CORRESPONDENCE

Cartoid Sinus Massage for Treatment of A-V Junctional Rhythm

To the Editor:—The report by Hill\(^1\) is interesting and supports previous reports demonstrating the effectiveness of propranolol in treating A-V junctional rhythm.\(^2\) This would appear to confirm the suggestion that A-V junctional rhythms during anesthesia may be triggered by increased activity in the A-V node mediated by increased sympathetic activity. Certainly pharmacological stimulation of the A-V node with β-adrenergic agonists is known to cause excitation of the node\(^3\) and the mechanism may be similar. In support of this hypothesis, I have observed the restoration of sinus rhythm in anesthetized children with accelerated junctional rhythm by the application of carotid sinus massage (CSM). This is illustrated by the following representative case history.

A 5-yr-old boy weighing 21 kg was anesthetized for repair of an injury to his right forearm. Following induction of anesthesia with 125 mg of thiopental and 0.05 mg of fentanyl and paralysis with 10 mg of atracurium, the trachea was intubated and the lungs ventilated to normocapnia with nitrous oxide, oxygen, and 0.5% halothane. After 50 min of uneventful anesthesia, the patient developed a junctional rhythm at a rate of 90 beats/min. Although there had been no significant change in heart rate, this was associated with a decrease in systolic blood pressure from 110 to 85 mmHg, presumably secondary to loss of the atrial contribution to ventricular filling. Sinus rhythm and blood pressure were restored by the application of 3 s of left-sided CSM similar to that recently described.\(^4\) During the remainder of the procedure, the patient reverted to junctional rhythm on three occasions, and on each occasion was immediately restored to sinus rhythm by further CSM.

One explanation for the success of CSM in accelerated junctional rhythm in this situation would be a suppression of increased excitability in the A-V node and subsequent "capture" of the ventricles by the sinus node. Pharmacological treatments of intraoperative junctional rhythm may all produce undesirable effects. It should be emphasized that no harmful effects from CSM have been observed in patients in this unit. CSM would not be recommended in slow junctional bradycardias where the etiology is more likely to be decreased excitability or conduction above the A-V node. CSM is a suitable technique in pediatric patients who in general lack ischemic impairment of the cardiac conducting system. Obviously it would not be a suitable technique in children with congenital conduction defects where further decreases in conduction could be hazardous. However, in general, properly conducted CSM is extremely safe.\(^5\) In common with previous experience with propranolol, it should be noted that CSM may have to be repeated to maintain a therapeutic effect.

In conclusion, CSM appears to be an effective method of treating accelerated A-V junctional rhythm in anesthetized children with a method of action analogous to that of propranolol. I would suggest that this use should be considered in this situation before resorting to pharmacological treatments.

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REFERENCES


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The FEF End-Tidal Carbon Dioxide Detector

To the Editor:—There are recent accounts\(^1,2,4,5\) indicating that unrecognized esophageal intubation is still a major cause of serious patient injury and litigation against anesthesiologists. The most reliable methods for ensuring correct placement of an endotracheal tube are either direct observation of passage of the tube into the trachea or the measurement of carbon dioxide (CO\(_2\)) in the expired gas mixture.\(^5\) As a result, electronically powered analyzers using infrared detection or mass spectroscopy are available in many operating rooms in North America. However, these devices are expensive, require maintenance and calibration, and are not always available, particularly in areas where tracheal intubation is carried out under difficult circumstances, e.g., intensive care units, emergency departments, ambulances, and resuscitation in the field.

In 1988 a pH-sensitive chemical indicator was developed that could detect CO\(_2\).\(^4\) The indicator was enclosed in a replaceable disc housing and connected to the gas stream between the endotracheal tube and the anesthetic circuit. We now describe the first commercial application of this technology—the FEF end-tidal CO\(_2\) detector manufactured by Fenem Airway Management Systems.

Figures 1 and 2 show the CO\(_2\) detector and a standard angle piece for comparison; inlet and outlet ports are standard 15 mm. The device is made of white plastic material with a dead space of 38 ml and contains

Perioperative Management of a Patient with Congenital Hypofibrinogenemia

To the Editor—Congenital severe hypofibrinogenemia and hypofibrinogenemia are rare disorders complicated by hemorrhage and/or thrombosis, which pose a significant therapeutic dilemma. Anesthesiologists generally only use fibrinogen infusions to increase the fibrinogen blood level, but thrombotic events have been described after those infusions in such patients.

A 57-yr-old, 75-kg weight male with congenital severe hypofibrinogenemia was scheduled for a right sympathectomy because of recurrent painful episodes of lower limbs ischemia. He had been suffering for years from both severe subcutaneous hematomas and peripheral arteritis. His sister also presented with hemorrhagic and thrombotic disease and died from postoperative pulmonary embolism. He had been given fibrinogen infusions for active bleeding nearly every 4 months during the last 20 yr; antifibrinogen antibodies were evaluated every 2 infusions, first by measuring postinfusion rise and half-life in plasma fibrinogen level, and second by a mixing test. No antifibrinogen antibodies could be detected. The serologic tests for HBV and HIV were negative. He had very low plasma fibrinogen levels as demonstrated...