Chapter

16

Subvalvular Aortic Stenosis

Rahul G. Baijal

Case Scenario

A 5-year-old male, who was a restrained rear-seat passenger, presents to the emergency department following a T-bone motor vehicle accident. The child's medical history is significant for repaired subvalvular aortic stenosis at age 3. The child is able to keep up with his older siblings and classmates without difficulty.

Primary assessment reveals a nonobstructive respiratory rate of 12 breaths/minute, SpO_2 99% on room air, heart rate of 140 beats/minute, and a Glasgow Coma Scale score of 14. Secondary assessment reveals a distended, tender abdomen along with bruising and tenderness over the right femur. Radiographic assessment confirms a right femur fracture while computed tomography of the abdomen reveals potential intraperitoneal air. The child is scheduled for an emergent exploratory laparotomy along with placement of an intramedullary nail in the right femur. Transthoracic echocardiography 6 months previously showed the following:

- *Moderate to severe subvalvular aortic stenosis with a peak velocity of 3.9 m/s*
- *40 mm Hg mean gradient across the aortic valve*
- *Normal ejection fraction (55%) with qualitatively normal diastolic function*
- *Moderate left ventricular hypertrophy*

Key Objectives

- Understand the pathophysiology of subvalvular aortic stenosis.
- Describe the natural progression of subvalvular aortic stenosis.
- Describe appropriate intervention(s) for subvalvular aortic stenosis.
- Understand echocardiographic findings in subvalvular aortic stenosis.
- Construct a perioperative plan for the patient with subvalvular aortic stenosis.

Pathophysiology

What is the epidemiology of subvalvular aortic stenosis?

Fixed subvalvular aortic stenosis (AS) is the second most common type of left ventricular outflow tract obstruction (LVOTO) following valvular AS, accounting for approximately 15% of all cases. Subvalvular AS has a male predominance with a ratio of approximately 1.5–2.5:1.

What is the pathology of subvalvular AS?

- ***Membranous subvalvular AS*** is the most common type, accounting for approximately 70%–80% of all cases of subvalvular pathology. (See Figure 16.1.) A thin, fibrous, circumferential membrane, often with attachments to the anterior mitral valve leaflet, is typically located 1–2 mm inferior to the aortic valve.

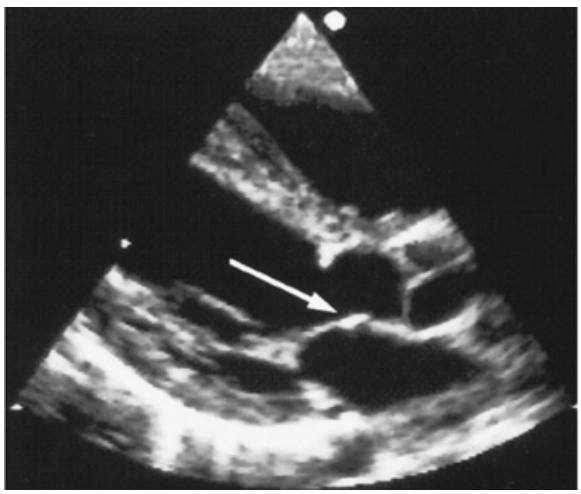

Figure 16.1 Parasternal long axis view of subvalvular aortic stenosis with membrane.

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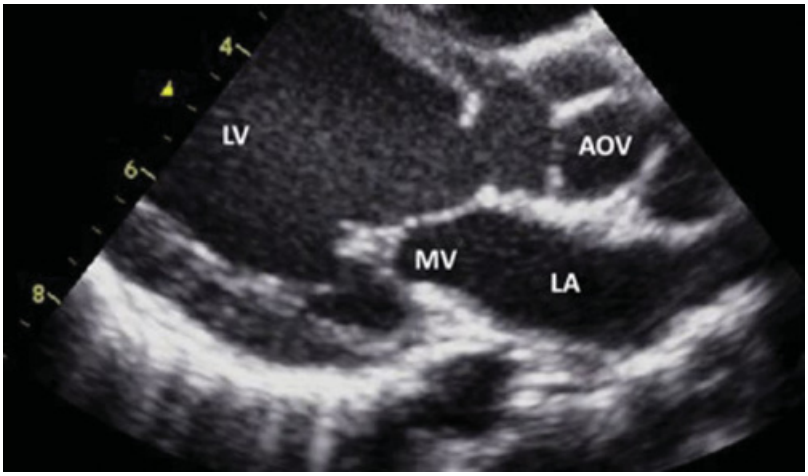

Figure 16.2 Parasternal long axis view of subvalvular aortic stenosis with fibromuscular ridge.

