

# d-Transposition of the Great Arteries (Arterial Switch)

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## Case Scenario

A 10-year-old male presents for elective upper endoscopy and colonoscopy. He has a 2-week history of persistent vomiting and weight loss with new-onset diarrhea. He has a history of dextro-transposition of the great arteries and underwent an arterial switch operation shortly after birth. He has been followed since by his cardiologist for residual pulmonic stenosis. He and his family report he is able to participate in sports but has begun to tire more quickly than his peers; he has felt considerably more fatigued since the start of the vomiting and diarrhea. His current vital signs include heart rate 100 beats/minute, respiratory rate 18 breaths/minute, and SpO<sub>2</sub> 99% on room air. His abdomen is soft, mildly distended, and nontender to palpation. He has mild eczema on his extremities.

Transthoracic echocardiography 1 week earlier showed the following:

- Peak velocity of 3.5 m/s and peak gradient of 50 mm Hg across the pulmonary valve
- Right ventricular hypertrophy
- Qualitatively normal biventricular function

## Key Objectives

- Understand the anatomy of dextro-transposition of the great arteries.
- Describe the arterial switch operation and identify potential sequelae.
- Describe the preoperative assessment and anesthetic management of a patient with repaired dextro-transposition.
- Understand considerations regarding performing procedures in remote or offsite locations in patients with repaired congenital heart disease.

## Pathophysiology

### What is transposition of the great arteries?

Transposition of the great arteries (TGA) is most accurately an umbrella term for all congenital cardiac lesions

whereby the aorta arises from the right ventricle (RV) and the pulmonary artery (PA) arises from the left ventricle (LV). This is termed ventriculoarterial discordance. Most typically the term TGA is used to denote dextro (d)-TGA, whereas other forms of TGA are called by their specific names, as discussed in the text that follows.

### What is d-TGA?

d-transposition of the great arteries is one of the most common cyanotic congenital heart lesions, characterized by **ventriculoarterial discordance**, the origination of the great vessels from the incorrect ventricles, in a heart with otherwise normal connections. Deoxygenated systemic blood flows from the vena cavae to the right atrium (RA), on to the RV, and then to the aorta. Oxygenated pulmonary venous blood enters the left atrium (LA), flows to the LV, and exits the PA. This anatomy results in a parallel circulation. (See Figure 21.1.) Fetal shunts between the parallel circulations, including a patent foramen ovale (PFO) and patent ductus arteriosus (PDA), are required to provide mixing between the separate circulations and thus some degree of oxygen delivery to the tissues. Without intervention, d-TGA is uniformly fatal in infancy, with a 30% mortality in the first week of life and 50% in the first month. Transposition represents 5% of all congenital heart lesions (32 per 100,000 live births) with a 2:1 predilection for males.

### What are other types of TGA and how are they distinct from d-TGA?

d-transposition is a morphologically distinct entity wherein the “d” denotes dextroposition of the bulbovertricular loop embryologically, resulting in atrioventricular concordance, ventriculoarterial discordance, and a typically anterior and rightward position of the aortic valve.

Congenitally corrected, or levo-TGA (l-TGA), is also characterized by ventriculoarterial discordance; however, the addition of atrioventricular discordance results in a series circulation, with the left-sided morphologic RV

responsible for systemic blood flow. (See Chapter 23.) Because l-TGA results in a series blood flow, it is a separate entity both anatomically and physiologically, as well as embryologically. Other congenital cardiac lesions that also have transposed great vessels include those with associated mitral or tricuspid atresia, double inlet left or right ventricle, and some forms of double outlet RV.

