Similarly, insulin therapy does not directly affect neuromuscular function clinically, but chronic complications of diabetes mellitus such as renal insufficiency could result in prolonged effects from nondepolarizing muscle relaxants. The patient in this case had normal renal function. In addition, she received atracurium, which is less dependent upon the renal route of elimination than other nondepolarizing neuromuscular blocking agents.

Theoretically, one concern involving the administration of succinylcholine in a patient with a muscle disorder is the excessive release of potassium secondary to depolarization of abnormal cells. Although this problem has not been documented in patients with dermatomyositis, we attempted to measure any changes in serum potassium following the administration of succinylcholine in our patient. Unfortunately, the blood sample was lost. However, no electrocardiogram changes or clinical signs of hyperkalemia were evident during the care of this patient.

In summary, the results of this case study demonstrate a normal onset, peak effect, and recovery from the neuromuscular blocking actions of succinylcholine and atracurium in a patient with active dermatomyositis. However, interactions between concomitant medications and muscle relaxants used intraoperatively may have influenced the results of this report through effects on neuromuscular transmission.

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The Effect of Positive End-expiratory Pressure on Right-to-left Shunting at the Atrial Level as Documented by Transesophageal Echocardiography

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The combination of a patent foramen ovale and positive end-expiratory pressure (PEEP) has been shown to be associated with an increased incidence of paradoxical embolism, significant shunting at the atrial level, and refractory hypoxemia. We report a case of intraoperative development of right ventricular failure secondary to right ventricular infarction during coronary artery bypass graft surgery. After the development of severe refractory hypoxemia, bedside transesophageal echocardiography (TEE) was performed and confirmed the presence of a right-to-left shunt at the atrial level. This case report demonstrates the deleterious effect of increasing PEEP on the shunt across a patent foramen ovale as documented by TEE.

CASE REPORT

A 74-yr-old man with a history of angina pectoris of recent onset was scheduled for coronary artery bypass graft surgery. He had a longstanding history of hypertension and non-insulin-dependent diabetes mellitus. His blood pressure was 148/86 mmHg with a regular heart
TABLE 1. Hemodynamic and Arterial Blood Gas Values During the First Postoperative Day

<table>
<thead>
<tr>
<th></th>
<th>6 PM</th>
<th>10 PM</th>
<th>2 AM</th>
<th>6 AM</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEEP (cmH₂O)</td>
<td>5</td>
<td>8</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Cardiac output (l/min⁻¹·m⁻²)</td>
<td>6.3</td>
<td>6.1</td>
<td>5.5</td>
<td>3.6</td>
</tr>
<tr>
<td>Right atrial pressure (mmHg)</td>
<td>17</td>
<td>18</td>
<td>19</td>
<td>21</td>
</tr>
<tr>
<td>Pulmonary arterial pressure (mmHg)</td>
<td>22/15</td>
<td>27/20</td>
<td>26/17</td>
<td>25/18</td>
</tr>
<tr>
<td>pH</td>
<td>7.47</td>
<td>7.52</td>
<td>7.45</td>
<td>7.36</td>
</tr>
<tr>
<td>PₐCO₂ (mmHg)</td>
<td>36</td>
<td>37</td>
<td>43</td>
<td>48</td>
</tr>
<tr>
<td>PₐO₂ (mmHg)</td>
<td>58</td>
<td>57</td>
<td>45</td>
<td>44</td>
</tr>
<tr>
<td>HCO₃⁻ meq/l</td>
<td>26.1</td>
<td>29.9</td>
<td>27.7</td>
<td>26.8</td>
</tr>
<tr>
<td>FiO₂</td>
<td>0.6</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
</tbody>
</table>

rate of 78 beats/min. His physical examination was unremarkable. His resting electrocardiogram demonstrated a left bundle branch block with left axis deviation. Selective coronary angiography showed severe triple vessel coronary artery disease with reasonably well-preserved left ventricular function. Ejection fraction was estimated to be 50%.

The patient subsequently presented for coronary artery bypass grafting. After insertion of two 10-G intravenous catheters and a 20-G right radial arterial catheter, anesthesia was induced with midazolam, fentanyl, and pancuronium. After insertion of a right internal jugular pulmonary artery catheter, the patient underwent coronary artery bypass grafting using saphenous vein segments. Anesthesia was maintained with fentanyl and pancuronium.

During the operation, while attempting to separate the patient from cardiopulmonary bypass, the surgeon observed that the right heart began to dilate. The systolic blood pressure decreased to 50 mmHg, and the cardiac index was noted to be 1.7 l/min⁻¹·m⁻². The pulmonary artery pressure was 40/24 mmHg. The patient developed atrial fibrillation, and several attempts at cardioversion were unsuccessful. Cardiopulmonary bypass was re instituted, and an additional saphenous vein graft was then anastomosed to the acute marginal artery. After another unsuccessful attempt at withdrawing cardiopulmonary bypass, placement of an intraaortic balloon pump and institution of dobutamine and epinephrine therapy finally made it possible to discontinue cardiopulmonary bypass. Contractility of the right ventricle was improved, and left ventricular contractility was good. The systolic blood pressure was 90 mmHg, and the cardiac index was now noted to be 2.1 l/min⁻¹·m⁻². The pulmonary artery pressure was now 80/18 mmHg. At this point the patient was hemodynamically stable and was transferred to the surgical intensive care unit.

