Chapter 19

Coarctation of the Aorta

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Case Scenario

A 9-month-old former full-term male presents for repair of nonsyndromic craniosynostosis. He was diagnosed via fetal ultrasound with a discrete coarctation of the aorta and underwent complete surgical repair with end-to-end anastomosis on day 10 of life. Postoperative transthoracic echocardiogram prior to discharge showed no residual gradient. His mother states that he has been feeding and growing well but she did not keep his 6-month cardiology follow up appointment. Current vital signs are heart rate 140 beats/minute, respiratory rate 25 breaths/minute, and SpO_2 97% on room air. The heart beat is regular with no perceptible murmur; the lungs are clear, and the abdomen is soft. He was seen in cardiology clinic today, and right upper extremity blood pressure was 105/70 mm Hg whereas blood pressure in the lower extremity was 70/40 mm Hg.

Transthoracic echocardiography revealed the following:

- *Mild aortic valve regurgitation*
- *Mildly decreased left ventricular systolic function with mild hypertrophy*
- *Turbulent flow below the subclavian artery origin; estimated peak systolic pressure gradient 27 mm Hg*

Key Objectives

- Understand the anatomy and physiology of coarctation of the aorta.
- Understand the natural history of coarctation of the aorta.
- Understand echocardiographic and catheterization data for evaluation of a coarctation gradient.
- Describe preoperative assessment and intraoperative management for this patient.
- Understand major concerns for craniosynostosis repair and how residual coarctation can affect anesthetic management.

Pathophysiology

What is the anatomy and physiology of coarctation of the aorta?

Coarctation of the aorta is defined as any form of narrowing of the aorta, but it is often a discrete narrowing of the aorta distal to the left subclavian artery, just past the point of insertion of the ductus arteriosus. It can occur in isolation or as part of a long segment of aortic arch hypoplasia, which can sometimes involve the transverse aortic arch. It is one of the most common forms of congenital heart disease, representing 4%–7% of cases [1]. If left untreated, aortic coarctation leads to chronic hypertension of the upper extremities, left ventricular (LV) pressure overload, LV hypertrophy, pulmonary edema, and the development of aortic collaterals. (See Figure 19.1A and B.)

What other cardiac defects can be associated with coarctation of the aorta?

The spectrum of clinical manifestations is highly variable and depends upon the presence of other associated lesions as well as the degree of obstruction at the level of the coarctation. In a series of severely symptomatic neonates isolated coarctation was seen in 40%, an associated ventricular septal defect (VSD) in 36%, and the remainder had more complex anomalies including atrioventricular septal defect, transposition of the great arteries, a hypoplastic ventricle, or double outlet right ventricle. Coarctation presenting in a child or an adult can occur either in isolation or with any other left-sided obstructive lesion such as bicuspid aortic valve, aortic valve stenosis, subaortic or supravalvular aortic stenosis. It can also be seen in patients with Turner syndrome or Shone complex. [1–3]. (See Chapter 20.)

https://doi.org/10.1017/9781108657341.020 Published online by Cambridge University Press

