Failure of Low Pressure Alarm Associated with the Use of a Humidifier

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One of the monitors specified as minimal standard for patient monitoring is the use of a low pressure alarm placed in the breathing circuit during mechanical ventilation.* This alarm serves as a warning in the event of a disconnection. A case is presented in which the use of a humidifier functioned to prevent the low pressure alarm from detecting a disconnection. Furthermore, a study delineating the clinical conditions associated with the potential for malfunction is presented.

CASE REPORT

A 26-year-old male suffered chest contusions, hemopneumothorax, and abdominal injuries from an auto accident for which he was undergoing a laparotomy. After insertion of an arterial cannula, central venous cannula, four peripheral iv, and placement of EKG electrodes, anesthesia was induced with ketamine and succinylcholine iv in a rapid sequence fashion and the trachea was intubated. Skeletal muscle paralysis was induced with pancuronium iv; anesthesia was maintained with iv ketamine, and fentanyl. Mechanical ventilation of the lungs was accomplished with a tidal volume of 900 ml and a frequency of 10 breaths/min using an Ohio ventilator. A low pressure alarm had been placed immediately distal to the inspiratory valve in a conventional circle system. A cascade humidifier had been placed in the inspiratory limb to warm and humidify inspired gases. At one point both anesthesia personnel had been engaged in rapid infusion of blood. It was then noted that a disconnection had taken place in the circle system between the "Y" and the endotracheal tube; yet the low pressure alarm had not sounded. Immediately before reconnection it was noted that a tidal volume delivered by the ventilator produced a system pressure of 20 cmH2O with no patient attached. No harm befell the patient as a result of this incident.

This case report illustrates a situation in which the use of a humidifier prevented the detection of a disconnection by the low pressure alarm. The following study details the circumstances under which a similar result might be obtained.

MATERIALS AND METHODS

Three Ohio anesthesia ventilators were tested. During the examination of each ventilator three inspiratory flow rates and four tidal volumes were used. The inspiratory flow control dial does not have specific graduated measurements; therefore, low, medium, and high flows were selected by setting the dial to the extreme left, middle, and extreme right, respectively. A (FLEISCH) pneumotachograph was used to determine the peak rates at each of these settings. The values were 27.8, 79.2, and 95.6 l/min.

The tidal volumes were 1,200, 1,000, 750, and 500 ml. The expiratory time and expiratory flow rates were adjusted to maintain a frequency of 8–10 cycles/s. Pressure in the breathing circuit with no patient attached was measured using an aneroid pressure gauge located on the inspiratory limb. The measurements were confirmed with a separate system consisting of a pressure transducer, fluid-filled tubing, and a monitor.

The cascade humidifier and the Portex Humid-Vent 1 were separately tested. The cascade humidifier was placed on the inspiratory limb. The Humid-Vent 1 was placed distal to the "Y" in the circuit. The Humid-Vent 1 was tested under dry conditions as well as water-saturated conditions. In both cases nothing was attached to the circuit at the patient position. The experiments were conducted with the low pressure sensor on the inspiratory limb and then repeated with the sensor on the expiratory side.

RESULTS

The Ohio Medical Products operations manual indicates that the low pressure alarm will sound if a pressure of 10 cmH2O has not been sensed for a period of 15 consecutive s. We found that in our machines the low pressure alarm sounded if a minimum pressure of 15 cm H2O was not detected at a rate of at least 4 breaths/min.

Table 1 shows the pressure developed during various combinations of inspiratory flow rates and tidal volumes.

<table>
<thead>
<tr>
<th>Tidal Volume (ml)</th>
<th>Inspiratory Flow (cm H2O)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Low</td>
</tr>
<tr>
<td>1,200</td>
<td>5.5</td>
</tr>
<tr>
<td>1,000</td>
<td>5.5</td>
</tr>
<tr>
<td>750</td>
<td>5.5</td>
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<tr>
<td>500</td>
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</tbody>
</table>

* Low pressure alarm did not sound.

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when the cascade humidifier was tested. When the sensor was on the inspiratory side between the inspiratory valve and the humidifier (fig. 1), low pressure alarm system did not warn of the disconnection during medium and high flow rates at any tidal volume between 500 and 1,200 ml (table 1). This occurred whether the disconnection was at position 1, 2, or 3. When the sensor was on the expiratory limb (between the patient connection and the expiratory valve), it consistently detected the disconnection because back pressure was not generated on that side of the circuit, i.e., distal to the cascade humidifier.

Table 2 depicts the results using the water-saturated Humid-Vent 1. Back pressure was generated only when the disconnection occurred distal to the Humid-Vent 1, i.e., the junction between the patient and Humid-Vent 1 (fig. 2). However, the pressure was transmitted to both sides of the airway circuit; therefore, the disconnection was not sensed when sufficient pressure was generated regardless of where the low pressure sensor was placed in the circuit. The results obtained using the saturated versus the dry Humid-Vent 1 were similar.

