Lesson 315: Assessment of the Neonate With Patent Ductus Arteriosus

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Read this article, reflect on the information presented, then go online and complete the lesson post-test and course evaluation before the termination date below. (CME credit is not valid past this date.) You must achieve a score of 80% or better to earn CME credit.

TIME TO COMPLETE ACTIVITY: 2 hours
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Professional Gaps

Treatment of symptomatic patent ductus arteriosus (PDA) is commonly successful with medical therapy. Occasionally, surgical intervention is indicated. Although the general anesthesiologist may only rarely be called on to participate in the care of a newborn with PDA, the condition is common enough that all anesthetic care providers should be well versed in neonatal physiology and management of this congenital abnormality.

Learning Objectives

At the completion of the activity, the reader will be able to:

1. Define patent ductus arteriosus (PDA) and its incidence
2. Recognize the risk factors for PDA
3. Discuss how the PDA closes after birth
4. Recognize the factors that lead to the DA reopening after closure
5. Name the morbidities that are associated with PDA
6. Describe the effect of PDA on systemic organs
7. Identify the clinical presentation of a neonate with PDA
8. Discuss the preoperative management of a neonate with PDA
9. Describe an appropriate intraoperative plan for neonates undergoing PDA ligation
10. List postoperative complication of PDA ligation

Case

A 2-week-old, 0.8 kg neonate was scheduled for ligation of a patent ductus arteriosus (PDA) in the neonatal intensive care unit (NICU). The patient was born at 25 weeks by cesarean delivery with an Apgar score of 5-5-8. His trachea was intubated immediately after delivery; an umbilical arterial
catheter and an umbilical venous catheter (UVC) were placed. He was extubated the next day, but had multiple apneic episodes with desaturations and bradycardia requiring reintubation for respiratory failure. The hospital course was complicated by intestinal perforation treated with an abdominal drain in the NICU and anemia of prematurity for which he received a blood transfusion. Echocardiography showed a moderate to large PDA. Cerebral ultrasonography was negative for intraventricular hemorrhage (IVH). Indomethacin was not given to treat the PDA because of the intestinal pathology. The patient had a radial arterial line, a peripherally inserted central catheter (PICC), and a 24 G peripheral IV. Vital signs were: blood pressure 50/20 mm Hg, pulse 156, SaO2 89% to 96%, respiratory rate 52 to 60, and temperature 36.4°C. The ventilator settings were: SIMV, rate 40, FiO2 30%, positive end-expiratory pressure 6, peak inspiratory pressure (PIP) 15, pressure support 5. Infusions included fentanyl 2 mcg/kg per hour, total parenteral nutrition, and lipids.

Introduction

A PDA is the connection between the lesser curvature of the aortic arch and the pulmonary artery (PA).\(^1\) It is an acyanotic congenital heart disease (CHD) if it is not accompanied with other complex cardiac defects. An isolated persistent PDA occurs in 1 in 2,000 to 5,000 live births and affects approximately 1 in 3 premature infants.\(^2\) The major risk factor is prematurity and the incidence is highest in neonates younger than 28 weeks postconceptual age, weighing less than 1,000 g, or both.\(^3\) Other factors include female gender (2:1), genetic disorders (Down syndrome, Carpenter syndrome, Holt-Oram syndrome, X-linked mutations), and intrauterine exposure to rubella.\(^3\) Incidental PDA could possibly be as high as 1 in 500 children. Although considered an anomaly, the presence of PDA may be required to maintain systemic circulation and oxygenation in infants with single ventricles and other complex CHD.

