The confirmed artery.

our clinical vascular and present severe received multiple and 11 patent other bronchial hemodynamic angiography usually occurred. Indeed, interruption of the aortic arch: clinicopathologic review of eight cases. Am J Cardiol 1971; 27:271-77

This information is important diagnostic value in cases of aortic arch interruption and should be recommended to patients unwilling to accept invasive cardiac investigation.

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Figure 3. Ascending aortogram showing the interruption (arrow) 2 cm distal to the origin of a large subclavian artery. AA = ascending aorta, IM = innominate artery, RC = right carotid artery, RS = right subclavian artery.

sixth thoracic vertebra 2.5 cm from the origin of the left subclavian artery. A 1-cm long segment of unenhanced soft tissue measuring 1 cm in diameter linked up the two ends of the interrupted aorta. There was calcification on the wall of the upper end of the distal thoracic aorta (Fig 2). The innominate and left subclavian arteries were large. Dilated internal mammary, intercostal, paravertebral, and bronchial collateral were noted.

He subsequently agreed to cardiac catheterization for complete assessment. The ascending aorta was approached from the right brachial artery and the distal aorta was catheterized from the right femoral artery. The ascending aortic pressure measured 160/90 mm Hg and the simultaneous distal aortic pressure 70/50 mm Hg. The hemodynamic findings were otherwise normal and there was no evidence of intracardiac shunting. Ascending aortogram (Fig 3) confirmed the abrupt interruption 2 cm distal to the origin of the left subclavian artery. There was delayed filling of the distal aorta via multiple collaterals. The patient refused surgical intervention and received maintenance antihypertensive treatment.

Discussion

Isolated aortic arch interruption without the presence of a patent ductus arteriosus had been considered a fatal condition. However, since the first case reported in 1964,11 other patients have been described in the literature.6 9 12 Type A interruption was common, although other varieties also occurred. Indeed, it has been postulated that some cases of type A interruption represented progression of severe coarctation to complete occlusion of the aorta.1 As shown in our patient, there was calcification on the wall of the upper end of the distal thoracic aorta, which was a relatively low-pressure system. It was possible that the present aortic interruption had followed severe coarctation, and the turbulent flow across the coarctation had caused vascular trauma and resulted in mural calcification. The clinical course described has been uniformly benign as in our case. The patients were all asymptomatic during infancy and usually presented with exertional claudication or parasthesia in childhood.14 15 Although cardiac catheterization and angiography give the best preoperative information in cases of aortic coarctation, CT has recently emerged as a valuable adjunctive diagnostic investigation.6 9 11 It is also useful in the postoperative period for evaluation of recurrent stenosis and complications such as dissection or aneurysm.9 As illustrated, CT also had important diagnostic value in cases of aortic arch interruption and should be recommended to patients unwilling to accept invasive cardiac investigation.

Airway Pressure Release Ventilation in a Patient with Acute Pulmonary Injury


Airway pressure release ventilation is a recently described method of ventilatory support. It allows spontaneous ven-
Ventilation with CPAP but differs from conventional ventilatory modes because, with APRV, peak inflation pressure never exceeds the level of CPAP, and airway pressure decreases, rather than increases, when tidal volume is delivered. The risk of pulmonary barotrauma and adverse hemodynamic effects associated with conventional modes of positive-pressure mechanical ventilation may be decreased because of lower peak inflation and mean airway pressures. We describe a patient in whom several risk factors for these complications were present who was treated successfully with APRV.

(Chest 1999; 96:679-82)

APRV = airway pressure release ventilation; psig = pounds per square inch gauge

Conventional modes of mechanical ventilation use positive pressure to inflate the lungs; however, high PIP and Paw predispose the patient to increased risk of barotrauma and adverse hemodynamic changes.1-3 Airway pressure release ventilation is a unique mode of ventilatory support that provides alveolar ventilation by intermittently decreasing airway pressure from a preselected level of CPAP.4-6 In contrast, conventional ventilatory techniques intermittently increase airway pressure when a Vr is delivered. The APRV allows spontaneous breathing during CPAP with or without supplemental ventilation; thus, the PIP never exceeds the baseline CPAP.