Clinical Pearl

***D-transposition** is characterized by ventriculoarterial discordance, the origination of the great vessels from the incorrect ventricles, in a heart with otherwise normal connections. This anatomy results in a **parallel circulation**. **L-TGA** is also characterized by ventriculoarterial discordance; however, the addition of atrioventricular discordance results in a **series circulation**, with the left-sided morphologic RV responsible for systemic blood flow.*

mixing of oxygenated and deoxygenated blood between the separate circulations. (See Figure 21.2.) The ductus arteriosus is almost always patent immediately after birth, and intravenous prostaglandin E<sub>1</sub> (PGE<sub>1</sub>) infusion is immediately initiated to maintain ductal patency. The PDA alone is unable to provide sufficient mixing, which requires robust bidirectional flow. As a result, patency at the atrial level (ASD and/or PFO) is necessary to provide countercurrent flow in equal volume to the flow across the PDA. A countercurrent flow across two shunts allows the possibility of sufficient mixing in the short term. Although the foramen ovale typically remains open due to higher pressures in the RA, it can be closed or restrictive. In these cases, emergent balloon atrial septostomy (BAS) is undertaken, generally in the cardiac catheterization laboratory, to enlarge the communication between the atria and allow adequate mixing. (See Figure 21.3.)

What is the newborn physiology of d-TGA?

D-transposition is characterized by a parallel circulation. There are three primary d-TGA subtypes that influence a newborn’s clinical presentation. (See Table 21.1.)

In infants with *TGA-IVS*, after birth the preexisting fetal shunts (PDA and PFO) become critical sources for

Table 21.1 d-TGA Subtypes and Incidences

Subtype	Incidence
d-TGA with intact ventricular septum (TGA-IVS)	60%–70%
d-TGA with ventricular septal defect (TGA-VSD)	25%–45%
d-TGA with VSD and left ventricular outflow tract obstruction (TGA-LVOTO)	5%–25%

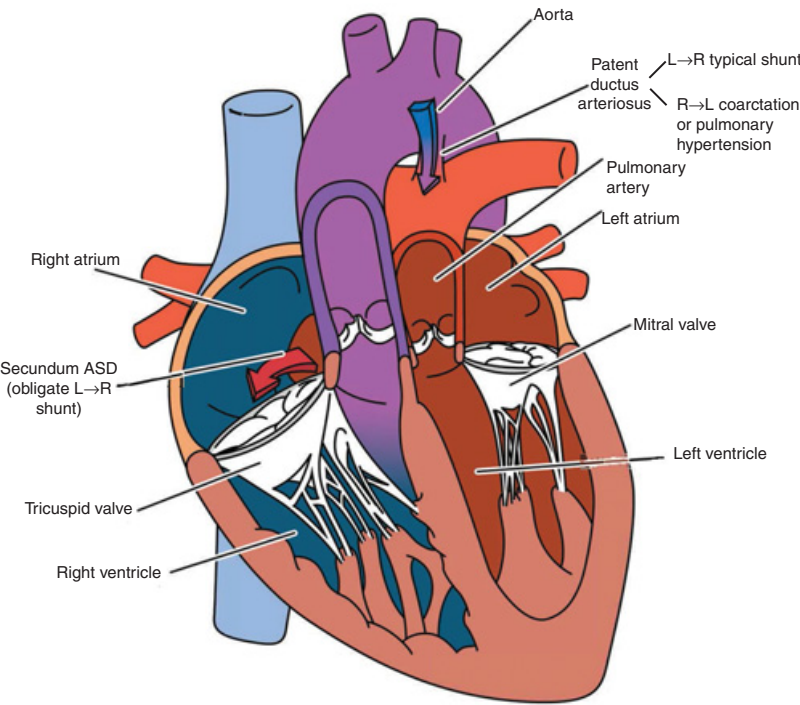
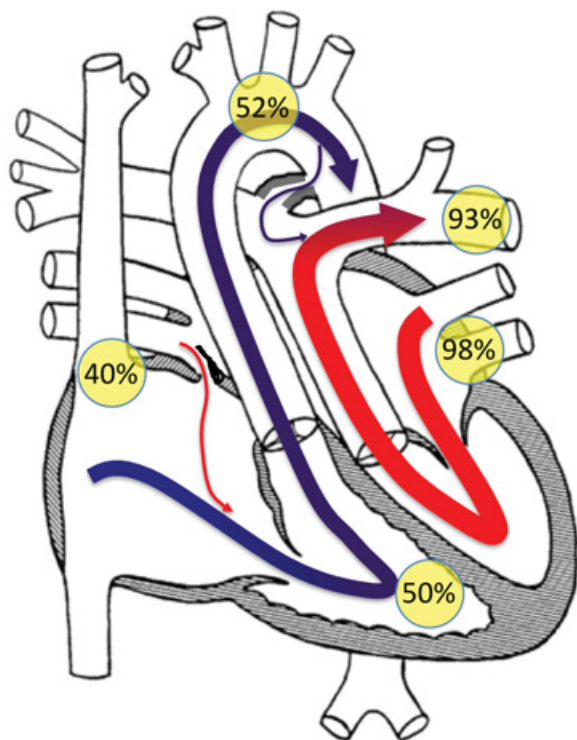
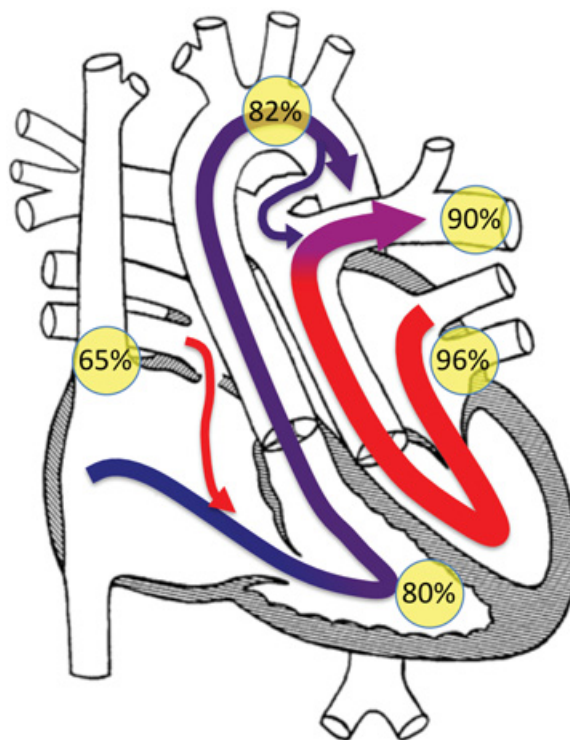