- A ***fibromuscular ridge***, located slightly more inferior to the aortic valve than the membrane, is the second most common type of subvalvular AS. (See Figure 16.2.)

Both types are acquired conditions. Turbulent flow and shear stresses within the left ventricular outflow tract (LVOT), producing endothelial damage, cellular proliferation, and collagen deposition, are theorized as the etiologies for the formation of both membranous and fibromuscular ridge subvalvular AS. The turbulent flow and the increased shear stresses are postulated to result from an abnormally shaped LVOT, where the angle between the long axis of the left ventricle (LV) and aorta is more acute than normal. The endothelial damage, cellular proliferation, and collagen deposition result in LVOTO, producing further turbulent flow and abnormal shear stresses. (See Figure 16.3.)

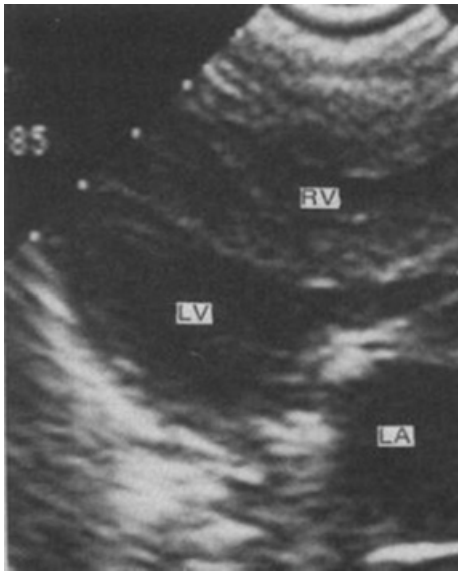

Figure 16.3 **Subvalvular aortic stenosis: Left ventricular injection**. An angiogram is performed in the left ventricle in the AP projection. The subaortic stenosis immediately below the valve is noted (arrows). Courtesy of Russel Hirsch, MD.

- ***Tunnel-type subvalvular AS***, extending for several centimeters below the aortic valve, is the most severe type of subaortic stenosis. (See Figure 16.4.) Tunnel-type obstruction is also acquired, typically following previous repair of congenital heart defects, including double outlet right ventricle, interrupted aortic arch, and Shone's complex. (See Chapter 20.) Turbulent flow and shear stresses caused by residual postoperative obstruction are again postulated as the etiology for tunnel-type obstruction.
- ***Other etiologies of subvalvular AS include*:**
  - Abnormal attachment of the anterior mitral valve leaflet or accessory atrioventricular valve tissue, as seen in cleft mitral valve or complete atrioventricular septal defect
  - Conal septal projection into the LVOT with a posterior malaligned ventricular septal defect (VSD)
  - Asymmetric septal hypertrophic cardiomyopathy

Aortic insufficiency (AI) develops in approximately 70% of children with subvalvular AS. (See Figure 16.5.) Aortic insufficiency develops secondary to long-standing damage to the aortic valve from the increased turbulent

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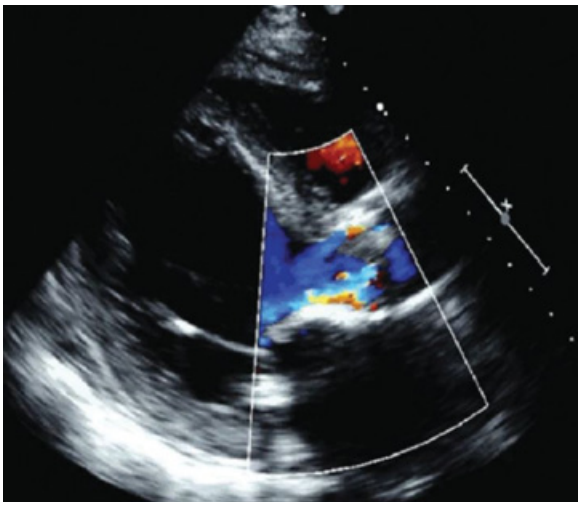