The following system is used to provide APRV: oxygen from a 50-psig gas source passes through a Venturi jet (Downs flow generator, Vital Signs), entraining sufficient ambient air to produce a continuous flow rate of 90 to 100 L/min. Regulation of the oxygen-air entrainment ratio controls the FIo2. The gas, heated to body temperature and humidified to 100 percent relative humidity, is directed to the patient's airway and finally through a low flow resistant threshold resistor expiratory pressure valve (Vital Signs).7

At regular preset intervals, an electronically controlled solenoid valve on the exhalation limb of the breathing circuit opens, and airway pressure (CPAP) is released (Fig 1). The solenoid valve can be fully opened or closed in less than 10 ms. When the solenoid valve opens, a sudden drop in airway pressure to ambient pressure or to a lower level of CPAP allows gas to leave the lungs and carbon dioxide to be excreted. When the solenoid valve closes, a rapid increase in airway pressure to the original level of CPAP causes the lung to reexpand.

The following case report demonstrates the usefulness of APRV for a patient with acute pulmonary injury.

CASE REPORT

A 32-year-old male scuba diver suffered near-drowning and apparent air embolism, which resulted in cardiopulmonary arrest. Following cardiopulmonary resuscitation at the scene, he was taken to a local hospital, where he was described as cyanotic and comatose. Subcutaneous emphysema of his neck and anterior chest wall was present. After initial stabilization, he was transferred to the Shands Hospital, University of Florida, where physical examination on admission revealed subcutaneous emphysema and decreased bilateral breath sounds. He responded only to deep pain and had a positive oculocephalic reflex ("doll's eyes" response) and minimally reactive pupils. The results of other examinations were unremarkable. A chest roentgenogram showed large right upper lobe and left perihilar infiltrates, pneumomediastinum, and subcutaneous emphysema. A CT scan of the head was normal.

Initial treatment included bilateral chest tube insertion, mechanical ventilation, and hyperbaric oxygen therapy. After three treatments with hyperbaric oxygen, the patient, becoming more responsive to pain, showed moderate neurologic improvement. His pupils were briskly reactive to light, and he moved his eyes and extremities spontaneously. A repeat CT scan of the head on the third day of hospitalization revealed no evidence of brain infarction or cerebral edema. Thereafter, hyperbaric oxygen therapy was discontinued.

On the fifth day of hospitalization, the right chest tube was removed. A few hours later, the patient became progressively

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**Figure 1.** Principles of APRV Venturi device provides gas at high continuous (cont) flow rate and controls FIo2. Gas flows through humidifier (not shown) and then to patient. On inspiratory limb of breathing circuit, threshold resistor valve with low resistance to flow controls level of CPAP A (top). Solenoid valve also attached to expiratory limb is closed, and patient is allowed to breathe spontaneously, as desired, with CPAP. B (middle), Solenoid valve opens; thus airway pressure and volume from lungs are released. C (bottom), Solenoid valve closes, airway pressure is restored, and patient is allowed to breathe with CPAP.
dyspneic and hypoxemic. A chest roentgenogram revealed a right pneumothorax, and the chest tube was reinserted. Because his body temperature rose to 40°C (104°F), specimens for culture were obtained. Sputum was positive for *Pseudomonas aeruginosa, Staphylococcus aureus*, and *Escherichia coli*; chest tube drainage also contained *Pseudomonas*; and cultures of blood and urine were negative. A regimen of therapy with cefazidine, nafcillin, and tobramycin was begun. Arterial blood gas partial pressures continued to deteriorate in spite of ventilatory support. Because he became combative and highly agitated, the patient was sedated with midazolam and paralyzed with pancuronium.

Initially, the patient was treated with IMV and CPAP. At an IMV rate of 14 breaths per minute, CPAP of 14 cm H₂O, and FiO₂ of 0.5, the Ve was 14 L/min, PaO₂ was 147 mm Hg, PaCO₂ was 55 mm Hg, PIP was 45 to 50 cm H₂O, and Paw was 21 cm H₂O (Fig 2); however, increases of IMV and CPAP resulted in hemodynamic instability, and pneumothorax worsened. The patient was then ventilated with APRV; pressure was released 14 times per minute, CPAP was 18 cm H₂O (the airway pressure was released from 18 to 3 cm H₂O; ie, the change in airway pressure was 15 cm H₂O during APRV), Ve was 5.4 L/min, and FiO₂ was 0.5. With these initial settings, PaCO₂ increased to 80 mm Hg. The rate of APRV was then increased to 50 releases per minute, the level of CPAP was increased to 23 cm H₂O (the airway pressure was released from 23 to 3 cm H₂O; ie, the change in airway pressure was 20 cm H₂O during APRV), and Ve was 14 L/min. At this Ve (the same as that during IMV and CPAP), the patient was well oxygenated, and PaCO₂ and Paw decreased from 55 mm Hg and 21 cm H₂O to 38 mm Hg and 12.4 cm H₂O, respectively (Fig 2). Eventually, when pancuronium was discontinued and the patient breathed spontaneously, Paw decreased further to 8.5 cm H₂O.