Fetal Circulation

Fetal circulation is characterized by a high pulmonary vascular resistance (PVR) and a low systemic vascular resistance (SVR). It varies from adult circulation by the existence of several shunts to carry oxygenated blood from the umbilical vein to the systemic circulation. These shunts include the ductus venosus (DV), the foramen ovale (FO), and the ductus arteriosus (DA). Beginning with the placenta, 50% of oxygenated blood passes through the DV to the right atrium (RA).\(^4\) RA configuration helps to direct well-oxygenated blood into the left atrium (LA) via the FO. Blood then travels into the left ventricle (LV), ascending aorta and finally to the brain and upper body. Superior and inferior vena cava blood flow returns to the right ventricle (RV) and into the PA. About 90% of this blood is shunted via the DA to the descending aorta to supply the lower body.\(^4\) As a result, RV output contribution to the fetal systemic circulation is double that of LV.\(^2\) Combined ventricular output to the lungs is only 7% because PVR is high and alveoli are collapsed and filled with amniotic fluid.\(^4\)

Ductal closure or constriction in utero can cause severe pulmonary hypertension and edema because of increased reactivity of the pulmonary vascular muscular layer. The resulting RV failure can lead to devastating conditions such as fetal hydrops and demise.\(^2\)

Postnatal Circulation and PDA Closure

An increase in SVR and a dramatic decrease in PVR lead to a transitional circulation in the first few days of life.\(^4\) These changes and high oxygen tension constrict the DA and reverse its flow, causing a left-to-right shunt. The DA functionally closes within 48 to 72 hours and anatomically by 2 weeks.\(^2\) In infants with
birth weights greater than 1,000 g, 67% of PDA closure occurs by 7 days and 94% by the time of discharge. In infants with birth weights less than 1,000 g, closure happens in 30% by discharge. In infants with birth weights less than 1,000 g, closure happens in 30% by discharge.\textsuperscript{1,2,5} Functional closure is mediated by several factors, including oxygen (which results in contraction of smooth muscle fibers via Ca\textsuperscript{2+}-induced constriction leading to intraluminal ischemic hypoxia), decreased circulating prostaglandins, and decreased ductal luminal pressure.\textsuperscript{2} It has been suggested that activated platelets forming an initial platelet plug have an important role in the primary closure of the DA.\textsuperscript{6} Anatomic closure occurs due to infolding of the endothelium and subintimal disruption and fibrosis.\textsuperscript{2} The resulting tissue persists as the ligamentum arteriosum. The DV and FO also close during the transition to the postnatal circulatory system. The FO may remain patent in 25% of normal infants.

Interference of this closure process can result in pulmonary hypertension and reversal to fetal circulation.\textsuperscript{4} Additional factors that promote and antagonize PDA closure are summarized in Table 1. Some of the factors that lead to persistence of the DA or reopening after closure include low oxygen tension, fluid overload, respiratory distress syndrome, acidosis, lung collapse, and inflammatory mediators as seen in conditions like necrotizing enterocolitis (NEC), severe infections and sepsis.\textsuperscript{7}

### Table 1. Factors Affecting DA Patency

<table>
<thead>
<tr>
<th>Factors That Oppose DA Closure</th>
<th>Factors That Promote DA Closure</th>
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<tbody>
<tr>
<td>Low oxygen tension</td>
<td>Oxygen</td>
</tr>
<tr>
<td>Acidosis</td>
<td>Normal pH</td>
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<tr>
<td>Lung collapse</td>
<td>Normal lung expansion</td>
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<tr>
<td>High PVR</td>
<td>Ductus vascular ischemia</td>
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<tr>
<td>High intraluminal pressure</td>
<td>PGI\textsubscript{2}</td>
</tr>
<tr>
<td>PGE\textsubscript{2}</td>
<td>Inhibition of PGE\textsubscript{2} (indomethacin, ibuprofen)</td>
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<tr>
<td>Nitric oxide</td>
<td>Endothelin</td>
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<tr>
<td>Carbon monoxide</td>
<td>Calcium channel stimulation</td>
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<tr>
<td>Presence of K\textsubscript{Ca\textsuperscript{2+}} channels</td>
<td>Catecholamines</td>
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<tr>
<td>cAMP</td>
<td>Rho kinase</td>
</tr>
<tr>
<td>cGMP</td>
<td></td>
</tr>
<tr>
<td>Thrombocytopenia</td>
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</table>

\textsuperscript{cAMP, cyclic adenosine monophosphate; cGMP, cyclic guanosine monophosphate; DA, ductus arteriosus; PGE\textsubscript{2}, prostaglandin E\textsubscript{2}; PGI\textsubscript{2}, prostacyclin; PVR, pulmonary vascular resistance}

Adapted from reference 2.

