Airway Pressure Release Ventilation (APRV)*
A Human Trial

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Airway pressure release ventilation (APRV) is a new approach to ventilatory support that provides artificial ventilation of the lungs by releasing positive airway pressure (Paw), thereby acutely reducing lung volume below functional residual capacity (FRC). The APRV system delivers continuous positive airway pressure (CPAP) and augments ventilation as an adjunct to CPAP in dogs with normal lungs. APRV provided ventilation and oxygenation equivalent to that provided by conventional positive pressure ventilation (PPV) and was superior to conventional ventilatory support with positive end-expiratory pressure (PEEP) in dogs with acute lung injury (ALI). We sought to determine APRV’s ability to support ventilation and oxygenation of patients recovering from the mild pulmonary dysfunction which follows cardiopulmonary bypass.

METHODS

After institutional approval, 14 consenting patients received radial arterial cannulae and balloon-tipped, flow-directed pulmonary artery catheters before cardiac revascularization operations. General anesthesia was induced with an intravenous infusion of fentanyl, 50 to 100 μg/kg or sufentanyl, 10 μg/kg, Pancuronium bromide, 0.1 mg/kg, provided muscle relaxation. Postoperatively, ventilation was controlled with a tidal volume of 12 ml/kg delivered at a rate sufficient to maintain pHa between 7.36 and 7.44. The fractional concentration of inspired oxygen (FiO2) was adjusted to keep PaO2 between 80 and 100 mm Hg. A pulse oximeter and an infrared capnograph continuously monitored arterial oxyhemoglobin saturation (SaO2) and end-tidal PCO2, respectively.

The patient resided in the intensive care unit for one hour and remained hemodynamically stable, arterial and mixed-venous blood gas and pH values were measured. Cardiac output was determined with a thermodilution technique using a 10 ml room-temperature injectate during exhalation. The peak Paw for every ventilatory cycle during one minute was averaged and recorded as the patient’s peak Paw Systemic (SVR) and pulmonary (PVR) vascular resistance, left (LVSW) and right (RVSW) ventricular stroke work, and stroke volume (SV) were calculated using standard equations. The APRV system was similar to that described previously. A Venturi device that was powered by a 90 psi oxygen source provided a constant air/oxygen mixture with a flow exceeding 90 L/min. Gas flowed through a heated, wick-type humidifier, past the patient and the closed APRV valve, and exited through a threshold resistor (CPAP) valve. The CPAP valve and high continuous gas flow created a 10 to 12 cm H2O elevation in airway pressure, and thus, increased FRC. When the APRV valve opened, airway pressure decreased from the CPAP level to near ambient, and the elastic recoil of the lungs and chest wall caused lung volume to decrease rapidly. After 1.5 seconds, the APRV valve again closed, CPAP was re instituted, and lung volume increased.

After data were collected during conventional PPV, the APRV circuit was attached to the patient’s tracheal tube with 10 to 12 cm H2O CPAP. Alveolar ventilation was provided by decreasing airway pressure to near-ambient for 1.5 seconds with a frequency sufficient to maintain PaCO2 and pHa within the same ranges specified during PPV. After 30 minutes of APRV, measurements were repeated. Each patient’s FiO2 remained constant throughout the study. Patients were weaned from ventilatory support using APRV within three to eight hours of arrival in the ICU. As narcotics and muscle relaxation waned and patients began to breathe spontaneously, the APRV rate was decreased gradually, as long as pHa remained greater than 7.36. Eventually, zero APRV breaths per minute were delivered, and the patients breathed spontaneously with 10 to 12 cmH2O CPAP.

Mean values during PPV and APRV were compared using Student’s t-test for paired samples.

