



FIGURE

to cause such phenomena than either fentanyl or sufentanil.⁵ Combining alfentanil with drugs that produce ganglionic stimulation (succinylcholine) or induction agents that produce central sympatholysis (propofol) may lower the opioid dose required to produce sinus node dysfunction. Anaesthetists employing these induction techniques should be aware of this possible interaction.

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Cardiac arrest after tourniquet release

To the Editor:

Reading the case reported by O'Leary¹ of a patient who developed acute pulmonary oedema after tourniquet release, I want to report a similar patient who died shortly after the release of a lower limb tourniquet.

Case report

Twenty three years ago, when I still was a resident, a healthy 40-year-old patient with a slight fever was operated upon for a swelling of the foot. Until then he had received only conservative treatment for an ankle fractured one week earlier. Anaesthesia was induced with thiopentone and maintained with N₂O/O₂/halothane and spontaneous ventilation. Monitoring, at that time, consisted of clinical observation, counting the respiratory frequency, feeling the radial pulse and measuring blood pressure. Because of the bleeding in the operative field, a tourniquet around the thigh was applied. The swelling appeared to be an infectious haematoma at the place of the fracture. Nothing unusual occurred during anaesthesia. After closure of the incision and the dressing of the wound the tourniquet was released 20 min after its application. Within minutes the respiratory frequency increased and the patient became cyanotic. Manual ventilation with 100% oxygen was commenced but the condition of the patient did not improve. The pulse could not be felt and the blood pressure could not be measured. Resuscitation measures failed. Post mortem examination revealed massive pulmonary emboli in both lungs. The lesson from both case reports is that when a tourniquet is applied to an infected limb its release can lead to severe respiratory and circulatory complications.

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