### Clinical Picture and Diagnosis

Small incidental PDAs may be completely asymptomatic. A large PDA may not exhibit its full hemodynamic effects in the first few days of life because of relatively high PVR, which results in a small left-to-right shunt. After this initial phase, PVR continues to decrease, resulting in a larger shunt and deterioration of the neonate’s condition by the “steal phenomenon.”\textsuperscript{2,4} The severity of PDA primarily depends on its size, but additional factors include shunt direction, duration of patency, extent of the steal phenomenon, and adequacy of compensatory mechanisms.\textsuperscript{2} The body attempts to adapt with an
increased heart rate, stroke volume, cardiac output, lung lymph flow, maintained contractility, and decreased SVR. Initially, redistribution of blood to vital organs decreases blood flow to the skin, bone, and skeletal muscles. When compensatory mechanisms are exhausted, a “hemodynamically significant” PDA develops, resulting in hypoperfusion to the kidneys, gastrointestinal (GI) tract, brain and heart. Hypoperfusion manifests as systemic hypotension, renal insufficiency and decreased urine output, NEC, IVH, myocardial ischemia, prolonged capillary refills, and metabolic acidosis. PDA is also characterized by increased pulmonary blood flow, pulmonary edema, worsening respiratory mechanics, and a need for increased ventilator support.

Premature infants are more subject to the deleterious effects of PDA due to impaired contractility and relaxation, elevated baseline heart rate, and less organized myocardial fibers.\(^2\) Additionally, immature lungs are less capable of removing circulating prostaglandins.

Classical physical findings of an isolated PDA include acyanosis with normal oxygen saturation, a vigorous peripheral pulse (due to increased pulse pressure), and a continuous machinery-like murmur best heard at the left upper sternal border.\(^1,2\) Worsening respiratory function is caused by transudation of fluid through pulmonary capillaries into interstitial and alveolar spaces due to increased pulmonary venous pressure. Preterm neonates also have less well-developed pulmonary lymphatic systems, which impair efficiency to clear excess interstitial fluid.\(^2\)

However, in mechanically ventilated preterm neonates, PDA evaluation solely by clinical exam is of limited value. Therefore, other modalities are required to diagnose and assess the severity of PDA. Transthoracic echocardiogram (TTE) is the most useful tool.\(^1\) Because the neonate may not demonstrate clinical deterioration in the first few days of life, TTE is done after birth and repeated a few days later to assess patency, direction and magnitude. Other TTE-based diagnostic criteria include LA to aortic root dimension ratio greater than 1:1.4, a ductal diameter greater than 1.4 mm/kg, LV enlargement, and diastolic flow reversal.\(^2\)

Other modalities include electrocardiography that may show signs of LA, LV and RV enlargement, and chest x-rays indicating cardiomegaly and increased lung markings.\(^1\) In older, stable infants, computed tomography angiography, magnetic resonance imaging, cardiac catheterization, or a combination of the 3, may be performed in complex or questionable anatomy, or if coarctation is suspected.\(^1\) Helpful laboratory studies may include B-type natriuretic peptide (BNP) and N-terminal pro-BNP.\(^5,8\) Morbidities associated with PDA are listed in Table 2.

