Isoflurane anaesthesia for a patient with long Q-T syndrome

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A case report of a patient known to have long Q-T syndrome (LQTS) undergoing elective oral surgery is presented. While nitrous oxide-narcotic, nitrous oxide-enflurane, or nitrous oxide-halothane techniques for anaesthetic management of LQTS have been previously reported, we report the use of nitrous oxide-isoflurane for the maintenance of anaesthesia. The authors feel that isoflurane is a safe anaesthetic agent for use in LQTS.

Key words

HEART: long Q-T syndrome, electrocardiography;
ANAESTHETICS, VOLATILE: isoflurane;
SURGERY: oral.

Congenital long Q-T syndrome (LQTS) with syncope episodes secondary to ventricular tachyarrhythmias following emotional or physical stress has been described.¹ Ventricular tachyarrhythmias typically follow stimulation of the sympathetic nervous system secondary to stress, including the stress of anaesthesia and surgery. Indeed, unrecognized or improperly managed congenital LQTS may lead to intraoperative ventricular tachyarrhythmias and even death.²

Although extensive medical literature on LQTS exists,¹ descriptions of anaesthetic management are limited. Review of the literature indicates there have been only seven patients reported with this syndrome who have received anaesthesia.²⁻⁷ These patients have been managed with nitrous oxide-enflurane, nitrous oxide-halothane, and nitrous

oxide-narcotic techniques. The following case is, to our knowledge, the first report of a patient with LQTS anaesthetized with isoflurane.

Case report

A twenty-seven-year-old woman was scheduled for elective mandibular osteotomy. The diagnosis of LQTS was made three years prior to admission after the patient presented with a history of syncope episodes since childhood. The patient had been well controlled on propranolol and diphenylhydantoin for three years. Three family members had LQTS with one death from the syndrome. There was no personal or family history of deafness. Electrocardiograms prior to admission documented long Q-T intervals with episodes of ventricular tachycardia. The patient's history was otherwise unremarkable. Physical examination on admission revealed a well-developed, well-nourished, white female in no acute distress. Heart rate was regular at 60 beats per minute with a blood pressure of 130/85 torr, respiratory rate was 16 per minute, and temperature was 36.7° C. The patient was 5'8" tall and weighed 57 Kg. Examination of the heart, chest, abdomen, and CNS were within normal limits. Haemoglobin (hgb), haematocrit, blood urea nitrogen, serum creatinine, sodium (Na), potassium (K), and calcium were within the normal range on admission. A standard 12 lead electrocardiogram (see Figure) showed a sinus bradycardia with a rate of 60 beats per minute. The Q-T interval was 0.44 s and there were prominent U waves.

Hydromorphone hydrochloride 0.5 mg, hydroxyzine hydrochloride 50 mg, and glycopyrrolate 0.2 mg intramuscularly were given two hours prior to surgery. On arrival to the preoperative holding area, we instituted continuous electrocardiographic monitoring. Diazepam 10 mg and morphine sulfate 2.5 mg intravenously were administered to alleviate

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the stress of preparing the patient for surgery. The patient's left radial artery was cannulated. The patient arrived in the operating room sedated but awake with a heart rate of 60 beats per minute and blood pressure of 125/75 torr. The patient's nose was sprayed with ten per cent xylocaine 0.25 ml and 0.5 per cent neosynephrine 0.25 ml. Anaesthesia was induced with thiamylal sodium 450 mg intravenously. A 7.0 mm endotracheal tube was placed through the nares and into the oropharynx. Succinylcholine 60 mg was administered and the patient was ventilated with 100 per cent oxygen through the tube. Xylocaine 100 mg was administered intravenously and the trachea sprayed with 3 ml of four per cent xylocaine and intubated. Following intubation, anaesthesia was maintained with 60 per cent nitrous oxide and 1–2.5 per cent inspired isoflurane concentration with assisted ventilation. There was no rise in blood pressure or heart rate following intubation. The usual surgical infiltration with epinephrine containing local anaesthetics was omitted. The blood pressure rose from 110/80 to 125/80 torr and the heart rate from 60 to 85 beats per minute following surgical incision. The electrocardiogram revealed sinus rhythm for the duration of the surgical procedure. The patient's blood pressure remained stable at approximately 110/65 torr with a heart rate of 70 beats per minute. Total crystalloid administered during surgery was 2100 ml with an estimated blood loss of 700 ml. Intraoperative laboratory values were pH 7.37, PCO_2 44 torr, PO_2 202 torr, Na 140 meq/L, K 4.1 meq/L and hgb 13.6 gm%.