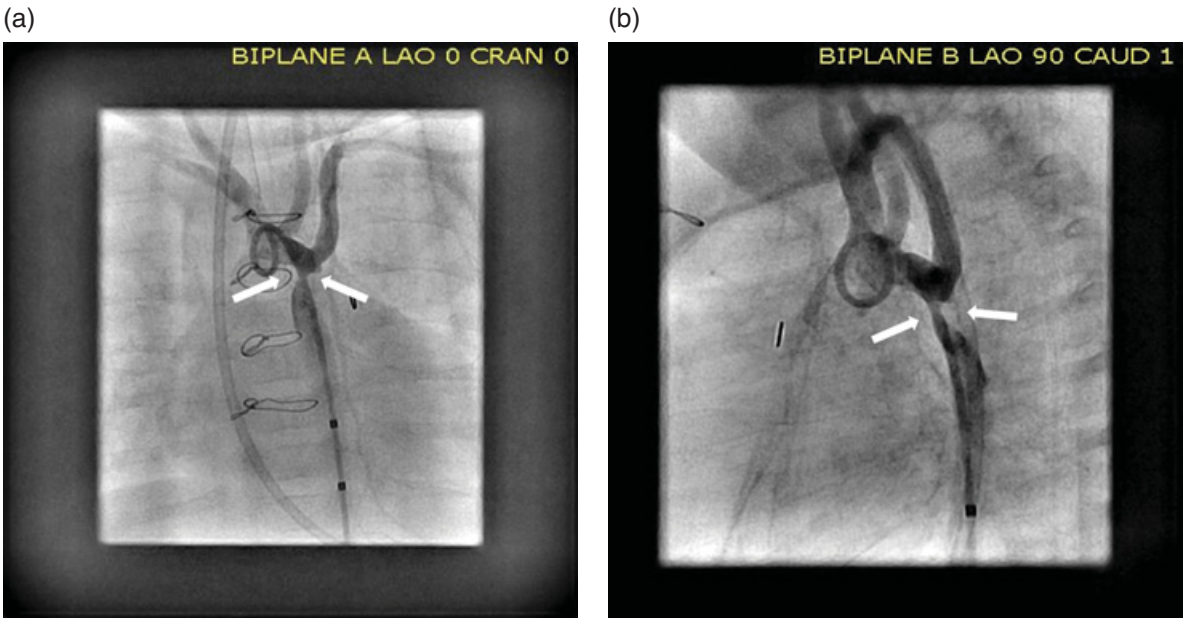

Figure 19.1 (A, B) Aortic coarctation: transverse aortic injection. An angiogram is performed in the AP and lateral projection. The arrows demonstrate the region of the recurrent coarctation at the aortic isthmus, distal to the origin of the left subclavian artery. Courtesy of Russel Hirsch, MD.

How is the diagnosis of coarctation of the aorta made?

The hallmark of coarctation of the aorta is upper extremity hypertension characterized by a differential gradient of at least 20 mm Hg between the systolic blood pressures of the upper and lower extremities. A neonate with coarctation may remain asymptomatic as long as the ductus arteriosus remains open; some neonates present in cardiogenic shock when the ductus closes. A delay in femoral pulses or auscultation of a cardiac murmur or click due to an associated defect may help the practitioner identify the coarctation. Patients may present with symptoms of left-sided heart failure including tachypnea, diaphoresis with feeds, hypotension, tachycardia, and hepatomegaly.

In children and adults aortic coarctation generally presents as hypertension. Murmurs from associated collateral vessels or other defects may be auscultated. Despite the differences in regional blood flow adequate perfusion is generally maintained due to the development of collateral circulation and by autoregulatory vasoconstriction in hypertensive areas and vasodilation in hypotensive areas. Most older patients are asymptomatic, but symptoms related to untreated hypertension such as headache, epistaxis and claudication can occur. If left untreated, the natural history involves the development of accelerated coronary artery disease, stroke, heart failure, and/or aortic dissection. The average survival age for people with untreated aortic coarctation is 35 years of age, with a 75% mortality rate by 46 years of age [4–5].

What echocardiographic and catheterization data can aid in evaluating the severity of coarctation?

Transthoracic echocardiography can establish the diagnosis of coarctation of the aorta by revealing areas of aortic narrowing and turbulent flow seen via color flow Doppler. Using continuous color flow Doppler, the maximum flow velocity and the pressure gradient across the narrowed area can be calculated. The presence of collateral vessels may be detected as well as associated cardiac anomalies. Pressure criteria for diagnosis include a trans-coarctation gradient >20 mm Hg or a gradient <20 mm Hg in the presence of collaterals or reduced cardiac output. Cardiac catheterization is considered the gold standard for diagnosis, with clinically significant aortic coarctation defined as a peak-to-peak pressure gradient difference ≥20 mm Hg. Given the invasive nature of catheterization, however, it is no longer indicated for diagnosis unless warranted for evaluation of associated defects. Although echocardiography is generally adequate for diagnosis in children, cardiac magnetic resonance imaging (MRI) or computed tomography (CT) imaging may be used as adjuncts in the evaluation of older children and adults as they not only clearly delineate

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the area and severity of coarctation but also describe associated collateral vessels [4, 5].

