CLINICAL REPORTS

is not far from 2 min following administration of lidocaine as in this study.

In conclusion, the results of this study demonstrate that intravenous injection of lidocaine (1 mg/kg) administered 2 min before endotracheal extubation prevents coughing and increases in blood pressure and pulse rate during and after extubation. Our findings suggest that intravenous administration of lidocaine prior to endotracheal extubation should be advantageous to the patient who has coronary-artery disease and cannot tolerate increases in cardiovascular dynamics.

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Exacerbation of Iatrogenic Hypercarbia by PEEP

RICHARD P. FOGDALL, M.D.*

The efficacy of positive end-expiratory pressure (PEEP) in the treatment of adult acute respiratory failure, and improvement of functional residual capacity (FRC), is well recognized.1,2 A PEEP device commercially available for intraoperative use† has been reported to be effective and safe.3

The following case report demonstrates a detrimental effect of this device upon carbon dioxide elimination in a cardiac patient, when combined with a malfunctioning anesthesia circle system. The summary of this case report will focus on the respiratory system.

REPORT OF A CASE

Fourteen days after mitral valve replacement and two-vessel coronary-artery-bypass grafting, a 64-year-old woman weighing 62 kg was scheduled for removal of an intra-aortic counterpulsation balloon. Past surgical and medical history included numerous myocardial infarctions, triple coronary-artery-bypass grafting two years previously, severe mitral-valve regurgitation secondary to papillary muscle infarction, and chronic congestive heart failure. Preoperatory respiratory support was provided, via a tracheostomy, with an MA-1® ventilator, utilizing an intermittent mandatory ventilation (IMV) mode and PEEP. Arterial blood-gas analysis showed satisfactory ventilation (table I). The patient was awake and oriented.

During transport to the operating room, ventilation was assisted by use of a Jackson-Rees modification of the Ayre's T-piece, and a 10-l/min flow of oxygen. In the operating room, ventilation was provided either manually or with an Air Shields® ventilator, utilizing the previously checked Ohio® anesthesia machine. Anesthetic medications included nitrous oxide, enfluran, diazepam, and pancuronium. Axillary temperature was 37°C throughout the procedure. Intraoperatively, 5 cm H2O PEEP was placed in the exhalation limb of the anesthesia circuit. After the application of PEEP, the patient appeared progressively agitated, began to resist mechanical ventilation, and the inspiratory peak pressures on the aspirator canister pressure gauge increased to 45 cm H2O. The oxygen analyzer in the inspired gas circuit confirmed the FiO2 expected from the flowmeter settings. Adequate oxygenation was confirmed by serial arterial blood-gas analyses, but progressive hypercarbia became evident (table I). Breath sounds, easily auscultated over the chest, were equal bilaterally. Ventilation coordinated well with visual impressions of inflation of the chest. No abnormality was discovered with the tracheostomy tube or cuff. Carbon dioxide was not being administered, and the CO2-absorption system and soda-lime appeared normal. The operation was completed, and the patient was transported to the intensive care unit, where ventilation was provided as preoperatively. Arterial blood-gas analyses twice during the first hour in the intensive care unit showed normocarbia, and were consistent with corresponding preoperative values (table I). There were no sequelae, and the patient made an uneventful recovery.

Postoperative evaluation of the anesthesia circuit demonstrated (beneath the fogged valve cover) that

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the inhalation valve poppet was jammed in the open position, and thus incompetent (fig. 1). We demonstrated effective to-and-fro ventilation through the circle system, without resistance of any kind. In addition, we demonstrated the lack of effectiveness of PEEP on the closed distal end of the circle system's Y-piece, even though the 5-cm H2O PEEP device was in place on the exhalation limb of the circle. The PEEP value was found to be fully effective in registering PEEP at the Y-piece, when the defective inhalation valve poppet was replaced with a normal component. We could find no abnormality of the mechanical ventilator, or the remainder of the anesthesia circuit, including the CO2-absorption system.

**DISCUSSION**

This case demonstrates a failure of CO2 elimination via the anesthesia circle system due to a malfunctioning inhalation circuit valve. In addition, it demonstrates exacerbation of hypercarbia by the use of a PEEP device in this circuit. Fortunately, there was no long-term effect on the patient.

