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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 O_2 concentration (FI_{O_2}). 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 cmH_2O, and two iv catheters were in place and the infant was receiving a dopamine infusion ($5\ \mu g \cdot kg^{-1} \cdot min^{-1}$) 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 35° 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 FI_{O_2}. 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 cmH_2O, arterial oxygen saturations were maintained in the mid-90% range with FI_{O_2} 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 FI_{O_2} 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.

* Assistant Professor of Anaesthesia.

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Address reprint requests to Dr. Hickey: Department of Anesthesia, Children's Hospital, 300 Longwood Avenue, Boston, Massachusetts 02115.

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TABLE 1. Perioperative Ventilatory Requirements and Arterial Blood Gas Values

Time	PIP/PEEP	Respiratory rate/FI_{O_2}	pH_a	$Paco_2$	Pao_2	Arterial saturation (%)
Preoperative (nursery)	40/5	39/.9	7.37	55	73	94
After induction	28/0	50/1.0	7.40	46	154	99
Chest closure and circulatory collapse	32/0	50/1.0	7.47	37	26	60
After right chest tube	32/0	50/1.0	7.32	42	30	45
After pericardial incision	29/0	50/1.0	7.24	44	176	97
Postoperative (nursery)	36/5	40/1.0	7.48	33	129	99

PIP: peak inspiratory pressure; PEEP: positive end-expiratory pressure: FI_{O_2}: fractional inspired O_2 concentration.

When the left chest was reopened, no free air was noted and lung movement was good, but the pericardium was bulging and tensely 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 manometer had recorded a maximum airway pressure of 35 cmH_2O 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.[4] 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.[3] Pulsus paradoxus is *not* observed in the newborn with tamponade secondary to pneumopericardium and was not observed in our case.[4] The case fatality rate is high, partly because diagnosis is often delayed.[4]

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 perivascular 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.[4]

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_2O produces critical cardiac tamponade.[5] 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.*[6] showed that pneumopericardium was related to PIP rather than PEEP and occurred in neonates with diseased lungs with PIP as low as 35 cmH_2O.

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

† Bricheteau: Observat d'hydropneumopericarde accompagne d'un bruit de fluctuation perceptible á l'orielle. Arch Gen Med 4:334, 1844.

in close proximity to the pericardial reflections of the great vessels, which allowed air already present in the perivascular sheaths to enter the pericardium. Although maximum airway pressures used during the procedure were no higher than those used preoperatively (PIP 35 cmH_2O), 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 unsuccessful. Placement of an anterior pericardial drainage tube under direct vision is the treatment of choice, but maintenance of long-term patency is difficult.[7] Recurrence is frequent because further barotrauma is generated by the underlying lung pathologic features and resultant prolonged 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 ventilation produced by prolonged occlusion of the open arm of Ayer's t-piece.[2] 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, continuous 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, incorporate both pop-off valves and reservoir bags to modulate airway pressures. Although modern systems have markedly reduced the incidence of high airway pressures, prolonged 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 monitoring 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 distant heart sounds. Although properly used modern anesthesia circuits markedly reduce the possibilities of barotrauma, they do not entirely prevent it in infants with diseased lungs. When modern circuits are used improperly, high airway pressures in any patient may produce tension pneumopericardium along with pneumothorax, pneumomediastinum, or subcutaneous emphysema.

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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 difficulty 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.

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 intensive

* Associate Professor of Anesthesia, University of Pennsylvania School of Medicine, and Senior Anesthesiologist, The Children's Hospital of Philadelphia.

† Assistant Professor of Anesthesia, University of Pennsylvania School of Medicine, and Assistant Anesthesiologist, The Children's Hospital of Philadelphia.

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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.

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