Figure 16.4 Parasternal long axis view of subvalvular aortic stenosis tunnel type.

flow and shear stresses. The thickening and distortion of the valve leaflets result in failure of leaflet coaptation or prolapse, producing AI. The peak echocardiographic Doppler gradient is the strongest predictor for AI in children with subvalvular AS.

Clinical Pearl

The peak echocardiographic Doppler gradient is the strongest predictor for AI in children with subvalvular AS.

What lesions may be associated with subvalvular AS?

See Table 16.1 for lesions associated with subvalvular AS.

What are the physiologic considerations with subvalvular AS?

Left ventricular outflow tract obstruction is the primary physiologic derangement in subvalvular AS, increasing LV systolic pressure and wall stress, which is directly proportional to LV pressure and indirectly proportional to LV wall thickness. Left ventricular wall hypertrophy is a compensatory mechanism that increases LV wall thickness to maintain constant LV wall stress in the setting of increasing LV systolic pressure.

$$T = Pr/2h,$$

where T = LV wall stress, P = LV systolic pressure, r = ventricular radius, and h = LV wall thickness.

Table 16.1 Lesions Associated with Subvalvular Aortic Stenosis

Lesion	%
Bicuspid aortic valve	40
Valvular aortic stenosis	28
Ventricular septal defect	24
Coarctation of the aorta	12
Patent ductus arteriosus	12
Atrial septal defect	4

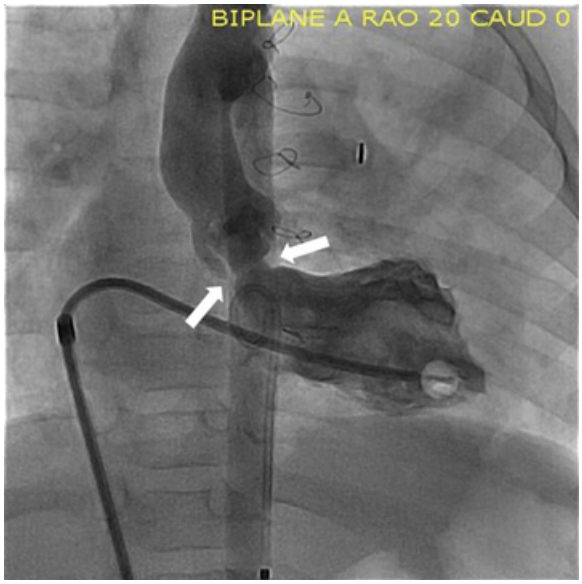

Figure 16.5 Parasternal long axis view of aortic insufficiency.

Left ventricular wall stress normalization initially maintains normal systolic and diastolic function despite an increased impedance to ejection. However, long-standing pressure overload to the LV results in ventricular remodeling with both diastolic and systolic dysfunction and subsequent clinical heart failure.

Subendocardial ischemia may also result from high intracardiac compressive forces that limit systolic coronary artery flow. Oxygen delivery to the subendomyocardium occurs during diastole, driven by the gradient between aortic diastolic pressure and left ventricular end-diastolic pressure (LVEDP). Increased myocardial oxygen demand is typically compensated for by increased oxygen delivery through coronary vasodilation. Further subendocardial coronary vasodilation in children with subvalvular AS during periods of stress is limited by the high intracardiac compressive forces. Subsequently, subendocardial oxygen delivery is determined by the duration of diastole and the

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gradient between aortic diastolic pressure and LVEDP. This diastolic gradient is additionally reduced from increased LVEDP secondary to decreased LV compliance due to compensatory hypertrophy. Hemodynamic data from a cohort of 80 children with AS demonstrated that myocardial oxygen delivery is determined by aortic valve area, diastolic function, and heart rate. All patients with severe AS with a heart rate <100 beats/minute demonstrated adequate oxygen delivery.