<table>
<thead>
<tr>
<th>Table 2. Morbidities Associated With Persistent Postnatal PDA</th>
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<tbody>
<tr>
<td><strong>General</strong></td>
</tr>
<tr>
<td>Altered postnatal nutrition</td>
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<tr>
<td>Altered postnatal growth</td>
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<tr>
<td><strong>Pulmonary</strong></td>
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<tr>
<td>Bronchopulmonary dysplasia</td>
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<tr>
<td><strong>Gastrointestinal</strong></td>
</tr>
<tr>
<td>NEC</td>
</tr>
<tr>
<td><strong>Neurology</strong></td>
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<tr>
<td>IVH</td>
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<tr>
<td>Decreased cerebral blood flow</td>
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<tr>
<td>Periventricular leukomalacia</td>
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<tr>
<td><strong>Renal</strong></td>
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<tr>
<td>Renal dysfunction</td>
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</table>

IVH, intraventricular hemorrhage; NEC, necrotizing enterocolitis; PDA, patent ductus arteriosus

**Treatment of the Isolated PDA**

**Medical Treatment**

Conservative medical therapy begins with fluid restriction and diuretics (furosemide or thiazides).\(^2,5,8\) If these are not effective, drugs that cause nonselective inhibition of prostaglandin synthesis
(cyclooxygenase-1 and -2 inhibitors) are used.\(^2,^7\) The most commonly used medications are indomethacin and ibuprofen. Recent reports suggest that high-dose acetaminophen may also have a role in increasing PDA closure.\(^5,^8\)

Indomethacin can be started prophylactically within 12 hours of birth or 2 to 3 days postnatal when PDA symptoms start to appear. Prophylactic treatment has been shown to reduce the incidence of severe early pulmonary hemorrhage, severe grades of IVH, the risk for developing symptomatic PDA, and the need for surgical ligation.\(^2\) Indomethacin is contraindicated in the presence of bleeding diathesis, NEC, renal failure or thrombocytopenia. It is effective in only 60% to 70% of preterm infants younger than 28 weeks of gestation. Before that age, the ductus wall is very thin and less responsive to the vasoconstrictive effect of hypoxia. Overall, its effectiveness is inversely related to the postnatal age at the time of treatment. With advancing postnatal age, dilator prostaglandins may not be the main factor in maintaining ductus patency.\(^2\)

Indomethacin significantly decreases cerebral, mesenteric, and renal blood flow, whereas ibuprofen has less of an effect on blood flow to the organs and is associated with a lower incidence of NEC. Ibuprofen, however, does not have the intracranial hemorrhage–sparing effect of indomethacin. Both medications may worsen renal function, inhibit platelet aggregation, and are associated with GI hemorrhage and perforation.\(^2\)

Symptomatic ductus can recur after the initial successful treatment with indomethacin. The rate is highest in the most immature neonates and correlates with the timing and completeness of ductus closure with the initial treatment.\(^2\) A second treatment can be given, but a surgical consult should be sought if it fails. Surgical treatment is also recommended for patients for whom nonsteroidal anti-inflammatory drugs are contraindicated.

**Surgical Treatment**

The chosen technique of surgical closure of PDA depends on the size of the patient, anatomy of the PDA, and institutional practice. Three approaches are described. A left thoracotomy, assuming levocardia is present, is usually reserved for preterm neonates and small infants, patients with a very large and tortuous PDA, or in the presence of aortic coarctation. Video-assisted thoracoscopic surgery is used in larger infants and children. Device closure is done via endovascular techniques in the cardiac catheterization laboratory and is used often for most of the small to moderate PDAs in larger infants and children.\(^1\) PDA closure usually takes place in the NICU to avoid any complications that may arise during transport of fragile, ventilated preterm neonates.

