are not effective after the reaction has begun, nor will they alter the responses due to other chemical mediators. General anesthesia with ether or cyclopropane does not prevent anaphylaxis, although the reaction may be modified by these agents. The prophylactic benefit of corticosteroids has not been well established. Schatz et al. showed that 5 of 42 (11.8 per cent) patients who had histories of anaphylactoid reactions to contrast media had repeat reactions when re-exposed to the media despite antihistaminic pretreatment. Zweiman et al. pretreated patients with histories of anaphylactoid reactions to contrast media with high-dose orally administered steroids for 18 hours before re-exposure. Repeat reactions were seen in one of nine (11 per cent) patients. Miller et al. reported a case in which high-dose oral steroid pretreatment for 72 hours was used successfully to prevent anaphylaxis on re-exposure to the contrast medium in a patient with a well-documented previous anaphylactic reaction.

Our patient had had a major anaphylactic reaction eight hours prior to her operation, and had been appropriately and successfully resuscitated. Although methylprednisolone, 100 mg, iv, used to treat the initial rash during catheterization, failed to prevent the subsequent anaphylaxis, a much larger dose of corticosteroid given intraoperatively with an antihistaminic agent prevented all clinical manifestations of a reaction to three injections of contrast material. The results in this case suggest that pretreatment with a large dose of corticosteroid and antihistamines may be beneficial in preventing anaphylaxis in patients known to have idiosyncratic reactions to contrast media. When emergency procedures requiring contrast studies are necessary for these susceptible patients, pretreatment in this fashion should be considered.

References


Anesthesiology
50:57–59, 1979

Ventricular Tachycardia Terminated by Exubation of the Trachea

SUSAN K. PALMER, M.D.,* AND KIITA D. PATEL, M.D.†

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-

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Theophylline 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

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

<table>
<thead>
<tr>
<th></th>
<th>1 Hour</th>
<th>2.5 Hours</th>
<th>5 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intubated</td>
<td></td>
<td>Intubated</td>
<td>15 min after extubation</td>
</tr>
<tr>
<td>Ventilation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assisted</td>
<td></td>
<td>Spontaneous</td>
<td>Spontaneous</td>
</tr>
<tr>
<td>Fco₂</td>
<td>0.4 Torr</td>
<td>0.7 Torr</td>
<td>0.7 Torr</td>
</tr>
<tr>
<td>pH</td>
<td>7.41</td>
<td>7.34</td>
<td>7.34</td>
</tr>
<tr>
<td>Paco₂</td>
<td>39 Torr</td>
<td>46 Torr</td>
<td>45 Torr</td>
</tr>
<tr>
<td>Pao₂</td>
<td>139 Torr</td>
<td>124 Torr</td>
<td>129 Torr</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>23 mmol/l</td>
<td>24 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>
<td>4.0 mmol/l</td>
</tr>
</tbody>
</table>
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 psychological 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

RONALD W. YAKAITIS, M.D.,* CASEY D. BLITT, M.D.,† JAMES P. ANGIULO, M.D.*

Inhalational induction followed by endotracheal intubation is a technique frequently employed in pediatric anesthesia. In an earlier study1 we introduced the term MAC₆₅, 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. MAC₆₅ for halothane was calculated to be 1.39 per cent at sea level. In the present report, similar methods were employed to determine MAC₆₅ 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 Enfluromatric vaporizer through a Jackson-Rees modification of an Ayres

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