Clinical Pearl

Cardiac catheterization is considered the gold standard for diagnosis, defining clinically significant aortic coarctation as a peak-to-peak pressure gradient difference ≥ 20 mm Hg. Given the invasive nature of catheterization, however, it is no longer indicated for diagnosis unless evaluation of associated defects warrants it.

What is "critical" coarctation? When should coarctation of the aorta be repaired?

Newborns born with critical aortic coarctation (Figure 19.2) are dependent on blood flow through a patent ductus arteriosus (PDA) to provide systemic blood flow to the distal aorta. Because maintaining ductal patency is essential, prostaglandin (PGE_1) infusion is continued until the time of repair. As they lack significant collaterals, neonates and infants are at risk for depressed ventricular function caused by high LV afterload when the PDA closes. If a critical coarctation goes undiagnosed, the patient may present in cardiogenic shock when the PDA closes.

Children and adults meeting the following criteria are considered to have disease significant enough to warrant repair:

- Gradient between upper extremity and lower extremity systolic blood pressure >20 mm Hg or mean Doppler gradient >20 mm Hg
- Upper extremity to lower extremity gradient or mean Doppler gradient >10 mm Hg with either:
  - Decreased LV systolic function or aortic valve regurgitation
  - Collateral flow

Early intervention is thought to decrease the risk of developing long-standing hypertension and its associated sequelae [5].

Clinical Pearl

A gradient between the upper and lower extremity systolic blood pressure or a peak-to-peak Doppler gradient of >20 mm Hg is the hallmark of aortic coarctation. However, a blood pressure gradient may not exist in the setting of collateral flow, aortic valve regurgitation, or decreased left ventricular systolic function.

How is the decision made between a surgical or cardiac catheterization approach to treating aortic coarctation?

The treatment of choice for neonatal coarctation is surgical repair. Compared to balloon angioplasty, surgical repair is

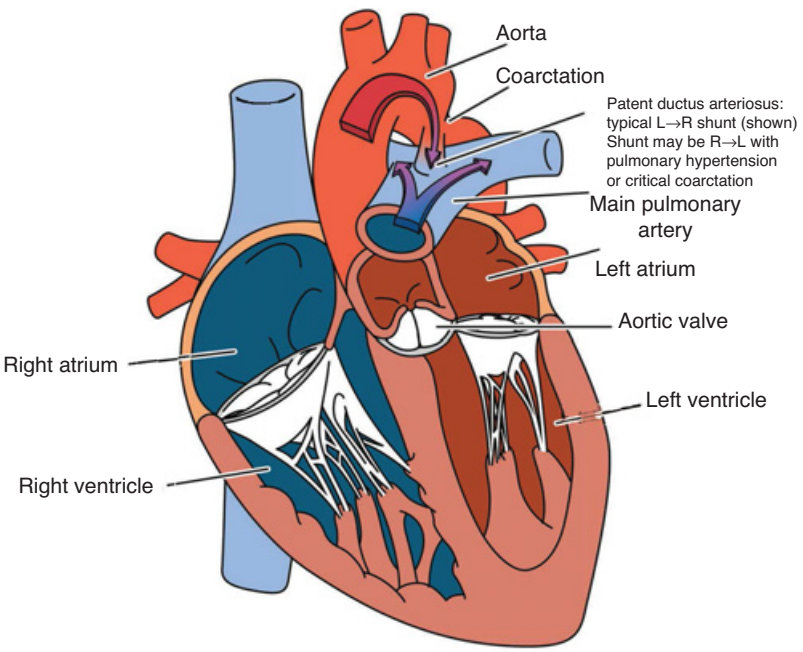

Figure 19.2 Coarctation of the aorta. Drawing by Ryan Moore, MD, and Matt Nelson.

