Intraoperative Tension Pneumopericardium with Tamponade after Ligation of Patent Ductus Arteriosus in a Premature Neonate

PAUL R. HICKEY, M.D.*

Although tension pneumothorax is a well-known cause of intraoperative cardiovascular collapse, tension pneumopericardium with cardiac tamponade may produce equally catastrophic results. Both of these potentially lethal complications occur in similar clinical settings and present similar clinical pictures. Although isolated tension pneumopericardium is a known complication of positive pressure ventilation in premature neonates with pulmonary disease in the intensive care setting,1 isolated pneumopericardium with tamponade occurring intraoperatively has not been previously reported. Several cases of intraoperative tension pneumopericardium and tamponade associated with pneumothorax and pneumomediastinum have resulted from very high airway pressures in infants.2 These multiple complications occurred during the era when breathing circuits without pop-off valves, such as the Ayer's t-piece, were widely used for controlled ventilation.2 We report an intraoperative case of isolated tension pneumopericardium causing tamponade without high airway pressures in a premature neonate after ligation of a patent ductus arteriosus (PDA).

REPORT OF A CASE

A premature, 4-day-old male neonate (28 weeks' gestation) weighing 1.2 kg was scheduled for ligation of a PDA. The infant had respiratory distress syndrome, a large PDA, required iv dopamine therapy, and was dependent on mechanical ventilation, requiring progressively higher inspiratory pressures and fractional inspired O2 concentration (FiO2). Because a grade IV intraventricular hemorrhage contraindicated pharmacologic closure of the PDA with indomethacin, surgical ligation was elected to decrease pulmonary blood flow and facilitate weaning from ventilator support. On the morning before the operative procedure, a chest roentgenogram showed the continued presence of severe bilateral hyaline membrane disease, but for the first time, pulmonary interstitial emphysema was noted. Review of the roentgenogram in the operating room showed no mediastinal, pleural, or pericardial free air, nor had any been seen previously.

An umbilical artery catheter, a 2.5-mm oral endotracheal tube with a leak at 35 cmH2O, and two iv catheters were in place and the infant was receiving a dopamine infusion (5 μg · kg⁻¹ · min⁻¹) on arrival in the operating room. The infant was placed on a warming blanket under warming lights while monitors were applied, which included electrocardiogram (ECG), pulse oximeter, temperature probe, blood pressure cuff, an esophageal stethoscope, and a transducer for the arterial blood pressure. For ventilation, a modified Mapleson D circuit was used, which included a heated humidifier and an airway pressure manometer, which recorded maximum airway pressure.

Anesthesia was induced with fentanyl (40 μg) and pancuronium (0.2 mg) iv without any notable change in vital signs (heart rate 150-160 beats/min, arterial blood pressure 50/30 mmHg, temperature 35º C). No additional anesthetic drugs were given during the case. Arterial oxygen saturations after induction were 98% and were subsequently maintained in the mid-90% range by adjusting FiO2. Perioperative arterial blood gases, pulse oximeter arterial saturations, and ventilatory requirements are shown in table 1. Ligation of the PDA proceeded uneventfully. The left lung was diffusely thickened and abnormal, but no free air was noted in the chest or pericardium during the procedure. Lung compliance did not change notably during retraction of the left lung. With the use of peak inspiratory pressure (PIP) of 25–30 cmH2O, arterial oxygen saturations were maintained in the mid-90% range with FiO2 of 0.8–0.9 without requiring positive end-expiratory pressure (PEEP). Arterial blood pressure increased briefly to 65/42 mmHg after ligation of the PDA before gradually decreasing to preligation values.