Clinical Pearl

Subendocardial oxygen delivery is determined by the duration of diastole and the gradient between aortic diastolic pressure and LVEDP. This diastolic gradient is additionally reduced from increased LVEDP secondary to decreased LV compliance due to compensatory hypertrophy. Tachycardia, particularly a heart rate >100 beats/minute, may be poorly tolerated in children with severe subvalvular AS.

What is the natural history of subvalvular AS?

Subvalvular AS is an acquired progressive disease, producing symptoms late in the disease course. Mild or moderate AS is typically asymptomatic. Symptoms with moderate to severe AS include dyspnea on exertion, angina, presyncope, syncope, and fatigue. Although the rate of disease progression may be variable, an increased gradient at diagnosis, attachment of the subaortic membrane to the mitral valve, aortic valve thickening, and decreased distance between the aortic valve and the subaortic membrane or fibromuscular ridge are risk factors for disease progression. Additionally, an increased mean gradient at diagnosis and increased time since diagnosis are risk factors for progression of AI.

What physical examination findings might be expected in subvalvular AS?

In children with moderate to severe subvalvular AS, the LV impulse will be displaced further laterally, and a systolic thrill may be appreciated over the base of the heart. The first heart sound is normal, and the second heart sound may be narrow, secondary to delayed aortic valve closure.

- A physiologically split S2 is the most reliable physical examination finding to *exclude* severe stenosis.
- S3 and S4 gallops are also present in children with LVOTO but do not correlate with the degree of stenosis in children <12 years of age. An S4 gallop in children >12 years of age may indicate severe stenosis and left ventricular diastolic dysfunction.
- A harsh crescendo–decrescendo systolic ejection murmur, loudest at the left mid-sternal border and radiating to the carotid arteries, is heard in subvalvular AS. The intensity of the murmur correlates with the severity of the obstruction. An early diastolic decrescendo murmur at the left lower sternal border may also be heard if AI is present.

Clinical Pearl

*A physiologically split S2 is the most reliable physical examination finding to **exclude** severe stenosis. The intensity of the harsh crescendo–decrescendo systolic ejection murmur correlates with the severity of the obstruction.*

Is electrocardiography a useful diagnostic tool?

Electrocardiographic findings are neither highly sensitive nor specific for severe stenosis. Only 74% and 70% of children with moderate or severe AS display LVH and strain, respectively, on ECG, whereas 24% and 10% of children with mild AS display LVH and strain, respectively.

What echocardiographic parameters are important in subvalvular AS?

Echocardiography is the only class I recommendation by the American Heart Association/American College of Cardiology (AHA/ACC) for the diagnostic evaluation of suspected AS and the monitoring of disease progression. An echocardiogram may determine not only the location and severity of the obstruction, but also the LV response to the obstruction, including hypertrophy and systolic and diastolic function. Doppler interrogation of the aortic valve accurately estimates disease severity and predicts the need for intervention in children.

The mean echocardiographic Doppler velocity more closely approximates the peak-to-peak pressure gradient obtained in the cardiac catheterization laboratory as an accurate reflection of disease severity, whereas the peak instantaneous echocardiogram Doppler velocity may overestimate the catheter-derived gradient.

Doppler interrogation may underestimate disease severity in the setting of severe myocardial dysfunction, multiple levels of obstruction, or the presence of a pop-off (e.g., an atrial or ventricular septal defect).

Preferred echocardiographic views are as follows:

- ***Parasternal long axis***: Aortic valve annulus and aortic root dimensions
- ***Parasternal short axis***: Aortic valve assessment

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Aortic valve gradient may be estimated by the Doppler velocity through the modified Bernoulli equation:

$$\Delta P = 4\, v^2,$$

where ΔP is the calculated gradient and v is either the interrogated mean or peak velocity.