After cessation of anaesthesia, the patient remained intubated with her jaw wired closed. She was transported to the recovery room, breathing spontaneously. On arrival in the recovery room, her blood pressure was 117/76 torr with a heart rate of 59 beats per minute and sinus rhythm on the cardiac monitor. The patient was placed on 40 per cent humidified oxygen via T-tube. Laboratory values in the recovery room were pH 7.35, PCO_2 46 torr, PO_2 175 torr, Na 139 meq/L, K 4.7 meq/L, and hgb 12.5 gm%. The patient remained intubated for 3.5 hours postoperatively without apparent discomfort. Xylocaine 100 mg intravenously was given prophylactically prior to extubation. The patient's heart rate increased to 105 beats per minute following extubation, but no ventricular ectopy was noted. Propranolol 1 mg intravenously slowed the heart

FIGURE Preoperative ECG showing sinus bradycardia at 56 beats per minute with 0.44s Q-T interval and prominent U waves.

rate to 88 beats per minute. The patient remained in the recovery room with electrocardiographic monitoring for 24 hours. The blood pressure and heart rate remained stable and there was no ventricular ectopy noted. All preoperative medications were resumed. The remainder of the patient's hospital course was uneventful and she was discharged home on the third postoperative day.

Discussion

With an increasing number of patients with LQTS being identified and coming for surgery, it is important for the anaesthetist to understand this syndrome and its anaesthetic implications. Although the exact pathogenesis remains unclear, it appears an imbalance in the adrenergic stimulation of the myocardium with either an increased left sympathetic tone or decreased right sympathetic tone is the most likely etiology.¹ Events leading to an increased sympathetic tone with an increase in circulating catecholamines may precipitate ventricular tachyarrhythmias in the patient with LQTS. Anaesthetic management should be directed toward minimizing sympathetic stimulation.

Several anaesthetic agents have been used in patients with LQTS. Halothane sensitizes the myocardium to circulating catecholamines and may precipitate ventricular tachyarrhythmias; hence, halothane is probably best avoided in patients with LQTS. Nitrous oxide-enflurane and nitrous oxide-narcotic techniques have been used without incident.^{3–7}

Because of its pharmacological properties, isoflurane should be a safe anaesthetic agent in patients with LQTS. Philbin and Lowenstein¹⁰ report no clinically significant Beta adrenergic stimulatory

effects with isoflurane in dogs. Joas and Stevens¹¹ report that isoflurane sensitizes the myocardium to exogenous epinephrine to a lesser extent than halothane and that as the depth of anaesthesia is increased, progressively more epinephrine is needed to induce ventricular arrhythmias. Johnston *et al.*¹² reported similar findings during isoflurane and halothane anaesthesia in humans.

We suggest that isoflurane is a safe anaesthetic in the presence of LQTS. However, further study is required to determine the optimal anaesthetic management of these patients.

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Résumé

On rapporte l'observation d'un malade porteur du syndrome du Q-T prolongé qui a subi de la chirurgie stomatologique de façon élektive.

Les publications médicales font état de techniques d'anesthésie pour ces malades employant l'association morphinique–protoxyde d'azote protoxyde–enflurane ou protoxyde–halothane. Nous présentons ici un cas de technique employant une combinaison protoxyde d'azote et isoflurane. Nous avons l'impression que l'isoflurane est un agent qu'on peut employer avec sécurité chez des malades porteurs de ce syndrome.