The pericardium and lung were unchanged in appearance before chest closure, and after closure no air leaks were noted from the left chest tube. During closure of the subcutaneous layers, arterial oxygen saturation abruptly decreased to 60% and then decreased further to 40% within several minutes, despite increases in FiO2 and good chest excursions. Simultaneously, the heart rate decreased to 70 beats/min and then to 60 beats/min, and heart sounds became faint while the arterial blood pressure decreased to 30/20 mmHg and then 25/18 mmHg despite an increase in the rate of the dopamine infusion. The wound was covered, the drapes removed, and the chest auscultated. Good bilateral breath sounds were heard. Heart sounds were distant, but no pericardial crunch or mill wheel murmurs were heard with either a precordial or esophageal stethoscope. The infant was grossly cyanotic, but no subcutaneous emphysema was noted in the neck or chest wall. Endotracheal tube position was checked by laryngoscopy and was noted to be at an appropriate depth in the trachea. Although transillumination of the thorax showed no definite pneumothorax on the right, a right chest tube was inserted because of the clinical picture and apparent patency of the left chest tube. No air under tension was noted on insertion of the right chest tube, and the infant's condition did not improve. Arterial blood gases drawn immediately before and after insertion of the tube showed poor oxygenation and developing metabolic acidosis (table 1). Continued hypotension, bradycardia, and cyanosis despite previous boluses of iv epinephrine and calcium prompted emergent reopening of the left thoracotomy.
TABLE 1. Perioperative Ventilatory Requirements and Arterial Blood Gas Values

<table>
<thead>
<tr>
<th>Time</th>
<th>PIP/PEEP</th>
<th>Respiratory rate/FEV&lt;sub&gt;1&lt;/sub&gt;</th>
<th>pH&lt;sub&gt;L&lt;/sub&gt;</th>
<th>P&lt;sub&gt;CO&lt;sub&gt;2&lt;/sub&gt;&lt;/sub&gt;</th>
<th>P&lt;sub&gt;AO&lt;/sub&gt;</th>
<th>Arterial saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperative (nursery)</td>
<td>40/5</td>
<td>59/1.9</td>
<td>7.37</td>
<td>55</td>
<td>73</td>
<td>94</td>
</tr>
<tr>
<td>After induction</td>
<td>28/0</td>
<td>50/1.0</td>
<td>7.40</td>
<td>46</td>
<td>154</td>
<td>99</td>
</tr>
<tr>
<td>Chest closure and circulatory collapse</td>
<td>32/0</td>
<td>50/1.0</td>
<td>7.47</td>
<td>37</td>
<td>26</td>
<td>60</td>
</tr>
<tr>
<td>After right chest tube</td>
<td>32/0</td>
<td>50/1.0</td>
<td>7.32</td>
<td>42</td>
<td>30</td>
<td>45</td>
</tr>
<tr>
<td>After pericardial incision</td>
<td>29/0</td>
<td>50/1.0</td>
<td>7.24</td>
<td>44</td>
<td>176</td>
<td>97</td>
</tr>
<tr>
<td>Postoperative (nursery)</td>
<td>36/5</td>
<td>40/1.0</td>
<td>7.48</td>
<td>33</td>
<td>129</td>
<td>99</td>
</tr>
</tbody>
</table>

PIP: peak inspiratory pressure; PEEP: positive end-expiratory pressure; F<sub>IO<sub>2</sub></sub>: fractional inspired O<sub>2</sub> concentration.

When the left chest was reopened, no free air was noted and lung movement was good, but the pericardium was bulging and tenses distended with air. When the pericardium was incised, air under pressure escaped and the vital signs dramatically improved; the arterial blood pressure immediately increased to 70/50 mmHg, the heart rate to 150 beats/min, and arterial oxygen saturation to 97%. After removal of a pericardial window, the left chest tube was repositioned in the anterior mediastinum to drain the pericardial space, and the chest was closed without further incident. At the conclusion of the case, the airway monitor had recorded a maximum airway pressure of 35 cmH<sub>2</sub>O during the case, consistent with the leak around the endotracheal tube.

