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Rapidly changing ECG in hyperkalaemia after succinylcholine

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A 58-year-old man was hospitalised in our intensive care unit for severe, acute respiratory distress syndrome caused by an influenza virus infection. After 31 days, he was gradually weaned off mechanical ventilation. However, on the day after extubation, he had to be reintubated because of further respiratory distress. Emergency intubation was performed after rapid sequence induction using the short-acting anaesthetic agent etomidate, intravenously, at a dose of 0·3 mg/kg, and the muscle relaxant succinylcholine, intravenously, at a dose of 1 mg/kg. Orotracheal intubation was done without any difficulty. However, immediately after intubation, a change was noted on the electrocardiogram (ECG) (figure) and the patient became pulseless. The ECG showed ventricular tachycardia, which developed into ventricular fibrillation. Cardiopulmonary resuscitation was initiated along with medical treatments to reduce his serum potassium concentration, which was 9·6 mmol/L; 1 h after the cardiac arrest, the serum potassium returned to within the normal range at 3·3 mmol/L. Of note, our patient had no contraindications to the use of succinylcholine before the reintubation—specifically, his serum potassium concentration was 3·5 mmol/L and he had no neurological signs or muscle weakness.

Closer examination of the ECG clearly showed the effects of hyperkalaemia on the heart during the incident: initially, the PR interval was prolonged and the T waves were tented (figure), next the P waves were lost, ST segment changes were seen, and the QRS complexes were prolonged (figure), and finally the QRS complex widened in a sine-wave configuration (figure). Succinylcholine is the first line neuromuscular blocking agent for rapid sequence induction because it is fast-acting. It causes depolarisation of the nicotinic acetylcholine receptors (AChRs) located in the neuromuscular junction (NMJ) leading to the efflux of intracellular potassium into the plasma. In cases of muscle denervation, including patients who have been in intensive care for a prolonged period, AChRs are upregulated in both the NMJ and throughout the muscle membrane; immature forms of AChRs are also released. These immature receptors are more sensitive to agonists with depolarisation occurring at lower concentrations and lasting for a longer time with no desensitisation. Succinylcholine in such situations can lead to a massive efflux of potassium from muscles to the plasma, with life-threatening consequences.

In cases of severe hyperkalaemia, treatment should include intravenous calcium salt—chloride or gluconate—to raise the threshold potential and to stabilise the myocardium, and drugs that promote cellular potassium reuptake—eg, insulin, sodium bicarbonate, and β2 agonists. This case serves as a reminder that succinylcholine should be used with caution in patients who have been in the intensive care unit for a prolonged period even without so-called intensive care unit acquired weakness.

Contributors
We all provided care for the patient. We all contributed to the search and review of the literature. A-FP was responsible for the figure and for writing the manuscript.

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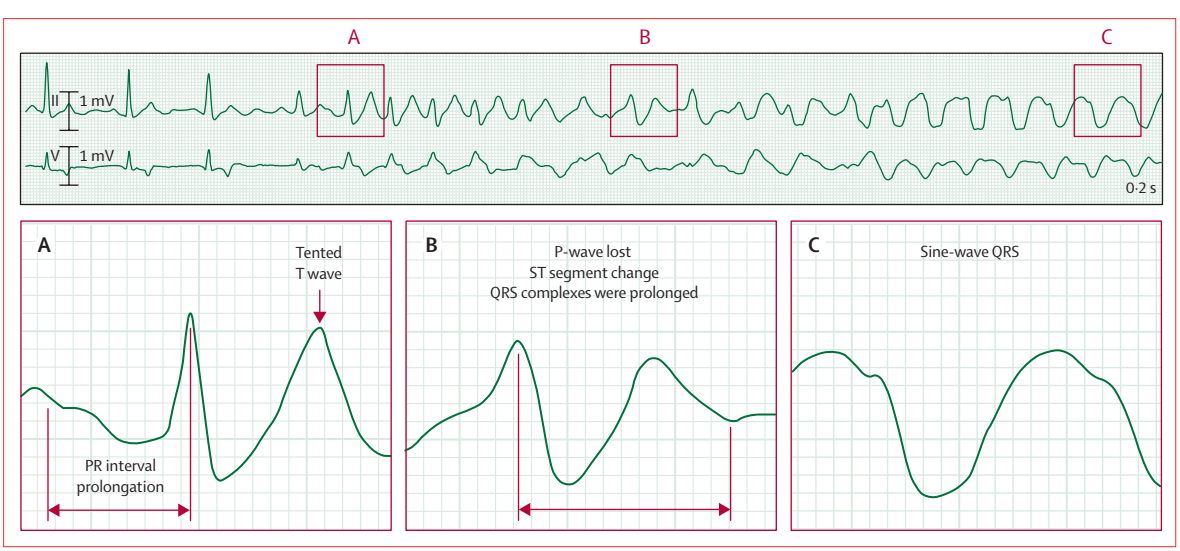

Figure: **ECG changes with fluctuations in serum potassium concentrations**
PR interval prolongation and tented T waves shown (A). P waves were lost, ST-segment changes were seen, and the QRS complexes were prolonged (B). Widening of the QRS complex occurred in a sine-wave configuration (C).