**DISCUSSION**

Cooper et al.,1 in a review of more than 1,000 preventable “critical incidents” during anesthesia, underscored the importance of a low pressure alarm. They reported that breathing circuit disconnection during mechanical ventilation was the most common overall incident. Of the incidents with substantive negative outcomes, at least 14 probably would have been detected more promptly if “some combination of monitoring based on oxygen sensing and surveillance of ventilation”1 had been in use. The authors suggested using an airway pressure monitor capable of sounding an audible alarm in the event of a disconnect. Likewise, the ASA guidelines also stress the importance of using alarm systems in assuring the adequacy of ventilation during general anesthesia.

The use of humidifiers in the operating room is commonplace and of proven benefit in protecting patients from the effects of inspired, cool, dry gases.2 Furthermore, they are also useful tools when trying to maintain the body temperature of patients during long cases.3 However, this equipment has airway flow impedance characteristics that can interfere with the low pressure alarm's ability to warn of an airway disconnection.

The current case illustrates a situation during which the use of a cascade humidifier prevented the detection of an airway circuit disconnection. Our study showed that the back pressure generated by both the cascade humidifier and the saturated Portex Humid-Vent 1 with medium to high inspiratory flow rates was sufficient to keep the low pressure alarm from sensing a disconnection. This similar outcome was obtained despite the differences in impedance characteristics between the two pieces of equipment. The cascade humidifier has both capacitance and resistance impedance characteristics, whereas the Portex Humid-Vent 1 is primarily a resistance impedance. The above situation could be alleviated when using the cascade humidifier by simply placing the sensor on the expiratory limb. However, if the disconnection occurred between the patient and the Portex Humid-Vent 1, then placement of the sensor on either the inspiratory or expiratory limb did not correct the problem.

This case demonstrates that under certain circumstances adhering to the ASA Standards for Intraoperative
monitoring does not always ensure patient safety. Specifically, a disconnection in the airway circuit may not be detected by the low pressure alarm when using medium to high inspiratory flow rates in conjunction with a cascade humidifier or Portex Humid-Vent 1. The use of redundant monitors of ventilation such as an esophageal stethoscope might be advised to provide early warning of patient disconnection. Although disconnect alarms are important, they are not a substitute for vigilance on the part of the anesthesiologist.

Cardiovascular Effects of Pimecuronium and Pancuronium in Patients Undergoing Coronary Artery Bypass Grafting

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Pimecuronium bromide (Arduan®) is a new, long-acting, nondepolarizing steroid muscle relaxant structurally related to pancuronium bromide.1 The neuromuscular potency of pimecuronium compared to pancuronium is 1: 1.5, respectively.2 Its neuromuscular effects, as well as its pharmacokinetics, are similar to those of pancuronium.2,4,5

Studies in animals suggest that pimecuronium does not induce hemodynamic changes related to histamine release or to an effect on the autonomic nervous system.5 In early human studies with doses as great as 1.5 × ED₉₅, pimecuronium provided adequate muscle relaxation without significant hemodynamic effects,6,7 although some degree of bradycardia has occasionally been reported.8 Whether bradycardia is accentuated by large doses of pimecuronium and beta-adrenergic blocking drugs or calcium channel blocking drugs is not clear.

In contrast, pancuronium may produce tachycardia, even in patients treated with beta-adrenergic blocking drugs,9 which may induce myocardial ischemia in patients with coronary-artery disease.10

The aim of the present study was to compare the cardiovascular effects of pimecuronium with those of pancuronium during induction of anesthesia with midazolam and fentanyl in patients undergoing coronary artery bypass grafting, and to determine whether or not pimecuronium causes bradycardia and hypotension.

MATERIALS AND METHODS

Informed consent was obtained from 30 ASA physical status 2 and 3 patients (mean ± SD age and weight, 59 ± 9 yr, 75 ± 14 kg, respectively) about to undergo elective coronary artery bypass surgery. Local hospital committee approval was also obtained. Excluded from the study were patients with unstable angina, clinical signs of left ventricular failure, valvular heart disease, known arterial hypertension, and patients with liver or kidney disease.

The patients were randomly assigned to one of the four treatment groups. Group 1 (n = 8) received pancuronium at a dose of 0.15 mg/kg (= ED₉₅ × 2); groups 2 (n = 6), 3 (n = 8), and 4 (n = 8) received pimecuronium at doses of 0.05 mg/kg (= ED₉₅ × 1), 0.1 mg/kg (= ED₉₅ × 2), or 0.15 mg/kg (= ED₉₅ × 3), respectively.6

Preoperative beta-adrenergic blocking drug and calcium channel blocking drug therapy was continued until the morning of surgery. All patients were premedicated with morphine 0.1 mg/kg im and diazepam 0.15 mg/kg po 1 h prior to surgery. After arrival in the induction room, supplemental oxygen was administered through a face mask, ECG electrodes attached, and a peripheral ve-