

## Lesson of the Week

### Suxamethonium is dangerous in polyneuropathy

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Artificial ventilation, tracheostomy, and bronchoscopy are often essential procedures in patients with polyneuropathy who develop respiratory failure. We report on four patients who had life-threatening ventricular arrhythmias during induction of general anaesthesia for these procedures.

#### Case reports

*Case 1*—A 23-year-old woman developed weak arms and legs and diplopia during her second pregnancy. On examination she had a mild peripheral sensory disturbance, definite distal muscle weakness, and absent tendon reflexes. Cerebrospinal fluid (CSF) protein was 2.0 g/l. The neuropathy relapsed over the next five years until she presented with deteriorating pulmonary function and an inability to cough due to muscle weakness, for which she required artificial ventilation. She was given atropine and thiopentone to induce anaesthesia and then paralysed with suxamethonium (succinylcholine) before intubation. During this procedure she developed a ventricular tachycardia, which returned promptly to sinus rhythm with intravenous lignocaine and DC cardioversion.

*Case 2*—A 42-year-old man presented with paraesthesia in the hands and feet and left-sided facial weakness. Over the next seven months his arms and legs became progressively weak, extending proximally. He was almost completely paralysed in the arms, legs, and trunk and had a peripheral sensory disturbance and absent reflexes. CSF protein concentration was raised at 2.84 g/l. Because he could not cough effectively and his respiratory function was deteriorating he required artificial ventilation. He was given atropine 0.3 mg, thiopentone 200 mg, and suxamethonium 75 mg intravenously before intubation. His pulse was lost immediately, and ECG monitoring showed ventricular tachycardia, which progressed to ventricular fibrillation. He was given intravenous lignocaine and cardioverted on four occasions before returning to sinus rhythm. No further arrhythmias were noted.

*Case 3*—A 75-year-old man was admitted with a five-month history of progressive weakness of the legs. On examination he had flaccid paralysis of all muscle groups with absent tendon reflexes. CSF protein concentration was 3.2 g/l. He developed purulent sputum and a chest radiograph showed consolidation in the right lower lobe. Because he was unable to cough and blood gas concentrations were falling he was intubated, artificial

**Suxamethonium is likely to cause ventricular arrhythmias in patients with polyneuropathy**

ventilation was started, and five days later a tracheostomy was performed. There were no recorded dysrhythmias during anaesthesia with thiopentone and suxamethonium for either of these procedures, but six days later the tracheostomy had to be refashioned and he was given a further anaesthetic (thiopentone 300 mg and suxamethonium 50 mg). During this procedure he developed ventricular tachycardia and then ventricular fibrillation. Sinus rhythm was restored with external cardiac massage and intravenous lignocaine.

*Case 4*—A 53-year-old woman presented with a six-month history of progressive weakness of the arms preceded by sensory symptoms in the hands and face. On examination she had appreciable proximal weakness of the arms and legs with absent reflexes. CSF protein concentration was normal. She had difficulty in coughing and developed purulent sputum. Chest radiographs showed collapse of the left lower lobe and it was necessary to remove secretions using rigid bronchoscopy. Intravenous anaesthesia was induced with atropine 0.4 mg, thiopentone 250 mg, and suxamethonium 50 mg. Immediately after the bronchoscope was inserted her pulse was lost. ECG monitoring showed ventricular tachycardia, which reverted spontaneously to sinus rhythm. Subsequent investigations showed that she had a relapsing polyneuropathy associated with paraproteinaemia.

#### Comment

Three possible causes of the ventricular arrhythmias in our patients were autonomic dysfunction, hypoxaemia, and hyperkalaemia after giving suxamethonium. Autonomic dysfunction in the Guillain-Barré syndrome has been severe enough to cause sudden death,<sup>1</sup> but ventricular arrhythmias have not been reported in these patients. Autonomic reflexes related to endotracheal intubation may have caused the arrhythmias. If this was the mechanism, however, we might have expected to see similar arrhythmia during the manipulation associated with subsequent tracheal aspiration, but this did not happen. All four patients had normal arterial partial pressures of oxygen (case 3 breathing 40% oxygen and the others breathing air) on the day that the anaesthetic was given and all were given oxygen before anaesthesia, which makes hypoxaemia an improbable cause of the arrhythmias. We think that the most likely reason for the ventricular arrhythmias was transient hyperkalaemia after intravenous suxamethonium although, unfortunately, no measurements of serum potassium concentrations were made at the time the arrhythmias occurred.