**Preoperative Management**

The neonate should be seen and examined in the NICU and a standard anesthesia history gathered from parents and the hospital chart. Vital signs should be noted to determine the patient’s baseline because significant hypertension or hypotension have been associated with IVH and other adverse outcomes. The anesthetic plan should take into consideration the presence of other congenital anomalies including facial, cerebral, renal, skeletal, or GI tract. Labs and other diagnostic studies, ventilator settings, IV lines, and rates of infusions should be reviewed and discussed with the surgeon and neonatologist. Blood products should be ordered, readily available, and checked before starting the procedure. A team approach and good communication are essential.
In the NICU, ventilation management includes minimizing FiO₂ to keep SpO₂ adjusted to the neonate’s postnatal age (usually between 89% and 95%) and maintaining normocarbia to decrease left-to-right shunting. Low FiO₂ also decreases the risk for the steal phenomenon, incidence of retinopathy of prematurity, and bronchopulmonary dysplasia.¹

**Intraoperative Management**

**Medications**

Total IV anesthesia is used for PDA ligation in the NICU. Emergency medications should be prepared in an appropriate diluted concentration, usually in small syringes according to the patient’s weight. These medications include, but are not limited to, the following: atropine 10 to 20 mcg/kg, calcium chloride 5 to 10 mg/kg or calcium gluconate 20 to 30 mg/kg, and epinephrine 1 to 5 mcg/kg. Other emergency medications like succinylcholine, phenylephrine, or ephedrine can be prepared at the anesthesiologist’s discretion. Inotropes such as dopamine or epinephrine should be immediately available.³ Albumin 5% may be used as a flush fluid.

Anesthesia medications include fentanyl (dosing varies widely among practitioners, but a usual starting dose is 5 to 20 mcg/kg, and the total dose may reach 50 mcg/kg), and vecuronium 0.1 to 0.2 mg/kg or rocuronium 1.2 to 1.5 mg/kg, with additional boluses as needed. Small doses of midazolam may be added. Inhalation anesthetics are not typically used in NICU cases because of myocardial depressant and hypotensive effects and because the regular anesthesia ventilator is not used for these very-low-weight neonates. Prophylactic antibiotics should be given before incision.

**IV Access**

Most of the time, the neonate has an indwelling PICC or UVC for infusions and nutrition. Additional access dedicated to anesthesia medications is preferred to avoid bolusing the total parenteral nutrition line. All IV tubing should be clear of air bubbles to avoid paradoxical air embolism to the coronary, cerebral, and peripheral vessels. A small-volume extension line should be used to obviate the need for large volume flushes after each drug administration. A 4-way stopcock is connected to the end of the extension and secured at the head of the incubator so medications, fluids, or blood can be administered without disrupting the surgery.

**Airway**

Airway management is paramount in these very small preterm neonates. The majority are already intubated for underlying medical conditions, but if no artificial airway is present they may be intubated either awake or postinduction with an appropriately sized uncuffed endotracheal tube (ETT). Alternative plans should be in place if the neonate has associated airway congenital anomalies. Suction and additional sizes of ETTs, laryngoscopes, and oral airways should be readily available.

Minimal leak around the ETT is indicated. If a large leak is present, ventilation can be inadequate after lung retraction, resulting in intraoperative desaturation. The ETT should be changed to a half size larger. Uncuffed ETTs are universally used in preterm infants, whereas cuffed ones can be used in larger neonates.
Monitors

Standard American Society of Anesthesiologists’ monitors are used with an additional pulse oximeter, one placed preductal (right hand) and the other postductal. The latter serves as an indicator of any blood flow interruption if the surgeon incorrectly test-clamps the aorta or the main pulmonary artery. A peripheral or umbilical arterial line is helpful, but not necessary. It allows real-time blood pressure monitoring when the ductus is being ligated as well as acid–base and electrolyte monitoring. Temperature monitoring is particularly important in these small neonates to detect and prevent hypothermia. ETCO₂ monitoring can be technically challenging in preterm neonates, but some NICUs possess appropriate equipment. It is also useful to detect pulmonary artery ligation or compression. Near-infrared spectroscopy allows for continuous measurement of tissue oxygenation. It may be used during and after PDA ligation to reflect perfusion status, and monitor fluctuations in real-time.

Anesthetic Induction and Maintenance

Anesthesia is typically induced with fentanyl and a muscle relaxant (see doses above) and maintained with additional boluses as necessary. If the neonate is not already intubated, an ETT is inserted and secured at an appropriate depth. A clinical technique to determine sufficient depth is by auscultating breath sounds bilaterally, gently advancing the ETT into the right bronchus, withdrawing the tube slowly while listening on the left side until breath sounds are present, and then extracting the ETT an additional few millimeters.

