Ventricular Tachycardia Terminated by Exubtation of the Trachea

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Although cardiac control is often explained in terms of intrinsic myogenic mechanisms, there is good experimental and clinical evidence that the sympathetic and parasympathetic nervous systems exert profound and immediate control over cardiac rate, rhythm, fibrillation threshold and contractility.† Arrhythmias are commonly seen in patients with endotracheal tubes in place. Ventricular tachycardia terminated by removal of an endotracheal tube has not yet been reported. We report such a case and discuss possible mechanisms in terms of current theories of airway reflexes and neurogenic cardiac control.

REPORT OF A CASE

A 45-year-old white man was admitted to the hospital with a history of incapacitating angina. His medications included nitro-
glycerin, propranolol, 80 mg. t.i.d., and isosorbide dinitrate, 5 mg. t.i.d. There was no previous history of congestive heart failure, hypertension, diabetes, or previous myocardial infarction. Laboratory data were within normal limits. The ECG showed symmetrical T-wave inversions in leads I, AVL, and V4 through V6. The T-waves were flattened in leads II, III, and AVF. Cardiac catheterization showed 90 per cent occlusion of the proximal left anterior descending coronary artery, total occlusion of the right coronary artery, and a poorly functioning left ventricle with an ejection fraction of 50 per cent. The patient was scheduled for an aorticcoronary bypass grafting procedure.

The ECG, a radial arterial pressure transducer, central venous pressure, and urinary output were monitored. Induction of anesthesia was accomplished with diazepam, pancuronium, and fentanyl. Intubation of the trachea was accomplished with a size 8 endotracheal tube after the larynx had been sprayed with 4 ml 4 per cent lidocaine. Anesthesia was maintained with N2O–O2, fentanyl, and halothane. The anesthetic and surgical courses were uneventful.

A three-vessel aorticcoronary bypass grafting procedure was performed in six and a half hours. The patient was taken to the intensive care unit in satisfactory condition and ventilated with a Servo 900B ventilator. One hour postoperatively, he was awake, with normal arterial blood gases (table 1), stable blood pressure, minimal bleeding, and a good urinary output. Mechanical ventilation was discontinued and the patient maintained stable spontaneous ventilation with 70 per cent O2 via T-piece with acceptable blood gases. He confirmed by nodding that the endotracheal tube was causing discomfort. He was given diazepam, and 10 mg in increments and morphine, 4 mg. iv. However, he became restless, coughed, and indicated by gestures that he wished to have the endotracheal tube removed.

The ECG monitor then showed abrupt ventricular tachycardia (fig. 1). Immediate elective extubation of the trachea by the anesthesiologist at the bedside resulted in a prompt conversion to a normal sinus rhythm. Although no antiarrhythmic agent was given, normal sinus rhythm was maintained. Arterial blood gases were stable (table 1), and the rest of the recovery period was uneventful. The patient was discharged from the hospital ten days later.

**DISCUSSION**

Manipulation of the airways can lead to cardiac arrhythmias. Mechanical stimulation of the nasal airway has been shown to cause reflex bradycardia, inhibition of ventilation, and laryngospasm in dogs. Electrical stimulation of the superior or inferior laryngeal nerve has been reported by many investigators to cause reflex bradycardia or even asystole.

In man, the empirical relationship between endotracheal intubation or extubation, cardiac arrhythmias, and sudden death is well known but poorly understood. Katz and Bigger have reviewed the subject of cardiac arrhythmias during anesthesia; they suggest that many patients will have at least a transient arrhythmia during airway manipulation.

Central nervous system participation in cardiac rhythm control was indicated by Verrier et al., who demonstrated that stimulation of the posterior hypothalamus in dogs caused a 40 per cent reduction in ventricular fibrillation threshold. In a clinical study, Lown et al. documented that mental events such as anxiety and guilt can reproducibly cause ventricular tachycardia. Psychologic stimuli caused and psychiatric treatment controlled cardiac arrhythmias in their patients.

