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Asystole after alfentanil-succinylcholine

To the Editor:

Alfentanil is a new narcotic capable of blunting intraoperative haemodynamic responses while allowing for return of spontaneous ventilation and consciousness after short surgical procedures. We have recently noted reports of severe bradycardia with the use of alfentanil combined with succinylcholine.[1,2] We also present such a case.

A 76-year-old 45 kg woman was scheduled for removal of an infected A-V graft in her left arm with repair of the brachial artery using an autologous saphenous vein patch. She had a long history of uncontrolled primary hypertension resulting in renal failure requiring dialysis. Haemodialysis was initiated after a failed four-month trial of peritoneal dialysis. An A-V graft had been inserted two weeks earlier but became infected. Antibiotics were started and she was maintained on peritoneal dialysis. Her volume status was labile and she demonstrated clinical signs of pulmonary oedema when her weight exceeded 50 kg. She denied a history of angina or myocardial infarction. Her blood pressure was variable ranging from 110/70–190/110 preoperatively. Despite her infected graft, there were no systemic signs of sepsis. Her volume status was optimized preoperatively. Her medications included captopril 50 mg P.O. t.i.d., nifedipine 40 mg P.O. b.i.d., propranolol 40 mg P.O. b.i.d., and digoxin 0.125 mg twice weekly. Preoperative serum electrolytes were normal including a potassium level of 3.8 $mEq \cdot L^{-1}$.

After premedication with lorazepam 2 mg orally 90 minutes preoperatively, she arrived in the operating room calm and alert. Standard monitors including a three-lead ECG pulse oximeter (Nellcor, model N100) and automatic blood pressure cuff (Critikon, Dinamap) were attached and an infusion of 0.9 per cent saline was begun. Oxygen was administered by mask followed by droperidol 1 mg IV, atracurium 4 mg IV then alfentanil 100 μg (22 $\mu g \cdot kg^{-1}$) which was injected over 40 sec. Thiopentone 75 mg was then given slowly to loss of eyelid reflex. The patient's lungs were ventilated by mask using positive pressure and a bolus of succinylcholine 100 mg IV was administered approximately 40 sec after the alfentanil had been injected. Positive pressure ventilation with 100 per cent oxygen was continued. Baseline blood pressure of 180 systolic, heart rate of 68 and oxygen saturation of 98 per cent were essentially unchanged as recorded by the automatic blood pressure cuff every 30 sec and the pulse oximeter. Suddenly, approximately 60 sec after the succinylcholine had been injected, the pulse tone of the pulse oximeter stopped, immediate inspection of the ECG revealed asystole which was confirmed by absence of the femoral and carotid pulses. Tracheal intubation was performed and the lungs were manually ventilated with 100 per cent oxygen. Immediately after intubation sinus rhythm resumed at a rate of 40 beats $\cdot min^{-1}$, increasing to 84 beats $\cdot min^{-1}$ after administration of atropine 0.6 mg IV. The blood pressure returned to 160 systolic and the pulse oximeter read 99 per cent saturation after return of sinus rhythm. Closed chest massage was never initiated. The asystole had lasted approximately 25 seconds. The remainder of the anaesthetic period and recovery room course were uneventful.

Fentanyl and its derivatives cause vagally mediated bradycardia especially after large bolus doses.[3] Succinylcholine can be associated with profound bradycardia mediated by this drug's stimulation of cardiac muscarinic receptors.[4] There have been two reports whereupon the combination of alfentanil in doses greater than 20 $mg \cdot kg^{-1}$ and succinylcholine have produced asystole rather precipitously.[1,2] With the sympathetic stimulation associated with intubation, sinus rhythm returned in our case and in the case reported by Rivard and Leowitz.[2] The combination of heightened vagal tone produced by alfentanil, muscarinic cardiac receptor stimulation produced by succinylcholine and β blockade with propranolol probably all contributed to the asystole seen in our patient. Rapid blood–brain equilibration occurs with alfentanil because 89 per cent of the drug is present as free base which rapidly crosses cell membranes.[5] Because of this rapid central effect, the vagotonic action of alfentanil may peak at the same time as the cardiac muscarinic stimulation of succinylcholine is maximal resulting in profound bradycardia.

We conclude that the combination of alfentanil boluses in doses greater than 20 $\mu g \cdot kg^{-1}$ and succinylcholoine may cause dangerous bradycardia or sudden asystole. We suggest that if this combination of drugs is to be used that alfentanil be administered slowly over a period of 1–2 min. As mentioned in previous reports the use of a neuromuscular blocker such as pancuronium with vagolytic properties as a defasciculant may be helpful in preventing asystole[1,2] but this remains to be proven clinically. nevertheless, atropine should always be immediately available and the anaesthetist should be ready to intubate quickly if asystole occurs.

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