Figure 21.1 Unrepaired d-TGA with intact ventricular septum. Drawing by Ryan Moore, MD, and Matt Nelson.



**Figure 21.2** d-TGA with intact ventricular septum. The ductus arteriosus and foramen ovale are restrictive, leading to poor mixing and severe hypoxemia. Courtesy of Greg Latham, MD.



**Figure 21.3** d-TGA-IVS with improved mixing after initiation of PGE<sub>1</sub> and a balloon atrial septostomy. Courtesy of Greg Latham, MD.

### Clinical Pearl

*The most common form of d-TGA, TGA-IVS, relies on shunting at the ductal and atrial level to adequately mix the parallel circulations. A prostaglandin E<sub>1</sub> infusion is usually required to maintain ductal patency, and a BAS may be required to create or maintain an atrial shunt.*

In patients with *TGA-VSD*, there are usually, but not always, ample saturations due to robust mixing at three levels: arterial (PDA), atrial (PFO), and ventricular (VSD). Therefore, these children may not be cyanotic at birth. In these cases, a trial of discontinuing PGE<sub>1</sub> is warranted to reduce the risk of excessive pulmonary blood flow (PBF) as pulmonary vascular resistance (PVR) decreases, therefore reducing the risk of heart failure.

Neonates with *TGA-LVOTO* may have adequate mixing from the VSD; however, the amount of PBF will be dependent on the degree of subpulmonic LVOTO, which may range from a mild to severe reduction in PBF, thus resulting in variable degrees of hypoxemia.

As PVR decreases after birth, children with either TGA-IVS or TGA-VSD will experience a gradual increase

in blood flow into the pulmonary side of the parallel circulation (pulmonary veins to LA, to LV, to PA, and back to pulmonary veins), which can lead to pulmonary congestion and heart failure.

