Update in Anaesthesia

CASE REPORT

Magnesium treatment of epinephrine-induced tachyarrhythmia during halothane anaesthesia

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SUMMARY
Halothane is known to be associated with ventricular arrhythmias during anaesthesia and also to lower the threshold for ventricular arrhythmias induced by epinephrine (adrenaline). Intravenous magnesium sulphate has been shown to be an effective treatment for a wide range of cardiac arrhythmias, including ventricular arrhythmias. We present a case report of a patient who developed a broad complex tachycardia after sub-mucosal injection of epinehrine-containing local anaesthetic solution during halothane anaesthesia, who was successfully treated with intravenous magnesium sulphate.

CASE REPORT
A 24-year-old, 50kg ASA grade 1 male presented for repair of cleft palate at Mulago Hospital, Kampala, Uganda. He had undergone a previous uneventful cleft lip repair and had no past medical history of note, in particular, no history of chest pain or palpitations. He worked as a farmer, had excellent exercise tolerance and there was no history of recent illness.

Anaesthesia was induced with thiopentone 500mg and suxamethonium 100mg and the trachea was intubated. Anaesthesia was maintained with 2% halothane in oxygen via a Boyle's machine with a circle system, using manual ventilation with an Ambu® bag. The patient was monitored with a pulse oximeter, manual sphygmomanometer and a precordial stethoscope. The palate was infiltrated with 10ml 0.5% lidocaine (lignocaine) with epinephrine 1:80,000 for haemostasis prior to the start of surgery (50mg lidocaine and 125mcg epinephrine). Approximately five minutes after the initial incision, the patient developed an irregular heartbeat suggestive of multiple ectopic beats, with his systolic blood pressure maintained at 140mmHg. No alternative volatile agent was available so the inspired concentration was reduced to 0.5%. 50mg intramuscular pethidine was administered and the ventilation rate increased to reduce the carbon dioxide level (although there was no end-tidal carbon dioxide monitor available). The ectopic beats continued and then about 5 minutes later the patient suddenly developed a sustained tachycardia at a rate of 180 beats per minute, associated with a weak but palpable pulse and a fall in systolic blood pressure to 60mmHg. The halothane was reduced further to 0.25% and 100mg intravenous ketamine was administered to maintain anaesthesia. An electrocardiogram (ECG) was brought from another theatre that demonstrated a regular broad complex tachyarrhythmia suggestive of ventricular tachycardia. Lidocaine 100mg IV was administered with no effect. There was no defibrillator and amiodarone was not available. Magnesium sulphate was brought from the labour ward and 25mg.kg^{-1} (1.25g) was administered by slow intravenous injection. By the time the injection was completed, the tachyarrhythmia had been terminated, approximately 10 minutes after it had started. The blood pressure was restored to normal and the operation was completed successfully without any further problem. The patient was monitored with ECG, blood pressure and pulse oximetry in the recovery room and observed for two hours with no recurrence of the arrhythmia. The patient made an uneventful postoperative recovery and was discharged home three days later.

DISCUSSION
Halothane remains the volatile agent of choice many developing countries and is unfortunately often administered without the benefits of ECG monitoring.[1] Halothane has well-documented effects on the heart, including myocardial depression, bradycardia, A-V conduction disturbances (via an effect on the SA and AV nodes), promotion of re-entrant tachycardia in association with myocardial ischaemia and reduction in the threshold for epinephrine-induced ventricular arrhythmias. The latter may be associated with high levels of endogenous epinephrine (light anaesthesia, high end tidal carbon dioxide levels), or epinephrine when injected subcutaneously or by sub-mucosal infiltration for surgical haemostasis.[2] The effects of halothane on the heart may be mediated by interactions with ion channels in the cardiac cells, particularly inhibition calcium channels responsible for contraction of the cardiac myocytes and maintaining the refractory period of the cardiac action potential, by promoting adrenergic receptor stimulation, or via inhibition of potassium channels to cause prolongation of the QT

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interval and malignant tachyarrhythmia.[3] Traditional methods to avoid halothane-induced myocardial side effects are to avoid deep halothane anaesthesia, avoid light anaesthesia by using a balanced anaesthesia technique with adequate analgesia, ensure adequate ventilation, and limit the dose of injected epinephrine to no more than 2mcg.kg^{-1}.

In the case described, the anaesthetic technique could have promoted the development of ventricular tachycardia through inadequate analgesia, ineffective ventilation due to lack of muscle relaxation in a young fit patient, and infiltration of the surgical site with a high concentration of adrenaline. This may have been avoided by a balanced anaesthesia technique using an analgesic agent prior to the start of the surgery, ideally a non-depolarising muscle relaxant, and a more dilute solution of epinephrine for wound infiltration (1:200,000 solution containing 5mcg.ml^{-1} epinephrine). There was no alternative volatile agent to halothane to maintain anaesthesia, so the concentration of halothane was reduced and pethidine and ketamine administered for analgesia and to maintain anaesthesia respectively.

Ventricular tachycardia is a potentially life threatening tachyarrhythmia that may deteriorate to ventricular fibrillation. The recommended treatment of an adult with stable ventricular tachycardia is chemical cardioversion with a loading dose of amiodaraone 300mg IV over 20-60 minutes, then 900mg amiodarone IV over 24 hours.[4] Lidocaine 100mg has been shown to be effective, but in only 30-40% of cases.[4] Synchronised electrical cardioversion is recommended for unstable VT, using an initial synchronised shock of 200J, increasing to 360J and loading with amiodarone if necessary. Unfortunately neither a defibrillator nor amiodarone were available during this case, and lidocaine was ineffective.

Magnesium is an essential cation in the body, and has a wide range of physiological functions, such as involvement in neuronal excitability, muscle contraction, control of bronchial and vasomotor tone, neurotransmitter release and cardiac excitability.[5] It is often said to act as a physiological calcium antagonist. It is indicated in the treatment of eclampsia, severe asthma and in particular, for the treatment of polymorphic ventricular tachycardia associated with prolonged QT syndrome. Magnesium acts as an effective membrane stabiliser, and has also been reported to be useful in the treatment of atrial and ventricular arrhythmias after cardiac and thoracic surgery, to reduce the ventricular rate in atrial fibrillation and Wolf-Parkinson-White and for digoxin induced arrhythmias. The patient was hypotensive so a small dose of magnesium was used in this case to avoid further fall in blood pressure (25mg.kg^{-1}), but it was effective in terminating the ventricular tachycardia immediately.

To our knowledge, this is the first case report to demonstrate that intravenous magnesium can be used terminate epinephrine-induced ventricular tachycardia during halothane anaesthesia in humans. Animal studies have suggested that magnesium can be used to reduce the duration of epinephrine-induced tachyarrhythmia during halothane anaesthesia,[6] but the exact mechanism is unclear. Magnesium is an inexpensive drug that is in common use in clinical practice, including in developing countries.

We recommend that patients receiving halothane anaesthesia should be closely monitored for arrhythmias, that deep halothane anaesthesia be avoided and that the dose of injected epinephrine to induce vasoconstriction at the surgical site should be kept within safe limits. Furthermore, we recommend that magnesium sulphate should be available in theatre when halothane is used and that intravenous magnesium sulphate should be considered early in the treatment of epinephrine-induced tachyarrhythmia during halothane anaesthesia.

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