https://doi.org/10.1017/9781108657341.020 Published online by Cambridge University Press

associated with fewer reinterventions, improved aortic arch growth, and a decreased risk of aneurysm formation and need for antihypertensive medication. While balloon angioplasty can be done in infants, it is essentially a palliative procedure in small, critically ill neonates with associated medical conditions that render them poor surgical candidates [1, 7]. Coarctation repair is generally performed via a left thoracotomy unless other associated cardiac defects will be simultaneously repaired on cardiopulmonary bypass via a median sternotomy. A variety of options for surgical repair exist including subclavian flap aortoplasty, aortic arch advancement with end-to-end or end-to-side reconstruction, tube graft interposition, and a resection of the coarctation with end-to-end anastomosis. Of these the latter is most commonly performed [7].

In older children the decision to treat aortic coarctation surgically versus catheterization lab intervention is more complex. Balloon angioplasty was introduced in the 1980s but was associated with a higher rate of recoarctation, aneurysm formation and aortic dissection. In the 1990s endovascular balloon-expandable stents were introduced in place of balloon dilation and have been associated with lower rates of recoarctation and aneurysm formation. An adult-sized stent can be placed in patients weighing >25 kg, and the stent can be redilated multiple times, eventually reaching adult size. This is not possible in neonates and infants, and simple balloon angioplasty remains the main transcatheter option for these patients [1, 3].

In patients >4 months of age but weighing <25 kg the decision to perform balloon angioplasty versus surgery depends upon the anatomy of the lesion and the expertise of the institution. Balloon angioplasty alone is associated with a higher rate of intimal tears and aneurysm formation compared with stent placement; however, in a patient with a discrete coarctation without evidence of arch hypoplasia it may be preferable to perform balloon angioplasty rather than a surgical approach via thoracotomy. Balloon angioplasty is, however, the first choice for treatment of residual or recurrent coarctation after surgical repair. Worldwide the preferred approach continues to be surgery [3, 5].

Does repair of aortic coarctation fix the problem or do residual concerns exist?

Despite successful surgical repair or transcatheter intervention, hypertension often persists. Hypertension is the most common sequela of coarctation, whether repaired or unrepaired. Systemic hypertension may not be consistently identifiable at rest and ambulatory blood pressure monitoring can be useful in identifying patients with a hypertensive response to exercise. Up to 80% of patients with prior coarctation intervention manifest an abnormally elevated upper extremity exercise blood pressure response and peak blood pressure is correlated with increased LV mass [4, 5].

Restenosis of the previously repaired or stented regions also occurs. In a retrospective comparison between balloon angioplasty and surgery for the treatment of neonatal coarctation in patients <4 months, 18% of patients who underwent surgery had recurrence of stenosis requiring follow-up angioplasty. Nevertheless, freedom from any intervention was significantly greater in the surgically treated cohort as opposed to the angioplasty cohort and the rate of complication was significantly lower [5, 6]. Among adult patients successfully treated for coarctation, long-term follow-up with MRI or CTA reveals that approximately 11% of patients may require reintervention for restenosis [4, 5].

In patients with a history of balloon angioplasty, aneurysms often occur in the ascending aorta. Patients who have undergone coarctation repair can develop aneurysms in the descending thoracic aorta or arch. Dissection can occur, presumably more likely in the setting of uncontrolled hypertension [5]. Multiple studies have demonstrated an increased frequency of intracranial aneurysm in adults with coarctation of the aorta as well. Approximately 10% of adult patients with coarctation of the aorta have intracranial aneurysms identified by MR angiography or CT angiography. Whether this is a byproduct of the coarctation itself or other factors such as hypertension is unclear [5].

What is evident is that lifelong follow up is needed not only for the prompt recognition and treatment of hypertension, cardiovascular disease, and postprocedural complications, but also for recurring or residual coarctation. Even with an excellent surgical repair or catheter-based intervention, hypertension remains common and predisposes the patient to myocardial infarction, stroke, and heart failure.

What is craniosynostosis and when should it be repaired?