### Clinical Pearl

*D-transposition relies on adequate mixing to prevent severe hypoxemia after birth. This generally requires patency of two out of three possible cardiac shunts (arterial level: PDA, atrial level: PFO, ventricular level: VSD) to allow circular mixing between the parallel circulations. Regardless, adequate oxygenation and perfusion may be tenuous prior to surgery.*

## What other anomalies are associated with d-TGA?

Regardless of the subtype, d-TGA may also be associated with aberrant coronary anatomy and/or ostial abnormalities, as well as accompanying aortic arch hypoplasia, coarctation, or interruption. Unlike most other complex congenital heart lesions, d-TGA usually appears in isolation without extracardiac anomalies or genetic syndromes.

## How and when is d-TGA repaired?

Because transposition physiology has been demonstrated to cause variable degrees of brain matter changes, early surgical repair is preferred. Days 3–5 of life are considered ideal, which strikes an appropriate balance, allowing reduction of postnatal PVR but occurring prior to the deconditioning of the LV that occurs as a result of the decreased afterload on the LV as it perfuses the pulmonary circulation.

Surgical management of d-TGA has changed over the past 40 years. Initially, d-TGA was repaired via a Mustard or Senning atrial switch operation. (See Chapter 22.) Today, the arterial switch operation (ASO) has become the mainstay of modern surgical management due to excellent outcomes, with more than 90%–95% of patients living into adulthood. The ASO results in anatomic correction (see Figure 21.4), whereby the great arteries are switched to create ventriculoarterial concordance, with reanastomosis of the coronary arteries to the neo-aorta. The ductus arteriosus, if patent, and any other residual septal shunts are closed.

### Clinical Pearl

*The arterial switch operation is the preferred repair for d-TGA and is appropriate in the majority of patients, with excellent surgical outcomes and more than 90% of patients surviving into adulthood.*

## Given that multiple types of d-TGA exist, can the ASO be used to repair all of them?

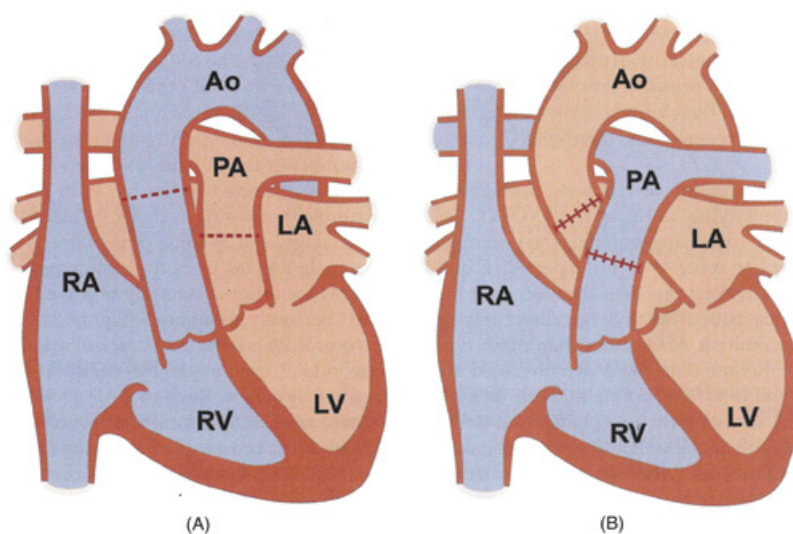
For the majority of d-TGA patients, the ASO is the appropriate surgical therapy. Patients with accompanying LVOTO may require more complex surgeries, as noted in the text that follows. (See Table 21.2.)

## What happens to patients with d-TGA and significant LVOTO?

It is important to understand that while performing the ASO in neonates with TGA-LVOTO will correct the ventriculoarterial discordance, it will also exchange what was pulmonic stenosis for neo-aortic stenosis. When the existing LVOTO is incompatible with sustaining systemic outflow, one of several surgical options may be chosen: the Rastelli, Nikaidoh, or réparation à l'étage ventriculaire (REV). Each of these three procedures has advantages and disadvantages, and the appropriate choice depends in part on the specific anatomy.

### Clinical Pearl

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**Figure 21.4** The arterial switch operation (ASO).