RESULTS

While patients were paralyzed and apneic, PPV and APRV supported ventilation and arterial oxygenation equally successfully in all cases. Mean arterial Po2,
Table 1—Arterial Blood Gas and pH during CMV and APRV (±SD) *

<table>
<thead>
<tr>
<th>Variable</th>
<th>PPV</th>
<th>APRV</th>
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<tbody>
<tr>
<td>PaO2 (mm Hg)</td>
<td>107 ± 16</td>
<td>116 ± 23</td>
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<tr>
<td>PaO2/FIO2 (mm Hg)</td>
<td>263 ± 40</td>
<td>281 ± 5</td>
</tr>
<tr>
<td>PaCO2 (mm Hg)</td>
<td>36 ± 3</td>
<td>38 ± 4</td>
</tr>
<tr>
<td>pHa</td>
<td>7.39 ± 04</td>
<td>7.38 ± 04</td>
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* Differences are not significant statistically.

Pco2, pH, and PaO2/FIO2 did not differ when apneic patients were ventilated with PPV or APRV (Table 1). Mechanical ventilatory rate and minute ventilation were similar during APRV and PPV while patients were apneic. However, mean peak Paw was greater during PPV (38 ± 6 cm H2O) (mean ± SD) than during APRV (11 ± 2 cm H2O) (p<0.0001). Measured and calculated hemodynamic variables were similar during PPV and APRV (Table 2). The SaO2 always exceeded 95 percent, regardless of ventilatory mode.

As patients emerged from anesthesia and sedation, they appeared comfortable while receiving APRV and while breathing spontaneously. No patient complained of dyspnea, and all denied shortness of breath, even when asked directly. All patients were weaned easily and successfully to CPAP after receiving APRV. No patient developed barotrauma.

**DISCUSSION**

Cardiopulmonary bypass results in mild acute lung injury by increasing lung water,1,4 reducing lung volume, and creating mismatching of ventilation and perfusion.5,6 After cardiopulmonary bypass, CPAP effects an increase in lung volume, lung compliance, and PaO2.6 Patients who receive narcotic-oxygen anesthesia require full ventilatory support in the immediate postoperative period, and partial ventilatory support thereafter, while narcosis and muscle relaxation resolve. Thus, patients who undergo cardiopulmonary bypass develop both homogenous, mild acute lung injury, and iatrogenic ventilatory failure; they are an excellent population for the study of ventilatory support techniques. Our results demonstrate that APRV is effective for both conditions, since it provided CPAP and the variable amounts of ventilatory support needed as drug effects waned. Moreover, APRV supported oxygenation and ventilation in apneic and in spontaneously breathing patients with mild acute lung injury, without depressing cardiac function to a greater extent than PPV. The efficacy of APRV in patients with mild acute lung injury suggests that it similarly may be effective in providing CPAP and variable ventilatory support to patients with more severe lung injury.

Patients with acute lung injury often require only CPAP to improve arterial oxygenation, so that a nontoxic FIO2 will suffice. The CPAP also may improve lung mechanics and reduce work of breathing. However, despite appropriate use of CPAP, some patients nevertheless require ventilatory assistance. Such patients usually need partial ventilatory support; total support rarely is necessary or desirable.8 When CPAP is added to conventional positive pressure ventilation as a secondary feature, it may not provide optimal oxygenation or ventilation and may depress cardiac function. In contrast, APRV was designed as a method to provide optimal CPAP levels as a primary goal and to augment alveolar ventilation should spontaneous breathing efforts prove inadequate. The patient may reap the mechanical and hemodynamic benefits of CPAP with unrestricted spontaneous ventilation. This suggests that APRV may provide effective gas exchange and ventilatory assistance to patients with acute lung injury with fewer detrimental effects that are often associated with the combination of PPV and PEEP.

The peak Paw required to ventila te patients' lungs during APRV was significantly lower than that recorded during PPV, representing a potential major advantage of APRV. Our subjects' lung-thorax compliance was decreased mildly. Therefore, it is not surprising that we did not observe barotrauma or cardiovascular depression during either PPV or APRV. However, in patients with low lung compliance, appropriately administered conventional PPV commonly generates high peak Paws. During APRV, peak Paw never exceeds the CPAP level, as long as a threshold resistor CPAP valve is employed. Therefore, we hypothesize that APRV will ventilate the lungs of patients with severe acute lung injury at lower peak Paws than either assist-control ventilation or intermittent man-
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REFERENCES