attempt to localize a carcinoid tumour. These included CT scanning of the abdomen, thorax and liver, ultrasound of the pelvis, needle biopsy of the thyroid, radiological examinations of the gastrointestinal tract and bronchoscopy, all to no avail. Her urinary HIAA continued to increase and therefore it was decided to perform an exploratory laparotomy.

Preoperative physical examination revealed a sinus tachycardia of 110 · min$^{-1}$, a blood pressure of 135/80 and normal heart sounds. The chest was clear to auscultation. The patient received ranitidine 150 mg the night before surgery in conjunction with her normal dose of octreotide. An additional dose of octreotide 100 μg was administered on the morning of surgery along with a further dose of ranitidine. Treatment with an $H_1$ blocker was omitted to prevent the theoretical risk of oversedation which may occur in carcinoid patients.[2] Premedication was with temazepam 20 mg and metoclopramide 10 mg.

Anaesthesia was induced with midazolam 50 mg, fentanyl 300 μg and vecuronium 8 mg. The trachea was intubated and the lungs were ventilated with isoflurane in oxygen and nitrous oxide. Apart from a slight initial decrease in blood pressure on induction of anaesthesia, she remained haemodynamically stable throughout the three-hour procedure. There was no evidence of bronchospasm. Postoperatively she remained overnight in the intensive care unit and went on to make an uneventful recovery.

We concur with the previous authors that octreotide probably makes an important contribution to the smooth perioperative course of carcinoid patients undergoing anaesthesia and surgery and we recommend its use in this situation.

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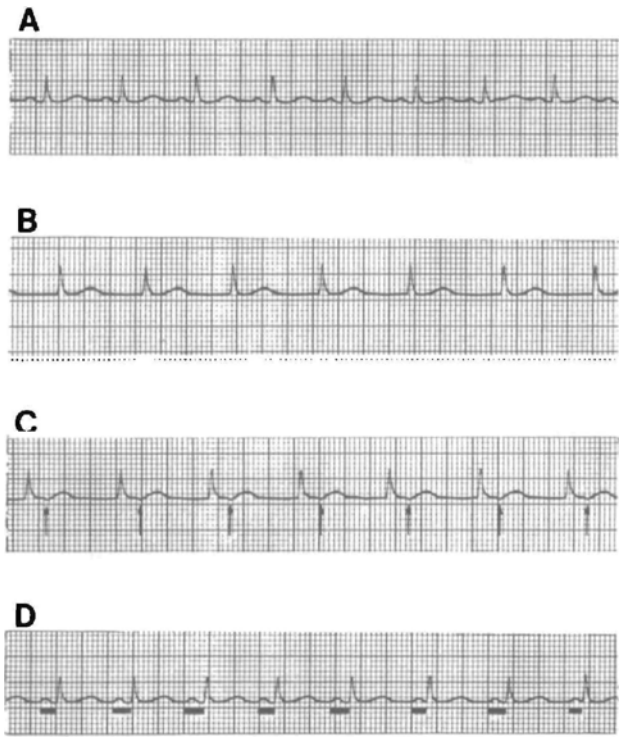
# *Sinus arrest following administration of alfentanil*

To the Editor:

There have been several reports of sinus node dysfunction observed following administration of alfentanil or sufentanil with succinylcholine during induction of anaesthesia.[1–4] These occurences have been ascribed to the combination of the sympatholytic effects of the potent opioid analgesics in combination with the vagotonic effects of succinylcholine. We wish to report three additional cases of sinus arrest following administration of alfentanil during induction of general anaesthesia.

Patients 1 and 2 were young, ASA Class 1 patients presenting for diagnostic laparoscopy as outpatients. Both received d-tubocurarine 3 mg, droperidol 1 mg, and then alfentanil 30 μg · kg$^{-1}$, thiopentone 4 mg · kg$^{-1}$, and succinylcholine 1.5 mg · kg$^{-1}$. Before their tracheas were intubated, the vocal cords were sprayed with aerosolized lidocaine. In both patients, there ensued a 10–15 sec period of asystole, terminated by the onset of a slow junctional escape rhythm. Administration of atropine 0.3 mg resulted in an accelerated junctional rhythm which then reverted to a normal sinus rhythm. Patient 3 was a 24-yr-old female presenting for uvulopalatopharyngoplasty. The patient was treated for a manic-depressive disorder with lithium carbonate, fluoxetine, and chlorpromazine. Following administration of d-tubocurarine 3 mg and droperidol 1 mg, anaesthesia was induced with alfentanil 20 μg · kg$^{-1}$, thiopentone 5 mg · kg$^{-1}$, and succinylcholine 2 mg · kg$^{-1}$. After tracheal intubation, the patient was noted to be in sinus rhythm with a heart rate of 70 bpm (Trace A) (see Figure). With introduction of the operating laryngoscope into the airway, there ensued a period of sinus arrest lasting approximately 20 sec with an accelerated junctional rhythm evident on the ECG (Trace B). Blood pressure was recorded at 75/40 mmHg. Subsequently, there was evidence of retrograde conduction into the atrium (Trace C, arrows at retrograde P waves) followed by a period of isorhythmic A-V dissociation (Trace D, variable PR interval). There was a spontaneous return to normal sinus rhythm.

Bradycardia is commonly seen in association with administration of rapid-acting opioid analgesics perioperatively.[1,2] This is presumed to result from the sympatholysis that they produce, creating a relative increase in resting vagal tone. Stimulation of the upper airway, however trivial, may further increase vagal tone, lead to sinus node suppression, and result in haemodynamically compromising bradydysrhythmias.[3,4] Alfentanil, because of its rapid onset, may be more likely




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'Leary[1] 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 $N_2O/O_2$/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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