Schematic representation of d-transposition of the great arteries anatomy before (A) and after (B) the ASO. AO, aorta; LA, left atrium; LV, left ventricle; PA, pulmonary artery; RA, right atrium; RV, right ventricle. From McEwan A. and Manolis M. *Anesthesia for transposition of the great arteries*. In Andropoulos D. B., Stayer S., Mossad E. B., et al., eds. *Anesthesia for Congenital Heart Surgery*, 3rd ed. John Wiley & Sons, 2015: 542–66. With permission.

**Table 21.2** Surgical Repairs for d-TGA Variants

Lesion	Surgical Repair
d-TGA with intact ventricular septum	ASO
d-TGA with intact ventricular septum and interrupted arch or coarctation	ASO with arch/coarctation repair
d-TGA with VSD	ASO with VSD closure
d-TGA with VSD and significant PS	Rastelli, REV, or Nikaidoh procedure

ASO, arterial switch operation; PS, pulmonary stenosis; VSD, ventricular septal defect.

The **Rastelli procedure** leaves the great vessels and semi-lunar valves in place and a VSD patch is used as a baffle to direct LV output across the VSD and to the anterior-rightward aortic valve that overlies the RV. Because the Rastelli baffle invades the RV outflow tract and precludes RV outflow to the leftward PA, the pulmonary valve is oversewn, and an external valved RV-to-PA conduit is placed. A disadvantage is that the synthetic external conduit obligates future reinterventions, including surgical conduit changes, percutaneous stenting of the conduit, and/or percutaneous transcatheter pulmonary valve replacements. Recurrent LVOTO is also a risk with the long LV outflow baffle.

The **REV procedure** is very similar to the Rastelli procedure but includes resection of the conal septum and directly connects the native PA to the RV, allowing for growth and eliminating the need for a prosthetic conduit. However, obligate pulmonary regurgitation will also eventually require reinterventions.

The **Nikaidoh procedure** is characterized by translocation of not only the great vessels but the aortic valve as well. The aortic root is harvested from the RV, and the LVOTO is relieved by dividing the posteriorly positioned pulmonary valve and outlet septum. The aortic valve, aorta, and coronary arteries are reimplanted above the reconstructed LV outflow tract, and the VSD and outlet septum are closed. Anastomosis of the main PA to the RV is accomplished via valved conduit or anastomosis of native (non-valved) PA-to-RV with patch augmentation. The Nikaidoh results in excellent outcomes with regard to the aortic valve and LV outflow tract, but reinterventions will be required for pulmonary regurgitation, as discussed with the Rastelli.

#### Clinical Pearl

*A small number of children with d-TGA and significant LVOTO are not suitable for an ASO repair and instead require surgical repair with a Rastelli, Nikaidoh, or réparation à l'étage ventriculaire (REV) procedure.*

## What are the long-term issues, if any, in patients with repaired d-TGA?

Children with TGA-LVOTO who require repair with a Rastelli, REV, or Nikaidoh procedure will have some degree of residual disease, as previously discussed. However, the remainder of this discussion will concern children post-ASO.

Although uncommon, early mortality after the ASO is caused in large part by acute coronary events. Beyond 3 months post-ASO, coronary complications are relatively rare, despite angiographic or radiographic evidence of some degree of coronary stenosis or “insufficiency” in 3%–11% of patients. The rate of clinically symptomatic coronary pathology, however, is low, and there is no agreed upon consensus for standardized coronary surveillance as of 2018.

Despite refinements in surgical techniques, progressive right ventricular outflow tract obstruction (RVOTO) remains a relatively common long-term morbidity. The obstruction can be subvalvular, valvular, or supravulvular in nature or exist as multilevel obstruction. The precise cause of RVOTO in most patients is not clear. Classically, the ASO is accomplished utilizing the Lecompte maneuver, which involves moving the main and branch pulmonary arteries anterior to the aorta while the aorta is transected. This results in the pulmonary trunk and arteries laying overtop the aorta in a nonspiral fashion, and while physiologically corrected, the lack of spiraling of the great vessels results in a different configuration from an embryologically normal heart. The Lecompte maneuver is done to prevent posterior compression of the PAs from the neo-aortic root. However, it is postulated that larger aortic roots may actually increase stretching of the PAs, or indeed that the lack of spiraling changes flow dynamics, of which pulmonary artery stenosis becomes a long-term consequence.