Suxamethonium-induced hyperkalaemia has been well

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documented in patients with upper motor neurone paralysis secondary to spinal cord injury, tumour, or cerebrovascular accidents.<sup>2</sup> Nicholson<sup>3</sup> described a child suffering from diffuse lower motor neurone disease who had a cardiac arrest three minutes after receiving intravenous suxamethonium. Sinus rhythm returned after external cardiac massage and the serum potassium concentration 15 seconds after the arrest was 7.9 mmol (mEq)/l. Experimental studies<sup>4</sup> suggest that denervation appears to sensitise the muscle cell membrane to suxamethonium, leading to the release of substantial amounts of potassium. This hyperkalaemia is a transient phenomenon lasting for only three to seven minutes and many of the dysrhythmias it causes resolve spontaneously.

It may be relevant that our four patients had all had severe muscle weakness for a long time. We have not found such arrhythmias in patients with acute polyneuritis in whom bronchoscopy, tracheal intubation, or tracheostomy was performed at an early stage in the illness under anaesthesia with thiopentone and suxamethonium. This observation is in keeping with the laboratory findings of Stone,<sup>5</sup> who showed that suxamethonium-induced hyperkalaemia in dogs with transected sciatic nerves and spinal cords was a time-related effect, maximal at 28 days. Clinical reports on patients with upper motor neurone lesions also suggest that suxamethonium-induced hyperkalaemia does not occur during the first three to four weeks of paralysis.<sup>1 6 7</sup>

Apart from these four patients we have ventilated only one other patient with relapsing polyneuropathy, a 34-year-old woman in whom two separate tracheostomies were performed two years apart under general anaesthesia. No arrhythmias occurred with these procedures but on neither occasion was the patient given suxamethonium.

Our case reports provide circumstantial evidence that

suxamethonium may produce life-threatening, though fortunately easily treated, ventricular arrhythmias in patients with chronic or relapsing polyneuropathies. Although this effect may not occur when suxamethonium is given soon after the onset of muscle weakness in acute cases, it seems wise to avoid suxamethonium and consider using a non-depolarising agent in all patients with polyneuropathy who require muscle relaxation during general anaesthesia.

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# Reading for Pleasure

## Grandfather's footsteps

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Sometimes between the pages  
I still smell frangipani,  
Between others, damp bamboo.

When, as a boy, I read my grandfather's diary and letters I had never heard of André Gide but consciously felt, as he did, that one read a writer not merely to get an idea of what he said but to go off with him and travel in his company. My grandfather was a Victorian and long dead in the 1930s when, through his diary, I joined him in Singapore on 28 May 1889 to "seek what riches might be hidden in the form of gold or other precious metals among the rocky highlands of Pahang." The journey started on the coasting steamer *Sappho*, which took us up the Straits of Malacca and picked its way into the jungle-hidden mouth of the Klang river. We disembarked at the village of Klang and travelled

the 19 miles to Kuala Lumpur by train. KL looks different now. In 1889 there was no hotel, and the resthouse was dirty and dilapidated. My grandfather and I were fortunate in that the Punjom Mining Company's agent invited us to stay with him and he helped us to enlist coolies and obtain provisions before we set out on foot into the interior.

When we left Kuala Lumpur by road, walking was easy, but this soon became a jungle track with rivers to be waded and baggage to be dragged by ropes up precipitous rocks, while leeches and violent thunderstorms compounded the discomforts of a journey which lasted many days and eventually finished in a dug-out canoe. I had to hide my head under the bedclothes when I heard his dog whine and tremble when we were being tracked by a tiger.

I was fascinated by his letters to Messrs John Little and Co of Singapore. "Please send: Claret (St Julien) 1 doz pints; Whisky (Old Glenlivet) 1 case; 1 keg corned beef; ½ doz table napkins; 1 butter dish; 1 sugar bowl." This and similar orders were being sent while a letter home records that "Jolly and Jones have been away in the jungle for eleven days but Jolly says they

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