Animal investigations, like those of Kolman et al., have confirmed that sympathetic nerve stimulation increases the vulnerability of the canine ventricle to fibrillation. Abildskov reported that electrical stimulation of cardiac sympathetic nerves caused QT-interval changes and associated increases in ventricular vulnerability to arrhythmia. They also documented that increased vagal activity could restore the fibrillation threshold to normal levels. Kolman's work on

**Table 1. Arterial Blood Gases in the Postoperative Period**

<table>
<thead>
<tr>
<th>Airway</th>
<th>Ventilation</th>
<th>Postoperative Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intubated</td>
<td>Assisted</td>
<td>1 Hour</td>
</tr>
<tr>
<td>Intubated</td>
<td>Spontaneous</td>
<td>2.5 Hours</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5 Hours</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 min after extubation</td>
</tr>
<tr>
<td>Fl2O</td>
<td>0.4 torr</td>
<td>0.7 torr</td>
</tr>
<tr>
<td>pO2</td>
<td>7.41</td>
<td>7.34</td>
</tr>
<tr>
<td>PacO2</td>
<td>39 torr</td>
<td>40 torr</td>
</tr>
<tr>
<td>PacO2</td>
<td>139 torr</td>
<td>124 torr</td>
</tr>
<tr>
<td>HCO3-</td>
<td>23 mmol/l</td>
<td>24 mmol/l</td>
</tr>
<tr>
<td>K+</td>
<td>3.9 mmol/l</td>
<td>4.0 mmol/l</td>
</tr>
<tr>
<td></td>
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<td>4.0 mmol/l</td>
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firmed that the balance of autonomic control is more important than the absolute level of sympathetic or parasympathetic activity.

Our patient may have had either psychologically or mechanically induced autonomic nervous system activity that predisposed the ventricle to tachycardia. Pain and anxiety could have caused increased sympathetic activity, which transiently increased vulnerability to cardiac arrhythmia. Extubation of the trachea may have been provided the stimulation necessary for an immediate shift in autonomic balance in control of the heart and terminated the arrhythmia. Removal of the endotracheal tube could have stimulated tracheal, laryngeal, and pharyngeal receptors, producing a burst of parasympathetic afferent activity. Reflex efferent parasympathetic discharge then might have counteracted the relative sympathetic overactivity and restored normal cardiac rhythm. Besides the burst of parasympathetic activity caused by the removal of the endotracheal tube, the psychologic relief obtained may have reduced the level of sympathetic activity. The ventricular tachycardia was terminated immediately with extubation of the trachea, and normal cardiac rhythm was maintained without the aid of the usual exogenous pharmacologic agents.

REFERENCES


End-tidal Enflurane Concentration for Endotracheal Intubation

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Inhalational induction followed by endotracheal intubation is a technique frequently employed in pediatric anesthesia. In an earlier study we introduced the term MAC_{ЕI}, defined as the end-tidal concentration of a gas or vapor needed by 50 per cent of the population to prevent all movement both during and immediately after laryngoscopy and endotracheal intubation. MAC_{ЕI} for halothane was calculated to be 1.39 per cent at sea level. In the present report, similar methods were employed to determine MAC_{ЕI} for enflurane in pediatric patients.

METHODS

Twenty-four studies were performed in 22 ASA I surgical patients, aged 2 to 6 years. Informed consent regarding the nature and risks of the study was obtained from the parent or guardian of each participant. Premedication consisted of atropine, 0.015 mg/kg. A precordial stethoscope was used to monitor heart and breath sounds. Blood pressure was measured indirectly, and lead II of the electrocardiogram was continuously displayed. Body temperature was monitored with a rectal thermistor. Induction of anesthesia was accomplished with enflurane, 4–5 per cent, and oxygen (5 l/min) delivered from an Enfluromatifer vaporizer through a Jackson-Rees modification of an Ayres