#### Clinical Pearl

*The most common cause of late reintervention post-ASO is to address RVOTO, often at the level of the branch pulmonary arteries.*

Less common long-term morbidities include LVOTO, neo-aortic root dilation, neo-aortic valve regurgitation, compression of the tracheobronchial tree, and primary pulmonary hypertension. Arrhythmias and bundle branch blocks are uncommon after ASO, as this surgery usually avoids significant manipulation of the nodal and conducting cardiac tissue. However, closure of large VSDs or performance of any of the other non-ASO techniques (Senning, Mustard, REV, Nikaidoh, Rastelli) portend a higher risk of long-term arrhythmias.

## What type of follow-up do these patients require after repair, and at what interval?

In addition to standard postoperative surgical follow-up, patients should have outpatient cardiology care at an interval recommended by the cardiologist. There are no current guidelines or recommendations for routine testing or follow-up in this patient population. The frequency of surveillance testing will generally be dependent on the presence of residual cardiac disease and could include annual electrocardiogram (ECG) and echocardiogram, with more dedicated imaging, such as cardiac magnetic resonance imaging, computed tomography angiography, or cardiac catheterization, based on the presence of symptoms or historical issues concerning for coronary, valvular, or overall cardiac function. On the other end of the spectrum, children with no evidence of residual cardiac disease may have infrequent cardiology visits.

The question of the maximal allowable time since last imaging or last cardiology visit prior to anesthesia for an elective procedure is difficult to define. In short, the child's family should be compliant with follow-up as recommended by his or her cardiologist. Additionally, if the child has new or progressive symptoms that are concerning for cardiac disease, consideration must be given to a cardiology visit or consultation prior to anesthesia, particularly for elective procedures.

## Anesthetic Implications

### What specific cardiac information should the preoperative assessment include?

In addition to the routine preoperative history and physical examination, the most recent echocardiogram should be reviewed to assess any residual right or left ventricular outflow tract obstruction, wall motion abnormalities, and biventricular function. An ECG can be reviewed for arrhythmias or the rare occurrence of ischemia, and records from the most recent cardiology visit should be reviewed when available. An assessment of current functional status can help gauge the degree of cardiopulmonary reserve and guide whether repeat imaging or further optimization of the child is necessary prior to elective surgery.

### What are the signs, symptoms, and diagnostic criteria of pulmonary stenosis?

The diagnosis of pulmonary stenosis (PS) is based on echocardiographic Doppler measurements across the stenotic pulmonary valve or other level of outflow tract. Clinically, the child may be noted to have decreased

exercise tolerance, shortness of breath and fatigue, chest pain, or poor growth. Findings on ECG may demonstrate RV hypertrophy with right axis deviation, a dominant R wave in V1, and deep S waves in V5 and V6.

Pulmonary stenosis is further characterized as mild, moderate, or severe, based on peak gradients of <40 mm Hg, 40–60 mm Hg, or >60 mm Hg, respectively. While these criteria generally apply to stenosis of the valve itself, the region of narrowing can occur anywhere along the RVOT, including superiorly to the branch pulmonary arteries. There is, however, a lack of agreement regarding optimal techniques to quantify and standardize data in these non-valvular regions. As such, clinical decision making for a child with possible multilevel RVOTO requires ongoing surveillance for changes and the development of symptoms over time to appropriately assess the need for intervention.

A peak gradient of 50 mm Hg or the presence of clinical symptoms is often used as a threshold for treatment of isolated PS. However, the treatment threshold is certainly multifactorial in a child with the development of postoperative PS that may exist at multiple levels and may require a more extensive surgical revision. Thus, the presence or severity of clinical symptoms, progression of RV hypertrophy, and rate of progression of PS will all be factors in assessing the need or timing for reintervention.

#### Clinical Pearl

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### How should patients and parents be counselled regarding their anesthetic risk?