On arrival at the surgical intensive care unit, the systolic blood pressure was 95 mmHg and the pulmonary artery pressure 26/20 mmHg. The pulmonary capillary wedge pressure was 16 mmHg; central venous pressure was 15 mmHg. Arterial blood gas analysis showed a pH of 7.34, PₐCO₂ of 54 mmHg, PₐO₂ of 112 mmHg, and a HCO₃⁻ of 18.2 meq/l while FiO₂ was 0.6, tidal volume 800 ml, respiratory rate 14 breaths/min, and PEEP 3 cmH₂O.

On the first postoperative day, the patient developed arterial hypoxemia that was refractory to increasing FiO₂. During the ensuing 12-h period, medical management included increasing the FiO₂ from 0.6 to 1.0. PEEP, which initially was set at 3 cmH₂O, was increased to a maximum of 12 cmH₂O; however, arterial PₐO₂ values continued to decrease. Auscultation of the lungs was remarkable for decreased breath sounds bilaterally in the lower lung segments. Chest x-ray revealed slight pulmonary vascular congestion, a left pleural effusion, and left lower lobe atelectasis. Hemodynamic data showed increasing right arterial pressures with relatively unchanged pulmonary artery pressures. The cardiac output decreased from 6.3 to 3.6 l/min⁻¹·m⁻² during this 12-h period (table 1).

These hemodynamic changes, with the refractory hypoxemia, were consistent with an intracardiac right-to-left shunt, and a bedside TEE was performed. TEE examination revealed 1) severe hypokinesis of both right and left ventricles; 2) right and left atrial enlargement; 3) a right-to-left shunt across a patent foramen ovale with a bulging interatrial septum into the left atrium; 4) moderate tricuspid regurgitation; and 5) a thrombus attached to the pulmonary artery catheter at the right atrial level and crossing through the septal defect into the left atrium (fig. 1).

At this point, while directly observing the interatrial defect with TEE color Doppler flow and transiently increasing the PEEP, we recorded an increasing size of the color Doppler jet of right-to-left shunt-

![Fig. 1. A thrombus attached to the pulmonary artery catheter at the right atrial level, crossing through the bulging foramen mem-
brane of the interatrial septum and the septal defect into the left atrium. AO = aorta; LA = left atrium; PFO = patent foramen ovale; RA = right atrium; arrows = right atrial thrombus attached to pulmonary artery catheter.](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931324/ on 01/13/2018)
At autopsy, an acute infarction of the posterior right ventricular wall and septum were noted, as was a patent foramen ovale measuring 5 mm in diameter.

**DISCUSSION**

Right-to-left shunting has been demonstrated previously across an atrial septal defect in patients sustaining an acute right ventricular insult causing refractory hypoxemia. Other factors contributing to this refractory hypoxemia include 1) mechanical as opposed to spontaneous ventilation; 2) pulmonary embolism; 3) vasoactive drugs such as nitroglycerin, which can decrease arterial
P_{O_2} by decreasing hypoxic pulmonary vasoconstriction or by decreasing systemic pressure and thereby left atrial pressure more than right heart pressures; 4) an increase in preload, which can increase the right-to-left atrial pressure gradient in the presence of right ventricular failure; and 5) PEEP.\textsuperscript{1,4,4}

The physiologic mechanism by which PEEP causes increased right-to-left shunting through a patent foramen ovale is not completely understood. It is known that PEEP increases end-expiratory lung volumes, thereby increasing pulmonary vascular resistance.\textsuperscript{5} At high PEEP, the increase in pulmonary vascular resistance has been shown to decrease right ventricular ejection fraction as well as increase right ventricular end diastolic pressure. This combination causes increased right atrial pressure, especially when right ventricular compliance is compromised, as occurred in our case report as a result of right ventricular infarction. As shown by Lemaire \textit{et al.},\textsuperscript{6} reversal of the normal left atrial to right atrial pressure gradient, as may occur secondary to any form of increased right atrial pressure and as may be worsened by increasing PEEP, causes shunting of venous blood through a patent foramen ovale. We believe that this was the most likely explanation of the right-to-left atrial shunting that occurred in our patient. It is possible that changing PEEP changed the relative position of the transducer and the jet, thereby simply enhancing the angle of interrogation and revealing more of the jet volume without actually altering the magnitude of the right-to-left shunt. In light of the worsening arterial hypoxemia with concomitant increases in PEEP, however, this seems an unlikely explanation.

Although the incidence of paradoxical embolism of venous thrombi to the systemic circulation via abnormal intracardiac channels is exceedingly rare,\textsuperscript{1} we were able to document by TEE the existence of a thrombus crossing the patent foramen ovale in our patient. The presence of this thrombus did not play a role in our patient's eventual demise. However, it served to point out the importance of its consideration whenever a patient presents with an intracardiac shunt.

This case underscores the importance of early consideration of an intracardiac shunt in the differential diagnosis of a patient with refractory hypoxemia that develops after a myocardial infarction.

\textbf{REFERENCES}