Craniosynostosis is a condition in which one or more of the sutures in an infant's skull fuse prematurely and alter the growth pattern of the skull. It can occur as an isolated sporadic event or as part of a larger syndrome such as Crouzon or Apert syndromes which also involve hypoplasia of the mid-face, skull base and limb abnormalities. These associated problems can lead to issues such as elevated intracranial pressure (ICP), airway obstruction, and feeding difficulties as well as abnormal behavioral and psychological development. The most significant surgical risks are blood loss and venous air embolism. The repair is not merely cosmetic and is performed at an early age to allow normal brain growth and cognitive development [7, 8].

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Anesthetic Implications

What preoperative evaluation is appropriate for this patient?

Preoperative evaluation should include a thorough assessment of preexisting medical conditions, medications, allergies, previous anesthetic or airway issues, relevant family history, and physical examination. Given this patient's prior coarctation repair and the existing noninvasive blood pressure (NIBP) gradient between upper and lower extremities, it is essential that during his cardiology visit an electrocardiogram, chest radiograph and transthoracic echocardiogram be obtained and reviewed.

With regard to the patient's craniosynostosis it is appropriate to look for clinical signs of increased ICP such as visual difficulties, nausea, vomiting, or somnolence. Although problems with increased ICP are unusual in infants with craniosynostosis, it can occur in older children or when fusion of multiple sutures exists. [7]

Preoperative laboratory evaluation should include a complete blood count and coagulation studies as well as a type and cross for packed red blood cells. Availability of blood should be confirmed prior to the start of the procedure and blood should be present in the operating room environment.

Does the restenosis of the coarctation need to be addressed prior to the craniotomy?

Given the patient's mildly diminished LV function and gradient between upper and lower extremity blood pressures, the results of the cardiology visit may prompt communication between physicians from neurosurgery, cardiology, and cardiac surgery regarding the appropriate sequence of interventions for this patient as he may need to undergo balloon angioplasty of the recoarctation in the cardiac catheterization laboratory prior to craniosynostosis repair. The existing upper extremity hypertension and relative hypotension below the level of the coarctation puts the patient at greater risk for perioperative complications during the craniosynostosis repair.

For a surgery involving significant blood loss, techniques such as permissive hypotension and acute normovolemic hemodilution have been employed in some centers. Neither has been found to be helpful in the setting of craniosynostosis repair mostly due to the small size of the patient, the increased risk of hemodynamic instability and the increased risk of venous air embolism. While neither permissive hypotension nor acute normovolemic hemodilution has gained widespread acceptance, hypertension should be avoided in the face of ongoing blood loss. In the presence of an aortic coarctation it will be important to maintain normal blood pressures above the coarctation and to avoid hypotension which would place the patient at risk for hypoperfusion of the kidneys, gastrointestinal tract, and spinal cord.

The general hemodynamic goals for craniosynostosis repair involve mild hypotension and euvolemia, attempting to decrease blood loss while maintaining adequate perfusion and minimal risk of venous air embolism. In this patient, however, the presence of the unrepaired recoarctation places the patient at risk for hypoperfusion to organs supplied by arteries distal to the recoarctation, thus an anesthetic strategy allowing mild hypotension may not be in the patient's best interest.

Clinical Pearl

The anesthetic goals for craniosynostosis repair include mild hypotension and euvolemia. The aim is to decrease blood loss while maintaining adequate perfusion and minimal risk of venous air embolism. The hemodynamic effects of the unrepaired recoarctation already put the patient at risk for hypoperfusion of the gastrointestinal tract, kidneys, and spinal cord and thus the use of deliberate mild hypotension may not be in the patient's best interest.

Are there special concerns that need to be communicated to the parents in the preoperative area?

On the day of surgery it should be confirmed that the patient is appropriately nil per os (NPO) and without recent signs of upper or lower respiratory infections or fevers. The patient's level of stranger anxiety should be assessed and if helpful an oral premedication such as midazolam may be administered. As with any preoperative evaluation, adequate time should be spent addressing any questions or concerns the parents have. Parents should be fully informed of the likelihood of transfusion. Although the child has already been through a major surgery and follow up visit to the catheterization lab, most likely without transfusion, there is a very high probability that during craniosynostosis repair he will require transfusion. The need for transfusion and potential development of coagulopathy may affect the ability to extubate the child at the end of the case.