The infant was transported to the neonatal nursery and arrived with little change in vital signs. No air leak was noted from either chest tube, and on postoperative chest roentgenogram, no pneumothorax, pneumomediastinum, pneumopericardium, or subcutaneous emphysema was evident. The infant was stable for 24 h postoperatively until the pneumopericardium spontaneously recurred despite the pericardial drainage tube. Subsequently, bilateral pneumothoraces and repeated recurrences of the pneumopericardium occurred, ventilatory pressures escalated further, and sepsis developed; the infant died 5 days postoperatively.

**DISCUSSION**

Without immediate pericardial decompression, most infants with pneumopericardium and cardiac tamponade will die rapidly. Although tension pneumopericardium is easily seen on chest roentgenogram, the time required to obtain a portable roentgenogram will not be tolerated by a moribund infant with tamponade. The classic mill wheel murmur heard with pneumopericardium was described in 1844 but usually occurs in complicated pneumopericardium because of the mingling of air and substantial amounts of fluid. Most reported cases of pneumopericardium with tamponade occur in neonates receiving assisted ventilation (81% of cases recently reviewed), but in only a small percentage of these cases was the classic murmur heard. Rather, the most frequent auscultatory findings reported were muffled, absent, or distant heart sounds, as occurred in the current case.

Tension pneumopericardium usually presents in infants as an abrupt clinical decline marked by hypotension, cyanosis, muffled heart sounds, bradycardia, and low voltage on the ECG. Pulsus paradoxus is not observed in the newborn with tamponade secondary to pneumopericardium and was not observed in our case. The case fatality rate is high, partly because diagnosis is often delayed.

Reopening the thorax is often crucial when cardiovascular collapse occurs in an infant after a cardiovascular procedure. This maneuver was delayed for several minutes in our case while endotracheal tube position was checked and a right chest tube was inserted to rule out definitively a right tension pneumothorax. Despite the presence of breath sounds and negative transillumination of the chest (both can be unreliable in small infants), the clinical picture prompted insertion of an immediately available chest tube on the right.

This infant was predisposed to the complications of barotrauma by his underlying lung pathologic features. The pulmonary interstitial emphysema seen preoperatively in this neonate results from the rupture of immature alveoli with subsequent dissection and tracking of air along the areolar tissue of the perivascularg sheaths in the lung. Distal dissection with rupture of visceral pleura produces pneumothorax, whereas central dissection may produce pneumomediastinum or, alternatively, pneumopericardium. In the presence of pulmonary interstitial emphysema, relatively little further central dissection may result in rupture through the pericardial reflection of the pulmonary veins, which produces the isolated pneumopericardium seen in infants on positive pressure ventilation.

Once air entry into the pericardial space is established, PIP in normal ranges is sufficient to cause tamponade. Air introduced experimentally into the pericardium of the dog at a pressure of 16.5 cmH<sub>2</sub>O produces critical cardiac tamponade. This is signaled by sudden decreases in heart rate, cardiac output, and aortic pressure and is caused by near equalization of end-diastolic pressures in all cardiac chambers. Cohen et al. showed that pneumopericardium was related to PIP rather than PEEP and occurred in neonates with diseased lungs with PIP as low as 35 cmH<sub>2</sub>O.

Although the pulmonary interstitial emphysema clearly set the stage, the immediate cause of the pneumopericardium in our case is unclear. We speculate that retraction and surgical exposure of the PDA disrupted tissue planes
in close proximity to the pericardial reflections of the great vessels, which allowed air already present in the perivas-
cular sheaths to enter the pericardium. Although maxi-
mum airway pressures used during the procedure were no higher than those used preoperatively (PIP 35
cmH₂O), these pressures were probably sufficient to cause
further air dissection, especially when the right lung was
dependent and the left lung retracted.

The tendency toward recurrence after initial relief of
air tamponade often makes treatment ultimately unsuc-
sessful. Placment of an anterior pericardial drainage tube
under direct vision is the treatment of choice, but mainte-
nance of long-term patency is difficult. Recurrence is
frequent because further barotrauma is generated by the
underlying lung pathologic features and resultant pro-
longed ventilatory support.