Severe aortic stenosis is defined as a peak velocity >4.0 m/s across the aortic valve or a mean gradient of 40 mm Hg across the valve. An aortic valve area ≤ 1.0 cm^2 or an indexed valve area of ≤0.6 cm^2/m^2 in the setting of severe myocardial dysfunction is consistent with severe stenosis.

Moderate aortic stenosis is defined as a peak velocity of 3.0–3.9 m/s or a mean gradient between 20 and 39 mm Hg.

Mild stenosis is defined as a peak velocity between 2.0 and 2.9 m/s or a mean gradient ≤20 mm Hg.

Clinical Pearl

Doppler interrogation of the aortic valve accurately estimates disease severity and predicts the need for intervention in children, but Doppler interrogation may underestimate disease severity in the setting of severe myocardial dysfunction, multiple levels of obstruction, or the presence of a pop-off (e.g., an atrial or ventricular septal defect).

How is subvalvular AS medically managed?

There is a limited role for medical management of LVOTO. Guidelines from the ACC/AHA recommend interval transthoracic echocardiography (TTE) to assess disease progression. A TTE is recommended every 6 months to 1 year in children with asymptomatic severe stenosis, every 1–2 years in asymptomatic moderate stenosis, and every 3–5 years in asymptomatic mild stenosis. Symptomatic patients require urgent evaluation. Asymptomatic children with mild stenosis may participate in all sports without restriction whereas all competitive sports should be avoided in patients with severe stenosis. Children with moderate stenosis without moderate or greater LVH on echocardiography, a normal stress test, and no strain on a baseline ECG may participate in sports with a low static component and low to moderate dynamic component.

Is there a role for interventional cardiac catheterization in subvalvular AS?

There is limited evidence that balloon aortic valvuloplasty is effective for subvalvular AS.

What is the surgical management in subvalvular AS?

The surgical approach to subvalvular AS repair depends on the type of obstruction. Although resection of the thin, fibrous membrane or fibromuscular ridge is a relatively straightforward procedure, indications for the procedure are unclear. Since the stenosis is progressive, some studies suggest early repair with a peak gradient as low as 20 mm Hg. Reoperation rates range between 0.6% and 1.8% per year, with risk factors for reoperation including an increased peak gradient at the time of diagnosis >40 mm Hg, early age at diagnosis, distance of <5 mm between the aortic valve and the membrane or fibromuscular ridge, and immediate postoperative LVOT peak pressure gradient >10 mm Hg. (See Figure 16.5.)

Tunnel type obstruction requires either a Konno or Ross–Konno operation (see Chapter 15) with resection of the conal septum, or revision of previously placed LV-to-aorta baffle. Reoperation rates in tunnel-type obstruction range from 15% to 50%. Overall recurrence rate is 20% and higher in children <10 years of age. New-onset AI is an indication for surgical repair. Some centers prefer to defer surgery in children with gradients <40–50 mm Hg without AI, or in children <5–10 years of age, given the high recurrence rate in patients <10 years of age.

Anesthetic Implications

What preoperative workup is appropriate for a child with subvalvular AS presenting for emergent surgery?

A history and physical examination may help elucidate the severity of the LVOTO. The patient/family should be interrogated for any symptoms of dyspnea on exertion, angina, presyncope, syncope, or fatigue. On physical examination, the quality and intensity of the murmur and any new findings are assessed and compared to previous examinations if possible. Previous echocardiographic data should be reviewed for Doppler interrogation of the aortic valve and the degree of stenosis, along with the presence of AI, LVH, and degree of myocardial systolic and diastolic dysfunction. Symptomatic patients require urgent reevaluation with an echocardiogram preoperatively. A limited point of care ultrasound via TTE allows rapid assessment of the degree of subvalvular AS and myocardial dysfunction if there is limited time for a more thorough examination before an emergent procedure.

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Clinical Pearl

Prior to emergent surgery, symptomatic patients with subvalvular AS require urgent reevaluation with an echocardiogram. A limited point of care ultrasound via TTE allows rapid assessment of the degree of subvalvular AS and myocardial dysfunction if there is limited time for a more thorough examination before an emergent procedure.

What are the fasting considerations for patients with subvalvular AS?

