Termination of supraventricular tachycardia by propofol

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It is known that propofol can have effects on atrioventricular node conduction. We report a case where administration of propofol was associated with termination of supraventricular tachycardia. Although a similar phenomenon has been reported in children, this is the first such case in an adult.

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Propofol has been shown to have a direct concentration-dependant inhibitory effect on the cardiac conduction system in isolated rabbit hearts. Although there have been instances of termination of supraventricular tachycardia (SVT) and suppression of premature atrial contractions during propofol anaesthesia in children, no such case has been reported in an adult.

Case report

A 68-yr-old man weighing approximately 80 kg presented to the emergency department with a 4 h history of central chest pain, suggestive of angina, and palpitations. He had a history of myocardial infarction 4 yr previously and was taking oral enalapril 20 mg daily for hypertension. He was a non-smoker and had not had any general anaesthesia before. He had taken solid food about 30 min before the onset of symptoms. On examination, he was oriented, afibrile with no pallor. His pulse rate was 160 min⁻¹ and his arterial pressure varied between 110/60 and 85/45 mm Hg. On auscultation, the breath sounds were normal with no added sounds. A 12-lead electrocardiogram had shown a rhythm suggestive of SVT with a heart rate of 170 min⁻¹. Adenosine 6 mg intravenously had transiently converted the rhythm to atrial flutter with a ventricular rate of 60 min⁻¹, which reverted to SVT (Fig. 1). Carotid sinus massage failed to have any effect on the rhythm. Serum concentrations of potassium, magnesium, and calcium were normal. As the arterial pressure was labile, it was decided that cardioversion would be more suitable than drug therapy.

Because of a potential full stomach, the anaesthetic plan was for a rapid sequence induction, cricoid pressure, and tracheal intubation followed by the cardioversion. After pre-oxygenation, cricoid pressure was applied and anaesthesia was induced with propofol. After about 100 mg of propofol had been given, it was noticed that the rhythm had converted to sinus (Fig. 1).

Succinylcholine was not given. As the patient was unconscious, he was turned on to the left lateral position and cricoid pressure maintained until he showed signs of return of protective airway reflexes and awakening. His arterial pressure was 90/60 mm Hg and the sinus rhythm persisted. The patient was kept under observation. After about 2 h, he complained of severe central chest pain along with electrocardiographic changes of myocardial ischaemia. He was transferred to the coronary care unit and underwent thrombolysis with streptokinase. The further clinical course was uneventful and the patient remained in sinus rhythm until discharge.

Discussion

There has been recent interest in the effects of propofol on the conduction system of the heart. This has partly been stimulated by the increasing use of propofol in cardiac electrophysiological studies along with radiofrequency catheter ablation (RFCA) to treat cardiac arrhythmias. Although it has been reported that propofol infusion made it difficult for the arrhythmias to be induced during such procedures, RFCA has been successfully performed in children under propofol anaesthesia for most arrhythmias except ectopic atrial tachycardia. Propofol was noted not to alter sinoatrial or atrioventricular conduction in paediatric patients undergoing RFCA when it was given after induction of anaesthesia with other agents.

In one of the first cases reported by Hermann and Vettermann, the child had a chronic ectopic atrial tachycardia of around 150 min⁻¹ that was being treated with verapamil and methyl-digoxin. The cardiac rhythm converted to sinus with a rate of 75 min⁻¹ during propofol...
anaesthesia, which reverted to tachycardia about 35 min after cessation of the anaesthetic. In our case, the rhythm remained in sinus after it was converted by propofol.

Several mechanisms have been proposed as an explanation for this effect of propofol. They include indirect effects such as attenuated sympathetic outflow, enhanced vagal tone, altered baroreceptor reflex sensitivity, and direct effects such as prolonged atrioventricular conduction. Animal studies have shown that propofol would be most effective at filtering atrial impulses during supraventricular tachydysrhythmias. Animal studies have also revealed different effects on neonatal and adult hearts with a 10 μM concentration of propofol affecting both but a 100 μM concentration affecting only the neonatal hearts. In adult humans, the mean blood concentration of propofol at the onset of unconsciousness after a bolus dose of 2 mg kg⁻¹ is approximately 56 μM and an adequate level of anaesthesia is maintained with blood concentrations ranging from 19–25 μM (but note that the concentration of free propofol will be substantially less than this because of the extent of plasma protein binding of the drug). Indeed, transient atrioventricular conduction block has been reported in an adult who received propofol. Paradoxically, propofol anaesthesia has also been associated with inducing SVT that deteriorated to ventricular tachycardia.

Although cricoid pressure was applied in this case, it is unlikely that it would have contributed to the termination of the rhythm for several reasons: the site of the cricoid pressure is away from that for carotid sinus massage, previous attempts at carotid sinus massage were unsuccessful and the conversion to sinus rhythm was noted during propofol injection.

In summary, this report describes a case of SVT in an adult, which converted to sinus rhythm during administration of propofol.

References