Sevoflurane is a potent volatile anaesthetic which was synthesized in the United States more than two decades ago. Wallin and colleagues described the physical, chemical and pharmacological characteristics of the agent [1]. The blood/gas partition coefficient has been reported to be 0.60–0.63, which is small enough to ensure fast induction and emergence from anaesthesia. The MAC of sevoflurane has been described as 1.71 %, AD₉₅ 2.07 % in oxygen, and 0.66 % and 0.94 %, respectively, in the presence of 63.5 % of nitrous oxide [2]. In common with other volatile anaesthetics, sevoflurane was shown to trigger malignant hyperthermia in susceptible swine [3]. Since May 1990, the anaesthetic has been available commercially for clinical use in Japan. We have anaesthetized 74 children with this agent and have had one case of clonic and tonic movements of the extremities during induction.

CASE REPORT

A 9-yr-old, 132-cm, 29-kg girl was scheduled to undergo elective plastic surgery for a postoperative scar caused by repair of a bilateral cleft lip. She had experienced general anaesthesia twice without problems: with oxygen–nitrous oxide–methoxyflurane at 4 months of age and with oxygen–nitrous oxide–enflurane at 2 yr. She had no history of seizure activity or drug allergies and was not taking medications. On her admission to hospital, routine laboratory biochemical tests were normal, including a serum CPK concentration of 171 iu litre⁻¹. Pre-operative ECG and chest x-ray were normal. In her family history she had an aunt who died shortly after appendicectomy under spinal anaesthesia at 19 years of age. Although the details of the aunt’s death were not clear, no indications were reported suggesting malignant hyperthermia. There was no family history of neurological disorders.

One hour before induction of anaesthesia, the patient was given atropine 0.2 mg i.m. and pentobarbitone 60 mg i.m. Upon arrival in the operating room she was well sedated. Anaesthesia was induced with oxygen 2 litre min⁻¹, nitrous oxide 4 litre min⁻¹ and incremental doses of 0.5–4.0 % sevoflurane (via an Ohmeda Sevotec 3, BOC Health Care, England), inspired via a face mask connected to a semi-closed circle system. SpO₂ was monitored continuously with a pulse oximeter, and was never less than 98 %. About 5 min after the start of induction when 2 % sevoflurane was being inhaled, the patient showed slow clonic movements of the upper extremities, as if she was in the excitement stage of anaesthesia. Ventilation was assisted easily and then controlled manually using a face mask. The concentration of sevoflurane was increased to 4 % and maintained for several minutes. The slow clonic movements of the upper extremities changed to tonic movements of the upper and lower extremities, with hyperextension of the upper extremities. As the patient had experienced enflurane anaesthesia without problems when aged 2 yr, we changed from 4 % sevoflurane to 1 % enflurane (after about 20 min of sevoflurane anaesthesia). The tonic movement was not affected and was sustained until enflurane was discontinued at 25 min of anaesthesia and the operation was cancelled. A second arterial sample at this time showed respiratory alkalosis (pH 7.55, PaO₂ 3.2 kPa, PaCO₂ 28.5 kPa, HCO₃⁻ 21.0 mmol litre⁻¹, base excess -0.8 mmol litre⁻¹). A cannula was inserted into the left saphenous vein and thiamyl 75 mg administered i.v., which did not cease or enhance the tonic movement. As the patient had experienced enflurane anaesthesia without problems when aged 2 yr, we changed from 4 % sevoflurane to 1 % enflurane (after about 20 min of sevoflurane anaesthesia). The tonic movement was not affected and was sustained until enflurane was discontinued at 25 min of anaesthesia and the operation was cancelled. A second arterial sample after disappearance of the tonic movement showed a slight respiratory alkalosis (pH 7.46, PaCO₂ 4.3 kPa, PaO₂ 76.3 kPa, HCO₃⁻ 22.6 mmol litre⁻¹, base excess -0.1 mmol litre⁻¹).
Throughout anaesthesia, ventilation was controlled easily with a face mask and rectal temperature remained normal (37.5–37.6 °C). Arterial pressure and heart rate did not change greatly from baseline values and the ECG showed no abnormalities. During and after emergence, the patient had no problems other than slight muscle pain in both arms, which continued until the next day. On the next day there were no biochemical abnormalities except an increased serum CPK concentration of 442 iu litre⁻¹, which decreased gradually to 156 iu litre⁻¹ on the fifth day after anaesthesia. Port wine urine was not observed. She was examined by a neurologist who reported no abnormal findings.

One week later, the patient was again scheduled for surgery. Anaesthesia was induced with fentanyl 75 µg i.v., midazolam 3 mg i.v. and thiamylal 50 mg i.v., and the trachea was intubated after administration of vecuronium 3 mg i.v. Anaesthesia was maintained for 7 h of surgery with 0.5–1.5% enflurane in oxygen 2 litre min⁻¹ and nitrous oxide 2 litre min⁻¹. The course of anaesthesia was uneventful and she had no postoperative problems.

**DISCUSSION**

Sevoflurane does not sensitise the myocardium to adrenaline, which implies relative freedom from cardiac arrhythmias caused by endogenous or injected catecholamines [1]. Its low blood/gas partition coefficient ensures ease and rapidity of induction and emergence from anaesthesia. These two properties have made the agent useful for anaesthetizing young children. Several authors have reported myoclonic movements or seizures during and after anaesthesia with enflurane, especially combined with respiratory alkalosis [4–9]. In sevoflurane-anaesthetized adult male volunteers, Holaday and Smith did not find any significant changes in several variables, including EEG [10]. Avramov and colleagues induced anaesthesia in five young male volunteers (23–25 yr) with 4% sevoflurane in oxygen and observed high amplitude, rhythmic slow waves on EEG at 1–3 min [11]. They did not describe any seizure movements in the volunteers. When incremental doses of the agent were administered (10 min each for 1, 2 and 4 %), high amplitude slow waves did not appear. In the present case, we induced anaesthesia with incremental doses of sevoflurane and nitrous oxide in oxygen. The rate of increase in concentration, however, was high, probably resulting in a condition similar to that produced by Avramov and colleagues [11].

The failure of thiamylal 75 mg i.v. to stop the tonic movement indicates that the movement was not caused by light anaesthesia. This finding is consistent with the results of animal experiments with enflurane and i.v. anaesthetics. Darimont and Jenkins reported that diazepam, thiopentone, methohexitone and ketamine enhanced EEG seizure activity produced by enflurane in cats [12]. Similar results were obtained with thiopentone by Furgang and Sohn, with the exception that thiopentone suppressed the spike activity of the EEG when the cats were at a greater depth of enflurane anaesthesia [13]. As the tonic movement in our patient was associated with respiratory alkalosis, subsequently we measured arterial blood-gas tensions during induction of anaesthesia with sevoflurane in concentrations up to 4 %. Of 14 children examined, seven had a PaCO₂ less than 4 kPa without manifesting any seizure-like movements.

It is not possible to determine the aetiology of the clonic and tonic movements noted in our patient, as we did not monitor the EEG. The movements might have been a manifestation of seizure activity in the CNS, or might have been myoclonus in the extremities, which was reported to occur without EEG seizure activities after i.v. administration of sufentanil in a 65-yr-old male with alcohol abuse [14].

**REFERENCES**