The remainder of the patient's clinical course was uneventful, and he was discharged. Rehabilitation therapy was required for residual central nervous system dysfunction sustained at the time of his cardiac arrest.

**DISCUSSION**

The development of APRV was stimulated by a better understanding, over time, of the pathophysiologic processes of acute pulmonary injury, of the usefulness of CPAP to treat this problem, and of the risks of high levels of PIP and Paw during mechanical inhalation. Current modes of positive-pressure ventilation (IMV, controlled mechanical ventilation, high-frequency jet ventilation, pressure-controlled ventilation, pressure support, and mandatory minute ventilation) expand the lungs by applying positive pressure to the airway that is higher than the baseline airway pressure. Although effectively producing adequate alveolar ventilation and oxygenation, this technique increases PIP and Paw, which may compromise cardiac output and may increase the incidence of pulmonary barotrauma. These considerations are especially pertinent for patients who have acute pulmonary injury with decreased pulmonary compliance. Airway positive release ventilation has been proposed as an alternative means of support for this group of patients.

In the patient we describe, barotrauma worsened during IMV. When APRV was instituted, a normal PaCO₂ was maintained with the same Ve and a significantly lower PIP and Paw, but with no adverse effect on the patient's hemodynamic status and no worsening of barotrauma. The amount of ventilatory assistance provided by APRV depends on (1) the pressure difference between CPAP and the pressure to which the system releases before represurizing; (2) the frequency with which airway pressure is released (the APRV rate); (3) the duration of pressure release; and (4) lung-thorax compliance. The appropriate level of CPAP is determined by evaluating respiratory mechanics and pulmonary gas exchange. Our current APRV device allows the application of up to 25 cm H₂O of CPAP (solenoid closed) and as little as 2 to 3 cm H₂O of pressure (solenoid open). This change in airway pressure allows gas to move passively in and out of the lungs.

Adjusting the APRV rate is similar to adjusting IMV; ie, the rate is decreased to augment spontaneous breathing to allow the patient to perform the maximal degree of spontaneous ventilation possible, or the rate is increased to maximize mechanical support; however, in contrast to IMV, increasing the APRV rate decreases Paw, because the system

**FIGURE 2. Arterial blood gas levels and Paw for patient treated with IMV and CPAP (in centimeters of water) and then with APRV. Asterisk indicates two numbers that are upper and lower release pressure. Dagger indicates two numbers that are Vt (in milliliters) during APRV and during spontaneous breathing, respectively. MECH RATE, Mechanical ventilatory rate (breaths per minute during IMV and releases per minute with APRV); and SPON RATE, patient's spontaneous breathing rate (breaths per minute). Note that PaCO₂ and Paw were much lower with APRV than with IMV and CPAP at same Ve (14 L/min).**

<table>
<thead>
<tr>
<th>CPAP: 14/3*</th>
<th>MECH: IMV</th>
<th>APRV</th>
<th>APRV</th>
<th>APRV</th>
<th>APRV</th>
<th>APRV</th>
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</thead>
<tbody>
<tr>
<td>RATE: 14</td>
<td>14</td>
<td>20</td>
<td>14</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SPON RATE: 0</td>
<td>0</td>
<td>0</td>
<td>24</td>
<td>42</td>
<td></td>
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</tr>
<tr>
<td>FiO₂: 0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.45</td>
<td>0.45</td>
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<td></td>
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<tr>
<td>VT: 1000</td>
<td>600</td>
<td>700</td>
<td>600/250</td>
<td>575/200</td>
<td></td>
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</tr>
<tr>
<td>VE: 14</td>
<td>8.4</td>
<td>14</td>
<td>14.4</td>
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is depressurized more frequently. Although APRV allows spontaneous breathing, the patient’s V\textsubscript{E} can be controlled by increasing the solenoid opening rate. Thus, APRV can be used for patients with neuromuscular paralysis; as with IMV, the APRV rate is decreased commensurate with the patient’s ability to resume spontaneous ventilation. When mechanical ventilation is no longer required, the solenoid valve is deactivated (i.e., continuously closed), and the system functions as a constant-flow CPAP system.

