thyromental distances were normal. Her clinical examination was otherwise normal. Preoperative 12 lead electrocardiograms (ECG) showed sinus rhythm with a rate of 60/min. Echocardiography (ECHO) demonstrated concentric left ventricular hypertrophy with mild aortic regurgitation. Chest radiograph, blood investigations including serum electrolytes and urine investigations were normal. Diazepam 10 mg was administered orally the night before and 60 minutes before surgery, along with morning dose of metoprolol and amlodipine. In addition, morphine 7.5 mg was administered intramuscularly.

In operating room, ECG trace showed sinus rhythm with heart rate (HR) of 54/min. BP was 140/70 mmHg and SpO$_2$ was 96% at room air. Anaesthesia was induced with 8% sevoflurane in 100% oxygen after administering 1.5 mg morphine intravenously. During induction, the HR dropped to 44/min. ECG tracing showed regular rhythm, absent P waves with narrow QRS complexes. BP was 120/70 mmHg. Sevoflurane concentration was reduced to 5% and satisfactory Proseal Laryngeal mask airway insertion (size 3) was accomplished with suxamethonium 100 mg. Ventilation was satisfactory without leak or obstruction. Capnograph showed a square wave form. Immediately following induction, ECG trace in lead II showed dissociated upright P waves which remained within QRS for several beats, then reappeared and recede from QRS complex. ECG also showed intermittent electrical alternans [Figure 1] and BP dropped to 86/50 mmHg. Atropine (0.6 mg) was administered intravenously as 0.3 mg

Figure 1: Real-time picture from the monitor shows Electrical alternans with absent P waves in ECG trace. The difference in heart rate (ECG trace) and pulse rate (Pulse oximetry trace) is seen due to electrical alternans. Real-time picture from the monitor shows the inspired and expired concentration of sevoflurane

boluses. HR picked up to 50/min but ISRAVD persisted. BP picked up to 110/70 mmHg. Meanwhile, anaesthesia was maintained with sevoflurane 2% and 50% nitrous oxide in 50% oxygen. Neuromuscular blockade was achieved with atracurium and ventilation was controlled. End-tidal carbon dioxide ($EtCO_2$) was maintained between 35 and 37 mmHg. Since the patient was haemodynamically stable, it was decided to proceed with surgery. PR (40-50 beats/min) and rhythm (ISRAVD) remained unaltered throughout the procedure that lasted for 3 hours. Atropine 0.6 mg and mephenteramine 3 mg were administered intravenously when the PR dropped below 40/min and systolic BP below 90 mmHg. These interventions were required twice during the procedure. A discrepancy was observed between the HR recorded by the ECG and PR recorded by the pulse oximeter. HR was 50 to 60 beats/min but PR was 38 to 40 beats/min. At the end of surgery, sevoflurane was discontinued and neuromuscular blockade was reversed with a mixture of atropine (1.2 mg) and neostigmine (2.5 mg). LMA was removed once the patient was completely awake. Few minutes after removal of the LMA, rhythm reverted to sinus with PR of 50/min and BP was 118/79 mmHg. Postoperative ECG and ECHO findings were similar to those done preoperatively. Serum electrolytes in immediate postoperative period were normal. Metoprolol and amlodipine were discontinued and enalapril 5 mg once daily was started by the cardiologist postoperatively. Patient was observed in intensive care unit for the next 48 hours. Throughout the postoperative course, HR and rhythm remained normal.

Case 2

A 5-year-old boy weighing 15 kg was scheduled for left inguinal hernia repair. Premedication administered was 7 mg of oral midazolam, 30 minutes before surgery. In the operating room, baseline PR was 88/min with sinus rhythm; SpO_2 99% on room air and NIBP, 96/62 mmHg. Lead II was monitored using three leads with a multiparameter monitor.