What are the most significant concerns during a craniosynostosis repair?

The surgical approach to craniosynostosis repair depends not only upon which sutures are fused but also institutional

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experience. All approaches involve elevation of the vascular periosteum. Once the osteotomy is performed, blood loss is usually slow and continuous. Dural sinuses are another potential source of blood loss. Bleeding from the sinuses can be dramatic and require an immediate response. Given their small size at time of repair it is not unusual for a patient to lose 25%–50% of their blood volume during repair, sometimes more [7, 8].

The patient's small size increases the impact of blood loss as well as the metabolic effects of transfusion. Cardiovascular function in infants is more sensitive to calcium homeostasis as well as acidosis. With a smaller and relatively less compliant heart than an older child or adult an infant has less cardiovascular reserve so the relative impact of a potassium load from older blood that has been in storage has greater consequence.

The most significant intraoperative concerns for an infant undergoing craniosynostosis repair include blood loss, coagulopathy, hemodynamic instability, and venous air embolism. Other important issues in small children include glucose regulation, acidosis, calcium homeostasis, hemodilution, and temperature regulation.

Clinical Pearl

During surgical repair of craniosynostosis the most important risks are blood loss and venous air embolism. It is imperative to plan for significant blood loss and to have packed red blood cells readily available in the operating room environment.

What drugs could be utilized for induction and maintenance of anesthesia?

An inhalation induction of anesthesia followed by placement of a peripheral intravenous (IV) line is generally well tolerated. A balanced anesthetic including volatile agents, opioids and muscle relaxant can be used throughout the case. Neuromonitoring is not employed for these cases and the use of total intravenous anesthesia is usually unnecessary. The goal is a readily titratable anesthetic that can be altered to accommodate varying levels of surgical stimulation and changing hemodynamic conditions.

What monitoring would be appropriate?

Monitoring should include American Society of Anesthesiologists standard monitoring along with placement of an arterial line. Although the coarctation has been repaired the preference for a right radial arterial line over a left-sided arterial line still exists. An NIBP cuff can be placed on the left arm or a lower extremity as well. Other indicators of adequate perfusion such as upper and lower extremity pulse oximeters would also be useful. Central venous pressure monitoring is not routine, but adequate access should be ensured for volume resuscitation, necessitating two peripheral IV lines at a minimum.

Although venous air embolism is a reported complication of craniosynostosis repair, the incidence of hemodynamically significant air embolism is rare. The patient should be carefully positioned, and table controls checked ahead of time so that the table can be put into Trendelenburg position quickly if necessary. Emergency drugs such as epinephrine diluted to 10 mcg/mL should be readily available. Sterile saline should be at hand for the surgeon to flood the field as necessary. Constant monitoring of end-tidal CO_2 is necessary and changes in end-tidal CO_2 or other signs of hemodynamic instability should be communicated to the surgeon immediately.

Although placement of a central venous line is not necessary for the repair, the evaluation of volume status should be a constant process via other mechanisms. Pulse pressure variation noted via the pulse oximeter and/or arterial line, maintenance of appropriate urine output and the development of a base deficit or lactic acidosis should be monitored and adequate volume resuscitation maintained. Regular arterial blood gases should be checked during the portion of the procedure involving significant blood loss. The hemoglobin and hematocrit may be deceptive in an underresuscitated child. It is not until one is already behind with volume resuscitation that a base deficit or lactic acidosis is observed. Although it is not possible to monitor cerebral near-infrared spectroscopy during a craniosynostosis repair, it may be helpful to place a sensor over the flank in an effort to optimize monitoring of lower extremity perfusion.

How does residual coarctation affect induction and management strategies?