Tension pneumopericardium occurs in association with
pneumothorax, pneumomediastinum, and subcutaneous
emphysema after very high airway pressures, and signs
of these complications may obscure those of tension
pneumopericardium. In our case, there were no excessive
airway pressures, and only tension pneumopericardium
occurred. Previous cases involved multiple complications
and high airway pressures during positive pressure ven-
tilation produced by prolonged occlusion of the open arm
of Ayer’s t-piece. This directly exposes the airway to the
pressures flowing from the anesthesia machine. Because
of the absence of a pop-off valve and reservoir bag, con-
tinuous vigilance is needed with Ayer’s t-piece to avoid
inadvertent high airway pressures. Modern anesthesia
circuits, such as the Mapleson D system we used, incor-
porate both pop-off valves and reservoir bags to modulate
airway pressures. Although modern systems have mark-
edly reduced the incidence of high airway pressures, pro-
longed occlusion of the expiratory limb (analogous to the
open arm of Ayer’s t-piece) or complete closure of the
pop-off valve can still result in very high airway pressures
and barotrauma.

This case report illustrates that in infants with lung
disease, especially in those with pulmonary interstitial
emphysema, intraoperative pneumopericardium with
tamponade may develop despite careful control and mon-
itoring of airway pressures. In these patients, tension
pneumopericardium as well as pneumothorax must be
considered when abrupt intraoperative cardiovascular
collapse occurs. Pneumopericardium with tamponade is
signaled by bradycardia, cyanosis, hypotension, and dis-
tant heart sounds. Although properly used modern anes-
thesia circuits markedly reduce the possibilities of baro-
trauma, they do not entirely prevent it in infants with
diseased lungs. When modern circuits are used improp-
erly, high airway pressures in any patient may produce
tension pneumopericardium along with pneumothorax,
pneumomediastinum, or subcutaneous emphysema.

REFERENCES
1. Lawson EE, Gould JB, Taesch HW: Neonatal pneumopericardium:
2. Loflin JW, Susen AF, Marcy JH, Sherman FE: Pneumopericardium
   resulting in cardiac tamponade. Ann Thorac Surg 37:
   511–518, 1984
4. Reppert SM, Ment LR, Todres ID: The treatment of pneumo-
5. Reed JR, Thomas WP: Hemodynamics of progressive pneumo-
6. Cohen DJ, Baumgart S, Stephenson LW: Pneumopericardium in
   neonates: Is it PEEP or is it PIP? Ann Thorac Surg 35:
   179–183, 1983
7. Emery RW, Foker J, Thompson TR: Neonatal pneumopericardium:

Anesthesiology
64:643–644, 1986

An Alternative Method to Secure an Endotracheal Tube in Infants with Midline Facial Defects

DAVID R. JOBES, M.D.,* AND SUSAN CRAIG NICOLSON, M.D.†

Small infants with large midline facial defects pose dif-
ficulty in securing an endotracheal tube. We describe a
method that solved this problem in our patient. We believe
it may be useful in similar cases.

* Associate Professor of Anesthesia, University of Pennsylvania
   School of Medicine, and Senior Anesthesiologist, The Children’s Hos-
   pital of Philadelphia.
† Assistant Professor of Anesthesia, University of Pennsylvania
   School of Medicine, and Assistant Anesthesiologist, The Children’s
   Hospital of Philadelphia.

Received from the Department of Anesthesia and Critical Care,

REPORT OF A CASE

The patient was an 11-week-old, 2.6-kg female infant with Wolf-
Hirschhorn syndrome (deletion of the short arm of chromosome 4).
With her trachea intubated, she was transported from the extensive

Accepted for publication December 17, 1985.
Address reprint requests to Dr. Nicolson: Department of Anesthesia and
Critical Care, Children’s Hospital of Philadelphia, 34th Street and
Civic Center Boulevard, Philadelphia, Pennsylvania 19104.

Key words: Anesthesia: pediatric. Intubation, endotracheal: tech-

Downloaded From: http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931399/ on 06/20/2017