The pressure release time can be set in seconds. A release time of 1.5 seconds was used for our patient because, in one report,\textsuperscript{7} after one second, the lungs emptied completely (expiratory flow ceased spontaneously as measured by pneumotachograph), and airway pressure stabilized at a lower level; by two seconds, airway pressure continued to remain stable, and no further gas left the lungs.

Airway pressure release ventilation previously has been compared with IPPV in anesthetized dogs.\textsuperscript{8} In normal lungs, APRV and IPPV achieved similar gas exchange and hemodynamic function. When acute pulmonary injury was induced, PIP was significantly lower and arterial oxygenation greater with APRV. Similar V\textsubscript{E} was delivered by both modes, but a lower PaCO\textsubscript{2} with APRV suggested decreased physiologic dead-space ventilation. Similarly, when our patient was ventilated with APRV, PaCO\textsubscript{2} was substantially lower than at the same V\textsubscript{E} with IMV (Fig 2).

We believe that patients with pneumothorax and other forms of barotrauma, as well as patients with hemodynamic instability or those with head injury and intracranial hypertension, will benefit from APRV. This mode of ventilatory support may serve as a bridge for patients who do not require full ventilatory support yet are unable to maintain spontaneous breathing during CPAP alone.

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Recurrent Acute Pulmonary Emboli in Association with Acute Myocardial Infarction

Djahangir J Ahdout, M.D.;* Prabodh M. Damani, M.D.;† and L. Barry Ulta, M.D.;‡

Left ventricular mural thrombi are a common complication of myocardial infarction, but right-sided mural thrombi have been reported only in blunt chest trauma, right ventricular catheterization, and pacemaker insertion. We describe a patient with AMI and subsequent right ventricular mural thrombi and ultimately pulmonary emboli. We believe a prospective study should be conducted first to evaluate the MI as a cause of right-sided mural thrombi and second to assess the right ventricle as a source of pulmonary emboli.

(Chest 1989; 96:693-84)

Right ventricular (RV) mural thrombi have been described at autopsy in association with RV myocardial infarction (RVMI),\textsuperscript{1,3,10-11} by 2D echocardiography in patients with blunt chest trauma\textsuperscript{4} and as an unusual complication of right-sided cardiac catheterization or the pacing catheter.\textsuperscript{5}

We report a case of RV mural thrombus secondary to an inferior infarction that resulted in pulmonary emboli with pulmonary infarction. Left ventricular mural thrombi are a known complication of MI. However, there is only one report in the literature, to our knowledge, regarding thrombus formation in the right ventricle following infarction; in this case, thrombus may have been stimulated by right-sided cardiac catheterization and pacing, as well as incomplete course of thrombolytic therapy.\textsuperscript{6}

CASE REPORT

A 66-year-old man with an unremarkable past medical history but known to be a heavy smoker was admitted to the hospital with sustained retrosternal chest pain and mild hypotension. Serial ECGs and enzyme studies (CPK 1159, 13.4 percent MB) confirmed the diagnosis of an inferior MI. The course of hospitalization was complicated by hypotension. Clear lung fields suggested the possibility of RV infarction, and fluids were administered with good response. He later developed mild left ventricular heart failure and atrial fibrillation, which responded to therapy with digoxin and quinidine as the patient converted to normal sinus rhythm. A 2D echocardiogram at that time demonstrated a dilated RV but no RV thrombus.

He was readmitted three days later for acute onset of right-sided pleuritic chest pain. Although no calf tenderness was found, ventilation-perfusion scans were consistent with pulmonary emboli. Repeated 2D echocardiogram now revealed RV mural thrombi, while RV dilatation had diminished (Fig 1 and 2).

After 12 days of full heparinization, the perfusion scan had normalized, and the patient was discharged receiving sodium warfarin (Coumadin). A third 2D echocardiogram again confirmed the presence of a right apical thrombus. He did well at home until 12 days after discharge, when his Coumadin dosage was decreased

*Pulmonary and Critical Care Fellow at University of California, Irvine Medical Center, Irvine.
†Clinical Instructor, Cardiology Department, Helen Fuld Medical Center, Trenton, NJ.
‡Clinical Instructor, Cardiology Department, Helen Fuld Medical Center, Trenton, NJ.
Reprint requests: Dr. Ahdout, Pulmonary Medicine, UCI Medical Center, 101 City Drive South, Orange, CA 92668