The American Society of Anesthesiologists (ASA) nil per os (NPO) guidelines are 8 hours for heavy meals, 6 hours for light meals, 6 hours for formula, and 2 hours for clear liquids for elective procedures. Excessive fasting should be limited in children with LVOTO, including subvalvular AS. Clear liquids should be administered liberally until 2 hours prior to surgery in children presenting for elective procedures. Extended periods without fluid intake or maintenance intravenous (IV) fluid hydration can significantly decrease preload or afterload, increasing the risk of hypotension resulting in decreased aortic diastolic blood pressure and subsequent myocardial ischemia. These NPO guidelines, however, do not apply for children presenting for emergent procedures. Children presenting for emergent procedures may be hypovolemic secondary to previous fasting or fluid or hemorrhagic losses. These losses may need to be replaced with fluid boluses or blood transfusion therapy preoperatively prior to the induction of anesthesia.

Clinical Pearl

Extended periods without fluid intake or maintenance intravenous fluid hydration can significantly decrease preload or afterload, increasing the risk of hypotension and resulting in decreased aortic diastolic blood pressure and subsequent myocardial ischemia. Excessive fasting should be limited in children with LVOTO, including subvalvular AS.

What are the primary anesthetic considerations in subvalvular AS?

Perioperative goals should entail reducing the physiologic trespass of surgery and anesthesia. Concentric LVH due to chronic pressure overload, a preload-dependent LV with poor myocardial compliance, and poor coronary artery reserve secondary to fixed LVOTO impede appropriate hemodynamic responses during surgery and anesthesia. Extended periods without fluid intake or maintenance IV fluid hydration can significantly decrease preload, increasing the risk of hypotension with decreased aortic diastolic blood pressure and subsequent myocardial ischemia.

Anesthetic goals include the following:

- ***Maintenance of adequate preload and systemic vascular resistance (SVR)***, or afterload, to ensure an appropriate aortic diastolic pressure for an adequate pressure gradient during diastole for coronary blood flow, and maintenance of myocardial contractility with a normal-to-low heart rate.
- ***Avoidance of decreases in SVR*** due to anesthetic agents as exaggerated hypotension may be seen secondary to a compromised compensatory increase in cardiac output from the fixed outflow tract obstruction.
- ***Avoidance of increased contractility with associated tachycardia***: Inadequate anesthetic depth may cause subendocardial ischemia from already maximally dilated subendocardial coronary arteries due to high intracardiac compressive forces. Tachycardia also prevents adequate early-diastolic filling of the LV that already has impaired relaxation necessary for early-diastolic filling, further reducing cardiac output.

Clinical Pearl

Anesthetic goals should include maintaining adequate preload and SVR, or afterload, to ensure an appropriate aortic diastolic pressure for an adequate pressure gradient during diastole for coronary blood flow, and maintenance of myocardial contractility with a normal-to-low heart rate.

Is premedication appropriate in this child?

This child is tachycardic: possible etiologies include pain, anxiety, hypovolemia, or a combination thereof. Premedication with IV, intranasal or oral agents, including midazolam, ketamine, opioids, or dexmedetomidine may reduce preoperative anxiety and decrease the risk of subsequent subendocardial ischemia secondary to tachycardia.

What induction technique is most appropriate for this child?

Although induction of anesthesia may be achieved with either inhalational agents, IV agents, or a combination of both, the method chosen should allow preservation of preload, afterload, and contractility with a normal-to-low heart rate. Hypotension, myocardial ischemia, and subsequent cardiac arrest may occur even with low incremental dosing of inhalational agents. A titrated IV induction offers optimal control of hemodynamics. Options for a titrated IV induction include etomidate, fentanyl, midazolam,

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dexmedetomidine, and ketamine. In this case, with an emergent surgery due to trauma, a rapid sequence induction utilizing narcotic along with etomidate and a rapid-onset neuromuscular blocking agent would be most appropriate. The use of vecuronium may be preferable due to the sympathomimetic effects of rocuronium. Administration of a preinduction IV fluid bolus of 20 mL/kg isotonic crystalloid, guided by heart rate response, pulse pressure variability on the pulse oximeter, or inferior vena cava collapsibility on point of care ultrasound, should be considered prior to induction to maintain normovolemia and prevent exaggerated effects on SVR. Up to three 20 mL/kg fluid boluses may be administered prior to vasopressor initiation.

