Correspondence

References


William Bayard 1814–1907

To the Editor:
Dr. William Bayard1 was certainly a leader in the medical community in New Brunswick. He was also one of the first to make use of ether anaesthesia, but the claim that he used it in 1844, two years before Morton demonstrated its use in Boston, is not substantiated by contemporary documentation. When McAvenney in 19052 wrote about Bayard’s use of ether, he did so sixty years after the event without citing his source: “The first time ether was administered in St. John for extracting teeth was in the office of the Vanbuskirks by Dr. William Bayard. . . . This was in 1844, shortly after Dr. Horace Wells, the American dentist, discovered surgical anesthesia.” He did not state that this was two years before ether was used in Boston, and Morton’s name was not mentioned.

The only occasion on which Bayard and Van Buskirk cooperated in the use of ether, which I have found,3 refers to a hospital operation which took place in March 1847, not in 1844: “Experience in establishing the beneficial effects of Ethereal Vapour during Surgical operations, and the use of it is receiving the highest professional sanction in Europe and the United States. In our own City, the experiments which have been already made, are confirmatory of the advantages of it . . . this fact was fully illustrated during an operation recently performed by Dr. Wm. Bayard, in the Hospital of this City and County . . . the patient inhaled the Vapour of Ether through a machine made by Mr. Van Buskirk, the dentist, who was present . . . as the public are interested in the question of good or evil connected with the use of Ethereal Vapour in Surgical operations, no apology is requisite for making a Newspaper the medium of reports of effects for the benefit of all, who from disease or accident, may have occasion and inclination to resort to it.”

My own research4 confirms MacDougall’s earlier opinion5 that the first use of ether in New Brunswick occurred in January 1847.6–8 No attempt has previously been made to establish priority for Van Buskirk or Bayard or Morton. Is it possible that McAvenney, writing sixty years after the event and without realizing the significance of the date, simply made an error when he stated that Bayard used ether in 1844?

Octreotide for Carcinoid Syndrome

To the Editor:
Drs. Watson, Badner and Ali reported the use of octreotide, a long-acting somatostatin analogue, in the management of a patient with an ovarian carcinoid tumour who presented for laparotomy and tumour resection.1 We have recently had experience with octreotide in a patient with carcinoid syndrome undergoing anaesthesia and surgery and we can confirm its efficacy for maintaining perioperative stability.

A 43-year-old female presented to the physicians with a one-year history of palpitations, hot flushes, tiredness and episodes of wheezing. Extensive investigations had failed to reveal the cause of her symptoms. However, she had a marginally raised urinary HIAA and was treated empirically with octreotide 50 µg subcutaneously o.d. Her symptoms improved dramatically on this treatment and the dose of octreotide was subsequently increased to 75 µg o.d. Further investigations were ordered in an
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 $110 \cdot \text{min}^{-1}$, 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 H2 blocker was omitted to prevent the theoretical risk of oversedation which may occur in carcinoid patients. 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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REFERENCES

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. These occurrences 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. 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. Alfentanil, because of its rapid onset, may be more likely