The patient is positioned in the right lateral decubitus by the surgeon while the anesthesiologist holds the head and the ETT. An appropriately sized axillary roll is made from 4x4 sponges or a small towel. Auscultation is repeated to confirm bilateral ventilation after positioning. A precordial stethoscope is beneficial in assuring adequate ventilation intraoperatively.

Mechanical ventilation is typically achieved by a NICU ventilator. The anesthesiologist should be familiar with this device because parameters need to be changed frequently for hypoxemia caused by lung retraction. FiO₂ and PIP are adjusted to keep SpO₂ at baseline. Although high FiO₂ leads to an increase in left-to-right shunt, it is frequently necessary intraoperatively. The anesthesiologist should inform the surgeon of any persistent desaturation so that lung retraction may be relieved until SpO₂ improves. Normocarbia should be maintained at 40 to 50 mm Hg.

Test-clamping of the PDA increases both systolic and diastolic blood pressures with a more pronounced change in diastolic pressures. If the aorta or the main PA is clamped, the arterial line or pulse oximeter waves in the postductal extremity disappear. Inadvertent clamping of one of the PA branches manifests as hypoxemia. The anesthesiologist must alert the surgeon immediately if either of these situations occurs.

Complications

Injury to the aorta or the main PA is fatal unless managed quickly by the surgeon; the anesthesiologist should be ready to transfuse blood immediately. Other complications include vagal-mediated bradycardia that may occur during retraction of major vessels or airways. Cessation of the inciting maneuver usually allows the heart rate to recover. Atropine may be required if the rate persists or cardiac output is negatively affected. The left recurrent laryngeal nerve can be injured during retraction, which may lead to the need for prolonged mechanical ventilation, tube feeding, and hospital stay.
Additional complications are listed in Table 3.

<table>
<thead>
<tr>
<th>Intraoperative</th>
<th>Postoperative</th>
<th>Long-Term Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desaturations</td>
<td>Atelectasis</td>
<td>Rib fusion</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Profound hypotension and increased ventilator support</td>
<td>Chest wall deformity</td>
</tr>
<tr>
<td>Ligation of the wrong vessel (aorta or left pulmonary artery)</td>
<td>Chylothorax</td>
<td>Scoliosis</td>
</tr>
<tr>
<td>Unilateral RLN injury and vocal cord paralysis (as high as 67% in preterm neonates &lt; 1,000 g)</td>
<td>Coarctation of aorta</td>
<td>Compromise of pulmonary function</td>
</tr>
<tr>
<td>Thoracic duct injury</td>
<td>Infection</td>
<td>Recurrence of patency</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Recurrence of patency</td>
<td></td>
</tr>
<tr>
<td>Pulmonary and/or paradoxical air embolism</td>
<td>BPD</td>
<td></td>
</tr>
<tr>
<td>Blood transfusion reactions</td>
<td>Possible impedence of lung growth</td>
<td>Pneumothorax</td>
</tr>
</tbody>
</table>

Table 3. Complications Associated With PDA Ligation

Postoperative Management

At the conclusion of surgery, the anesthesiologist holds the ETT during drape removal. The neonate is positioned supine and bilateral lung auscultation should be performed to ensure appropriate ETT position and lung ventilation. Thoracotomy tubes are not typically left in place. A chest x-ray allows a check of the position of the ETT and excludes pneumothorax.