Reporting from multiple centers and databases has implicated congenital and acquired cardiac disease as primary risk factors for anesthetic morbidity and mortality in children. However, congenital heart disease (CHD) clearly represents a wide range of disease types and surgical repairs. Subgroup analysis demonstrates that those patients with single ventricle physiology, obstructive left heart lesions, pulmonary hypertension, or cardiomyopathy appear to be most vulnerable. Although no long-term data are specifically reported for anesthetic risk in children or adults post-ASO, it is intuitive that children and adults with repaired CHD who are fit and have no residual defects are at low risk, with increasing risk correlating with the presence and

severity of residual or comorbid disease. Ultimately, the child's American Society of Anesthesiologists physical status remains a strong predictor for the risk of anesthetic complications, emphasizing the importance of each patient's preoperative assessment.

#### Clinical Pearl

*Post-ASO patients with no residual defects are at low risk for anesthetic complications, with increasing risk directly correlating to the presence and severity of comorbid disease.*

## What are the general anesthetic considerations for patients with PS?

Outflow tract obstructions cause an increase in ventricular afterload. The impact to the underlying ventricle, in this case the RV, depends on the severity and the rate of progression of stenosis. Slowly progressive PS results in gradual RV hypertrophy to compensate for the increased afterload, and the RV is typically well adapted to sustain physiologic demands, including exercise, despite the increased afterload. The child discussed in this chapter appears to fall into this category, with moderate to severe PS but preserved RV function and the ability to continue athletics, albeit with increased fatigue. With continued significant stenosis, progressive RV hypertrophy is eventually accompanied by impaired relaxation, a tenuous oxygen supply-demand relationship, and eventual risk of RV ischemia and failure. On the other hand, if a child has acute onset of severe PS, the risk of acute RV failure exists given that the RV has not had time to develop compensatory mechanisms to adapt to the severely increased afterload.

Anesthetic risk in the setting of PS, therefore, depends on the severity of PS and the underlying RV function, and many would consider RV function to directly correlate with risk of morbidity and mortality under anesthesia. Important goals during all phases of the anesthetic include normovolemia to maintain adequate preload, maintenance of normal sinus rhythm to optimize the atrial contribution to RV filling, and avoidance of tachycardia for optimization of both RV filling time and RV ejection across the stenosis. Special caution should be exercised during induction and emergence, as these times are more commonly fraught with hemodynamic instability.

#### Clinical Pearl

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*of normovolemia for adequate preload, maintenance of normal sinus rhythm to optimize the atrial contribution to RV filling, and avoidance of tachycardia for optimization of both RV filling time and RV ejection across the stenosis.*

## Should additional monitors or invasive access be placed?

The use of a 5-lead ECG increases myocardial ischemia detection, is noninvasive, and as such is easily warranted for any patient at risk for ischemia under anesthesia. The need for and use of invasive monitors is dictated by both the surgical procedure and the presence of patient comorbidities. This child underwent a straightforward ASO repair and presents with PS, preserved RV function, and the ability to participate in sports. Given the short, non-invasive nature of the EGD and colonoscopy, invasive monitoring would not be warranted.

## Are there any specific considerations for induction of anesthesia?

A patient with d-TGA post-ASO has normal anatomic circulation. Increased detection of myocardial ischemia can be obtained with a 5-lead ECG, but other specific considerations regarding the induction of anesthesia will depend on the presence of any residual cardiac disease, including outflow tract obstruction(s).

In this patient, the severity of his PS is the concerning cardiac manifestation, and anesthetic goals should be tailored as discussed earlier. Preload, afterload, and contractility are the key ingredients to maintain RV performance. Preload is managed with intravenous (IV) hydration. Further increases in RV afterload should be prevented, which means maintaining a low PVR via the normal mechanisms under anesthesia. Tachycardia may be reduced with administration of anxiolytic premedication if indicated and preoperative fluid hydration. Slow titration of anesthetic induction agents allows for close monitoring of hemodynamics and treatment if perfusion and oxygen delivery are compromised. The 10-year-old in this scenario will likely behave quite normally with a gentle inhalation or propofol-based induction. The child's history of vomiting must also be factored into the induction plan.