A balanced anesthetic technique combining volatile anesthetic and IV agents for maintenance of anesthesia is appropriate as long as SVR is maintained and tachycardia is prevented. ST changes on ECG may herald subendocardial ischemia, and afterload should be increased with agents that increase SVR such as phenylephrine or vasopressin. Infusions may be utilized if necessary to maintain SVR. Phenylephrine may be the vasopressor of choice, as it not only improves coronary perfusion by increasing SVR but also improves LV filling dynamics by possibly increasing left atrial pressure through reflex bradycardia. Tachycardia without hypotension should also be treated aggressively to improve early-diastolic filling of the left ventricle, ideally with short-acting $ß_1$ agents.

What additional monitoring may be necessary? Is a preinduction arterial line appropriate prior to emergent surgery?

This child has moderate-to-severe AS with a peak velocity of 3.9 m/s and a mean gradient of 40 mm Hg with preserved myocardial function. This child is also presenting for two invasive procedures that may both have a propensity for significant blood loss and fluid shifts: an exploratory laparotomy and an intramedullary pinning of a femur fracture. Invasive monitoring, including an arterial line for hemodynamic monitoring and volume assessment via pulse pressure variation will be necessary not only for the procedure but also for appropriate monitoring of moderate-to-severe AS. Although a preinduction arterial line may be ideal to monitor hypotension during induction of anesthesia, placement of an awake arterial line (even with local anesthetic infiltration, anxiolytic, and ultrasound guidance) may elicit a detrimental tachycardic response in a child who is already tachycardic. A central venous line should be placed to monitor fluid status with central venous pressure trends and for vasopressor administration, given the expected blood loss and fluid shifts with the planned procedures. Although transesophageal echocardiography is an invaluable tool to monitor wall motion abnormalities, intracardiac volume, valvular and myocardial function, the limited experience of many providers may limit this modality intraoperatively. However, limited point of care ultrasound with TTE may allow rapid assessment of intracardiac volume and myocardial function with myocardial collapse.

What perioperative pain management strategies are appropriate?

Perioperative pain management is not only critically important for functional recovery, but also to alleviate pain-associated tachycardia that is detrimental to LV early-diastolic filling in patients with AS. Although neuraxial anesthesia may provide both sensory and motor blockade for this child for both the exploratory laparotomy and the intramedullary rod placement, it should be used judiciously in a child with moderate-to-severe AS. Several case reports describe the successful use of neuraxial anesthesia in patients with AS, however sympathetic blockade decreasing SVR may produce exaggerated hypotension secondary to a compromised compensatory increase in cardiac output from the fixed outflow tract obstruction, resulting in subsequent myocardial ischemia. A peripheral nerve block for perioperative pain may offer less risk of hypotension, particularly for surgical procedures where expected blood loss and fluid shifts are expected. A truncal nerve block with an indwelling catheter, such as a quadratus lumborum or transversus abdominal block, along with a femoral nerve block and an indwelling catheter would provide coverage perioperatively for the exploratory laparotomy and the femoral intramedullary rod pinning. Postoperative mechanical ventilation should be considered in children who require significant intraoperative cardiopulmonary support, including significant changes in positive pressure ventilation, vasopressor support, or ongoing fluid or blood product resuscitation. In this instance IV analgesia with opioids, α-agonists, or ketamine would be appropriate.

What is the appropriate venue for postoperative recovery?

Decisions regarding postoperative extubation will depend on the length of surgery, blood loss and intraoperative fluid shifts, and hemodynamic stability during the procedure. If vasopressor support has been required during surgery, postoperative care in an intensive care unit should be considered and arranged. Assuming the child is at cardiopulmonary baseline, consideration can be given to

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extubation in the operating room with postoperative care in the post-anesthesia care unit.

Suggested Reading

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