If a blood pressure gradient exists between upper and lower extremities, it is important to know the patient's underlying baseline pressure both above and below the coarctation with the goal being to "keep him where he lives." Under such circumstances the surgery would be performed at a higher cerebral pressure than might be favored by the surgeon, but the potential need for additional blood transfusion and ideal surgical conditions is offset by the necessity of maintaining adequate kidney, spine, and gastrointestinal perfusion. Under such circumstances, regular monitoring of arterial blood gases becomes even more critical.

https://doi.org/10.1017/9781108657341.020 Published online by Cambridge University Press

Clinical Pearl

When a residual gradient exists, it is best to get a sense of the patient's baseline pressures and "keep him where he lives." Continue to monitor upper and lower extremity pressures throughout the case and pay close attention to other indicators of perfusion such as urine output and development of lactic acidosis.

What inotropic or vasoactive infusions might be helpful?

Antifibrinolytics such as tranexamic acid have been shown to reduce blood loss and the need for transfusion in some studies. The loading dose of tranexamic acid varies between 10 and 100 mg/kg, followed by an infusion of 5–10 mg/kg/hour for the duration of the surgery. Although the use of a nicardipine infusion for potential control of residual upper extremity hypertension could be considered, an appropriate level of hemodynamic control can generally be achieved with a combination of inhaled anesthetic and narcotic [8].

Are there transfusion issues that are unique to infants?

Transfusion guidelines are similar in children and adults. Unless a child suffers from complex cyanotic heart disease, is premature, or requires invasive respiratory or hemodynamic support, transfusion for a hemoglobin level >10 g/dL is usually unnecessary. Transfusion for a hemoglobin level of 6 g/dL or below is indicated. In the setting of ongoing surgical blood loss, regular arterial blood gases along with other sources of clinical information help to guide transfusion. In small children it is helpful to calculate the estimated maximum allowable blood loss to utilize as a guideline along with regular arterial blood gases.

$$\textbf{Allowable blood loss} = [\text{EBV} \times (H_i - H_f)]/H_i,$$

where
H_i = initial hemoglobin and H_f = final hemoglobin
EBV = estimated blood volume.

All blood used in young children should be leukocyte-depleted, cytomegalovirus negative, and washed or stored less than 2–3 weeks. It should be warmed and filtered prior to administration to the patient.

If the apparent blood loss exceeds half of the patient's EBV, a new set of baseline labs should be sent *STAT* and the provider should consider ordering platelets or fresh frozen plasma (FFP). Although the need for red blood cell transfusion is common, the need for transfusion of platelets, FFP or cryoprecipitate occurs less frequently. Nevertheless, platelet transfusion is appropriate if the blood loss approaches the patient's blood volume or if the platelet count is known to be <50,000. The usual therapeutic dose of platelets in children under 10 kg is 10 mL/kg. When INR is >1.5 times normal, effective hemostasis can generally be achieved with a minimum of 30% of the normal level of factor concentration. Administration of 10 mL/kg FFP is generally appropriate in small children. If fibrinogen levels drop below 80 mg/dL, one unit of cryoprecipitate per 10 kg of body weight or 0.1 unit/kg can be administered and should correct fibrinogen concentration by approximately 50 mg/dL [7].

Where should the patient recover after surgery and what concerns might the managing team have?

Patients are generally extubated after craniosynostosis repair. Factors that can delay extubation include large volume fluid shifts due to transfusion, facial swelling due to patient positioning or underlying airway obstruction concerns due to associated airway defects such as midface hypoplasia. Even if extubated, the patient should still be transferred to a unit with a high level of care. Postoperative coagulopathy is an ongoing concern. Children with a coarctation are at risk for hypertension even after repair, which may complicate postoperative hemostasis. Dramatically elevated levels of norepinephrine have been noted following coarctation repair and are thought to be due to baroreceptor adaptation. The use of nonsteroidal antiinflammatory drugs should be avoided. Attention should be paid to postoperative electrolyte disturbances; hyponatremia can result from intraoperative crystalloid infusions as well as syndrome of inappropriate antidiuretic hormone [4, 7].

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Suggested Reading

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