## If the patient aspirated during induction, what are the specific risks in a child with PS?

In this case, the risk of aspiration is additive to the risk of PS. Aspiration of a large volume or particulate matter can cause obstruction of both the large and small airways,

leading to atelectasis, hypoxemia, and hypercapnia. Each of these acutely increases PVR and risks abruptly increasing RV afterload and thus inducing RV strain or failure. Systemic hypoxemia further risks inadequate myocardial oxygen delivery, particularly in those with RV hypertrophy and elevated RV diastolic pressure (wall tension). While healthy children can be quite ill after a large-volume aspiration event, those with preexisting cardiopulmonary disease will likely fare worse.

## Should positive pressure ventilation be avoided in this type of patient?

Positive pressure ventilation (PPV) decreases preload and increases RV afterload, which should be considered in those with existing RVOTO or RV dysfunction. On the other hand, hypoventilation with resultant hypercapnia and hypoxemia will increase PVR and also risk increasing the overall RV afterload. Thus, a reasonable approach is to allow spontaneous ventilation when appropriate for the procedure and when the patient is able to maintain adequate oxygenation or ventilation; if neither of these factors are met, it may be preferable to control ventilation. While the impact of PPV is a prudent question to consider in children with CHD, the impact is generally not a consideration for those with repaired defects, including repaired d-TGA, without residual cardiac abnormalities. In this case, the anesthesiologist must weigh the risks and benefits present, also considering the child's history of recurrent vomiting.

## Is it appropriate to perform this case in a remote location?

Although the ASO for children with d-TGA provides anatomic correction with typically normal to near-normal physiologic function, there can be residual dysfunction as in this child. An understanding of the patient's current cardiac function, as well as the particular geographic constraints and proximity of the gastrointestinal (GI) suite to assistance, must be considered before agreeing to perform an anesthetic in a GI suite that may or may not be in a location remote from operative services. The appropriateness of care in a free-standing GI suite (not within a hospital) for patients with residual cardiac disease must be weighed with even greater caution.

## Is outpatient surgery appropriate for this patient?

Overall, the appropriateness of outpatient surgery always depends on the presence of comorbidities, procedure type, need for postoperative pain management or physiologic

support, and social issues at home. In this case, the 10-year-old boy has preserved RV function and is able to exercise, although with early fatigue. Nothing in this history is an absolute contraindication to outpatient care, especially considering the brevity and noninvasiveness of the procedure and lack of pain management requirements afterwards. A conservative but reasonable approach is to require a longer recovery period post-procedure, and if the anesthetic is uncomplicated and the child has "returned to baseline" without worrisome signs or symptoms, the child may be discharged home. Those with repaired d-TGA without residual cardiac comorbidities are certainly candidates for outpatient surgery, while those with signs of RV failure may require admission or prolonged observation post-procedure.

### Clinical Pearl

*A conservative but reasonable approach is to require a longer recovery period post-procedure, and if the anesthetic is uncomplicated and the child has "returned to baseline" without worrisome signs or symptoms, the child may be discharged home.*

## Suggested Reading

- Angeli E., Raisy O., Bonnet D., et al. Late reoperations after neonatal arterial switch operation for transposition of the great arteries. *Eur J Cardiothorac Surg* 2008; **34**: 32–6.
- Cohen M. S., Eidem B. W., Cetta F., et al. Multimodality imaging guidelines of patients with transposition of the great arteries from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. *J Am Soc Echocardiogr* 2016; **29**: 571–621.
- Gottlieb E. A. and Andropoulos D. B. Anesthesia for the patient with congenital heart disease presenting for noncardiac surgery. *Curr Opin Anesth* 2013; **26**: 318–26.
- Latham G. L., Joffe D. C., Eisses M. J., et al. Anesthetic considerations and management of transposition of the great arteries. *Semin Cardiothorac Vasc Anesth* 2015; **19**: 233–43.
- Morgan C. T., Mertens L., Grotenhuis H., et al. Understanding the mechanism for branch pulmonary artery stenosis after the arterial switch operation for transposition of the great arteries. *Eur Heart J Cardiovasc Imaging* 2017; **18**: 180–5.
- Warnes C. A., Williams R. G., Bashore T. M., et al. ACC/AHA 2008 Guidelines for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines (Writing Committee to develop guidelines on the management of adults with congenital heart disease). *Circulation* 2008; **118**: 714–833.