Inhalation induction of anaesthesia was performed with sevoflurane in 100% oxygen (fresh gas flow 5 l/min) through T piece circuit with child breathing spontaneously at a rate of 20 to 24/min. Inspired concentration of sevoflurane was increased in 2% increments every minute up to 8% concentration. The $EtCO_2$ was between 31 and 33 mmHg, as measured through the angle piece interfaced between the face mask and the breathing circuit. After breathing 8% sevoflurane for 2 minutes, the child did not show any motor response to jaw thrust. Intravenous access was secured. Sudden drop in HR to 56/min was noticed and the ECG revealed the presence of junctional rhythm with absent P waves and regular narrow QRS complexes. At that moment, SpO_2 was 99% and BP was 66/36 mmHg. LMA (size 2) insertion was accomplished in the first attempt. Sevoflurane was discontinued and isoflurane was started. Intravenous fentanyl (30 μg) was administered and anaesthesia was maintained with 1.5% isoflurane in a mixture of 50% nitrous oxide in oxygen (fresh gas flow rate 5 l/min). Regular sinus rhythm was restored 2 minutes after discontinuation of sevoflurane. Sinus rhythm persisted thereafter. Spontaneous ventilation was maintained throughout the procedure and LMA was removed at the end. The child recovered well.

Discussion

American College of Cardiology/American Heart Association recommends using volatile agents during non-cardiac surgery for the maintenance of general anaesthesia in haemodynamically stable patients at risk for myocardial ischemia.[8] Anticipating a difficult airway and considering the patient's cardiac status, and associated hypertension, sevoflurane was chosen for induction of anaesthesia. ISRAVD was recorded during induction of anaesthesia with sevoflurane which reverted back to sinus rhythm after discontinuation of sevoflurane during emergence.

ISRAVD has been observed with the use of volatile agents.[9] In ISRAVD, 2 pacemakers (sinus and AV nodes) fortuitously discharge at the same or nearly similar rate, without antegrade or retrograde conduction across the AV node. Either slowing of the sinus node discharge rate or the emergence of a slightly faster subsidiary pacemaker controlling the ventricles is the common initiating event.[10] Electrocardiographically, the dissociated and upright 'P' waves approaches the narrow QRS complex, disappears within it for several beats, then

reappears and recedes from the QRS and this sequence may be repeated.[11] The same was observed in the ECG recording in the present case [Figure 1].

From the limited number of experimental studies, the development of ISRAVD and the particular electrocardiographic patterns resulting from there are dependent upon relative discharge rate of the dominant atrial and subsidiary pacemakers, presence or absence of retrograde conduction, chronotropic response to atrial stretch, baroreceptor-mediated autonomic nervous system output and responsiveness of the sinus node.[10]

High incidence of nodal rhythm (20%) and bradycardia with use of sevoflurane has been reported in unpremedicated infants. The onset of nodal rhythm was significantly earlier in the high-concentration technique than with incremental induction technique. On the other hand, Nakaigawa *et al.* demonstrated that up to 2 MAC (minimal alveolar concentration) sevoflurane does not affect cardiac conduction system significantly.[8] In both our patients, 8% sevoflurane was used for induction that possibly resulted in ISRAVD and junctional rhythm. ISRAVD was possibly precipitated by the volatile agent affecting Ca^{2+} release from the sarcoplasmic reticulum, resulting in depression of Ca^{2+} slow inward current in the myocardium.[12] In patients on Ca^{2+} channel and β-adrenergic blocker therapy, the arrhythmogenic potential of sevoflurane is likely to be enhanced as Ca^{2+} channel blockers inhibit the entry of Ca^{2+} into the cell or its mobilisation from intracellular stores, while β-adrenergic blockers depresses sinus rate and AV node conduction.[13]

Discontinuing the volatile agent is possibly the best method of treating ISRAVD. Abe *et al.* reported a case of intraoperative torsades de pointes ventricular tachycardia and ventricular fibrillation during sevoflurane anaesthesia, which persisted with DC shock but reverted to sinus rhythm after 10 minutes of discontinuation of sevoflurane and after the patient was awakened.[6] A discrepancy was observed between the HR and the PR due to intermittent electrical alternans in the ECG. It is therefore wise to rely on the PR rather than the HR in such cases to treat bradycardia. Haemodynamic alterations associated with ISRAVD are usually minor and well tolerated.[11] Rarely, sevoflurane-induced suppression of baroreceptor reflex activity[14] might result in desynchronisation between the atria and ventricle causing haemodynamic instability which may require electrical pacing.

To conclude, sevoflurane in high concentration (8%) should be used with caution in children and in conditions where primary pacemaker activity is suppressed, as in elderly. Caution should also be exercised with its use in patients on Ca^{2+} channel and b blocker therapy.

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