An incompetent valve on the inhalation limb of an anesthesia circle system will allow exhaled gas to enter the inhalation limb during patient exhalation, and thus this gas will mix with fresh gas, producing partial rebreathing during the subsequent breath (fig. 2). Kerr and Evers have shown that one incompetent circle system valve can increase inspired CO2 concentrations from a normal value of 0.1–0.3 per cent to approximately 1.5 per cent. This would be expected to increase Paco2 by approximately 10 torr, as was seen early in our case. The addition of PEEP to the exhalation limb of this malfunctioning circle system will impair exhalation, and thus the amount of exhaled gas regurgitated into the inhalation limb will increase, compounding the rebreathing problem, and leading to increased hypercarbia (fig. 2). A patient struggling and exhaling forcefully should make the problem even worse, as the easiest escape of exhaled gas is via the inhalation limb.

Was the progressive increase in Pco2 in our case caused only by the expected normal effects of PEEP? Triclet et al. have shown that the application of 10 cm H2O PEEP to patients after mitral valve replacement increases Paco2 approximately 6 torr, and increases Vd/VT (deadspace-to-tidal volume ratio) approximately 9 per cent. Thus, only modest increases in Paco2 from PEEP alone have been expected. Clearly, the 27-torr increase in Paco2 in our case, after the application of PEEP, would occur only with concomitant circle-system valve incompetency (fig. 2). In addition, because of the valve incompetency in our case, an increase in the patient's Vd would not occur during PEEP.

Hypercarbia produced some cardiovascular effects in this patient. However, it is doubtful that PEEP itself

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**Table 1. Ventilatory and Arterial Blood-gas Variables in the Intensive Care Unit and during Anesthesia**

<table>
<thead>
<tr>
<th>Location and Timing</th>
<th>Ventilator</th>
<th>Peak Pressure (cm H2O)</th>
<th>Tidal Volume (ml)</th>
<th>Frequency (/Min)</th>
<th>PEEP (cm H2O)</th>
<th>Arterial Blood-gas Values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Pco2</td>
</tr>
<tr>
<td>Intensive care unit,</td>
<td>MA-1</td>
<td>30</td>
<td>700</td>
<td>8 (IMV)</td>
<td>7.5</td>
<td>0.4</td>
</tr>
<tr>
<td>preoperatively</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Operating room</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After 60 min</td>
<td>Air Shields</td>
<td>38</td>
<td>900</td>
<td>10</td>
<td>0</td>
<td>0.5</td>
</tr>
<tr>
<td>After 90 min</td>
<td>Air Shields</td>
<td>45</td>
<td>900</td>
<td>10</td>
<td>5</td>
<td>1.0</td>
</tr>
<tr>
<td>After 120 min</td>
<td>Manual</td>
<td>45</td>
<td>—</td>
<td>12</td>
<td>5</td>
<td>1.0</td>
</tr>
<tr>
<td>After 150 min</td>
<td>Air Shields</td>
<td>36</td>
<td>1,000</td>
<td>10</td>
<td>5</td>
<td>1.0</td>
</tr>
<tr>
<td>Intensive care unit</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After 12 min</td>
<td>MA-1</td>
<td>34</td>
<td>1,000</td>
<td>10</td>
<td>7.5</td>
<td>0.5</td>
</tr>
<tr>
<td>After 60 min</td>
<td>MA-1</td>
<td>30</td>
<td>900</td>
<td>9</td>
<td>4</td>
<td>0.5</td>
</tr>
</tbody>
</table>
had any direct circulatory effect, since it failed to increase the airway pressure at the circle Y-piece. The direct cardiovascular effects of PEEP during controlled ventilation have been discussed by others.3-7

Preoperative testing of the totally closed anesthetic circle system, by applying positive pressure to the ventilation bag while looking for leaks, does not test valve competency. Nor does breathing to and fro through the circle system, unless the valves are visually checked for movement and proper closure. The valves cannot always be trusted. Function, understanding, and testing of circle absorption systems and valves are discussed by Dorsch and Dorsch.8

Continuous end-tidal CO2 monitoring would have been informative in the early detection of CO2 accumulation in our patient, and the detrimental effect of PEEP on CO2 elimination. In addition, routine monitoring of the airway pressure at the level of the endotracheal tube, instead of within the absorption system, is important in assessing the presence of elevated airway pressures after the application of a PEEP device to a circle system with competent valves. As an added suggestion, when there are doubts about malfunctions of equipment, vis-à-vis "malfunctions" of the patient, switching to a simple "bag-and-oxygen" ventilation system should offer advantages both in diagnosis and in therapy.

This case demonstrates a failure of CO2 elimination via the anesthetic circle system, secondary to an incompetent inhalation valve. In addition, it demonstrates an exacerbation of the resulting hypercarbia by a combination of a PEEP device with the malfunctioning valve.

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