In the early postoperative period, most neonates experience an improvement in blood pressure and SpO2 with lower FiO2 requirements. In 25% to 30% of cases of neonates with a birth weight less than 1,000 g, this period is followed by one of cardiopulmonary deterioration 6 to 14 hours postoperatively before eventual improvement.2 This phase is characterized by severe hypotension requiring vasopressors and/or inotropes and increased ventilator support. The causes of this decompensation are unknown, but the speculated underlying mechanisms are altered loading conditions, increased SVR, decreased LV output and stroke volume, downregulated cardiovascular adrenergic receptors, relative adrenal insufficiency, and altered vascular tone due to anesthesia. PDA size is one of the strongest predictors for decompensation. Other known risk factors include decreased gestational and postnatal age, and increased ventilator support prior to surgical ligation.

Extubation should occur when the patient recovers from muscle relaxant and fentanyl effects and meets extubation criteria typically on postoperative day 0 or 1. It should not be attempted until resolution of hemodynamic instability, which can take several days.

According to the infective endocarditis guidelines from the American Heart Association, no prophylaxis is required in acyanotic patients with unrepaired or repaired PDA.10

Management of the Case Presented

A large air leak was present around the uncuffed 2.5 ETT, so it was exchanged for a 3 uncuffed tube after induction of general anesthesia by IV fentanyl and rocuronium. The procedure was uneventful except for a few episodes of desaturations, no lower than 85%, during lung retractions, for which PIP and FiO2 were increased accordingly. At the conclusion of surgery, the ETT was exchanged for a 2.5 uncuffed tube and PIP and FiO2 titrated down. The postoperative course was stable and the patient was extubated 4 days later.
Concluding Statement

Surgical management and provision of anesthesia to a neonate may seem frightening to the clinical anesthesiologist who is not a cardiac specialist. However, with a good understanding of the physiology involved and adherence to basic principles of pediatric anesthesia, these infants have a good outcome.

Dr. Elizabeth A.M. Frost, who is the editor of this continuing medical education series, is clinical professor of anesthesiology at The Mount Sinai School of Medicine in New York City. She is the author of Clinical Anesthesia in Neurosurgery (Butterworth-Heinemann, Boston) and numerous articles. Dr. Frost is past president of the Anesthesia History Association and former editor of the journal of the New York State Society of Anesthesiologists, Sphere. She is also editor of the book series based on this CME program, Preanesthetic Assessment, Volumes 1 through 3 (Birkhäuser, Boston) and 4 through 6 (McMahon Publishing, New York City).

REFERENCES

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Post-test

1. The ductus arteriosus is a connection between the _____.
   a. pulmonary artery and aorta
   b. superior vena cava and pulmonary artery
   c. left and right atriums
   d. placenta and inferior vena cava

2. Which of the following is NOT a risk factor for a patent ductus arteriosus (PDA)?
   a. Female gender
   b. Prematurity
   c. Rubeola exposure
   d. Down syndrome

3. In utero, what is the right ventricle’s contribution to the systemic circulation compared with that of the left ventricle?
   a. Varies, depending on weight of the baby
   b. Double
   c. Equal
   d. Half

4. Patency of the ductus arteriosus (DA) is maintained by ____.
   a. high oxygen tension
   b. low pulmonary vascular resistance
   c. low intraluminal pressure
   d. prostaglandin E₂

5. The ductus arteriosus functionally closes between ____ after delivery:
   a. 6 and 10 h
   b. 48 and 72 h
   c. 2 and 4 wk
   d. 1 and 3 mo
6. _____ does NOT result in reopening of previously closed DA.
   a. Oxygen
   b. Sepsis
   c. Necrotizing enterocolitis
   d. Fluid overload

7. Which extremity is used to monitor preductal blood pressure?
   a. Right arm
   b. Left arm
   c. Right leg
   d. Left leg

8. PDA is associated with decreased blood flow to the following organs EXCEPT:
   a. Brain
   b. Lungs
   c. Kidneys
   d. Intestine

9. Which of the following will increase after PDA ligation?
   a. Venous return
   b. Stroke volume
   c. Pulmonary blood flow
   d. Blood pressure

10. _____ is a compensatory mechanism for PDA.
    a. Low heart rate
    b. Low stroke volume
    c. Low systemic vascular resistance
    d. Low cardiac output